Migrant Labor and Sexually Transmitted Disease: AIDS in Africa*

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Acquired immune deficiency syndrome (AIDS) is worldwide, but the clinical and epidemiological pattern of the disease in Africa is different from that in developed areas. "Type 1 AIDS" occurs in industrialized North America and Europe; it has a distinctive sex ratio (16:1) and risk pattern of IV drug use and sexual practices. "Type 2 AIDS" occurs in Third World countries, particularly in eastern, southern, and central Africa. It is characterized by an entirely different sex ratio (1:1) and by distinctively different risk patterns. Both epidemics are caused by the HIV-1 virus. The key concept for understanding the origins of the differences between Type 1 and Type 2 AIDS is the migratory labor system in eastern, central, and southern Africa. This system causes long absences, increased family breakdown, and increased numbers of sexual partners. Historically the organization of this labor market has created a population which suffers from epidemics of sexually transmitted diseases. These historical patterns are presented as evidence for the contemporary transmission of AIDS. When contemporary AIDS and HIV-1 seropositivity prevalence data are examined, a systematic temporal and geographic pattern emerges for the AIDS epidemic in Africa. Despite a paucity of good data, the prevalence data from eastern, central, and southern Africa support the thesis of migrant labor's role in the transmission of AIDS.

The following paper integrates four areas of history, research, methodology, and theory. First, it incorporates a scientific understanding of the HIV-1 virus and the action of this virus in the creation of acquired immune deficiency syndrome (AIDS). The modes and methods of transmission in both Africa and the United States are the basis of understanding in the following discussion.

Second, in combination with an understanding of the biology of the HIV-1 virus and of the AIDS that results, this paper uses an analysis of epidemiology known as "historical materialist epidemiology." Accordingly it assumes the primacy of economic life, economic patterns, and economic development. This economic foundation sets the basic parameters of health and illness in society. This method of approach is derived from work done by such early thinkers as Frederick Engels, Rudolf Virchow, and Salvador Allende (Waitzkin 1983, pp. 65–85). This early work was followed in the 1970s and 1980s by a great deal of research and writing on disease and health. The historical materialist approach was taken in a series of articles concerning coronary heart disease and hypertension (Eyer 1975; Eyer and Sterling 1977; Schall and Kern 1986), cancer (S. Epstein 1976, 1978), suicide (Hopper and Guttmacher 1979; Waldron and Eyer 1975), occupational health and safety (Chavkin 1984; Navarro 1982; Navarro and Berman 1983; B. Smith 1981), women's longer life span as compared with men's (Waldron 1986), and the expansion and contraction of economic cycles and mortality rates (Eyer 1977; Waldron and Eyer 1975). This approach may be described as a method that "relates patterns of death and disease to the political, economic, and social structures of society" (Waitzkin 1978, p. 272). Special applications have been made to Third World

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societies, relating underdevelopment, dependency theory, and health (Doyal 1981; Navarro 1974; Turshen 1977).

Third, this paper's assertions are based on a study of the historic, social, and economic development of eastern, central, and southern Africa, the Africa of migrant labor and the labor reserve. The development process has been described by dependency theory; therefore this paper is grounded in dependency theory (Amin 1976; Foster 1986; Frank 1967; Leys 1975; Rodney 1982; Saul and Arrighi 1973). World systems theory has elaborated on dependency theory; the work in this paper also complements the studies in world systems theory on the household in the world economy (Friedman 1984; Martin 1984; J. Smith 1984; Wallerstein 1984; Wallerstein and Martin 1979). The structure of the household, its size, and its varying composition, as well as degree of proletarianization, wage and nonwage work, and migrancy, are integrated into the following analysis with considerations of health, well-being, and disease. These household structures are defined by their place in the world system; consequently, it is argued in the subsequent presentation, health and disease are defined by place—core or periphery—in that system.

Finally, an understanding of the medical and population geography of central, eastern, and southern Africa is integrated into the thesis advanced in this paper (see Hall and Langlands 1975; Kabera 1982; Langlands 1975; Meyer 1988; Prothero 1977, 1983; White 1978). The population's geographical movements in these regions of Africa, often termed oscillations rather than migrations, give rise to the characteristic concentrations of labor around plantations, mines, and other capital investments. These movements, interacting with historical development patterns, give rise to the labor reserve, the rural farming area which often is in decline because of the patterns established by dependency and migrant labor (P. Epstein and Packard 1987). This pattern of movement and labor market affects Africans' family and sexual patterns (see Murray 1977, 1980, 1981); as I argue here, it creates a population that is susceptible to sexually transmitted disease (STD), AIDS in particular. A study of past spatial patterns—the medical geography of STDs—will reveal the historical relationship of STDs to migrant labor. This pattern, it is argued, has been repeated in the AIDS epidemic of the 1970s and 1980s.

AFRICA AND AIDS

Acquired immune deficiency syndrome is epidemic in the United States, but it is more than an epidemic. AIDS is a pandemic: it is *worldwide* in distribution and began almost simultaneously in a number of areas in the world. World Health Organization (WHO) officials estimate that "over 250,000 cases of AIDS have already occurred, that between five and 10 million people worldwide are infected with the AIDS virus and that within the next five years about one million new AIDS cases can be expected" (Mann, Chin, Piot, and Quinn 1988, p. 82). By March 1, 1988, there were 81,433 reported AIDS cases in 133 countries (WHO 1988).

AIDS has appeared in the Pacific Oceania complex of islands and Australia, in South and Central America, and, although delayed, in Asia. Nowhere, however, was its appearance more ominous than in Africa (Andersen, May, and McLean 1988).

It is extremely difficult to judge the exact extent of AIDS in Africa, either geographically or in the population (Konotey-Ahulu 1987, pp. 206–207). Rwanda, Burundi, the Congo, Zimbabwe, Botswana, Zaire, Zambia, Kenya, and Uganda seem to be particularly afflicted (Mann and Chin 1988; Mann et al. 1988). These countries form what has been called "the AIDS belt" across eastern, central, and southern Africa.

AIDS was first discovered to be present in Africa when it was diagnosed in upper-class Africans seeking treatment in European hospitals. The first African cases were diagnosed in Europe shortly after the first diagnosis of AIDS occurred in the United States (Quinn, Mann, Curran, and Piot 1986). African AIDS, however, has two distinctive aspects in relation to the AIDS epidemic in the United States.

First, the sex ratio of those who have AIDS in Africa is approximately 1:1, an equal number of females and males (Biggar 1986; Brunet and Ancelle 1985; Curran, Morgan, Hardy, Jaffe, Darrow, and Dowdle 1985; Mann and Chin 1988; Mann et al. 1988; Mann, Francis, Davachi, et al. 1986; Mann, Francis, Quinn, et al. 1986; Quinn 1987; Quinn et al. 1986). This ratio is in sharp

contrast to the 16:1 ratio of males to females in Europe and the United States (Quinn et al. 1986). The disparity is related to the different risk patterns associated with African and with North American AIDS cases. In Africa neither homosexuality, homosexual sexual patterns, nor IV drug use are associated with AIDS or found as a means of transmission for the AIDS virus (Biggar 1987; Brunet and Ancelle 1985; Carswell 1987; Clumeck et al. 1985; Kreiss 1986; Moodie 1988; Quinn 1987).

When the illness is studied epidemiologically, primarily *sexually active heterosexuals* are seen to evidence the highest incidence of AIDS in Africa. Females who have AIDS in Africa tend to be younger than males, and often are single. There appears to be a higher incidence of AIDS among prostitutes than among African women generally (Biggar 1986; Brunet and Ancelle 1985; Quinn 1987; Quinn et al. 1986). Because women contract AIDS in Africa much more often than in North America, and because it appears that AIDS can be transmitted across the placenta from the infected mother to the fetus, there are many more children with AIDS in Africa than in the United States (Mann, Francis, Davachi, et al. 1986); in fact, children constitute almost one-third of all AIDS cases in Africa (Hilts 1987). As in the United States, AIDS in Africa appears to occur much more frequently in large cities than in the rural areas, at least at the present (although this may be a reporting bias, as we will discuss later). As in Europe and the United States, those who are discovered to have the AIDS virus frequently have a medical history of previous venereal diseases and appear to be more sexually active than those without AIDS (Quinn 1987; Quinn et al. 1986). The patterns of AIDS infection in the industrialized countries of North America and Europe is so distinctive in relation to the pattern in Africa, Haiti, and parts of Central and South America that the former is often termed "Pattern 1 AIDS" and the latter "Pattern 2 AIDS" (Mann and Chin 1988; Mann et al. 1988; Piot, Plummer, Mhalu, Lamboray, Chin, and Mann 1988).

AIDS in Africa has a second distinctive aspect. In the United States and in Europe a definite series of "opportunistic" infections occurs with AIDS, such as Kaposi's sarcoma (a skin cancer), toxoplasmosis (a parasitic disease spread by cats and chickens, which is usually harmless for those not suffering from AIDS), or a type of pneumonia. In Africa, in contrast, the opportunistic infections usually do not include any of these. Generally the infections suffered by AIDS patients in Africa involve the stomach or the digestive system, some skin diseases (in some cases Kaposi's sarcoma), tuberculosis, herpes zoster, and meningitis (Quinn et al. 1986). Thus in Uganda, AIDS is termed "slim disease" because of the wasting that occurs as a result of these infections. AIDS in Africa is epidemiologically and clinically quite distinct from AIDS in Europe or in North America. In fact, "the definition of AIDS used in the West needs to be broadened for use in Africa" (Norman 1985).

If the biological agent of the disease is the same in North America and Europe as in Africa—and research has proved that this is the case (Biggar 1986)—why do AIDS in Africa and AIDS in the United States look and act so different? Why is the same biological cause linked to such different demographic and clinical results? In short, why are there Pattern 1 and Pattern 2 AIDS epidemics? The answer lies in the social/historical environment in which the biological cause, the HIV virus, acts.

When AIDS is viewed in this way, one can advance a strong thesis that the main historical fact of African social, political, and economic reality is the situation of underdevelopment and the position of dependency in which many African nations find themselves with respect to the core capitalist countries. This relationship of underdevelopment and dependency, particularly as it affects African health, labor market organization, and rural agricultural development, has largely determined the pattern of the disease in Africa.

DEPENDENCY AND AIDS

First we will examine African historical and economic development in terms of industrialization and the development of a labor market. Then we will discuss the effect of this development on rural and agricultural areas. This background will help us to understand how these factors interact with the AIDS virus.

The pattern of industrial development in southern, eastern, and central Africa, which was followed by colonization of this area by

the European powers and has continued to the present, was based largely on a migrant labor system (Crush 1984; Davidson 1983; Doyal 1981, pp. 11–119; Freund 1984, 1988; Gugler, 1968; Gutkind and Wallerstein 1976; Karugire 1980; C. Leys 1975; R. Leys 1974; Mamdani 1976; Parpart 1983; Roberts 1979; Sathyamurthy 1986; Stichter 1985; Turshen, 1977, 1984; Wallerstein and Martin 1979). Mining, railroad work, plantation work, and primary production facilities absorbed capital investment and became enclaves of development in an immense, underdeveloped continent. Just as these industries absorbed capital, they absorbed large quantities of labor from the rural areas, concentrating great numbers of male workers. The effect on these men and on their families is described well by Lesley Doyal (1981) in *The Political Economy of Health:*

> The migrant labor system affected Africans' lives in many fundamental ways. Whatever the miseries of industrialization in Britain, it was usually possible for workers to keep their families together, but this has not been the case in third world countries. In Tanganyika, for example, male workers were typically recruited from designated labour supply areas great distances from the centres of economic activity. This entailed prolonged family separations which had serious physical and psychological repercussions for all concerned. The populations of African towns 'recruited by migration' were characterized by a heavy preponderance of men living in intolerably insecure and depressing conditions and lacking the benefits of family life or other customary supports (p. 114).

Through the migrant labor process and the enclave development system, the African workers, largely male, concentrated at the site of industry, agriculture, or extraction. In many cases the resulting depletion of males from the rural villages and farms caused a marked deterioration in women's ability to carry on alone and to provide for their families. A reduction in labor available to do farm work resulted from the men's employment-related migration (Stichter 1985). The dependency ratio (the number of very young and very old) increased in the rural areas and in the labor reserves (Murray 1981). In many cases, women began to change crops; although these crops were prolific and could feed many mouths, with the added benefit of requiring less labor input, they were largely carbohydrates rather than protein. Cassava is such a crop (Stichter 1985). This change in cropping patterns led to protein deficiencies and compromised the immune systems of persons living in the labor reserves (McCance and Rutishauser 1975). In fact, some studies show that simply the increased labor demand on women, with the reduction in time to attend to their children's nutritional concerns, leads to declines in children's nutritional status whether or not cropping patterns and crops have changed (Vaughn and Moore 1988).

Lack of labor to assist in land clearing produced overcropping of already-cleared areas, deterioration of the land, and loss of fertility. The loss of fertility also contributed to a reduction in yield, which further encouraged the growing of higher-yield crops on poorer soils, although these crops were less nutritious than in the past (Stichter 1985). A "bachelor wage" system usually produced little money income for migrant laborers to send back to a family on the labor reserve; thus the labor reserve subsidized other sectors of the economy to its own detriment (Stichter 1985). This cycle continues in a process of declining agriculture.

In addition, and particularly in recent decades, many unmarried rural women saw no means of adequate support for a family and thus emigrated to the city at a young age (see Murray 1981; Stichter 1985). Family conflict and separation also have caused many young women to migrate to the cities and to concentrations of male labor. Unfortunately, however, very few women have found wage labor or work of any kind (Doyal 1981, p. 116; Stichter 1985); there is little wage employment for women in the developing African economy (Stichter 1985, pp. 144–78). Some—perhaps many—of these women become prostitutes and enter the marginal or secondary labor market in the areas surrounding the large concentrations of men and development (Doyal 1981; Stichter 1985).

The combination of the migrant labor system with a heavy prepondernance of male laboring jobs and long familial separations caused a breakdown in family and sexual patterns in central and southern Africa (Murray 1981). An explosion of both prostitution and sexually transmitted diseases (STDs) in these populations occurred well before the

AIDS virus made an appearance (Bennett 1975; Brown, Zacarias, and Aral 1985; Rampen 1978; Sajiwandani and Babbo 1987; Sathyamurthy 1986). As Doyal 1981 states:

> . . . venereal disease became—and still remains—a major health problem in many parts of the third world where it was previously unknown. It is hardly surprising that the disruption of the economic and personal foundations of family life led to the disintegration of long-established marital and sexual patterns. In this context, the growth of prostitution represented one form of adaptation to the intolerable strains faced by men and women alike. In the case of male migrants, the absence of their wives was compounded by the fact that their new environment was almost exclusively masculine. This unequal sex ratio made it difficult for men to establish stable sexual liaisons with women, and encouraged prostitution. For the women involved, prostitution was usually a matter of sheer economic necessity . . . The structure of the colonial economy made it virtually impossible for women to sell their labour . . . most were compelled to live off the low wages of male workers. Often this involved either formal or informal prostitution (p. 115–16).

The development of the migrant labor and industrial pattern led to serious health consequences, particularly to an epidemic of sexually transmitted diseases in central and southern Africa. This pattern was clear even in 1981, when Doyal wrote the above (Osoba 1981).

In addition to the "pull" effect of industrialization and the migrant labor markets and the "push" effect of declining agriculture with reduced labor inputs, a "push" effect was produced by capital's takeover of African agriculture (Loewenson 1988; Sanders and Davies 1988). As in many Third World countries, the best and most fertile lands are used by capital to produce agricultural goods for export. Local food production is marginalized on poor land; rural labor needs become seasonal; as a result of large-scale monoculture and mechanization, rural "overpopulation" is "pushed" into urban slum areas. In these areas poor health, disease, and malnutrition abound. Employment is difficult, especially for women; family life may become difficult (Stichter 1985). The result, as with the "pull" effect of large capital developments, is the social creation of a population especially vulnerable to venereal disease and particularly to AIDS (P. Epstein and Packard 1987, pp. 10–17).

As a consequence of the migrant labor pattern and the capitalist takeover of rural African agriculture, AIDS had a ready population that suffered from an unusually high level of sexually transmitted diseases. Because of residual lesions and injury, the higher incidence of previous venereal disease (especially if untreated) increases the likelihood of contracting AIDS (Quinn et al. 1986, pp. 957–58; Stamm, Handsfield, Rompalo, Ashley, Roberts, and Corey 1988).

The manner in which labor is handled in migrant labor situations makes the resulting epidemics much worse. Such a labor system, in which migrants recruited from rural areas surround industrial and extractive developments, does not require care for the health or the safety of the working population. As long as labor power is in surplus in the rural and urban areas because of the expansion of capitalist agriculture and the existence of the labor reserve, injured or incapacitated workers can be replaced easily by subsequent migrants. Workers who no longer can work under such conditions simply return to their villages (Doyal 1981, p. 119). Further, workers' ability to combat poor working conditions and to demand health benefits from employment is severely compromised because migrant laborers are historically difficult to unionize or to organize for resistance to employers (Stichter 1985).

Urban slum dwellers, who lack medical care because of unemployment, also return home regularly when ill, carrying urban disease back to their rural villages (Doyal 1981). Thus a woman who is a prostitute and who becomes ill is very likely to return to her home village to be cared for by relatives.

In the case of sexually transmitted diseases and many other illnesses, this return home has tragic consequences. Tuberculosis and STDs are carried back to the village, infecting areas where they have not been seen before and striking populations that lack resistance or previous exposure. "Whether or not these migrants survived their diseases, the diseases invariably survived them, often spreading rapidly among an increasingly susceptible population" (Doyal 1981, p. 119).

Therefore there is little question that AIDS

strikes a population which is not only susceptible to sexually transmitted diseases but which is also structured socially to hasten the transmission of the HIV-1 virus. Such structuring of a population to make it vulnerable to STDs is an integral part of the peripheral status of eastern, central, and southern Africa. As argued above, the social structuring of the family unit and labor migrancy are related closely to partial proletarianization and incomplete separation from the land, characteristics of a peripheral labor force (Friedman 1984; Martin 1984; J. Smith 1984; Wallerstein 1984; Wallerstein and Martin 1979).

Past data on prevalence of sexually transmitted diseases show clearly that the pattern of STD spread described above has occurred. For instance, in studies completed in the 1960s and early 1970s, the primary risk factor for gonorrhea in eastern Africa was male labor migrancy, with a pool of infection in the female prostitute population (Bennett 1962; Verhagen and Gemert 1972). The pattern of spread from urban and labor concentrations to rural areas also was confirmed for gonorrhea (Bennett 1964). Prevalence data also establish that higher rates of STDs are present around labor concentrations.

An especially well-documented example in west Africa may suffice to show this relationship. In Cameroon, migrant workers "were and are considered to be a major cause of a high incidence of venereal disease among the population in the plantation environs" (DeLancey 1978, p. 172). This is the area of labor concentration in Cameroon; DeLancey regards the return of migrant laborers to rural areas as a problem in this area. Further, a 1949 health survey showed a 4.2 percent rate of gonorrhea infection in rural areas far from these labor concentrations, in contrast to 13.1 percent and 10.4 percent rates in areas directly surrounding the concentrations. In 1948 a 21 percent rate of gonorrhea infection was found in the heart of the migrant labor district in Cameroon (DeLancey 1978, p. 179). Labor migrancy in west Africa has declined since the 1940s, however, so this pattern may no longer be in evidence in that area.

Urethral stricture is a common long-term result of STD, particularly of gonorrhea. A spatial mapping of this condition for Zambia, in east central Africa, shows that it is most prevalent in the copper belt of Zambia, the primary labor concentration area. The second highest area of prevalence is in the northeast of Zambia, a labor reserve area (Kibukamusoke 1975). Further, the condition is much more prevalent in Uganda in the labor concentration area: it forms a half moon around Kampala and Lake Victoria, and shows secondary concentrations in the West Nile district and in Kigezi and Ankole. Migrant labor is drawn from these areas into the Kampala area (Kibukamusoke 1975).

Thus the pattern of the eastern and central African labor market—a migrant labor system based on labor reserves, surpluses of laborers, and a laboring population separated only partially from the land—may account for many of the differences between the AIDS epidemic in the United States in the African population. In Africa as in North America, AIDS required a population that was vulnerable to sexually transmitted diseases. In each case the virus first made its inroads into these populations. In Africa, a population structured socially by capitalist agriculture and a labor market created in its present form by dependency development gave AIDS the foothold it required. The prevalence of malnutrition, malaria, measles, and other diseases that resulted from the collapse of local food production and from Africa's dependency development pattern has resulted in a population whose health is continually compromised. Large numbers of persons have immune systems weakened not only by diseases but also by malnutrition, a condition which also may help to set the stage for AIDS infection (P. Epstein and Packard 1987; Hall and Langlands 1975; McCance and Rutishauser 1975). In North America, a gay population that had had a sexually transmitted disease problem for at least a decade was the foothold for the HIV virus (Altman 1986; Shilts 1987). In each case the initial stages of the epidemic were determined by the population in which the virus was able to establish itself. In both cases it appears that although these populations affect profoundly the perceptions of AIDS, the HIV-1 virus will not confine itself to these sites. They are simply the populations that were most vulnerable at the outset.

This model for the spread of the AIDS epidemic relies on the scientific understanding of the HIV-1 virus and of its transmission capabilities. We know that this virus is spread in much the same way as hepatitis B, by body

fluids and particularly during sexual activity. We also know that previously untreated STDs, especially when they may cause breaks in the skin or ulcers, increase the likelihood of infection on exposure to HIV-1 (Schoub et al. 1988; Simonsen 1988; Stamm et al. 1988). Both in Africa and in the United States, large numbers of sexual partners increase the possibility of infection with HIV-1 and the resulting development of AIDS (Carswell 1987; Curran 1985).

This model of the AIDS epidemic in eastern and central Africa also relies on an understanding of historic patterns of development in this area. The context of dependency development, combined with studies of medical and population geography for the region, gives an outline of the probable high-prevalence areas in countries in east Africa and indicates which groups in the population will have high prevalence and high incidence rates of HIV-1 infection. The following section will discuss in greater detail the implications of this model in medical and population geography and in population subgroups by presenting a series of hypotheses regarding the prevalence rates of HIV-1 infection, primarily in Uganda and in neighboring Burundi and Rwanda in eastern Africa.

AIDS: THE PATTERN OF AN EPIDEMIC IN AFRICA

If the model that was presented above its valid for the AIDS epidemic in eastern and central Africa, the pattern in which the epidemic appears should show some evidence of the expected modes of transmission. In other words, if the epidemic occurs geographically in areas with high concentrations of male migrant labor, and particularly among males and females who have high levels of heterosexual activity, then high prevalence or incidence rates should appear first geographically in areas where labor is concentrated. Further, two population groups in these areas should be affected: the male migrant laborers themselves and their partners, usually female prostitutes living near the enclave of development. Thus a high rate of infection in these two groups would fit the model.

Further, if the spread of the HIV-1 virus occurs through the return of male migrant laborers and female prostitutes to their villages due to illness, the second geographic sites that should begin to show high rates of infection are those areas in eastern and central Africa known as labor reserves. If we assume that the rural areas are infected with HIV-1 after the labor concentration areas are infected, these rural areas should show lower prevalence rates than the labor concentration areas. Yet the labor reserves—the migrant labor pool areas—should show higher rates of infection than surrounding rural areas which do not provide migrant labor. Thus prevalence of HIV-1 in the areas from which migrants are drawn by contract in order to provide the workers necessary for production should lag somewhat behind the areas where these workers labor. When the workers return home carrying HIV-1, it is assumed in this model that they will pass the infection on to their partners and to others after their return.

The prostitutes from these areas of male migrant labor concentration also tend to return home when illness prevents them from continuing their work. This trend may be less definite because prostitutes are not dismissed formally. They may continue prostitution activities and delay returning home, even though they are quite ill. They may be able to continue working and generating some income, although their capacity may be reduced considerably.

The brief discussion in this paper concerning the historical, social, and economic development of eastern, central, and southern Africa points in a number of directions for further research and study. As a start, available data concerning HIV-1 seropositivity and AIDS prevalences can be used to test the pattern of the epidemic. Do the available data on seropositivity and AIDS prevalence fit the pattern that would result from the description presented above? We can use this description to generate the following 14 hypotheses based on the above argument:

1. Geographically, areas of migrant labor concentration should show high prevalence rates of HIV-1 infection. These sites include mining areas, commercial plantations, and some large cities.
2. Geographic areas from which migrant laborers are drawn should show high rates of prevalence for HIV-1 infection.
3. The prevalence of HIV-1 infection in labor reserves should be higher than in

rural areas that do not form labor reserves.

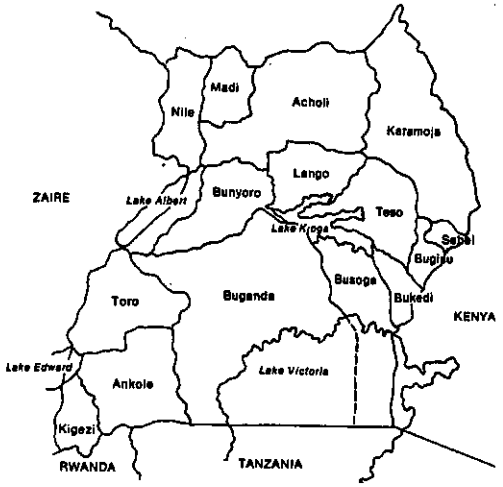
4. Geographic areas from which young women migrate to become prostitutes should show high prevalence levels of HIV-1 infection. These rates should be higher than for other rural areas. Again, the rate of HIV infection in rural labor reserves should be higher than in areas of stable commodity production by peasants.
5. Because of earliest transmission by migrant labor to the rural labor reserves, HIV-1 seropositivity should appear earlier in these areas than in surrounding rural areas or in areas of stable peasant agricultural commodity production. This difference may be due to temporal differences in transmission as well as to reduction in the number of sexual partners once a migrant laborer returns home.
6. The prevalence rates for areas where migrant labor is concentrated should be somewhat higher than for the rural labor reserves, the areas from which migrant labor is drawn.
7. HIV-1 seropositivity should appear earliest in the areas where migrant labor is concentrated, such as mining areas, plantations, and cities.
8. Migrant laborers themselves should show higher rates of infection than other groups in the population.
9. For male migrant workers, the rate of HIV-1 seropositivity may increase with length of labor contract or time away from the family.
10. There should be a lower rate of prevalence among permanent migrant laborers who are allowed to take their families to the job site.
11. Prostitutes concentrated in areas where male migrant labor is located should show higher rates of infection than other women in the population. Further, prostitutes located in areas of migrant labor concentration should show higher levels of HIV-1 seropositivity and AIDS than prostitutes in areas migrant labor does not concentrate. These rates should bias the age and marital status data concerning women who are infected with HIV-1. Therefore infected women should be younger and more often single than infected men.
12. Higher prevalences and earlier occurrences should appear among those who previously suffered sexually transmitted diseases (STDs). This population is constructed socially and biologically to transmit these diseases.
13. There should be lower prevalences among persons on the rural labor reserves who have not migrated for laboring contracts, and among their families.
14. HIV-1 seropositivity should evidence a complex pattern of spread. The epidemic will not spread from rural areas to the city, as would be posited by a disease which is endemic for long periods of time in rural areas and which breaks out into the larger population because of social disruption. Such a pattern should appear earlier and in higher prevalences in rural areas. Rather, HIV-1 seropositivity should evidence a movement from areas of labor concentration to rural labor reserves and outward from these latter foci of infection to the general rural community. The degree of movement of HIV-1 seropositivity and AIDS into a labor reserve should depend on the quantity of migrant labor employed from that reserve, the length of time of contracts or absences, the age of laborers who migrate, the type of employment to which migrants are moving, the number of women leaving for the city and the work they obtain, and the rate of return of ill women and migrant laborers. The direction of movement should depend on where most of the migrant labor originates, where recruiting occurs, and from what areas women are leaving. In other words, numerous factors that influence the sexual and migratory patterns in eastern, central, and southern Africa will affect the spread and transmission of HIV-1 seropositivity and AIDS. The principal organizing factor, however, is the fact of labor migrancy and the labor reserve. From this central point one can begin to investigate the variations and the complex movement of the epidemic.

DATA AND PREVALENCE SURVEYS

Do the available data support the 14 hypotheses presented above? Do the actual AIDS prevalence and seropositivity studies conducted in eastern, central, and southern Africa conform to these hypotheses? Caution is advised because, as already mentioned, data are sometimes unreliable and fragmentary. In addition, as mentioned earlier, the ELISA test for HIV-1 immune response may be much less reliable in Africa than elsewhere (Biggar et al. 1985). Of course, in Uganda the use of the WHO case definition (Berkeley, Okware, and Naamara 1989), which does not rely on laboratory work, makes this unreliability less of a problem.

Further, the process of HIV-1 transmission, which was outlined above, is dynamic. Much of the data concerns prevalence, which can be examined spatially or geographically; incidence data are relatively scarce. Prevalence data are inherently static, not dynamic, and we can only infer the dynamic process from the static data. It is rare, however, to find truly adequate data in social science; therefore let us pursue the available data to see if they agree with the above model and with the hypotheses that the model has generated. In the discussion that follows refer to Figure 1, a map of Uganda, including district names. See also Figure 2 which shows the labor concentration areas and the labor reserve areas in Uganda. This map is derived from historical accounts and migration data from the Ugandan census (Kabera 1982; Mamdani 1976; Gimui 1982).

FIGURE 1. Map of Uganda



Hypotheses 1, 2, 3, 4, 5, 6 & 7

These hypotheses concern the geographical and temporal distributional evidence for the pattern of HIV-1 transmission outlined above. Geographical distribution is capable of eliciting transmission patterns in some diseases (Hall and Langlands 1975; Hunter 1974; McGlashan 1972; Meade 1980; Pyle 1979).

It is clear that AIDS cases and HIV infection generally have been most serious in cities in Uganda and in much of eastern Africa (Berkley et al. 1989; Biggar 1986; Economist 1987; Georges et al. 1987; Harden 1986; Harden 1987; Mann, Francis, Quinn, et al. 1986; Mugerwa and Giraldo 1987; Mugerwa, Widy-Wirski, and Okwake 1987; Van de Perre 1984). These are areas of labor concentration. This conclusion is also confirmed by cumulative prevalence data from Uganda (see Figure 3), which establish that the highest prevalence rate of AIDS in Uganda is contained in the half-moon area along the lakeshore of Lake Victoria, particularly in the Masaka and Kampala-Entebbe areas (Berkley et al. 1989, p. 83).

The problem with these data is that they reflect not only the possible reality of the epidemic but also the availability of medical facilities in these regions of Africa. Most medical facilities that are capable of recognizing and diagnosing AIDS in central and eastern Africa are concentrated almost exclusively in cities (Hall and Langlands 1975; Navarro 1974). Thus these areas will show a predominant number of diagnosed cases. Individuals from rural areas may not even be able to travel to the cities for diagnosis and may be either misdiagnosed or not treated at all. It has been noted frequently that AIDS patients in eastern Africa are more well-to-do than the average population (Georges et al., 1987; Harden 1987; Van de Perre 1984). This finding also may reflect the comparative ease with which the most affluent classes are able to enter the medical system in contrast to poorer or rural individuals.

In Uganda, however, it has been claimed that the prevalence of HIV-1 seropositivity does not seem as high in Kampala as elsewhere (Economist 1987), and that the southwest areas of the country have higher HIV-1 prevalence rates. This conclusion seems to be refuted by recent, more comprehensive studies and cumulative prevalence data (Carswell 1987; Berkley et al. 1989) (see

FIGURE 2. Labor Concentrations and Labor Reserves in Uganda

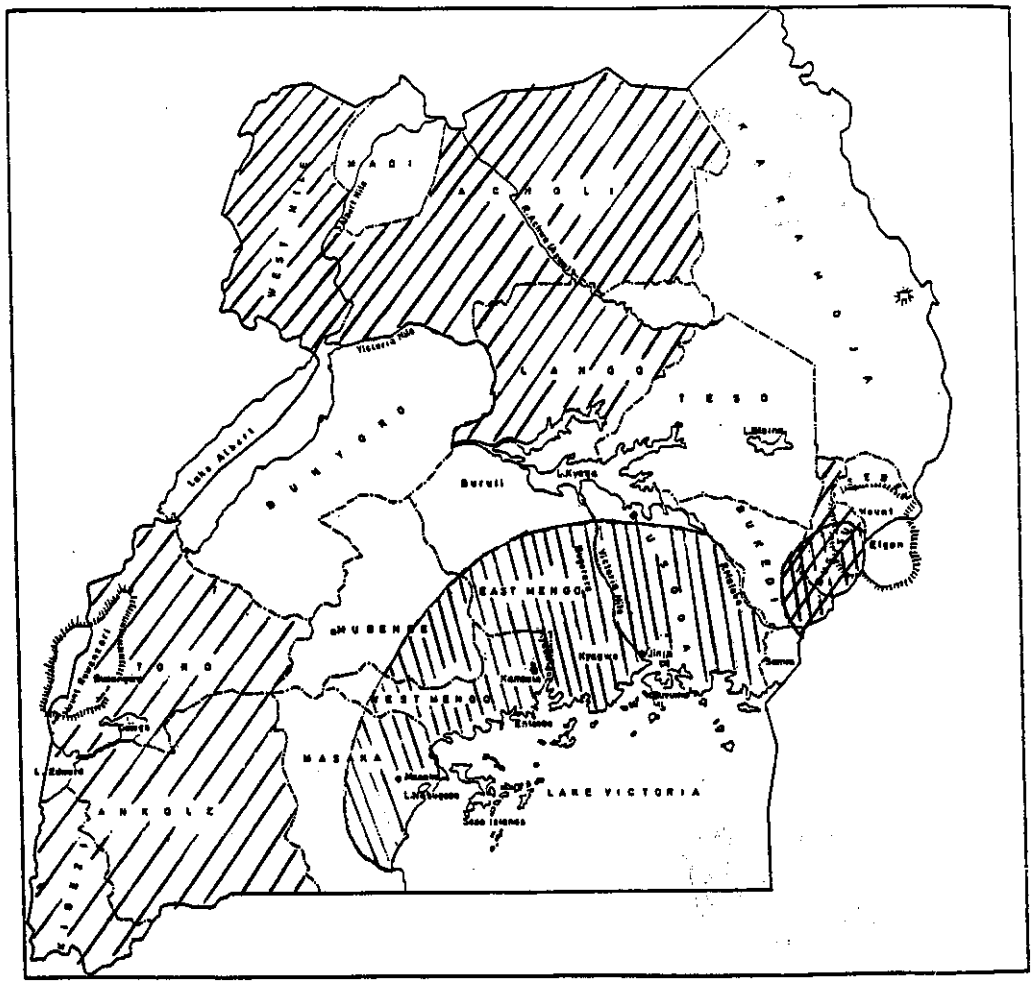
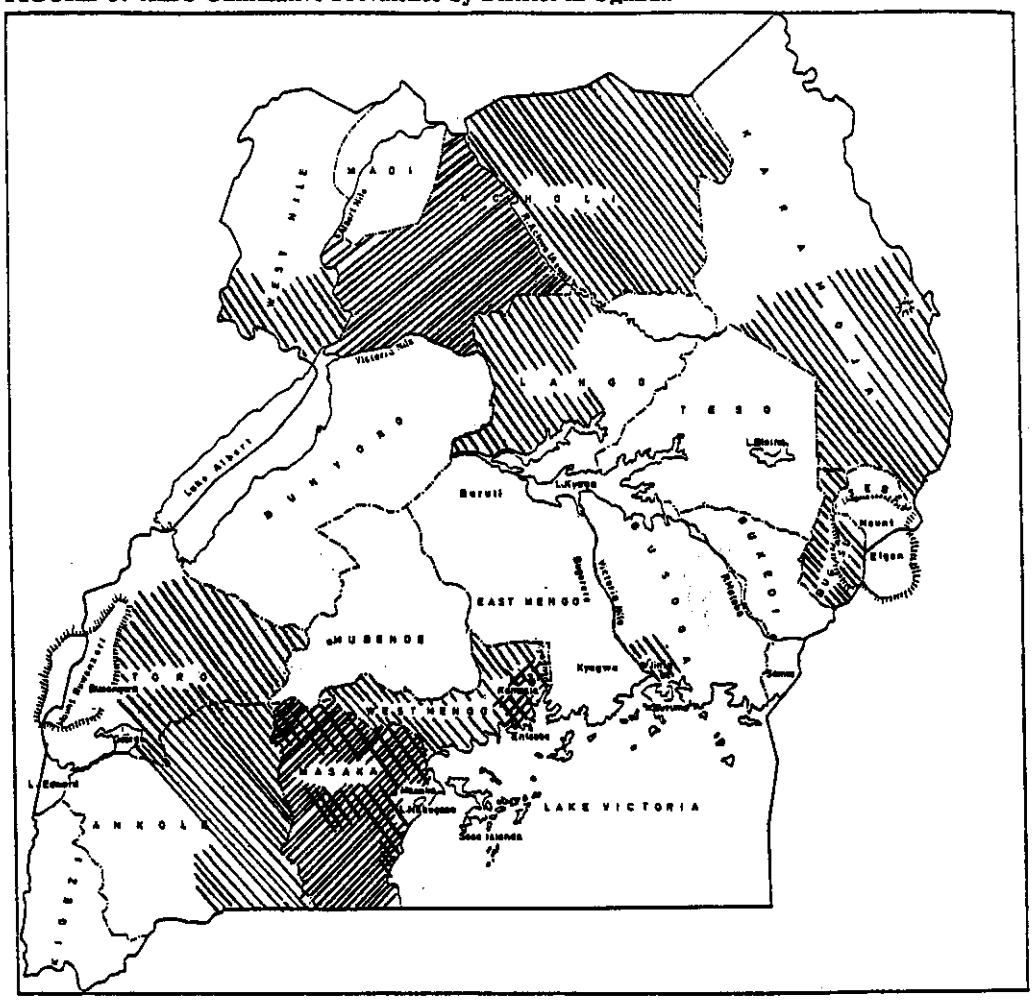


Figure 3). In general, rural residents, particularly in the peasant farming areas of central Uganda, seem to have a rather low rate of HIV-1 seropositivity and AIDS (Berkley et al., 1989; Carswell 1987; Harden 1987; Van de Perre 1984). In Rwanda and Zaire the infection rate in the country is far lower than the very high rate in the cities (Specter 1986). We can say, however, that rural southwestern Uganda appears to have very high prevalences of AIDS cases and HIV-1 infection (Berkley et al. 1989; Economist 1987; Serwadda, Seqankambo, Carswell, Bayley, Tedder, Weiss, Mugerwa, Lwegaba, Kirya, Downing, Clayden, and Dalgleish 1985). It has been estimated that one-third of this population is infected with HIV-1 (Economist 1987). It is clear from the cumulative prevalence data of AIDS cases that the southwest areas, particularly Anchole and Toro, have quite high rates of AIDS cases, in the 11-50/100,000 range. These two areas are labor reserves with high rates of circulating migration for work in the core developed areas in Uganda.

In studies of prostitutes in the town of Rakai, an area of major labor concentration south of Kampala, some 86 percent were HIV-1 positive (WHO 1988). Barmaids were 80 percent seropositive in this same area. It is

FIGURE 3. AIDS Cumulative Prevalence by District in Uganda



easy to see the high cumulative rates of AIDS cases in this area on the map of Uganda (see figure 3). In fact, this area south of Kampala has one of the highest rates of AIDS in Uganda, comparable to the Kampala-Entebbe area (see Figure 3).

The countries of Burundi and Rwanda have not established an AIDS surveillance system such as the one in place in Uganda. However, the WHO has collected some cumulative figures for AIDS cases in these two countries. These figures were accumulated until 31 December 1988 and they establish that Burundi's prevalence rate of AIDS cases is 13/100,000, with a total of 1408 cases of AIDS (WHO 1989, p. 113). This rate is comparable to the high rate found in Ugandan labor reserves such as the West Nile, Lango, Acholi, Bugisu, Toro, and eastern Ankole. Further, the data show that Burundi has the second highest national cumulative prevalence rate of AIDS cases in Africa, higher than the overall rate for Uganda (WHO 1989, p. 113).

Rwanda, according to these figures, has a cumulative AIDS case rate of only 2.8/100,000, placing it at a much lower rate than other labor reserves. Similarly, the labor reserve in Kigezi, the extreme southwest of Uganda, has a low rate of 1-10/100,000. This case may reflect a diminished rate of circulating migrant labor and increases in permanent settlement outside Kigezi (Kabera 1982). This is only speculation, however, as we have no really accurate figures for the trends in migratory labor from this district in the 1970s and 1980s.

The low figures for both Rwanda and the Kigezi district may reflect underreporting, although this is more likely for the Rwanda figures. Underreporting in Rwanda seems very likely since the infection rate in Kigali, Rwanda, has been estimated at 25 percent (Van De Perre 1984). The infection rate in rural areas of Rwanda has been estimated to be very low (Van De Perre 1984; Harden 1987), so it might be the combination of these two rates which produce the somewhat lower rate. In the early 1980s Rwandan prostitutes had the world's highest infection rate among this subgroup with an 80 percent reported infected in 1983–84 (Mann, et al. 1986a; Padian 1988, p. 414). This level of infection was not reached by any other prostitute population studied until 1987 when 535 incarcerated women studied in Nairobi, Kenya showed an 85 percent infection rate (Padian 1988, p. 414). It is possible that the early (1983–84) figure for Rwandan prostitutes reflected ELISA tests which were not properly interpreted due to malaria contamination. This seems unlikely, however, due to Rwanda's high altitude and its relative low level of malaria infection. The reported rate of HIV infection in the WHO figures may actually underestimate the infection rate in Rwanda, especially because Rwanda has not shown the same degree of openness increasingly evident among other Eastern African nations with regard to AIDS.

Underreporting seems less likely in the Kigezi district, since the area has been served (at least in the 1960s, the most recent report) by two government hospitals, two mission hospitals, two health centers and a number of dispensary/maternity units (Hall and Langlands 1975, p. viii). This might be an area for further research. However, the surveillance system in Uganda is passive, not legally required, and may underreport cases (Berkeley et al. 1989, p. 82). The Kigezi District, however, would have to have a systematic underreporting bias to explain its lower rate of AIDS cases compared to other labor reserves. At the present, this systematic bias is unknown and the low rate may be seen as unsupportive of the hypotheses put forward on geographical distribution in the case of Kigezi.

In the southern areas of West Nile District, cumulative AIDS case prevalence figures establish that the rate is in the 11-50/100,000 range. In the northern areas, the rate is lower, 1-10/100,000 (see Figure 3). This, despite the fact that it has been claimed that there is a low rate of infection in the entire area of the West Nile (Carswell 1987). Why this variation and relatively low rate in the northern section of the West Nile?

There is some indication that the Tanzanian invasion and subsequent civil war, which started in 1978 and continued until 1985, impeded the movement of commerce and migrant labor from the West Nile district to the Kampala area, thus delaying the spread of HIV-1 (Avirgan and Honey 1982; Carswell 1987; Mamdani 1988). Further, the Nile River itself often forms a "formidable barrier to communication" and interchange (Carswell 1987). Unlike many previous wars and conflicts, the fighting in Uganda may have slowed down the spread of disease by slowing the flow of migrant laborers from the West Nile area into Kampala. Certainly the present prevalence of AIDS in the West Nile area is lower than in the Kampala area. There is some indication that the army may be spreading HIV-1 in its movements into the northwest to suppress the recent rebellion in this area of Uganda (Economist 1987). The generally high rate of AIDS cases in the southern section of the West Nile, combined with the difficulties in commerce in the 70s and 80s may be seen as supportive of the hypotheses which we have advanced for temporal and spatial distribution of the AIDS epidemic.

Many cite Acholi in central-northern Uganda as the leading area of migratory labor (Kabera 1982; Mamdani 1976). Acholi's cumulative rate of AIDS cases is second only to that of the labor concentrations around Kampala-Entebbe and Masaka (see Figure 1), with a 51-100/100,000 rate in the western sections of Acholi. The western section of Lango and the eastern section of Acholi also contain

unusually high cumulative rates of AIDS at 11-50/100,000. Both of these findings support the hypotheses on spatial and temporal distribution of AIDS.

In eastern sections of Uganda, a high rate of cumulative AIDS case prevalence, 11-50/100,000, occurs in Bugisu area, also a major labor reserve and a source of migrant labor. This high rate extends northward into the Karamoja District which is not a major labor reserve area. In general, however, these results are supportive of hypotheses one through seven.

It is difficult to determine if the syndrome occurred earlier in the urban labor concentrations than in the labor reserves, but one can argue that the higher cumulative prevalence rates in the Kampala-Entebbe and Masaka labor concentrations are consistent with an earlier appearance. Certainly, it can be argued that AIDS seems to have had a longer time to reach high epidemic levels in these labor concentrations.

When considering countries other than Uganda, Burundi, and Rwanda, in areas of labor concentration such as the copper mines of Zambia and Zaire, the evidence is consistent with a higher rate of spread of HIV-1 (Harden 1987). In Lusaka, the capital of Zambia, the rate for blood donors was 15 percent in 1986. In the northern copper belt areas, the rate of HIV-1 seropositivity among blood donors was 13 percent (Harden 1987; Melbye, Mads, Bayley, Manuwele, Clayden, Blattner, Tedder, Njelesani, Mukelabai, Bowa, Levin, Weiss, and Biggar 1986). Zambia has always had a clearer pattern of labor concentrations and labor reserves than Uganda, because the copper belt has dominated the economic development of that country (Kay 1967; Mamdani 1976; Parpart 1983; Roberts 1979). Unfortunately, we have little data from labor reserve areas in Zambia to compare with the data from the copper belt. In Zaire it is clear that the rural prevalence of HIV-1 is low (Nzilambi et al., 1988) and that urban prevalence, at least in Kinshasa, is quite high (Mann et al., 1986a). Hardly any data exist in Zaire, however, to allow comparison of these results with those in labor reserve areas.

Hypotheses 8, 9, and 10

Migrant laborers themselves should show higher rates of HIV-1 infection than others in the population; these data should correlate with family factors and with length of contracts. As with the other hypotheses, data on this point are scarce. There are only two studies that concentrate on migrant laborers in eastern, southern, and central Africa; we may use national data to draw some conclusions concerning this group.

In 1986, 74 truck drivers for a large freight company in Kampala (Uganda) were tested for HIV-1. Some 55.9 percent of the drivers were seropositive (Carswell 1987; Economist 1987). The overall HIV-1 infection rate for Kampala was estimated in 1986 to be approximately 10 percent (Harden 1986). Thus, we can say that in Uganda truck drivers have a much higher rate of infection than others in the population. These drivers and their mates, known as "turnboys," were either Ugandan or Kenyan nationals and had large numbers of sexual partners, particularly prostitutes (Carswell 1987).

In South Africa, the country with the most highly "developed" system of migrant labor, we might expect clearer findings in regard to HIV-1 seropositivity. South Africa has a very systematic labor reserve policy, termed a "homelands" policy; workers usually are banned from living with their families near where they contract to work. Thus the familial separation is enforced more strictly in South Africa, particularly in the mining regions.

In a study of South African migrant miners, 30,000 miners were selected randomly for HIV-1 testing. Some labor reserve areas and some countries showed a seropositive rate in the migrant labor group as high as four percent. In 1987, however, South Africa reported an overall infection rate of 0.1 per cent, with only 46 cases of full-blown AIDS. Therefore it would appear that the AIDS epidemic is concentrated in the migrant labor population in South Africa. This pattern is recognized by South African medical authorities (Ijsselmuiden et al. 1988; Sher 1986).

Finally, with regard to migrant laborers, the countries of Rwanda and Burundi, particularly the former, seem to have a serious problem with AIDS infection (Biggar 1987; Brunet and Ancelle 1985; Harden 1987; Mann et al. 1988a and b; Quinn et al. 1986; WHO 1989). It is noteworthy that these two mountainous countries send massive numbers of migrant laborers to the labor concentration

areas of Uganda. In fact, they constitute labor reserves for Uganda; some 380,000 Rwandans and 140,000 Rundi have lived in Uganda. These individuals come to Uganda as migrant laborers and usually are only temporary residents. A large proportion of the migrant laborers in the areas around Kampala come from these two countries (Hall and Langlands 1975, pp. 7–8). The high cumulative prevalence of AIDS cases in Burundi and the likelihood of high prevalences in Rwanda argue strongly for the hypothesis that AIDS prevalence will be high among these migrant laborers.

It is impossible to determine from these data whether the earliest cases of AIDS in South Africa or in central or eastern Africa occurred among migrant laborers. If we assume, however, that the AIDS epidemic is new in many of these areas (see Carswell, Sewankambo, Lloyd, and Downing 1986), we may say that the prevalence data indicate where the epidemic began. Thus although cases may show up earlier in isolated groups in South Africa, the prevalence among migrant laborers argues for an early appearance in this group. Unfortunately, there are hardly any data on other migrant laborers or on their family and sexual patterns; nor are these data correlated with length of contract, working conditions, and other factors. This lack of data militates against truly definitive conclusions.

Hypothesis 11

One of the best-documented facts about the AIDS epidemic in Africa is its incredibly high prevalence rate among prostitutes in all countries. Prostitutes are at high risk for HIV-1 infection (Mann et al. 1988b) and are recognized world-wide as transmitters of HIV-1 (Carael, Van de Perre, Lepage, Allen, Nsengumuremyui, Van Goethem, Ntahorutaba, Nzaramba and Clumeck 1988; Day 1988; Hudson, Hennis, Kataaha, Lloyd, Moore, Sutehall, Whetstone, Wreghitt and Karpas 1988; Padian 1988). In different areas of the world the practice of prostitution varies from the United States where prostitutes develop a professionalization associated with "marked distance between their working and private lives (Day 1988, p. 421)," to the much more informal situation in Sub-Saharan Africa, where prostitution is difficult to define (Day 1988, p. 424).

Rates of infection among some prostitute groups range from 27 percent in Kinshasa, Zaire to 66 percent in Nairobi, Kenya and 88 percent in Butare, Rwanda (Mann, Francis, Quinn, et al. 1986). Overall infection rates in Rwanda are approximately 25 percent in Kigali, although rural infection rates are almost zero (Harden 1987; Van de Peere 1984). In a survey of 327 persons concluded in 1986 in the Central Africa Republic, 19 percent of prostitutes were seropositive, compared to a 4 percent overall infection rate for prostitutes in the Central Africa Republic (Specter 1986). In Tanzania a 1986 study of 225 prostitutes established a 29 percent seropositivity rate (Padian 1988, p. 414).

A study in Kenya showed a 4 percent rate of HIV-1 infection in Nairobi prostitutes in 1980 (Kreiss 1986). In 1987 the prevalence among 115 prostitutes in Nairobi was 65 percent. None of these women had been infected by HIV-1 only one year before. More than one-half of the new cases were attributed to the presence of genital ulcers and to the use of birth control pills, which apparently increase the probability of HIV-1 infection (Specter 1986).

The infection rate for women overall is consistently much lower than for prostitutes, as would be expected. The rate for sexually active persons of both sexes is approximately 20 percent (Mann et al. 1988, p. 85). In fact, women of childbearing age evidenced approximately a 10 to 15 percent infection rate in 1986 in Kampala, Uganda (Harden 1987). In one hospital in Kampala, 13 percent of 1,000 pregnant women were found to be HIV-1 seropositive in 1986. The same hospital showed a prevalence of 24 percent one year later among 170 pregnant women (Blaine 1987; Carswell 1987).

In all of the countries of this region, the mean age of men with seropositivity was greater than the mean age of seropositive women (Quinn 1987; Quinn et al. 1986; Mann, Francis, Quinn, et al. 1986a). Women account for most of the AIDS cases in the 20–29 age group in Kinshasa, Zaire. Most of the cases in the 40–49 and 50–59 age groups are male, although both of these groups generally contain smaller cohorts of AIDS cases than the 20–29 group (Mann, Francis, Quinn et al. 1986). Among those suffering from AIDS, women are more likely to be

unmarried than men (Mann, Francis, Quinn, et al. 1986; Quinn 1987; Quinn et al. 1986).

All of this evidence points to a sexually transmitted disease in which the number of partners increases the risk of exposure to the infection (Carswell 1987; Clumeck et al. 1985; Kreiss 1986; Serwadda et al. 1985; Van de Perre 1984). Prostitutes, having large numbers of partners, are particularly at risk, but the pattern also fits the larger social model which has been advanced. Young women migrating to concentrations of male migrant labor become prostitutes in order to earn a living in an environment in which women are rarely employed in wage labor or other "legitimate" jobs.

Hypothesis 12

There is little question that persons who were previously infected by sexually transmitted diseases are at higher risk for HIV-1 infection because of the continued high number of sexual partners. (Pepin, Plummer, Brunham, Piot, Cameron, and Ronald 1989). This pattern may be evidence of a life situation, such as migrant laboring, which gives rise to an unstable sexual life. Further, as mentioned earlier, this group also increases its risk if sexually transmitted diseases are untreated or if lesions and ulcerations are present to allow infection more easily.

Hypothesis 13

Unfortunately, it is not possible to compare migrant workers with persons from the same labor reserve area who do not migrate because there seems to be little information reflecting tests of migrant workers themselves. Thus at present we have little ability to compare prevalence rates in this way.

Hypothesis 14

There is little question of the complex nature of the AIDS epidemic in central, eastern, and southern Africa (Biggar 1987; Carswell 1987). This complexity may blind observers to the existing patterns and without geographic, historic, and economic information about the region observers cannot understand the map of cumulative prevalences of AIDS.

Yet on the basis of much of the data dealing with urban and rural prevalences of HIV-1 seropositivity and AIDS cases, it is quite clear that a rural-to-urban spread pattern is not present. Urban infection rates are considerably higher than rural (Berkley et al. 1989; Carswell 1987; Carswell et al. 1986; Clumeck et al. 1985; Nzilambi et al. 1988; Peterson et al. 1987; Recene et al. 1987; Van de Perre 1984; Wender, Schneider, Gras, Fleming, Hunsmann, and Schmitz 1986). Therefore these data provide additional refutation of the argument that the HIV-1 virus was somehow endemic in rural Africa and then broke out into the broader community because of war and social disruption. Such an argument is not supported by the prevalence patterns we have seen here. It is much more logical to argue that this disease spread as a result of the complex migratory labor patterns in the eastern, central, and southern African economy. This would explain the complex pattern of cumulative AIDS prevalences seen in Uganda. The effects of these patterns on sexual practices and on the stability of the Africa family in these regions help to set up the pattern of AIDS epidemic, whose outline we can see from the scanty evidence available.

There seems to be substantial evidence, particularly in geographical cumulative prevalence data, as well as in HIV-1 seropositivity prevalence data among some specific population groups and geographical areas, that would support the model which we presented for the AIDS epidemic in Eastern Africa. In a rather striking manner, this evidence supports the concept that the HIV-1 virus, and the resulting AIDS, first struck the labor concentrations in Uganda and then moved outward to the labor reserves, carried by migrant laborers and prostitutes as they return to their birthplaces for care and assistance with illnesses consequent to infection with this virus.

CONCLUSION AND SUMMARY

The Western press and (to some extent) the Western scientific establishment have attempted to locate the origins of the HIV-1 virus, the biologically causative agent for AIDS, in Africa. In my opinion this attempt is based on a "victim-blaming" mentality and

is fundamentally racist (Hunt 1988). The desire to see serious disease as coming from somewhere else, as an invasion, as a foreigner, is widespread (Sontag 1978, 1989; Brandt 1985). The "African origins" theory is based on many weak arguments; almost all have been disproved, including the genetic relationship of the HIV-1 virus to the simian variety of retrovirus found in the green monkey of equatorial Africa (see Gallo 1987; Mulder 1988). The "African origins" myth also arose because cases of AIDS were discovered earlier in Africa than in Europe or in North America. This argument, too, has been refuted decisively (see Carswell et al. 1986; Garry et al. 1988; Huminer, Rosenfeld, and Pitlik 1987; Hunt 1988).

One of the remaining props of the "African origins" myth is the pattern of spread of the disease in eastern, central, and southern Africa. The thesis of this paper provides an alternative explanation to the myth insofar as the myth is supported by the rapid or early spread of HIV-1 seropositivity in Africa. The historical pattern of sexually transmitted disease in eastern, central, and southern Africa is related intimately to dependency development and to migrant labor. It provides a much more adequate explanation for the rapidity and the complexities of the spread of HIV-1 seropositivity in Africa than do any assumptions about the origins of HIV-1. "African origins" theories contradict the evidence not only genetically and clinically, but also epidemiologically.

In the foregoing discussion an alternative epidemic pattern has been presented for AIDS in eastern, central, and southern Africa. This pattern has appeared in other areas where migrant labor markets were combined with dependency development and where workers were separated incompletely from the land; it has occurred, for instance, in western Africa. This pattern is rooted in migratory labor markets and is based in systematic economic, historic, and social development. Previous epidemics of STDs occurred in eastern, central, and southern Africa, resulting from the conjunction of the same historic, economic, and social factors. Those epidemics demonstrate medical/geographic and temporal patterns that are based in the movements of labor between concentrations of work and industry and the labor reserve.

The data on HIV-1 seropositivity and AIDS prevalence in Africa are sparse, scattered, small in scale, and often inaccurate. Yet even in view of these limitations, the available data on HIV-1 seropositivity and AIDS surveillance and testing in eastern, southern, and central Africa support the historical pattern and the labor market relationship in the AIDS epidemic now ravaging Africa. The highest prevalence rates of AIDS and HIV-1 seropositivity occur in the concentrations of industry and development in Uganda. In the rural labor reserves, particularly in Burundi, Rwanda, and southwestern Uganda, very high prevalence rates of HIV-1 seropositivity and AIDS also occur, but those rates are not as high as in the industrial and developed areas. Labor reserves in parts of Uganda where prevalence rates of seropositivity and AIDS are low show isolation from traditional labor migration because of war and social disruption over the last decade. The general rate of rural infection in eastern, southern, and central Africa is quite low.

There is some evidence of high rates of HIV-1 seropositivity among migrant laborers themselves. Certainly, prostitutes have very high rates of seropositivity and AIDS, as would be expected by most social theories of STDs. Further, those who suffered from previous STDs appear more likely to contract HIV-1 seropositivity and therefore AIDS. This interaction in the AIDS epidemic reinforces the historic patterns of STDs already occurring in this region in Africa.

In conclusion, the epidemic of HIV-1 seropositivity in Uganda and in other countries of the area is a social event, not simply a biological occurrence (see Stark 1977). Although a biological understanding of HIV-1 is necessary, it is inadequate. The epidemic of HIV-1 seropositivity and AIDS in Africa must be understood socially, in its historically specific context, or not at all.

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