The Social Ecology of AIDS in Africa

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"Societies are complex, and complexity calls for caution" (Susser, 1987:171)

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The incidence of AIDS in populations

The practice of epidemiology, according to Geoffrey Rose, is based on the question "Why did *this* patient get *this* disease at *this* time" (Rose, 1985). Epidemiologists can be quite good at detecting the causes of illness by comparing individuals with different levels of exposure to hypothesised risk factors or infectious agents. This is how we know that HIV causes AIDS, and how we know that certain behaviours are associated with a higher or lower risk of transmission of HIV.

When we look at AIDS epidemics as social phenomena, however, an entirely different etiological question arises: "Why do some populations have much AIDS, while it is rare in others?" This type of question was asked by Rose when comparing the incidence of arterial hypertension between Kenyan nomads and civil servants in London. The answer, he found, "has to do with determinants of the population mean. What distinguishes the two groups has nothing to do with the characteristics of individuals; it is rather a shift of the whole distribution—a mass influence acting on the population as a whole" (Rose, 1985: 33). The determinants of this shift are the main subjects of reflection for "social epidemiology," a field of study that is still in its infancy and has not yet developed a clear theoretical framework (Krieger, 2001).

Whatever the reason for a particular "shift in distribution," it may appear rather insignificant and small yet yield spectacular differences in the population incidence of disease. This has been demonstrated for heart disease (Rose, 1985), suicide (Durkheim, 1951), homicide (Marmot, 1998), psychiatric illness (Rose, 1992), diabetes (McKinlay and Marceau, 2000), alcoholism (Marmot, 1998), syphilis (Kilmarx et al., 1997), and many other conditions of ill health. It is at the base of one of the most important discoveries in public health—the prevention paradox: "A preventive measure that brings large benefits to the community offers little to each participating individual" (Rose, 1992: 12).

If we select a few locations in sub-Saharan Africa with reasonably consistent series of epidemiological surveillance data for HIV and plot these data on a graph, the design we are most likely to obtain looks suspiciously like a box full of snakes. There is no recognisable pattern. Figure 1 shows an example of HIV prevalence over a nine year period among women attending ante-natal clinics in Zinguinchor (Sénégal), Abidjan (Côte d'Ivoire), Francistown (Botswana), and Mbarara (Uganda) (UNAIDS/WHO 2000).

This heterogeneity of HIV epidemics is well known. There are, of course, many explanations: The different time of introduction of HIV; the properties of different viral strains; the prevalence of underlying genital tract pathology; the prevalence of male circumcision, population mobility, and variations in behavioural norms. But one-dimensional causal theories are inept in predicting the profile and course of HIV epidemics. There are many examples of local epidemics that seem to defy conventional theories of risk. For instance:

- *Early age of sexual initiation among girls is associated with high prevalence of HIV.* In Ghana, about 60 percent of girls aged 15 to 19 are sexually active, compared to only about 30 percent in Zimbabwe (Singh et al., 2000). Yet, the HIV prevalence in Zimbabwe is more than five times as high as in Ghana.
- *Low prevalence of male circumcision is associated with high prevalence of HIV.* In Malawi, male circumcision is almost universal among the Yao and Lomwe people in the South while it is rarely practised in the North. Yet the HIV prevalence is generally higher in both rural and urban areas of the Southern Region than in the North. (UNAIDS/WHO 2000)
- *The prevalence of HIV in a society is related to the average number of sexual partners outside marriage and stable relationships.* Men and women in Côte d'Ivoire report consistently more "irregular sexual partners" than in Zambia (Caraël, 1995), yet the HIV prevalence in Lusaka is more than twice the rate in Abidjan.
- *The prevalence of HIV decreases when people use condoms more frequently.* Between 1994 and 1998 only 5.1 percent of Ugandans reported that they used a condom during intercourse with their last non-regular partner, compared to 22.2 percent of Kenyans and Zambians (combined population). Meanwhile, HIV prevalence in Uganda was falling rapidly while it was static in Zambia and climbing in Kenya (Stoneburner, 2000).

Sometimes we can make causal inferences on the basis of average behavioural profiles. For instance, the fact that settlements along major trucking routes in Africa have a higher than average HIV prevalence is most certainly related to the typical sexual behaviour of the men who drive trucks along these roads and the women who live in these settlements. But once we arrive at a behavioural

explanation, we still do not know why people at this time in this particular location behave in a way that puts their health in serious peril. Without this knowledge our ability to intervene is restricted to exhortation for behavioural change; an approach of doubtful effectiveness.

The complexity of this situation was stated bluntly by President Thabo Mbeki of South Africa in his opening speech of the XII International Conference on AIDS in Durban: “As I listened and heard the whole story told about our country, it seemed to me that we could not blame everything on a single virus” (Mbeki, 2000). An intelligent remark by a careful observer. Yet, it fuelled a storm of protest that most certainly damaged his political career.

In fact, even the most vociferous critics of President Mbeki have to admit that they have neither the theory nor the data to explain the widely diverging patterns of AIDS epidemics in Africa or anywhere else. What our preoccupation with examining risk factors has provided us so far is, in the words of McMichael, an explanation and quantification for the “bobbing of corks on the surface of waters, while largely disregarding the stronger undercurrents that determine where, on average the cluster of corks ends up along the shoreline of risk” (McMichael, 1995: 633).

There is, of course, the possibility that all epidemiological curves of HIV would look similar were it not for the success of national AIDS prevention efforts. Would the epidemic in Ziguinchor look like the epidemic in Abidjan had it not been for the political commitment of the Government of Sénégal to fight AIDS? Would the epidemic in Mbarara today look like the epidemic in Francistown had it not been for the vigorous response to AIDS by the Museveni Government in Uganda? We will never know and there is certainly no reason to discount or belittle the efforts of national prevention programmes. However, the profile of the HIV epidemic in Mbarara, Uganda looks a lot like the profile of the epidemic in Bukoba, Tanzania. The HIV prevalence in Ziguinchor, Sénégal is no lower than the prevalence in Nouakchott, Mauritania. It is highly unlikely that programming in Uganda and Sénégal spilled across the borders into Tanzania and Mauritania. The more likely explanation is that there are similarities within the two pairs of neighbouring locations that somehow resulted in similar experiences of HIV epidemics.

The social ecology of AIDS

“Ecology” is a descriptive term applied to complex relationships between organisms and their environment (Hawley, 1950). Its application to social phenomena, i.e. the conceptualisation of “social ecology” started about 25 years ago by a group based at the University of California at Irvine (Whiteley, 1999). Whiteley, quoting Dave Taylor, one of the founders of the social ecology theory, lists the six principles of social ecology analysis as follows:

- Identify a phenomenon as a social problem
- View the problem from multiple levels and methods of analysis
- Utilise and apply diverse theoretical perspectives
- Recognise human-environment interactions as dynamic and active processes
- Consider the social, historical, cultural, and institutional contexts of people-environment relations
- Understand people’s lives in an everyday sense

For example, an enquiry into breast cancer in a community, would look at the genetic make up, the diet, the social profile, the ethnic mix, the availability and accessibility of health services, the types of community support structures, the level of exposure to environmental toxins or radiation, the experiences of women who had undergone mastectomies, etc. The sources of information for such an enquiry would have to come from the field of genetics, nutrition, behavioural psychology, public health, anthropology, and many more. Each field would bring to it its unique theoretical perspective.

Such an enquiry would generate quite different results from a classical epidemiological study that may only tell us that the community has a high incidence of breast cancer because the women have too few children and eat too much fat. A social ecology analysis, on the other hand, would give us an idea why these and other risk factors for breast cancer are particularly prevalent in this community. What are the trade-offs for women in this community? Is the increased risk of breast cancer an acceptable trade off for the career opportunities afforded by not having children? What is reasonable nutritional advice, given the means and customs of the community? What is the experience of women with breast cancer? How do they cope and can the coping be improved?

When it comes to research on AIDS, we should think that an analysis of the social ecology is the standard and dominant form of enquiry. Pronouncements that AIDS is a social issue are ubiquitous and are part of any discourse on AIDS that aspires to political correctness. Every political document on AIDS written since 1995 includes the word “multi-sectoral” which, at least in theory, promises multiple levels and methods of analysis. Yet, beyond the jargon, the elements of a social ecology framework are rarely incorporated into the questions about and responses to AIDS. Our gaze remains “obsessively fixed on the individual and his/her responsibility” (Waterston, 1997). In fact, within the last five years we may have entered into “a new era of creeping absolutism,” where “biomedical advances are given premature credit for what they can achieve in HIV control” (Donovan and Ross, 2000:1897).

Why are most efforts to address AIDS in Africa based on reductionist analyses of causation at the individual level, although there is so much lip service paid to the “social” dimension? It is evident that there is more commercial interest in devising and implementing programmes centred on individuals. The real and potential profits in the pharmaceutical and biotechnology sector are well known and often talked about. But even non-profit development contractors delivering programmes for large donor agencies (from condom marketing to STD control, to voluntary counselling and testing) have an economic interest in the growth of these projects. By contrast, the economic opportunities created by an ecological and social policy analysis are relatively minor. While many people like to talk about cultural and social determinants of AIDS, there is not much fiscal incentive for anybody to translate this discourse into activities.

This, however, is not the only reason. There are also epistemological and methodological problems with the application of a social ecology paradigm to the issue of AIDS. We are quite clear about the outcome we want to explain and ultimately influence: the population incidence of HIV in a society. This information is available or can be obtained. The fact that we are measuring prevalence rather than incidence is irrelevant in the long run. But what are the exposure variables? “All societies are complex and multi-faceted. Each has innumerable features that might be measured and tested for their potential contributions to the configuration of health and disease” (Susser, 1987:194).

How can we do justice to this level of complexity? What are we looking for when we are looking for the cause of high or low population incidence of HIV? The tools of epidemiological risk factor analysis allow us to determine which among a number of chosen factors are “significant,” that is, highly likely to be associated with the level of HIV prevalence and not just by chance. This method does not tell us whether this is a causal association or the direction of the causation. More importantly, the method does not tell us which factor to look at in the first place. Whatever we select to be examined most certainly reflects our own bias and ideology. And if we select and test enough factors, we will always find a few apparently significant associations. That is an unalterable law of probability.

Epidemiological techniques of risk factor analysis are quite adequate in researching the causes of HIV infection in individuals. After all, the potential exposure variables are rather limited. These techniques, however, are woefully inadequate in constructing a causal theory for population incidence of HIV. Whatever set of exposure variables we examine will be based on our own theoretical or ideological biases. This does not mean that the findings will be wrong, they are just insufficient and biased by our own pre-selection. There is little doubt, for instance, that the prevalence of male circumcision influences the profile of HIV epidemics (Weiss, 2000), but how useful is this information in trying to understand and respond to the spread of HIV in a particular social context? People circumcise little boys for many different reasons and under many different circumstances. These reasons and circumstances themselves are linked to other factors that also have an impact on HIV transmission. Examining the effect of male circumcision by “holding everything else constant” is a useful approach for some purposes. Unfortunately in life “everything else” is never constant, and that complexity is lost in the analysis.

One large study of multivariate risk factor analysis was conducted by Over (Over, 1998). His analysis of urban HIV prevalence rates in 50 countries found eight aggregate variables that were able to explain more than half of the differences in national urban HIV prevalence rates:

- The age of the epidemic
- The per capita Gross National Product (GNP)
- The proportion of the population that is foreign born
- The proportion of the population that is Muslim

- The Gini coefficient[1]
- The male – female literacy gap
- The urban male/female gender ratio in the 20 to 39 age group
- The size of the military forces in proportion to the total urban population

The analysis confirmed much that was already known, offered some new insights, and raised many questions. However, the number of cases (50 countries) is small, especially when examining multiple variables. The selected exposure variables are but a few characteristics of 50 widely divergent societies. And almost half of the variation between the countries remains unexplained.

In 1994, a "study group on the heterogeneity of HIV epidemics in African cities" was formed to find explanations for the observed difference in the rate of spread of HIV. The findings of the group were published in 2001 as a doctoral thesis (Buvé, 2001), and soon after as a supplement to the Journal AIDS (Caraël and Holmes, 2001). The group selected population samples of about 1,500 young adults in four African cities. Two study populations, Yaoundé in Cameroun and Cotonou in Bénin, were known for their relatively low HIV prevalence. These study populations had HIV prevalence rates of 4.4 and 6.6 percent respectively. The other two cities, Kisumu in Kenya and Ndola in Zambia were known for their high HIV prevalence. The study populations in these two cities had HIV prevalence rates of 27.3 and 31.2 percent respectively. Using the population samples, the study group examined factors likely to affect the rate of HIV transmission in the four cities according to standardised research protocols. These included variables of sexual behaviour such as age of first intercourse and prevalence of concurrent sexual partnerships, prevalence of specific genital tract infections, male circumcision rates, and the prevalence of commercial sex.

The results of the "four city study" are surprising. Sexual behaviour, sexual practices, and condom use appear to have no impact on the rate of spread of HIV. For instance, in Yaoundé, a city with low HIV prevalence, men and women reported more lifetime and more concurrent sexual partners, and men reported more intercourse with sex workers than in Ndola and Kisumu where HIV prevalence was about five times higher. Condom use was about equal in all four cities. There were only three factors associated with higher HIV prevalence within each study population that were also more common in the "high prevalence" cities than in the "low prevalence" cities. These were lack of male circumcision, high prevalence of Herpes Simplex Virus II (HSV 2) infection, and being married.

It is plausible that the findings that the prevalence of male circumcision and of HSV 2 infection are important factors for the dynamics of HIV epidemics. The relationship between high HIV prevalence and early marriage, on the other hand, is unlikely to be causal, unless early marriage is always associated with early sexual debut. But the limitations of the study are clear. Randomly choosing comparable study populations in all four sites and comparing the frequency of individual characteristics among the four populations excludes any social characteristic from the analysis other than frequencies of behaviours. We know that there are more polygamists in Cotonou, that women in Kisumu marry at a younger age, and that men in Ndola are more likely to use a condom with a non-spousal partner. But we know nothing about the context in which these behaviours occur.

Deconstructing societies into quantifiable components or aggregating individual risk factor frequencies into a "social analysis" is the predominant approach used in positivist scientific reasoning. Positivism is based on the belief that if you observe, describe, quantify, and explain all the component parts of a system, you will generate an objectively real understanding of it. If you observe enough regularities of events to say that *x* causes *y*, you will be able to eventually decipher all universal laws of nature. About universality Susser noted: "Above the level of molecules, no biological entity can conform entirely to universal laws because of overarching contexts and the interaction between levels within a biological structure. The banal fact is that each society is influenced by its economic, political, and cultural circumstances as well as by its mix of peoples, climate, and topography. What is most universal is least biological and, most of all, least human" (Susser and Susser, 1996:675). We will have to come to terms with the fact that positivist causal reasoning is not very applicable to the study of populations within an ecological framework.

[1] The Gini coefficient is a measure of income inequality. It is the ratio of the area between the Lorenz curve and the 45 degree line and the area of the entire triangle. The coefficient approaches zero as the distribution of income approaches absolute equality.

If we cannot construct universal causal theories for the population incidence of HIV by analysing and comparing enough small generic units of the population, we are left with ecologic studies that relate the profiles of HIV epidemics to a description of the social environment in which they occur. This type of enquiry is not new. In the social science literature it is referred to as a constructivist or naturalistic enquiry (Lincoln, 1992). We will have to overcome three major obstacles. The first is the widespread belief that "ecologic analyses are not to be trusted" (Poole, 1994:715). The second is the view that anything that is not quantified is not "scientific." The third is the tendency of politicians and international civil servants to interpret and present reality in terms of their own success and, by implication, in terms of other people's failure.

We recently tried to reconstruct a historical view of the HIV epidemic in Zimbabwe using the approach of social ecology, that is, viewing the epidemic from multiple levels and methods of analysis, applying diverse theoretical perspectives, and considering the social, historical, cultural, and institutional contexts of people-environment relations (Decosas and Padian, 2001). The study, based entirely on the review of existing data, showed a major problem of applying a social ecology perspective: the question of scale. Zimbabwe has a mature, wide-spread HIV epidemic with a very high national HIV prevalence rate of approximately 25 percent among the young adult population. The colonial and post-colonial history of the country, of the distribution of wealth, the decade of economic decline, the uprooting of Shona culture by colonialist invasion, the disruption of families by the migrant labour system, and the highly divisive and parochial politics of the recent post-colonial era are all plausible causal contributors to the high population incidence of HIV. However, there is too much heterogeneity within the country, in terms of social organisation and in terms of the profile of HIV, to create a picture that goes beyond generalities. The description offers some explanations for the severe HIV epidemic experienced by the population of Zimbabwe but it does not provide much detailed guidance as to what can be done about it.

A similar analytic approach was taken by Barnett and Whiteside in their comparison of the five HIV epidemics in the UK, Botswana, Uganda, India, and the Ukraine (Barnett and Whiteside, 1999). The five case studies illustrate how susceptibility to HIV epidemics and vulnerability to the impact of AIDS vary from society to society and influence the profile of the population incidence of HIV. However, as the authors point out, the results of such an analysis are too general for practical purposes unless they are refined by additional parameters. This refinement is achieved by the development of a theoretical framework that aggregates the social exposure variables into two main parameters: The level of wealth and the degree of social control, order, or cohesion. The framework, described under the name "Jaipur Paradigm" in somewhat greater detail in a later publication (Barnett, Whiteside, and Decosas, 2000), has reasonable power to predict the shape of HIV epidemics at the national level, but it appears that its real value is in its application to social settings on a smaller scale: Refugee camps, villages, neighbourhoods in large cities.

Finally, a more empirical and data-driven approach to social ecology analysis was conducted in the United States in order to explain county-specific variations in the prevalence of syphilis (Kilmarx et al., 1997; Thomas et al., 1999). In practical terms, the main distinction between syphilis and HIV epidemics is the fact that the quality and availability of health care services directly influence syphilis prevalence. Before the advent of widely available anti-retroviral therapy, the main impact of heath care services on HIV epidemics was mediated by the medical treatment of conditions facilitating HIV transmission. Nevertheless, there are more commonalties than differences in the analysis of the population incidence of HIV infection and syphilis.

The first study (Kilmarx et al., 1997) was a multivariate analysis of sociodemographic characteristics to explain the variation of syphilis prevalence among approximately 3000 counties in the United States. The study used the same technique as the study by Over of social determinants of urban HIV prevalence described above (Over, 1998). It yielded five social, demographic and geographic variables that were able to explain 71 percent of the county differences in syphilis rates:

- Proportion of non-Hispanic Black population
- County location in the South
- Proportion of urban population
- Proportion of Hispanic population
- Proportion of births to women below 20 years of age

As in the Over study, these variables are interesting but offer little guidance for prevention programming. However, the researchers did not stop there. The second study in the series (Thomas et al., 1999) examined the 12 counties in the sample that had significantly higher or significantly lower syphilis prevalence than predicted by the regression model. This time the researchers used an entirely qualitative study design primarily involving observation and unstructured interviews. They found an array of additional social characteristics that were linked to high prevalence of syphilis:

- Highly unequal distribution of political power among different ethnic groups
- Poor or absent social organisation of minority ethnic groups
- Lack of employment opportunities
- A high level of inequality in income and social status
- Physical, cultural, and language barriers to access to health services
- Little interaction between social service agencies and the community
- Discord and competition between different social service providers and between service providers and community organisations (primarily churches)

These are exciting findings. They offer real opportunities for social programming in the locations of high syphilis prevalence. They also complete a theoretical framework that fits the data obtained by Over, the situation we found in Zimbabwe, and the framework developed by Barnett and Whiteside. The common thread found in all studies is that high population incidence of HIV (or syphilis) is associated with poor social cohesion. In national level studies this factor may be masked. Over's "proportion of Muslims," associated with low HIV prevalence, is likely a measure of social control and homogeneity. His "proportion of foreign born population," associated with high HIV prevalence, is likely measure of social diversity. What the Thomas study tells us is that the important exposure variables in the causal chain of a community's health profile are the way social groups in the community are organised and the way social, political, and economic power is distributed among them, rather than the level of diversity of community members. All four studies also found an association of a high level of economic inequality with high HIV or syphilis prevalence. Economic inequality is, however, one of the characteristics of non-cohesive societies. In fact, Wilkinson argues that poor social cohesion is the link between the well documented association of high levels of income inequality and poor health (Wilkinson, 1999).

Social cohesion

In Uganda, the overall HIV prevalence among women attending antenatal clinics peaked in 1991 at about 21 percent, a level of infection that is not surpassed in many countries. Still, while countries such as Zambia have experienced relatively stable antenatal HIV prevalence rates at about the same level (19 to 20 percent) from 1992 to 1998, the rates in Uganda declined relatively quickly, reaching a level of below 10 percent in 1998. There is good evidence and a general consensus that the main reason for the unprecedented rapid decline in Uganda was a shift in the behavioural profile of the population (Stoneburner, 2000). The question that has not been answered to anyone's satisfaction, however, is why this shift occurred so much sooner and faster in Uganda than in other countries. The