**Johnson & Wales University**
**ScholarsArchive@JWU**

Health & Wellness Department Faculty
Publications and Research

College of Health & Wellness

2010

# Concurrent partnerships as a driver of the HIV epidemic in sub-Saharan Africa? The evidence is limited

Mark N. Lurie
*Brown University*

Samantha R. Rosenthal
*Johnson & Wales University - Providence*, Samantha.Rosenthal@jwu.edu

Follow this and additional works at: https://scholarsarchive.jwu.edu/health_fac

Part of the Medicine and Health Sciences Commons

COMMENTARY

# Concurrent Partnerships as a Driver of the HIV Epidemic in Sub-Saharan Africa? The Evidence is Limited

Mark N. Lurie · Samantha Rosenthal

## Introduction

Sub-Saharan Africa contains only 3% of the global population but an alarming 68% of the world's adults and children living with HIV/AIDS (UNAIDS 2007). Sixty-eight percent of adults and children newly infected with HIV in 2007 also reside in this region (UNAIDS 2007). Although only 5% of adults are infected with HIV in Sub-Saharan Africa, no other region in the world has an HIV prevalence greater than 1% (UNAIDS 2007). There is also significant heterogeneity of HIV prevalence within sub-Saharan Africa with most countries in Southern Africa having an adult prevalence that exceeds 15% and some with adult prevalence as high as 27% (UNAIDS 2007).

Demographers and epidemiologists have been puzzled for decades over what factors explain the vastly different HIV epidemics seen across the globe. Some of the many plausible factors include high rates of other sexually transmitted infections which facilitate HIV acquisition and transmission, poor access to quality health care services, insufficient or ineffective prevention programs, poverty, high levels of migration and lack of male circumcision (Lurie et al. 2003; Piot et al. 2007; Wasserheit 1992; Williams et al. 2006). Since HIV is spread predominantly through sexual contact, sexual behavior has been the major research focus. Recently, Mah and Halperin (2008) and

others have proposed that concurrent sexual relationships—those that overlap in time—are a major driver of the HIV epidemic in sub-Saharan Africa. In this article we review the concurrency hypothesis and the evidence put forth by Mah and Halperin. We conclude that, despite their assertion that "enough evidence exists," the evidence base is in fact weak and that more targeted research with more refined definitions is needed.

Over the years researchers have investigated the effect of several aspects of sexual behavior on HIV transmission, including the frequency of sexual intercourse, the type, number and duration of partnerships, condom use, sexual mixing and network dynamics. The idea that concurrency may be important in HIV epidemic dynamics first entered the literature in the early 1990s (Watts and May 1992; Hudson 1993), but it was the work of Kretzschmar and Morris that receives the most attention (Kretzschmar and Morris 1996). Using microsimulation models, Morris and Kretzschmar found that, compared to a sexual network where most relationships are serially monogamous, in a network with a high degree of concurrency—defined by the authors as "simultaneous sexual partnerships"—HIV would spread more quickly (Morris and Kretzschmar 1997, 2000; Kretzschmar and Morris 1996).

Mah and Halperin's argument that concurrency *can* drive an epidemic stems from the insight that HIV will spread more rapidly in sexual networks with more partnerships that overlap in time compared to networks in which serial monogamy predominates (Mah and Halperin 2008). But whether concurrency actually *is* driving the epidemic in Africa is an entirely different question that mathematical modeling alone cannot address. Yet from these initial observations of the impact of different networks and partnering arrangements on HIV epidemic

M. N. Lurie (✉) · S. Rosenthal
Department of Community Health, International Health Institute,
Warren Alpert Medical School of Brown University,
121 S. Main Street, Rm. 221, Providence, RI 02912, USA
e-mail: Mark_Lurie@Brown.edu

dynamics, Mah and Halperin have concluded that concurrency is *the* driving force in sub-Saharan Africa's epidemic (Mah and Halperin 2008; Halperin and Epstein 2004). Others have gone so far as to accuse UNAIDS of failing to address concurrency in their recommended prevention interventions, and of *deliberately* covering up the evidence of the importance of concurrency (Epstein 2007, 2008).

Concurrency theory is controversial because there is no substantial evidence: (a) that levels of concurrency are significantly higher in Africa than elsewhere; and (b) that the observed levels of concurrency explain the African epidemics. Efforts to better understand these dynamics are further complicated by the fact that concurrency is difficult to measure and quantify and often vaguely and inconsistently defined. Much work remains to be done to accurately quantify the true contribution of concurrency to the current HIV epidemics in Africa. An examination of the literature reveals that the totality of evidence on which these claims are made is shaky and sometimes contradictory.

In reality, HIV epidemic dynamics are complex and unlikely to be explained by a single variable. Instead, a combination of factors likely drives the epidemic—with some factors playing a more important role in some geographic areas than in others. Nevertheless, understanding the main drivers of the epidemic is important in shaping future outreach programs, prioritizing interventions, and determining appropriate resource allocation. In order to appropriately critique the arguments put forth by Mah and Halperin (2008), it is important to first examine how concurrency has been defined and measured in the studies that have addressed concurrent sexual partnerships.

## Defining Concurrency

The fact that concurrency has been defined many different ways in the literature complicates the evaluation of concurrency as a driver of the HIV epidemic in Africa. Some authors define concurrency simply as sexual relationships that overlap in time (Halperin and Epstein 2004; Carter et al. 2007). Other definitions incorporate a specific time period of overlap, such as relationships that overlap for a period of at least 1 month. These types of definitions include only *ongoing* sexual relationships that overlap in time, while excluding "casual" partners or relationships of very short duration such as one or two sexual acts (Mah and Halperin 2008). The variety of definitions obviously hampers comparisons across studies.

Defining concurrency is difficult because it represents a spectrum of different types of sexual relationships with varying duration and overlap. Not all concurrency, however, results in the same level of risk on an individual or on members of a sexual network. Concurrency can include overlapping sexual relationships that last from as little as one sex act to decades. Participants in concurrent sexual relationships can have as few as two partners in their lifetime or as many as hundreds. For these reasons, it is particularly important that we strictly define concurrency and recognize that it can occur in many different scenarios, some of which may actually be protective.

Consider several different relationships, each broadly considered concurrent, but each conferring very different levels of risk on the individuals involved, and vastly different implications for HIV epidemic growth. The first example is a man who is in a long-term, polygamous relationship with two wives, none of whom have additional sexual partners. Or alternatively, a migrant man who has a wife at home and a long-term partner at work. The relationships in both of these examples are clearly concurrent—meeting the simplest definition that they overlap in time. However, these men can infect at most two other susceptible individuals since their sexual network extends only to two women, neither of whom has additional partners.

In a network with these kinds of concurrent relationships, $R_0$, the basic reproductive number, cannot exceed two. What is important then is not just the connectedness between individuals, but the broader network in which these relationships occur—in essence, your partner's partners (Aral 1999). And even a network with all concurrent partnerships will not necessarily result in a large epidemic if the concurrency is such that people have only a few relationships of long duration that overlap in time. In short, not all concurrency is alike, and some forms of concurrency may actually be protective. Mah and Halperin (2008) have categorized all concurrency as contributing to larger HIV epidemics, yet it is clear that it is essential to differentiate between different types of concurrent partnerships as not all concurrent partnerships pose the same risk.

## Measuring Concurrency

Concurrency is notoriously difficult to measure. Measuring sexual partnerships in general is complex—the validity and reliability of self-reported sexual behavior may be low, as is true for other sensitive topics (Cleland et al. 2004). Measuring concurrency adds an additional complication because it requires participants to recall the start and end dates of relationships, which may not always be easy or accurate (Garnett and Johnson 1997). Several different operational definitions for concurrency have been used in the literature.

One frequently used method asks study participants to recall how many sexual partners they had in the past 3 months. If the participant reports more than one partner,

the relationships are considered concurrent (Kalichman et al. 2007; Colvin et al. 1998). This very broad method of defining concurrency fails to differentiate between actual concurrency and serial monogamy. For example, a participant who reports two different sexual partners in the past 3 months may have: (a) had an ongoing sexual relationship with both partners throughout that period; or (b) ended a relationship at the beginning of that period and begun another thereafter.

A second method asks participants to identify any sexual relationship, without regard to duration, that occurred simultaneously with another ongoing sexual partnership (for example, Harrison et al. 2008). Carter et al. (2007) asked participants about their most recent three sexual partners in the past 3 months. For each sex partner reported, the participants were asked, "While sexually active with this person, did you have any other sexual partners?" Those participants who answered "yes" were considered to have had a concurrent sexual partnership. This method, unlike the first, differentiates between serially monogamous relationships and concurrent relationships. This method can also vary the time period or number of previous partners referenced according to the investigator's preference. One key shortcoming of this method, however, is that the duration of partnerships is unknown and the duration of overlap is unknown. This is critical information because a one-sex-act concurrent sexual encounter has very different implications for HIV transmission dynamics compared to a 1- or 2-year long concurrent sexual relationship.

A third method that is frequently used to measure concurrency asks study participants to recall the start and end dates of all sexual partnerships. This detail provides information about the number of partners, the timeline of partnerships, and precise periods of overlap. This is the most robust method of the three, but it also has limitations. Nelson et al. (2007) compared direct question methods for measuring concurrency with calendar methods and found that 29% of those reporting concurrent partners by direct question did not do so by the calendar method and 26% of those reporting concurrent partners by the calendar method did not do so by direct question. These and other data do not provide convincing evidence that we can accurately measure concurrency with any of the three methods used.

A unique and robust sociocentric approach to measuring sexual networks was undertaken in Likoma Island, Malawi (Helleringer and Kohler 2007), where adults were asked to identify up to five of their most recent sexual partners. Researchers then traced those partners from a roster of the island population. This unique and creative approach to measuring networks found that half of all sexually active respondents were connected in a large network component, and that more than a quarter were linked through multiple independent chains of sexual partnerships. Nevertheless

this approach is probably feasible only among relatively small isolated populations.

The multiple methods used for measuring concurrency in reality tend to measure quite different operational definitions for concurrency, yet have repeatedly been compared across regions. Without consistent measurement methods and definitions of concurrency, it is problematic to compare rates of concurrency across regions and across the world, as Mah and Halperin (2008) have done in arguing for support of the concurrency theory. To highlight some of these discrepancies in measurement, it is necessary to examine some empirical evidence.

## Empirical Evidence

Several study designs have been used to better understand the prevalence of concurrency and the role that concurrency may play in HIV epidemics. First, several studies have estimated the prevalence of concurrency in a population—that is, the proportion of people involved in concurrent sexual relationships in a given time period. Secondly, some studies have examined the individual-level association between being involved in concurrent relationships and the likelihood of having HIV. Third, very few studies have looked for an ecological association between the prevalence of concurrency in a population and the overall HIV prevalence in a population.

Descriptive studies that quantify the prevalence of concurrency in a population are most common and most cited to support Mah and Halperin's argument. These studies use a representative sample of a population to quantify the proportion of people who are participating in concurrent sexual relationships. Yet studies that quantify the amount of concurrency in a population—and that do not quantify HIV in that population—cannot and should not be used to argue that concurrency drives the HIV epidemic. The mere existence of concurrency says nothing about its role in HIV epidemic dynamics. In addition, these studies rarely use an operational definition of concurrency that includes the degree of overlap in sexual relationships. Finally, much of the data collected across studies are not comparable because of the inconsistent definition and measurement of concurrency.

These inconsistencies result in a wide range of reported rates of concurrency. For example, using a broad definition of concurrency, 11.3% of married Nigerian men aged 15–59 reported having "had extramarital sex in the past 12 months," (Mitsunaga et al. 2005). Among men in Kisumu, Kenya aged 18–24 who had sex in the past 12 months, 63% reported being in concurrent relationships (Mattson et al. 2007). The Kenya concurrency rates are much higher in part because the operational definition of

concurrency was taken from a *lifetime* sexual history. Information about every sexual relationship since sexual debut was collected for up to 12 partners. Concurrent partnerships were defined as partnerships in which the start and end dates of any two partners overlapped (Mattson et al. 2007).

This example shows that comparisons of concurrency rates between studies may not be valid. And the rates of concurrency in males do not accurately estimate rates in females since reported concurrency is significantly higher among men than women (UNAIDS 2003). Engaging in concurrent sexual relationships is also strongly associated with marriage. Therefore concurrency rates in unmarried individuals may not reflect those of married individuals (Sandoy et al. 2008).

In their recent review of global sexual behavior, Wellings et al. (2006, 2007) did not find enough comparable data to assess rates of concurrency in different regions of the world. However, the authors did find that the number of lifetime partners is lower in Africa than in industrialized countries, and that the prevalence of multiple partnerships is generally higher in industrialized countries. In addition, more men and women in Africa are sexually abstinent, with two-thirds of the population reporting recent sexual activity compared to three-quarters of the population in industrialized countries. This review of sexual behavior in 59 countries does not support the concurrency hypothesis because it shows that on average, African adults are less sexually active and have fewer lifetime partners than their counterparts in industrialized countries (Wellings et al. 2006, 2007).

In contrast, Halperin and Epstein (2004) reported that "of increasing interest to epidemiologists is the observation that in Africa men and women often have more than one—typically two and perhaps three—concurrent partnerships that overlap for months or years. This pattern differs from that of the serial monogamy more common in the west, or the one-off casual commercial sexual encounters that occur elsewhere." No reference is provided for the assertion that there is more concurrency in Africa than elsewhere.

Fewer studies have examined the association between participation in concurrent sexual relationships and individual HIV prevalence. The Kisumu, Kenya study found that those who ever had a concurrent partnership were slightly more likely to be HIV-positive than those who never had a concurrent partnership (age-adjusted OR, 95% CI: 1.7, 1.0–2.9), but the differences were only marginally statistically significant (Mattson et al. 2007). Mattson et al. (2007) defined concurrent partnerships as those which overlapped in time and were determined from dates obtained in interviews. In contrast, a study of men aged 15–26 in Eastern Cape, South Africa found no association between having had one or more *khwapheni*, or

"concurrent partners who are kept secret from a main partner" in the past 12 months and being infected with HIV (Jewkes et al. 2006).

These studies provide no information about the effects of population-level concurrency on the size of an epidemic. More importantly, these cross-sectional studies cannot determine whether engaging in concurrent sexual partnerships is an independent risk factor for having HIV because temporality is unclear and *association* does not equal *causation*.

Even fewer studies have investigated the population-level impact of concurrency on the size of the epidemic. By far the most rigorous of these studies was the Africa five-city study (Lagarde et al. 2001), which among other things tested the association between concurrent sexual partnerships and the prevalence of HIV infection in five urban communities of sub-Saharan Africa. Participants aged 15–49 in five African cities were given a standardized questionnaire measuring socio-economic characteristics and sexual behavior over the past 12 months, and were tested for HIV.

If concurrency was a main driver of the epidemic, then the levels of concurrency would necessarily have to be higher in countries with high HIV prevalence compared to those in low prevalence countries, *but they were not*. The prevalence of HIV infection in these cities varied dramatically, as did the proportion of relationships that were concurrent at the time of the interview. But the proportion of relationships that were concurrent was higher in the low HIV prevalence cities than it was in the high HIV prevalence cities (Lagarde et al. 2001).

Lagarde et al. utilized a measure of concurrency proposed by Kretzschmar and Morris, $k$, where $k = \sigma^2/\mu + \mu - 1$. $k$ measures the fraction of sexual partnerships that are concurrent at any given point in time using the mean ($\mu$) and the variance ($\sigma^2$) of the number of partners. The $k$ index was not higher in Kisumu and Ndola (where HIV prevalence was 25.9 and 28.4%, respectively) than it was in Cotonou, Yaounde and Dakar (where HIV prevalence was 3.4, 5.9, and 1%, respectively). Lagarde et al. were therefore *unable to find evidence that concurrent sexual partnerships are a major determinant of the size and spread of the epidemic*. The authors also constructed an individual indicator of concurrency to estimate an individual's propensity to keep or dissolve ongoing partnerships before engaging in another relationship. Like the $k$ index, this measure did not differ significantly between HIV infected and uninfected people.

Mah and Halperin dismiss the conclusions of this study on methodological grounds, but it may be their results, not the methods, to which they object. First, they claim that "the study measured *current* concurrency, rather than concurrency over a specific recall period." In fact, the study questioned participants about spouses and

non-spousal partners *over the past 12 months*. Mah and Halperin also criticized this study because "concurrency was only measured in the index case and not his/her partners." Although having complete information on the entire sexual networks in these communities would be optimal, measuring concurrency and HIV infection among the 9,643 participants *and* all of their partners would hardly be logistically or economically feasible.

Finally, Mah and Halperin criticize the study for having "no measure of partnership overlap recorded." This criticism is unfounded because the authors measured the duration of each non-spousal partnership over the past 12 months using the calendar method. The sum of time periods of overlapping partnerships for each individual was computed, averaged by community and compared to HIV prevalence. While this study has its limitations, it is the best empirical evidence and only population-level study that we have so far. And the authors concluded that they "could not find evidence that concurrent sexual partnerships were a major determinant of the rate of spread of HIV in 5 Africa cities" (Lagarde et al. 2001).

Similarly, a recent study using nationally representative Demographic and Health Surveys (DHS) and AIDS Indicator Surveys (AIS) in 22 countries (all but one of which was in Africa) concluded that "the prevalence of concurrency does not seem correlated with HIV prevalence at the community level or at the country level, neither among women nor among men" (Mishra and Assche 2009).

The lack of association found in these studies shows that the concurrency theory does not meet the dose-response relationship standard of Hill's (1971) criteria for causality and there is yet to be another population-level study that demonstrates this phenomenon. Studies like these are also subject to other potential biases—it is possible that other effects, for instance male circumcision or other, unknown or unmeasured variables, could have overwhelmed the true effects due to concurrency.

## Qualitative Evidence

The qualitative data presented by Mah and Halperin is hardly convincing. For while qualitative data may serve to highlight the "socio-cultural nature of concurrent relationships" it says nothing about the prevalence of these relationships in different cultural contexts, nor the degree to which these relationships have anything to do with HIV risk or transmission. Indeed qualitative interviews with the *right* people in nearly *any* country would reveal that some people are involved in concurrent relationships, which tells us a little, but not much. Furthermore, what tends to get reported in qualitative studies are the "interesting cases" which are often not representative and say little about the distribution of local social norms. It is therefore unlikely that individual qualitative studies will provide the level of evidence needed to conclude that concurrency is what is driving the epidemic.

## Modeling Concurrency

Existing empirical data does not support the theory of concurrency. Rather, the evidence that supports the argument of Mah and Halperin comes only from theoretical mathematical models. Even the results of mathematical models that explore concurrency are mixed; some find that concurrency is important and others find that factors such as number of partners or the patterns of age mixing are more important than concurrency (Morris and Kretzschmar 2000; Ghani and Garnett 2000).

Perhaps the first simulation model to examine the impact of different levels of concurrency in a population on the size of the HIV epidemic over time was published in the late 1990s (Kretzschmar and Morris 1996; Morris and Kretzschmar 1997, 2000). These models have frequently been cited as proof of the argument that concurrency drives the HIV epidemic. The model was intended to distinguish the effects of serial monogamy from those of concurrency at varying degrees. The authors ran 10 different simulations, comparing serial monogamy to varying degrees of concurrency (ranging from 0 to 66% of partnerships being concurrent). They concluded that concurrent partnerships exponentially increase the number of infected individuals and thus the initial growth rate of the epidemic. In addition, when one-quarter of partnerships are concurrent, the typical epidemic is three times as large by the end of a 5-year simulation compared to the same simulation with only serially monogamous partnerships.

These findings powerfully endorse the idea that concurrency *can* drive an epidemic because the degree of concurrency is shown to increase disease transmission far more than serial monogamy. It is important, however, to consider some of the parameters used in the model; as with all models, the choice of realistic parameters is complex. In this model, individuals involved in concurrent relationships were assumed to have between zero and four partners at a given time and an average partnership duration of 6–7 months. These assumptions may be unrealistic. The authors use 1994 data from Uganda for their baseline estimates (Morris and Kretzschmar 2000). These data actually present a different picture of baseline risk. The mean duration of spousal relationships was 239 months (9.9 years), and the mean duration of non-spousal relationships was 28.4 months, much longer than the parameters used in the model.

There is also reason to think that sexual activity rates are not as high as those used in the model. The model assumes

that every partnership consists of one sex act per day, meaning that a person with four concurrent partners has sex with each of the four partners every day. This assumption is highly unlikely. For example, in a recent nationally representative South Africa survey, 92% of men and 90% of women aged 15–24 report having sex less than five times in the past month (Pettifor et al. 2005).

In addition, the model assigns a constant rate of transmission of 0.05 per sex act which is significantly higher than documented rates of transmission (Deuchert and Brody 2007; Hollingsworth et al. 2008). For example, Leynaert et al. 1998 estimate that during each stage of HIV: acute (3 months), latent (8 years), and full-blown AIDS (2 years), the transmission probabilities are 0.0022, 0.0007, and 0.0015, respectively, with the average infection probability, (regardless of stage of disease and differences in male-to-female versus female-to-male transmission) being 0.0009. A simple calculation illustrates the impact of this vastly overestimated transmission probability: using Kretzschmar and Morris' transmission probability of 0.05 per sex act, the probability that an infected individual will infect his or her partner over a 6-month period with 3 sex acts per week is greater than 98%. By contrast, using Leynaert's more realistic transmission probability of 0.0009, the probability that an infected individual will infect his or her partner over a 6-month period with 3 sex acts per week is <7%.

It is also important to note that the latent stage of disease is by far the longest, and has the lowest transmission probability, far below the 0.05 estimate. Boily et al. (2009) recently estimated that infectivity in the acute and late phases of HIV infection were 9.2 and 7.3 times larger, respectively, than for the asymptomatic phase. The Morris and Kretzschmar model therefore could be improved by using more realistic transmission probabilities and by varying transmission probabilities throughout the course of the disease.

In 2000, Ghani and Garnett constructed an individual-based sexual partner network simulation model to test the effect of different sexual network behaviors on the risk of acquiring and transmitting sexually transmitted diseases (Ghani and Garnett 2000). This model showed that the degree of concurrency in sexual partnerships was significantly associated with risk of disease acquisition and transmission. Yet the same model showed that the number of sexual partners an individual has is even more important than concurrency. These results indicate that disease acquisition is driven by number of partnerships, whether concurrent or monogamous.

The most recent simulation model by Doherty et al. (2006) compared the effects of concurrency and mixing on disease prevalence. The simulation divided the population into three groups according to level of sexual activity: high,

moderate, and low, each with a different transmission probability. Mixing was defined as the formation of partnerships across different sexual activity groups. Doherty et al. concluded that mixing had a greater impact on disease prevalence than concurrency for all groups, and concurrency was a significant factor only for the lowest sexual activity group. These findings present mixing—not concurrency—as an important driver of HIV spread.

Another stochastic individual-based microsimulation model was created by Korenromp et al. (2000) using the model, STDSIM (van der Ploeg et al. 1998). This model compared sexual networks with: (1) prostitution and concurrent short relationships; and (2) those with no prostitution and concurrent short relationships to those with prostitution and serially monogamous short relationships. The model adjusts transmission probabilities by disease stage, type of sex act, and the presence of specific STIs. This robust model also accounts for several extraneous variables that are often neglected in other models, such as life expectancy, fertility, age of sexual debut, divorce rates, prostitution rates, condom use rates, and preferential assortative mixing by age. The results of this microsimulation model revealed that both sexual networks with short concurrent relationships had significantly higher rates of HIV prevalence after 30 years than the sexual network with only serially monogamous short relationships (Korenromp et al. 2000). Despite these findings, it cannot be determined whether these differences in HIV prevalence can be attributed to concurrency or a greater number of sexual partnerships because the number of partnerships was not held constant across sexual networks.

The reviewed mathematical models show that concurrency *can* play a part in HIV transmission. However, taken together, they do not wholly support the theory that concurrency *is* a critical driver of the Africa HIV epidemic. Several models that included concurrency found that other factors (i.e. the number of partners or the patterns of mixing) were more important driver; none of these models were mentioned by Mah and Halperin. Indeed, like the empirical data, the results from these models are far from conclusive.

## Conclusions

Taken together, the evidence that concurrency is driving the Africa AIDS epidemics is limited. There is as yet no conclusive evidence that concurrency: (1) is associated with HIV prevalence; (2) increases the size of an HIV epidemic; (3) increases the speed of HIV transmission; (4) increases the persistence of HIV in a population; or (5) that this relationship has a large magnitude of effect.

That concurrency *could theoretically* play a dominant role in transmission of HIV through networks is certainly

true, but this should not be taken to mean that *it is or it has* played that role. Little evidence supports the hypothesis that sexual behavior differs dramatically in Africa compared to the rest of the world, nor that sexual behavior in Africa is different in countries with high versus low HIV prevalence. And it bears repeating that the most rigorous field studies on this topic did not find evidence for an association between concurrency and HIV prevalence in five diverse African cities (Lagarde et al. 2001) or in 22 countries (Mishra and Assche 2009).

Theoretical plausibility—provided by the mathematical models—does not provide sufficient evidence to conclude that what *could* happen is actually what *has* happened. Without strong data showing that people have more concurrent partnerships in Africa than elsewhere and that places with high levels of concurrency also have high levels of HIV, we can only conclude that under certain conditions concurrency *may* be a significant driver of the HIV epidemics in sub-Saharan Africa.

To definitively answer this question, additional studies are needed. First, improved methods for measuring sexual behavior and particularly partnership duration and overlap are needed. Without a common definition of concurrency, it is futile to make valid comparisons across populations and between studies. Second, better study designs must be used. Current data comes from cross-sectional and ecological studies only. Longitudinal studies that prospectively measure concurrency and incidence of HIV infection are needed to validly assess whether concurrency causes increased transmission of HIV. Once there is evidence that concurrency is a causal factor that increases the risk of HIV infection, it will make sense to measure the effect of population-level concurrency on HIV epidemics. Whether concurrency is a significant driver of the HIV epidemic in sub-Saharan Africa is a question that has yet to be answered. Finally, designing prevention interventions around concurrency [as Mah and Halperin (2008) and others (Epstein 2007; Halperin and Epstein 2004) have suggested] without a better understanding of the intricacies of the relationship between concurrency and HIV transmission may well not produce the intended result of preventing new HIV infections.

## References

Aral, S. O. (1999). Sexual network patterns as determinants of STD rates: Paradigm shift in the behavioral epidemiology of STDs made visible. *Sexually Transmitted Diseases, 26*(5):262–264. doi:10.1097/00007435-199905000-00004.

Boily, M. C., Baggaley, R. F., Wang, L., Masse, B., White, R. G., Hayes, R. J., et al. (2009). Heterosexual risk of HIV-1 infection per sexual act: Systematic review and meta-analysis of observational studies. *The Lancet Infectious Diseases, 9*(2):118–129. doi:10.1016/S1473-3099(09)70021-0.

Carter, M. W., Kraft, J. M., Koppenhaver, T., Galavotti, C., Roels, T. H., Kilmarx, P. H., et al. (2007). "A bull cannot be contained in a single kraal": Concurrent sexual partnerships in Botswana. *AIDS and Behavior, 11*:822–830. doi:10.1007/s10461-006-9203-6.

Cleland, J., Boerma, J. T., Carael, M., & Weir, S. S. (2004). Monitoring sexual behavior in general populations: A synthesis of lessons of the past decade. *Sexually Transmitted Infections, 80*(Suppl. 2):1–7. doi:10.1136/sti.2004.013151.

Colvin, M., Abdool Karim, S. S., Connolly, C., Hoosen, A. A., & Ntuli, N. (1998). HIV infection and asymptomatic sexually transmitted infections in a rural South African community. *International Journal of STD and AIDS, 9*:548–550. doi:10.1258/0956462981922683.

Deuchert, E., & Brody, S. (2007). Plausible and implausible parameters for mathematical modeling of nominal heterosexual HIV transmission. *Annals of Epidemiology, 17*:237–244. doi:10.1016/j.annepidem.2006.10.011.

Doherty, I. A., Shiboski, S., Ellen, J. M., Adimora, A. A., & Padian, N. S. (2006). Sexual bridging socially and over time: A simulation model exploring the relative effects of mixing and concurrency on viral sexually transmitted infection transmission. *Sexually Transmitted Diseases, 33*(6):368–373. doi:10.1097/01.olq.0000194586.66409.7a.

Epstein, H. (2007). The invisible cure. Why we are losing the fight against AIDS in Africa. New York: Picador.

Epstein, H. (2008). AIDS and the irrational. *British Medical Journal, 337*:1265–1267. doi:10.1136/bmj.a2638.

Garnett, G. P., & Johnson, A. M. (1997). Coining a new term in epidemiology: Concurrency and HIV. *AIDS (London, England), 11*:681–683. doi:10.1097/00002030-199705000-00017.

Ghani, A. C., & Garnett, G. P. (2000). Risks of acquiring and transmitting sexually transmitted diseases in sexual partner networks. *Sexually Transmitted Diseases, 27*(10):579–587. doi:10.1097/00007435-200011000-00006.

Halperin, D., & Epstein, H. (2004). Concurrent sexual partnerships help to explain Africa's high HIV prevalence: Implications for prevention. *Lancet, 364*(9428):4–6. doi:10.1016/S0140-6736(04)16606-3.

Harrison, A., Cleland, J., & Frohlich, J. (2008). Young people's sexual partnerships in KwaZulu/Natal, South Africa: Patterns, contextual influences, and HIV risk. *Studies in Family Planning, 39*(4):295–308. doi:10.1111/j.1728-4465.2008.00176.x.

Helleringer, S., & Kohler, H.-P. (2007). Sexual network structure and the spread of HIV in Africa: Evidence from Likoma Island, Malawi. *AIDS (London, England), 21*(17):2323–2332. doi:10.1097/QAD.0b013e328285df98.

Hill, A. B. (1971). *Statistical evidence and inference. Principles of medical statistics* (9th ed.) (pp. 309–323). New York: Oxford University Press.

Hollingsworth, T. D., Anderson, R. M., & Fraser, C. (2008). HIV-1 transmission, by stage of infection. *The Journal of Infectious Diseases, 198*(5):687–693. doi:10.1086/590501.

Hudson, C. P. (1993). Concurrent partnerships could cause AIDS epidemics. *International Journal of STD and AIDS, 4*(5): 249–253.

Jewkes, R., Dunkle, K., Nduna, M., Levin, J., Jama, N., Khuzwayo, N., et al. (2006). Factors associated with HIV sero-positivity in young, rural South African men. *International Journal of Epidemiology, 35*(6):1455–1460. doi:10.1093/ije/dyl217.

Kalichman, S. C., Ntseane, D., Nthomang, K., Segwabe, M., Phorano, O., & Simbayi, L. C. (2007). Recent multiple sexual partners and

HIV transmission risks among people living with HIV/AIDS in Botswana. *Sexually Transmitted Infections, 83*:371–375. doi:10.1136/sti.2006.023630.

Korenromp, E. L., van Vliet, C., Bakker, R., de Vlas, S. J., & Habbema, J. D. F. (2000). HIV spread and partnership reduction for different patterns of sexual behaviour—a study with the microsimulation model STDSIM. *Mathematical Population Studies, 8*(2):135–173.

Kretzschmar, M., & Morris, M. (1996). Measures of concurrency in networks and the spread of infectious diseases. *Mathematical Biosciences, 133*:165–195. doi:10.1016/0025-5564(95)00093-3.

Lagarde, E., Auvert, B., et al. (2001). Concurrent sexual partnerships and HIV prevalence in five urban communities of Sub-Saharan Africa. *AIDS (London, England), 15*(7):877–884. doi:10.1097/00002030-200105040-00008.

Leynaert, B., Downs, A., de Vincenzi, I., et al. (1998). Heterosexual transmission of human immunodeficiency virus: Variability of infectivity throughout the course of infection. *American Journal of Epidemiology, 148*(1):88–96.

Lurie, M., Williams, B., Zuma, K., et al. (2003). The impact of migration on HIV-1 transmission: A study of migrant and non-migrant men, and their partners. *Sexually Transmitted Diseases, 40*(2):149–156. doi:10.1097/00007435-200302000-00011.

Mah, T. L., & Halperin, D. T. (2008). Concurrent sexual partnerships and the HIV epidemics in Africa: Evidence to move forward. *AIDS and Behavior, 22*. Epub ahead of print.

Mattson, C. L., Bailey, R. C., Agot, K., Ndinya-Achola, J. O., & Moses, S. (2007). A nested case-control study of sexual practices and risk factors for prevalent HIV-1 infection among young men in Kisumu, Kenya. *Sexually Transmitted Diseases, 34*(10):731–736.

Mishra, V., & Assche, S. B.-V. (2009). Concurrent sexual partnerships and HIV infection: Evidence from national population-based surveys. DHS Working Paper 62. Accessed at http://www.measuredhs.com/pubs/pdf/WP62/WP62.pdf on May 5, 2009.

Mitsunaga, T. M., Powell, A. M., Heardm, N. J., & Larsen, U. M. (2005). Extramarital sex among Nigerian men" polygyny and other risk factors. *Journal of Acquired Immune Deficiency Syndromes, 39*(4):478–488. doi:10.1097/01.qai.0000152396.60014.69.

Morris, M., & Kretzschmar, M. (1997). Concurrent partnerships and the spread of HIV. *AIDS (London, England), 11*:641–648. doi:10.1097/00002030-199705000-00012.

Morris, M., & Kretzschmar, M. (2000). A microsimulation study of the effect of concurrent partnerships on the spread of HIV in Uganda. *Mathematical Population Studies, 8*(2):109–133.

Nelson, S. J., Manhart, L. E., Gorbach, P. M., et al. (2007). Measuring sex partner concurrency: It's what's missing that counts. *Sexually Transmitted Diseases, 34*(10):801–807. doi:10.1007/978-1-59745-040-9.

Pettifor, A. E., Rees, H. V., Kleinschmidt, I., et al. (2005). Young people's sexual health in South Africa: HIV prevalence and sexual behaviors from a nationally representative household survey. *AIDS (London, England), 19*:1525–1534. doi:10.1097/01.aids.0000183129.16830.06.

Piot, P., Greener, R., & Russell, S. (2007). Squaring the circle: AIDS, poverty and human development. *PLoS Medicine, 4*(10):1571–1575. doi:10.1371/journal.pmed.0040314.

Sandoy, I. F., Dzekedzeke, K., & Fylkesnes, K. (2008). Prevalence and correlates of concurrent sexual partnerships in Zambia. *AIDS and Behavior, 8*. Epub ahead of print.

UNAIDS. (2003). AIDS epidemic update: December 2003. Geneva: UNAIDS.

UNAIDS. (2007). AIDS epidemic update: December 2007. Geneva: UNAIDS.

van der Ploeg, C. P. B., van Vliet, D., de Vlas, S. J., Ndinya-Achola, J. O., Fransen, L., et al. (1988). STDSIM: A microsimulation model for decision support on STD control. *Interfaces, 28*: 84–100. doi:10.1287/inte.28.3.84.

Wasserheit, J. N. (1992). Epidemiological synergy: Interrelationships Between Human Immunodeficiency Virus Infection and other Sexually Transmitted Diseases. *Sexually Transmitted Diseases, 19*(2):61–77. doi:10.1097/00007435-199219020-00001.

Watts, C. H., & May, R. M. (1992). The influence of concurrent partnerships on the dynamics of HIV/AIDS. *Mathematical Biosciences, 108*(1):89–104. doi:10.1016/0025-5564(92)90006-I.

Wellings, K., Collumbien, M., Slaymaker, E., et al. (2006). Sexual behavior in context: A global perspective. *Lancet, 368*:1706–1728. doi:10.1016/S0140-6736(06)69479-8.

Wellings, K., Slaymaker, E., Bajos, N., Collumbien, M., & Singh, S. (2007). Global sexual behavior. *Lancet, 369*(9561):557. doi:10.1016/S0140-6736(07)60270-0. author reply 557.

Williams, B. G., Lloyd-Smith, J. O., Gouws, E., Hankins, C., & Getz, W. M. (2006). The potential impact of male circumcision on HIV in Sub-Saharan Africa. *PLoS Medicine, 3*(7):e262. doi:10.1371/journal.pmed.0030262.