SOCIAL INTERACTIONS AND SOCIAL DISEASES

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Abstract

In this paper, we review what is known about the role of social and sexual networks in the diffusion of sexually transmitted diseases and clarify areas of demographic research that would benefit from new theoretical approaches, new data and new methods. Our focus is on HIV and other sexually transmitted diseases (STDs) that are among the most rapidly growing diseases through out the developing world with particularly high prevalences currently in sub-Saharan Africa (Behrman et al. 2006), but we also draw from research done elsewhere that is potentially useful to demographers. Our general conclusion adds to the evidence that sexual networks are the primary mechanism through which HIV is spread and transformed in Sub-Saharan Africa (SSA) by taking into account the influence of social networks on the structure and composition of sexual networks. We also conclude that theoretical perspectives from the network literature should lead to new data collection and the use of methodologies that are not standard in this area in order to understand better the social dynamics of social diseases and to be able to move from considering only associations to causal effects.

1 Social interactions

There is no doubt that the emergence of HIV/AIDS presents a challenging new situation that is associated both with considerable uncertainty about infection risks and with pressure to adopt innovative behaviors to moderate these risks. Not only is HIV/AIDS a new disease with a distinct epidemiology, but the strict prevention prescriptions promoted by international organizations and national governments—abstinence before marriage, fidelity after, and, if these are unacceptable, consistent condom use—are often at odds with pre-existing notions of the good life and norms/traditions about husband-wife interactions, sexual relations, reproduction and family organization (Caldwell 2000; Watkins 2004). The literatures on social networks and social interaction thus suggest that communication and interaction in social networks are likely to be important mechanisms through which individuals in developing countries in general and in sub-Saharan African countries in particular learn about the disease, its implications and consequences for individuals and families, and acceptable and effective strategies to reduce risk. At least two sources of empirical evidence support this hypothesis. *First*, experimental and empirical studies have revealed strong influences of peers on risk assessments, expectations and subjective beliefs (Fiske and Taylor 1991; Nisbett and Ross 1985; Rabin 1998; Scherer and Cho 2003). *Second*, in many developing countries where new ideals of small family size and new methods of fertility control have been introduced in the past few decades, several studies have documented that individuals have turned to others to help to evaluate these risks associated with low versus high fertility, and new versus old methods of fertility control. For example, analyses of both qualitative and survey data from Thailand, Ghana and Kenya provide evidence that women chat with each other about family planning and family size (Entwisle et al. 1996; Montgomery and Casterline 1993; Rutenberg and Watkins 1997; Watkins 2000) and AIDS (Watkins 2004; Watkins and Schatz 2001). Related studies on the determinants of contraceptive use in high-fertility areas have found strong influences of social interactions

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on demographic attitudes and behaviors (e.g., Entwisle et al. 1996; Kohler et al. 2001; Montgomery and Chung 1998; Munshi and Myaux 2006). Though qualifications are needed because the studies do not all control for the endogenous choices of social network partners (e.g., see Brock and Durlauf 2001; Manski 2000), on the whole these studies suggest that social networks play an important role in shaping the diffusion of family planning and low fertility in developing countries.

The mechanisms by which social networks affect the diffusion process can be summarized under the headings "social learning", "joint evaluation", and "social influence" (Bongaarts and Watkins 1996; Montgomery and Casterline 1996). For instance, at early stages of an innovation or social change such as the spread of HIV, individuals are likely to seek information about the transmission mechanisms of the disease, the risks of infections, and potential prevention strategies to reduce the risk of infection. A potential source of this information is interactions with friends, neighbors, or other members of an individual's social network. "Social learning" thus is defined as the process by which individuals (*a*) learn about the existence, epidemiology, risks, and prevention strategies of a new disease such as HIV?AIDS, and (*b*) reduce the uncertainties associated with the adoption of new behaviors that might reduce infection risks by drawing on the experience of network partners. Watkins (2004) argues that in contemporary rural Malawi the basic epidemiological facts of HIV/AIDS—that it is a sexually transmitted virus and that it is fatal—are no longer challenged but widely accepted. In contrast, the dominant prevention prescriptions promoted by international organizations and national governments—abstinence, fidelity, and condom use—are vociferously challenged. By "joint evaluation", we mean that within social networks, friends, relatives and neighbors jointly evaluate the strict prevention prescriptions: they (*a*) re-interpret them such that they are meaningful in the local context; (*b*) moderate them such that they appear more realistic; and (*c*) extend the list of prevention strategies with innovative approaches. For example, they consider whether fewer, more carefully selected, partners might substitute for strict abstinence and fidelity; whether religious communities might provide support for resisting temptations to engage in risky behavior; whether divorcing a spouse believed to be infected is preferable to consistent condom use within marriage. Finally, "social influence" emphasizes that preferences regarding sexual behaviors, gender relations or other HIV/AIDS-related behaviors are potentially affected by the opinions and attitudes that prevail in an individual's social environment. For example, network partners may express their disapproval of a man having sex with commercial sex workers, on the grounds that this exposes himself and his spouse to the risk of HIV/AIDS infection. Individuals may therefore change their preferences in response to normative pressures after interactions with others about the threat of HIV/AIDS. The direction of this social influence can differ in different stages of epidemic, and it can importantly differ across gender and family structure. For instance, the analyses by Helleringer and Kohler (2005) suggest that marriage patterns play a major role in defining the mechanisms of social interactions, and that women in the patrilocal region of Malawi (Rumphi in the northern region) have fewer opportunities to learn about threats to their sexual health though social interactions because networks are deeply embedded in her spouse's family and therefore impose tighter forms of social control and social influence. Kohler et al. (2006) use longitudinal survey data from rural Kenya and Malawi, and investigate whether social interactions—and especially the extent to which social network partners perceive themselves to be at risk—exert causal influences on respondents' risk perceptions and on one approach to prevention, spousal communication about the threat of AIDS to the couple and their children. The study explicitly allows for the possibility that important characteristics, such as unobserved preferences or community characteristics, determine not only the outcomes of interest but also the size and composition of networks. The key findings of the analyses include: *First*, and foremost, the analysis shows that social networks have significant and substantial effects on individuals' AIDS risk perceptions even when we control for unobserved factors that also may determine the nature of the social networks. Thus, to understand the dynamics and diffusion of behavioral change in response to AIDS it is essential to incorporate the impact of social networks. The failure to do so may lead to misunderstanding the dynamics of behavioral change. *Second*, this effect of social networks extends to the area of spousal communication about AIDS risk, and interactions with network partners—independent of network partners' risk assessments—tend to increase the probability of husband-wife communication about the disease. *Third*, the effects of social networks that are have found contribute to a better understanding

of diffusion. These effects are generally nonlinear and asymmetric. They are particularly large for having at least one network partner who is perceived to have a great deal of concern about AIDS. The inclusion of additional network partners with the same level of concern or with less concern generally has much smaller or insignificant effects. An exception to this asymmetry occurs in the network effects on spousal communication, where network partners, independent of their risk perception, have strong and significant effects. *Fourth*, social networks are associated with important social multiplier effects that reinforce the effects of AIDS prevention programs. For females, for instance, about one fifth of the program efforts influence on respondents' AIDS risk perception is mediated through social networks.

2 Social diseases

Theoretical network models have shown that individuals' positions within sexual networks, and the structural characteristics of the network itself, are important determinants of HIV infection risks and disease dynamics (Ghani and Garnett 2000; Kretzschmar and Morris 1996; Newman 2002). Several features of sexual networks that are predicted by these models to enhance the spread of HIV have been empirically documented in SSA including *concurrency* of sexual partnerships (Morris 1997), *skewed degree distributions* of sexual networks (Anderson and May 1991; Jones and Handcock 2003b), and *large and robust connected components* (Moody et al. 2003; Moody and White 2003).

We begin our review of the literature with the classical models of mathematical epidemiology (e.g., Anderson and May 1991; Bailey 1975), and then draw on our own work in rural Malawi as well as the work of others to show that some basic assumptions of these models are violated. In the most influential model, that of Anderson and May (1991), two key measures to study epidemics are are (1) the basic reproduction number, R_0, and (2) the final size of an epidemic s_∞. The basic reproduction number, R_0, is the expected number of secondary infections arising from a single, typical infectious individual in a completely susceptible population (Heesterbeek 2002). In a well-mixed and socially unstructured population, R_0 is the product of three quantities: the transmissibility τ, the rate of contact between susceptible and infectious individuals $\bar{c}$ and the duration of infectiousness δ. Epidemics are nonlinear phenomena and R_0 is a threshold parameter. When $R_0 > 1$, an epidemic is certain in a deterministic model and has non-zero probability in a stochastic model. Strategies for disease control and eradication are aimed at bringing R_0 below the threshold of unity, i.e, when the average infection generates fewer secondary infections than necessary for replacement and the epidemic fades. In the well-mixed and unstructured case, the final size of the epidemic is given by the implicit equation $\log(s_\infty) = R_0(s_\infty - 1)$, which has exactly two roots on the interval $[0\ 1]$ when $R_0 > 1$. The smaller of these roots is the proportion of the population remaining uninfected at the end of an epidemic.

One basic assumption of the classical models is that partners are selected randomly from a well-mixed and socially unstructured population. Our research in rural Malawi has shown that this is certainly not the case; rather, social processes, and particularly conversations in local social networks, play a key role in identifying and evaluating potential sexual partners (Watkins 2004). Even before the advent of AIDS, partners were selected by consulting with others about the behavior of a potential partner—for example, since sexual relationships were a form of evaluating potential marriage partners, the "good behavior" of the potential sexual partner was relevant for a man's choice to make a proposal to a woman, and for the woman's choice of accepting or rejecting his proposal. With the arrival of AIDS, the rules have been adapted in response to the threat of contracting a fatal disease. Thus, in making partnership choices, people draw on local knowledge of the health of individuals (e.g., are they known to have had a sexually transmitted infection?) as well as local knowledge of their sexual biography (e.g., did a previous sexual partner die of AIDS?)

Because the selection of sexual partners is a social process, one in which people employ varied and elaborate rules to choose their partners, the sexual networks through which HIV is transmitted in transmission dynamics in real populations are not well described by the classical epidemiological model. In particular, different rules of partner selection can produce sexual networks that either facilitate or impede the disease dynamics. Consider a number of examples drawn from the literature: (1) Rules of assortative mating can

structure a network into communities within which the disease spreads rapidly, but across which the spread is slow (Laumann et al. 1994; Laumann and Youm 1999; Morris 1993); (2) small world studies have shown that networks characterized by bridges joining otherwise disjoint clusters (Watts and Strogatz 1998; Watts 1999), can lead to thresholds and rapid disease diffusion to distant subpopulations; (3) robust networks, i.e., groups of persons tied together by more than one path in the sexual network, can decrease the ability to control the spread of HIV because redundant connections continue to transmit HIV even after some transmission paths are broken or eliminated (Moody et al. 2003; Potterat et al. 2002); (4) skewed degree distributions, i.e., networks containing individuals with very a high number of partners (high degree network members), can result in epidemics driven by promiscuous individuals (e.g., Liljeros et al. 2001; for a critical perspective, see Handcock and Jones 2004; Jones and Handcock 2003a).

While it is possible to study HIV disease dynamics taking these characteristics of human sexual networks into account using simulation models (Hethcote et al. 1991) and structured epidemiological models (Morris 1993), the lack of empirical information on sexual network structures for SSA limits the ability to properly calibrate these models, and to use them to study disease dynamics based on empirically-informed parameters. Despite the theoretical claims about the importance of networks for transmitting disease, the ability to empirically study HIV/AIDS infection risks and disease dynamics in SSA from a network perspective remains very limited because available data are only cross-sectional, often based on small populations, frequently restricted to ego-centric rather than complete networks, and with the exception of a pilot study by Helleringer and Kohler (2006b), not based on an integrated design that includes tracing of sexual networks, HIV testing, and extensive socioeconomic data for all members a population. Thus, without taking the newer theoretical perspectives into account, and without new data and methods with which to test these theoretical perspectives, we can only speculate about the changes of sexual networks during individuals' life-courses, the implications of different network structures for disease dynamics, the effect of individuals' sexual/social network position on the adoption of AIDS prevention strategies, the evolution of the HIV-1 virus as it is transmitted through sexual networks, and the potentially complex effects that intervention programs may have on the structure of sexual and social networks in a population, and ultimately the spread of the virus.

In 2005/06, Helleringer and Kohler (2006b) conducted a pilot study on Likoma Island on sexual and social networks to highlight, for the first time in a sub-Saharan context, to demonstrate some of the important findings that can emerge from an empirical network-based study of HIV. Likoma Island is located next to Chizumulu Island in the northern region of Lake Malawi. The population of the two islands is a little above 10,000 people and is comprised primarily of the Nyanja and Tonga ethnic groups. The overall level of economic development is quite low, and fishing is the main source of income for most households. On Likoma, 9% of young adults aged 18–35 are infected with HIV. The population of the islands is extremely young, as 50% of all inhabitants are below 15 years old, and there is a noticeable excess of women in Likoma and Chizumulu. Schooling is relatively widespread: most men between ages 15 and 49 have completed primary school. The median age at first birth in Likoma is 18 years, and among these births more than 50% take place out-of-wedlock. Indeed, marriages in Likoma happen relatively late (median age is 21 years for women and 26 for men) and are somewhat fragile (1/3 of marriages have ended after 10 years). Transportation to Likoma and Chizumulu Island is quite limited as only one boat travels weekly to the mainland of Malawi, although a few small canoes make daily trips to the Mozambican shore. Despite these constraints, however, inhabitants of the islands travel frequently: 2/3 of males and more than 1/2 of females had gone to mainland Malawi in the year prior to the survey, while almost 1/2 of males and 1/3 of females had gone to Mozambique over that same time span. Inhabitants of Chizumulu also frequently travel to Likoma (80% of them had gone there within the last year), in part because the maize outlet is in Likoma, but the converse is not true.

A striking characteristic of the sexual network elicited by the pilot study for Likoma Island is the existence of a giant connected component (Figure 1), in which 68% of male respondents and 58% of female respondents are embedded. The size of this component is significant because it determines the upper bound of a potential epidemic, i.e., the maximum number of people at risk if a disease is introduced within the population. However, there are *no* hubs, i.e., high degree individuals with a large number of network part-

ners, through which this connectivity arises. To the contrary, even though the giant component in Figure 1 contains most of the individuals with multiple sex partners, the vast majority of members of this structure report having had only one or two partners over the last 3 years. A giant cluster nevertheless emerges because somebody's unique partner may have other partners, who in turn have other partners, etc. While individuals in the giant cluster have a low rate of partner change, their risk of contracting HIV might be significantly higher than if they were part of smaller disjoint components. This observation is relevant in itself, but it is also important in the context of this proposal because it illustrates the need to collect data on (quasi) complete networks to analyze individuals' location within networks, and the local HIV risk-environments resulting from these network positions.

Even if giant components within a network exist, it is unlikely that all members of this component would become infected with HIV once the virus has entered the network. First, HIV has a fairly low infectivity (Gray et al. 2001), and second, the temporal ordering of relationships creates "breaks" in the paths the virus can travel (Moody 2002). However, if there also exist *cycles* in a sexual network—i.e., groups of person that are connected by more than one path—then the structure can enhance the potential epidemic spread of HIV (Moody et al. 2003). During a recent debate on the modes of HIV transmission in SSA, Brewer et al. (2003) argued that this structural feature was absent from sexual networks in SSA. In contrast to this assertion, however, the Likoma pilot study revealed an abundance of cycles and multiple paths between respondents. For instance, 45% of the members of the giant component were part of a *bicomponent* (Figure 1)—a highly cyclical structure in which individuals are connected together by (at least) two independent paths—that connects more than one third of the young adult population (40% of males and 30% of females) in the study villages.

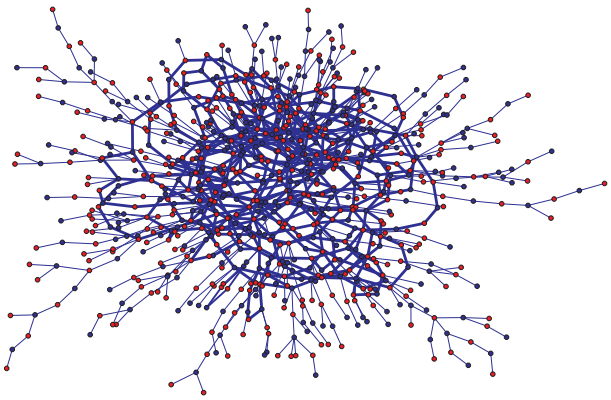

Figure 1: *Giant component*, including 68% of male respondents and 58% of female respondents. Individuals are represented by dots, and sexual relationships by lines; individuals in the large *bicomponent* (i.e. connected by more than one path) are linked by thick lines

Concurrent partnerships are widespread in Likoma, as in other parts of Malawi and SSA. At the time of the survey, respondents were involved in an average of 1.2 partnerships (Helleringer and Kohler 2006a). Calculating κ, the index of concurrency developed in Kretzschmar and Morris (1996), we found that almost 80% of active partnerships were concurrent. Concurrency in Likoma is considerably more widespread among young adults, but contrary to previously reported findings for SSA (Halperin and Epstein 2004), there is no marked sex difference in concurrence rates (25% of men, and 20% of women report more than one active relationship). Married women were 58% less likely to be concurrently involved with two or more partners than unmarried women, while marriage for men is associated with higher rates of concurrency.

Table 1 tabulates HIV prevalence by the number of sexual partners and the membership in small components of the Likoma sexual network (i.e., isolates, dyads, triads), the main component outside the bicomponent, and the bicomponent (connected by thicker lines in Figure 1). The table shows that the prevalence of HIV infection increases with the number of partnerships of a respondent (degree), and varies significantly with the respondent's position in the network. Moreover, while the prevalence of HIV is generally higher among members of the main component than among individuals in dyads or triads, respondents who are members of the bicomponent of the graph are *less* likely to be HIV positive than other respondents. Several factors may help explain this—at first sight counterintuitive—pattern. On the one hand, members of the bicomponent tend to be significantly younger than the rest of the population (age homophily); thus, while their network position may put them at increased risk of HIV infection, the short exposure implies still low HIV prevalence among their group, despite high connectivity. On the other hand, once infected, HIV-positive in-

dividuals may also drift to the periphery of the network (or into smaller components) as a result of choosing fewer partners, being selected as partner less frequently (Reniers 2005), or at advanced stages of the disease, due to poor health and reduced sexual activity.

3 Analysis, modeling and interpretation issues:

The central point of the following discussion is that special data in combination with sophisticated techniques of social network analysis, epidemiological modeling and econometric estimation are essential for assessing the role of sexual networks in fostering the spread and diversification of HIV, as well as evaluating the effects of changes in behaviors on the course and severity of the epidemic.

Table 1: HIV prevalence and network position, $N = 596$

Number of sexual partners		Network position: Small	Main	Bicomp.	Total
0	HIV prev.	0%	—	—	0.0%
	N	16			16
1	HIV prev.	6.5%	10.0%	—	7.5%
	N	107	40		147
2	HIV prev.	12.3%	11.9%	3.4%	10.3%
	N	57	59	29	145
3+	HIV prev.	12.5%	20.0%	4.3%	9.0%
	N	40	65	184	289
N		219	164	213	596

Network positions: *Small:* members of small components (isolates, dyads, triads); *Main:* members of the giant component, outside the bicomponent (Figure 1); *Bicomp.:* members of the bicomponent (**??**).

3.1 Analyses of data quality of sexual behavior reports:

Inaccurate reporting of sexual behaviors is a widespread problem in SSA (Cleland et al. 2004; Gregson et al. 2004; Mensch et al. 2003; Nnko et al. 2004) and other contexts. Inaccurate reporting of sexual activity is problematic because it not only undermines analyses that attempt to document and explain the behaviors, but also compromises program evaluations that attempt to determine whether interventions designed to improve reproductive health are effective. To reduce the potential measurement error for sexual behaviors and other sensitive behaviors, and to provide an assessment of the potential extent of measurement error for the analyses, various strategies have been proposed and in a few cases, used: (1) *Audio computer-assisted self-interviewing* (ACASI) techniques can be used for the collection of social and sexual network data (e.g., see Bloom 1998; Mensch et al. 2003). (2) *Reports of each party in a sexual relationship* can be used to asses the extent of discordance, and thus potentially misreporting, as well as the extent to which misreporting of sexual behaviors varies by the type of relationship and characteristics of individuals. In particular, because sexual relationships represent a *dyad* within a network, accurate reporting of sexual behaviors would imply that each relationship is reported by both members of the dyad. In reality, however, reports of sexual relationships are often discordant: they are reported by one, but not the other partner. Concordance rates, for instance, were around 50–88% for recent sexual and needle-sharing relationships among members of high-risk groups in the U.S. (Adams and Moody 2006; Bell et al. 2000), and around 47–67% in Helleringer and Kohler's (2006) study on the island fo Likoma in Malawi. (3) *microsimulation models*, such as (Bracher et al. 2003a,b), can be used to test the internal consistency of a variety of determinants and outcomes. For example, by employing such a model, one can test whether reported sexual behavior of relatively isolated components of the network structure is more or less consistent with HIV status in general than in the more integrated components of the networks.

3.2 Econometric analyses of networks and HIV/AIDS-related behaviors:

A major tool for investigations of social interactions and social diseases is econometric estimates. Particularly valuable are such estimates that exploit the longitudinal data, and capture the dynamic developments over segments of the life cycles of the sample members in the presence of unobserved endowments. Estimated relationships might include (*i*) the effect of an individual's position in sexual or social networks on important AIDS-related behaviors (e.g., onset of sexual activity, marriage, condom use, partner choice and

partner characteristics), (*ii*) the extent to which behaviors such as sexual activity, condom use, or the transition to marriage affect HIV-infection risks, and (*iii*) the effect of life-course transitions during adolescence and young adulthood (e.g., onset of sexual activity, first stable sexual relation, marriage, migration, HIV infection, death of partner) on the structure of an individual's social and sexual networks, and his/her position in these networks. In such estimates, individual fixed effects, instrumental variables and knowledge of the variance-covariance structure of the error matrix from the network data can be used to improve estimates of the determinants of one's position in the network structure and of the consequences of one's position in the network structure for HIV-infection risks and important AIDS-related behaviors.

3.2.1 Limitations of existing studies:

A number of existing studies in this field use selected samples, such as those who choose to attend antenatal clinics. The vast majority of studies in this field also abstract from dynamic multi-period life-cycle considerations with persistent fixed characteristics, such as genetic endowments related to health and tastes for taking risks. These limitations of existing studies imply biased or conceptually unsatisfactory estimates. For example, consider estimating the following relationship between some outcome such as one's perceived probability of having HIV, denoted H, and characteristics of one's networks N: $H_t = \beta N_t + \gamma X_t + \varepsilon_t$, where N_t is a vector of social and sexual network characteristics and X_t is a vector of other covariates included as controls in this regression; β and γ are parameter matrices to be estimated; the subscript t refers to the period in which the data were collected and ε_{it} is the disturbance term. Most studies estimating the impact of networks on outcomes such as the perceived probability of having HIV assume that N_t and ε_t are uncorrelated, i.e. $E[N_t \varepsilon_t] = 0$. However, ε_t is likely to consist of at least three components, μ, ν_j and η_t: μ denotes characteristics of the person not accounted for in N_t or X_t; for example, innate health; innate cognitive abilities; preferences related to socialization and sexual interaction; ν_j reflects locality specific factors that affect H_t but do not appear in X_t; for example, the risk environment, expectations regarding the returns from human capital investments; and η_t is a random disturbance term. Note that $E[N_t \varepsilon_t] \neq 0$ either because $E[N_t \mu_j] \neq 0$ or because $E[N_t \nu_j] \neq 0$, or both. Consequently, cross-sectional estimates of β are inconsistent and provide no meaningful evidence of the impact of a person's networks on his/her perceived probability of having HIV. We outline below a conceptual and analytical framework that explicitly incorporates and controls for the roles played by μ_j and ν_j to obtain consistent estimates of β,

3.2.2 General conceptual framework:

The conceptual framework for such econometric analyses considers a multi-period abstraction of processes over a segment of the life cycle in which in each period the individual's preferences (that are separable across periods) are maximized subject to end-of-the previous period capital stocks (state variables) and current and expected future period prices broadly defined and stochastic shocks. In each period t there is determined a vector Y_t of outcomes that include current outcomes such as the perceived current probability of having HIV H_t, current sexual interactions S_t, current consumption C_t, etc. and end-of-period capital stocks K_t. The essence of capital stocks is that they are carried over into future periods. Capital stocks include human, social, physical and financial capital. Human capital stocks include intellectual capital or education, physical capital (such as weight and body composition, i.e., capital in a biological sense), migratory status, marital status, and work experience. Weight, body composition, education and other forms of human capital are the outcomes of investments that are made in light of expected future payoffs as in standard human capital models (Becker 1967, 1991; Behrman et al. 1982, 1995; Grossman 1972). Social capital stocks include social and sexual networks that also reflect behavioral decisions in light of future payoffs, parallel to standard human capital models.

Within this framework, the outcomes for a particular individual in the t^{th} period can be viewed as the outcome of *dynamic decision rules* that are a function of *prices* P_t (broadly defined), *resources* R_t and *stochastic terms* U_t that the decision maker has faced in the past, faces in the current period, and expects to face in the future and *production technologies* (see below). *Prices* broadly-defined include market prices (such

as the cost of acquiring a calorie of energy from different foods), the cost of time, the risk environment (including with respect to contagious diseases) and expectations (such as for investments in human capital). *Resources* include individual resources (designated by a superscript i; e.g., innate health, own human capital social and sexual networks), family resources (designated by a superscript f; e.g., human capital of other family members, land and other assets and other stocks from the end of the previous period; and community resources (designated by a superscript c; e.g., health related services, options for changing networks, disease environment). Resources and prices may be *observed* (designated by a superscript o) or *unobserved* (designated by a superscript u) in the data. Thus the vector R (and likewise P) can be rewritten in terms of a number of subvectors—R^{io}, R^{iu}, R^{fo}, R^{fu}, R^{cu}, R^{co}. Consequently, Y_t can be viewed as the result of dynamic decision rules in which the right-side variables are P_t, R_t, and U_t. Information regarding past prices, past resources, and past stochastic terms at the individual, family, household and community levels is embodied in the capital stocks at the end of the previous period K_{t-1} that are the start-of-the-period resources for the current period. The current-period changes in individual and household capital stocks are determined in the current period, so they are not included among the right-side variables in these decision rules. Therefore for each period, linear approximations to these dynamic decision rules can be written as

$$Y_t = \alpha_0 + \alpha_1 P_t^o + \alpha_2 P_t^u + \alpha_3 K_{t-1}^{io} + \alpha_4 K_{t-1}^{fo} + \alpha_5 K_{t-1}^{fu} + \alpha_6 R_t^{co} + \alpha_7 R_t^{cu} + U_t, \quad (1)$$

where the α_i each are matrices of coefficients of the variables (themselves vectors), the elements in the vector Y include components relating to perceived probability of having HIV, current sexual relations, time use, consumption, productivity, etc., for the relevant individual and α_3 includes a subvector of coefficient estimates for end-of-previous-period social capital. For example, to estimate the impact of networks on own perceived probability of having HIV or sexual relations, we will estimate relation (1) with perceived probability of having HIV or sexual relations for Y_t and networks at the end of the previous period for a component of K_{t-1}^{io} to obtain an estimate of the related coefficient α_3.

3.2.3 Some general issues for estimating dynamic decision rules and their resolution:

Valid estimates of the critical parameters in relation (1) are essential to understand better the impact of social and sexual networks on outcomes of interest. However, various possible problems, if not addressed, could undermine the consistency of the estimates. For instance, valid estimates of relation (1) require recognizing (*i*) that there is a lagged version of this relation that determines the end-of-previous period networks, and (*ii*) how the process of network formation is related to still earlier periods. Analyses therefore need to be cognizant of the fact that networks at the end of the previous period are the outcomes of previous behavioral decisions in response to a number of observed and unobserved variables. In the next sections, we outline in more detail this and other problems that are most pernicious, but are ignored in standard estimates in the literature, and we discuss strategies to deal with them. Although at a statistical level, all the problems reflect the possibility of correlation of one or more regressors with the regression disturbance term (thereby making estimation procedures such as ordinary least squares inconsistent), we describe them in categories based on their underlying causes.

3.2.4 Endogeneity and omitted variable bias:

If unobserved right-side variables affect the outcome Y_t and are correlated with observed right-side variables in the same relation, and if these correlations are not controlled or eliminated in the estimation procedure, biased estimates result. The disturbance term in the actual estimation includes not only stochastic terms that are distributed independently of the observed right-side variables such as U_t, but also other terms related to unobserved characteristics because the "compound disturbance term" includes whatever is not observed in the data that enters into the relation. For instance, if innate health endowments and preferences for discounting the future directly affect the perceived probability of having HIV and also affect previous investments in social and sexual networks, then estimation methods must be used to control for the latter dependence to obtain consistent estimates of the effects of networks the perceived probability of having HIV. Three strategies

to deal with such problems include: (*a*) *Measurements for variables that usually are unobserved:* Some data sets have information on data on individual expectations, risk perceptions and detailed network-based measures of the risk environment. These data permit including such variables directly in the K^{io}_{t-1} vector instead of the K^{iu}_{t-1} vector in relation (1), which reduces the probability that the usual observed components of the K^{io}_{t-1} such as network characteristics are not in part proxying in the estimates for these usually-unobserved factors. Of course, as with other elements of the K^{io}_{t-1}, it is important to explore whether these variables are endogenous or exogenous (e.g., innate characteristics or formed in early childhood) to the relations being estimated. (*b*) *Fixed effects:* Longitudinal data permit control for unobserved fixed factors (e.g. innate health endowments, preferences, culture) that affected the end-of-the previous period capital stock (e.g., networks, health) in previous periods and that affect the current period outcomes (e.g., perceived probability of having HIV) directly. There are many examples of studies in the literature (e.g., Behrman, Kohler, and Watkins 2002; Kohler, Behrman, and Watkins 2006), in which control for such unobserved fixed effects alters the estimates substantially—implying that if the true model has such fixed effects, the practice in much of the literature of ignoring them is likely to lead to inconsistent estimates. (*c*) *Instrumental variable (IV) estimates:* If there are time varying determinants that affect, say, both the perceived probability of having HIV and networks, there may be simultaneity bias even with control for fixed effects. What are needed are instruments that affect sufficiently the right-side variables but do not directly affect the dependent variables. Some data sets include some instruments through the experiments. Measures of previous periods' risk environments, beauty, and prices also are candidates for being instruments if there also is control for fixed effects.

3.2.5 Sample attrition and selection for observing key variables:

Sample attrition and selection have the potential to invalidate inferences that can be drawn if the attrition or selection is not random with regard to the behavior being studied. Consider the canonical selection model that consists of a selection equation, $L^*_t = \beta_2 + \beta_3 X_t + \beta_4 Z_t + U^*_t$, and the relation of interest, $Y_t = \beta_0 + \beta_1 X_t + U^{**}_t$ (Y_t observed only if $L^*_t < 0$). As a result, the outcome variable, Y_t, is observed only for a subset of the entire sample, those for whom the latent index variable, L^*_t, is less than zero. If the error terms U^{**}_t and U^*_t are correlated, then estimation of$Y_t = \beta_0 + \beta_1 X_t + U^{**}_t$ ignoring the selection leads to inconsistent parameter estimates and thus incorrect inferences. What is of ultimate concern is not the level of attrition nor whether the means differ between "stayers" and "leavers" but whether, and to what extent, the attrition invalidates the inferences we make using these data. We will address sample attrition in two ways. *First*, if specifications of relation (1) include covariates that are associated with attrition, estimation biases due to attrition are reduced. Conditional on the maintained assumptions about the functional form, attrition selection on observed right-side variables does not lead to attrition bias (Fitzgerald et al. 1998a,b). *Second*, correction procedures for attrition and selection on observables are available that reweight observations based on the latent propensity to leave the sample as estimated from a probit for attrition as a function of baseline characteristics (Behrman et al. 2006, 2005; Fitzgerald et al. 1998a,b).

3.2.6 Measurement error in right-side variables:

Random measurement error in a right-side variable tends to bias the coefficient estimates of that variable towards zero (though, in a multivariate framework, the bias is unsigned). Fixed and instrumental variable procedures that are described above can control for such erros. In addition, fixed effect analyses can control for certain systematic measurement errors, such as fixed individual tendencies to over- or underreport sexual relations or other sensitive behaviors used as right-side variables.

3.3 Analyses of network structures:

In addition to the econometric analyses described above, an important concern of this review will be to describe analyses of the structure of the sexual networks within which HIV diffuses, and attempts to understand how sexual network structures evolve as the aggregate result of a large number of individual decisions

taking place in a changing environment (e.g., population composition, aggregate shocks, social networks, life-course transitions). These approaches involve manipulations and decompositions of the matrix of relations derived from linkages of survey data to obtain summary measures of connectivity and clustering. Indices such as density, geodesics, distribution of component sizes as well as the presence of cycles (among others) help summarize the overall structure, while various measures of centrality summarize the position of individuals within this structure (Wasserman and Faust 1994). Multidimensional scaling and principal components analysis are used to explore potential partitioning of the network into cohesive subgroups (based for example on preferences for"similar" partners) and how these may change over time (Freeman 2005). While mostly descriptive, these analyses are important for the formulation of hypotheses to be tested in a statistical framework. In addition, several network measures—e.g., network densities or the size of cycles or bicomponents—are closely connected to disease, and therefore provide important indicators of the intensity of HIV risks in the population. In addition to these descriptive analyses of networks, statistical and simulation models will be used to investigate network clustering and changes in the network structure over time (e.g., Handcock et al. 2003; Keeling and Gilligan 2000; Newman 2002).

4 Conclusions

In this paper, we review what is known about the role of social and sexual networks in the diffusion of sexually transmitted diseases and clarify areas of demographic research that would benefit from new theoretical approaches, new data and new methods. Our focus is on HIV and other sexually transmitted diseases (STDs) that are among the most rapidly growing diseases through out the developing world with particularly high prevalences currently in sub-Saharan Africa (Behrman et al. 2006), but we also draw from research done elsewhere that is potentially useful to demographers. Our general conclusion adds to the evidence that sexual networks are the primary mechanism through which HIV is spread and transformed in Sub-Saharan Africa (SSA) by taking into account the influence of social networks on the structure and composition of sexual networks. We also conclude that theoretical perspectives from the network literature should lead to new data collection and the use of methodologies that are not standard in this area in order to understand better the social dynamics of social diseases and to be able to move from considering only associations to causal effects.

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