Damned if You Do, Damned if You Don't: Religion and HIV-Risk Network Structure

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Recently researchers have begun investigating the relationship between religion and HIV. Many of these studies evaluate the role of religious organizations in the development of HIV prevention and intervention messages (Green 2003; Hearn 2002; Leibowitz 2002; Parry 2003; Pfeiffer 2004). To date, Trinitapoli (2006) is the only study to model individual HIV infection by religious affiliation and participation. Other previous work focuses on the link in less direct fashion. In an attempt to capture the broad-scale influence, Gray (2004) showed that among the few indicators he modeled that proportion Muslim was the only one negatively associated with HIV prevalence for 38 countries in SSA. The vast majority of studies however explore the link between religious participation and particular "risky" behaviors. For example, several studies show the association between religious affiliation and delayed sexual onset (Agha, Hutchinson, and Kusanthan 2006). Garner (2000) for example shows that among four churches in Kwa-Zulu Natal, South Africa, the Pentecostal church has lower levels of extra- and pre-marital sexual partnerships than other congregations. Hill and colleagues (Hill, Cleland, and Ali 2004) find a similar effect in Brazil for evangelicals, a category which largely overlaps with Pentecostalism in their sample. Others question whether there is a relevant connection between religious participation and declines in risky behaviors (Lagarde et al. 2000).

These studies all conceptualize HIV-risk as an individual level property. While reductions in risk behaviors are a vital component to reversing the HIV epidemic, such behavior modification (or others, including condom use) is only effective if universally adopted. The simulations here demonstrate how those who do not adopt such changes and engage in "risky" behaviors can affect risk both for themselves *and* those who have adopted behavior modifications such as faithfulness or condom use. This focus will move beyond the current literature's focus on how religious participation is helpful *or* harmful in regard to the present HIV epidemic, presenting one mechanism by which both can happen simultaneously. I draw on data that establishes the link between religion and HIV-risk behaviors to build simulation models of risk network structure. I illustrate network properties and generate epidemic curves that demonstrate how observed reductions in risk behaviors that result from religious participation can actually generate little-to-no effect on group level risk, and in certain circumstances can even correspond to increases in group level risk. I then incorporate estimated differences between reported and actual behavior changes into the models to investigate how pervasive norms in religious communities that are not universally observed can generate similar increases in group-level risk, based on changes in risk-network structure.

Existing work recognizes that HIV is contracted through pathogens that only pass through blood and other bodily fluids. Only particular types of contact can therefore put a person at risk of HIV infection, and those are the sorts of behaviors that present models of HIV attempt to explain. The assumption is that if researchers can understand the causes of these particular sets of behaviors, then models of intervention and prevention

can be better conceptualized. The implied model suggests that infection risk is a function of individual risk behaviors, which can be stated in probabilistic terms as:

$$\mathbf{P_{i(inf)} = C_i \times S_C + f(i, r, c, n) + e_i}, \qquad \mathbf{(1)}$$

where $P_{i(inf)}$ is the probability that an individual (*i*) will get infected as a function of: (a) C_i, the number of risky behaviors in which *i* engages; (b) S_C, the susceptibility of infection in a single exposure through contact by a particular risk behavior; and f(i, r, c, n) which represents a combination of any number of other factors, which could vary at the individual (i), religious organization (r), community (c), or even national (n) levels. Such models typically attempt to estimate C_i, as "risky" behaviors, for example as the number of extramarital or extra-relational sexual partnerships of an individual (i). Other times, they model a proxy for $C_i \times S_C$, for example as the adoption of condom use within such behaviors, which reduces S_C. Researchers estimate these models with any number of parameters among their independent variables (the f(…) portion of Eq.1); and have recently explained a considerable amount of variation in risky behaviors that is associated with religious participation (e.g., Agha et al. 2006; Hill et al. 2004; Trinitapoli and Regnerus, forthcoming), and have even begun to link these to HIV infection rates (Trintapoli 2006). Such models however tell only part of the story, and in effect are based more on the epidemiology of *non*-infectious diseases, like cancer, than on infectious diseases, like HIV.

This application of a "cancer model" of the relationship between risk behaviors and actual risk to the case of HIV and other infectious diseases has been discussed elsewhere (e.g. Morris 2004), but as yet has not been incorporated into the literature connecting religion and HIV. What these models fail to incorporate is that $P_{i(inf)}$ is a function not only of an individual's "risky" behaviors, but also of a number of properties of the alters (*j*) with whom individuals (*i*) engage in such behaviors. If for example an individual has 100 extra-marital partners, none of whom are infected, the risk of infection for that individual, S_C, is zero, even though their number of partners (C_i) is high. A better representation of the probability of infection for an individual would be:

$$\mathbf{P_{i(inf)} = \sum_j \left\{ C_{ij} \times S_{Cij} \mid P_{j(inf)} \right\} + f(i, j, r, c, n) + e_i + e_j}, \qquad \mathbf{(1a)}$$

where the differences from Eq. 1 are the inclusion of the sum ($\sum_j$) for all of *i*'s alters (*j*) of: (a) C_{ij}, the number of an individual's contacts (C_i) with each of *i*'s alters (*j*); (b) the inclusion of infection rates which are partner specific (S_{Cij}) and not uniform across all "risky" behaviors; and (c) the dependence of these infection rates on the given probability that each alter is infected ($\mid P_{j(inf)}$). For the present paper, I only incorporate the differences in Eq. 1 and 1a that occur in the (a) and (c) terms, although the independent variables included in later models could also incorporate properties of each alter f(*j*) as well as properties of each of the other factors separately for *i* and *j* (e.g., f(r_i, r_j)).[1]

There are numerous factors beyond C_i, which could constrain C_{ij}. This paper focuses on the way religious affiliation contributes to those constraints, and incorporates a range of estimates of those effects in the simulated networks described below. First, while previous work has established a link between religious affiliation and sexual partnering, no studies to date have shown how those individual level "risk" behaviors equate to changes in structural properties of risk. This first focus of the paper will

[1] Modeling pairwise infection probabilities (S_{Cij})

therefore examine how changes in C_{ij} (number of "risky" behaviors) change estimates of population level infection risk. Second, by incorporating the known prevalence rates of HIV infection for the population into the network models, I can also estimate the effects such changes can have on the probability that those behaviors take place with an infected partner ($P_{j(inf)}$), which is a difficult property to incorporate into individual models (for one approach, see Trinitapoli 2006).

It is well known that epidemiological models based on individual behaviors alone misestimate STI risk (Aral 2002), and that sexual network structure can explain differences that are not observed in individual level models. For example, Laumann and Youm (1999) find that sexual partnerships among blacks more frequently serve as network bridges than for whites. They show that even beyond individual level risk behaviors, the populations where blacks draw risk partners put them at much greater risk of STI than is the case for whites. Similarly, Moody and adams (2006) show that in one high risk population different risky behaviors differentially connect the network. They show that sexual ties form bridges across otherwise disconnected portions of the population, while needle sharing partnerships (which have higher transmission rates per contact) provide largely redundant connectivity, and therefore do not contribute substantially to the observed networks overall risk potential. To date no research examines how religious affiliation can influence the partnering patterns of sexual partnerships, which may similarly drive structural components of STI/HIV risk. The premise of this paper is therefore to combine the observed differences that arise in number of sexual partnerships by religious affiliation into models of risk network structure. While these changes in individual behaviors have been the focus of previous research investigating the link between religion and HIV-risk, little is known about how they contribute to network properties and therefore the structural properties of risk. In the simulations that follow, I demonstrate how the declines in risky behaviors associated with some religious affiliations can coincide with increases in group level risk.

I generate a series of networks, representing three populations, which are based on characteristics observed in data from the Malawi Diffusion and Ideational Change Project – Wave 3 (Networks 2006) (2004). The MDICP is a longitudinal household survey, which in wave 3 surveyed 1542 women and 1081 of their husbands in 119 villages in three rural districts of Malawi. For each simulated population, gender, religious affiliation and HIV status are each randomly assigned and held constant through variation of each of the other simulation parameters. The number of sexual partners for each simulated respondent is then assigned according to a series of reports on sexual partnership data from the Malawi Diffusion and Ideational Change Project, Wave-3 (described below). For each type of sexual partnership reporting, I assign sexual partners in the simulated populations to approximate the observed distribution of partnerships first randomly, then constrained by gender, then constrained by gender and religious affiliation. This generates a series of 240 individual-gender-religious affiliation constrained distributions of number of sexual partnerships. For each generated distribution, I then simulate 100 networks with the observed properties.

For each produced network, I calculate the size of the largest connected component and bi-component, and the number of people who are members of

components of at least size three and bicomponents of at least size four.[2] A component of a graph is a subset of the graph where at least one path connects all nodes, and is the largest possible range for epidemic spread. Similarly, a bicomponent of a graph is a subset of the nodes in the graph where at least two node-independent paths connect each node. Some have suggested bi-components as a minimal measure of potential STD-cores (e.g., Moody et al. 2006; Moody and White 2003). For each individual in the network, I then tabulate their membership in components of size three or larger, bicomponents of size four or larger, the largest connected component and the largest connected bicomponent. Finally, for each individual in each graph, I calculate his or her minimum number of steps away from an HIV infected individual. I then summarize each of these individual measures across religious affiliation as estimates of structural-risk properties. These simulations show that while religious affiliation has been reported to alter number of sexual partnerships, these changes do not produce corresponding changes in structural components of risk.

[2] A component of size three is the smallest group that reflects individuals who are, or who are tied to someone who is, involved in non-monogamous relationships. Four nodes as the smallest possible bicomponent here represents both the data's inclusion only of reports of heterosexual partnerships, and the property of bicomponents, which requires that they have at least four nodes to include 2 node-independent paths.

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