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Research Article

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Sexual networks, partnership mixing, and the female-to-male ratio of HIV infections in generalized epidemics: An agent-based simulation study

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Abstract

BACKGROUND
Empirical estimates of the female-to-male ratio of infections in generalized HIV epidemics in sub-Saharan Africa range from 1.31 in Zambia to 2.21 in Ivory Coast. Inequalities in the gender ratio of infections can arise because of differences in exposure (to HIV-positive partners), susceptibility (given exposure), and survival (once infected). Differences in susceptibility have to date received most attention, but neither the relatively high gender ratio of infections nor the heterogeneity in empirical estimates is fully understood.

OBJECTIVE
Demonstrate the relevance of partnership network attributes and sexual mixing patterns to gender differences in the exposure to HIV-positive partners and the gender ratio of infections.

METHOD
Agent-based simulation model built in NetLogo.

RESULTS
The female-to-male ratio of infections predicted by our model ranges from 1.13 to 1.75. Gender-asymmetric partnership concurrency, rapid partnership turnover, elevated partnership dissolution in female-positive serodiscordant couples, and lower partnership re-entry rates among HIV-positive women can produce (substantial) differences in the gender ratio of infections. Coital dilution and serosorting have modest moderating effects.

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CONCLUSION
Partnership network attributes and sexual mixing patterns can have a considerable effect on the gender ratio of HIV infections. We need to look beyond individual behavior and gender differences in biological susceptibility if we are to fully understand, and remedy, gender inequalities in HIV infection in generalized epidemics.

1. Introduction

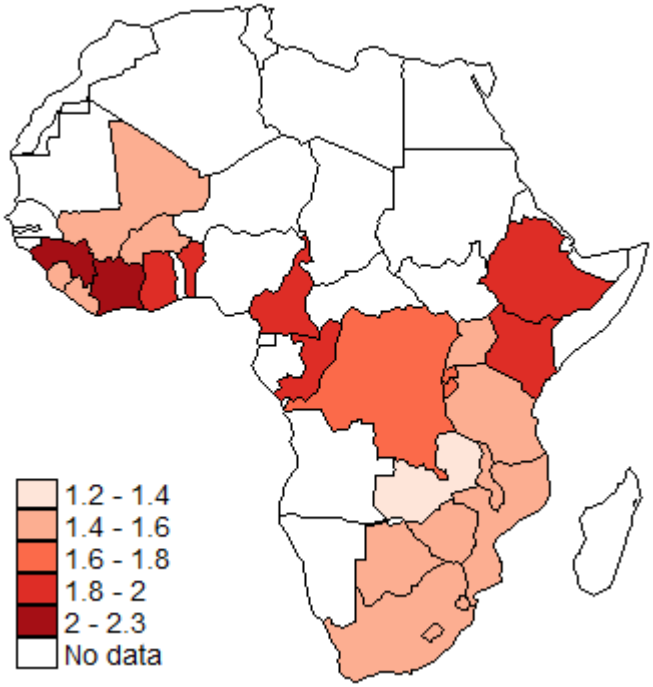
About 60% of adults living with HIV in sub-Saharan Africa are women, and that corresponds to a female-to-male ratio of infections of 1.48 (UNAIDS 2010).[4] Empirical estimates of the gender ratio of infections in African populations with an HIV prevalence level above 1% range from 1.31 in Zambia to 2.21 in Ivory Coast (Figure 1). The highest gender ratios are found in a string of African countries along or just above the equator. Interestingly, the gender ratio does not exceed 1.60 in the southeastern African countries that are most severely affected by the AIDS epidemic. Our understanding of this variation in the gender ratio of infections in generalized epidemics is rather weak and that is partly because two-gender serosurveys have only been conducted on a regular basis since the availability of rapid HIV testing technologies (i.e., since the early 2000s). However, caution is necessary when interpreting empirical estimates of the gender ratio of infections because they may be affected by relatively high male non-response bias (Barnighausen et al. 2011; Reniers and Eaton 2009).[5] Distortions in empirical estimates of the gender ratio can also arise from the restricted age range to which these apply (often 15−49) because the female-to-male ratio of prevalent infections is generally lower (in some cases reversed) at older ages.[6]

[4] By contrast, women account for 35% of all HIV positives in Central and South America (UNAIDS 2010). This estimate corresponds to a female-to-male ratio of infections of 0.54, and it is well understood that this large difference with generalized epidemics is related to the modes of transmission: the female-to-male HIV prevalence ratio is usually above unity in epidemics where heterosexual intercourse is the primary channel for new infections, and it is much lower in concentrated epidemics where sex between men or needle sharing among drug users are important epidemic drivers.

[5] It was precisely because of concerns over high refusal rates that the WHO initially chose to conduct anonymous surveillance in antenatal clinics (Chin 1990).

[6] Elaborating further on methodological issues in the measurement of gender inequity in HIV risk, it is worth noting that the gender ratio of prevalent infections, just as HIV prevalence itself, is a measure of the stock of infections over the whole reproductive age range. It thus includes recent as well as old infections, and it is dependent on the age structure of the population and gender differences in the survival of HIV positives (see below). Further, it is possible that higher levels of current infection in females in cross-sectional studies conceal equal or even higher cumulative HIV incidence among men (Gregson and Garnett 2000).

Figure 1: Female-to-male ratio of HIV prevalence in men and women of reproductive age (15−49, 2003−2011)



Data sources: ORC Macro (2013), Shisana (2005), and CSO [Botswana] (2009). Estimates are restricted to countries with a female HIV prevalence above 1%.

Aside from the data artifacts listed above, three categories of explanation exist for the relatively high female-to-male ratio of HIV infections in generalized epidemics: (1) women's exposure to infected men is greater than men's contact with HIV-positive women, (2) women's susceptibility or acquisition probability per coital act with an HIV-infected partner is higher than that of men, and (3) HIV-positive women survive longer than HIV-positive men. In this contribution we study some of the mechanisms that contribute to the first of these explanations, namely differences in exposure to HIV-positive partners. Our evidence comes from agent-based simulations wherein we model (gender-symmetric and gender-asymmetric) partnership concurrency, partnership turnover, coital dilution, HIV status-dependent partnership formation and dissolution, and serosorting as causal factors of interest. We do not intend to be exhaustive or explain empirically observed differences in the gender ratio of infections between

populations. Instead, we isolate a number of exposure factors that have not received much attention in discussions of the gender ratio of infections and demonstrate their pertinence. Before introducing the model we briefly review what is known about gender differences in exposure, susceptibility, and survival.

Gender differences in the survival of HIV positives arise because men tend to be infected at older ages (see below), and an advanced age at infection is negatively correlated with survival post-infection (Gregson and Garnett 2000; Todd et al. 2007). Gender differences in survival may also result from differences in the uptake or adherence to antiretroviral therapy (ART).[7]

Women's greater susceptibility is attributed to a number of biological mechanisms, including differences in genital immunology that are well described in the literature (Chersich and Rees 2008; Higgins, Hoffman, and Dworkin 2010; Yi et al. 2013). A variety of co-factors may alter susceptibility to HIV infection, including the presence of both viral and bacterial sexually transmitted infections (STI) (Cohen 2004; Glynn et al. 2001; Hertog 2008; UNAIDS/WHO 2000)[8] and male circumcision (Auvert et al. 2005; Hertog 2008). The contributions of pregnancy (Gray et al. 2005; Marston et al. 2013; Morrison et al. 2007) and hormonal contraceptives to women's disproportionally high infection rates are less certain (WHO 2012). Other factors with repercussions for HIV transmission pertain to the sex act itself, including vaginal versus anal sex, the use of vaginal drying agents, and forced sex (Chersich and Rees 2008). Finally, gender differences in ART uptake might alter the gender ratio of infections because onward transmission is reduced due to viral suppression.

Several studies of serodiscordant couples in high-income countries have confirmed the gender difference in susceptibility (Mastro and de Vincenzi 1996; Nicolosi et al. 1994), and it is now often assumed that women's acquisition risk per coital act is at least twice as high as that of men. However, estimates of the gender ratio of transmission probabilities per coital act for low-income countries are much more diverse and not consistently above unity. The sources for that heterogeneity are not well understood (Boily et al. 2009; Powers et al. 2008).

Of the class of explanations that revolve around gender differences in exposure, age mixing is best researched. Because men are often older than their female partners the exposure to HIV is possibly higher for women than for men, and age mixing has thus been proposed as an explanation for the relatively high prevalence rates in young women in particular (gender differences in HIV prevalence are more modest or even reversed in late adulthood) (Clark 2004; Gregson et al. 2002; Kelly et al. 2003; Leclerc-

[7] Early reports suggest that ART use is indeed higher among infected women than among men (Braitstein et al. 2008; Mills, Ford, and Mugyenyi 2009).

[8] STIs can influence the gender ratio of infections because they are more prevalent in women, or because they affect male-to-female transmission more than female-to-male rates.

Madlala 2008).[9] At least one study from Kwazulu-Natal challenges this view (Harling et al. 2014).

Studies addressing gender differences in other exposure factors are fairly limited. Worth noting, however, is the proposition that the gender ratio of infections is related to epidemic maturity: during the early phase of an epidemic, HIV infection is thought to be concentrated in female sex workers and their partners so that male prevalence exceeds that of women. With time, new HIV infections shift to long-term partners of those sex worker clients and the female-to-male ratio of infections may increase as a result of that (Carpenter et al. 1999; Gregson and Garnett 2000). This hypothesis thus revolves around the structure of sexual networks and its implications for the gender ratio of infections as the epidemic establishes itself in a population.

Network structure is also the prime concern in this study. More specifically, we compare the gender ratio of infections in monogamous sexual networks and sexual networks with different levels of gender-symmetric and gender-asymmetric partnership concurrency.[10] We introduce the distinction between gender-symmetric – assumed in the early modeling by Morris and Kretzschmar (1997) – and gender-asymmetric partnership concurrency because the latter is characteristic of populations that practice polygynous marriage. Empirical estimates of partnership concurrency, both formal (marriage) and informal, are also much higher for men than for women (Sawers 2013).[11] We also assess the compensating effect of a reduction in the per partner number of sex acts during episodes of concurrency (hereafter named 'coital dilution').[12]

We extend the analysis of concurrency effects to other attributes of sexual networks, including the rate of partnership turnover and HIV-status-based partnership mixing. The importance of fast partnership turnover for epidemic propagation has been known for quite a while (May and Anderson 1987), but its implications for the gender ratio of infections has not been described to date. We expect that rapid partnership turnover will maximize gender differences in HIV prevalence, provided that the susceptibility of men and women is indeed different: as partnerships last longer, the

[9] Young women's incidence rates are also high because of the immaturity of their genital tract, which elevates susceptibility. In other words, a relatively early sexual debut and age mixing increases women's exposure to HIV as well as transmission efficiency. In addition, a negative correlation exists between the age at infection and disease progression, and because women tend to be infected at younger ages they survive longer as HIV positives, which, in turn, elevates the gender ratio of infections in cross-sectional studies (Gregson and Garnett 2000; Todd et al. 2007).

[10] We refer to Morris and Kretzschmar (1997), Lurie and Rosenthal (2010), Mah and Halperin (2010), Sawers and Stillwaggon (2010), and Boily, Alary, and Baggaley (2012) for different points of view about the importance of partnership concurrency as an epidemic driver.

[11] A number of studies have suggested that reporting bias inflates these gender differences (Nnko et al. 2004), but gender differences are likely to persist even in the absence of reporting issues.

[12] Coital dilution is common in polygynous marriages; the evidence for coital dilution in other forms of partnership concurrency is mixed (Delva et al. 2013; Gaydosh, Reniers, and Helleringer 2013; Reniers and Tfaily 2012).

virus is more likely to spread to seronegative partners of HIV-positive individuals irrespective of the transmission probability, and the gender ratio of infections will come to depend more on other attributes of the network structure such as partnership concurrency.

A final set of sexual mixing patterns under consideration relate to HIV-status-based partnership formation and dissolution, namely (1) elevated dissolution rates in serodiscordant couples,[13] (2) lower partnership formation rates of HIV positives, and (3) serosorting or homogamy based on HIV status. HIV-status-based partnership choices will become an increasingly important factor in the epidemiology of HIV as the uptake of HIV testing and counseling (HTC) increases, but from previous research we also know that individuals often act on imperfect information about their own or others' HIV status (Watkins 2004). There are at least five studies that have suggested that partnership dissolution rates (through widowhood and divorce or separation) are significantly higher in serodiscordant couples, and particularly so in female positive serodiscordant couples (Carpenter et al. 1999; Floyd et al. 2008; Grinstead et al. 2001; Mackelprang et al. 2013; Porter et al. 2004). We will retain this gender imbalance in our simulations. Similarly, we develop scenarios with lower partnership formation rates among HIV-positive women. Such a pattern may arise from HIV-related morbidity, but also because those who are known or suspected to be HIV-positive are less desirable partners or withdraw from the partnerships market on their own initiative. Two studies from rural Malawi have identified these HIV-status-based partner recruitment strategies (Anglewicz and Reniers 2014; Reniers 2008). In combination with elevated dissolution rates in serodiscordant couples, the disproportionate recruitment of HIV-negative women into new partnerships causes the drift of HIV positives from the core to the periphery of sexual networks.[14] The drift of HIV-positive women also explains the relatively high HIV prevalence rates in the divorced and widowed compared to (re)married women in cross-sectional studies (de Walque and Kline 2012). Serosorting, in turn, has been well described among men who have sex with men in concentrated epidemics (Parsons et al. 2005; Suarez and Miller 2001), but has received little attention as a mediating factor in populations with generalized epidemics (Reniers and Helleringer 2011).

[13] Elsewhere we have argued that elevated partnership dissolution in serodiscordant couples reduces the spread of HIV (Reniers and Armbruster 2012).

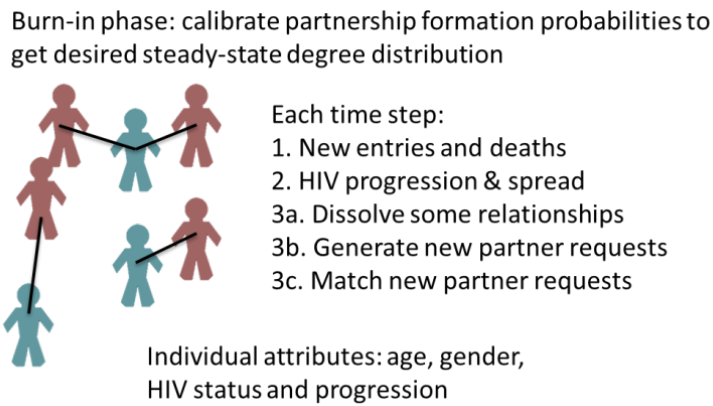
[14] We have borrowed this expression from Helleringer and Kohler, who offer it as one of the explanations for the distribution of HIV positives in their sexual network study of the Likoma Island in Lake Malawi. They found an over-representation of certain socioeconomic groups (e.g., older respondents, women, widows) in the sparser regions of the sexual network and suggest that they might have been infected when they were "closer to the dense regions of the networks but subsequently drift into smaller disjoint components" (Helleringer and Kohler 2007: 2330).

2. Methods

We demonstrate the effect of the sexual network structure and mixing patterns described above with a discrete-time agent-based model with one-month time steps built in NetLogo (Wilensky 1999). The code for the model is published in the NetLogo Modeling Commons as the HIV and Polygamy model[15].

The simulation tracks the characteristics of adult men and women and models their partnerships (Figure 2). We only account for heterosexual relationships and do not distinguish between formal and informal sexual partnerships. Each relationship has a constant hazard rate of dissolving, and each individual may have up to three relationships simultaneously. We do not, in other words, attempt to model networks with highly sexually active core groups such as commercial sex workers, but model a low degree network that is more characteristic of a generalized epidemic.

Figure 2: Modeled entities and processes



The rates for forming new partnerships are automatically selected so that the desired distribution of the number of partners for men and women in a user-defined scenario is attained. Table 2 contains the partnership degree distribution for each scenario. Before the start of the simulation of HIV transmission, the relationship part is run for 10 years to ensure that the initial partnerships distribution is in a steady state. Entry and exit rates from partnerships can be made dependent on HIV status, as is also

[15] http://modelingcommons.org/browse/one_model/4339

the case for the choice of future partners. Within the constraints defined in the scenario settings, partner choice is random.

HIV transmission is a key part of the model. The simulation tracks the HIV status of each individual as well as the stage of infection: acute, chronic, or AIDS. The acute stage and the final AIDS stage are 8 times and 4 times as infectious as the chronic stage, respectively. A woman in the chronic stage will infect any HIV-negative partner with a probability of 0.019 each month. The monthly transmission probability from an untreated man in the chronic stage to his partner is 0.038. In accordance with (some) empirical findings, we thus assume a greater susceptibility of women compared to men. These transmission parameter settings do not match one particular study, but fall in the range of values that have been reported in the literature (Boily et al. 2009; Wawer et al. 2005). Note also that these are monthly transmission probabilities and not the probabilities per coital act.

In scenarios with coital dilution, the probabilities are multiplied by a factor that represents the per partner reduction in coital frequency in partnerships with concurrency. More precisely, we multiply the monthly transmission probability by 0.8 if the index person has two partners, and by 0.6 if he or she has three partners. These assumptions imply that more concurrency is associated with a larger number of coital acts in the simulated population, and differ from another coital dilution modeling study that rests on the stronger assumption that higher levels of concurrency lead to a reduction in the population-level number of coital acts (Sawers, Isaac, and Stillwaggon 2011).

The model is initialized by randomly allocating HIV infection to 5% of the subjects. We have chosen 5% because we are primarily interested in the dynamics of generalized epidemics and not so much in the conditions that explain or characterize the early expansion of an HIV epidemic. The time of infection for these seed cases is set to match historical estimates of incidence in Williams et al. (2006). An infected individual is in the acute stage for the first three months after seroconversion; the last 10% of the lifespan is considered to be the final AIDS stage.

Our model focuses on adults aged 15−50. We assume that the number of males and females are equal, and match the 2009 age distribution for Zambia (UNPD 2013). We do not model specific patterns of age mixing in partnership formation. Individuals may die from AIDS or from causes unrelated to AIDS at an annualized rate of 6 per 1,000. To account for AIDS-related mortality, the length of a seroconvertor's lifespan is chosen from a Weibull distribution with a mean of 9.7 years and shape factor of 2.25. These parameter settings hold the middle ground between several empirical estimates, and assume that treatment is not available (Morgan et al. 2002; Todd et al. 2007). One further constraint is that the simulated population is held constant: each person who dies or turns 50 re-enters the population as an uninfected 15-year old individual of the same

gender without existing relationships. This constraint also precludes any appreciation of the implications of mother-to-child transmission for the gender ratio of infections among adults.

The global model settings and key scenario assumptions are summarized in Tables 1 and 2. We distinguish three types of sexual network: populations with monogamous partnerships only, populations with gender-asymmetric partnership concurrency (only men have concurrent partners), and populations with gender-symmetric concurrency. In each of these networks we vary the mean number of partnerships, and in the concurrency networks we also vary the level of concurrency. The quantity of partnerships is indexed by the mean number of partnerships per individual at any point in time (m), and the concurrency level is measured by the percentage of partnerships in the population that are concurrent (k) as defined by Morris and Kretzschmar (1997). A scenario with gender-asymmetric concurrency of level k=40 (and m=0.9) represents a population wherein 24% of the adult men have more than one partner at any point in time. In Togo, one of the countries with the highest polygyny rates in sub-Saharan Africa, 25% of the men aged 15−59 have more than one spouse (Anipah et al. 1999). In the symmetric case, k=40 implies a sexual network where about 12% of both men and women have multiple partnerships. Note that the scenarios with low and high levels of concurrency also differ in terms of the mean number of partnerships per person. The latter has implications for the interpretation of the results that we highlight later.

Table 1: Global model settings

Attribute	Value
Population size	1250
% HIV positive at t_0	5%
% on ART	0%
Female-to-male monthly HIV transmission rate (chronic stage)	1.9%
Male-to-female monthly HIV transmission rate (chronic stage)	3.8%
Acute infectivity ratio	8
AIDS stage infectivity ratio	4
Survival time post-HIV infection	Weibull with mean=9.7 years and shape=2.25
Background mortality rate	6 per 1,000
Simulation length	25 years

Populations with a low rate of partner change are those where the monthly partnership dissolution rate through separation or divorce is d=0.0167. We contrast those with networks wherein the monthly dissolution rate is d=0.0556. These separation rates translate into average partnership durations (in the absence of death) of 5 and 1.5 years, respectively. These levels are not necessarily chosen to match empirically observed patterns for entire populations, but could represent the partnership turnover rates in sub-populations.

Table 2: Scenario-specific settings

Mean number of partners (m) and level of concurrency (k)							
		Monogamy		**Asymmetric concurrency**		**Symmetric concurrency**	
	# partners	m=0.8, k=0	m=0.9, k=0	m=0.8, k=15	m=0.9, k=40	m=0.8, k=15	m=0.9, k=40
Male	0	0.2	0.1	0.3	0.4	0.25	0.25
	1	0.8	0.9	0.62	0.36	0.71	0.63
	2	-	-	0.06	0.18	0.03	0.09
	3	-	-	0.02	0.06	0.01	0.03
Female	0	0.2	0.1	0.2	0.1	0.25	0.25
	1	0.8	0.9	0.8	0.9	0.71	0.63
	2	-	-	-	-	0.03	0.09
	3	-	-	-	-	0.01	0.03

Coital dilution factor

Reduction in the monthly probability of HIV transmission (per partnership) if either partner has one or two concurrent sexual relationships

Partnership turnover

Partnership dissolution (divorce/separation) rates per month:

Low: 0.01665 → mean partnership duration: 5 years

High: 0.05560 → mean partnership duration: 1.5 years

Serosorting

Odd ratio of forming a new partnership with someone of the same (versus different) serostatus: 2

HIV-positive women's drift out of the partnerships market

Divorce/separation odds ratio in female-positive serodiscordant (F+M-) couples versus (F-M-couples): 3

Female-positive remarriage odds ratio (versus HIV- women): 0.5

Three parameters are used for manipulating HIV-status-based partnership mixing patterns. Scenarios with serosorting assume that the odds of forming a partnership with someone of the same serostatus are twice as high as the odds of forming a partnership with someone who is HIV serodiscordant. The drift of HIV-positive women is controlled by two parameters: one that sets elevated dissolution rates in female-positive serodiscordant couples (odds ratio=3) and one that sets lower remarriage odds in HIV-positive compared to HIV-negative women (odds ratio=0.5). For serosorting we do not have good empirical estimates to guide us with the parameter settings. By contrast, the settings for the drift of HIV-positive women are informed by empirical estimates from Porter et al. (2004).

We first present long-term epidemic trajectories (25 years) for populations with different partnership network structures, concurrency level, and partnership turnover rates. This is followed by an OLS regression analysis of the gender ratio of infections after 10 years. Models where the gender ratio of infections is log transformed produced a slightly better fit, but we have given preference to presenting the model with the dependent variable in its original form because of the easier interpretation of the coefficients. This choice does not affect any of the substantive conclusions. The regressions are separately done for the three types of sexual network structure (monogamy, gender-asymmetric concurrency, and symmetric concurrency) to allow for all two-way interactions with other predictors.

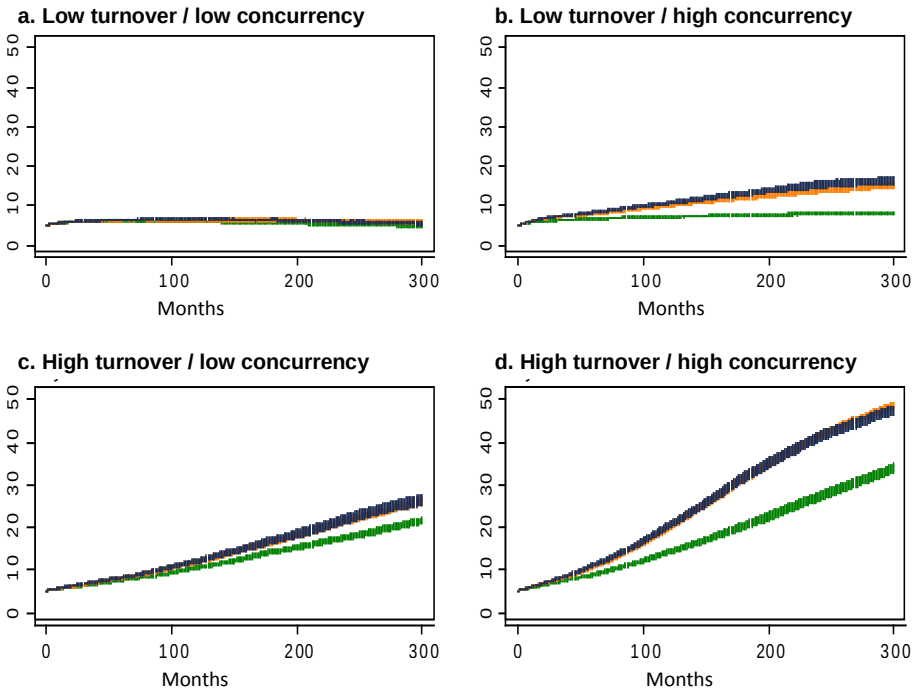
3. Results

The first outputs are trends in HIV prevalence (Figure 3). It is useful to gain an understanding of the magnitude and trajectory of the epidemics that are predicted by our simulations, even though it is not the focus of this study. In the epidemics portrayed in Figure 3 we do not assume any coital dilution or HIV-status-based partnership mixing. The plots highlight the contribution of partnership concurrency as well as elevated partnership turnover rates to the magnitude of the HIV epidemic. A comparison of strict monogamy (green bars) with the two concurrency scenarios (orange and blue bars) in each panel shows that partnership concurrency increases the epidemic size, but its effect is rather small at low levels of concurrency, and particularly in networks with low partnership turnover (the HIV epidemic never takes off in Figure 3a). In sexual networks with k=40, the epidemic size after 25 years is almost twice that of a monogamous population with the same average number of partners per individual (Figures 3b and 3d). The differences between symmetric and asymmetric concurrency are negligible (with the exception perhaps of networks with high concurrency and a low

partner change rate), and these results corroborate the findings of Santhakumaran et al. (2010).

The differences between networks with rapid and slow partnership turnover are more striking: irrespective of the network structure (monogamy, asymmetric or symmetric concurrency) and concurrency level, sexual networks with fast partnership turnover produce epidemics that are roughly four times as large (at 25 years) as in their variant with a low rate of partner change (Figure 3c versus 3a, and Figure 3d versus 3d).

Figure 3: HIV prevalence (95%-confidence interval) in sexual networks with low and high partnership turnover and various degrees of partnership concurrency, by network structure



The net effect of an increase in the mean number of partnerships from 0.8 to 0.9 partnerships per person can be evaluated for partnership networks with monogamy (only), and by comparing Figure 3a with 3b, and Figure 3c with 3d. Its effect is marginal in networks where partnership turnover is slow, but elevates the epidemic size after 25 years by about a third in populations where the partnership change rate is high.

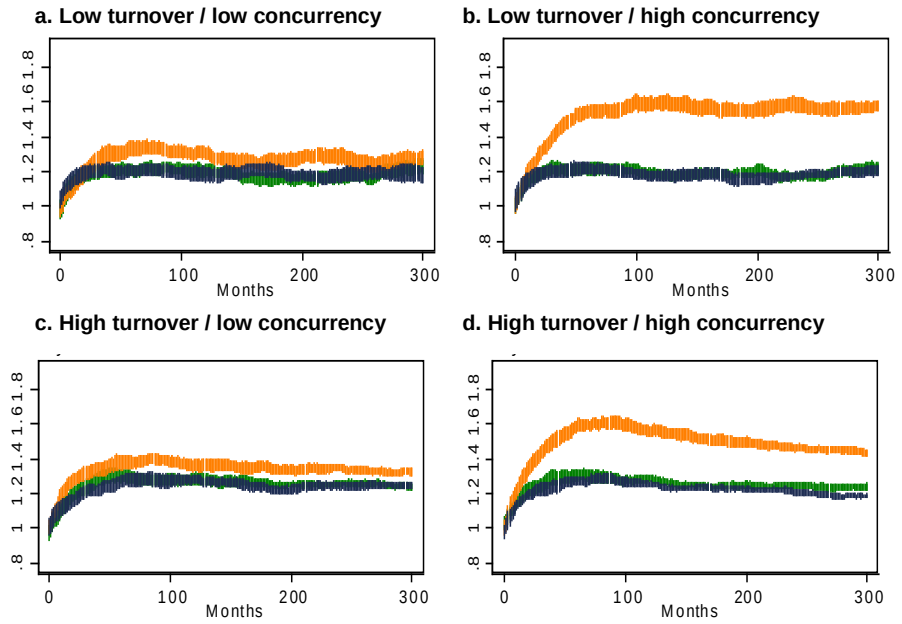
Trends in the gender ratio of HIV infections are illustrated in Figure 4. In populations with strict monogamy and gender-symmetric concurrency, the gender ratio of infections quickly rises from unity to about 1.20 (somewhat higher in networks with a higher partnership turnover rate). Thus, even though the monthly male-to-female transmission probability is set to be twice as high as the female-to-male transmission probability, the HIV prevalence in women is only about 20% higher in women than in men. The reason is that relationships usually last longer than a few months, and the importance of gender differences in transmission probabilities per coital act declines as the lifespan of partnerships increases. Networks with a higher rate of partnership turnover will therefore maximize gender differences in the susceptibility to HIV infection.

Differences in the gender ratio of infections in networks with low and high rates of partnership turnover are quite small compared to those induced by gender-asymmetric partnership concurrency: at high levels of partnership concurrency, the female-to-male ratio of infections even increases to 1.60. However, in a network with high concurrency and rapid partnership turnover (Figure 4d) the gender ratio decreases somewhat following a peak after 80 months. This decline is due to the disproportionally high female mortality associated with their elevated HIV prevalence, and the modeling assumption that all deaths and HIV-positive exits (at age 50) are substituted by an HIV-negative 15-year-old of the same sex. Given that the modeled HIV prevalence in a network with high partnership turnover and high concurrency is quite high (Figure 3d), this mortality effect is probably larger than we might observe in empirical populations, but it is nonetheless plausible that the gender ratio of infections declines somewhat once mortality starts taking its toll in populations where more women are infected than men.

The factors affecting the gender ratio of infections are further explored by means of an OLS regression analysis of the gender ratio after 10 years (Table 3). The regression models were fitted separately for each type of network structure (monogamy, asymmetric concurrency, and symmetric concurrency), which de facto allows for all two-way interactions between the other predictors and the network structure. Other interaction effects are less important and have been suppressed. The dataset for this analysis is based on 20 simulation runs for each combination of parameter settings. Even though there is no sampling variability in simulated data, and significance tests can be misleading given that we could readily inflate the sample size, we present the

coefficients with their 95% confidence interval to give readers a sense of the heterogeneity in the coefficient estimates that the simulations produce.

Figure 4: The female-to-male ratio of infections (95% confidence interval) in sexual networks with low and high partnership turnover and various degrees of partnership concurrency, by network structure



Legend: = monogamy, = gender-asymmetric concurrency, = gender-symmetric concurrency. Bars represent 95% confidence intervals. See Table 2 for a definition of the scenarios.

The explained variance (adjusted R-squared) in the female-to-male ratio of prevalent infections after 10 years ranges from 0.32 (asymmetric concurrency) to 0.58 (symmetric concurrency), which indicates that there is considerable random variation and that the gender ratio can be quite difficult to predict, even in simulations where all the parameters are determined without error.

The value of the intercepts – i.e., the expected values of the HIV gender ratio when all other covariates are set to 0 – ranges from 1.14 for populations practicing gender symmetric partnership concurrency to 1.38 for populations with asymmetric concurrency. The relatively large and positive coefficient for the variable identifying scenarios with high asymmetric partnership concurrency (and mean number of

partnerships) underscores that gender asymmetry in the sexual network structure can produce substantial differences in the gender ratio of infections. The effect of gender asymmetric concurrency on the gender ratio of infections is somewhat compensated by coital dilution, but, with the parameter settings that were used here, its effect is rather modest.

In a strictly monogamous population, the coefficient for an increase in the mean number of partnerships (the concurrency level is per definition zero) is negative, indicating that the importance of differences in biological susceptibility between men and women dissipates as men and women spend a larger fraction of their reproductive lives in a union.

Table 3: OLS regression of the gender ratio of infections after 10 years, by network structure

VARIABLES*	Monogamy	Asymmetric	Symmetric
Concurrency and coital dilution			
Intercept	1.25	1.38	1.14
	(1.231 - 1.269)	(1.358 - 1.401)	(1.123 - 1.166)
↑ Concurrency/Quantum	-0.1	0.11	0
	(-0.115 - -0.086)	(0.093 - 0.126)	(-0.014 - 0.018)
Coital dilution	-0.01	-0.06	0.03
	(-0.028 - 0.002)	(-0.079 - -0.046)	(0.009 - 0.042)
Partnership turnover rate			
↑ partnership turnover	0.06	0.01	0.11
	(0.042 - 0.071)	(-0.003 - 0.030)	(0.091 - 0.123)
HIV-status-based mixing			
↑ Divorce (F+M-)	0.13	0.12	0.24
	(0.119 - 0.148)	(0.101 - 0.134)	(0.227 - 0.259)
↓ F+ Remarriage	0.11	0.11	0.22
	(0.096 - 0.126)	(0.096 - 0.129)	(0.207 - 0.239)
Serosorting	-0.02	-0.02	-0.02
	(-0.035 - -0.005)	(-0.036 - -0.003)	(-0.034 - -0.002)
N	1,280	1,280	1,280
Adjusted R-squared	0.382	0.321	0.584

Notes: 95% confidence intervals in parenthesis.

* All predictor variables are dichotomies: ↑ *Concurrency/Quantum*: scenarios with high concurrency and mean number of partnerships (versus low); *coital dilution:* scenarios with coital dilution (versus without); ↑ *partnership turnover:* scenarios with high partnership turnover (versus low); ↑ *Divorce (F+M-)*: scenarios with an elevated separation rate in female-positive serodiscordant (versus without); ↓ *F+ Remarriage:* scenarios with reduced re-entry into partnerships for HIV-positive women (versus without); *Serosorting:* scenarios with serosorting (versus without). More detail about the scenarios is given in Table 2.

From Figure 4 we learned that populations with high rates of partner change tend to maximize the gender ratio of infections. In the regression analysis this is confirmed by small but positive coefficients. Its effect seems to be the smallest for populations

practicing asymmetric concurrency, and that is because these networks produce particularly large epidemics with disproportionally high AIDS mortality among women. In the simulations these deaths are replaced by 15-year-old HIV-negative subjects of the same gender, which keeps the gender ratio of infections in check.

The drift of HIV-positive women from the partnership market has a substantial effect on the gender ratio of prevalent infections. Depending on the network structure, the drift elevates the gender ratio of infections by 0.23 to 0.46 points. In our simulations, the exclusion or retreat of HIV-positive women from the partnership market increases the demand for HIV-negative women, who are now disproportionally recruited into new partnerships and exposed to HIV infection. The effect of the drift is particularly strong in networks with symmetric partnership concurrency, because HIV-positive women are now less likely to act as central nodes with multiple partnerships, which further reduces the exposure for men in the population. The effect of serosorting on the gender ratio of infections is small, if not negligible.

The female-to-male ratios of infection predicted by our model range from 1.13 for a network with gender-symmetric concurrency, low partner turnover, and without the drift of HIV-positive women, to 1.75 for a population with gender-asymmetric concurrency, high partnership turnover, and the drift of HIV positive women.

4. Discussion

Gender differences in the susceptibility to HIV infection have received considerable attention and are deemed important determinants of gender differences in HIV prevalence in generalized epidemics. However, the empirical support for gender differences in the acquisition probabilities per coital act is fairly limited, particularly for low-income countries. In addition, relatively large gender differences in susceptibility do not result in equally large differences in HIV prevalence. It can be analytically shown that the steady-state gender ratio of infections converges towards the square root of the susceptibility ratio in the simplest possible heterosexual differential equation transmission model with random partnership mixing. Put more simple, this means that gender differences in acquisition probabilities per coital act will start to matter less as partnerships last longer. In our simulation study this is corroborated by the fact that the estimated gender ratio of infections 10 years into the epidemic is 1.25 in the baseline monogamous sexual network, even though the monthly male-to-female transmission probability was set to be twice that of female-to-male transmission. The higher gender ratio of infection in populations with an elevated rate of partnership turnover lends further support to this conclusion, and confirms that networks with rapid partnership turnover will maximize gender differences in susceptibility.

Empirical estimates for the gender ratio of infections in most southeastern African populations with large HIV epidemics range between 1.40 and 1.60, and it is thus unlikely that differences in susceptibility fully account for these large gender differences in HIV prevalence. Co-factors that are known or believed to affect the transmission probabilities include male circumcision, the prevalence of STIs, and hormonal contraceptives. Women may also survive longer with HIV, which is related to their relatively young age at infection. All these factors could in theory raise empirical estimates of the gender ratio of infections observed in generalized epidemics, but were not the subject of this study.

Another class of explanation focuses on gender differences in the exposure to HIV-positive partners, and it is in this realm that our simulations contribute most to the understanding of gender differences in HIV prevalence. Best described in the literature is age mixing, which explains the relatively high prevalence rates in young women. Using agent-based simulations, we further demonstrate the importance of (1) gender-asymmetric partnership concurrency and (2) the drift of HIV-positive women out of the partnership market. Coital dilution (in networks with asymmetric partnership concurrency only) and serosorting have relatively modest moderating effects on the gender ratio of infections.

The scenarios with asymmetric concurrency and the drift of HIV-positive women are informed by empirical studies that consistently identify higher levels of partnership concurrency among men compared to women, and the gradual exclusion or retreat of HIV-positive women from partnerships. However, the results also depend on the assumption that the population-level quantity of partnerships is not correlated with the network structure or the partnership mixing patterns that are modeled. This constant partnerships assumption is common in sexual network simulation studies because it allows us to isolate the net effects of the network attribute of interest (e.g., Morris and Kretzschmar 1997), but it remains largely unverified by empirical studies. We return to this issue below.

The asymmetric concurrency effect on the gender ratio operates via two mechanisms. First, asymmetric concurrency exposes seronegative concurrent partners of a man to elevated transmission probabilities associated with the high viral load during the acute phase if that man has just acquired HIV from (one of) his other partner(s).[16] The second mechanism is intricately related to the modeling assumption that keeps the number of partnerships constant across the different sexual network types. Under this assumption, more male concurrency implies more isolated male nodes

[16] See Eaton, Hallett, and Garnett (2011) and Goodreau et al. (2012) for a comprehensive discussion of the interaction between partnership concurrency and acute infection.

in the network,[17] and women will on average spend more time in partnerships than men (which increases their relative exposure to HIV). It is not clear to what extent this assumption is realistic, however. In an empirical study of the effects of polygyny on HIV epidemic propagation, we could not find much evidence for the 'monopolizing polygynists' hypothesis (i.e., the proposition that polygyny among older men leads to a deprivation of sexual partners for the younger men) (Reniers and Tfaily 2012). We do not know of other studies that have addressed this question, and it would be precipitous to entirely discard the validity of the constant partnerships assumption on the basis of one study only.

The drift of HIV-positive women from the partnership market consists of two processes that operate in parallel: (1) the relatively high partnership dissolution rates among female-positive serodiscordant couples, and (2) the relatively low partnership formation rates among HIV-positive women. As we have shown, this phenomenon could have considerable effects on the gender ratio of infections (particularly in networks with gender-symmetric concurrency), but it also rests on the assumption that the demand for partners in the population is unchanged. Given the constant partnership distribution in the simulated populations, the drift leads to the substitution of HIV-positive women in partnerships by HIV-negative women, and that will increases HIV-negative women's exposure to HIV-positive men, as well as decrease male exposure to HIV-positive women.

Using variation in the simulated attributes of sexual networks and sexual mixing patterns, we model populations with a gender ratio of infections ranging from 1.13 to 1.75, and that suggests that these factors can indeed help explain some of the heterogeneity in the gender ratio of infections currently observed in empirical data. Even though the nature of our evidence comes with the disclaimer that it is entirely based on simulated data that rely on a number of simplifying assumptions, it suggests that we need to look beyond individual behavior or gender differences in biological susceptibility if we are to fully understand, and remedy, gender inequalities in HIV infection in generalized epidemics. Such remedies may have to target upstream or distal determinants of the inequalities between men and women that are discussed elsewhere (UNAIDS, UNFPA and UNIFEM 2010), but we hope that our study sheds light on the mechanisms through which they operate.

[17] See Reniers and Watkins (2010) for an illustration of sexual networks with strict monogamy, gender-asymmetric concurrency, and symmetric concurrency.

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References

Anglewicz, P. and Reniers, G. (2014). HIV status, gender, and marriage dynamics among adults in Rural Malawi. *Studies in Family Planning* 45(4):415−428. doi:10.1111/j.1728-4465.2014.00005.x.

Anipah, K., Mboup, G., Mawuéna Ouro-Gnao, A., Boukpessi, B., Adadé Messan, P., and Salami-Odjo, R. (1999). *Enquête démographique et de santé Togo, 1998*. Calverton, Maryland, USA: Direction de la Statistique et Macro International Inc.

Auvert, B., Taljaard, D., Lagarde, E., Sobngwi-Tambekou, J., Sitta, R., and Puren, A. (2005). Randomized, controlled intervention trial of male circumcision for reduction of HIV infection risk: The ANRS 1265 Trial. *PLoS Medicine* 2(11): e298. doi:10.1371/journal.pmed.0020298.

Barnighausen, T., Bor, J., Wandira-Kazibwe, S., and Canning, D. (2011). Correcting HIV prevalence estimates for survey nonparticipation using Heckman-type selection models. *Epidemiology* 22(1): 27−35. doi:10.1097/EDE.0b013e3181ffa201.

Boily, M.C., Alary, M., and Baggaley, R.F. (2012). Neglected issues and hypotheses regarding the impact of sexual concurrency on HIV and sexually transmitted infections. *AIDS and Behavior* 16(2): 304−311. doi:10.1007/s10461-011-9887-0.

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

Braitstein, P., Boulle, A., Nash, D., Brinkhof, M.W.G., Dabis, F., Laurent, C., Schechter, M., Tuboi, S.H., Sprinz, E., and Miotti, P. (2008). Gender and the use of antiretroviral treatment in resource-constrained settings: findings from a multicenter collaboration. *Journal of Women's Health* 17(1): 47−55. doi:10.1089/jwh.2007.0353.

Carpenter, L.M., Kamali, A., Ruberantwari, A., Malamba, S.S., and Whitworth, J.A.G. (1999). Rates of HIV-1 transmission within marriage in rural Uganda in relation to the HIV sero-status of the partners. *AIDS* 13(9): 1083−1089. doi:10.1097/00002030-199906180-00012.

Chersich, M.F. and Rees, H.V. (2008). Vulnerability of women in southern Africa to infection with HIV: biological determinants and priority health sector interventions. *AIDS* 22 Suppl 4: S27−40. doi:10.1097/01.aids.0000341775.94123.75.

Chin, J. (1990). Public health surveillance of AIDS and HIV infections. *Bulletin of the World Health Organization* 68(5): 529−536.

Clark, S. (2004). Early Marriage and HIV Risks in Sub-Saharan Africa. *Studies in Family Planning* 35(3): 149−160. doi:10.1111/j.1728-4465.2004.00019.x.

Cohen, M.S. (2004). HIV and sexually transmitted diseases: lethal synergy. *Topics in HIV medicine* 12(4): 104−107. doi:10.1136/sti.2004.011171.

CSO [Botswana] (2009). 2008 Botswana AIDS impact survey III: Statistical report. Gaborone: Central Statistics Office

de Walque, D. and Kline, R. (2012). The Association Between Remarriage and HIV Infection in 13 Sub-Saharan African Countries. *Studies in Family Planning* 43(1): 1−10. doi:10.1111/j.1728-4465.2012.00297.x.

Delva, W., Meng, F., Beauclair, R., Deprez, N., Temmerman, M., Welte, A., and Hens, N. (2013). Coital frequency and condom use in monogamous and concurrent sexual relationships in Cape Town, South Africa. *Journal of the International AIDS Society* 16: 18034. doi:10.7448/IAS.16.1.18034.

Eaton, J.W., Hallett, T.B., and Garnett, G.P. (2011). Concurrent sexual partnerships and primary HIV infection: a critical interaction. *AIDS and Behavior* 15(4): 687−692. doi:10.1007/s10461-010-9787-8.

Floyd, S., Crampin, A.C., Glynn, J.R., Mwenebabu, M., Mnkhondia, S., Ngwira, B., Zaba, B., and Fine, P.E. (2008). The long-term social and economic impact of HIV on the spouses of infected individuals in northern Malawi. *Tropical Medicine and International Health* 13(4): 520−531. doi:10.1111/j.1365-3156.2008.02030.x.

Gaydosh, L., Reniers, G., and Helleringer, S. (2013). Partnership Concurrency and Coital Frequency. *AIDS and Behavior* 17(7): 2376−2386. doi:10.1007/s10461-013-0525-x.

Glynn, J.R., Carael, M., Auvert, B., Kahindo, M., Chege, J., Musonda, R., Kaona, F., and Buve, A. (2001). Why do young women have a much higher prevalence of HIV than young men? A study in Kisumu, Kenya and Ndola, Zambia. *AIDS* 15(Supplement 4): S51−60. doi:10.1097/00002030-200108004-00006.

Goodreau, S.M., Cassels, S., Kasprzyk, D., Montaño, D.E., Greek, A., and Morris, M. (2012). Concurrent partnerships, acute infection and HIV epidemic dynamics among young adults in Zimbabwe. *AIDS and Behavior* 16(2): 312−322. doi:10.1007/s10461-010-9858-x.

Gray, R.H., Li, X., Kigozi, G., Serwadda, D., Brahmbhatt, H., Wabwire-Mangen, F., Nalugoda, F., Kiddugavu, M., Sewankambo, N., Quinn, T.C., Reynolds, S.J., and Wawer, M.J. (2005). Increased risk of incident HIV during pregnancy in Rakai, Uganda: a prospective study. *Lancet* 366(9492): 1182−1188. doi:10.1016/S0140-6736(05)67481-8.

Gregson, S. and Garnett, G.P. (2000). Contrasting gender differentials in HIV-1 prevalence and associated mortality increase in eastern and southern Africa: artefact of data or natural course of epidemics? *AIDS* 14(Supplement 3): S85−S99.

Gregson, S., Nyamukapa, C.A., Garnett, G.P., Mason, P.R., Zhuwau, T., Caraël, M., Chandiwana, S.K., and Anderson, R.M. (2002). Sexual mixing patterns and sex-differentials in teenage exposure to HIV infection in rural Zimbabwe. *Lancet* 359(9321): 1896−1903. doi:10.1016/S0140-6736(02)08780-9.

Grinstead, O.A., Gregorich, S.E., Choi, K.H., and Coates, T. (2001). Positive and negative life events after counselling and testing: the Voluntary HIV-1 Counselling and Testing Efficacy Study. *AIDS* 15(8): 1045−1052. doi:10.1097/00002030-200105250-00013.

Harling, G., Newell, M.-L., Tanser, F., Kawachi, I., Subramanian, S., and Bärnighausen, T. (2014). Do age-disparate relationships drive HIV incidence in young women? Evidence from a population cohort in rural KwaZulu-Natal, South Africa. *JAIDS Journal of Acquired Immune Deficiency Syndromes* 66(4): 443−451. doi:10.1097/QAI.0000000000000198.

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

Hertog, S. (2008). Sex ratios of HIV prevalence: evidence from the DHS in *Population Association Annual Meeting*. New Orleans, LA.

Higgins, J.A., Hoffman, S., and Dworkin, S.L. (2010). Rethinking gender, heterosexual men, and women's vulnerability to HIV/AIDS. *American Journal of Public Health* 100(3): 435−445. doi:10.2105/AJPH.2009.159723.

Kelly, R.J., Gray, R.H., Sewankambo, N.K., Serwadda, D., Wabwire-Mangen, F., Lutalo, T., and Wawer, M.J. (2003). Age differences in sexual partners and risk of HIV-1 infection in rural Uganda. *Journal of Acquired Immune Deficiency Syndromes* 32(4): 446−451. doi:10.1097/00126334-200304010-00016.

Leclerc-Madlala, S. (2008). Age-disparate and intergenerational sex in southern Africa: the dynamics of hypervulnerability. *AIDS* 22: S17−S25. doi:10.1097/01.aids.0000341774.86500.53.

Lurie, M.N. and Rosenthal, S. (2010). Concurrent partnerships as the driver of the HIV epidemic in sub-Saharan Africa? The evidence is limited. *AIDS and Behavior* 14(1): 17−24. doi:10.1007/s10461-009-9583-5.

Mackelprang, R.D., Bosire, R., Guthrie, B.L., Choi, R.Y., Liu, A., Gatuguta, A., Rositch, A.F., Kiarie, J.N., and Farquhar, C. (2013). High Rates of Relationship Dissolution Among Heterosexual HIV-Serodiscordant Couples in Kenya. *AIDS and Behavior*:1−5.

Mah, T.L. and Halperin, D.T. (2010). Concurrent sexual partnerships and the HIV epidemics in Africa: evidence to move forward. *AIDS and Behavior* 14(1): 11−16. doi:10.1007/s10461-009-9617-z.

Marston, M., Newell, M.L., Crampin, A., Lutalo, T., Musoke, R., Gregson, S., Nyamukapa, C., Nakiyingi-Miiro, J., Urassa, M., Isingo, R., and Zaba, B. (2013). Is the risk of HIV acquisition increased during and immediately after pregnancy? A secondary analysis of pooled HIV community-based studies from the ALPHA network. *PLoS One* 8(12): e82219. doi:10.1371/journal.pone.0082219.

Mastro, T.D. and de Vincenzi, I. (1996). Probabilities of sexual HIV-1 transmission. *AIDS* 10(Suppl A): S75−82. doi:10.1097/00002030-199601001-00011.

May, R.M. and Anderson, R.M. (1987). Transmission dynamics of HIV infection. *Nature* 326(6109): 137−142. doi:10.1038/326137a0.

Mills, E.J., Ford, N., and Mugyenyi, P. (2009). Expanding HIV care in Africa: making men matter. *Lancet* 374(9686): 275−276. doi:10.1016/S0140-6736(09)61348-9.

Morgan, D., Mahe, C., Mayanja, B., Okongo, J.M., Lubega, R., and Whitworth, J.A. (2002). HIV-1 infection in rural Africa: is there a difference in median time to AIDS and survival compared with that in industrialized countries? *AIDS* 16(4): 597−603. doi:10.1097/00002030-200203080-00011.

Morris, M. and Kretzschmar, M. (1997). Concurrent partnerships and the spread of HIV. *AIDS* 11(5): 641−648. doi:10.1097/00002030-199705000-00012.

Morrison, C.S., Wang, J., Van Der Pol, B., Padian, N., Salata, R.A., and Richardson, B.A. (2007). Pregnancy and the risk of HIV-1 acquisition among women in Uganda and Zimbabwe. *AIDS* 21(8): 1027−1034. doi:10.1097/QAD.0b013e3280f00fc4.

Nicolosi, A., Correa Leite, M.L., Musicco, M., Arici, C., Gavazzeni, G., and Lazzarin, A. (1994). The efficiency of male-to-female and female-to-male sexual transmission of the human immunodeficiency virus: a study of 730 stable couples. Italian Study Group on HIV Heterosexual Transmission. *Epidemiology* 5(6): 570−575. doi:10.1097/00001648-199411000-00003.

Nnko, S., Boerma, J.T., Urassa, M., Mwaluko, G., and Zaba, B. (2004). Secretive females or swaggering males? An assessment of the quality of sexual partnership reporting in rural Tanzania. *Social Science and Medicine* 59(2): 299−310. doi:10.1016/j.socscimed.2003.10.031.

ORC Macro (2013). Demographic and Health Surveys: StatCompiler. Calverton, MD: Macro International Inc.

Parsons, J.T., Schrimshaw, E.W., Wolitski, R.J., Halkitis, P.N., Purcell, D.W., Hoff, C.C., and Gomez, C.A. (2005). Sexual harm reduction practices of HIV-seropositive gay and bisexual men: serosorting, strategic positioning, and withdrawal before ejaculation. *AIDS* 19 Suppl 1: S13−25. doi:10.1097/01.aids.0000167348.15750.9a.

Porter, L., Hao, L., Bishai, D., Serwadda, D., Wawer, M.J., Lutalo, T., and Gray, R. (2004). HIV status and union dissolution in sub-Saharan Africa: the case of Rakai, Uganda. *Demography* 41(3): 465−482. doi:10.1353/dem.2004.0025.

Powers, K.A., Poole, C., Pettifor, A.E., and Cohen, M.S. (2008). Rethinking the heterosexual infectivity of HIV-1: a systematic review and meta-analysis. *Lancet Infectious Diseases* 8(9): 553−563. doi:10.1016/S1473-3099(08)70156-7.

Reniers, G. (2008). Marital strategies for regulating exposure to HIV. *Demography* 45(2): 417−438. doi:10.1353/dem.0.0002.

Reniers, G. and Armbruster, B. (2012). HIV status awareness, partnership dissolution and HIV transmission in generalized epidemics. *PLoS One* 7(12): e50669. doi:10.1371/journal.pone.0050669.

Reniers, G. and Eaton, J. (2009). Refusal bias in HIV prevalence estimates from nationally representative seroprevalence surveys. *AIDS* 23(5): 621−629. doi:10.1097/QAD.0b013e3283269e13.

Reniers, G. and Helleringer, S. (2011). Serosorting and the evaluation of HIV testing and counseling for HIV prevention in generalized epidemics. *AIDS and Behavior* 15(1): 1−8. doi:10.1007/s10461-010-9774-0.

Reniers, G. and Tfaily, R. (2012). Polygyny, partnership concurrency, and HIV transmission in Sub-Saharan Africa. *Demography* 49(3): 1075−1101. doi:10.1007/s13524-012-0114-z.

Reniers, G. and Watkins, S. (2010). Polygyny and the spread of HIV in sub-Saharan Africa: a case of benign concurrency. *AIDS* 24(2): 299−307. doi:10.1097/QAD.0b013e328333af03.

Santhakumaran, S., O'Brien, K., Bakker, R., Ealden, T., Shafer, L.A., Daniel, R.M., Chapman, R., Hayes, R.J., and White, R.G. Polygyny and symmetric concurrency: comparing long-duration sexually transmitted infection prevalence using simulated sexual networks. *Sexually Transmitted Infections* 86, no. 7 (2010): 553–558.

Sawers, L. (2013). Measuring and modelling concurrency. *Journal of the International AIDS Society* 16: 17431. doi:10.7448/IAS.16.1.17431.

Sawers, L., Isaac, A.G., and Stillwaggon, E. (2011). HIV and concurrent sexual partnerships: modelling the role of coital dilution. *Journal of the International AIDS Society* 14(1): 44. doi:10.1186/1758-2652-14-44.

Sawers, L. and Stillwaggon, E. (2010). Concurrent sexual partnerships do not explain the HIV epidemics in Africa: a systematic review of the evidence. *Journal of the International AIDS Society* 13(1): 34. doi:10.1186/1758-2652-13-34.

Shisana, O. (2005). *South African national HIV prevalence, HIV incidence, behaviour and communication survey, 2005*. Human Sciences Research Council.

Suarez, T. and Miller, J. (2001). Negotiating risks in context: a perspective on unprotected anal intercourse and barebacking among men who have sex with men−where do we go from here? *Archives of Sexual Behavior* 30(3): 287−300. doi:10.1023/A:1002700130455.

Todd, J., Glynn, J.R., Marston, M., Lutalo, T., Biraro, S., Mwita, W., Suriyanon, V., Rangsin, R., Nelson, K.E., and Sonnenberg, P. (2007). Time from HIV seroconversion to death: a collaborative analysis of eight studies in six low and middle-income countries before highly active antiretroviral therapy. *AIDS* 21: S55−S63. doi:10.1097/01.aids.0000299411.75269.e8.

UNAIDS (2010). UNAIDS report on the global AIDS epidemic, 2010. Geneva: Joint United Nations Programme on HIV/AIDS (UNAIDS).

UNAIDS, UNFPA, and UNIFEM (2010). Women and HIV/AIDS: Confronting the crisis. Geneva: Joint United Nations Programme on HIV/AIDS (UNAIDS).

UNAIDS/WHO (2000). Consultation on STD Interventions for Preventing HIV: What is the Evidence? In: *Best practice collection*. Geneva: UNAIDS/WHO.

UNPD (2013). World Population Prospects: The 2012 Revision. New York United Nations.

Watkins, S.C. (2004). Navigating the AIDS epidemic in rural Malawi. *Population and Development Review* 30(4): 673−705. doi:10.1111/j.1728-4457.2004.00037.x.

Wawer, M.J., Gray, R.H., Sewankambo, N.K., Serwadda, D., Li, X., Laeyendecker, O., Kiwanuka, N., Kigozi, G., Kiddugavu, M., Lutalo, T., Nalugoda, F., Wabwire-Mangen, F., Meehan, M.P., and Quinn, T.C. (2005). Rates of HIV-1 transmission per coital act, by stage of HIV-1 infection, in Rakai, Uganda. *The Journal of Infectious Diseases* 191(9): 1403−1409. doi:10.1086/429411.

WHO (2012). Hormonal contraception and HIV. Technical statement. Geneva: World Health Organization.

Wilensky, U. (1999). *NetLogo. http://ccl.northwestern.edu/netlogo/*. Northwestern University, Evanston, IL: Center for Connected Learning and Computer-Based Modeling.

Williams, B.G., Korenromp, E.L., Gouws, E., Schmid, G.P., Auvert, B., and Dye, C. (2006). HIV infection, antiretroviral therapy, and CD4+ cell count distributions in African populations. *The Journal of Infectious Diseases* 194(10): 1450−1458. doi:10.1086/508206.

Yi, T.J., Shannon, B., Prodger, J., McKinnon, L., and Kaul, R. (2013). Genital immunology and HIV susceptibility in young women. *American Journal of Reproductive Immunology* 69(Supplement 1): 74−79. doi:10.1111/aji.12035.