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Gender asymmetry in concurrent partnerships and HIV prevalence

Ka Yin Leung[a,b,*], Kimberly A. Powers[c], Mirjam Kretzschmar[b,d]

[a] Utrecht University, PO Box 80010, 3508 TA Utrecht, The Netherlands
[b] University Medical Center Utrecht, PO Box 85500, 3508 GA Utrecht, The Netherlands
[c] The University of North Carolina at Chapel Hill, 2105D McGavran-Greenberg Hall, Campus Box 7435, Chapel Hill, NC 27599-7435, USA
[d] National Institute of Public Health and the Environment, PO Box 1, 3720 BA Bilthoven, The Netherlands

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ABSTRACT

The structure of the sexual network of a population plays an essential role in the transmission of HIV. Concurrent partnerships, i.e. partnerships that overlap in time, are important in determining this network structure. Men and women may differ in their concurrent behavior, e.g. in the case of polygyny where women are monogamous while men may have concurrent partnerships. Polygyny has been shown empirically to be negatively associated with HIV prevalence, but the epidemiological impacts of other forms of gender-asymmetric concurrency have not been formally explored. Here we investigate how gender asymmetry in concurrency, including polygyny, can affect the disease dynamics. We use a model for a dynamic network where individuals may have concurrent partners. The maximum possible number of simultaneous partnerships can differ for men and women, e.g. in the case of polygyny. We control for mean partnership duration, mean lifetime number of partners, mean degree, and sexually active lifespan. We assess the effects of gender asymmetry in concurrency on two epidemic phase quantities (R_0 and the contribution of the acute HIV stage to R_0) and on the endemic HIV prevalence. We find that gender asymmetry in concurrent partnerships is associated with lower levels of all three epidemiological quantities, especially in the polygynous case. This effect on disease transmission can be attributed to changes in network structure, where increasing asymmetry leads to decreasing network connectivity.

1. Introduction

Sexual behavior plays an essential role in the transmission of HIV and other sexually transmitted infections (STIs) in a population. There is large diversity in sexual behavior, leading to varying risks of acquiring or transmitting infection. Some behavior predominantly influences transmissibility and susceptibility, such as condom use. Other behavior, such as concurrency, influences the population network structure.

Whether concurrent partnerships are driving HIV epidemics in sub-Saharan Africa has been discussed and debated for almost 20 years (e.g. Morris and Kretzschmar, 2000; Halperin and Epstein, 2004; Lurie and Rosenthal, 2010; Mah and Halperin, 2010; Sawers and Stillwaggon, 2010; Kretzschmar et al., 2010; Epstein and Morris, 2011; Goodreau, 2011; Boily et al., 2011; Reniers and Watkins, 2010; Kretzschmar and Caraël, 2012). The biological explanation behind an important role for concurrency is plausible: in the setting of serial monogamy, a newly infected individual is typically still in a partnership only with his/her infector during the acute phase of HIV, i.e. the elevated infectiousness in the first few weeks/months immediately after infection. In contrast, when partnerships overlap in time, newly infected individuals can transmit infection to their susceptible partners in this acute phase. It has been hypothesized and shown through mathematical modeling (Eaton et al., 2011) that the interaction between acute infectiousness and concurrent partnerships could potentially enhance the spread of HIV in the population.

Some forms of concurrency such as polygyny (men may have multiple partners at a time while women are monogamous) naturally lead to gender asymmetry in concurrent behavior. An ecological study of 34 countries in sub-Saharan Africa suggested that this form of concurrency is negatively correlated with HIV prevalence in the population (Reniers and Watkins, 2010). A recent modeling study (Reniers et al., 2015) comparing polygyny to gender-symmetric concurrency also concluded that polygyny is associated with lower HIV prevalence. Besides polygyny and gender-symmetric concurrency, however, there is a broad range of concurrency patterns existing in populations. In particular, men

* Corresponding author. Present address: Department of Mathematics, Stockholm University, 106 91 Stockholm, Sweden.
E-mail addresses: kayin.leung@math.su.se (K.Y. Leung), powersk@email.unc.edu (K.A. Powers), m.e.e.kretzschmar@umcutrecht.nl (M. Kretzschmar).

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are often found to report more concurrent partners than women, but women may also have more than one partner at a time (Morris and Kretzschmar, 2000; Glynn et al., 2012; Maughan-Brown, 2013). It is not straightforward to predict how this 'intermediate' type of asymmetric concurrency might affect population-level HIV transmission. We therefore sought to assess through mathematical modeling how varying types of gender asymmetry in concurrent partnerships – including polygyny – affect HIV disease dynamics in a population.

As most HIV epidemics in sub-Saharan Africa are now endemic, our main aim is to study the relationship between gender-asymmetric concurrency and endemic prevalence. Moreover, we study the basic reproduction number R_0 and the relative contribution of the acute stage of HIV to R_0. How does gender asymmetry affect the population prevalence; are more asymmetric populations associated with lower levels of HIV prevalence (as found in Reniers and Watkins, 2010)? In this study we are interested in the qualitative behavior and we do not attempt to make any quantitative predictions about disease prevalence. We use a mathematical model for the spread of HIV on a dynamic sexual network for a heterosexual population where we control for sexually active lifetime, mean partnership duration, mean degree, and mean lifetime number of partners. To parameterize the model we use existing data on sexual behavior from a study population in Malawi and published estimates of HIV infectivity and natural history parameters. Different scenarios in the maximum number of simultaneous partners are investigated.

2. Methods

2.1. Model

We use a mathematical model to study the effect of gender asymmetry in concurrency on transmission dynamics in the population. To this end, we use a previously introduced model for infection dynamics on a dynamic network that takes into account partnership formation and separation as well as demographic turnover (Leung et al., 2012, 2015). General analytical results for the spread of infections on networks have mainly been for static networks (e.g. Diekmann et al., 1998; Ball and Neal, 2008; House and Keeling, 2011; Miller et al., 2012). Early exceptions are e.g. Altmann (1995) and Volz and Meyers (2007) who consider disease transmission on dynamic networks without demographic turnover. Our model, which takes into account both partnership changes and demographic changes, can be seen as a generalization of pair formation models that describe sequentially monogamous populations (which were first introduced in epidemiology by Dietz and Hadeler, 1988).

In this paper, we extended the model of Leung et al. (2012, 2015) to a heterosexual population with a 1:1 sex ratio and a two-stage infectious disease. Concurrent partnerships are modeled by allowing a maximum number of partners that an individual can have at the same time; we call this number the partnership capacity. We assume that there are two partnership capacities n_m and n_f for men and women in the population, respectively, and we varied these values across model scenarios (while holding the values of n_m and n_f fixed across individuals within a scenario). Newly infected individuals are in the first phase of infection and then progress to the chronic phase after an exponentially distributed amount of time. An infectious individual remains infectious for the rest of his or her sexually active life. We refer to the first and second phase of infection as the acute and chronic phase, respectively. The acute phase is characterized by a higher transmission rate than the chronic phase.

The network structure is stable over time, which leads to stable degree distributions for men and women (the degree of an individual is the number of partners that he or she has). The mean degree was kept constant throughout this study. However, the specific links within the network are dynamic in time due to individuals forming new partnerships and separating from existing partners during the course of their life. Furthermore, there is demographic flow due to individuals entering and leaving the sexually active population. Infection is assumed to have no impact on partnership formation or separation nor on mortality.

The model is deterministic in nature. It is fully characterized by a set of parameters comprising sexual behavior and infection parameters. Individuals are characterized by sex-specific behavioral parameters; see Sections 2.2 and 2.3. We assume that the infection parameters (i.e. transmission rates and disease progression) do not depend on gender (Hughes et al., 2012; Powers et al., 2008; Quinn et al., 2000).

Concurrency is measured using the partnership-based concurrency index (Morris and Kretzschmar, 1997; Leung et al., 2012; Leung and Kretzschmar, 2015). It measures the mean number of additional partners of an individual in a randomly chosen partnership and can be expressed in terms of the mean and variance of a degree distribution. Here we distinguish between concurrency indices κ_P^m and κ_P^f for men and women, respectively, and the concurrency index κ_P^{popul} for the population as a whole. We vary the gender symmetry of concurrency in the population by varying n_f and n_m.

We have previously derived explicit expressions for the degree distributions, partnership-based concurrency indices, basic reproduction number R_0, and relative contribution of the acute stage to R_0 in terms of model parameters (Leung et al., 2012, 2015). The endemic prevalence is characterized implicitly as solution of a fixed point problem that can be computed numerically.

A more detailed and technical description of the model can be found in Appendix A of the supplementary data.

2.2. Scenarios

Throughout this study we keep mean partnership duration d_P, sexually active lifespan $\bar{L}$, and mean lifetime number of partners $\bar{\theta}$ in the population fixed. This also means that mean degree in the population is fixed (Eq. (A.2) in Appendix A of the supplementary data). The scenarios are defined by varying the sex-specific quantities n_m, n_f, L_f, and L_m (which also varies θ_f and θ_m, Eq. (A.1) in Appendix A.1 of the supplementary data).

We focus on gender asymmetry in the maximum number of partners men and women may have at the same time, i.e. in partnership capacities n_f and n_m. Mean partnership capacity is fixed across scenarios at $\bar{n} = (n_f + n_m)/2 = 4$. The extremes are the asymmetric polygynous situation $(n_f, n_m) = (1, 7)$ (i.e. women are monogamous) and the symmetric situation $(n_f, n_m) = (4, 4)$. Note that, since infection parameters are gender independent, asymmetry in the opposite direction ($(n_f, n_m) = (7, 1)$ etc.) is automatically included by switching the role of males and females in the results.

Mathematical models of heterosexual HIV transmission in sub-Saharan African settings commonly assume a sexually active population between the ages of 15 and 49 years (Eaton et al., 2012), corresponding to an average sexually active lifespan $\bar{L}$ of 35 years. Differences in the duration of sexual activity by sex are not well defined. Given the uncertainty in sexual lifespans for men and women, we modeled three scenarios: one in which both men and women had a mean sexual lifespan of $L_f = L_m = 35$ years; one in which the sexually active lifespan was $L_f = 33$ years for women and $L_m = 37$ years for men; and one in which the sexually active lifespan was $L_m = 33$ years for men and $L_f = 37$ years for women. Larger differences in sexually active lifespans were considered in the sensitivity analysis of Appendix C of the supplementary data (see Fig. C.6).

Table 1
The 12 different scenarios. Corresponding lifetime number of partners are $\theta_f = 3.3$ and $\theta_m = 3.7$ for $L_f = 33$ years, $L_m = 37$ years, $\theta_f = 3.5 = \theta_m$ for $L_f = 35 = L_m$ years, and $\theta_f = 3.7$ and $\theta_m = 3.3$ for $L_f = 37$ years, $L_m = 33$ years, see also Section 2.3.

	$L_f = 33$ years $L_m = 37$ years	$L_f = 35$ years $L_m = 35$ years	$L_f = 37$ years, $L_m = 33$ years
$(n_f, n_m) = (1, 7)$	A1	A2	A3
$(n_f, n_m) = (2, 6)$	B1	B2	B3
$(n_f, n_m) = (3, 5)$	C1	C2	C3
$(n_f, n_m) = (4, 4)$	D1	D2	D3

Together, gender asymmetry in partnership capacities and sexually active lifespans give rise to twelve different scenarios; these are summarized in Table 1.

2.3. Parameterization of the model

Mean partnership duration is based on self-reported data on steady partnership duration collected in a study of acute HIV detection strategies and longitudinal HIV viral dynamics at an STI clinic in Lilongwe, Malawi. Sampling strategies and study procedures have been described previously (Fiscus et al., 2007; Pilcher et al., 2007; Powers et al., 2007, 2011a,b). As our model assumes a constant partnership dissolution rate, we calculated the slope of a linear curve fitted to the reported duration of steady partnerships (in days) on the x-axis and the – ln(partnership survival) on the y-axis, see Appendix B of the supplementary data. We estimated a weighted mean partnership duration d_P of 3.92 years across men and women as the inverse of this slope.

Since we are interested in qualitative behavior and we do not aim to make any quantitative predictions about disease prevalence in this study, the mean lifetime number of partners $\bar{\theta}$ is calibrated to an endemic HIV prevalence of around 13% for scenarios B2 and C2, corresponding to estimated HIV prevalence in Malawi in 2010 (DHS Program, 2010). This leads to a mean lifetime number of partners $\bar{\theta} = 3.5$. Consistency between mean lifetime number of partners and sexual lifespan then determines the mean lifetime number of partners θ_m and θ_f for men and women, these are found below Table 1 (see also Eq. (A.1) in Appendix A.1 of the supplementary data).

Note that our choice of keeping mean sexual behavior parameters constant also leads to a constant mean degree across all scenarios, which is the same for men and women (Eq. (A.2) in Appendix A.1 of the supplementary data). Variance, and therefore the concurrency index, does differ across the scenarios; see Section 3.1 for details and a discussion.

Infection is characterized by the transmission rates β_1, β_2 and duration of the acute phase d_A (Table 2). We let $\beta_1 = 2.76$/year, $d_A = 2.9$ months, and $\beta_2 = 0.106$/year (Hollingsworth et al., 2008). In Appendix C.3 of the supplementary data we conducted sensitivity analysis using infectivity parameter estimates from a different, more recently published study (Bellan et al., 2015). In Bellan et al. (2015) a much lower transmission rate for the acute stage of infection was estimated but a higher transmission rate for the chronic stage of infection (yielding a comparable total infectivity). Qualitatively, this did not change our conclusions.

Table 2
Model parameters and values. Variation around the parameter values are addressed in the sensitivity analysis in Appendix C of the supplementary data.

Parameter	Description	Estimate
n_m	Partnership capacity for men	Varied (see Table 1)
n_f	Partnership capacity for women	Varied (see Table 1)
d_P	Mean partnership duration	3.92 years
L_f	Sexually active lifespan for women	Varied (see Table 1)
L_m	Sexually active lifespan for men	Varied (see Table 1)
$\bar{L} = \frac{1}{2}(L_f + L_m)$	Sexually active lifespan	35 years
θ_f	Mean lifetime number of partners for women	Varied (see Table 1)
θ_m	Mean lifetime number of partners for men	Varied (see Table 1)
$\bar{\theta} = \frac{1}{2}(\theta_f + \theta_m)$	Mean lifetime number of partners	3.5
β_1	Transmission rate acute phase	2.76/year
d_A	Duration acute phase	2.9 months
β_2	Transmission rate chronic phase	0.106/year

Parameter values are summarized in Table 2. Variation around the parameter values are addressed in the sensitivity analysis in Appendix C of the supplementary data.

3. Results

The three different sexually active lifespan cases $L_f < L_m$, $L_f = L_m$, and $L_f > L_m$ lead to very similar results for network structure, R_0, and the relative contribution of the acute phase to R_0. Therefore, in both Sections 3.1 and 3.2, we focus on the sexually active lifespan case $L_f = L_m$, i.e. scenarios A2, B2, C2, and D2. In Section 3.3 all 12 scenarios (defined in Table 1) are considered. A sensitivity analysis was carried out in Appendix C of the supplementary data, which shows that the results presented in this section are qualitatively robust to variations in parameter values.

3.1. Network structure

As the overall degree distributions (across women and men) are very similar across scenarios B2, C2, and D2, we show only scenarios A2 and D2 in Fig. 1. Our definition of scenarios leads to a constant mean degree for men and women across all 12 scenarios and it is given by $\bar{\theta}/\bar{L}d_P = 0.39$ (note that this is independent of gender and partnership capacity). The variance for men and women does differ across the scenarios. Variance values for scenarios A2, B2, C2, and D2 are summarized in Table 3 (variance values for the other eight scenarios are similar).

We find that the degree distribution for men is similar across all four scenarios, illustrated by the two most extreme scenarios

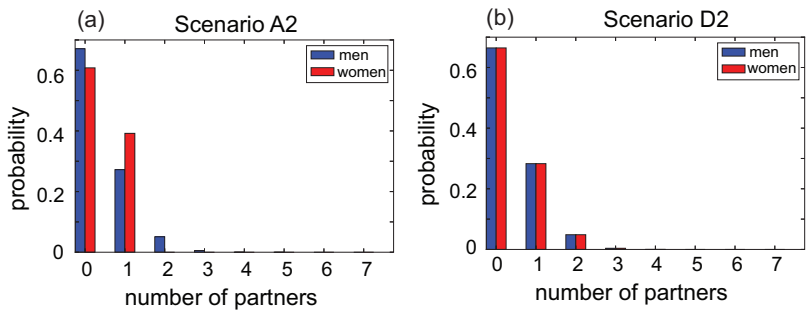

Fig. 1. The degree distributions for (a) scenario A2 and (b) scenario D2 calculated from the model parameters.

Table 3
The degree variance for women (f) and men (m) for the scenarios A2, B2, C2, and D2 (Table 1). Mean degree is 0.39 for both men and women across all scenarios.

A2		B2		C2		D2	
f	m	f	m	f	m	f	m
0.24	0.38	0.32	0.37	0.35	0.37	0.36	0.36

A2 and D2 and the degree variance in Table 3. The key difference across scenarios is in the degree distributions for women: in A2, women are monogamous and can have either zero or one partner, which is distinctly different from scenarios B2, C2, and D2, where there are fractions of women with more than one partner at a time.

The degree distribution for men and women together are important in making up the network structure, this is illustrated by Fig. 2. In Fig. 2 networks for scenarios A2 and D2 are visualized by simulating cross-sections of the networks using degree distributions of Fig. 1. Network structure is significantly different in the two scenarios A2 and D2. In scenario D2, longer 'chains' of partnerships can be found, e.g. a chain consisting of six individuals. This is absent in scenario A2 where women have at most one partner at a time (so chains consist of at most three individuals). Note that Fig. 2 represents snapshots of one randomly chosen point in time as the networks are evolving in time with partnership formation and dissolution as well as individuals entering and leaving the sexually active population. Furthermore, single individuals are omitted from the network figures to better show the partnerships. Finally, although partnership capacity choices in principle allow for much larger connected components, our parameter values in this study are such that this is rarely the case; there is only a very small fraction of the population with more than two partners (Fig. 1). Nevertheless, the small proportion of longer chains in the D2 network result in substantially higher rates of disease transmission in the non-polygynous scenarios (Sections 3.2 and 3.3).

Next, we consider the level of concurrency for the scenarios A2, B2, C2, and D2. Different aspects of concurrency, such as the mean number of concurrent partnerships and the mean duration of overlap, are captured by the degree distribution in the population (Fig. 1 and network parameters such as the mean partnership duration and lifetime number of partners). From the degree distributions in Fig. 1, we observe that only small fractions of men and women have concurrent partnerships, i.e. more than two partners, across all scenarios, and a large proportion of the population is without any partner. We combine these aspects of concurrency and measure the level of concurrency with the partnership-based concurrency index (cf. Morris and Kretzschmar, 1997; Leung et al., 2012; Leung and Kretzschmar, 2015, see also Appendix A of the supplementary data). The results are presented in Fig. 3.

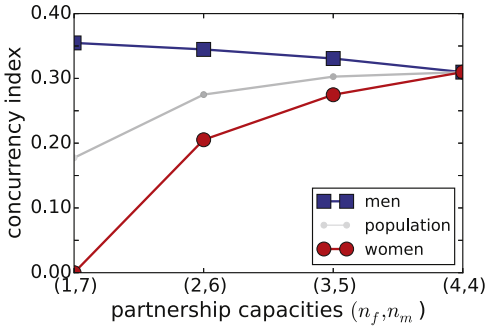

Fig. 3. The concurrency index κ_p^m for men, κ_p^f for women, and κ_p^{popul} for the population, in scenarios A2, B2, C2, and D2.

Note that female concurrency is zero for $n_f = 1$ (monogamy) and increases for $n_f = 2, 3, 4$. Male concurrency is highest in the most asymmetric scenario A2 and decreases with more symmetric scenarios. Similar to the case for women, this decrease in concurrency is caused by a decrease in the maximum number of simultaneous partnerships ($n_m = 7, 6, 5, 4$). The decrease in male concurrency is less than the increase in female concurrency. Therefore, with increasing equality of men and women in the numbers of partners they may have at the same time, the overall population-level concurrency index κ_P^{popul} increases, even if average lifetime numbers of partners remain constant.

3.2. R_0 and the contribution of the acute phase to R_0

The beginning of the epidemic is studied by considering R_0 and the relative contribution of the acute phase to R_0 for scenarios A2, B2, C2, and D2 in Fig. 4. R_0 increases with increasing symmetry in the population. In all four scenarios R_0 is above the epidemic threshold value one.

Transmission of disease during the primary phase is strongly determined by concurrency (combine Fig. 3 with Fig. 4). Only if concurrent partners are available during the primary phase can the infection be transmitted in that phase, because separation and finding a new partner requires a much longer time. In the polygynous scenario the network structure is made up of short chains of partnerships (involving a maximum of three individuals, see also Fig. 2). Therefore, it is reasonable that primary infection will have little impact in the polygynous scenario A2. The acute phase does play a

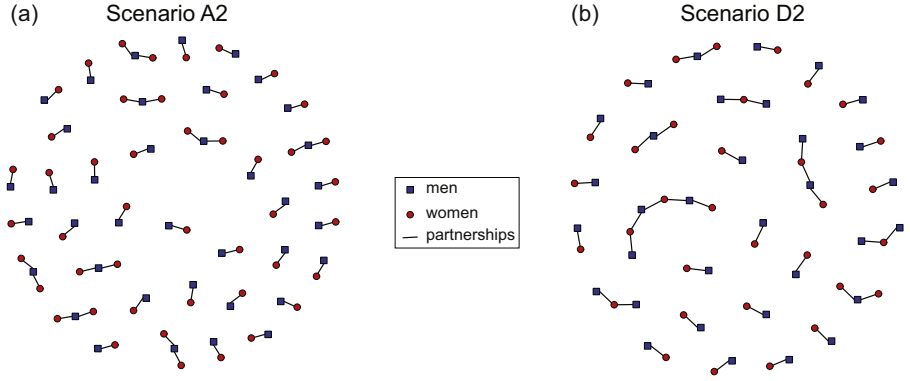

Fig. 2. Simulated configuration networks. The networks are shown at one point in time for a population size of 200 individuals from given degree distributions for scenarios A2 and D2. In (a) and (b) networks for scenarios A2 and D2, respectively, are simulated. At any point in time, a large part of the population is single (see also the degree distributions in Fig. 1: the probability that a man is single is around 0.66 in both scenarios, and the probability that a woman is single is 0.60 and 0.66 in scenarios A2 and D2, respectively). In network A2 there are 62 single males and 54 single females while in network D2 there are 67 single males and females. For clarity of the figures, these single individuals are not displayed in the two figures.

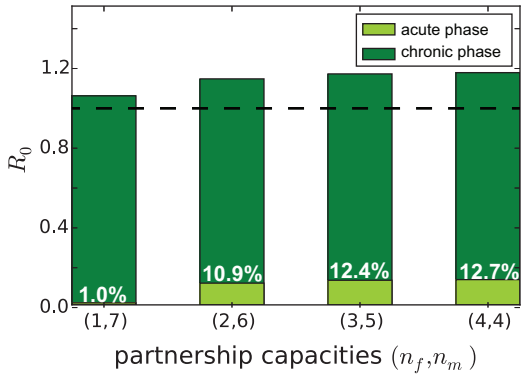

Fig. 4. R_0 in the four scenarios A2, B2, C2, and D2. The dashed line represents the epidemic threshold value of one. The light and dark green indicate the fraction of R_0 that is caused in the acute and chronic phase of infection, respectively, while the height of the bar represents R_0. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

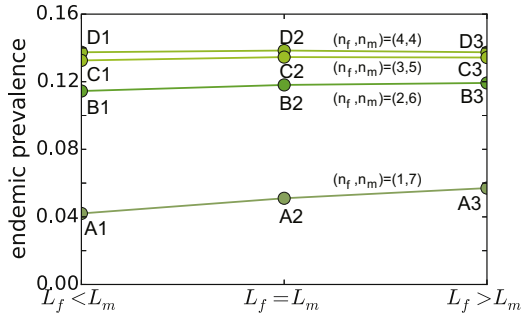

Fig. 5. The endemic prevalence in the population for the 12 different scenarios plotted as a function of sexually active lifespan with partnership capacities fixed. The corresponding scenario (see Table 1) is denoted for each point in the graph.

significant role in the other three scenarios with a contribution of up to ∼13% to R_0.

3.3. Endemic prevalence

In Fig. 5 population HIV prevalence is considered as a function of the concurrency and sexually active lifespan scenarios.

Similar to the quantities explored in Sections 3.1 and 3.2, from the results in Fig. 5 we conclude that gender asymmetry in sexually active lifespans have little effect on population prevalence (compare e.g. scenarios B1, B2, and B3 to each other, these scenarios have the same gender asymmetry in concurrency but different sexually active lifespan scenarios). Moreover, we see that gender asymmetry in concurrent partnerships does have an important effect on population prevalence (compare e.g. scenarios A1, B1, C1, and D1 to each other, where gender asymmetry in concurrent partnership is varied but sexually active lifespans are the same). In particular, we find that the polygynous setting A is associated with much lower levels of disease prevalence compared to the other concurrency settings B, C, and D. This is consistent with the network structure in setting A compared to the other settings (see also Fig. 2). In setting A, the longest chains of partnerships involve at most three individuals (men having more than one partner). Potentially much longer chains of partnerships can be found in the other settings (see Fig. 2 for a simulated cross-section of a network structure in scenario D2; scenarios B2 and C2 are in between A2 and D2 in terms of their network structure).

We investigate the gender asymmetry further by considering endemic prevalence separately for men and women as a function of partnership capacity scenarios in Fig. 6. The fact that endemic prevalence is in a reasonable range (DHS Program, 2010) is a consequence of our parameter value choice for $\bar{\theta}$ (recall Section 2.3: we *calibrated* $\bar{\theta}$ to an endemic disease prevalence of around 13% in the population for the scenarios B2 and C2; see Fig. 6). Our interest was in the qualitative behavior, in particular we are interested in comparing the endemic prevalences of scenarios A2, B2, C2, and D2 relative to each other. We find that, qualitatively, gender asymmetry in partnership capacity is associated with lower endemic prevalence in the population. Again, this implies that more equally distributed concurrency behavior can lead to higher epidemiological outcomes.

Next, there is also gender asymmetry in disease prevalence. In Fig. 6(b) and (c) female prevalence is higher than male prevalence for all four partnership capacity combinations. We find that the female-to-male ratio of HIV prevalence ranges from 1 in scenario D2 (symmetric sexual lifespans and partnership capacities) to 1.22 in scenario A3 ($L_f > L_m$ and greatest asymmetry in partnership capacities). This gender ratio is largest in Fig. 6(c). However, in Fig. 6(a) for $(n_f, n_m) = (3, 5)$, and (4, 4), female prevalence is slightly lower than male prevalence. Sensitivity analysis shows that this is a general result for partnership capacity combination $(n_f, n_m) = (4, 4)$ and $L_m > L_f$ (Fig. C.6 in Appendix C of the supplementary data). On the other hand, the same analysis shows that for $(n_f, n_m) = (1, 7)$, female prevalence is always larger than male prevalence regardless of gender differences in sexually active lifespans (so $(n_f, n_m) = (2, 6), (3, 5)$ lie somewhere in between). The female-to-male ratio of HIV prevalence is only slightly skewed towards women in Fig. 6. However, we considered only relatively small differences in lifespans between men and women. We find that the female-to-male ratio of HIV prevalence becomes much more skewed towards women when the female-to-male ratio of sexually active lifespans also increases (Fig. C.6 in Appendix C of the supplementary data).

We conclude that not only is gender asymmetry in concurrent partnerships associated with lower disease prevalence, it also affects the female-to-male ratio of prevalence. Gender asymmetry in concurrent partnerships is associated with an increased gender asymmetry in disease prevalence. While gender asymmetry in sexually active lifespans has little effect on total population prevalence, it does affect the gender ratio in disease prevalence.

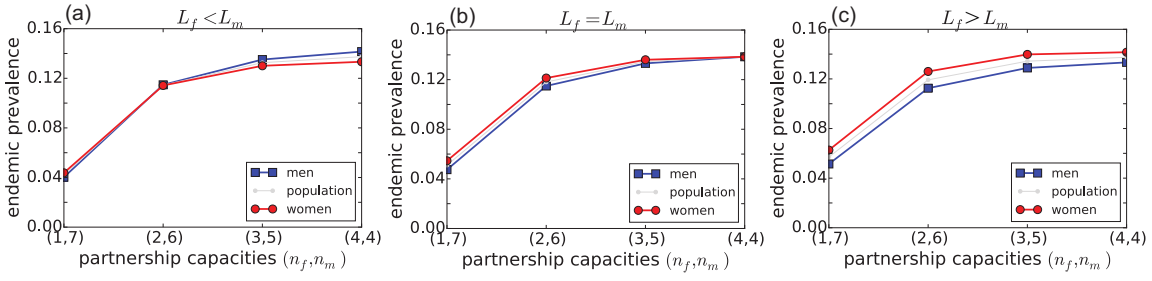

Fig. 6. The endemic prevalence as a function of different partnership capacity combinations with sexually active lifespans (a) $L_f < L_m$, (b) $L_f = L_m$ and (c) $L_f > L_m$.

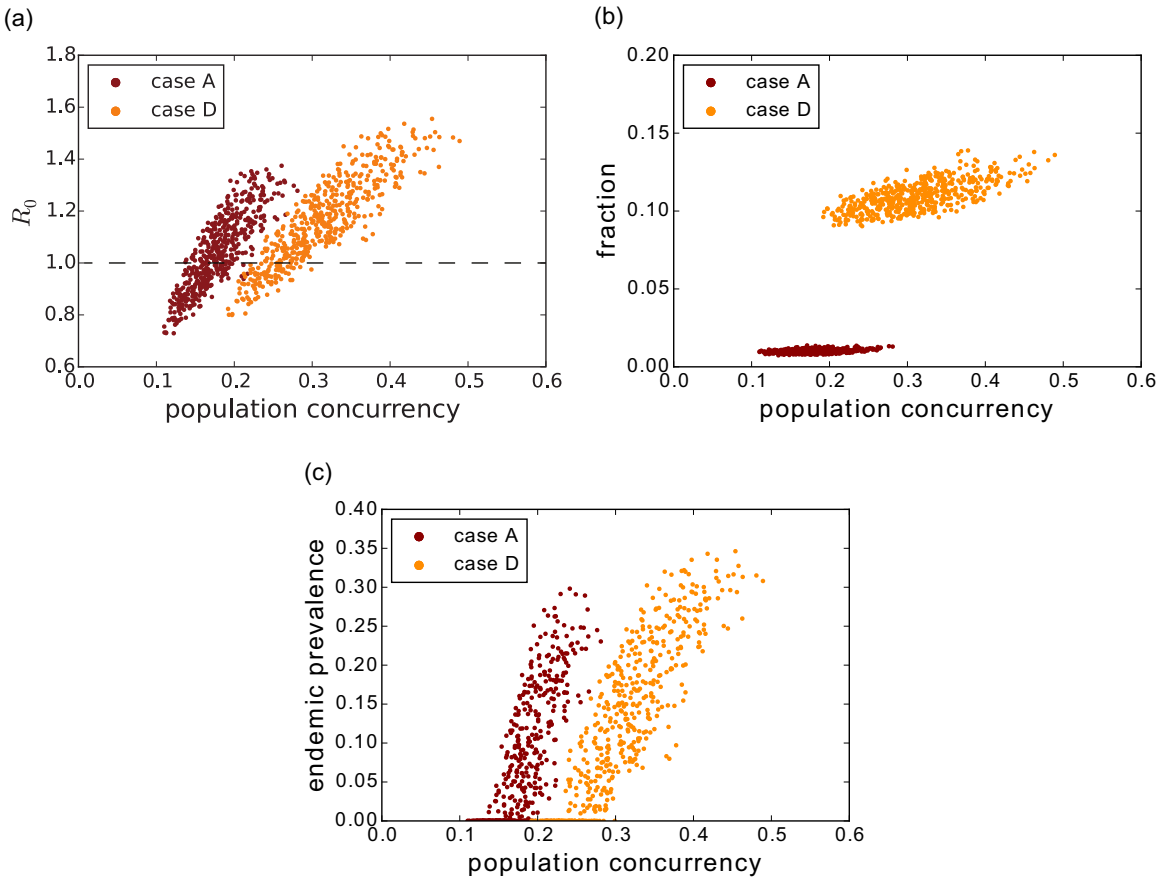

Fig. 7. Correlation between population concurrency and (a) R_0, (b) the relative contribution of the acute phase to R_0, and (c) population prevalence, when varying model parameters in the sensitivity analysis (see Appendix C of the supplementary data). Case A corresponds to the asymmetric polygynous setting and case D corresponds to the symmetric setting.

3.4. Concurrency and epidemiological outcomes

Taking the results of Sections 3.1–3.3 together, we conclude that the epidemiological quantities R_0, the relative contribution of the acute phase to R_0, and the endemic prevalence are increasing with increasing concurrency in the population (combine Fig. 3 with either Fig. 4, or Fig. 6). The effect of gender-asymmetric concurrency on epidemiological parameters thus appears to be in part mediated through increased overall concurrency. Moreover, in our sensitivity analysis where we allow for variation in all parameter values simultaneously, we find a very strong correlation between population-level concurrency and R_0 and population disease prevalence; see Fig. 7(a) and (c) and Appendix C of the supplementary data for details. This suggests that, although all sexual behavior parameters separately have an impact on prevalence, the synergistic combination of these parameters in the concurrency index κ_P^{popul} yields a much more pronounced correlation. This correlation is much less strong (but clearly still existent) when considering the relative contribution of the acute phase to R_0; see Fig. 7(b).

4. Conclusion and discussion

In this study we investigated the effect of gender asymmetry in the maximum number of simultaneous partnerships an individual can have, the so-called partnership capacity, on HIV disease dynamics. Our study was qualitative in nature and cannot be used to make any quantitative statements. Motivated by the findings of Reniers and Watkins (2010), we were especially interested in the extreme case of polygynous unions, i.e. $(n_f, n_m) = (1, 7)$, with additional interest in other types of asymmetries. We found that all three epidemiological outcomes R_0, the relative contribution of the acute phase to R_0, and endemic prevalence are much lower in the polygynous scenario than in more gender symmetric cases. These findings highlight polygyny as a special case of asymmetric concurrency that may be particularly protective at the population level. However, the epidemiological outcomes were also lower in the other two gender asymmetric scenarios $(n_f, n_m) = (2, 6)$ and $(3, 5)$ compared to the symmetric scenario $(n_f, n_m) = (4, 4)$. Our model results show that any level of asymmetry leads to lower endemic prevalence than a population with full symmetry. Therefore, consistent with the findings of Reniers and Watkins (2010), our results support that polygyny, even if practiced only by a part of the population, can indeed reduce HIV prevalence as compared to a fully mixed population where both sexes have equal numbers of concurrent partners. However, based on our study, where we controlled for many network statistics such as mean partnership duration, it is not possible to say how the effect of polygyny in the model extends to a causal explanation for the ecological correlation between polygyny and prevalence in Reniers and Watkins (2010).

We also investigated the influence of gender asymmetry in sexually active lifespans L_f and L_m for women and men, respectively. Women tend to enter the sexually active population at a younger age than men (Demographic and Health Surveys, 2015). At the same time, men tend to remain sexually active at older ages when women are no longer so (Demographic and Health Surveys, 2015). Since it is not clear how these effects together exactly influence mean times that men and women spend

in the sexually active population, we considered three different cases (but see Lindau and Gavrilova, 2010 for an estimation of sexually active lifespans for a heterosexual population in the USA).

Nevertheless, gender asymmetry in sexually active lifespans did not qualitatively change our findings about gender asymmetry in partnership capacities and the association with the epidemiological quantities under consideration. Additionally, we found that in case of gender symmetric sexually active lifespans ($L_f = L_m$) or longer sexually active lifespans for women than men ($L_f > L_m$), the female-to-male ratio of HIV prevalence is skewed towards women. The sensitivity of HIV prevalence ratios to differences in sexual lifespans when we considered a broader range (Fig. C.6 in Appendix C of the supplementary data) indicates that more empirical information – and attention in modeling – on sexually active lifespans and their effects is needed to better understand these durations.

Consistent with our findings, HIV prevalence data indeed shows that more women than men are living with HIV in sub-Saharan Africa, e.g. in Malawi (UNAIDS, 2013; DHS Program, 2010). Empirical estimates of the female-to-male ratio of infections in sub-Saharan African populations range from 1.31 to 2.21 (Reniers et al., 2015). Many factors can lead to these differences, and one of them could be the difference in concurrent partnerships between men and women. The ratios in our study are smaller than the empirical estimates, most likely because we only investigated the effect of gender asymmetry in concurrent partnerships.

Our main focus was on the effect of gender asymmetry in partnership capacities on HIV prevalence while controlling for mean partnership duration, mean lifetime number of partners, mean degree, sexually active lifespan, and infection parameters. We were not trying to isolate the effect of these asymmetries independently of overall concurrency, rather we wanted to see the total effect of these asymmetries (even if partially mediated through changes in overall concurrency). We found that any of the sexual behavior parameters, such as lifetime number of partners and partnership duration correlate positively with the epidemiological quantities. There is a particularly strong correlation between population-level concurrency with R_0 and population disease prevalence (Section 3.4). Similar questions on gender asymmetry in concurrency and HIV prevalence were addressed in Reniers et al. (2015). Their focus was on comparing polygyny and gender-symmetric concurrency and their effect on the female-to-male ratio of HIV prevalence using an agent-based modeling approach. In our study we looked at other types of gender-asymmetric concurrency beyond polygyny. Moreover, in our study, the levels of concurrency in the population were determined by sexual behavior parameters that were kept constant throughout different scenarios. As a consequence, gender asymmetry led to a slightly lower level of overall concurrency in the population compared to gender symmetry. Therefore, results from our study are not directly comparable with Reniers et al. (2015). However, consistent with our findings, Reniers et al. (2015) found that higher levels of concurrency are associated with higher disease prevalence.

There are of course many factors that can play a role in determining disease dynamics. For example, age mixing (where young women tend to form partnerships with older men) has been proposed as an explanation in creating the difference in HIV prevalence in men and women (Kelly et al., 2003; Leclerc-Madlala, 2008; Maughan-Brown et al., 2014). But note that there are also longitudinal cohort studies finding no increase in HIV acquisition among young women with older partners in South Africa (Jewkers et al., 2012; Harling et al., 2014; Balkus et al., 2015). In our model, partner acquisition (or separation) does not depend on the age of individuals at all. Changes in behavior over the course of the epidemic and heterogeneity in behavior across individuals of a given sex are also not considered. Obviously these factors may also be of influence, although non-linearities in epidemic systems make it difficult to predict the exact nature of these influences (but see Reniers et al., 2015 where they find that behavior change among HIV-positive women can enlarge the gender asymmetry in disease prevalence).

Our model also did not differentiate between long- and short-term partnerships. With respect to partnership duration, we did perform sensitivity analysis that showed that endemic prevalence and R_0 increase with increasing partnership duration. In a population with only short term partnerships, endemic transmission cannot be maintained (without increasing numbers of partners). However, we hypothesize that in a population with a combination of short and long partnerships, there could be a synergistic effect of the two, with short-term partnerships enabling transmission during primary infection and long-term partnerships ensuring a sufficiently high reproduction number. Future modeling work that includes different types of partnerships will allow analyses of gender-asymmetric concurrency effects on epidemiological parameters within this type of more realistic system.

Another model aspect worth mentioning is that we did not include any disease-related mortality, which would shorten the infectious period. As a consequence, infectious individuals would remain in the sexually active population for a shorter time, causing fewer secondary cases than if there is no disease-related mortality. Inclusion of this phenomenon could then lead to lower levels of disease prevalence. However, assuming that disease-related mortality affects both genders in the same way, we believe that it will not influence the relationships demonstrated in this paper.

In conclusion, gender asymmetry in the maximum number of concurrent partnerships can affect the epidemic and endemic states of HIV transmission dynamics, with these effects being partially mediated through changes in overall concurrency levels. Our findings suggest that increased asymmetry is associated with decreased endemic prevalence, R_0, and the acute-phase contribution to R_0, particularly in the special case of polygyny. This provides potential mechanistic explanations for associations between polygyny and key epidemiological measures of disease transmission. The findings in this study help to round out prior empirical and modeling studies by adding considerations of scenarios between the two extremes of symmetric concurrency and polygyny. We also find that gender imbalances in concurrency may partially explain higher observed HIV prevalence in females vs. males in contexts where sexual lifespans for females are at least as long as for males. Taken together, our findings indicate that improved empirical understanding of male-vs-female concurrency patterns and sexual lifespans, combined with our modeling insights about the effects of these quantities on HIV transmission dynamics, could improve understanding of observed epidemiological patterns across HIV-endemic settings.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.epidem.2017.01.003.

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