Polygyny and the spread of HIV in Sub Saharan Africa: A case of benign concurrency

Georges Reniers
Susan Watkins

Polygyny and the spread of HIV in Sub Saharan Africa: A case of benign concurrency

Georges Reniers, Office of Population Research, Princeton University

Susan Watkins, California Center for Population Research, University of California-Los

Angeles

Corresponding author:

Georges Reniers

Office of Population Research, Princeton University

257 Wallace Hall, Princeton, NJ 08544

tel: +1 609 258 5513, fax:+1 609 258 1039

e-mail: greniers@princeton.edu

Word count – abstract : 214

Word count – main text (excl. abstract, acknowledgements, references, tables/figures): 3489

Objectives

Most of our understanding about the effect of concurrent sexual partnerships on the spread of HIV derives from mathematical models, but the empirical evidence is limited. In this contribution, we focus on polygyny, a common and institutionalized form of concurrency, and study its relationship with HIV prevalence at the ecological level.

Methods

First, we describe country-level variation in the prevalence of polygyny and HIV. Second, we test the relationship between HIV and polygyny at the sub-national level using country fixed-effects regression models with data from nineteen Demographic and Health Surveys.

Results

The ecological association between polygyny and HIV prevalence is negative at the country as well as sub-national level: HIV prevalence is lower in countries where the practice of polygyny is common, and within countries it is lower in areas with higher levels of polygyny. Proposed explanations for the protective effect of polygyny include the distinctive structure of sexual networks produced by polygyny, the disproportionate recruitment of HIV positive women into marriages with a polygynous husband, and the lower coital frequency in conjugal units of polygynous marriages.

Conclusion

Existing models of concurrency are not sufficiently specific to account for the protective effect of polygyny on the spread of HIV, and require refinements before they are to be used for informing HIV prevention policies.

Introduction

The practice of concurrent sexual partnerships is considered to be a critical driver of the HIV/AIDS epidemic in high prevalence countries in sub-Saharan Africa (SSA) [1-3]. The upsurge of interest in concurrency is based on insight from persuasive mathematical models demonstrating that overlapping sexual partnerships are more efficient loci for the spread of infectious diseases [4, 5]. Under serial monogamy, the spread of the virus beyond the couple is interrupted until the relationship dissolves and a new partnership is formed. In contrast, a salient feature of concurrency is that it inflates the number of individuals who are directly or indirectly connected at any point in time. As a result the virus can be transmitted relatively quickly through the sexual network. In addition, there is biological evidence that high levels of viral load in acute infection increase the probability of transmitting HIV [6, 7], which becomes particularly pertinent if individuals have overlapping partnerships. The conviction that concurrency is critical has gained support from the observation that there is both more concurrency and higher HIV prevalence in SSA than elsewhere [1, 8, 9].

These arguments are suggestive indeed, but the weak link in the case for a concurrency effect is the lack of direct empirical evidence linking concurrency to the faster and more pervasive spread of HIV [10, 11]. A few studies failed to identify a consistent concurrency effect [12-14], but these often focused on the relationship between the respondent's (concurrent) partnerships and his or her own HIV status, whereas the concurrency model stresses the importance of overlap in the respondent's partnerships for transmitting HIV to someone else. In other words, these studies largely assessed the role of concurrency as a risk factor for HIV *acquisition*, but failed to test whether concurrency has a

measurable effect on the *transmission* of HIV. Sexual network studies based on partner-tracing designs (i.e., designs whereby the partners of an index case are traced and tested for HIV) should in principle facilitate more rigorous tests, but substantial populations need to be covered to produce convincing statistical evidence [15].

Our point of entry in this debate is to focus on a special form of concurrency, namely polygyny, and to examine its association with HIV prevalence. Polygyny is an institutionalized form of concurrency that is probably more accurately (although not perfectly [16]) reported than informal partnerships. Moreover, polygynous marriages are likely to be less transient than informal partnerships, and they account for a substantial share of all concurrent partnerships in SSA [12, 17]. In a survey conducted in Zambia in 1998, for example, 17.8% of rural Zambian men aged 25-49 reported more than one ongoing relationship; when polygynous marriages were excluded, the percentage of concurrent relationships dropped to 9.1% [17].

We thus propose that an analysis of the association between polygyny and HIV provides a valuable empirical contribution to understanding the relationship between overlapping sexual partnerships and HIV transmission. We focus on the ecological relation between polygyny and HIV because (1) most readily available datasets are ego- or respondent-centered, and these are by design not useful for demonstrating the effect of concurrency on HIV transmission (cfr. supra), and (2) HIV prevalence as a measure of epidemic severity is an attribute of aggregates rather than individuals.

Data and methods

We first present country-level associations between HIV prevalence and polygyny using secondary data sources. Second, we seek to confirm our findings at a lower level of aggregation (the survey cluster) using data from nineteen African Demographic and Health Surveys (DHS) and HIV/AIDS Indicator Surveys (AIS) with individually-linked survey and HIV serostatus data. The DHS and AIS use a two-stage randomized cluster sample design. Survey clusters are the smallest area units comparable to enumeration areas in a national census. A cluster contains around 100 households, and there are on average 379 (standard deviation = 80) clusters per survey in the data used here. In the second sampling stage, a predetermined number of households are selected from each cluster. In each household, all eligible women (aged 15-49) and a selection of eligible men (aged 15-54 or 15-59) are interviewed. Between 20 and 40 women are typically interviewed per survey cluster [18]. DHS and AIS data, survey instruments, and other documentation can be retrieved from the Measure DHS website (http://www.measuredhs.com). A disadvantage of the DHS and AIS, is the lack of detail on marriage and partnerships (e.g., no full marriage histories) and, sometimes also, the lack of standardized questions (e.g., the wording of the question about current marital status has slightly changed over time). The DHS and AIS are nonetheless important resources for studying concurrency because they constitute the largest collection of comparable datasets from African countries with partnership information linked to HIV serostatus. In our analysis we combine consensual and formal unions, and the definition of the polygynous character of the union is based on a question about the number of (co-)wives. We refer to a study by

Timæus and Reynar [16] for a discussion of the correspondence in survey responses to these questions between husbands and wives.

We use DHS and AIS data in conjunction with a count-response model with a negative binomial distribution to model the number of prevalent infections per survey cluster with the logarithm of the total number tested per cluster as an exposure or offset parameter. A negative binomial distribution is a generalization of the Poisson model with an additional parameter that accounts for over-dispersion in the data [19]. One source of over-dispersion in count models is event-dependency. In this case, event-dependency is produced by the infectiousness of HIV, and –to the extent that partnership markets are geographically bound– the ensuing correlation between an individual's likelihood of being HIV positive and the HIV prevalence in the survey-cluster to which (s)he belongs. Empirically, over-dispersion can be diagnosed when the variance of the count distribution is larger than its mean. We start with an analysis of pooled –but sex disaggregated– datasets for the nineteen countries, and include dummies for all but one country to obtain unconditional fixed-effects estimates. We also present the results from country-wise regression analyses.

The regression framework allows us to control for selected characteristics that have previously been identified as risk factors of HIV infection. The most plausible are age, the type of place of residence [20], the extent of male circumcision [21-23], the interval between the age at sexual debut and first marriage [24], the prevalence of other sexually transmitted infections (STIs) [25, 26], religion [27], mobility or migration [28], and the prevalence of non-marital sex. Age is measured as the sex-specific median age in each survey cluster. The type of place of residence is an indicator variable that contrasts urban and rural clusters. Our

measures of first marriage and sexual initiation are survey cluster life table values of the median age at these events (values are imputed at the age of the oldest individual plus one for clusters where over 50% of respondents are self-reported virgins or single). The other indices are operationalized as percentages. The measure of religion distinguishes between Islam and other religions, and the prevalence of STIs is based on self-reports of genital sores, ulcers or discharge. It has been categorized (0%, 0-10% and >10%) because its distribution is highly skewed. Mobility is measured as the percentage of interviewed men (the information is not available for women) who spent more than one month away from their home. The measure of non-marital sex is based on self-reports of married respondents. The prevalence of STIs, male mobility and non-marital sex all pertain to the year before the survey.

In the regressions models, we define the prevalence of polygyny is defined as the average of the percentage of married men and married women in polygynous unions. We average male and female centered-definitions of polygyny because it smoothes some of the measurement error and random variation, and because it accounts for two aspects of polygyny, namely the *incidence* (the proportion of men with more than one wife), and the *intensity* of polygyny (the average number of wives per polygynyst) [29].

Results

The scatterplots in Figure 1 suggest that polygyny and HIV prevalence are negatively correlated: western and central African countries have generally higher levels of polygyny and lower HIV prevalence than populations of eastern and southern Africa, and vice versa. The relationship holds for female-centered definitions of polygyny (the percentage of

women with one or more co-wives) as well as male-centered definitions (the percentage of men with more than one wife).

Figure 1

This country-level analysis is, however, a weak test of the polygyny-HIV association because there are several possibly confounding factors that are unaccounted for. In Table 1, we thus present results from regression models that aim to circumvent the omitted variable bias. In these analyses, we change the level of aggregation from the country to the survey cluster-level, and regress the sex-specific count of HIV positives in each survey cluster on the prevalence of polygyny. We first present crude effects, followed by effects adjusted for the statistical controls described earlier. Figure 2 summarizes the distribution of most of these variables. We present the regression parameters in exponentiated form or incidence rate ratios (IRR), followed by their z-statistic. Control variables are only included in the full (adjusted) models if they are significant for at least one of the sexes.

Figure 2

Table 1

The results in Table 1 corroborate the negative association between polygyny and HIV prevalence. In the presence of other statistical controls, the IRR is 0.995 (95% confidence

interval: 0.991-0.998) for men and 0.995 (95% confidence interval: 0.993-0.998) for women. This translates into a 0.5% decrease in HIV prevalence for each one percentage point increase in the prevalence of polygyny. This is a considerable effect given the heterogeneity in the prevalence of polygyny within countries: the smallest 10-90th percentile range for the prevalence of polygyny across clusters is 19.1 percentage points (Rwanda), and the largest value is 57.2 points (Burkina Faso)(see also Figure 2).

The effects of the other covariates largely operate in the expected direction. Age has a positive effect, indicating that clusters with older respondents have a higher HIV prevalence (the effect is curvilinear for men). HIV prevalence is generally higher in urban areas, and male circumcision has a protective effect. Although the percentage of Muslims tends to be negatively correlated with HIV prevalence, its effect is not significant after adjusting for other controls. A more thorough evaluation of religion should probably include a measure of religious involvement [30], but that is not available in the DHS. The effect of this measure of male mobility is not significantly either. Late marriage is protective (particularly for women), but its effect should be interpreted in conjunction with the parameter estimate for the duration between first sex and first marriage. That suggests that late marriage will only contain the spread of HIV if it is not accompanied by pre-marital sexual activity. As could be expected, the latter is positively correlated with HIV prevalence. STI symptoms are another important covariate of HIV prevalence, and its effect appears stronger for women than for men (this could be due to sex differences in the reporting of STI symptoms). The percentage of married men and women reporting a non-marital sex partner

is also a predictor of HIV prevalence. Importantly, none of these controls eliminate the apparent protective effect of polygyny at the population-level.

We extended the analyses in two respects. First, we estimated the model with all statistical controls for each country and sex separately, and plotted the z-statistic for the effect of polygyny in these regressions in Figure 3. Despite the relatively small number of clusters per country, seven of the parameter estimates are negative and significant at the 10% level (z-score ≤ -1.64), and another seven are significant at the 5% level (z-score ≤ -1.96). None of the parameter estimates are positive and statistically significant, and that gives further support to the results from the pooled analysis. Second, we fitted models with self-reports of STI symptoms instead of HIV as the outcome (pooled analysis for all countries). In these models, the effect of polygyny is insignificant, suggesting that the mediating effect of polygyny might be different for HIV and other STIs.

Figure 3

Discussion

Our analyses establish a negative ecological relationship between polygyny and HIV prevalence at two levels of aggregation. At the country-level, the association is most obvious, but it is confirmed by analyses that only exploit within-country variation and control for other factors that are suspected to affect the spread of HIV (e.g., the prevalence of STIs, male circumcision, and the length of the pre-marital sexually active period). These findings

are perhaps unexpected, and it is worth reflecting on the conditions that would render them consistent with predictions of the concurrency model.

We begin by acknowledging that concurrency could be driving our results if survey clusters with lower levels of polygyny are characterized by higher levels of informal concurrency and vice versa. Whereas we included statistical controls for the prevalence for pre-marital and extra-marital sex, we cannot rule out that these measures affected by reporting bias. Prior analyses of the relationship between polygyny and extra-marital sex are, not conclusive either [31, 32]. A more interesting point relates to the sexual network structure produced by polygyny. On the one hand, polygyny generates a pattern of dissasortative mixing whereby high-degree nodes (men who already have one or more partners) are paired with low degree nodes (women). As Morris and Kretzschmar have shown [33], dissasortative mixing is likely to *increase* the final epidemic size compared to a random mixing model. This would not bring us closer to reconciling our findings with predictions from their concurrency model, were it not for another respect in which polygyny constitutes a special case. Barring extra-marital relationships, polygyny produces a system of gender-asymmetric dissasortative mixing: this implies that the size of the largest temporally connected sexual network component will not exceed the maximum number of wives of any of the individual men in the population (see also [34]). Thus polygyny, in effect, creates small isolates of concurrent partnerships in which the virus is trapped until one or more of the (infected) spouses start a new relationship (Figure 2C). Gender-symmetric concurrency produces larger temporally connected network components (Figure 2B), and that facilitates a faster and more pervasive spread of the virus.

Figure 4

The arguments presented so far imply that polygyny hinders epidemic growth compared to a scenario of gender-symmetric concurrency. It is unlikely, however, that the sexual network structure alone can fully account for the negative statistical relationship between polygyny and HIV observed in this study. For a number of plausible complementary effects, we refer to analyses of individual-level data that suggest that there are two other features of polygyny that influence the spread of HIV over and above the structural network effects discussed above [31, 35]. *First,* polygynous marriage systems are in large part sustained by the rapid remarriage of divorcees and widows (often as second or third wives of polygynous men, and sometimes via the practice widow inheritance) [36, 37]. Because higher marriage order and widowhood are positively correlated with HIV status [38-40], the addition of new wives is likely to introduce HIV into what might have been an HIV-free monogamous marriage. This would, of course, fuel the epidemic, were it not for a *second,* and counterbalancing characteristic of polygynous marriages that delays the spread of the virus. We conveniently label it a *coital dilution* effect [41, 42]: compared to a monogamous man, a polygynous husband divides his time between two or more wives, which inevitably leads to a reduction in the coital frequency with each wife. Just as coital dilution is claimed to affect fertility in polygynous marriages [43-45], it could reduce HIV incidence in serodiscordant couples with a polygynous husband. The reduction in coital frequency not only arises from the resource constraints on a polygynous husband's coital budget, it may

also result from the relatively old age of husbands in polygynous unions, and, more interestingly, from a conscious decision to reduce the risk of transmitting HIV (e.g., the new husband of an inherited spouse may be aware of the cause of death of his wife's previous husband). Together, these two mechanisms produce a mixing pattern whereby HIV positive women are disproportionately recruited into polygynous marriages where coital frequency is lower. As a result, seronegative *individuals* in polygynous marriages may face greater exposure to HIV than those in a monogamous marriage, but the *population* level effects of polygyny on the spread of HIV are beneficial on average. The policy implications of this finding will depend on whether individuals ought to be the primary beneficiaries of public health policy interventions, or, the population at large.

Other avenues through which polygyny may affect the spread of HIV deserve more careful consideration than we have been able to provide. Polygynous marriage systems may, for example, exert greater control over female sexuality or restrict younger men's access to women, and, as a result, not only reduce the coital frequency in conjugal units of polygynous marriages, but also among those who are single. Given that the polygyny effect persists after accounting for the duration of the pre-marital sexual interval and the prevalence of non-marital sex, this mechanism is, however, unlikely to fully account for the polygyny effect observed in this study. We also recognize that polygyny could have a different effect at different stages of the epidemic, just as it has been demonstrated that disassortative mixing on the number of existing partners can have different effects on the take-off and leveling of a simulated epidemic [5]. Similarly, we have not explored whether the mediating effect of

polygyny is dependent on the distribution of wives per polygynous husband (i.e., whether it

is also dependent on the intensity of polygyny)

Because of the cross-sectional nature of the DHS and AIS, we are limited to a current

status measure of polygyny (and most other control variables) whereas prevalent HIV

infection is the cumulative result of past exposure. It is unlikely, however, that the

prevalence of polygyny has changed over time such that the association between polygyny

and HIV prevalence would have reversed entirely. We tested this competing hypothesis by

analyzing the correlation between changes in the practice of polygyny and HIV prevalence.

Of the countries with more than one DHS, the proportion of married women in polygynous

marriages declined in all but one (Eritrea). The proportion of men in polygynous unions only

increased in Guinea. The annual rate of change in the prevalence of polygyny between the

first and the DHS with information on polygyny is, however, not significantly correlated with

HIV prevalence (p=0.21 and p=0.85 for women and men, respectively).

Despite these open questions and limitations, it is clear from the rather benign

relationship between polygyny and HIV that refinements to the description of concurrency

effects on the spread of HIV are in order. Polygyny produces a specific pattern of sexual

mixing with outcomes that are not accommodated by existing models. We believe that

future models of concurrency need to incorporate (1) variability in sexual mixing patterns

and how these correlate with concurrency and HIV status, and (2) the heterogeneity across

union types in coital frequency. More realistic models of concurrency need to account for (3)

the variable infectiousness by duration since infection as well. Such models might also help

us understand whether, and if so why, the relationship between polygyny and HIV is different from its relationship with other STIs (as our analyses seem to suggest).

In the above, we have described polygyny as a special type of concurrency, but it is not an uncommon form of concurrency in SSA. Our study indicates that polygynous marriages may require special treatment in epidemiological models of concurrency, particularly if they are to inform public health policies. Policies that target concurrency in a generic fashion are likely to be as culturally insensitive as 19th and 20th century missionary efforts to ban polygyny. In addition, the negative association identified in this study suggests that they may have counterproductive public health implications. We conclude by invoking the practice of widow inheritance as a concrete illustration. Whereas the re-entry of widows into the marriage or partnership market implies a non-negligible risk of transmitting HIV, an important social function of widow inheritance is to provide a safety net for the surviving spouse, who may or may not have been previously infected [46]. Women in populations where the practice is common are embedded in the lineage of their husband and their livelihood is, at least in principle, independent of their husband's survival. If a widow remarries her former husband's relative, she is likely to become a higher-order spouse in a polygynous marriage, which, as we have argued above, implies a reduction in her sexual activity level. In the absence of widow inheritance (and adult male children), a widow may need to seek an alliance with another man to secure her own and possibly her children's livelihoods, and that process may induce an even higher risk of transmitting HIV.

Acknowledgements

We wish to thank Jimi Adams, Ron Brookmeyer, Jeffrey Eaton, Stephane Helleringer and

Jenny Higgins for useful comments on earlier versions of the manuscript.

Figure 1: Prevalence of HIV (age 15-49, both sexes) and polygyny (by sex), Sub-Saharan Africa
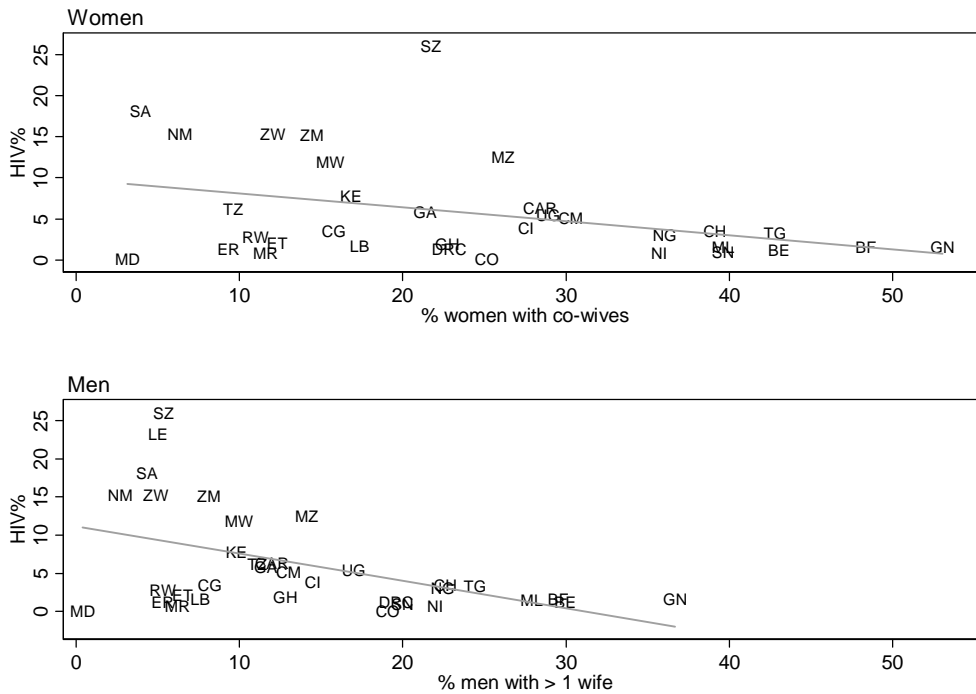
Notes: Estimates of adult HIV prevalence (both sexes combined) are UNAIDS estimates for 2007[47], and the estimates of prevalence of polygyny come from a recent DHS survey. The year of the survey is listed following the country label used on the graph: Benin (BE, 2006), Burkina Faso (BF, 2003), Cameroon (CM,2004), Central African Republic (CAR, 1994-95), Chad (CH, 2004), Comoros (CO, 1996), Congo (CG, 2005), Ivory Coast (CI, 2005), Democratic Republic of Congo (DRC, 2007), Eritrea (women only, ER, 2002), Ethiopia (ET, 2005), Gabon (GA, 2000), Ghana (GH, 2003), Guinea (GN, 2005), Kenya (KE, 2003), Lesotho (LE, 2004, men only), Liberia (LB, 2007), Madagascar (MD, 2003-04), Malawi (MW, 2004), Mali (ML, 2006), Mauritania (MR, 2000-01), Mozambique (MZ, 2003), Namibia (NM, 2006-07), Niger (NI, 2006), Nigeria (NG, 2003), Rwanda (RW, 2005), Senegal (SN, 2005), South Africa (SA, 2003), Swaziland (SZ, 2006-07), Tanzania (TZ, 2004-05), Togo (TG, 1998), Uganda (UG, 2006), Zambia (ZM, 2007), Zimbabwe (ZW, 2005-06).

Figure 2: Sample descriptive statistics (10-90th percentiles range and median) by country and sex
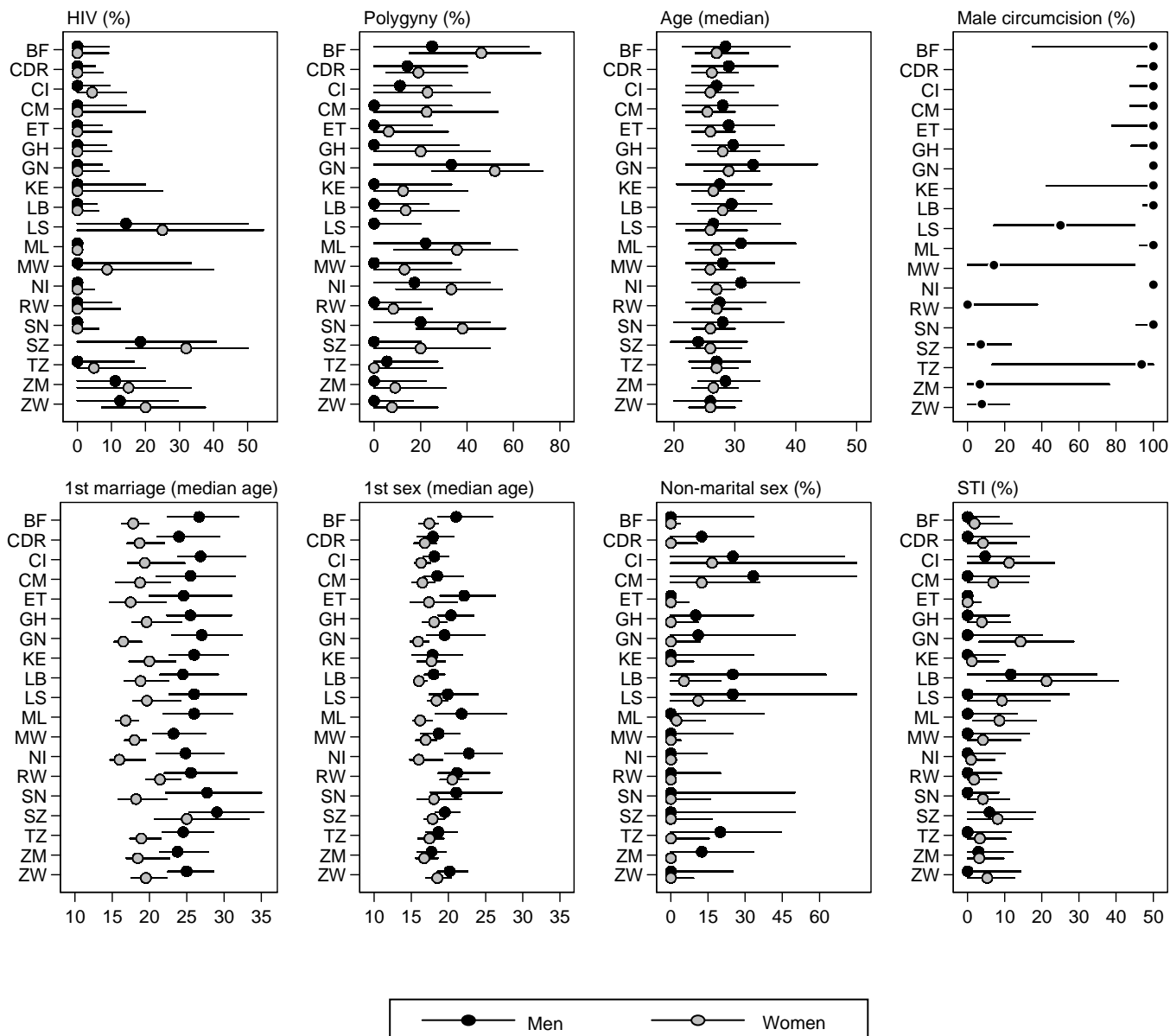
Notes: The unit of analysis is a survey cluster. The range between the 10th and 90th percentile is depicted by the horizontal lines; the dots represent the median for men and women, respectively. Countries included: Burkina Faso (BF, 2003), Democratic Republic of Congo (DRC, 2007), Ivory Coast (CI, 2005), Cameroon (CM, 2004), Ghana (GH, 2003), Guinea (GN, 2005), Kenya (KE, 2003), Liberia (LB, 2007), Lesotho (LS, 2004), Mali (ML, 2006), Malawi (MW, 2004), Niger (NI, 2006), Rwanda (RW, 2005), Senegal (SN, 2005), Swaziland (SW, 2006-07), Tanzania (TZ, 2004-05), Zambia (ZM, 2007), Zimbabwe (ZW, 2005-06). The question about the number of co-wives was not included in the female questionnaire for Lesotho. The distribution for the type of place of residence is not shown: survey clusters are either entirely urban or rural, and the 10-90th percentile range spanning both values for all countries.

Table 1: Unconditional country fixed-effects negative binomial regression models predicting the count of HIV positives per survey cluster (pooled analysis for 19 African countries)

Variables	Women Unadjusted IRR	Adjusted IRR	Men Unadjusted IRR	Adjusted IRR
% Polygynous	0.993***	0.995***	0.992***	0.995***
	(-6.201)	(-3.908)	(-4.789)	(-3.313)
Age [a]	1.084	1.018***	1.216***	1.152***
	(1.565)	(4.041)	(6.638)	(4.837)
Age - squared [a]	0.999	n.s.	0.997***	0.998***
	(-1.473)		(-5.679)	(-3.807)
Urban (versus rural)	1.619***	1.610***	1.584***	1.511***
	(15.92)	(14.10)	(11.52)	(9.403)
% Circumcised (men)	1.000	0.998***	0.998**	0.996***
	(-0.409)	(-3.354)	(-2.125)	(-4.625)
% Muslim	0.998**	n.s.	0.998*	n.s.
	(-2.230)		(-1.858)	
Mobility (% of men >1 month elsewhere)	1.000	n.s.	0.999	n.s.
	(0.0405)		(-0.595)	
Median age 1st marriage				
Women	1.030***	0.925***	1.016**	0.944***
	(6.683)	(-6.316)	(2.514)	(-3.586)
Men	1.029***	n.s.	1.015***	n.s.
	(7.300)		(2.811)	
Difference median age 1st marriage –				
Women	1.037***	1.095***	1.021***	1.058***
	(7.231)	(6.864)	(2.909)	(3.285)
Men	1.034***	1.023***	1.023***	1.019***
	(9.160)	(6.011)	(4.637)	(3.680)
% Reporting STI symptoms (versus 0%)				
Women: ≤10%	1.226***	1.189***	1.260***	1.228***
	(5.027)	(4.302)	(4.259)	(3.836)
>10%	1.345***	1.314***	1.612***	1.522***
	(6.116)	(5.651)	(7.526)	(6.717)
Men: ≤10%	1.084**	n.s.	1.106**	n.s.
	(2.197)		(2.114)	
>10%	1.123***	n.s.	1.328***	n.s.
	(3.015)		(5.708)	
% Reporting non-marital sex				
Women	2.393***	1.588***	2.410***	1.444*
	(6.040)	(3.146)	(4.531)	(1.888)
Men	1.543***	1.278***	1.776***	1.496***
	(5.971)	(3.318)	(5.925)	(4.106)
N (missing)		6867(300)		6812(299)

Notes:
*** p<0.01, ** p<0.05, * p<0.1, z-statistics (based on the sum of the outer product of the gradient vectors (OPG) variance estimator) in parentheses, n.s.: predictor is not significant in the adjusted regression models for either sex and omitted [a] Age is defined as the survey-cluster median age of the male respondents in the models for men, and median age of female respondents in the models for women. Male respondents' median age was not a significant predictor of the count of HIV positives in the model for women and vice versa. See text for variable definitions. See Figure 2 for a list of countries included in the analysis. For Lesotho, the % in a polygynous union is entirely based on responses for men (the question was not asked to women).

Figure 3: The adjusted effect (z-score) of the prevalence of polygyny on the count of HIV positives per survey cluster, by country and sex
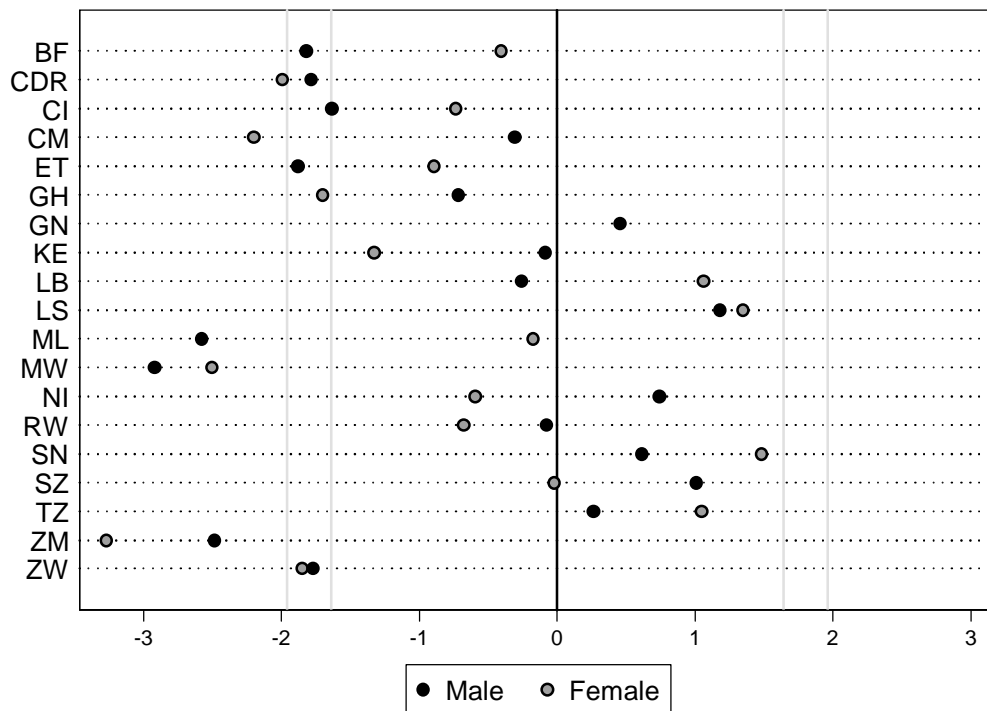
Notes:
See Figure 2 for the list of countries and their abbreviations. z-scores are based on country and sex specific negative binomial regression models with all (significant) controls from the adjusted model in Table 1. z-scores ≥ 1.64 or ≤ -1.64 are significant at the 10%-level, z-scores ≥ 1.96 or ≤ -1.96 are significant at the 5%-level. Negative s-scores are indicative of a negative effect of polygyny. For Lesotho, the % in a polygynous union is entirely based on responses for men (the question was not asked to women).

Figure 4: Simulated sexual network structure (cross-section) under monogamy (A), gender symmetric concurrency (B), and gender asymmetric concurrency (i.e., polygyny, C).

<div align="center">A.   B.   C.</div>
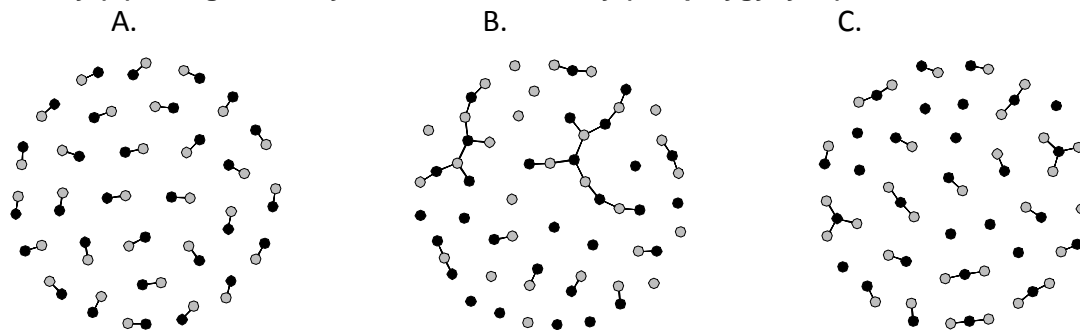
Notes:

Each network consists of 30 men (●), 30 women (◎) and 30 –given the constraints on partnership concurrency– randomly assigned heterosexual partnerships (◎–●). Graphs generated with the networksis package in R [48].

References:

1. Halperin DT, Epstein H. Concurrent sexual partnerships help to explain Africa's high HIV prevalence: implications for prevention. *Lancet* 2004,364:4-6.

2. Shelton JD. Ten myths and one truth about generalised HIV epidemics. *Lancet* 2007,370:1809-1811.

3. Epstein H. The invisible cure. New York: Farrar, Straus and Giroux; 2007.

4. Watts CH, May RM. The influence of concurrent partnerships on the dynamics of HIV/AIDS. *Math Biosci* 1992,108:89-104.

5. Morris M, Kretzschmar M. Concurrent partnerships and the spread of HIV. *AIDS* 1997,11:641-648.

6. Wawer MJ, Gray RH, Sewankambo NK, Serwadda D, Li X, Laeyendecker O*, et al.* Rates of HIV-1 transmission per coital act, by stage of HIV-1 infection, in Rakai, Uganda. *The Journal of Infectious Diseases* 2005,191:1403-1409.

7. Wilson DP, Law MG, Grulich AE, Cooper DA, Kaldor JM. Relation between HIV viral load and infectiousness: a model-based analysis. *Lancet* 2008,372:314-320.

8. Mah TL, Halperin DT. Concurrent sexual partnerships and the HIV epidemics in Africa: Evidence to move forward. *AIDS Behav* 2008, (epub ahead of print).

9. Carael M. Sexual behaviour. In: *Sexual behaviour and AIDS in the developing world. Findings from a multisite study*. Edited by Cleland J, Ferry B. London: Taylor and Francis; 1995:75-123.

10. Lurie MN, Rosenthal S. Concurrent partnerships as the driver of the HIV epidemic in sub-Saharan Africa? The evidence is limited. *AIDS Behav* 2009,forthcoming.

11. UNAIDS. Consultation on concurrent sexual partnerships. Recommendations from a meeting of the UNAIDS Reference Group on Estimates, Modeling and Projections held in Nairobi, Kena, April 20-21. In. Geneva: UNAIDS Reference Group on Estimates, Modeling and Projections 2009.

12. Mishra V, Bignami-Van Assche S. Concurrent sexual partnerships and HIV infection: evidence from national population-based surveys. Calverton, MD: Macro International Inc; 2009.

13. Lagarde E, Auvert B, Carael M, Laourou M, Ferry B, Akam E, *et al.* Concurrent sexual partnerships and HIV prevalence in five urban communities of Sub-Saharan Africa. *AIDS* 2001,15:877-884.

14. Kelly RJ. Determinants of HIV transmission in rural Uganda: Circumcision, concurrency and age differences in sexual partners [PhD Thesis]. Baltimore: Johns Hopkins University; 2001.

15. Helleringer S, Kohler HP, Kalilani-Phiri L. The association of HIV serodiscordance and partnership concurrency in Likoma Island (Malawi). *AIDS* 2009,23:1285-1287.

16. Timæus IM, Reynar A. Polygynists and their wives in sub-Saharan Africa: An analysis of five Demographic and Health Surveys. *Population Studies* 1998,52:145-162.

17. Sandoy IF, Dzekedzeke K, Fylkesnes K. Prevalence and correlates of concurrent sexual partnerships in Zambia. *AIDS Behav* 2008, (epub ahead of print).

18. Macro International. Sampling manual. In: *DHS-III basic documentation*. Calverton, MD: Macro International Inc.; 1996.

19. Hilbe J. Negative binomial regression. New York: Cambridge University Press; 2007.

20. Carael M. Urban-rural differentials in HIV / STDs and sexual behaviour. In: *Sexual cultures and migration in the era of AIDS: anthropological and demographic perspectives*. Edited by Herdt G. Oxford: Oxford University Press; 1997.

21. Moses S, Bradley JE, Nagelkerke NJ, Ronald AR, Ndinya-Achola JO, Plummer FA. Geographical patterns of male circumcision practices in Africa: association with HIV seroprevalence. *Int J Epidemiol* 1990,19:693-697.

22. Auvert B, Taljaard D, Lagarde E, Sobngwi-Tambekou J, Sitta R, Puren A. Randomized, controlled intervention trial of male circumcision for reduction of HIV infection risk: the ANRS 1265 Trial. *PLoS Med* 2005,2:e298.

23. Gray RH, Kigozi G, Serwadda D, Makumbi F, Watya S, Nalugoda F, *et al.* Male circumcision for HIV prevention in men in Rakai, Uganda: a randomised trial. *Lancet* 2007,369:657-666.

24. Bongaarts J. Late marriage and the HIV epidemic in sub-Saharan Africa. *Popul Stud* 2007,61:73-83.

25. Fleming DT, Wasserheit JN. From epidemiological synergy to public health policy and practice: the contribution of other sexually transmitted diseases to sexual transmission of HIV infection. *Sex Transm Infect* 1999,75:3-17.

26.   Over M, Piot P. HIV infection and sexually transmitted diseases: Population, Health and Nutrition Division, Population and Human Resources Department, World Bank; 1991.

27.   Gray PB. HIV and Islam: is HIV prevalence lower among Muslims? *Soc Sci Med* 2004,58:1751-1756.

28.   Decosas J, Kane F, Anarfi JK, Sodji KD, Wagner HU. Migration and AIDS. *Lancet* 1995,346:826-828.

29.   Van de Walle E. Marriage in African censuses and inquiries. In: *The demography of tropical Africa*. Edited by Brass W, Coale A, Demeney P*, et al.* Princeton: Princeton University Press; 1968:183-238.

30.   Trinitapoli J, Regnerus MD. Religion and HIV risk behaviors among married men: Initial results from a study in rural sub-Saharan Africa. *Journal for the Scientific Study of Religion* 2006,45:505-528.

31.   Reniers G, Tfaily R. Polygyny and HIV in Malawi. *Demographic Res* 2008,19:1811-1830.

32.   Mitsunaga TM, Powell AM, Heard NJ, Larsen UM. Extramarital sex among Nigerian men: polygyny and other risk factors. 2005,39:478-488.

33.   Morris M, Kretzschmar M. Concurrent partnerships and transmission dynamics in networks. *Soc Networks* 1995,17:299-318.

34.   Morris M, Kretzschmar M. A microsimulation study of the effect of concurrent partnerships on the spread of HIV in Uganda. *Mathematical Population Studies* 2000,8:109-133.

35.   Reniers G, Tfaily R. Polygyny and the spread of HIV in sub-Saharan Africa. In: *PAA Annual Conference*. New Orleans, LA; 2008.

36.   Goldman N, Pebley A, Lesthaeghe RJ. The demography of polygyny in Sub-Saharan Africa. In: *Reproduction and social organization in Sub-Saharan Africa*. Edited by Lesthaeghe R. Berkeley: University of California Press; 1989:212-237.

37.   Pison G. La démographie de la polygamie. *Population* 1986,41:93-122.

38.   Reniers G. Marital strategies for regulating exposure to HIV. *Demography* 2008,45: 417-438.

39.   Lopman BA, Nyamukapa C, Hallett TB, Mushati P, Spark-du Preez N, Kurwa F*, et al.* Role of widows in the heterosexual transmission of HIV in Manicaland, Zimbabwe, 1998-2003. *Sex Transm Infect* 2009,85 Suppl 1:i41-48.

40. Quigley M, Munguti K, Grosskurth H, Todd J, Mosha F, Senkoro K, *et al.* Sexual behaviour patterns and other risk factors for HIV infection in rural Tanzania: a case-control study. *AIDS* 1997,11:237-248.

41. Blower SM, Boe C. Sex acts, sex partners, and sex budgets: implications for risk factor analysis and estimation of HIV transmission probabilities. *J Acquir Immune Defic Syndr* 1993,6:1347-1352.

42. Josephson SC. Does polygyny reduce fertility? *Am J Hum Biol* 2002,14:222-232.

43. Musham HV. Fertility of polygamous marriages. *Popul Stud* 1956,10:3-16.

44. Barrett JC. Fecundability and coital frequency. *Popul Stud* 1971,25:309-313.

45. Pebley AR, Mbugua W. Polygyny and fertility in sub-Saharan Africa. In: *Reproduction and social organization in sub-Saharan Africa*. Edited by Lesthaeghe R. Berkeley: University of California Press; 1989:338-364.

46. Palmore E. Cross-cultural perspectives on widowhood. *J Cross Cult Gerontol* 1987,2:93-105.

47. UNAIDS. Report on the global AIDS epidemic. Geneva: UNAIDS; 2008.

48. Admiraal R, Handcock MS. networksis: a package to simulate bipartite graphs with fixed marginals through sequential importance sampling. *JSS* 2008,24.