Community HIV Prevalence and Parity Progression

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Short Abstract: The emergent literature on how HIV prevalence in the community affects fertility includes a wide array of mixed results, but is starting to favor the null hypothesis of no general effect on fertility. However, an overall zero effect can conceal radically different effects for women of different parities. More specifically, high HIV prevalence could delay first births by delaying the initiation of condomless sex, but it could speed the transition from the first birth to desired family size. Women who have already been exposed to risk of acquiring HIV sexually might be motivated to complete childbearing quickly future reproductive capacity is most uncertain. I estimate effects of community HIV prevalence on parity progression in 20 sub-Saharan African countries in random effects models and 15 countries in fixed effects models.

The prevailing belief that the HIV/AIDS epidemic will accelerate fertility decline in sub-Saharan Africa derives largely from estimates based on the lower fertility of infected women without considering community-level effects of HIV prevalence. However, the total fertility impact of HIV depends more on how the general population responds than on fertility differentials by individual HIV serostatus (Fortson 2009; Juhn, Kalemli-Ozcan and Turan 2008; Young 2007). The emergent literature on how HIV prevalence in the community affects fertility includes a wide array of mixed results, but is starting to favor the null hypothesis of no general effect on fertility. However, an overall zero effect can conceal radically different effects for women of different parities. More specifically, high HIV prevalence could delay first births by delaying the initiation of condomless sex (National Research Council and National Institute of Medicine 2005; Young 2005), but it could speed the transition from the first birth to desired family size. Women who have already initiated childbearing have also been exposed to risk of acquiring HIV sexually. Such women might be motivated to complete childbearing quickly where HIV prevalence is high and thus future reproductive health is uncertain (Rutenberg, Biddlecom and Kaona 2000). Women who know they are infected but who still desire to have children may also adopt a faster tempo of childbearing (Allen et al. 1993; Keogh et al. 1994).

Data and methods

Available surveys. Since 2003, the DHS has included HIV test results at the individual level for nationally representative samples in 20 sub-Saharan African countries.[1] There is wide variation in HIV prevalence at the national level in these data (see the second column of table 1 below). All 20 countries can be included in random effects models estimating the effect of community HIV prevalence on fertility. The random effects estimates adjust for observations being correlated within communities.

[1] 18 in standard DHS surveys plus the AIDS Indicator Surveys in Tanzania (2003 & 2007).

Table 1: Countries with nationally representative HIV data from DHS

Country and year	HIV prevalence at DHS survey	Additional HIV data collection	WFS?	Oldest DHS	HIV prevalence in 1990†
Western Africa					
Benin 2006*	1.2%	Not yet	Yes	1996	No data
Burkina Faso 2003	1.9%	Not released	No	1992/93	1.8%
Côte d'Ivoire 2005	4.7%	Not released	Yes	1994	2.0%
Ghana 2003	2.2%	No	Yes	1988	0.0%
Guinea 2005	1.5%	No	No	1992	0.2%
Liberia 2007	1.6%	No	No	1986	0.0%
Mali 2006	1.3%	2001*	No	1987	0.0%
Niger 2006	0.7%	No	No	1992	0.1%
Senegal 2005	0.7%	No	Yes	1986	0.0%
Middle Africa					
Cameroon 2004	5.3%	Not released	Yes	1991	0.9%
Congo Democratic Republic 2007	1.4%	No	No	2007	No estimate
Eastern Africa					
Ethiopia 2005	1.4%	No	No	2000	0.7%
Kenya 2003	6.7%	Not released	Yes	1988/89	No estimate
Malawi 2004	11.8%	Not released	No	1992	2.3%
Rwanda 2005	3.0%	No	Yes	1992	9.2%
Tanzania 2004	7.0%	2007	No	1992	4.8%
Zambia 2007	14.3%	2001/02*	No	1992	9.0%
Zimbabwe 2005/06	18.1%	No	No	1988	14.5%
Southern Africa					
Lesotho 2004	23.1%	No	Yes	2004	0.4%
Swaziland 2006/07	25.9%	No	No	2006/07	0.5%

† Source: http://apps.who.int/globalatlas/predefinedReports

*HIV test results cannot be linked to individual interview data in data sets before 2003, nor is the HIV data available at the individual level from Benin 2006

It is also desirable to control for community fixed effects—aspects of communities related to fertility that do not change over time but that if omitted could bias the results. Estimates of community level effects are generally more subject to omitted variable bias than individual level effects because of both data limitations and smaller n at the higher level. Therefore, controlling for fixed effects is very important, but it requires having multiple observations per community. This imposes two important limitations: first, there must be more than one survey per country, and second, the same communities must be identifiable in both data sets. In almost all countries, the smallest "communities" available for analysis across time are provinces (or their equivalent; naming varies among countries; there are 3-14 such geopolitical areas per country).

At present, there are only three countries for which nationally representative HIV data is available for two time points, and for each of them there are other important data limitations. For Mali and Zambia, HIV data are not available at the individual level from the first data collection.

Thus I can still estimate the effect of community HIV prevalence on fertility, but a portion of this will be biological rather than behavioral (due to the subfecundity of infected women). For Tanzania, the 2003 AIDS Indicator Survey (AIS) did not include a birth history like the 2007 Tanzania AIS and standard DHS. Preliminary analysis reveals surprisingly consistent results from using a "last birth" approach as is necessary for Tanzania 2003 compared to a more rigorous one with greater data demands. Both approaches are detailed below; I previously implemented them using data from Kenya and Lesotho (DeRose 2009).

Additional countries can be included where data is available from long enough ago that assuming zero HIV prevalence is reasonable. Column 4 of table 1 shows the eight countries that had World Fertility Surveys (WFS) fielded between 1977 and 1983.[2] Fixed effects can be employed in an additional four countries by assuming zero HIV prevalence in early DHS data collections. This last step will be done with caution. Fortson (2009) assumed zero HIV prevalence before 1990, arguing a fertility response to HIV-related mortality risk was unlikely because of low HIV prevalence in the early 1980s coupled with survival times of about ten years in the absence of treatment. I found her argument questionable given the high rates of HIV prevalence in Eastern Africa in 1990 (see last column in table 1 above): it is highly possible that AIDS morbidity was already impacting community life. However, the availability of both a WFS and an early (1988/89) DHS in Kenya allowed for testing whether the choice of baseline mattered in fixed effects models. I ran regressions patterned after Fink and Linnemayr (2009), but for only Kenya (rather than their five country pooled sample). I then substituted DHSI data from Kenya for the WFS baseline. The assumption of zero HIV prevalence in 1978 seems unquestionable; still assuming zero HIV prevalence in 1988/89 yielded nearly identical results. This exercise lent credibility to Fortson's approach, but in Zimbabwe fixed effects models are not possible except using 1988 data, and HIV prevalence is estimated to have been 14.5% in 1990. Thus adding the three West African countries to the fixed effects analysis using early DHS data as the baseline seems unproblematic, but I will present overall results with and without Zimbabwe.

In sum, fixed effects models are possible in 15 of the 20 countries. Some of these require more data compromises than others. Nonetheless, there are 12 countries for which at least two of the time points have all the necessary data. The other three are all in East Africa: Tanzania where the 2003 survey did not have complete birth histories; Zambia where HIV testing cannot be linked to individual records in the 2001/02 survey and the 1992 survey might be too late to represent fertility unaltered by community HIV; and Zimbabwe where the 1988 surveys may be similarly too late.

Methods

I start with an approach that estimates the total effect of HIV prevalence, and then restrict the sample to measure parity-specific effects. My basic model is a multilevel discrete-time hazard model where observations are three-month intervals taken from the DHS birth histories. Women

[2] Fink and Linnemayr (2009) used five WFS countries. I add Rwanda which was not originally part of the WFS, but has the same core questionnaire. I am also able to add Benin by using community HIV from the 2006 survey taken from published reports (as well as anticipating the 2009 survey), and Lesotho by using the capital region (Maseru) and all other areas as communities—a compromise that is not as bad as it sounds because a) Lesotho's 10 districts are smaller than the provinces in other countries, b) HIV prevalence is high (over 17%) throughout the country, and c) extensive migration makes Lesotho's communities generally less distinct.

contributed a maximum of eight such intervals, as they were followed from two years before the survey unless they were younger than 15 at that time, in which case they were followed from age 15. Intervals commencing less than 10 months after the previous birth are also omitted. The individual-level outcome is given by:

$$\log(P_{ij}/(1-P_{ij}))=m_0+m_1G_{1j}+ \ldots +m_kG_{kj} + U_{0j}+ b_{1j}x_{i1j} + \ldots +b_{bj}x_{ibj} \quad (1)$$

where P_{ij} is the probability that a woman i in community j gives birth in the interval. The grand mean of the log odds for community *j* is represented by $m_0+m_1G_{1j}+ \ldots +m_kG_{kj} + U_{0j}$, with the intercept being allowed to vary between communities in the random effects models and with a set of dummy variables for community in the fixed effects models. Deviations from that grand mean according to individual characteristics (x_{ibj}'s) are given by the rest of the equation. The m_n's are coefficients on community characteristics (G_n's), and the community-level errors are represented by the U_{0j}'s. Individual sero-status is one of the x_{ibj}'s, and the community HIV prevalence rate is one of the m_nG_n's. The coefficients on cross-level interactions are shown as b_{nj}'s above, but they are really the sum of the effect associated with the particular x_{inj} and that variable interacted with the community-level HIV prevalence rate.

At the individual level, the woman's age is measured in five-year intervals. The number of years of completed education will be grouped 0-1, 2-4, 5-7, 8-10, and 11 or more years and represented by a vector of dummies. I also control parity at the start of the observation period, household wealth,[3] and Muslim religion (see (Westoff and Cross 2006). At the community level, I control for urban residence, average household wealth, and community education (measured as in Kravdal (2002)). The epidemic was initially most severe in areas that were more advanced socioeconomically, probably because of greater mobility (Ainsworth, Filmer and Semali 1998; Hargreaves and Glynn 2002). These communities would have lower fertility even in the absence of any effects from HIV. In the fixed effects analysis, I also follow Fink and Linnemayr (2009) in including an interaction between province and survey to control for heterogeneous paths of fertility decline irrespective of the epidemic. Community HIV prevalence will be included initially as a vector of dummy variables for each percent of the community infected, i.e., <1%, 1-2%, etc. Further representation of the percent infected will be determined by any thresholds discovered by this analysis.

The basic model estimates the total effect of HIV prevalence through all pathways. Adding a control for individual HIV status leaves the effect of HIV prevalence on the infected and the uninfected alike. In previous work, I included a cross-level interaction term between women's own HIV status and community HIV prevalence, but the effect of community HIV does not seem to vary with women's own status which is not surprising given that many do not know their serostatus and many seropostive women nonetheless wish to continue childbearing (Desgrées du Loû 2005; Yeatman 2007). Adding an additional control for province child mortality also helps remove supply-side effects from the coefficient on community HIV prevalence.

[3] I cannot control for household wealth when using WFS data. When relying solely on DHS data, I use Sarah Giroux's wealth index which is comparable across countries. I chose this over the DHS wealth index that measures relative wealth within countries.

To determine whether higher HIV prevalence motivates faster completion of childbearing, I will stratify the basic model by parity rather than simply controlling for parity. My focus will be on the second and third birth intervals because the majority of women in every country in my sample progress to at least the third birth. I expect that the fertility-stimulating effects of HIV prevalence will be strongest for women who have already commenced childbearing (and therefore already been exposed to the risk of acquiring HIV sexually), but have not yet completed it. Additionally, I will add a control for marriage and an interaction between individual marital status and community HIV prevalence: married women in sub-Saharan Africa find condomless sex virtually mandatory, so they are the subset that would be most motivated to complete childbearing early. Unmarried women face a tradeoff between increased risk and their childbearing goals.

If faster progression from parity one to parity three were simply a tempo effect, completed fertility need not be higher for women who time their births earlier (they may have lower ASFRs when they reach their late 30s than do contemporary women in their late 30s whose earlier childbearing was presumably less affected by the epidemic). Therefore, I also plan to estimate the effects of HIV prevalence on ideal family size. If women were seeking only to time childbearing earlier, ideal family size should be less affected by HIV prevalence than early parity progression is. Earlier childbearing could, of course, also result in faster population growth through later unintentional childbearing and shorter mean length of generation.

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