Fertility Differentials by Education and Community HIV Prevalence

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Short Abstract: Both individual and community education can be expected to condition the impact of HIV prevalence on fertility. Existing literature seems to suggest that the epidemic will only solidify the negative relationships between socioeconomic status and fertility that we have all grown accustomed to. However, when HIV-related mortality reduces the returns to schooling by truncating the time horizons for such returns (and introducing uncertainty about them), educated individuals who would otherwise have educated their children are the most likely to experience changes in the costs and benefits of children. Fertility differentials at the individual level could therefore alter radically. Whether better-educated communities respond differential to HIV prevalence also receives empirical investigation here.

Fink and Linnemayr (2009) introduced the notion that the fertility response to community HIV prevalence could vary by education. They showed that educated women in communities with high HIV prevalence curtailed their fertility significantly more than uneducated women. However, their main fixed effects models were based on observing communities at two points assuming zero HIV prevalence in the World Fertility Surveys from 1978-1982. This was before the onset of fertility decline in all five countries in their sample, at a point where a few years of education was associated with significantly higher fertility (because of its affects on traditional postpartum practices), and more education had no significant effect on fertility. Therefore, when they interacted HIV prevalence with education, the interaction term was also capturing change in the meaning of education over time: between the WFS and DHS data collections in the 21st century that included HIV status, a statistically significant negative relationship between education and fertility emerged. When I included change in the meaning of education over time in their models, the more educated then had a significantly *positive* response to community HIV prevalence. Thus the HIV/AIDS epidemic has the potential to reduce, eliminate, or reverse the well-known inverse relationship between education and fertility, particularly among the uninfected subpopulation.

Literature Review. HIV positive women have lower fertility for both biological and behavioral reasons, plus they are much more likely to die during their reproductive years. These differentials by HIV serostatus were the basis of early empirical work concluding that the epidemic would help reduce fertility (e.g., (Camlin, Garenne and Moultrie 2004; Hunter et al. 2003; Lewis et al. 2004; Terceira et al. 2003; Zambuko and Mturi 2005), even though multiple ways the HIV epidemic could alter fertility behavior among the uninfected were recognized (Cleland, Ali and Capo-Chichi 1999; Desgrées du Loû 2005; Grieser et al. 2001; Ntozi 2001; Rutenberg, Biddlecom and Kaona 2000; United Nations Population Division 2002; Young 2005).[1]

[1] Fertility enhancing pathways identified in this literature include proving healthiness by producing healthy children (most relevant where HIV testing is rare), shortening postpartum abstinence to protect monogamy, avoiding or shortening breastfeeding because of fear of mother-to-baby transmission, uncertainty about future HIV status motivating faster early childbearing, and fear of child loss motivating hoarding strategies. Fertility suppressing pathways include avoiding childbearing because of fear of mother-to-baby transmission, concern for the welfare of

More recent literature recognizes that the total fertility impact of HIV depends more on how the general population responds to the presence of the epidemic than on fertility differentials by serostatus (Fortson 2009; Juhn, Kalemli-Ozcan and Turan 2008; Young 2007). While the emergent literature on the effects of community HIV prevalence on fertility is inconclusive, it generally indicates that the epidemic will not accelerate fertility decline in sub-Saharan Africa. In fact, the only published work documenting a negative effect of HIV prevalence on fertility is that of Alywn Young on South Africa (Young 2005) and also 27 sub-Saharan countries with generalized epidemics (Young 2007). His work has been forcefully and persuasively criticized by Sebnum Kalemli-Ozcan who has shown that his results depend on residual errors: for South Africa, the effect of community HIV actually turn positive and significant when the time period is restricted to avoid a discrete jump in the HIV data that leads to a mechanical downward trend in the residuals (Kalemli-Ozcan 2009). Similarly, clustering standard errors by country eliminates statistical significance from his multi-country study because positively serially correlated residuals led to underestimated standard errors (Kalemli-Ozcan 2006[2]).

Several studies have supported the null hypothesis that fertility does not vary significantly with community HIV prevalence (Ahuja et al. 2009; Fortson 2009; Juhn et al. 2008; Magadi and Agwanda 2007). Others have found results that vary by age and parity (Sneeringer and Logan 2009a), specification (Kalemli-Ozcan 2006), and education or wealth (Fink and Linnemayr 2009). The single most important shortcoming of the existing literature is a failure to translate Fink and Linnemayr's insight regarding the potential for differential fertility responses by socioeconomic status from the micro level to the macro level. There is substantial reason to believe that both community education levels and community education trajectories condition the fertility response to community HIV prevalence.

First, with respect to community education *levels*, regions with higher education levels had lower fertility prior to the HIV/AIDS epidemic. The well-known inverse relationship between education and fertility pertains at both the individual and the community level—better educated communities have even lower fertility than would be predicted based on fertility differentials by education together with educational composition of the community (Kravdal 2002). In sub-Saharan Africa where fertility decline commenced late—South Africa being the only sizable country[3] where substantial decline at the national level occurred before 1980 (United Nations 2009)—this means that, in general, more educated communities are farther along in their fertility transitions. Importantly, the least educated communities are likely to be ones in countries where fertility has declined less than 10% from its peak, i.e., in countries that have not entered the fertility transition according to the Princeton rule. Even where the onset of the fertility transition has officially occurred, communities experiencing the early stages of fertility decline are ones where older cohorts representing older fertility norms are alive and possibly influential. In short, it may be easier to return to high fertility where it is a norm of the recent past rather than the distant past. However, empirical analysis of the HIV/fertility relationship has not to date

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existing children, increased obligations to take in orphaned children of extended family members, and increased value of women's time due to HIV-induced labor scarcity.

[2] The May 2009 version of Kalemli-Ozcan's 2006 NBER Working Paper includes the re-analsysis of Young's 2007 data.

[3] The others were Djibouthi, Mauritius, Mayotte, and Reunion.

considered how long fertility has been declining.

Of further relevance with respect to community education levels, there is a strong correlation between education and other resources: better endowed regions may be better able to absorb the costs of HIV morbidity and mortality. Alternatively stated, economic development may provide more options for a multiphasic response to mortality *increase*. Recall that Kingsley Davis (Davis 1963) postulated that a fertility response to mortality decline would be delayed while people maintained fertility norms while coping with demographic pressure by other means like migration. In a similar way, regions with relatively advanced fertility transitions might try to preserve norms like investing in a small number of children while pre-transition or early transition regions might not resist the shift back to quality from quantity as strongly. Moreover, more developed regions might be able to afford to continue to invest in quality during the epidemic while poorer regions might lack that as an option. Richer regions may also simply have less mortality increase for a given HIV prevalence because of better health care, especially increasingly available antiretrovirals. And finally, the chance that the supply of contraceptive services will be compromised by the demands that AIDS morbidity puts on health care systems is greater in the poorer regions.

Second, with respect to community education *trajectories*, much of the existing literature controls out one of the possible pathways through which HIV can affect fertility, namely reducing investments in education. Work that controls for education in order to establish the effect of HIV on fertility independently of education (Fink and Linnemayr 2009; Juhn et al. 2008; Magadi and Agwanda 2007; Sneeringer and Logan 2009a; Young 2005, 2007) misestimates the total effect of HIV on fertility because poor educational trajectories—i.e., slower expansion of education and even declining enrollments—are at least in part caused by the epidemic (Ahuja et al. 2009; DeRose 2005; Ferreira et al. 2003; Fortson 2008). Fortson's (2008) analysis substantiates that although there are multiple pathways that could produce a negative relationship between HIV prevalence and education, the most likely explanation is higher mortality risk decreasing the utility of investment in education.[4] Kalemli-Ozcan (2006) has an excellent discussion of why higher *adult* mortality in particular would stimulate fertility, drawing heavily on (Meltzer 1992) and (Soares 2005): when parents expect to be alive when their children finish schooling, they gain more by sacrificing to provide schooling than when their own longevity is uncertain; moreover, the returns to schooling are on average lower for the children themselves when the time horizons for returns are truncated by HIV mortality.

Conceptual Framework and Hypotheses. The conceptual framework guiding this investigation is simply that fertility results from factors influencing the supply of children, the demand for children, and the costs of fertility regulation. For each of these three factors, HIV prevalence can theoretically have both positive and negative effects as reviewed briefly in this section. The analysis will not conclusively sort out the pathways through which HIV prevalence influences fertility—the key hypotheses are about how changes in fertility *demand* would be expressed in fertility patterns.

[4] Most importantly, she shows moves beyond a large previous literature on orphan's schooling to show effects in the general population that depend on HIV prevalence rates.

HIV/AIDS decreases the supply of children among the infected by reducing individual fecundity, truncating reproductive careers, and increasing child mortality. Moreover, child survivorship among uninfected children can be compromised by fatalism brought on by high disease prevalence and diluted resources for medical care. Less and later sexual intercourse occurs in areas of higher HIV risk (Mensch, Grant and Blanc 2006; Westoff and Cross 2006). On the other hand, shorter abstinence (Cleland et al. 1999; DeRose 2007; Desgrées du Loû and Brou 2005) and breastfeeding (Ntozi 2001) increase the supply of children. Adult mortality also increases the supply of children per surviving adult.

There has been a great deal of concern that the costs of fertility regulation would increase with HIV/AIDS because both governments and donors would have less funds available for family planning because of the burdens imposed by the epidemic (Cleland and Sinding 2005; Ng'weshemi et al. 1997). However, family planning programs have been shown to be better preserved than other public health efforts (Richey 2004). Further, where contraceptive use has declined as in Kenya, little could be attributed to difficulties obtaining supplies (Westoff and Cross 2006). Efforts to combat the epidemic also increase the supply of free condoms.

Finally, the effects of *adult* mortality will influence fertility demand. (Child mortality influences fertility supply.) Labor scarcity due to adult mortality can increase returns to work and thus the opportunity costs of childrearing, leading to decreased demand for children (Young 2005). Fink and Linnemayr (2009) further argue that parents would choose more consumption and fewer children in the face of HIV because returns to childbearing decrease considerably when parents lose adult children. Despite these pathways through which the epidemic can reduce fertility demand, parents faced with decreased returns to childbearing might also invest less in them and thus have more of them. The shift from quality to quantity would also reduce the burden of taking in foster children, and less education means lower opportunity costs of childrearing in future generations.

Hypotheses:

1) the effect of HIV prevalence on fertility will be more negative/less positive in countries where fertility decline commenced in the 1980s, i.e., before the epidemic was widespread. Retaining low fertility norms is more likely where they are better established, and the effects of HIV/AIDS on supply and costs are less likely to vary with stage of the fertility transition.

2) HIV/AIDS reduces incentives to invest in child quality, thus stimulating fertility. The estimated effect of HIV prevalence will be more negative/less positive when current enrollments are controlled, as these gauge parental investment. Further, educated parents will be most likely to respond to reduced incentives to invest in education because they are those who would have been most likely to educate their children in the absence of the epidemic.

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.[5] 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.

[5] 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.[6] 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

[6] 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, 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,[7] 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.

[7] 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.

Effects of stage of fertility decline. Six of the twenty countries—including at least one from Western, Eastern, and Southern Africa—entered the fertility transition according to the Princeton rule in the period from 1985-90. I will estimate the effect of HIV prevalence in this subsample of countries and test whether it differs from the effect in the other fifteen countries. This will determine whether the fertility response to HIV prevalence depends upon whether the transition was underway prior to the epidemic becoming widespread.

Effects on fertility through educational trajectories. I will add current school enrollments (from the DHS household files) to the basic model. The community education control is based on the attainment of adults, but fertility decisions also depend on the extent to which the current generation of children is being educated (Caldwell 1980). While many sub-Saharan countries show evidence of education decline in at least some of their provinces, Malawi shows no such decline: it stands in contrast to Tanzania and Zimbabwe as a high HIV prevalence country without this other negative trend. Among low prevalence countries, there has been little education decline in Burkina Faso, Ghana, and Senegal, but much in Mali and Niger (DeRose and Kravdal 2007). There is also substantial variation between countries in recent efforts to promote education as mandated by Millennium Development Goals (e.g., Kenya's dropping of primary school fees in 2003). Controlling for both children's enrollments and adult attainment answers the question of whether children's enrollment matters independently (a difficult question historically because of colinearity when educational progress was monotonic). More importantly, comparing the estimated effects of HIV prevalence with and without this control will indicate whether there is an indirect effect of HIV prevalence on fertility via children's enrollments, i.e., whether the epidemic stimulates fertility by encouraging quantity rather than quality.

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