# The Effect of HIV/AIDS on Fertility via Children's School Enrollments

**Laurie F. DeRose**

**Maryland Population Research Center**
**University of Maryland, College Park**

Abstract: Much of the existing literature controls out one of the possible pathways through which HIV can affect fertility, namely reducing investments in education. Returns to schooling have become highly uncertain because of increased mortality in the prime productive years. Community HIV prevalence is a strong determinant of uncertainty in returns to schooling. Work that controls for education in order to establish the effect of HIV on fertility independently of education 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. I estimate the effect of community HIV prevalence on fertility *via its effect on school enrollments* employing multilevel random effects models using Demographic and Health Survey data from 20 sub-Saharan African countries.

How HIV prevalence affects fertility behavior is still unknown, and yet this is a crucial question for the future of world population growth. A large share of future growth will occur in sub-Saharan Africa even if fertility continues to decline, but declines seem to have stalled in several countries. The region has the highest HIV prevalence rates and the highest fertility rates in the world. The effect of the HIV/AIDS crisis on fertility will condition the timing and pace of fertility decline in the region most consequential for world population size in the 21st century.

Further, the HIV/AIDS crisis in sub-Saharan Africa holds more potential than any other historical mortality change to inform what we know about the mortality/fertility relationship. Adult mortality has increased everywhere that HIV is prevalent, but increases in child mortality have been more variable. In the past, mortality improvements at various ages were correlated. We therefore do not know whether parents had fewer children when they expected more of them to survive or when they expected them to survive long enough to make investing in education worthwhile. It used to be that children who attended school generally survived long enough to reap returns from schooling and to support their parents in old age. With the introduction of HIV/AIDS, returns to schooling have become highly uncertain because of increased mortality in the prime productive years.

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 is more dependent on fertility responses among the general population than on the fertility effect of positive serostatus (Fortson 2009; Juhn, Kalemli-Ozcan and Turan 2008; Young 2007). The newly emerging consensus is that the epidemic does *not* in fact alter fertility in the general population. However, my previous work identifies important differentials in the fertility response to the epidemic that indicate schooling may be a key omitted variable biasing our understanding of the HIV/fertility relationship.

First, the estimated effect of HIV prevalence among women over 20 is significantly negative. Insignificant findings obtain when all age groups are analyzed together. Younger women are those for whom continuing schooling and initiating childbearing are generally mutually exclusive. Thus if the epidemic reduces incentives to invest in schooling, then an otherwise negative effect of HIV prevalence would be offset among the youngest women. Evidence that HIV prevalence has a negative impact on schooling is strong, and Jane Fortson's forthcoming work establishes that this is an effect on the general population, not confined to orphans and caregivers (Fortson Forthcoming).

Second, how different socioeconomic groups respond to HIV prevalence also points in the direction of HIV-induced reductions in enrollments mattering for fertility decisions. Those with no education are the most likely to reduce their fertility in the face of HIV/AIDS, while those with more than four years of schooling do not (DeRose 2010). Because those who have been educated themselves would otherwise be most likely to educate their children, it is possible this pattern is the result of a shift back from quality to quantity strategies among those who would have chosen quality in the absence of the epidemic.

The goal of the current paper is to estimate the effect of community HIV prevalence on fertility *via its effect on school enrollments*. I hypothesize that the epidemic would contribute to fertility decline more if it did not stimulate fertility by reducing school enrollments. This research increases our understanding of how HIV/AIDS affects fertility, but it is additionally important because it addresses questions regarding HIV/AIDS and economic development. Alwyn Young's work (Young 2005, 2007) generated a great deal of controversy because he argued

that HIV-induced fertility limitation would contribute to economic development on the continent. His opponents have undermined his conclusion by arguing that HIV/AIDS does not in fact cause lower fertility in the general population. My work will likely substantiate a fertility-reducing effect of living in communities with high HIV prevalence, but without supporting hopes for subsequent economic development: if HIV/AIDS interferes with the intergenerational transmission of education, it reduces the human capital stock of future generations. Further, the narrowing of fertility differentials by education in the face of HIV/AIDS can also mean that even if educational expansion were achieved, it might not reduce fertility as much as it has historically: fertility declines could easily stall in an environment where returns to education are compromised.

## Background

The proposed research contributes to understanding the mortality/fertility relationship, informs what we know about the progress of transition from high to low fertility, and disentangles the effects of children's and adults' education on fertility. The progress of sub-Saharan Africa's fertility transition will depend on the fertility response to increased mortality. Other crises have been shown to speed fertility decline (Guzmán 1994; Mari Bhat 1998; Martine 1996; Rutenberg and Diamond 1993), but these have not involved extensive deterioration of human capital as is present in contemporary sub-Saharan Africa with declining educational attainments and increased mortality.

The notion that fertility would continue to decline if it dropped by at least 10% was derived from observing prosperity-led fertility transitions on the European continent (Coale and Watkins 1986). Transitions that were accelerated by economic crisis were underway before the crises hit (Guzmán 1994; Mari Bhat 1998; Martine 1996; Rutenberg and Diamond 1993). In sub-Saharan Africa, 17% of the population live in the eight countries that have yet to experience the onset of fertility transition and an additional 17% live in the six countries that entered the fertility transition only in the 21st century.[1] The pace of fertility decline is also highly variable across the remaining two-thirds of the region. This paper takes into account that the impact of the HIV/AIDS mortality crisis depends upon whether it occurred before or after substantial fertility decline. Uneducated individuals have been shown to have lower fertility in communities with higher HIV prevealence only where fertility transition was well underway prior to 1980 when the epidemic began to spread (DeRose 2010). Thus, fertility-suppressing effects of the epidemic may only pertain in a subset of afflicted countries.

In addition to providing the possibility for further unraveling of the mortality/fertility relationship, the HIV/AIDS epidemic also breaks the long-standing correlation between adult education and children's education. Fertility transition has generally taken place in communities where education was becoming increasingly common. With children therefore becoming more expensive, disentangling this effect from direct effects of the parents' own (higher) education was difficult. The epidemic has retarded and even reversed educational progress in parts of sub-Saharan Africa (Ahuja, Wendell and Werker 2009; DeRose 2005; Ferreira, Santos and Pessoa 2003; Fortson 2008), and this unfortunate natural experiment allows us to better assess whether the enrollment rates of children matter separately from the attainment of adults.

Moreover, 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

[1] Peak TFR from United Nations (2009); onset of fertility transition defined as when TFR falls 10% below its peak. Current population size and TFRs from Population Reference Bureau (2009).

Linnemayr 2009; Juhn et al. 2008; Magadi and Agwanda 2007; Sneeringer and Logan 2009; 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 (forthcoming) argues that among the multiple pathways that could produce the negative relationship between HIV prevalence and education, the most likely is higher mortality risk decreasing the utility of investment in education.[2] 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 potentially truncated by HIV mortality. Community HIV prevalence is a strong determinant of uncertainty in returns to education.

The fertility response to the epidemic then includes indirect effects through schooling trajectories. The importance of this pathway can be tested by comparing estimates of the effect of community HIV prevalence with and without controls for children's schooling.

## 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.[3] There is wide variation in HIV prevalence at the national level in these data (see the second column of table 1 below). Sub-national variation is even greater. The number of communities available for analysis, 3-14 provinces (or their equivalent) per country, allows for estimating effects of community HIV prevalence on fertility.

[2] Most importantly, she moves beyond a large previous literature on orphan's schooling to show effects in the general population that depend on HIV prevalence rates.
[3] 19 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 |
|---|---|
| **Western Africa** | |
| Burkina Faso 2003 | 1.9% |
| Côte d'Ivoire 2005 | 4.7% |
| Ghana 2003 | 2.2% |
| Guinea 2005 | 1.5% |
| Liberia 2007 | 1.6% |
| Mali 2006 | 1.3% |
| Niger 2006 | 0.7% |
| Senegal 2005 | 0.7% |
| Sierra Leone 2008 | 1.5% |
| **Middle Africa** | |
| Cameroon 2004 | 5.3% |
| Congo Democratic Republic 2007 | 1.4% |
| **Eastern Africa** | |
| Ethiopia 2005 | 1.4% |
| Kenya 2003 | 6.7% |
| Malawi 2004 | 11.8% |
| Rwanda 2005 | 3.0% |
| Tanzania 2007 | 7.0% |
| Zambia 2007 | 14.3% |
| Zimbabwe 2005/06 | 18.1% |
| **Southern Africa** | |
| Lesotho 2004 | 23.1% |
| Swaziland 2006/07 | 25.9% |

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

## Methods

I employ a multilevel discrete-time hazard model where the observations are three-month intervals from the three years before the survey. Women contributed a maximum of twelve such intervals, but may have contributed fewer as those commencing when they were younger than 14.25 or already pregnant are omitted. The individual-level outcome in the random effects models is given by:

$$\log(P_{ij}/(1-P_{ij}) = bx_{ij} + e_{ij} + mG_j + U_j + year \quad (1)$$

where:

$P_{ij}$ is the probability that a woman i in community j conceives in the interval
$b$ is the vector of coefficients on the individual characteristics $(x_{ij}'s)$
$e_{ij}$ represents individual-level errors
$m$ is the vector of coefficients on the community characteristics $(G_j's)$
$U_j$ is the community random term

The community random term $U_j$ allows the intercept to vary between communities, thus testing whether cross-sectional variation in HIV prevalence predicts cross-sectional variation in fertility outcomes while correcting for correlation between observations from the same community. Communities are defined as provinces, and there are 185 provinces across the 20 countries.

At the individual level, the woman's age in years and an age squared term is also included to capture the non-linear relationship between age and fecundity. The number of years of completed education is be grouped 0, 1-3, 4-6, 7-11, and 12 or more years and represented by a vector of dummies. I also control parity at the start of the observation period, duration of exposure (since age 14.25 or the most recent birth), household wealth,[4] Muslim religion (see (Westoff and Cross 2006), and urban residence.

At the community level, I control for average household wealth and community education (see below). 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.

The basic model estimates the total effect of HIV prevalence on recent fertility through all pathways. With a control for individual HIV status, the coefficient on HIV prevalence represents the effect on the general population—both infected and uninfected—and should be less negative/more positive, as it no longer includes the effects of individual subfecundity among seropositive women. 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 seropositive women nonetheless wish to continue childbearing (Desgrées du Loû 2005; Yeatman 2007). Adding an additional control for province early childhood (under 3) mortality also helps remove supply-side effects from the coefficient on community HIV prevalence.

I include cross-level interaction terms between individual education and community HIV prevalence (Fink and Linnemayr 2009) because educated parents would be more likely to respond to reduced incentives to invest in education . I also create a dummy variable distinguishing the countries where peak fertility occurred in 1970-75 or before from those where sustained fertility decline began later (see Appendix A) because my work has shown that fertility effects of the HIV/AIDS epidemic are not consistent across these settings.

Finally, I include a community-level variable created from the DHS household files that measures children's education. Because only about half of the countries have data on whether children are currently in school, I follow Fortson (2008) in constructing a variable for the proportion of school age children who are not more than two years behind correct grade for age. In models where adult education is also controlled, this variable does not measure overall community education, but rather *current* community education while controlling for *past* community education. I again follow Fortson in using only pre-1980 birth cohorts when calculating adult education: these are the cohorts whose own education would likely have been completed before the epidemic caused widespread increases in adult mortality. For example, someone born in 1978 would have entered school around 1985: while they could have still be enrolled in the 1990s when AIDS mortality became quite noticeable in many countries, any effects would be modest compared to those in later cohorts who would not have completed primary education before pronounced mortality increase. Given the timing of the Demographic and Health Surveys with nationally representative HIV data, pre-1980 birth cohorts will be

[4] I choose Sarah Giroux's wealth index, which is comparable across countries, over the DHS wealth index that measures relative wealth within countries. See Appendix B.

approximately 25 and older at interview (with some variation between countries based on the date of survey).

Using these two quite different community education variables, I can assess whether children's education impacts fertility net of adult attainments. Of even greater importance in the context of the current research, any *change* in the coefficient on community HIV prevalence when adding a control for children's education gauges how much of the fertility response to the epidemic operates through current school enrollments.

I also plan to test whether the lack of fertility response among women under 20 to HIV prevalence in their communities can be explained by lower enrollments. *Ceteris paribus*, we should see a stronger negative relationship in this age group than among older women because living in communities with high HIV prevalence can provide incentive to delay the initiation of condomless sex. Instead, there is no effect of HIV prevalence in this age group, and that might be explained by lower opportunity costs to childbearing among women who are not in school.

## Expected findings

I expect to find evidence of a shift back from quality to quantity strategies among educated respondents in countries with mature fertility transitions. This response is far less likely 1) among individuals who would not have invested much in their children's education anyway, and 2) in countries where fertility is still relatively high. I also expect to uncover a negative effect of HIV prevalence on fertility among the youngest women when controlling for their own enrollments. This would not mean that HIV prevalence had a net negative impact on the initiation of childbearing, but it would explain why it does not.

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