Higher Community HIV Prevalence Predicts Higher Recent Marital Fertility in Kenya

Laurie F. DeRose
University of Maryland

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I estimate the effect of community prevalence of HIV on recent martial fertility in Kenya. The general consensus in the existing literature on the relationship between HIV and fertility is that the relationship is negative, and even that the HIV/AIDS epidemic has contributed to fertility decline in severely affected countries (see Terceira et al. 2003; Zambuko and Mturi 2005). The negative individual-level relationship between HIV and fertility is fairly unambiguous: what few proximate determinants would contribute to higher fertility (like earlier intercourse) are outweighed by a set of others that produce lower fertility among the infected (higher rates of miscarriage, lower coital frequency because of illness, widowhood, divorce, lower fecundity). Some of the best studies have concluded that the overall fertility of HIV positive women is 25-40% lower than among the uninfected (Allen et al. 1993; Ryder et al. 1991; Zaba and Gregson 1998). However, the community-level relationship between HIV and fertility is also believed to be negative, but with far less evidence.

Two main factors have constrained previous work on the community-level impact of the HIV/AIDS epidemic on fertility. First, community-level effects cannot be measured when sero-prevalence data is available only for small geographic areas. In order to estimate the effects of different HIV prevalence levels, there must be variation between communities in the infection rates. The Demographic and Health Surveys (DHS) now include HIV testing, providing the first estimates of HIV prevalence for nationally representative samples of reproductive-age adults. Second, the contribution of HIV to fertility declines has been calculated using the assumption that fertility in the absence of the epidemic would have been similar to fertility among non-infected women (see Lewis et al. 2004; Terceira et al. 2003; Zambuko and Mturi 2005). This method therefore measures the fertility-suppressing effect associated with own infection while assuming that community HIV prevalence rates have no effect on the fertility behavior of non-infected women. Clearly, assuming a zero effect is not the same as demonstrating one.

The literature on how the HIV epidemic affects fertility via behavioral mechanisms (rather than simply biological ones) acknowledges a variety of pathways through which the epidemic could motivate *higher* fertility. These include proving healthiness through the production of healthy children (particularly important where most do not know their sero-status, as is generally the case in sub-Saharan Africa), succeeding in reproduction before becoming infected, replacing dead children, and greater fear of child loss because of high community mortality rates. Nevertheless, a careful review of this literature concluded that these positive effects on fertility desires were not normative (United Nations Population Division 2002): concerns about the effect of pregnancy on maternal health, fear of vertical transmission, concern for the welfare of existing children, and increased obligations to take in children of extended family members could all curtail fertility desires. Therefore, there is ample reason to believe that the epidemic would not, on balance, motivate higher fertility among the uninfected. Nonetheless, empirical work

on this question is limited (United Nations Population Division 2002) and Kenya has experienced recent fertility increase that is not well understood (Westoff and Cross 2005).

Data and Methods

The data are from the 2003 Kenya Demographic and Health Survey (DHS). Kenya is currently the only high-prevalence country where HIV test results can be linked to individual DHS survey data. Earlier data from Zambia showed that the *variation* in fertility was higher in communities with higher HIV prevalence (DeRose 2005)—a finding consistent with higher fertility among the uninfected and lower fertility among the infected—but such a relationship could not be confirmed when the individual-level relationship between HIV and fertility could not be estimated.

Using the Kenyan data, I employ multi-level linear regression model[1] to estimate the determinants of recent fertility (births in the three years prior to survey). I model recent fertility as a function of the woman's own sero-status as well as the HIV prevalence in the woman's community.

I limited the analytic sample to married women aged at least 18 at interview to insure women were at appreciable risk of birth during the three-year observation period. By analyzing the impact of HIV on marital fertility, I am not capturing the total impact of the epidemic: clearly, the proportion of women married can depend on HIV mortality rates among both women and men; divorce can also result from infection or fear of infection. Differences in levels of these and other proximate determinants by infection status are not taken into account as would be ideal (see discussion in Lewis et al. 2004). Nevertheless, much of the literature assumes that the fertility behavior of uninfected women is not affected by the epidemic, and estimating the effects on marital fertility tests this assumption for a very large subset of the reproductive-age population.

Community Characteristics

There were 400 sampling clusters in the 2003 Kenya DHS with an average of 21 women per cluster, but only an average of 11 per cluster were tested for HIV. I stabilized the cluster estimates of percent infected using data from 15 regions based on province and residence (treating rural and urban areas of the seven of the eight provinces separately since HIV prevalence rates are markedly higher in urban areas; Nairobi had only urban areas). The relative weight given to the province/residence estimates versus the cluster estimates was determined by the distance between the number of observations in the cluster and 50. Kravdal (2004) gives evidence supporting the appropriateness of basing community estimates on the small samples available for DHS clusters, but I nonetheless used only the province/residence estimates where the number of observations in a cluster was below 5.

The community level control variables are rural residence and province.

[1] Very similar results were obtained using a logistic regression model with whether or not there had been a birth in the observation period as the outcome variable.

Individual Variables

I control for the age of the woman in single years. The woman's education is categorized as none (ref), primary, and secondary and higher. Other individual variables a wealth index (a linear asset index weighted by principle components), parity at the start of the observation period (three years ago), a dummy variable for child deaths in the observation period, and a dummy variable for Muslim religion (see Westoff and Cross 2005).

Dependent Variable

I analyzed the determinants of births during the three years prior to interview. Although some of the women testing HIV positive at interview may not have been infected throughout the period for which fertility is measured, this factor should bias downward the coefficient for own infection while having little impact on the community-level effects of infection rates.

Results

The variation in HIV infection rates between communities in Kenya is fairly large. The percentage HIV positive ranged from 0-26% in the 400 sampling clusters and from 0-24% in the 15 province residence areas. None of the blood samples from the women in Northeast Province tested positive for HIV in either rural (n=133) or urban (n=58) areas. In urban Nyanza, 24% tested HIV positive, and in Nairobi 11%.

Being infected with HIV is associated with lower recent fertility (0.168 fewer births). The mean number of births in the past three years was 0.431, meaning that HIV infection is associated with 39% lower fertility; this is quite consistent with previous literature on individual-level effects. However, living in an area where HIV prevalence is a percentage point higher is associated with recent fertility that is 0.019 births higher. When these individual and community level effects are considered jointly, the model predicts that fertility in urban Nyanza was elevated by 0.42 births because of the epidemic (0.019*24=0.456 and the fertility-suppressing effect of own infection pertains to only 0.24 of the sample –0.168*0.24=-0.040). In Nairobi, the epidemic was estimated to contribute 0.19 additional births per woman over the past three years. I provide these estimates for illustrative purposes only, as effects among married women do not measure the total impact of the epidemic. Nonetheless, it is clear that a fertility-enhancing behavioral effect associated with living in areas of high HIV prevalence can outweigh the fertility-suppressing effects of own infection.

Table 1: Population averaged models with robust standard errors

Outcome	Births in last 3 years
Individual-level characteristics	
HIV positive	-0.168**
Age	-0.034***
Education	-0.056
Primary	-0.039
Secondary and higher	-0.099
Wealth index	-0.050
Parity at beginning of observation period	0.011
Child death in observation period	0.245***
Muslim	0.175*
Community-level characteristics	
Intercept	1.311***
HIV prevalence	0.019*
Rural residence	0.132*
Province (ref=Nairobi)	
Central	-0.081
Coast	-0.047
Eastern	0.016
Nyanza	-0.163
Rift Valley	0.030
Western	0.067
North Eastern	0.004

* significant at p≤.05; ** significant at p≤.01; *** significant at p≤.0.001

Conclusions and Plans for Further Estimation

The TFR increased in Kenya between 1998 and 2003. Westoff and Cross's (2005) analysis provided evidence that this reflected an increase in wanted fertility rather than supply-side problems, but the factors motivating higher fertility have remained unclear. This analysis indicates that married women in Kenya may have had higher recent fertility in response to the HIV levels in their communities (despite the fact that infected women have markedly lower fertility). In further analysis, I plan to investigate whether this is a quantum effect or a tempo effect. If HIV creates an environment where reproductive careers feel more uncertain because of own mortality risk, married women may speed up childbearing without aiming for higher completed fertility. This hypothesis will be tested with more detailed analysis of the effects at different parity levels. The higher levels of childhood mortality in communities with a more severe HIV/AIDS epidemic could also motivate higher fertility if risk were generalized by prospective parents, but the utility of insurance strategies is, of course, compromised by vertical transmission. Nonetheless, I will test for insurance effects by 1) including childhood mortality (levels and change) as a community-level variable and 2) testing whether childbearing beyond reported ideal family size is a function of HIV prevalence rates.

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