Direct correspondence to:
Laurie F. DeRose
2112 Art-Sociology Building
University of Maryland
College Park, MD 20742-1315
Phone: (301) 424-2252
Lderose@umd.edu

HIV prevalence, investment in education, and fertility trajectories

LAURIE F. DEROSE

University of Maryland, College Park

Maryland Population Research Center

This work was funded, in part, with a Seed Grant from the Maryland Population Research Center, with support from the National Institutes of Health and Human Development Grant number R24 HD41041-04.

Abstract

The future of fertility transition in sub-Saharan Africa depends in part on the fertility response to HIV/AIDS. The prevailing wisdom is that, if anything, the epidemic will speed the transition. However, fertility decline has slowed and even reversed in some countries on the continent. Moreover, literature supporting a negative fertility effect of HIV/AIDS assumes lower fertility among the infected and unaltered fertility among others. In contrast, my previous work using Kenyan data showed that with individual sero-status controlled, higher community HIV prevalence rates predicted higher fertility. Data from Lesotho and Tanzania are used in the current paper to test whether the fertility-enhancing effect of community HIV rates pertain in countries that have not experienced recent fertility increase (and whether fertility decline is slowed by less investment in education). I also test the validity of findings from random effects models by making assumptions that allow for inclusion of fixed effects.

The future of fertility transition in sub-Saharan Africa depends in part on the fertility response to the HIV/AIDS epidemic. The prevailing wisdom is that, if anything, the epidemic will speed the transition. However, fertility decline has reversed in Kenya where HIV levels are high by world standards (moderate by African standards), and other African transitions have slowed or stalled (Bongaarts 2006). Moreover, most previous estimates of the impact of the HIV/AIDS epidemic on fertility have been calculated under the assumption that the fertility among uninfected women represents the levels of the entire population in the absence of the epidemic (see Terceira et al. 2003; Zambuko and Mturi 2005). This method assumes that community HIV prevalence rates have no effect on the fertility behavior of uninfected women. I first tested this assumption using Demographic and Health Survey (DHS) data from Kenya using multilevel models that included both individual sero-status and community HIV prevalence as predictors of recent fertility (DeRose 2006). I found that Kenyan women aged 20-34 who lived in communities with higher HIV prevalence rates had *higher* recent fertility. Here I propose to further explore the effect of community HIV prevalence on fertility and whether it helps explain recent fertility trajectories in sub-Saharan Africa. First, I will test whether the results from Kenya obtain using DHS data from two additional high prevalence countries: Lesotho and Tanzania. Second, I will estimate models that include community fixed effects for Kenya and Tanzania. For both sets of models, I devote attention to possible pathways through which the epidemic could affect fertility. Of particular importance for stalled fertility transitions more generally, I include changes in education. In some communities in sub-Saharan Africa, young women entering their reproductive years have fewer years of education than their predecessors. This negative trend has emerged in response to economic hardship, but may also be fueled by the HIV/AIDS epidemic as investing in education makes less sense when the time horizon for returns to education is less certain.

Background

The fertility-suppressing effects of HIV infection at the individual level have been well documented in previous literature, with a number of possible biological as well as behavioral mechanisms identified (Carpenter et al. 1997; Gray et al. 1998; Hunter et al. 2003; Terceira et al. 2003). Social scientists have also suggested a variety of pathways through which the epidemic could motivate higher fertility among the *uninfected*. Communities with high rates of HIV infection motivate individual women to: (1) prove their "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; see Grieser et al. 2001); (2) time childbearing early so as to fulfill fertility desires before becoming infected (Rutenberg et al. 2000); and (3) increase fertility because of greater fear of child loss in a community with high mortality rates (United Nations Population Division 2002; see also Sandberg (2005) whose work suggests higher *variance* in mortality could impede fertility limitation). Countering these "fertility-enhancing" pathways are factors that might depress fertility in high HIV prevalence communities. Higher fertility desires might be outweighed by commonly cited concerns regarding the effect of pregnancy on maternal health, fear of mother-to-child transmission, concern for the welfare of existing children, and increased obligations to take in children of extended family members (Grieser et al. 2001; Rutenberg et al. 2000; United Nations Population Division 2002). Fertility might also be lowered where scarcity of labor (caused by HIV mortality) increases the value of women's time and therefore the opportunity costs of childbearing (Young 2005).

Although it seems to be commonly believed that on balance the HIV/AIDS epidemic has contributed to fertility limitation, the effects of community HIV prevalence rates on the behavior of the uninfected could not be tested until data with sero-status from a wide range of communities became available. As indicated above, the Kenyan data revealed that higher prevalence of HIV in the community corresponded to higher fertility among women aged 20-34. In contrast, both younger and older women

had significantly lower fertility where HIV was higher (DeRose 2006). This pattern seems consistent with a desire to complete childbearing while still healthy. The youngest women do not accelerate their childbearing, probably because attempting to do so would simultaneously increase risk of infection. But as women enter their twenties and have almost all entered sexual unions—most marital unions—reducing chances of infection by attempting to avoid unprotected sex becomes unfeasible. It is also likely undesirable in Kenya where the average woman still bears five children. If a twenty-year old woman plans on three-year birth intervals and average fertility, she is looking at a decade and a half of childbearing. Somewhere like urban Nyanza province where 21% of reproductive-age women are HIV positive, an uninfected 20-year-old may lack confidence that she will reach her early thirties still uninfected and still able to bear uninfected children. In contrast in the rural areas of Coast province where only 5% of her peers are infected, she may not be as uncertain about her future health status. After women have born their desired number of children, there is no longer a need to increase childbearing in response to the epidemic. HIV-infected newborns in sub-Saharan Africa usually die early in their second year of life, so a woman can be reasonably sure she has produced uninfected offspring in fairly short order. High childhood mortality rates in her community would be unlikely to affect her childbearing because a primary source of mortality risk can be ruled out early on (the effects of childhood mortality rates will nonetheless be tested).

An independent literature on the effects of HIV on schooling has also developed. This work points toward temporary schooling disruptions associated with caregiving during illness (Burke and Beegle 2004; Sundar 2005; Yamano and Jayne 2003), and also highlights the possibility of longer term effects because of the need for children's incomes when parents are no longer able to earn (Ainsworth et al. 2005; Bicego et al. 2003; Mishra et al. 2004). Declines in school enrollments can also occur as a result of economic hardship, either as a result of lower adult productivity in the wake of the epidemic or independently of HIV/AIDS. Economic crisis has been implicated in declining primary and secondary enrollments in many areas throughout the sub-Saharan Africa. Men's schooling attainments have declined on average across the region (National Research Council and National Institute of Medicine 2005), and education decline for women has occurred in about a quarter of communities (DeRose and Kravdal 2007). Education decline has emerged in countries with very low HIV prevalence rates like Niger and Madagascar, but it is also pronounced in higher HIV countries like Kenya, Tanzania, Zambia, and Zimbabwe. In Zambia, education reversals have been more extreme in areas with higher HIV prevalence (DeRose 2005).

The importance for fertility of the effect of HIV on schooling is twofold. First, less educated women are more likely to engage in childbearing early both because they are not enrolled in school and because educational attainment suppresses fertility (see, e.g, Blossfeld and Huinink 1991). Second, investments in schooling are less likely where the time horizon for returns to these investments is shorter on average (see Ainsworth et al. 2005; Soares 2005; Strauss and Thomas 1995); less schooling reduces childrearing costs and could promote fertility.

The literatures on the effects on HIV on fertility and the effects of HIV on schooling are not yet connected, despite the important feedback effects between fertility and schooling. When HIV-related education decline is conceptualized only as a response to hardship in the families of HIV-infected individuals, the potential fertility-stimulating effect of shorter average earnings horizons in areas with generalized epidemics is overlooked. But it is highly plausible that reproductive strategies could shift more toward "quantity" in a context where investments in "quality" are more risky. The problem in the literature on the effect of HIV on schooling is parallel to the problem in the literature on the effect of HIV on fertility: children with non-infected parents are used as the reference group, and therefore ways

that the epidemic may affect the whole community are missed. The literature on the effects of HIV on fertility also suffers by not considering the fertility effects of education decline outlined above.

Data

In order to estimate the effects of HIV prevalence in the community, there must be variation between communities in the infection rates. Such estimates were not possible when sero-prevalence data were available for only small geographic areas. Appropriate data for estimating fertility effects of the epidemic have recently become available as the Demographic and Health Surveys (DHS) now include HIV test results at the individual level for nationally representative samples in Burkina Faso, Cameroon, Ghana, Kenya, Lesotho, and Tanzania. Until data from Lesotho and Tanzania were released, only Kenya had sufficient sub-national variation in community infection rates to analyze effects of community HIV prevalence on fertility. Now similar analysis can be performed in two additional relatively high HIV countries. Although I intend to incorporate low prevalence countries into future analysis, the focus of the current paper is to establish whether the fertility-stimulating effect of high HIV pertains in Lesotho and Tanzania as it does in Kenya.

The sampling clusters in DHS data correspond to Standard Enumeration Areas from population censuses. Within randomly selected clusters, the demographic composition of each household is ascertained by a household questionnaire, and interviews are attempted with all women aged 15-49. The women's questionnaire includes, among other things, various socioeconomic indicators and a complete birth history. In half of households, questionnaires are also administered to all men aged 15-59, and all reproductive age men and women in these same households were eligible for HIV testing. In Kenya, there were 3964 women and 3581 men present in households when HIV blood samples were collected. Eighty-four percent of both men and women consented to the blood testing. Across 400 sample clusters, this provided an average of 16 test results per cluster. Similar sample size is anticipated at the cluster level for Lesotho and Tanzania. The analysis will be repeated at the district level where sample size is more adequate where possible (at least in Kenya).

Methods

The current investigation does not seek to estimate the total impact of the HIV/AIDS epidemic. Instead, it focuses primarily on the hypothesis that communities with higher HIV prevalence have higher recent fertility.

It is important to note that the total fertility rate (TFR) is a synthetic cohort measure of how many children a woman would have if she lived her entire reproductive career under the current age-specific fertility rates. As such, it estimates the average number of children for women who complete their reproductive years. In fact, HIV-related mortality limits the number of women who will complete their reproductive years. Thus, it is quite possible for a higher TFR to co-exist with smaller young generations by surviving women having more children and those with reproductive careers truncated by death having fewer.

Random-Effects Models

I first estimate random-effects multilevel models that include a woman's own sero-status at the individual level and the proportion that are HIV positive in the cluster, averaged from the individual data. Kravdal (2004) gives evidence supporting the appropriateness of basing community estimates on the small samples available for DHS clusters, but because HIV testing was not done in all households (see data description above), I stabilized the cluster rates using rates from rural and urban areas of

provinces (following Pande 2003 who borrows strength from larger geographic areas; see also DeRose 2005. 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. Where the cluster had more than 50 observations, I simply used the cluster estimate; where less than five, only the province/residence estimate.) I include data from Kenya, Lesotho, and Tanzania with a set of dummy variables for country. I also run separate models by country.

The dependent variable in my analyses is recent fertility (births in the three years prior to the survey). Since HIV status is known only at interview, projecting it very far into the past would be problematic. I estimate discrete-time hazard models where observations are three-month intervals taken from the DHS birth histories. Women contributed a maximum of twelve such intervals each, as they were followed from three years before the survey unless they were younger than 15 at that time (when risk of birth is quite low), in which case they were followed from age 15. If a woman had given birth less than 10 months before the start of an interval, she was not considered at risk during that interval.

At the individual level, I control for the age of the woman as a continuous variable. The number of years of completed education is grouped 0-1, 2-4, 5-7, 8-10, and 11 or more years and represented by a vector of dummies. Other individual variables include a principle components weighted household wealth index (based on consumer durables and housing quality), parity at the start of the observation period, and a dummy variable for Muslim religion (see Westoff and Cross 2005 for religious variation in Kenya's recent fertility increase).

Estimates of the effects of community-level characteristics are even more likely than individual-level estimates to suffer from omitted variable bias. In part that is because smaller sample size at the community level precludes implementing a large number of controls. Here I will control for a set of characteristics that could bias the effects of HIV prevalence: urban residence, community wealth (averaged from the household wealth index), and community education (measured among 25-34 year-olds to avoid being affected by HIV-related education decline). The epidemic was initially most severe in areas that were more advanced socioeconomically, probably because of greater mobility (Ainsworth and Semali 1998; Hargreaves and Glynn 2002). These communities would have lower fertility even in the absence of any effects from HIV. I include dummy variables for province to control for fixed effects in larger geographic units. I will also include cross-level interaction terms between the woman's own HIV status and the community HIV prevalence rate to test the hypothesis that community prevalence affects women differently according to their own sero-status. It is, however, possible that *behavioral* mechanisms are very similar between infected women (many of whom do not know their status at the time of observation) and uninfected women.

The individual-level outcome is given by

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

where P_{ij} is the probability that a woman i in region j has a child within the observation interval of three months. The grand mean of the log odds for cluster *j* is represented by $m_0+m_1G_{1j}+ \ldots +m_kG_{kj} + U_{0j}$, and 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 cluster-level errors are represented by the U_{0j}'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.

Possible pathways for a positive fertility response to HIV prevalence among uninfected women

Earlier fertility. If higher recent marital fertility in areas with high HIV prevalence reflects a desire to complete childbearing early because later childbearing might be risky or impossible if the woman contracted AIDS, then the fertility response will depend on age. Younger women in high HIV prevalence communities will show higher birth rates while older women will be less affected. The age-specific effects of community HIV prevalence are tested using cross-level interaction terms between the individual woman's age group and the community HIV prevalence rate. This pathway is given the most attention here because the findings from Kenya indicated strong age-specific effects (see description above of DeRose 2006).

Childhood mortality. My childhood mortality measure is the proportion of deaths among births in the last three years in the cluster. As above, these cluster-level estimates are stabilized using a weighted average with larger geographic regions (the rural or urban portion of the province in which the cluster is located). Childhood mortality is introduced to previously estimated models to see if the effect of community HIV prevalence is explained in part by its influence on childhood mortality.

Childhood mortality is endogenous to fertility. This is more problematic at the individual level than at the community level, but still needs consideration here. I choose to measure current childhood mortality rates rather than recent change in those rates (even though some of the theory points to the importance of mortality change) because recent change could be particularly determined by the fertility behavior among the infected (e.g., lower childhood mortality where HIV positive women become aware of their status and refrain from childbearing).

Education decline. Similarly, I introduce the trend in community education to previously estimated models. Education trend is defined as the difference between the proportion of 15-24 year-olds who have completed four years of education minus the proportion of 25-34 year-olds with the same attainment. Primary schooling often extends beyond age 15 in sub-Saharan Africa because of late enrollments and grade repetition, but almost all who will complete four years of education have done so by age 15 (National Research Council and National Institute of Medicine 2005). If education decline in high HIV areas reflects only difficulties families burdened by illness and death face in keeping their children in school, then community education trend should be unrelated to fertility among the uninfected. If, however, the relationship between community HIV rates and fertility of the uninfected is partly explained by education decline, then there is support for the hypothesis that investments in children and opportunity costs to early and higher fertility change when adult longevity is uncertain.

Incorporation of fixed-effects

The results of the random effects models described above may be biased by community-specific features that influence both fertility and the spread of HIV. For example, Nyanza Province has the highest HIV prevalence rates in Kenya, but it was also experiencing a slower fertility transition than the rest of the country even before the HIV epidemic was advanced enough to have a plausible fertility impact. More generally, where fertility is highly valued protective behavior may be more difficult to implement. Ideally, nationally representative data HIV data would be available for more than one point in time so that there would be sufficient sample size to include a set of community variables in the model and still have enough statistical power remaining to estimate the effects of other community characteristics (like HIV prevalence). Such data will not be available anytime soon, and the fertility

effects of the epidemic are important to understand now as the future of fertility transitions on the continent depends in part on the fertility response to the epidemic.

As an alternative to having true longitudinal HIV data, I propose to treat the oldest available DHS data as representative of a no AIDS scenario. While this is not precisely correct by any means—the first DHS in Kenya was in 1989, and in Tanzania, 1992—the epidemic had not progressed much in those early years. Therefore, by pooling data from the earliest and most recent DHS in Kenya and Tanzania (Lesotho has had only one DHS) and assuming zero prevalence in the early data, I am able to include community fixed effects while retaining enough degrees of freedom to also include the same community level variables as in my random effects models. This will serve as an important test of the robustness of my earlier findings for Kenya as well as any emergent findings from the newly available data.

Because the sampling clusters have changed over time, the fixed effect models will need to use data aggregated at a higher level. The random effects models will first be re-estimated with rural and urban areas of provinces as the "communities." Community level control variables other than HIV prevalence may need to be dropped because of the loss of sample size when moving to an analysis with fewer communities. Community fixed effects will then be introduced. In the way, the sensitivity of the estimated effects of community HIV prevalence can be assessed separately from other differences between the random effects and fixed effects models.

Bibliography

Ainsworth, M., K. Beegle, and G. Koda. 2005. "The Impact of Adult Mortality and Parental Deaths on Primary Schooling in North-Western Tanzania." *The Journal of Development Studies* 41: 412-439.

Ainsworth, M., and I. Semali. 1998. "Who is Most Likely to Die of AIDS? Socioeconomic Correlates of Adult Deaths in Kagera Region, Tanzania." in *Confronting AIDS: Evidence in the Developing World*, edited by M. Ainsworth, L. Fransen, and M. Over. Brussels: European Union.

Bicego, G., S. Rutstein, and K. Johnson. 2003. "Dimensions of the Emerging Orphan Crisis in Sub-Saharan Africa." *Social Science & Medicine* 56: 1235-1247.

Blossfeld, H.-P., and J. Huinink. 1991. "Human Capital Investments or Norms of Role Transition? How Women's Schooling and Career Affect the Process of Family Formation." *American Journal of Sociology* 97: 143-168.

Burke, K., and K. Beegle. 2004. "Why Children Aren't Attending School: The Case of Northwestern Tanzania." *Journal of African Economies* 13: 333-355.

Carpenter, L. M., J. S. Nakiyingi, A. Ruberantwari, S. S. Malamba, A. Kamali, and J. A. G. Whitworth. 1997. "Estimates of the Impact of HIV Infection on Fertility in a Rural Ugandan Population Cohort." *Health Transition Review* Volume 7: 113-126.

DeRose, L. F. 2005. "Bifurcated Fertility in Areas with Higher HIV prevalence? Evidence from the 2001-2002 Zambia Demographic and Health Survey." Paper presented at the XXV International Population Conference of the IUSSP. Tours, France.

DeRose, L. F. 2006. "Higher Community HIV Prevalence Predicts Higher Recent Marital Fertility in Kenya." Paper presented at the Population Association of America Annual Meeting in Los Angeles, March.

DeRose, L. F., and Ø. Kravdal. 2007. "Educational Reversals and First Birth Timing in Sub-Saharan Africa: A Dynamic Multilevel Approach." *Demography* 44: 59-77.

Gray, R. H., M. J. Wawer, D. Serwadda, N. Sewankambo, C. Li, F. Wabwire-Mangen, L. Paxton, N. Kiwanuka, G. Kigozi, J. Konde-Lule, T. Quinn, and C. Gaydos. 1998. "Population-Based Study of Fertility in Women with HIV-1 Infection in Uganda,." *The Lancet* 351: 98-103.

Grieser, M., J. Gittelsohn, A. V. Shankar, T. Koppenhaver, T. K. LeGrand, and R. Marindo. 2001. "Reproductive Decision Making and the HIV/AIDS Epidemic in Zimbabwe." *Journal of Southern African Studies, 27(2), 225-243.* 27: 225-243.

Hargreaves, J. R., and J. R. Glynn. 2002. "Educational Attainment and HIV-1 Infection in Developing Countries: A Systematic Review." *Tropical Medicine and International Health* 7: 489-498.

Hunter, S.-C., R. Isingo, J. T. Boerma, M. Urassa, G. M. Mwaluko, and B. Zaba. 2003. "The Association Between HIV and Fertility in a Cohort Study in Rural Tanzania." *Journal of Biosocial Science* 35: 189-99.

Kravdal, Ø. 2004. "Child Mortality in India: The Community-Level Effect of Education." *Population Studies* 58: 177-192.

Mishra, V., F. Arnold, F. Otieno, A. Cross, and R. Hong. 2004. "Education and Nutritional Status of Orphans and Children of HIV-Infected Parents in Kenya." DHS Working Papers No. 24. Calverton, MD: Macro International and USAID.

National Research Council and National Institute of Medicine. 2005. *Growing Up Global: The Changing Transitions to Adulthood in Developing Countries*. Washington, DC: The National Academies Press.

Pande, R. P. 2003. "Selective Gender Differences in Childhood Nutrition and Immunization in Rural India: The Role of Siblings." *Demography* 40: 395-418.

Rutenberg, N., A. E. Biddlecom, and F. A. D. Kaona. 2000. "Reproductive Decision-Making in the Context of HIV and AIDS: A Qualitative Study in Ndola, Zambia." *International Family Planning Perspectives* 26: 124-130.

Sandberg, J. 2005. "Network Mortality and Nonnumeric Response Concerning Expected Family Size." *Demography* 42: 737-756.

Soares, R. R. 2005. "Mortality Reductions, Educational Attainments, and Fertility Choice." *American Economic Review* 95: 580-601.

Soares, R. R. forthcoming. "The Effects of Longevity on Schooling and Fertility: Evidence from the Brazilian Demographic and Health Survey." *Journal of Population Economics*.

Strauss, J., and D. Thomas. 1995. "Human Resources: Empirical Modeling of Household and Family Decisions." in *Handbook of Development Economics*, edited by J. Berhman and T. N. Srinivasan. Amsterdam: Elsevier.

Sundar, R. 2005. "The Household Impact of HIV/AIDS on the Education of Children—A Study of High HIV Prevalence States of India." Paper presented at the XXV International Population Conference of the IUSSP. Tours, France.

Terceira, N., S. Gregson, B. Zaba, and P. R. Mason. 2003. "The Contribution of HIV to Fertility Decline in Rural Zimbabwe, 1985-2000." *Population Studies* 57: 149-164.

United Nations Population Division. 2002. "HIV/AIDS and Fertility in Sub-Saharan Africa: A Review of the Research Literature." ESA/P/WP.174. New York: Department of Economic and Social Affairs.

Westoff, C. F., and A. R. Cross. 2005. "The Stall in the Fertility Transition in Kenya." Paper presented at the Population Association of America Annual Meeting in Philadelphia.

Yamano, T., and T. S. Jayne. 2003. "Working-age Adult Mortality and Primary Sschool Attendance in Rural Kenya.". Discussion Paper Series on International Development Strategies No. 2003-006. Foundation for Advanced Studies on International Development.

Young, A. 2005. "The Gift of Dying: The Tragedy of AIDS and the Welfare of Future African Generations." *Quarterly Journal of Economics* 120: 423-466.

Zambuko, O., and A. J. Mturi. 2005. “The Effect of the HIV/AIDS Epidemic on Fertility in South Africa.” Paper presented at the XXV International Population Conference of the IUSSP. Tours, France.