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Ruben Castro University of Pennsylvania, rcastro@sas.upenn.edu

Jere R. Behrman University of Pennsylvania, jbehrman@econ.upenn.edu

Hans-Peter Kohler University of Pennsylvania, Dept. of Soc, HPKOHLER@POP.UPENN.EDU

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Abstract

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Keywords

Africa, AIDS, Child health, Child quality, Child quantity, Children, Fertility, HIV, Malawi, Malawi Longitudinal Study of Families and Health, Maternal morbidity, Mortality, Mothers, Schooling

Disciplines

Demography, Population, and Ecology | Social and Behavioral Sciences | Sociology

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Perception of HIV risk and the quantity and quality of children:

The case of rural Malawi

Ruben Castro Jere R. Behrman Hans-Peter Kohler

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Abstract. The empirical literature on HIV and the quality (Q) and quantity (N) of children generally reports negative associations for Q and unclear associations for N. We focus our analysis on the effects of HIV, as a predictor of mother and child mortality, on investments in child Q and N. We develop a Q-N model within which higher *mothers*' mortality predicts lower N while higher *child* mortality predicts lower Q. Those effects together make reasonable the expectation of negative influences of higher HIV likelihood on child Q and N. Based on longitudinal micro data on mothers and their children in rural Malawi we find that variation in mothers' reported HIV risk reduces both child quality, as reflected in children's schooling and health, *and* child quantity, when the perceived risk is already moderate or high. The effects are sizable, and, in the case of Q (schooling and health) are found in children and teenagers, as well as boys and girls, while in the case of N are found for young and mature women.

Key words: HIV, child quantity, child quality, Malawi, fertility, schooling, child health.

Introduction

High HIV prevalence and infection risks in a number of Sub-Saharan African countries have created considerable concern about the consequences of the epidemic for economic growth and wellbeing. Many consequences of HIV for *infected* adults and children are fairly well established, ranging from schooling declines and increased malnutrition to changes in sexual behavior¹.

But HIV rates can also have consequences for the *non-infected* population. There may be macro level impacts of HIV through changes in health care and schooling provision, changes in the level of income-per-worker, increases in poverty and of course higher mortality². All those changes can also induce changes in individuals' behaviors, in particular if individuals update their expectations about the future based on perceptions of HIV risks³. Thus, though a majority of the population is HIV negative, they still might be affected by the HIV epidemic through their expectations about the future as well as other mechanisms.

Some of the possibly important effects of such changes in expectations may be on the quantity and quality of children, which is the topic of this paper. Conclusions of previous studies on this topic are somewhat mixed, especially for fertility. Kalemli-Ozcan (2006) analyzes regional HIV rates⁴ among Sub-Saharan African countries and concludes that higher fertility and lower child schooling

¹ Studies that report schooling declines include Evans and Miguel (2007), Yamano, Shimamura and Sserunkuuma (2006) and Case and Ardington (2005). Mishra, *et al.* (2007) analyzes malnutrition while Weinhardt *et al* (1999), and Oster (2007) study changes in sexual behavior.

² Some examples include Case and Paxson (2008) on health care, Papageorgiou and Stoytcheva (2005) and Young (2005) on income, and Whiteside (2002) on poverty.

³ The median time from seroconversion to death is estimated to be around 10 years, before antiretroviral therapy, and it has being recently informed by the UNAIDS that only 37% of people in sub-Saharan Africa eligible for treatment were able to access life-saving medicines in 2009".

⁴ He uses both incidence and, among pregnant women, prevalence rates.

attainment are associated with higher HIV rates. Although a pathway is not specified, the author emphasizes replacement behaviors in which mothers choose higher child quantity and lower child quality because HIV rates are linked to maternal expectations regarding child mortality.

Forston (2008, 2009) also uses aggregate data, but tries to establish causality from HIV to schooling and fertility. She finds that the schooling difference between pre- and post-1981 birth cohorts is negatively associated with current HIV prevalence rates. Thus, if the HIV rates are exogenous to the schooling decision, the inverse relation between HIV rates and schooling is confirmed. But, somewhat surprising, the relation seems to hold only in countries in which the HIV rate is relatively low, rather than relatively high. She finds, in contrast, that the difference between fertility for preand post-1990 birth cohorts is unrelated to HIV rates. Thus no child quantity effect is found. Durevall (2008), on the other hand, finds Malawian HIV rates are negatively associated with fertility rates, with the exception of young woman with no children, for whom the probability of a first birth increases.

These studies investigate overall relations between HIV and child quantity or quality. Such relations, however, can reflect a number of different pathways, so the results cannot be easily interpreted with regard to the mechanisms underlying the empirical associations. For example, child quality and quality might be affected by supply-side variables, such as teacher and health worker availability and school⁵ and health services funding⁶. They also might be affected by demand-side variables, such as changes in parents' investments in their children due to, for example, changes in expected mortality.

⁵ Kelly (2000) reports increased teacher mortality, though Bennell's (2005) results contrast with these findings. Grassly *et al* (2003) report impacts on schools funding.

⁶ Chen et al (2004) report impacts on health sector workers and Case (2008) on health services.

Young (2005) considers South Africa, a country with one of the highest HIV rates. He proposes a pathway from HIV to lower fertility through higher condom use and through higher wages (produced by the decrease in available workers); a long-run simulation leads to a smaller population with higher wages and higher human capital. Another pathway is proposed by Grant (2008), who finds that Malawian parents who learned their HIV status in 2004 subsequently were more likely to send their children to school, regardless of the HIV status. She interprets this result as reflecting the effect of lower uncertainty about HIV on investments in children.

In this paper we focus on perceived HIV risks as a predictor of higher mortality, an idea commonly mentioned in this field. We note that in a theoretical model such as in Becker and Lewis (1973) and Willis (1973), higher *child* mortality is ambiguously related to child quantity and quality. We add to this type of model the role of *mothers*' mortality, because higher HIV risk implies higher mortality for both mothers and their children. We propose that as mothers became more likely to die, children's quality is re-valued in terms of their wellbeing as orphans⁷. Using a simple two-period model, we illustrate that simultaneous rises in mothers' and child mortality are likely to produce a negative impact on child quantity and quality.

The empirical literature on links between HIV rates and child quality focuses on schooling. However, another important investment in children's human capital is in their health, which may have important long-run effects in poor populations (e.g., Hoddinott *et al.* 2008, Maluccio *et al.* 2009, Behrman *et al.* 2009). Therefore in this paper we include indicators of children's health as well as of their schooling in our empirical representation of their human capital.

⁷ De Lannoy (2005) presents related qualitative evidence.

Estimates of the impact of HIV risks on child outcomes in the previous literature may be hard to interpret because unobserved factors such as risk aversion and ability could affect both expectations regarding future health status and decisions related to the quantity and quality of children. To better control for such non-observed confounding factors, we use child-level longitudinal data, including mother-level measures of HIV risk. To our knowledge, this is the first use of this kind of longitudinal data to investigate the impact of mothers' perceptions of HIV risk on the quantity and quality of children. Our results indicate that mothers' self perceived risk of HIV is inversely linked to child quality *and* quantity.

The organization of the paper is as follows. Section 1 discusses the implications of children's and mothers' expected mortalities within a child quantity and quality model. Section 2 introduces the data. Section 3 presents child-level fixed-effects regressions of the impact of mothers' reported HIV risk on children's schooling and health and mother-level fixed-effects regressions of the impact on fertility. Section 4 uses mothers with completed fertility (40 or more years old) to estimate the two-period model introduced in section 1. The last section concludes.

Section 1: Mother and Children Mortality in the Q-N framework.

The general background for our analyses is provided by the "new home economics" (e.g, Becker and Lewis 1973 and Willis 1973) that postulates a trade-off between child quality and child quantity. Specifically, we assume in our model that survival is uncertain and that mothers maximize their twoperiod utility by deciding how much to consume in each period (C_1 , C_2) and how much to invest in child quality (*Q*) and quantity (*N*) during period one. The mothers' perceived probabilities of dying before reaching period two are H_m for themselves and H_i for their children. Mothers' total utility is a function of a) period 1's consumption, b) period 2's consumption and period 2's quantity and quality of children, in the scenario that the mother survives to period 2, and c) period 2's welfare of children, in the scenario that the mother does not survives to period 2. In all cases, it is assumed that the utility from children is higher if the children's probabilities of surviving are higher:

$$U(C_1) + (1-H_m)[U(C_2) + (1-H_c)W^s] + H_m(1-H_c)W^{ns}$$
, (1)

where W^s and W^{ns} are the utilities that mothers derive from their surviving children in the scenario of surviving and not surviving, respectively. Both W^s and W^{ns} are functions of Q, N, and the relative preference of Q versus N, noted by w^s and w^{ns} respectively. It is assumed that the overall expected utility from children is decreasing in H_m . To simplify the discussion, the total utility of mothers is restated as:

$$U(C_1) + f(H_m)[U(C_2) + (1-H_c)W],$$
 (2)

where $f(H_m)$ is a decreasing function of H_m , such that f(1)=k>0. Higher H_m or H_c makes it less likely that mothers will derive period two's own consumption utility or period two's children-associated utility, respectively⁸. Under different scenarios of mother and child mortality, however, the relative investments on Q and N can change: the function W depends on N, Q and w, the relative preference

⁸ In case the mother dies, there are C_2 resources available, for example, for children to consume. The model presented in this section, however, does not include children's consumption explicitly. But the inclusion of children's consumption would not change the mayor lessons learned in this exercise.

of Q versus N, which is itself a function of H_m and H_c . For example, mothers may favor N more relative to Q if they perceive H_{ℓ} to be higher.

Holding w constant, a rise in H_m decreases the total utility derived in period two and thus less resources are allocated to both C_2 and investments in children. Similarly, holding w constant a rise in H_{ι} decreases the utility from children, W, and thus fewer resources are allocated to investments in children. We call this negative influence of increases in H_m and H_i on investments in children the "investment effect".

In addition, a rise in H_m or H_c can change the relative preferences for Q over N through the function w. If w changes, the optimal distribution of total investments in children among Q and N changes. For simplicity, it is assumed that changes in w do not change the total investments in children⁹. A rise (drop) in w implies that more of the total resources invested in children are allocated to Q (N). We call the influence of changes in *w* the "*relative preference effect*".

For example, assume that $W = N^a Q^\beta$ and that the budget constraint is of the form $T = C_1 + C_2 + P_n N + C_2$ $P_{qQ} + P_{qr}NQ$, where T is total income, α/β is the form that the function implies for the preferences for Q relative to N, P_n and P_q are the fixed prices of N and Q and P_{qn} is the quality-dependent price of quantity. The first-order conditions with respect to C_{μ} , Q and N show that while the investments effect is negative¹⁰, the relative preference effect depends on the change in w. Period two's Q/N

⁹ Changes in the relative preferences over Q and N could change the total amount to be invested in children if the marginal utility of investments in children, W', changes. Nevertheless, since a change in w implies a rise in preferences for one factor and a drop for the other, the change in W' should be small.

¹⁰ The first-order conditions with respect to C_{l} , Q, N, respectively, are: (4')

U'-λ=0

 $f(H_m)[(1-H_c)W'(w_qNw_nQw_q-1)] - \lambda(P_q+P_{qn}N) = 0$ (5')

 $f(H_m)[(1-H_c)W'(w_nNw_n-1Qw_q)] - \lambda(P_n+P_{qn}Q) = 0$ (6')

ratio, shown in Equation 3, is positively associated with w. Therefore if w increases there is a positive relative preferences effect for Q relative to N, while the opposite is true if w decreases:

$$Q/N = w P_n / [P_{qn}N(1-w) + P_q]$$
 (3)

Figure 1 presents an example where a rise in H_c induces a decrease in w. The TI curves represent all the combinations of Q and N that are attainable at a given level of investment in children, while the Q/N curve, given by Equation 3, represents the optimal combinations of Q and N at a given level of w. Assume that the starting point is A, where the optimization of resources determines a certain level of investments in children that defines TI₀ that together with the original $w = w_0$ determines the optimal combination of Q and N. Higher H_c induces two changes. First, it induces an *investment effect*, which moves the equilibrium from A on TI₀ to B on TI₁ holding w constant at w_0 . Second, it induces a *relative preference effect* as, in this example, w drops from w_0 to w_1 , which moves the equilibrium from Bto C holding investments in children constant along TI₁.

[Figure 1 Here]

In words, the example in Figure 1 shows that the investment effect induces a drop in Q and N, while the relative preferences effect drops Q further while increasing N. Therefore, the total effect for Q is negative while the total effect for N is ambiguous.

Equation 5' plus 6' imply Equation 3. Equation 4' plus 5' implies that $f(H_m)[(1-H_d)W'(w_qN^{un}Q^{uq-1})] / (P_q+NP_{qq}) = U(C_l)$ '; If H_m rises then the left side decreases and thus $U(C_l)'$ decreases, which implies that C_l increases; Because of Equation 3, adjustments in Q and N go in the same direction so both Q and N decrease. Therefore total investment in children decreases. The analysis of a rise in H_c goes along the same lines.

In the framework presented in this section, a *simultaneous* rise in H_m and H_c unambiguously produces a negative *investment effect*, since both investment effects are negative and thus reinforce each other. But it will also produce an ambiguous *relative preferences effect*, since both preference effects could go either way.

The literature suggests that higher child mortality H_{e} decreases the valuation of Q relative to N because human capital investments in child Q such as education take longer to pay back than investments in N (see, for example, Datar, Ghosh and Sood 2007). Thus a rise in H_{e} is likely to be associated with a drop in w, as shown in Figure 1. Higher likelihood of maternal mortality H_{m} may enhance the importance of the wellbeing of children in the case of orphanhood (De Lannoy 2005 presents qualitative evidence about this point.). Thus if an orphan's wellbeing is greater if their human capital is higher, a rise in H_{m} is likely to be associated with a rise in w. Therefore H_{m} and H_{e} 's relative preference effects increase and decrease w, respectively.

As a result, if H_m 's and H_c 's relative preference effects counteract each other at least to some extent, while their investments effects reinforce each other, it is reasonable to expect that a rise in H_m and H_c will induce lower levels of Q and N.

Section 2: Data and Measures

The Malawi Longitudinal Study of Families and Health (MLSFH) provided the data for our estimation. MLSFH has conducted so far six waves (1998, 2001, 2004, 2006, 2008, 2010)¹¹, containing rich demographic information on all members residing in the sample households, along

¹¹ The 2010 wave is not yet available.

with information on transfers of money and help, social networks, income, labor participation, schooling, household wealth and consumption, capital accumulation, attitudes in relation to HIV/AIDS, and many other variables.

The initial MLSFH survey in 1998 included approximately 1,500 ever-married women and 1,100 spouses of these women¹². Subsequent waves were augmented to include new spouses (2001), adolescents (2004), and the spouses of married adolescents (2006). In 2006, the MLSFH included more than 3,200 male and female respondents aged 17 to 60 who were asked about a wide range of demographic, health, and socio-economic characteristics. Comparisons with the Malawi Demographic and Health Survey showed that the MLSFH sample population is reasonably representative of the rural Malawi population (Anglewicz et al., 2009). Data from Wave 3 (2004), 4 (2006) and 5 (2008) are used in this paper, and the longitudinal data across these multiple MLSFH waves will allow us to control in our estimates for fixed effects, such as those that might confound the estimated impact of mothers' subjective survival probabilities on children's human capital.

Data from the MLSFH was matched across three waves to create the data used in this paper. To match children in wave 2008 with the same children in 2006 and the same children in 2004 was possible because of data on the first and last names of people listed in the household roster. Children were matched using names, ages, sex, and birth order. Because not all data were available in every wave, and because the spelling of names is not always exactly identical across waves, the matching was not undertaken with a computerized algorithm, but was done case by case instead. Two processes were undertaken simultaneously. First, names were designated the principal matching

¹² Detailed descriptions of the MLSFH sample selection, data collection, and data quality are provided on the project website http://www.malawi.pop.upenn.edu/, in a Special Collection of the online journal Demographic Research that is devoted to the MLSFH (Watkins et al. 2003), and in a recent follow-up publication that incorporates the 2004 and 2006 MLSFH data (Anglewicz et al., 2009).

variable; so to be consider matched, a minimum similarity in spelling was required. Second, a quality indicator for the quality of the match was assigned to each matched child, with the match being a) low quality, if no other data than the spelling itself was available to establish the match, and the spelling itself was not sufficiently similar across waves, b) medium, if any other variable was available (age, sex, birth order) to establish the match or, if no other data was available but the spelling matched very closely, and c) high, if two or more variables were available to establish the match. Only children of medium and high quality of match are included in this study, which represent 89% of the total matched cases. The results for matches of medium and high quality are:

[Table 1 Here]

Over five thousand three hundred children were matched between the 2006 and 2008 waves. The number of matched children between the 2004 and 2006 waves was smaller, restricted by the smaller number of children included in the 2004 wave, in which only resident children were included in the household roster.

Mothers' perceptions of HIV risks

Our study is in a high HIV prevalence area, where HIV is widely perceived to be the primary cause of early mortality for prime-age adults. Choosing a measure of individual-level HIV risks to be used in this study, however, is not straightforward because several possibilities exist in the data. The natural first option might seem to be the actual HIV status. Such a measure was collected by MLSFH through blood tests in 2004, 2006 and 2008. But there are some disadvantages in using the actual HIV status for the analyses in this paper: First, the key variable in our approach is mothers' survival perceptions; and there are reasons to believe that our respondents might not know or might not believe or understand the results of HIV tests¹³. Second, there is likely to be self selection in decisions to get tested¹⁴. Third, the actual HIV infection status at a point in time does not provide information about the differential risks that those who are not infected at that time have about future possible infections.

The MLSFH data provide another more interesting option: individuals' perceptions of being infected with HIV, which has been repeatedly asked in the 2004, 2006 and 2008 survey rounds. The wording of the question is: "How likely are you of being infected with HIV now?": 0 (no likelihood) 1 (low) 2 (medium) and 3 (high)¹⁵. This variable shows considerable variability across respondents, and despite some overestimation of the overall probability of being infected with HIV, subjective HIV infection probabilities have been shown to vary consistently with various HIV risk factors, risky behaviors and other predictors of HIV infection (Anglewicz and Kohler, 2009; Delavande and Kohler 2009).

We use three HIV subjective likelihood variables to compute a measure of HIV risk at the individual level. The three components are current subjective HIV likelihood, subjective HIV likelihood of future infections and spouses' reports on current subjective HIV likelihood. Each of these variables are recoded for our purpose as 1 ("medium" or "high") and 0 ("no likelihood" and "low"), and then the three are summed. The final variable, thus, goes from zero to three. The reason for recoding

¹³ Tabulations in De Paula, Shapira and Todd (2008) show that more than half the individuals receiving HIV positive test results in 2004 in the next wave in 2006 classify themselves as zero or low likelihood of having HIV.

¹⁴ For example, Matovu et al (2005) and Nyblade et al (2001) find self selection in testing in Uganda.

¹⁵ The exact wording of the question depends on the language of the interview. Most of the interviews were conducted in Tumbuka and Chichewa (District of Rumpi, north Malawi), Chichewa (District of Mchinji, central Malawi) and Ciao (District of Balaka, south Malawi).

each component on a zero/one basis is to focus on extreme answers ("medium" or "high") under the assumption that they are more informative¹⁶.

Maternal HIV likelihood is finally rescaled as 0 to 1. For unmarried women, for whom the husbands' likelihood of HIV risk is not observed, the constructed "HIV likelihood" goes from 0 to 2, instead of 0 to 3. This fact is taken into account when rescaling from 0 to 1.

Mothers' health, included as a control in our robustness tests, is taken from a widely-used question: "How well do you rate your general health?": 1 (very poor), 2 (poor), 3 (normal), 4 (good) and 5 (very good). As a subjective, self-reported variable, some shortcomings in interpretation may arise, even if fixed effects are added in the regressions to control for variations across individuals in how they relate these categories to different objective health categories. However, this variable significantly predicts mortality even after controlling for several other health indicators (Idler and Benyamini 1997, Singh-Manoux 2007).

Quality of children (Q)

For children's human capital we use two measures: grades of schooling attained (or schooling attainment) and general health level, as reported by the mothers. Schooling attainment is probably a fairly good report on the true variable, with studies in other contexts suggesting noise to signal ratios of 10% or less (e.g., Ashenfelter and Krueger 1994; Behrman, Rosenzweig and Taubman 1994; Behrman et al. 2010). In 9.3% of the cases, the gain in schooling between 2008 and 2006 was

¹⁶ We are not aware of evidence about the better predictive power of these extreme answers; Iddle and Benyamini (1997) summarize evidence in relation to self reports on health.

"minus one"; zero gain in schooling was assumed in those cases. In 3.0% of the cases, the gain in schooling between 2008 and 2006 was lower than minus one; those cases were eliminated.

Maternal reports on children's health are likely to incorporate larger measurement errors, as they are subjective perceptions. The child health variable used in this study is computed from the answers to "What is your child's general health?"¹⁷, "is your child usually ill?", and "has your child been ill in the past six months?". The first of these variables is recoded as 0: "very good" or "good", and 1: "poor" or "very poor", while the latter two are coded as 1 ("yes") and 0 ("no"). Each variable is then summed for each child. Children's health is finally rescaled as 0 to 1. There is the possibly of a spurious association between maternal HIV likelihood and child health reports if maternal HIV likelihood is correlated with mothers' health, and mothers in bad health are more likely to report their children as being in bad health for given objective health status (Waters et al 2000). This effect could be mitigated by including mothers' health and fixed effects as control variables.

Quantity of children (N)

For our analysis of the quantity of children, only mothers who were not pregnant and did not have children less than one year of age in period one (2006 survey) are considered. Women who were already pregnant or had children less than a year old at the time of the 2006 survey probably had smaller possibilities to be pregnant again in 2008, and thus the effect of changes in perceived HIV risk may be too small to be detected with our sample size.

¹⁷ Recoded as 0: "Very good" or "Good", and 1: "poor" or "very poor".

For the women whom we consider, a dichotomous fertility variable is used with "1" if pregnant in 2008 or they gave birth between 2006 and 2008, and "0" otherwise. The information on pregnancy in 2008 is taken directly from a survey question, while births after 2006 are inferred from the information in the household roster.

Descriptive statistics

The main descriptive statistics are in Table 2:

[Table 2 Here]

Between 2006 and 2008, mothers' perceptions of HIV risk increased by about a tenth, mothers' perceptions of child health on average hardly changed, and reported child schooling attainment increased by an average of 0.8 grades, which is consistent with a substantial proportion of children observed in 2006 attending school afterwards but also a 56% proportion not completing school grades afterwards.

Section 3: Regression of Q and N on Mothers' Perceived HIV Risk

Our basic regression has a dynamic structure in which a disturbance term, the change in mothers' perceived HIV risk between 2004 and 2006 and changes in observed control variables between 2004

and 2006 are posited to affect with a lag changes in child quality and quantity between 2006 and 2008:

$$\Delta \text{DEP}_{8/6} = \alpha + \beta * \Delta H_{6/4} + \gamma * (\Delta H_{6/4})^2 + \delta * \Delta \text{controls}_{6/4} + e, \qquad (4)$$

where $\Delta DEP_{8/6}$ is the change between 2006 and 2008 in child schooling attainment, child health or mothers' fertility; $\Delta H_{6/4}$ is the change between 2004 to 2006 in mothers' perceived risk of HIV that is included in level and squared form to allow for nonlinear effects; $\Delta controls$ are the changes between 2004 and 2006 in observed initial conditions and *e* is an exogenous shock that is assumed to be random. $\Delta H_{6/4}$ is included in level and squared to capture nonlinearities in the association between perceived HIV risk and the dependent variable. Implicit in Equation 4, we are controlling for unobserved fixed effects at the child-level or mother-level that might affect the levels of *Q* and *N*. These fixed effects might include, for example, risk aversion or ability, which might affect the levels of both maternal perceived HIV risks and the dependent variables directly. We note that these fixedeffects estimates are significantly different from random-effects estimates for all regressions in this study¹⁸.

We include changes in four *controls* for possibly confounding factors that are observed in the data. First, the prior level of child quality might play an important role in determining mothers' decisions, and this role might be correlated with maternal perceived risk of HIV. For example, Datar et al (2007) show that higher mortality gaps among children in rural India produce, later on, higher gaps in schooling, because of parents' resource allocations. If higher risk of HIV is correlated with child

¹⁸ The significance of the differences in the coefficients of interest between the fixed- and the random-effects models are investigated by the Durbin-Wu-Hausman test. The random errors model used to conduct the test also included other regressors: age of the mothers, number of siblings, regional fixed effects (one for each of the three regions included in the survey) and HIV perceived status.

mortality for reasons different than the ones analyzed in this study, omitting this variable might bias the results. Thus, $\Delta Q_{6/4}$ is included as a control variable¹⁹.

Second, shocks in wealth might affect both maternal HIV perceptions and child human capital. Moreover, because all variables used in this study are self reported, misreports of other variables such as child human capital could be correlated with the level of reported wealth. The controls for wealth that are included pertain to livestock, an important form of wealth in rural Malawi (that may be adjusted more quickly than some assets): numbers of cows, goats, pigs, and poultry, separately²⁰.

Third, mothers with poor health may underreport their children's human capital (Waters et al 2000), and also might expect lower wages and higher medical costs, which produce similar implications as do wealth shocks. Besides, children of such mothers might also be required to stay at home to take care of sick adults or to replace them in household duties, with effects on their Q. Self-reported general health therefore is included to control for mothers' health; household medical expenses as a proportion of total expenses are included to control for overall health of household members.

Fourth, a pure time effect is added to control for changes in the administration of the survey or supply-side changes in schooling or health services.

¹⁹ Q_2 and Q_1 , as dependent variables, are taken from MLSFH 2008 and MLSFH 2006. Q_1 and Q_0 , as control variables, are taken from MLSFH 2006 and MLSFH 2004. But, MLSFH 2004 does not contain the required measure of child' health. Thus, the included control for child health (Q'_1 and Q'_0) are the answers to "is your children ill?", as included in MLSFH 2006 and 2004.

²⁰ Another possible wealth indicator is household crop production. But given that the survey was conducted in the dry season, crop accounts have to rely on retrospective reports that probably have considerable measurement error as usually is the case for near-subsistence agriculture. Also crop production is affected considerably by weather fluctuations that may obscure the relation with longer-run wealth. Nevertheless, crop variables were included in exploratory regression analysis (not shown) but they showed little significance.

The results are shown in Table 3. Children are grouped by different age intervals because age is related to different processes of investments, dependence and vulnerability. Also, if children's marginal productivities, which are not observed, affect mothers' decisions, then using age intervals and controls should attenuate the unobserved variable bias.

[Table 3 Here]

The effect of perceived risk of HIV is displayed at H=0.5. At lower levels of H, the effects are not significant; 27% and 14% of women in our sample, in 2004 and 2006 respectively, have an H of 0.5 or higher. When levels of H are 0.5 or more, therefore, higher maternal perceived HIV risk is clearly associated with lower child quality and lower child quantity. Except for child health, adding controls does not produce a substantial change in terms of significance though some of the magnitudes of the point estimates are affected. Child health is affected by mothers' perceptions of HIV risks for a somewhat different age range than is child schooling. The most precise effect for child health is for the 8-12 years old age range, while schooling is affected for the 5-16 years old age range. For both kinds of child quality, the estimated coefficients have similar magnitudes between the two age intervals, without significant differences between the age ranges considered. Likewise the estimates do not differ significantly by gender of the children.

Mothers' fertility for very young mothers (18-21) and young mothers (22-25) shows no significant results, while fertility for mothers of middle age (26-35 years old) ranges seem influenced by mothers' perceived risk of HIV. The effect on relatively older women (39 to 45 years old), is smaller and not significant, perhaps due to sample size since fertility at that age is in general very low. The lack of significant results among the younger mothers might be related to the "hastening" fertility

behavior of Malawian young women discussed by Dureval (2008) and Ueyama and Yamauchi (2009)'s marriage behavior. Both of these studies conjecture that young women are responding in part to the epidemics by accelerating their marriages and fertility.

The interpretation of the magnitudes of the estimates requires some elaboration. The H variable is zero if the women reported no likelihood of current HIV, no likelihood of future infection and the husband also reported no likelihood of current HIV. High likelihood reported for any one of these three variables maps into an increase of 0.33 in the H variable. Based on the estimates in Table 3 with controls, an increase in H by 0.33, when H is 0.5 or higher, is associated with a decline of about 0.33 * -0.3 = -0.1 grades of schooling attainment, i.e. roughly around 10% of the average increase in schooling between 2006 and 2008 for the children in our data. An increase in H by 0.33 is associated with a decline of 0.33 * -0.16 = -0.05 in child health over two years, which is comparable to a decrease of 7% of average child health. An increase in H by 0.33 is associated with a change of 0.33 * -0.32 = -11% in the probability of getting pregnant over the next two years for women in the 26-35 years old age range, which is a sizable effect considering that the average probability of getting pregnant during in this time interval in our sample was 41%.

Section 4: An estimate of the relative preference effect

In the previous section we estimated a linear model for the impact of the change in HIV risk preferences on the changes in child quantity and quality. The resulting estimates comprise both the *investment effect* and the *relative preference effect* that are discussed in section 1. From the theoretical

discussion in section 1, a negative impact of HIV on both Q and N could be observed with or without a change in the relative preferences for Q over N. In this section we attempt to explore whether a change in the HIV likelihood is associated with a change in preferences for Q. To achieve this goal, the model is simplified in the following ways. *First*, only older women, whose fertility is presumably fixed²¹, are used, thus ruling out decisions about child quantity. *Second*, the model in section 1 is slightly modified to include a specific mechanism for the change in preferences:

$$U(C_{1}) + (1-H_{m})[U(C_{2}) + (1-H_{c})W(Q)] + H_{m}*V_{m}(Q_{2}) + (1-H_{c})*V_{c}(Q_{2})$$
(6)

In equation 6, utility derived from children is a weighted average of W and V, the utility in the scenario of surviving and not surviving, respectively, where the weights depend on H_m and H_c . The utility in the scenario of not surviving, V, is experienced in period one and represents the mothers' expectation over the situation of their children in the case in which she dies. *Third*, we use the HIV likelihood in place of both H_m and H_c . *Fourth*, HIV, which is a categorical variable, is mapped into probabilities of dying using the rule that "0" corresponds to 1 percent, while anything higher corresponds to 10 percent; the results in Table 4 are fairly robust to different rules²². And finally, the following forms of U, W, V_m and V_c are assumed: $b_c log(C_1)$, $b_m log(Q_2)$, $b_{cm} log(Q_2)$ and $b_n log(Q_2)$, where all coefficients are nonnegative. Replacing these functions in the equilibrium condition derived from equation 6 and the budget constraint given above in section 2 yields²³:

²¹ Only women of completed-fertility life span are included in the analysis. The minimum age is set at 40 years old, instead of 45 or more, to attain a minimum sample size.

²² MLSFH 2006's respondents report, by selecting a number of beans from zero to ten, their likelihood of dying within the next one, five or ten years. On average, the higher risk of own mortality, associated with HIV likelihood higher than zero, is 10 percent. We also assume that this represents the children's mortality risk.

²³ The first-order conditions for equation 6 with respect to C_1 and Q, yield the following equilibrium condition: $U(C_1)' = HIV[V_m(Q_2)' + V_m(Q_2)' - 2W(Q_2)'] + W(Q_2)'(1 + HIV^2).$

$$Q_{2} = \underbrace{(b_{vm} + b_{vc} - 2b_{w})/b_{c}}_{\mu} * HIV*C_{1} + \underbrace{b_{w}/b_{c}}_{\pi} (HIV^{2}*C_{1} + C_{1}) + u$$
(7)

Equation 7 is the equilibrium condition between child quality in period two, and consumption and the HIV likelihood in period one. The disturbance term u is included to represent a random shock that affects Q_2 in period two that is not anticipated by the mother when she makes her decisions in period one. If there is no change in preferences associated with a change in HIV, then the V function should be zero (i.e., $b_{vm} + b_{vc} = 0$). The estimate of $\mu + 2\pi$ is the estimate of $(b_{vm} + b_{vc})/b_c$. While the components of this ratio cannot be identified with our data, the sign and significance of $b_{vm} + b_{vc}$ is given by the sign and the significance of this ratio under the maintained assumption that the marginal utility of consumption (and therefore b_c) is strictly positive. This seems a very weak assumption for the very low-income population being studied. As in section 4, child fixed effects are included in the estimates and the same control variables are used, with the exception of wealth, whose correlation with consumption produced unstable results. The results are:

[Table 4 Here]

Although the results are somewhat imprecise (perhaps due to the small sample sizes), Table 4 shows that $\mu+2\pi$ is significant and positive for both schooling and health. The parameter estimates for girls' health are bigger in size and have greater significance than do those for boys' health, but this is reversed for schooling, where the parameters for boys are big and significant, and those for girls are not significant.

Conclusion and Discussion

While children and adults infected with HIV are disadvantaged in several dimensions, non-infected individuals also could be affected by the HIV epidemic. This is an important point because most of the population across different countries even in the AIDS belt in Sub-Saharan Africa is HIV negative. One possibly important channel for such effects is the impact of maternal perceived HIV risk, interpreted as mortality risk, on investments in child quantity (N) and child quality (Q). We consider this channel in this paper.

We first discuss that, under our conceptual approach, higher *child* mortality predicts lower Q and has no prediction for N. We add to this previous theoretical result the role of mothers' perceived mortality risk because HIV risk implies higher mortality for both mothers and their children. We show that, under our conceptual approach, higher *mothers*' mortality risk predicts lower N but has no prediction for Q. Both child and mother's effect of higher mortality can be decomposed as the summation of a) an overall reduction on child investment related to the smaller likelihood of surviving, and b) a re allocation of child resources to N or Q depending on whether child or maternal mortality increases; if the child and mother mortality effects act together the reallocation effects counteract each other to some extent while the overall reductions reinforce each other, therefore it is possible that both Q and N will be negatively associated with HIV risk.

We estimate a dynamic linear regression between mother's HIV risk "today" and children's Q or N "tomorrow". To better control for observed and non-observed confounding factors, we use childlevel longitudinal micro data collected in rural Malawi in 2004, 2006 and 2008. Maternal selfperception of HIV risks is our key right-side variable, though we also control for fixed effects and some important observed variables such as prior child health in our estimates of Q.

Our results indicate that mothers' self-perceived risk of HIV is linked to lower levels of child quality *and* quantity, but only when the perception is moderate to high, which includes 27% and 14% of women in our sample from 2004 and 2006, respectively. Child schooling is negatively associated with this perceived risk for boys and girls between 5 and 16 years old. Child health is negatively associated with this risk for boys and girls in a slightly narrower age interval, 8-12, though the association is still visible in the 13-18 age interval. The likelihood of giving birth in the two years after 2006, for mothers who were not pregnant and had not had a recent delivery in 2006, also is found to be negatively associated with perceived HIV risk. The point estimates imply that each HIV-risk-question answered "high" rather than "low" is associated with a 10 % smaller increase in child schooling, a 7% decrease in child health, and a 25% reduction in the likelihood of births over a two-year period.

Therefore, increases in individuals' perceived risk of HIV induce smaller investments in children's health and schooling when the perceived risk is moderate or high. A policy that aims at increasing the education and awareness of HIV, however, could either increase or reduce the perceived risk.

Conceptually, while rises in H_m and H_c probably induce lower valuation of children as a whole, in one hand, they also induce a change in relative preferences for quality over quantity that could partially or even entirely counteract the lower valuation of either Q or N. In consequence the overall negative effect discussed in the previous paragraph could arise with or without changes in relative preferences. Under certain assumptions, we estimated parameters linked to the preferences for Q relative to N and found them significant for boys and girls' health and schooling. Thus HIV likelihood, as a predictor of mortality, could change relative preferences. However, apparently this effect cancels itself (because H_m 's effect counteracts H_c s effect) and/or is too small to counteract the overall negative impact of HIV on Q and N.

Among the shortcomings of this study, one is the validity of the reported risk of HIV, a concept that respondents might not understand well. Nevertheless, this variable appears significant in all regressions, consistently with a negative sign, even though controls for mothers' health, wealth, time effects, medical expenses and child or mother fixed effects are included. Another possible problem is that the relation between HIV risk and mortality may be changing as the probability of access to medication for HIV increases. A third problem might be that our small sample sizes, especially for the study of child health, might result in imprecise estimates. Also, HIV risk is not uniquely interpreted as a predictor of mortality; it could also be a predictor, for example, of poorer health or lower wealth. Finally, because of the inter temporal nature of investments and human capital, we consider only the effects of 2004/2006 variation in HIV risk on 2006/2008 variation in Q or N. However, some of the effects might show up earlier or later.

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Children report	ed*:	Children reported**:
Wave 3 (2004)	3,615	Wave 4 (2006) 6,285
Wave 4 (2006)	6,449	Wave 5 (2008) 6,753
Matched***:	2,902	Matched***: 4,837

Table 1: Matched children

*: Includes only mothers answering both wave 3 and 4. **: Includes only mothers answering both wave 4 and 5. ***: Includes matches of medium and high quality.

		Mean	N. Obs
Survey year	2006	2008	
Children's schooling attainment (grades)	3.8	4.5	1,752
Mothers' perception of child's health	0.66	0.67	1,907
Child male	50%	50%	
% Not pregnant or birth in last year	58%		
Pregnant or birth since 2006*		46%	
Survey year	2004	2006	
Mothers' perceived HIV likelihood	0.22	0.14	844
Mothers' general health	2.41	2.46	
Mothers' age	36.2		
Average number of children**	3.1		
Household consumption***	2,010	3,243	

Table 2: Descriptive statistics

*Includes only women who were not pregnant and did not have a child less than one year old in 2006 survey. **includes all children, even those not included in our sample ***The data contain consumption in the last three months. 3,000 Kwacha \approx 22 US dollars. This mean value excludes the 12% of the respondents, who reported zero.

	With	Without controls			With controls		
Age	Effect at H=0.5	p- value	Obs	Effect at H=0.5	p- value	Obs	
Child health							
8-12	-0.03	0.51	1,501	-0.18	0.06	1,061	
13-18	-0.04	0.42	1,241	-0.10	0.25	849	
Child schooling							
5-9	-0.54	0.00	1,473	-0.32	0.03	1,040	
10-16	-0.39	0.01	1,693	-0.40	0.03	1,132	
Mothers' fe	Mothers' fertility						
18-21	-0.26	0.23	61	0.13	0.29	50	
22-25	0.22	0.42	128	-0.04	0.88	93	
26-35	-0.35	0.00	341	-0.32	0.06	237	
36-41	-0.06	0.30	281	-0.04	0.61	214	
	Girls			Boys			
Child health							
8-12	-0.18	0.10	433	-0.14	0.32	413	
13-18	-0.06	0.71	292	-0.17	0.06	334	
Child schoo	Child schooling						
5-9	-0.53	0.02	573	-0.65	0.00	567	
10-16	-0.48	0.06	589	-0.39	0.07	621	

Table 3: Results of regressing changes in Q and N on changes in maternal perceived risk of HIV. The effect of HIV is displayed at H=0.5*

*. All regressions include the time period and control for a child or mother fixed effect. Other controls include changes in mothers' reported health (categorized), total number of cows, total number of pigs, total number of goats, total number of poultry, the percentage of total consumption for medical expenses and, in the case of children's quality, the initial child quality. A robust estimator of the covariance matrix is used. The maternal perceived HIV risk takes five values from 0 (very low) to 1 (very high). Age intervals for which no significant results were found for child quality are omitted. The regressions for girls and boys in the lower half of the table include controls.

Girls			Boys			
Child heal	lth					
Age	μ+2π	p- value	Obs	μ+2π	p- value	Obs
8-12	3.8	0.02	195	1.9	0.23	193
13-18	6.0	0.04	191	2.4	0.06	266
Child schooling						
Age	μ+2π	p- value	Obs	μ+2π	p- value	Obs
5-9	-1.3	0.68	127	10.3	0.05	140
10-16	-4.3	0.32	253	7.9	0.09	259

Table 4: Changes in preferences over Q (mothers of age 40 or more)

* Linear child fixed effect regressions are used, with a robust estimator of the covariance matrix. Consumption is top-coded at its 95 percentile value, and includes adult spending on fabric, clothes, shoes, agricultural goods and funeral-related expenses. Controls include mothers' reported health (categorized), percentage of total consumption dedicated to medical expenses and, in the case of child quality, the initial child quality. Perceived HIV risk is recoded as 1%, if the original value was zero, and 10% otherwise. **Figures**

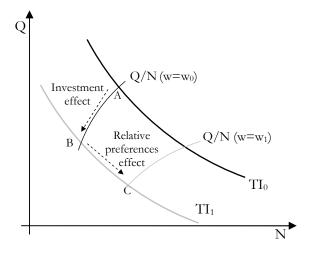


Figure 1: Graphical example. A rise in H_{ι} that induces a drop in w

*Q/N: Optimum path of Q and N when total investments in children change and relative preferences are constant. TI₀ and TI₁: combinations of Q and N that are attainable with constant levels of investments in children.