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A PORTFOLIO APPROACH TO MORTALITY SHOCKS AND FERTILITY CHOICE: THEORY AND EVIDENCE FROM AFRICA

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A Portfolio Approach to Mortality Shocks and Fertility Choice: Theory and Evidence from Africa

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Abstract

The effects of the HIV/AIDS epidemic on fertility in Africa remains ill understood. To align the contrasting findings of recent empirical research, we develop a portfolio model that captures the potential trade-off between "quantity" and "quality" of offspring. According to this theoretical model, the overall impact of mortality shocks on fertility is heterogeneous, and involves changes in human capital investment strategies. A key prediction is that investment switching and fertility impacts are conditional on income levels. We use African panel data to test the implications of the model, and find strong support for key model predictions. In particular, the impact of HIV prevalence on both fertility and human capital investments varies with income in a manner that is consistent with model predictions.

Key words: HIV/AIDS, Fertility, Portfolio, Development

JEL Code: D8, I12, J13

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1 Introduction

HIV/AID represents a first order challenge for human welfare and economic development, especially in Africa. Worldwide, more than 30 million people are infected with HIV, and Sub Saharan Africa is home to about two-thirds of these infected people. Adult HIV prevalence in Africa is approximately 5% (UNAIDS and WHO 2007), but this aggregate statistic conceals considerable heterogeneity. For example, the prevalence rate among prime age workers is as high as 20% in Southern Africa. It is estimated that HIV/AIDS killed about 2 million Africans in 2005, and that HIV/AIDS-related mortality has driven a decline in life expectancy to pre-1970 levels (US Census Bureau, 2005). It seems intuitive that such a mortality shock will not only cause enormous suffering among affected populations, but also involves large economic costs - possibly jeopardizing development prospects for generations to come. An improved understanding of the consequences of the HIV/AIDS mortality shock is important to help policy makers design interventions to cope with these costs and challenges.

To appreciate the long-term consequences of the HIV/AIDS epidemic, analysts need not only consider its effects on mortality and longevity, but also its impact on fertility. Indeed, this relationship is considered to be "one of the most important missing pieces in the puzzle of AIDS and development" (Kalemli-Ozcan and Turan (2011), p.64). The literature has not reached a consensus regarding the effect of HIV/AIDS on fertility, and the empirical evidence is mixed. While some studies find that fertility decreases in response to the increased mortality risk (Young (2005) and Young (2007)), other studies argue there is no significant effect, or that behavioral responses may invite an increase in fertility rates (Fortson (2009), Kalemli-Ozcan (2012) and Juhn, Kalemli-Ozcan and Turan (2013)). As discussed below, these differences in econometric outcomes may be attributed to differences in HIV prevalence data, variation in econometric specifications, and variation in countries included in the sample (Kalemli-Ozcan (2012)). In this paper we focus on the latter possibility, and consider the possibility that the relation between HIV/AIDS and fertility is heterogeneous - varying across the African continent in a systematic fashion. Specifically, we probe whether the relation between mortality shocks and fertility is conditional on income levels, so that cross-country studies seeking to tease out average effects may hide important underlying patterns and relations in the data. While the theory is framed in terms of the HIV/AIDS mortality shock, we emphasize the intuition is more general and may also be used to understand the impact of positive shocks to longevity, such as eventuating from new developments in medicine.

The objective of this paper is twofold. First, we develop a portfolio model to study fertility, allowing heterogeneous treatments so that the effect of mortality shocks on fertility varies with pre-shock income levels. A key element of the model is that households choose both quantity and quality of their offspring, so our model builds on work by Becker and Lewis (1973) and Becker and Barro (1988). We find that fertility may increase or decrease in response to increased life expectancy, depending on income levels, and that quantity-quality trade-offs are driving these outcomes. Specifically, choices with respect to both fertility and human capital accumulation are determined by the so-called Sharpe ratio associated with offspring of low and high quality. We demonstrate that, for intermediate income levels, mortality shocks will invite a downward adjustment in human capital investments and a matching upward response in fertility. In contrast, households in low and high income categories - to be made more precise below - respond by reducing fertility. This finding may help to reconcile the conflicting evidence on the fertility-effects of mortality shocks that has heretofore characterized the literature.

Second, we test our theoretical model using a large sample of African countries. We find strong empirical support for key model predictions. Most importantly, the effect of the HIV/AIDS mortality shock on fertility is heterogeneous and varies with income – while both the poorest and rich countries respond to the shock by decreasing fertility, we show that mid-income countries respond by increasing fertility and simultaneously "switching" from high-quality to low-quality offspring.

The paper is organized as follows. In section 2 we summarize the evolving literature on the impact of the HIV/AIDS mortality shock on fertility, and use this to motivate our theoretical model. In section 3 we present our theoretical model, and derive key testable implications. In section 4 we introduce our data and outline our identification strategy. In section 5 we present our empirical results, focusing on both fertility and human capital accumulation. Section 6 concludes.

2 Literature Overview

Fertility choice is a classical theme in economics. Modern economic thinking about fertility goes back to pioneering work by Becker and Lewis (1973) who studied the trade-off between child quantity and quality. Building on this idea, endogenous fertility has been an element of, for example, long-term growth theory (e.g., Becker and Barro (1988), Galor and Weil (1999)). The quantity-quality model of fertility typically suggests fertility should increase in response to increased mortality risk (reduced longevity). As returns to investments in human capital fall, parents choose to invest less in each child, but may also decide to have more children.

Obviously, the relation between HIV/AIDS and fertility is much more complex than this. In the context of this epidemic other "micro" effects are relevant as well. For example, there is a direct physiological (biological) effect, associated with reduced fecundity and perhaps lower energy levels to engage in sexual activities. Sexual behavior may also change as people seek to reduce the risk of infection (by foregoing unprotected sex, reducing fertility as a by-product), or if mothers who are aware of HIV transmission channels try to avoid giving birth to infected children. In addition, there may be important "macro" effects if mortality shocks are sufficiently severe. Young (2005) and Young (2007), for example, points to increasing capital to labor ratios. As returns to labor increase, so do the opportunity costs of time for women, which will invite a (further) fall in demand for offspring. This mechanism explains why a relatively short-lived shock may have persistent fertility effects (think of the plague in Europe). In sum, theory does not produce an unambiguous prediction concerning the effect of increased HIV/AIDS mortality risk on fertility – the relation may be positive or negative, or these opposing effects may offset each other so that a zero net effect results.

This ambiguity is reflected in empirical analysis. This work received an important impetus by the work of Young, who argued that the HIV/AIDS mortality shock represents an economic boon to Africa. Combining country-level variation in HIV prevalence and individual-level fertility data, he studied the economic consequences of the HIV/AIDS epidemic for South Africa (Young (2005)) as well as for a sample of 29 African countries (Young (2007)). A key element of the story is a drop in fertility resulting from the epidemic. Physiological effects play a minor role in this process, and Young attributes the bulk of the fall in fertility to a reduced demand for children. This is reflected in an increased demand for contraceptives (including, but not limited to, condoms) and a reduction in survey-based expressed fertility. The mechanism linking mortality to fertility includes lower dependency ratios (increasing effective participation) and higher capital to labor ratios. Even after controlling for the loss of prime age working age adults and the epidemic's effect on human capital accumulation, Young (2007) concludes a net windfall gain eventuates, which may be used to increase current consumption or to finance patient care programmes: "the behavioral response to the HIV epidemic creates the material resources to fight it" (p.322).

Not surprisingly, this analysis has proven controversial and has been contested. Crucially, the negative relationship between HIV/AIDS and fertility appears to be absent in analysis based on more recent rounds of Demographic and Health Surveys (DHS). These data contain improved estimates of HIV prevalence based on individual testing of survey respondents, rather than voluntary testing of pregnant women. Moreover, the most recent fertility estimates suggest a stall, or even reversal, in African demographic transition patterns. Focusing on a sample of 13 African countries, Juhn et al. (2013) find no effect of community HIV prevalence on fertility choices of HIV-negative women.[1] Kalemli-Ozcan (2012) uses a panel of 44 African countries

[1]Own infection status negatively affects future fertility (presumably a physiological effect), but this effect is too small to significantly affect overall estimates of the relation between HIV/AIDS prevalence

and probes various model specifications (exploiting between and within-country variation) as well as different HIV data, and finds there is no robust relation between HIV prevalence and fertility - some models produce positive outcomes, others negative ones, but most models do not produce any significant association. These results support earlier evidence by Fortson (2009), who using a sample of 12 African countries, concludes "there is no evidence that living in an area with high levels of HIV has a sizable effect on the fertility of those who are not directly affected (i.e., infected) by the virus" (p.172).

Hence, the literature contains different – sometimes opposite – estimates of the overall effect of mortality shocks on fertility. As mentioned, this divergence can to some extent be attributed to the nature of HIV prevalence data across DHS rounds. Recent data, based on individual testing of respondents, are more accurate, and allow distinguishing between the effect of own infection status versus that of community-wide HIV prevalence (Juhn et al. (2013)). But there are other reasons for the lack of consensus. For example, assumptions with respect to HIV prevalence for the pre-1990 period, for which no HIV data are available, matter for model outcomes (Kalemli-Ozcan and Turan (2011)), as do assumptions with respect to the identification strategy – whether the analyst focuses on cross-country or within-country variation, and on assumptions with respect to clustering of standard errors (Kalemli-Ozcan (2012)).

Yet another reason for conflicting outcomes is variation in the sample of countries included in the analysis. If the effect of mortality shocks on fertility is conditional on country characteristics, such as income levels, then an aggregate analysis that lumps together all countries might obscure rather than reveal key relationships (see Table 1 for an overview of the countries involved in the various studies). Moreover, different sub-samples drawn from the overall sampling frame may produce different estimates. We will probe this latter possibility in our own empirical work below.

Before presenting our portfolio-approach to mortality shocks and fertility, we briefly introduce another relevant strand of the literature on the consequences of mortality shocks. In addition to affecting the number of children, mortality shocks may affect the level of investments in human capital per child - the possibility of a quantity-quality trade-off discussed above. Young (2007) is optimistic about the adverse effects of the epidemic on the quality of children. Analysing the accumulation of human capital among orphaned children, he concludes HIV prevalence "does not appear to exert any consistent dampening effect on the accumulation of human capital" (p.305). But this result has been disputed as well. Using more recent data, Fortson (2011) considers the effect of HIV/AIDS induced mortality risk among African children and youths (not just among orphans), and concludes it slows down human capital ac-

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and fertility.

cumulation. This is reflected across a range of indicators: years of schooling, school attendance, primary school completion, and progress through school. While these outcomes are consistent with a reduction in the expected return to investments in human capital, they may also be due to changes in family resources, or to changes in market prices (e.g. wages). Regardless of the exact channel, the overall effect appears large and significant, for both orphans and non-orphans. For related insights, refer to Meltzer (1992), Bell, Devarajan and Gersbach (2006), and Jayachandran and Lleras-Muney (2009).

Insert Table 1 approximately here.

3 Theoretical Model

3.1 Portfolio Model of Fertility Choice

We assume agents view children as investment goods rather than durable (consumption) goods. The assumption is considered to be realistic particularly for developing countries, where the pension systems and financial markets are underdeveloped (c.f. Portner (2001)). So parents invest in human capital of their children, and expect a certain return after their offspring has matured. Like other investments, the rate of return on investing in offspring is uncertain and several factors, including disease, may inhibit human capital from maturing. Consider an agent with mean-variance utility preferences. Her portfolio consists of two assets: a risky asset, children, and a risk-free asset, possibly a salaried job or accumulated savings. The utility maximization problem is given by:

$$\max_{\alpha_t} \quad \mathrm{E}_t(r_{p,t+1}) - \frac{\theta(w_t)}{2}\sigma^2_{p,t+1} \tag{1}$$

$$\text{s.t.} \quad r_{p,t+1} = r_{f,t+1}(w_t) + \alpha_t(r_{t+1} - r_{f,t+1}(w_t)) \tag{2}$$

where w_t is initial wealth of parents, which can be taken as both human and physical capital. $r_{p,t+1}$ and $\sigma_{p,t+1}$ are the return and the variance of the portfolio. r_{t+1} is the return on raising children; and $r_{f,t+1}(w_t)$ is the risk-free rate, which we treat as an increasing deterministic function of w_t (reflecting that high physical or human capital facilitates access to high-return investment opportunities, or better salaried jobs, c.f. Claessens (2006) and Sahn and Alderman (1988)). We also assume the coefficient of relative risk aversion, γ to be a decreasing function of wealth (DRRA utility), and that $r_{t+1}|\mathcal{F}_t, \sim N(\mu, \sigma^2)$. It is easy to verify that the following holds,

$$r_{p,t+1}|\mathcal{F}_t, \sim N(r_{f,t+1}(w_t) + \alpha_t(\mu - r_{f,t+1}(w_t)), \alpha_t^2\sigma^2). \tag{3}$$

where $\mathcal{F}_t$ represents the information set up to time t. The distribution of the return on the portfolio in Equation (3) allows us to rewrite the optimization problem as:

$$\max_{\alpha_t} \quad r_{f,t+1}(w_t) + \alpha_t(\mu - r_{f,t+1}(w_t)) - \frac{\theta(w_t)}{2}\alpha_t^2\sigma^2. \tag{4}$$

Solving for α_t yields,

$$\alpha_t = \frac{\mu - r_{f,t+1}(w_t)}{\theta(w_t)\sigma^2} = \frac{\lambda(w_t)}{\theta(w_t)\sigma}, \tag{5}$$

where λ is the so-called Sharpe ratio for investments in (the human capital of) children. Reflecting the dependence of the risk free asset on wealth, this ratio is a decreasing function of wealth, so we have:

$$\frac{\partial r_{f,t+1}}{\partial w_t} > 0, \qquad \frac{\partial \lambda}{\partial w_t} < 0, \qquad \frac{\partial \theta}{\partial w_t} < 0.$$

Equation (5) shows that the fraction of wealth allocated to bearing and raising children increases with the attractiveness of offsping, as captured by the Sharpe ratio (λ) but decreases with its riskiness (σ) and the agent's degree of risk aversion (θ). As both λ and θ decrease with wealth levels, it is not evident how wealth affects investments in children. Denoting the number of children by n_t and the cost of raising a child by C_t respectively, we obtain,

$$n_t = \frac{w_t\lambda(w_t)}{C_t\theta(w_t)\sigma}. \tag{6}$$

Next, assume there are two types of children - a "high-quality" type (H) and a "low-quality" type (L) where quality simply reflects investment in human capital during (early) childhood. Denote these investment costs by C_t where, obviously, $C_H > C_L$. The return to these types is defined as r_{t+1}, where $r_{H,t+1} \sim N(\mu_H, \sigma_H)$ and $r_{L,t+1} \sim N(\mu_L, \sigma_L)$, and where $\mu_H > \mu_L$ and $\sigma_H > \sigma_L$.[2] In words, raising high-quality type children yields higher expected returns, but also implies incurring higher costs and generating higher risks. As a benchmark model we assume agents can only raise one type of children, so they treat all children equally at least when they

[2]Throughout this paper, we use subscript "L" to represent variables associated with low-quality children and subscript "H" to represent variables associated with high-quality children.

are born. According to the model, the optimal number of type-specific children is given by:

$$n_{H,t} = \frac{w_t \lambda_H(w_t)}{C_{H,t}\theta(w_t)\sigma_H}, \tag{7}$$

$$n_{L,t} = \frac{w_t \lambda_L(w_t)}{C_{L,t}\theta(w_t)\sigma_L}. \tag{8}$$

The maximized expected utility from high and low quality of offspring are, respectively:

$$\mathrm{E}_t U_H^* = r_{f,t+1}(w_t) + \frac{\lambda_H(w_t)^2}{2\theta(w_t)}, \tag{9}$$

$$\mathrm{E}_t U_L^* = r_{f,t+1}(w_t) + \frac{\lambda_L(w_t)^2}{2\theta(w_t)}. \tag{10}$$

Proposition 1. *If $\lambda_H > \lambda_L$, the agent chooses asset H; If $\lambda_H < \lambda_L$, she chooses asset L; Otherwise, she is indifferent between asset L and asset H.*[3]

Proposition 1 states that the decision on the type of offspring only depends on the attractiveness of children as an investment opportunity, as reflected by the Sharpe ratio.

Proposition 2. *If the agent chooses asset L ($\lambda_L > \lambda_H$), then $n_L > n_H$. Otherwise, the relationship between n_L and n_H is ambiguous.*

The proof of Proposition 2 is as follows. The conditions $C_H > C_L$ and $\sigma_H > \sigma_L$ ensure that the denominator of n_H is larger than that of n_L. Therefore, if $\lambda_L \geq \lambda_H$, $n_L > n_H$. In contrast, if $\lambda_L < \lambda_H$, the relationship becomes ambiguous. Proposition 2 implies that agents choosing low-quality type offspring have more children than agents chosing the high-quality type (quantity-quality trade-off).

Figure 1 shows how optimal fertility varies with wealth. For low wealth levels, $\lambda_H = \lambda_L$ so the agent chooses low-quality offspring. As wealth increases, so does fertility, reflecting greater ability to invest in offspring. However, Sharpe ratios also change, as depicted in Figure 2. Specifically, both Sharpe ratios decrease with the wealth level, but the high-quality ratio falls more slowly so that eventually the two curves (may) intersect. When that happens, so that $\lambda_H = \lambda_L$, then agents switch from low to high-quality offspring, and fertility drops abruptly (Figure 1). Further increasing wealth implies that, again, fertility increases which simply reflects that wealthier households can afford to invest more in offspring.

Insert Figure 1 approximately here.

Insert Figure 2 approximately here.

[3] The result immediately follows from a comparison between Equation (9) and Equation (10).

3.2 Individual Responses to a Mortality Shock

We can actually apply this framework to analyze the fertility responses to various sorts of shocks in developing world, where the children are more likely to be taken as investment goods. But in this paper, we narrow our focus and only elucidate the effects of a mortality shock on fertility decisions. In what follows, a mortality shock reflects a permanent increase in the probability that offspring will die before becoming productive (or, alternatively, it lowers the expected return on investments in human capital). We assume the shock leaves the variance of returns unaffected. Since the return to the high-quality type is characterized by higher variance, the decrease in expected return of the high-quality type is larger than that of the low-quality type. Denote these shocks to the returns on high and low-quality types by m_H and m_L, respectively. We assume that[4]

$$r(w) = a + bw, \tag{11}$$

$$m_H = cm_L, \tag{12}$$

$$\theta(w) = \frac{d}{w}, \tag{13}$$

where $a > 0$, $b > 0$, $c > 1$, and $d > 0$. The Sharpe ratios before and after the mortality shock associated with both types are given by:

$$\lambda_H^B(w) = \frac{\mu_H - a - bw}{\sigma_H}, \qquad \lambda_L^B(w) = \frac{\mu_L - a - bw}{\sigma_L}; \tag{14}$$

$$\lambda_H^A(w) = \frac{\mu_H - cm_L - a - bw}{\sigma_H}, \qquad \lambda_L^A(w) = \frac{\mu_L - m_L - a - bw}{\sigma_L}, \tag{15}$$

where λ_H^B and λ_L^B represent the before-shock Sharpe ratios for the high and low-quality types, and where λ_H^A and λ_L^A denote the after-shock Sharpe ratios. In what follows we will consistently use the superscript B (A) to denote before (after) mortality shock variables. The critical wealth level where parents switch from the low to the high-quality is obtained by equating λ_H^B and λ_L^B (λ_H^A and λ_L^A after the shock), so we get:

$$w^B = \frac{\sigma_H \mu_L - \sigma_L \mu_H}{b(\sigma_H - \sigma_L)} - \frac{a}{b}, \tag{16}$$

$$w^A = \frac{\sigma_H \mu_L - \sigma_L \mu_H + (c\sigma_L - \sigma_H)m_L}{b(\sigma_H - \sigma_L)} - \frac{a}{b}. \tag{17}$$

We assume that $c\sigma_L - \sigma_H > 0$, then $w^B < w^A$, which ensures that after the mortality shock, some parents switch from high to low-quality type but not the other way around. The

[4] To ease the notation, we suppress the time representation "t" in what follows.

type-switching region induced by mortality shocks is $[w^B, w^A]$ and the width of the region, I, is given by,

$$I = \frac{(c\sigma_L - \sigma_H)m_L}{b(\sigma_H - \sigma_L)} = \frac{(c-1)\sigma_L m_L}{bD_\sigma} - \frac{m_L}{b}, \tag{18}$$

where $D_\sigma = \sigma_H - \sigma_L$. It is easy to verify I is increasing in c and decreasing in both D_σ and b. Figure 3 illustrates how type-switching is affected by the mortality shock. Compared to the before-shock case (with $w^B = 1.4$) the intersection of the two Sharpe ratios moves to the right after the mortality shock, and now occurs at a greater level of wealth (with $w^A = 1.9$). Hence, a fraction of the population, endowed with intermediate levels of wealth between $w = 1.4$ and $w = 1.9$, switches its investment strategy and opts for low-quality offspring rather than the high-quality type after the mortality risk of their offspring has been shocked up. The poor ($w < 1.4$) and rich ($w > 1.9$) are unaffected in terms of the type of children they desire, and continue to opt for the low and high-quality type, respectively.

Insert Figure 3 approximately here.

3.3 Mortality Shocks: Aggregate Response

We now turn to the aggregate fertility response to a mortality shock, paying special attention to the pivotal role of wealth as a determinant of investments in human capital. Assume wealth in a country is uniformly distributed; $w \in [\underline{w}, \bar{w}]$. For simplicity, we normalize $\bar{w} - \underline{w}$ to one, so that aggregate wealth of a country with upper bound $\bar{w}$ is given by,

$$W(\bar{w}) = \int_{\bar{w}-1}^{\bar{w}} w \mathrm{d}w = \bar{w} - \frac{1}{2}. \tag{19}$$

Hence, there is a one to one correspondence between the upper bound, $\bar{w}$ and the total wealth of the country, W. Aggregate fertility, then, is given by,

$$N = \begin{cases} \frac{1}{dC_L\sigma_L^2}\int_{\bar{w}-1}^{\bar{w}} w^2(\mu_L - a - bw)\mathrm{d}w, & \text{if} \quad \bar{w} \leq w^B; \\ \frac{1}{dC_L\sigma_L^2}\int_{\bar{w}-1}^{w^B} w^2(\mu_L - a - bw)\mathrm{d}w + \frac{1}{dC_H\sigma_H^2}\int_{w^B}^{\bar{w}} w^2(\mu_H - a - bw)\mathrm{d}w, & \text{if} \quad w^B < \bar{w} \leq w^B + 1; \\ \frac{1}{dC_H\sigma_H^2}\int_{\bar{w}-1}^{\bar{w}} w^2(\mu_H - a - bw)\mathrm{d}w, & \text{if} \quad \bar{w} > w^B + 1, \end{cases}$$

This outcome is graphically depicted in Figure 4. As aggregate wealth levels increase, fertility first increases, then decreases, and finally increases again. These dynamics capture the interplay between the direct income effect (raising demand for offspring) as well as the indirect effect via the incentive to invest in human capital. Across a range of wealth levels there exists a tradeoff between quantity and quality of offspring.

Insert Figure 4 approximately here.

Insert Figure 5 approximately here.

How does a mortality shock affect aggregate fertility? The model yields positive and negative effects, which may help us to understand the ambiguous empirical evidence that heretofore dominates the literature. Figure 5 shows the aggregate fertility for countries with different levels of wealth, before and after the mortality shock. N^B and N^A, are the aggregate fertility before and after the mortality shock, respectively, and W^B and W^A are the matching threshold wealth levels.

As shown in the top panel, a mortality shock moves the entire fertility distribution to the right. In the bottom panel we summarize the implications in terms of changes in fertility (i.e., fertility after the shock minus fertility before the shock). This net effect may be positive or negative-depending on the income level. Hence, the impact of HIV/AIDS on fertility is heterogeneous, and should vary across samples (that is; when different samples contain countries with different income levels). In samples dominated by lowest-income countries, the mortality shocks lowers the average fertility rate. However, samples containing sufficient intermediate income countries may display a positive average treatment effect. We will return to this issue below.

Define the "transition region" at aggregate level as the region between the two critical income levels, W^B and W^A, where countries switch from full investment in low-quality children to having both types. This is the region where fertility would be falling in the absence of the mortality shock, but is actually increasing in the presence of the mortality shock. We can prove that, as long as the cost of raising a high-quality child is sufficiently large (relative to the cost of raising a low-quality type), the transition region extends to $\bar{w} = w^B$.[5] For this reason, we assume

$$C_H > lC_L, \tag{20}$$

where

$$l = \frac{\sigma_L^2}{\sigma_H^2} \frac{(\mu_H - a)(w^B)^2 - b(w^B)^3}{(\mu_L - a)(w^B - 1)^2 - b(w^B - 1)^3}. \tag{21}$$

[5]Proof can be found in Appendix.

The one to one correspondence between the upper bound $\bar{w}$ and aggregate wealth of the country, W implies that the threshold before the mortality shock is given by,

$$W^B = \int_{w^B-1}^{w^B} x\mathrm{d}x = w^B - \frac{1}{2}. \tag{22}$$

Similarly, the threshold after the mortality shock is given by,

$$W^A = \int_{w^A-1}^{w^A} x\mathrm{d}x = w^A - \frac{1}{2}. \tag{23}$$

Therefore, as long as $c\sigma_L - \sigma_H > 0$, $W^A > W^B$, which ensures that the transition region between W^B and W^A in Figure 5 always exists. Obviously, for poor countries ($W_t < W^B$), the aggregate fertility increases monotonically with the aggregate wealth level irrespective of whether there are mortality shocks, or not. Moreover, as shown in the bottom panel, the difference between the number of children after and before the mortality shock decreases because of the direct mortality effect. The proof is as follows. If $1 < \bar{w} \leq w^B$, N^B and N^A are given by,

$$N^B = \frac{1}{dC_L\sigma_L^2}\int_{\bar{w}-1}^{\bar{w}} w^2(\mu_L - a - bw)\mathrm{d}w, \tag{24}$$

$$N^A = \frac{1}{dC_L\sigma_L^2}\int_{\bar{w}-1}^{\bar{w}} w^2(\mu_L - a - bw - m_L)\mathrm{d}w. \tag{25}$$

Thus, the difference between N^B and N^A is,

$$N^A - N^B = -\frac{m_L}{3dC_L\sigma_L^2}(3\bar{w}^2 - 3\bar{w} + 1). \tag{26}$$

It can be easily verified that $(N^A - N^B)$ is a decreasing function of $\bar{w}$ for $\bar{w} \in (1, w^B]$.

The top panel of Figure 5 demonstrates that total fertility before the mortality shock decreases over the interval $[W^B, W^A]$, while after the mortality shock fertility increases. Hence, to ensure that the intersection of N^B and N^A exists between W^B and W^A, the aggregate fertility associated with wealth level before the mortality shock (denoted by $N^{B,A}$) should be smaller than that after the mortality shock (denoted by $N^{A,thre}$). $N^{A,thre}$ is given by,

$$\begin{aligned} N^{A,thre} &= \frac{1}{dC_L\sigma_L^2}\int_{w^A-1}^{w^A} w^2(\mu_L - a - bw - m_L)\mathrm{d}w \\ &= \frac{1}{dC_L\sigma_L^2}\left[\frac{\mu_L - m_L - a}{3}((w^A)^3 - (w^A-1)^3) - \frac{b}{4}((w^A)^4 - (w^A-1)^4)\right]. \end{aligned} \tag{27}$$

The value of $N^{B,A}$ depends on the relation between w^B and $w^A - 1$. In what follows, we discuss two cases: $w^B > w^A - 1$ and $w^B \leq w^A - 1$.

1. $w^B > w^A - 1$. There exist m_l^{thre} and m_u^{thre} such that $m_l^{thre} < m_L < m_u^{thre}$, which implies that, to ensure the existence of transition, mortality shocks cannot be too small nor too large. The intuition is that if mortality shocks are "too big", even type-switching agents will not increase their number of children as income increases because of the extremely low return on offspring. In contrast, if the shocks are "too small", every agent only slightly reduces her offspring, regardless of the type, and type-switching does not occur.

2. $w^B \leq w^A - 1$. There exists m^{thre} such that $m_L < m^{thre}$. Since $w^B \leq w^A - 1$ ensures that shocks cannot be very small, we only have to rule out that mortality shocks are too large. If mortality shocks are "too big", even type-switching agents are not willing to increase their offspring due to the low expected return.

3.4 Testable Hypotheses from the Model

Our model, as summarized in Figure 5, predicts that after a mortality shock the fertility rate decreases for very poor countries. This drop in fertility increases with wealth (region P in the Figure). Slightly richer countries, such as those in region Q, also experience that the mortality shock reduces fertility, but for these countries the rate of decrease is decelerating with wealth. Finally, for intermediate-wealth countries, or those in Region R, fertility increases after the mortality shock. Overall, as depicted in the bottom panel of Figure 5, as wealth increases, the effect of mortality on fertility may be represented by a quadratic function. Based on the U-shape pattern that we observe on the left side of Figure 5 (where we assume African countries are located), we derive the first hypothesis.

Hypothesis 1. *The negative effect of HIV/AIDS on total fertility rate is accelerating with wealth first, then the negative effect becomes decelerating with wealth, finally the effect turns to be positive.*

We expect that more complex patterns can be found in datasets that contain a greater spread in wealth levels - that is; in countries containing African and non-African countries (while suffering from a similarly dramatic mortality shock). Moving further towards the right of Figure 5 we observe that fertility appears to not respond to mortality for a range of wealth levels (that is; to the right of w^A), and eventually to drop (again).

Another pattern contained in Figure 5 concerns the effect of mortality on human capital accumulation, which we will proxy with school enrollment. According to the model, type-switching is an important reason for changes in fertility following the mortality shock. Hence, we do not predict school enrollment to drop seriously for very poor countries (characterized by low-quality types before and after the shock) or for rich countries (where both before and after the shock most children are of the high-quality type).[6] In both poor and rich countries, the size of the population is affected, but not its composition. However, the same is not true for intermediate-wealth countries, which are characterized by massive dis-investment in human capital, and the associated switch from high to low-quality type offspring. The share of low-quality type children in the population increases, and school enrollment falls accordingly. This observation forms the basis for our second hypothesis:

Hypothesis 2. *A mortality shock lowers the accumulation of human capital, for intermediate wealth countries, but not for poor ones.*

4 Data and Identification

4.1 Data

Some of previous literature is based on individual-level data, mainly from the DHS (see section 2). However, this database does not contain much data about income or wealth. Specifically, while it contains information on wealth, this information is only available after 2005 (DHS V and DHS VI), which is not helpful to us because the HIV shock hit Sub-Saharan Africa in the 1980s. For this reason we use country-level data for our main analysis, and only use individual-level data for a simple robustness check. Specifically, we use panel data for 44 countries from Sub-Saharan Africa, for the period 1975 to 2007. HIV prevalence ($prev\_hiv$) data are from UNData. Income level (inc), education level ($prise$, $fprise$, $mprise$, $secse$), infant-child mortality rate ($infmor$ and $u5mor$), total fertility rate (tfr) are from the World Bank Database. Table 2 summarizes the descriptive statistics. The sample has at least 792 observations. Both the coefficients of variation for $prev\_hiv$ and inc exceed one, indicating the variation is sufficiently large to capture the effects of HIV and income.

Insert Table 2 approximately here.

[6] Again, because we focus on a panel of African countries, we do not expect to observe this response of "rich countries" in our data.

4.2 Identification and Specification

To identify Hypothesis 1, we follow Fortson (2009) and use a dummy variable, $post\_90$, which is set equal to one for observations after 1990 to identify the effect of HIV prevalence on fertility. This rests on the assumption that HIV prevalence in Sub-Saharan Africa was very low in the 1980s, and most of the HIV-induced mortality occurred after 1990 (as did, presumably, the behavioral response we are interested in). If so, we may use 1990 to identify the before-after effect of HIV. We also introduce an interaction term capturing the product of income level (gross national income per capita, inc), and the $post\_1990$ dummy variable, to identify how income mediates the impact of the mortality shock. We use average income level (gross national income per capita) as a proxy of wealth, since income is highly correlated with wealth, especially at the aggregate level. Hypothesis 1 predicts the effect of income should be quadratic. We introduce time-varying co-variates to cope with potential omitted variable bias and, since we have a panel of country level data, we also use fixed effects. This amounts to the following specification:

$$\begin{aligned}\log\left(tfr_{it}\right) = \alpha_0 + \alpha_1 post\_90_{it} + \alpha_2 inc_{it} \times post\_90_{it} + \alpha_3 inc_{it}^2 \times post\_90_{it} \\ + \alpha_4 inc_{it} + \alpha_5 inc_{it}^2 + \beta' \mathbf{x}_{it} + \alpha_i + \varepsilon_{it},\end{aligned} \tag{28}$$

where tfr_{it} is total fertility rate for country i in year t. Since tfr is bounded by zero, we take its log so that the error term more closely resembles a normal distribution. Next $\mathbf{x}_{it}$ is a matrix of co-variates, consisting of education and the infant-child mortality rate. Education level controls for other preference to human capital than income concerns. We have two measures of education; primary school enrollment rate, $prise$ and secondary school enrollment rate, $secse$. According to Zhang (1990), Doepke (2005) and others, infant-child mortality rate controls for the replacement effect, which can lead to high fertility rates if infant mortality is high. We have two such measures of the infant-child mortality rate; $infmor$ and child mortality rate under 5, $u5mor$. Finally, α_i is the country fixed effect. We may transform the specification into:

$$\log\left(tfr_{it}\right) = \alpha_0 + \left(\alpha_1 + \alpha_2 inc_{it} + \alpha_3 inc_{it}^2\right) \times post\_90_{it} + \alpha_4 inc_{it} + \alpha_5 inc_{it}^2 + \beta' \mathbf{x}_{it} + \alpha_i + \varepsilon_{it}. \tag{29}$$

The effect of the mortality shock on total fertility rate is quadratic in income levels. If our hypothesis holds, then α_1 and α_2 should be negative, and α_3 is positive. As income rises, fertility first decreases sharply, then decreases more slowly, and finally increases. We refer to Equation (29) as Specification I in what follows.

To avoid the measurement error issue in Young (2005), as pointed out by Kalemli-Ozcan and Turan (2011), we also employ another measure of HIV/AIDS. That is, we use the time-varying measure of the prevalence of HIV, denoted as $prev\_hiv$. This implies specification II, where we again expect the effect of the mortality shock to be quadratic in income levels:

$$\begin{aligned}\log(tfr_{it}) = \alpha_0 + \alpha_1 prev\_hiv_{it} + \alpha_2 inc_{it} \times prev\_hiv_{it} + \alpha_3 inc_{it}^2 \times prev\_hiv_{it} \\ + \alpha_4 inc_{it} + \alpha_5 inc_{it}^2 + \beta' \mathbf{x}_{it} + \alpha_i + \varepsilon_{it}.\end{aligned} \tag{30}$$

We also expect the effect of mortality shock is quadratic to income level.

To provide further evidence to Hypothesis 1, we narrow our focus on the second part of the hypothesis. Above a certain income level, the drop in fertility rate due to mortality shock slows down with income rising, and eventually the effect turns positive. Taking a similar strategy as above, we use the $post\_90$ dummy to identify the effect of mortality shock, then introduce the interaction term $inc \times post\_90$ to capture the change mediated by income. Specification III is:

$$\log(tfr_{it}) = \gamma_0 + \gamma_1 post\_90_{it} + \gamma_2 inc_{it} \times post\_90_{it} + \gamma_3 inc_{it} + \rho' \mathbf{x}_{it} + \gamma_i + \epsilon_{it} \quad if\ inc > i\bar{n}c, \tag{31}$$

where $i\bar{n}c$ is a threshold above which the negative effect starts to decelerate with income. It can be transformed into

$$\log(tfr_{it}) = \gamma_0 + (\gamma_1 + \gamma_2 inc_{it}) \times post\_90_{it} + \gamma_3 inc_{it} + \rho' \mathbf{x}_{it} + \gamma_i + \epsilon_{it} \quad if\ \ inc > i\bar{n}c. \tag{32}$$

If our hypothesis holds, γ_1 should be negative and γ_2 should be positive. Of course we can also use $prev\_hiv$ to estimate this model, which produces Specification IV:

$$\log(tfr_{it}) = \gamma_0 + \gamma_1 prev\_hiv_{it} + \gamma_2 inc_{it} \times prev\_hiv_{it} + \gamma_3 inc_{it} + \rho' \mathbf{x}_{it} + \gamma_i + \epsilon_{it} \quad if\ \ inc > i\bar{n}c. \tag{33}$$

To identify Hypothesis 2, we also take 1990 as the shocking time, and use the five-year lagged variable of $post\_90$ to capture the effect of mortality on human capital. Following Fortson (2011), present school enrollment rate depends on HIV prevalence five or more years ago, when the offspring was born. We take primary school enrollment as the dependent variable, yielding Specification V:

$$\log(prise_{it}) = \theta_0 + \theta_1 post\_90_{it} + \theta_2 inc_{it} + \theta_i + \nu_{it} \qquad if\ inc > i\bar{n}c, \tag{34}$$

where the primary school enrollment rate is denoted by $prise$. We do not only test the effect of HIV/AIDS on overall enrollment, but also on female enrollment and male enrollment separately. If Hypothesis 2 holds, then above a certain income level where type-switching occurs, the parameter θ_1 should be negative.

5 Empirical Results

5.1 Cross-Country Evidence

Table 3 reports the estimation results of Specification I. Since we have two measures for education and infant-child mortality, we have four combinations of control variables. In column 1, we take $prise$ and $u5mor$ as co-variates, and find that the estimation results are perfectly in line with our predictions. The coefficient to $post\_90$ is negative, indicating that HIV reduced fertility when income is low. The coefficient of interaction term $inc \times post\_90$ is negative, and that of $inc^2 \times post\_90$ is positive. These coefficients provide the U-shape curve of mortality shocks discussed above. All the estimated parameters are significant. In column 2, we use $secse$ and $u5mor$ as controls. In column 3, $prise$ and $infmor$ are used, and $secse$ and $infmor$ are used in column 4. Overall, the estimation results tend to be consistent with our predictions, and most coefficients are statistically significant. The signs of the coefficients in column 2 are also of the right sign, but not significant. This may be due to multi-collinearity. The correlation of $inc \times post\_90$ and $inc^2 \times post\_90$ is 0.94, so multi-collinearity concerns may eventuate, lowering the efficiency of our estimations.

To avoid potential measurement error problems, we next employ $prev\_hiv$ as measure of HIV, or test Specification II. Table 4 shows the estimation results. Across the columns in this Table we use the same combinations of controls as in Table 3. Overall, as before, the estimated coefficients are consistent with Hypothesis 1. That is, the coefficients of $prev\_hiv$ and $inc \times prev\_hiv$ are consistently negative and significant. In two out of four specifications, the coefficient of $inc^2 \times prev\_hiv$ is significantly positive. Based on the parameter in column 1, Figure 6 depicts how fertility is affected by the mortality shock across a range of income levels. The effect is negative for low incomes. As income rises, the effect drops further, until average income equals $5,940 per year (or about twice the average income level in our sample). As income increases further, households switch from high to low quality offspring, and the negative effect of mortality shock shrinks. Finally, for average incomes exceeding $15,286 per year, we observe that the HIV epidemic raises fertility rates. This is just within sample observations for our panel, but it is evident that most countries in our sample are far removed from this critical income level.

Insert Table 3 approximately here.

Insert Table 4 approximately here.

Insert Figure 6 approximately here.

Table 5 and Table 6, which focus on the dynamics in regions Q and R (in the top panel of Figure 5), further support Hypothesis 1. When income exceeds a certain threshold, the negative effect of HIV on fertility is decelerating with income, and eventually turns positive. We use *post*_90 and *prev*_*hiv* to identify the effect of the mortality shock, and the effect of income is now identified by the interaction term $inc \times post\_90$ and $inc \times prev\_hiv$. We simply take $2000 per year as the threshold. The results in Table 5 fit our prediction well – across almost all columns we obtain significant coefficients of the "right sign". The empirical findings of Table 6 are less robust, but we find these results are sensitive to the threshold chosen. For example, when we raise the threshold to $8000 per year, the estimations become significant and tend to confirm the earlier insights (results not shown but available on request).

Insert Table 5 approximately here.

Insert Table 6 approximately here.

Next, turn to Hypothesis 2, dealing with the impact of mortality on human capital investments. Results are reported in Table 7. The theory predicts that, for average income exceeding a certain level, HIV/AIDS negatively affects human capital accumulation because of type-switching. We take primary school enrollment as a measure of investment in human capital, and consider also female enrollment rate (column 1 and 4) and male enrollment rate (column 2 and 5) separately. We again take $2000 per year per capita as the threshold. In the first three columns, we use *inc* to capture income, and in the next three columns we use a five-year lagged term of *inc* to reflect that perhaps enrollment rates are determined by both present income as well as income levels at the time when fertility choices were made.

All estimation results strongly support Hypothesis 2 - the coefficient of of *inc* is positive and that of *post*_90 is negative. All the coefficients are statistically significant. Based on this model, we can estimate the effect of mortality shocks on school enrollment, conditional on income. For example, for an income level of $5000, enrollment rates go down by 5% compared to the before mortality shock situation. Another finding is that the reduction in enrollment for girls is significantly larger than for boys.

Insert Table 7 approximately here.

We have done ample checks to probe the robustness of our findings. A sample of these results is presented in Table 8 (qualitatively similar results are found for model specifications with other controls–details available on request). First, since both the income and mortality shock can have lagging effects, we employ lagged terms of inc and $prev\_hiv$ or $post\_90$ to re-estimate Specification I. This approach should also attenuate concerns about potential endogeneity of income and HIV prevalence variables. In columns 1 and 2, we use lagged terms for both income and the mortality shock, and find rather similar results as those reported in Table 4 and Table 3. All the coefficients are consistent with Hypothesis 1, and most are significant. In columns 3 and 4 we only use the lagged term of income, and this estimation also supports our predictions.

In columns 5 and 6 we use lagged term income and mortality to test Specification III and IV, using a threshold of $2000 per year. In columns 7 and 8, we only use the lagged term of income. As before, we find that all the estimated parameters are in line with our predictions, and in most cases, coefficients are significant. Next, since the time span of our data exceeds 30 years, time series issues may emerge. As a simple test, we re-estimate Specifications I using only data for the period 1980-2000. As shown in column 9, the estimated coefficients are still consistent with our predictions. Finally, we use upper and lower estimates of HIV prevalence provided in the literature to estimate Specifications I-IV. Results are provided in Table 9 and, again, they support our main predictions.

Insert Table 8 approximately here.

Insert Table 9 approximately here.

5.2 Individual Evidence

As a further robustness check we also probe the association between HIV and fertility using individual-level data. As mentioned, no wealth or income data are available for the pre-2005 DHS rounds, so we cannot capture the shock in 1980s. Instead, we adopt a cross-section specification, and refrain from making strong statements about causal inference - the results in this section are only provided to illustrate there are similar patterns in the individual-level data as in the aggregate data. The individual level data are summarized in Table 10. The model we estimate follows Juhn et al. (2013):

$$fertility_{irc} = \kappa_0 + \kappa_1 prev\_hiv_{rc} + \phi' \mathbf{x}_{irc} + D_c + D_r + \mu_{irc} \tag{35}$$

where κ_1 is predicted to be negative for poor families, and positive for rich ones. We employ data from the most recent Demographic and Health Survey (DHS) in Cameroon and Senegal, which includes information on household wealth. Three measures are taken to capture the fertility choice of woman i in region r and country c: number of birth in last year, last five year and the whole life. *prev_hiv* is the prevalence of HIV in the region of residence. We also include regional and national dummies, and control for other co-variates, such as education, age and so on. Figure 7 shows us the rough correlation between the number of births in last five years and the local prevalence of HIV. The two curves are negatively correlated for low wealth levels. However, and consistent with the theory, as wealth increases, the association changes and turns positive. Table 11 provides similar patterns. In Column 1 to 5, we equally divided the sample into five groups based on wealth levels. HIV prevalence is negatively associated with fertility for the poorest 20% of families, which is in line with our hypothesis. Also consistent with the theory, coefficients are positive for other wealth groups, but here the coefficients are not (so) significant. We speculate this reflects modest variation in regional prevalence of HIV (relative to the sample size). In addition, we did several robustness checks (alternative grouping methods, Tobit and Probit models), and found that the associations are always robust.

Insert Table 10 approximately here.

Insert Figure 7 approximately here.

Insert Table 11 approximately here.

6 Discussion and Conclusion

The empirical literature contains a range of estimates of the impact of the HIV/AIDS epidemic on fertility in Africa – varying from negative to positive. One of the reasons for this embarrasing richness of results, we argue, might be a heterogeneous treatment effect. Specifically, we explore whether the fertility impact of HIV/AIDS may vary with African income levels. The mechanism we propose to link mortality to fertility, via income, is a so-called portfolio model of offspring in which households choose both the quantity and quality of their offspring. When households can choose between low-quality and high-quality children (distinguished by investments in human capital), aspiring households can adjust their portfolio across two margins in the face of a mortality shock – they can adjust their fertility as well as the quality of their children.

Our main result is that, for households within a certain income range (the so-called "intermediate income" household), a mortality shock indeed invites a two-fold revision of their portfolio. They will choose to have more children but invest less per child (i.e., they choose low rather than high quality offspring). Households who are either poorer or richer than these intermediate income households adjust their portfolio only across one margin and, interestingly, their fertility response may potentially offset the response of the intermediate income type. Specifically, because offspring represents a less valuable asset following the mortality shock, both poor and rich households will respond by reducing fertility, investing in risk-free assets instead. The overall fertility response, at the national level, therefore depends on aggregate income (as well as the distribution of income across the three types of households). Our empirical analysis is somewhat rough as it is mainly based on aggregate statistics (household-level income panel data are unfortunately not matching our HIV prevalence data well) and focuses only on average income levels within countries. Nevertheless, our empirical results provide support for the theoretical predictions.

Our estimations suggest that critical switching points are within the range of data of our sample. Specifically, we predict that mortality results in under-investment in human capital for a range of incomes exceeding some $6000 per year. Most African countries in our sample actually have average income levels that are (much) lower than this threshold. However, average income levels conceal a great diversity of income within African countries, so within most countries certain social groups (relatively privileged groups, to be sure) may already decide to under-invest in the education of their offspring because of the risk implied by such investments. Moreover, rapid economic growth across the African continent in recent years suggests that an increasing share of the population will gradually enter the income range where rational under-investment prevails. Large-scale investments in medicine and medicine-related infrastructure to reduce HIV/AIDS-related mortality may thus not only be advocated on humanitarian grounds – such efforts could also invite complementary private investments in human capital.

A Figures

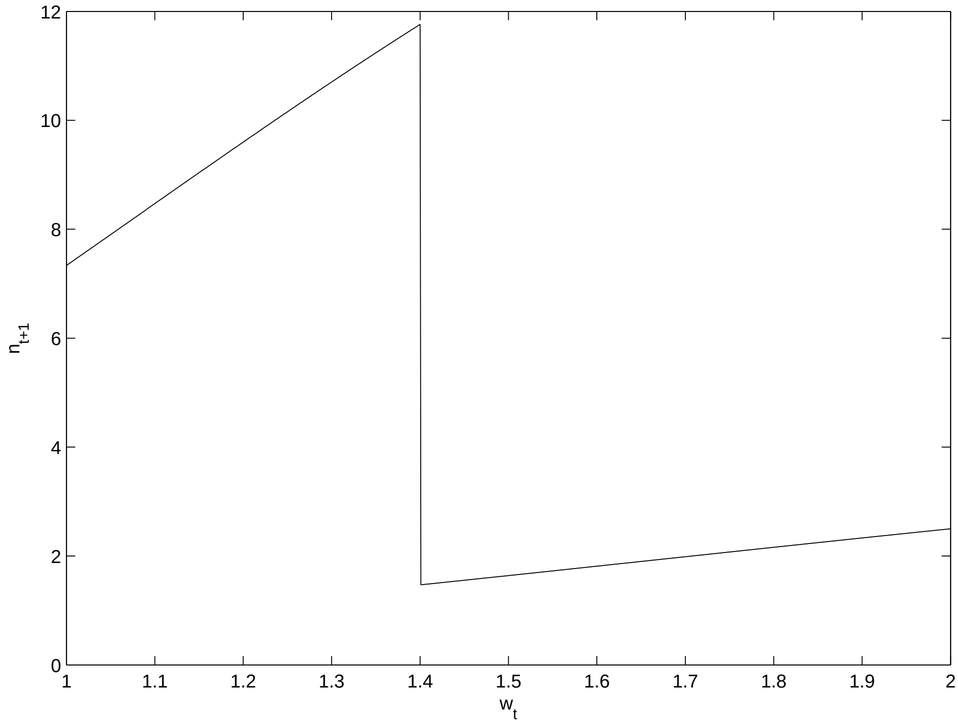

Figure 1: Number of children for agents with different wealth levels.

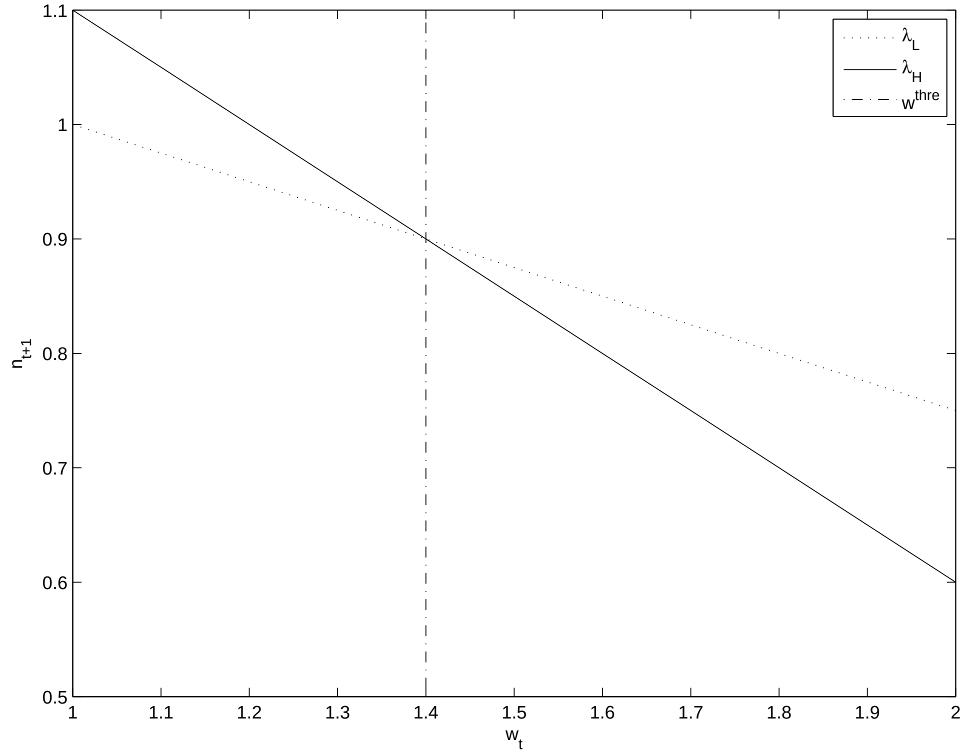

Figure 2: Sharpe ratios for different individual wealth. λ_H (dotted line) and λ_L (solid line) are Sharpe ratios for high-quality and low-quality children, respectively. w^{thre} is the critical wealth level for type-switching.

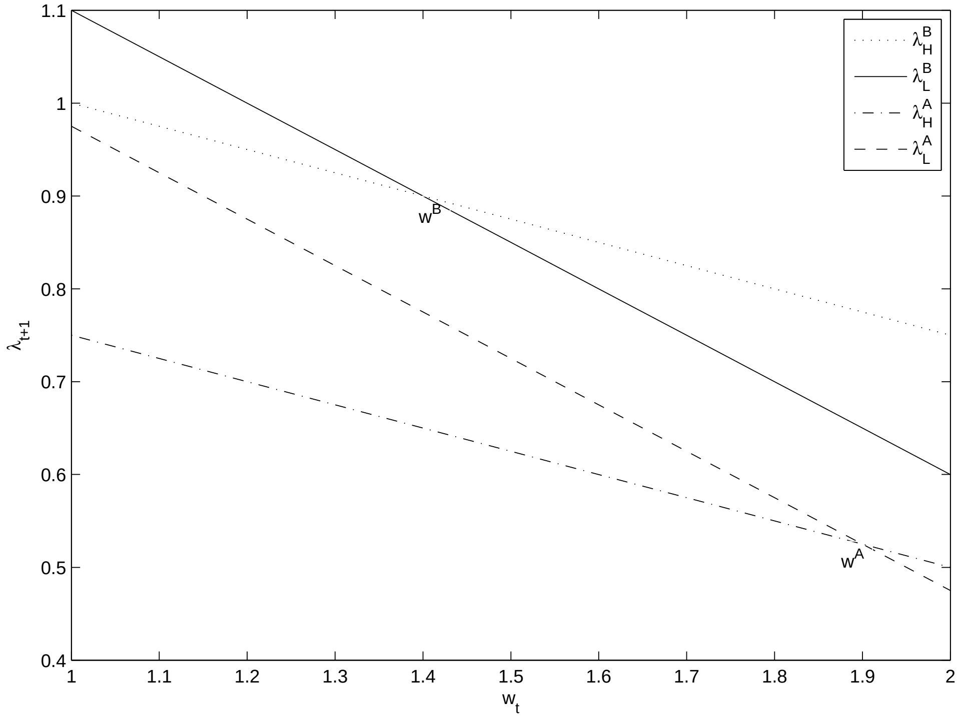

Figure 3: Sharpe ratios for different individual wealth and effect of mortality shock. λ_H^B (dotted line) and λ_L^B (solid line) are the Sharpe ratios for high-quality and low-quality children before the mortality shock. λ_H^A (dash-dotted line) and λ_L^A (dashed line) are the Sharpe ratios for high-quality and low-quality children after the mortality shock. w^B (w^A) is the critical individual wealth level for type-switching before (after) the mortality shock.

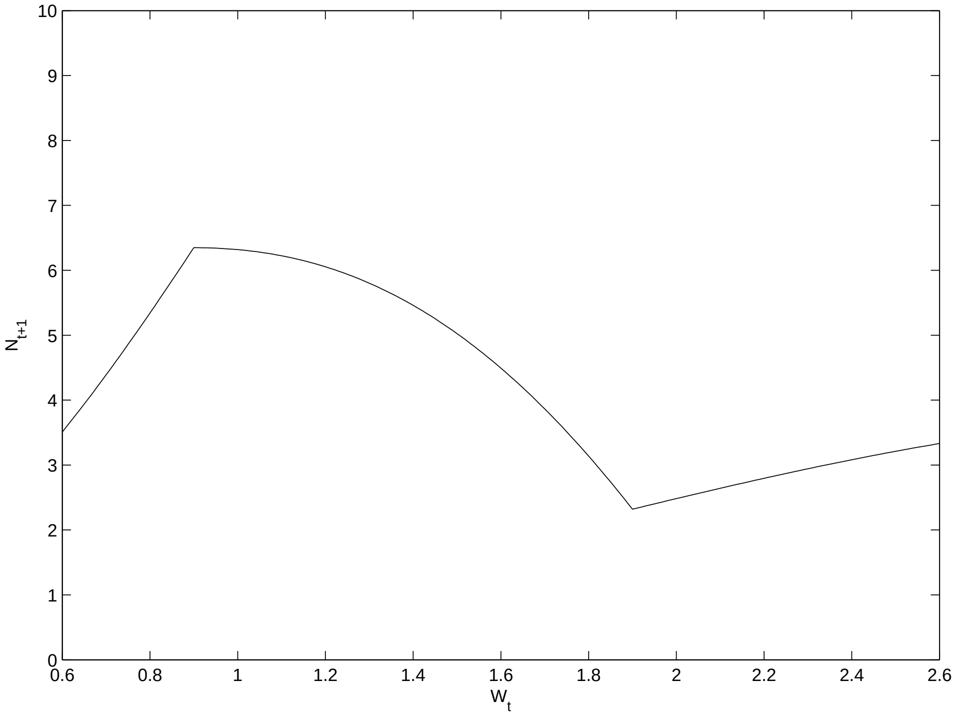

Figure 4: Aggregate fertility for countries with different wealth.

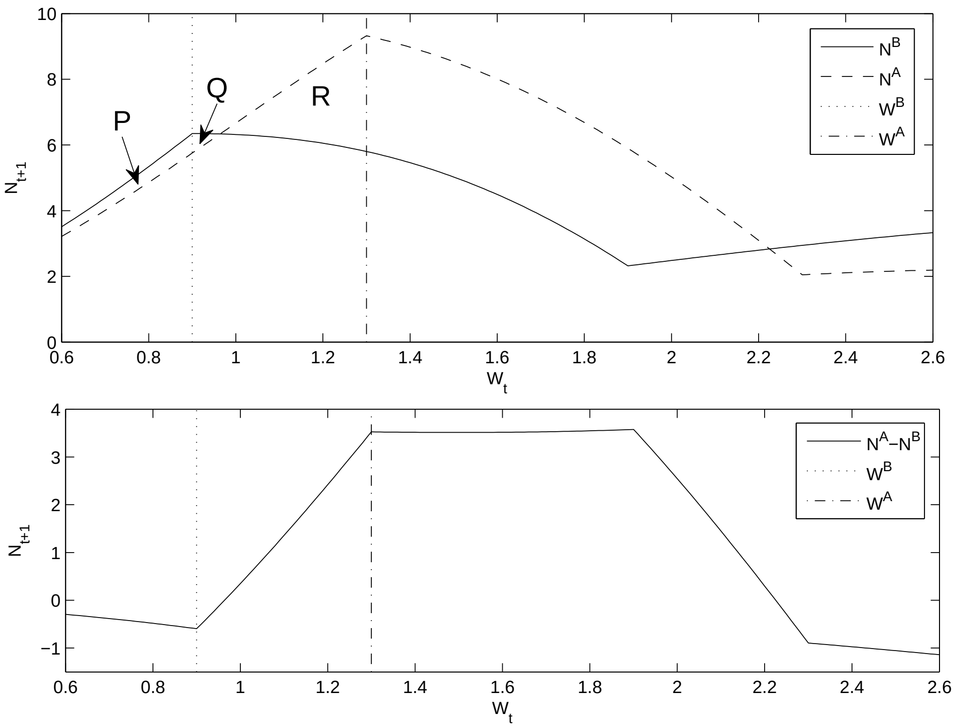

Figure 5: Aggregate fertility for countries with different wealth before and after the mortality shock. N^B (solid line) and N^A (dashed line) are the aggregate fertility before and after the mortality shock. W^B (dotted line) and W^A (dash-dotted line) are the critical aggregate wealth levels for type-switching before and after the mortality shock.

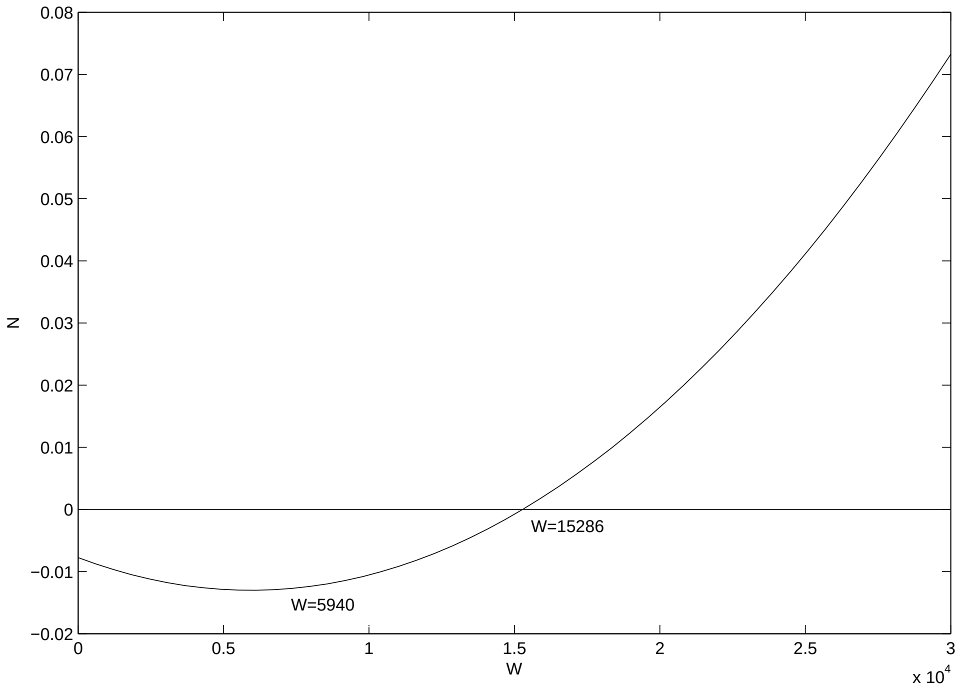

Figure 6: HIV effect on fertility changes with income rising (based on estimated parameters).

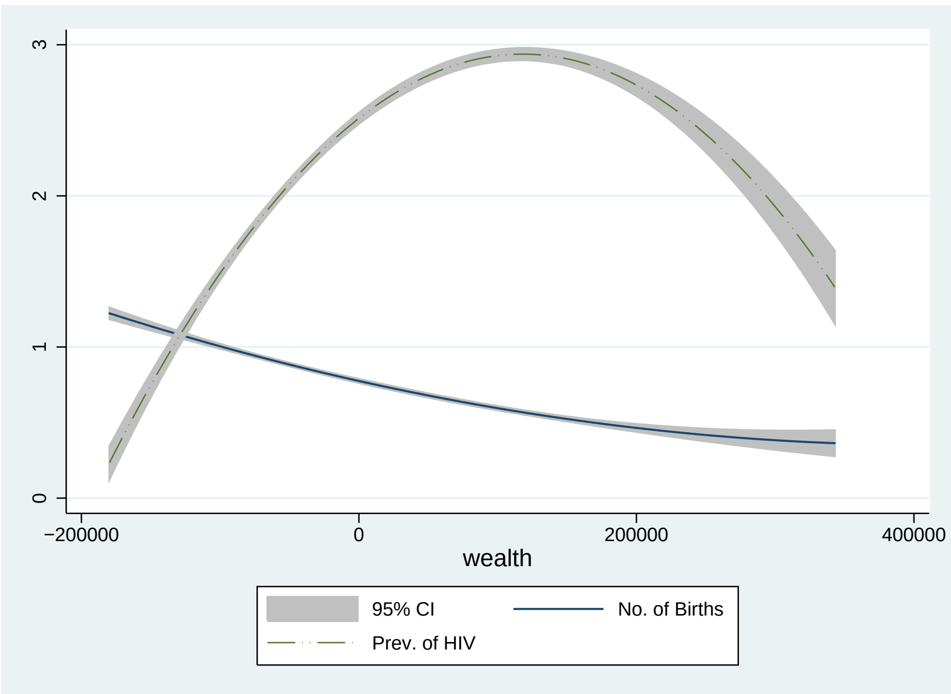

Figure 7: Association between number of birth and HIV for different wealth groups.

B Tables

Table 1: Summary of Literature

Ranking	Country	Juhn et al (2013)	Durevall et al (2011)	Kalemli-Ozcan et al (2011)	Fink et al(2008)	Kalemli-Ozcan (2012)	Fortson (2009)
1	Mozambique					✓	
2	Ethiopia	✓				✓	✓
3	Niger	✓				✓	✓
4	Eritrea						
5	Malawi	✓	✓			✓	✓
6	Mali					✓	✓
7	Uganda					✓	
8	Rwanda	✓				✓	✓
9	Burkina Faso	✓				✓	
10	Chad					✓	
11	Guinea	✓				✓	
12	Sierra						
13	Togo					✓	
14	Madagascar						
15	Tanzania					✓	✓
16	Guinea-Bissau						
17	Comoros						
18	Sudan						
19	Zambia					✓	✓
20	Ghana	✓			✓	✓	✓
21	Benin					✓	
22	Kenya	✓			✓	✓	✓
23	Senegal	✓			✓	✓	
24	Gambia						
25	Nigeria					✓	
26	Lesotho	✓					
27	Cote d' Ivoire	✓			✓	✓	✓
28	Mauritania						
29	Cape Verde						
30	Cameroon	✓			✓	✓	✓
31	Djibouti						
32	Congo						
33	Angola						
34	Namibia					✓	
35	Swaziland						
36	Mauritius						
37	Botswana						
38	South Africa			✓		✓	
39	Gabon					✓	
40	Equatorial						
Results	Individual	ambiguous	negative	positive	ambiguous		
	Country or Region				weak	ambiguous	weak

Table 2: Descriptive Statistics - Aggregate Data

Variable	Obs	Mean	Std. Dev.	Min	Max
tfr	1452	5.873623	1.165725	1.66	8.293
prev_hiv	792	5.078409	6.247504	0.1	27.3
low_hiv	792	4.433333	5.922549	0.1	25.8
high_hiv	792	5.859848	6.536551	0.1	28.6
inc	843	2561.663	3137.459	285.545	18359.15
prise	1161	80.73995	32.91283	13.3561	207.731
fprise	1106	73.91807	35.17613	10.13384	168.0079
mprise	1106	86.54604	31.08558	16.49015	176.9076
secse	907	24.49522	18.54848	1.65224	95.69964
u5mor	1342	153.4171	63.44948	15.6	339.2
infmor	1342	92.63867	32.08939	13.4	178.8

Table 3: Effect of HIV on Total Fertility Rate (I)

VARIABLES	(1)	(2)	(3)	(4)
inc	-4.81e-05***	7.19e-05***	-4.11e-05***	7.53e-05***
	(1.46e-05)	(1.58e-05)	(1.44e-05)	(1.57e-05)
$post\_90$	-0.0672***	-0.0852***	-0.0498***	-0.0712***
	(0.0163)	(0.0157)	(0.0158)	(0.0155)
$inc \times post\_90$	-5.09e-05***	-1.45e-05	-5.52e-05***	-1.91e-05**
	(8.97e-06)	(8.86e-06)	(8.76e-06)	(8.76e-06)
$inc^2 \times post\_90$	3.07e-09***	9.72e-10	3.40e-09***	1.25e-09*
	(6.96e-10)	(6.60e-10)	(6.82e-10)	(6.55e-10)
$prise$	-0.000825***		-0.000576**	
	(0.000296)		(0.000280)	
$u5mor$	0.000891***	0.000276		
	(0.000220)	(0.000183)		
inc^2	1.09e-09	-3.32e-09***	7.48e-10	-3.53e-09***
	(8.57e-10)	(8.43e-10)	(8.43e-10)	(8.36e-10)
$secse$		-0.00969***		-0.00939***
		(0.000546)		(0.000546)
$infmor$			0.00265***	0.00118***
			(0.000399)	(0.000351)
Observations	740	573	740	573

Dependent variable is $log(tfr)$, Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 4: Effect of HIV on Total Fertility Rate (II)

VARIABLES	(1)	(2)	(3)	(4)
inc	-1.24e-06	-0.000110***	-0.000106***	2.40e-06
	(1.96e-05)	(1.99e-05)	(1.99e-05)	(1.95e-05)
prev_hiv	-0.00776***	-0.0114***	-0.00999***	-0.00676***
	(0.00174)	(0.00206)	(0.00208)	(0.00173)
$inc \times prev\_hiv$	-1.77e-06**	-1.42e-06*	-1.66e-06**	-2.00e-06***
	(7.11e-07)	(8.22e-07)	(8.25e-07)	(7.09e-07)
$inc^2 \times prev\_hiv$	1.49e-10***	0	6.69e-11	1.69e-10***
	(5.68e-11)	(6.71e-11)	(6.76e-11)	(5.67e-11)
secse	-0.00757***			-0.00755***
	(0.000557)			(0.000545)
u5mor	0.00127***	0.00241***		
	(0.000187)	(0.000232)		
inc^2	-4.59e-10	5.77e-09***	5.56e-09***	-6.21e-10
	(1.13e-09)	(1.21e-09)	(1.20e-09)	(1.12e-09)
prise		-8.70e-05	-0.000207	
		(0.000300)	(0.000289)	
infmor			0.00439***	0.00249***
			(0.000417)	(0.000339)
Observations	370	495	495	370

Dependent variable is $log(tfr)$, Standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 5: Effect of HIV on Fertility for Countries Excluding the Poorest (I)

VARIABLES	(1)	(2)	(3)	(4)
inc	-1.55e-05**	1.86e-07	-1.34e-05**	4.46e-07
	(6.49e-06)	(8.19e-06)	(5.90e-06)	(7.30e-06)
prev_hiv	-0.0282***	-0.0293***	-0.0301***	-0.0320***
	(0.00282)	(0.00311)	(0.00255)	(0.00278)
inc×*prev_hiv*	1.06e-06***	3.78e-07	1.45e-06***	1.09e-06***
	(3.19e-07)	(3.68e-07)	(3.00e-07)	(3.51e-07)
secse	-0.00308***		-0.00237***	
	(0.000923)		(0.000852)	
u5mor	0.00471***	0.00641***		
	(0.000674)	(0.000727)		
prise		0.000669		0.000448
		(0.000597)		(0.000533)
infmor			0.00990***	0.0128***
			(0.00111)	(0.00114)
Observations	109	128	109	128

Dependent variable is $log(tfr)$, Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 6: Effect of HIV on Fertility for Countries Excluding the Poorest (II)

VARIABLES	(1)	(2)	(3)	(4)
$post\_90$	-0.171***	-0.131***	-0.166***	-0.118***
	(0.0423)	(0.0455)	(0.0424)	(0.0454)
inc	1.60e-06	-3.17e-05***	1.54e-06	-3.28e-05***
	(7.19e-06)	(7.09e-06)	(7.22e-06)	(7.04e-06)
$inc \times post\_90$	5.20e-06	-1.31e-05**	7.43e-06	-1.07e-05*
	(5.79e-06)	(5.67e-06)	(5.86e-06)	(5.77e-06)
$secse$	-0.00710***		-0.00726***	
	(0.00106)		(0.00106)	
$u5mor$	-0.00108	-0.00134		
	(0.000797)	(0.000915)		
$prise$		0.000217		-0.000280
		(0.000894)		(0.000894)
$infmor$			0.00121	0.00249
			(0.00153)	(0.00172)
Observations	165	191	165	191

Dependent variable is $log(tfr)$, Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 7: Effect of HIV on Human Capital

VARIABLES	log(fprise)	log(mprise)	log(prise)	log(fprise)	log(mprise)	log(prise)
inc	2.05e-05***	3.29e-05***	2.42e-05***			
	(5.07e-06)	(5.26e-06)	(5.37e-06)			
inc_{t-5}				1.23e-05	2.30e-05***	1.76e-05**
				(7.59e-06)	(8.00e-06)	(7.66e-06)
$post\_90_{t-5}$	-0.0693***	-0.0495***	-0.0499***	-0.0777***	-0.0579***	-0.0677***
	(0.0148)	(0.0153)	(0.0153)	(0.0182)	(0.0192)	(0.0180)
Observations	185	185	191	146	146	150

Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 8: Robustness Check (I)

VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
$post\_90$				-0.0687***			-0.159***		-0.0529***
				(0.0156)			(0.0438)		(0.0132)
$prev\_hiv$			-0.00598***					-0.0314***	
			(0.00176)					(0.00282)	
$inc \times post\_90$									-1.80e-05**
									(7.66e-06)
$inc^2 \times post\_90$									1.30e-09**
									(6.12e-10)
inc^2									-1.88e-09**
									(8.30e-10)
inc									5.04e-05***
									(1.49e-05)
inc_{t-1}	2.10e-05	7.53e-05***	3.17e-05	8.95e-05***	3.39e-06	-8.16e-06	6.71e-06	-1.13e-05*	
	(2.07e-05)	(1.59e-05)	(2.06e-05)	(1.65e-05)	(7.72e-06)	(6.58e-06)	(7.78e-06)	(6.59e-06)	
$prev\_hiv_{t-1}$	-0.00658***					-0.0326***			
	(0.00166)					(0.00259)			
$post\_90_{t-1}$		-0.0802***			-0.157***				
		(0.0154)			(0.0427)				
$inc_{t-1} \times prev\_hiv_{t-1}$	-2.44e-06***					2.01e-06***			
	(7.08e-07)					(3.26e-07)			
$inc_{t-1} \times post\_90_{t-1}$		-1.52e-05*			5.69e-06				
		(8.63e-06)			(5.89e-06)				
$inc_{t-1} \times prev\_hiv$			-2.44e-06***					1.63e-06***	
			(7.53e-07)					(3.38e-07)	
$inc_{t-1} \times post\_90$				-1.88e-05**			6.95e-06		
				(8.81e-06)			(6.03e-06)		
${inc^2}_{t-1} \times prev\_hiv_{t-1}$	2.13e-10***								
	(5.81e-11)								
${inc^2}_{t-1} \times post\_90_{t-1}$		9.72e-10							
		(6.49e-10)							
${inc^2}_{t-1} \times prev\_hiv$			2.08e-10***						
			(6.25e-11)						
${inc^2}_{t-1} \times post\_90$				1.13e-09*					
				(6.52e-10)					
$secse$	-0.00751***	-0.00911***	-0.00794***	-0.00969***	-0.00681***	-0.00332***	-0.00767***	-0.00254***	-0.00677***
	(0.000553)	(0.000559)	(0.000545)	(0.000548)	(0.00110)	(0.000914)	(0.00108)	(0.000921)	(0.000724)
$infmor$	0.00240***	0.00104***	0.00251***	0.00114***	0.00116	0.0103***	0.000983	0.0107***	0.000227
	(0.000338)	(0.000363)	(0.000339)	(0.000358)	(0.00156)	(0.00110)	(0.00161)	(0.00120)	(0.000541)
${inc^2}_{t-1}$	-1.63e-09	-3.43e-09***	-2.12e-09*	-4.10e-09***					
	(1.15e-09)	(8.58e-10)	(1.20e-09)	(8.82e-10)					
Observations	346	548	366	548	165	108	165	113	

Dependent variable is $log(tfr)$, Standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 9: Robustness Check (II)

VARIABLES	(1)	(2)	(3)	(4)
inc	1.34e-06	-3.21e-07	-1.59e-05**	-1.27e-05**
	(1.97e-05)	(1.89e-05)	(6.17e-06)	(5.20e-06)
high_hiv	-0.00499***		-0.0254***	
	(0.00163)		(0.00247)	
$inc \times high\_hiv$	-2.31e-06***		9.92e-07***	
	(6.72e-07)		(3.01e-07)	
$inc^2 \times high\_hiv$	1.80e-10***			
	(5.41e-11)			
low_hiv		-0.00712***		-0.0359***
		(0.00178)		(0.00248)
$inc \times low\_hiv$		-2.31e-06***		2.08e-06***
		(7.36e-07)		(2.83e-07)
$inc^2 \times low\_hiv$		1.99e-10***		
		(5.87e-11)		
secse	-0.00737***	-0.00732***	-0.00220**	-0.00282***
	(0.000560)	(0.000541)	(0.000902)	(0.000733)
inc^2	-6.23e-10	-5.25e-10		
	(1.14e-09)	(1.09e-09)		
infmor	0.00257***	0.00253***	0.00859***	0.0108***
	(0.000345)	(0.000336)	(0.00111)	(0.00100)
Observations	370	370	109	109

Dependent variable is $log(tfr)$, Standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 10: Descriptive Statistics - Individual Data

Variable	Obs	Mean	Std. Dev.	Min	Max
birth5	31114	0.7735746	0.8876717	0	6
birth1	31114	0.1783763	0.3931079	0	3
total birth	31114	2.726168	2.806152	0	17
prev_hiv	31114	2.293241	2.517678	0	7.2
age	31114	15.89002	14.63769	0	49
educ	31114	23.59112	13.94372	1	46
urban	31114	0.3660089	0.4817197	0	1
living children	31114	1.618063	1.894924	0	13
wealth	31114	13307.58	100924.9	-179749	342939

Table 11: The Effect of HIV on Fertility - Individual level

VARIABLES	(1) richest	(2) richer	(3) middle	(4) poorer	(5) poorest
prev_hiv	0.00306	0.00465	0.000792	0.00332	-0.0322***
	(0.00459)	(0.00521)	(0.00697)	(0.00939)	(0.0123)
age	-0.00609***	-0.00489***	-0.00611***	-0.00546***	-0.00937***
	(0.000624)	(0.000599)	(0.000631)	(0.000750)	(0.00126)
educ	-0.000169	-0.000132	-0.000444	-0.000815**	-0.00325***
	(0.000297)	(0.000326)	(0.000362)	(0.000408)	(0.000455)
urban	0.0324**	0.0177	-0.00347	-0.0321	-0.104**
	(0.0162)	(0.0140)	(0.0129)	(0.0211)	(0.0432)
livingchildren	0.0506***	0.0417***	0.0401***	0.0353***	0.0667***
	(0.00329)	(0.00308)	(0.00303)	(0.00342)	(0.00491)
wealth	-2.22e-07**	1.32e-07	-4.21e-07*	-3.74e-07	-1.68e-07
	(1.01e-07)	(2.36e-07)	(2.31e-07)	(3.23e-07)	(4.74e-07)
national dummies					
regional dummies					
Observations	6,226	6,222	6,222	6,222	6,222

Dependent variable is number of births last year, Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 12: Benchmark Parameter Values

Parameters	Description	Values
a	Risk-free rate parameter	0
b	Risk-free rate parameter	0.05
c	Ratio of mortality shocks to two types of children	4
d	Risk aversion parameter	3
μ_H^B	Mean return on high type children before mortality shocks	0.25
μ_L^B	Mean return on low type children before mortality shocks	0.16
σ_H	Volatility (riskiness) of high type children	0.2
σ_L	Volatility (riskiness) of low type children	0.1
m_L	Mortality shocks to mean return on low type children	0.05
C_H	Cost of high type children	2
C_L	Cost of low type children	0.5

C Parameter Values

All of the figures are made using the parameter values shown in Table 12.

D Conditions for Local Maxima at $\bar{w} = w^B$

The number of children associated with the aggregate wealth level of W^B, $N^{B,thre}$, is given by,

$$N^{B,thre} = \frac{1}{dC_L\sigma_L^2}\int_{w^A-1}^{w^A} w^2(\mu_L - a - bw)\mathrm{d}w. \tag{36}$$

As any country wealthier than W^B has both types of children, the number of children associated with $W^B + \Delta w$ is,

$$\begin{aligned} N^B(W^B + \Delta w) = &\frac{1}{dC_L\sigma_L^2}\int_{w^B-1+\Delta w}^{w^B} w^2(\mu_L - a - bw)\mathrm{d}w \\ &+ \frac{1}{dC_H\sigma_H^2}\int_{w^B}^{w^B+\Delta w} w^2(\mu_H - a - bw)\mathrm{d}w. \end{aligned} \tag{37}$$

To ensure the local maxima is obtained at $\bar{w} = w^B$, the difference between $N^{B,thre}$ and $N^B(W^B + \Delta w)$ must be negative,

$$N^{B,thre} - N^B(W^B + \Delta w) = \frac{1}{dC_L\sigma_L^2}\int_{w^B-1}^{w^B-1+\Delta w} w^2(\mu_L - a - bw)\mathrm{d}w - \frac{1}{dC_H\sigma_H^2}\int_{w^B}^{w^B+\Delta w} w^2(\mu_H - a - bw)\mathrm{d}w > 0. \tag{38}$$

From Equation (38), we have,

$$C_H > lC_L, \tag{39}$$

where

$$\begin{aligned} l &= \lim_{\Delta\to 0}\frac{\sigma_L^2}{\sigma_H^2}\frac{\int_{w^B}^{w^B+\Delta w} w^2(\mu_H - a - bw)\mathrm{d}w}{\int_{w^B-1}^{w^B-1+\Delta w} w^2(\mu_L - a - bw)\mathrm{d}w} \\ &= \frac{\sigma_L^2}{\sigma_H^2}\frac{(\mu_H - a)(w^B)^2 - b(w^B)^3}{(\mu_L - a)(w^B-1)^2 - b(w^B-1)^3}. \end{aligned} \tag{40}$$

E Conditions for Intersection of N^A and N^B

E.1 Case 1: $w^B > w^A - 1$

In this case, the country with wealth of W^A has both types of children. Therefore, $N^{B,A}$ is given by,

$$\begin{aligned} N^{B,A} &= \frac{1}{dC_L\sigma_L^2}\int_{w^A-1}^{w^B} w^2(\mu_L - a - bw)\mathrm{d}w + \frac{1}{dC_H\sigma_H^2}\int_{w^B}^{w^A} w^2(\mu_H - a - bw)\mathrm{d}w \\ &= \frac{1}{dC_L\sigma_L^2}\left[\frac{\mu_L - a}{3}((w^B)^3 - (w^A-1)^3) - \frac{b}{4}((w^B)^4 - (w^A-1)^4)\right] \\ &\quad + \frac{1}{dC_H\sigma_H^2}\left[\frac{\mu_H - a}{3}((w^A)^3 - (w^B)^3) - \frac{b}{4}((w^A)^4 - (w^B)^4)\right]. \end{aligned} \tag{41}$$

From Equation (17), we have:

$$m_L = ew^A + f, \tag{42}$$

where

$$e = \frac{b(\sigma_H - \sigma_L)}{c\sigma_L - \sigma_H} > 0, \tag{43}$$

$$f = \frac{a(\sigma_H - \sigma_L) - \sigma_H \mu_L + \sigma_L \mu_H}{c\sigma_L - \sigma_H}. \tag{44}$$

Equation (42) implies that m_L can be expressed as an increasing linear function of w^A. Using this relation and the condition $N^{A,thre} > N^{B,A}$, we obtain:

$$g_1(w^A)^4 + h_1(w^A)^3 + i_1(w^A)^2 + j_1 w^A + k_1 > 0, \tag{45}$$

where

$$g_1 = -\frac{3b}{4}\left(1 - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}\right) < 0, \tag{46}$$

$$h_1 = (\mu_L - a) - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}(\mu_H - a) - 3e, \tag{47}$$

$$i_1 = 3(e - f), \tag{48}$$

$$j_1 = 3f - e, \tag{49}$$

$$k_1 = -f + \left[\frac{C_L\sigma_L^2}{C_H\sigma_H^2}(\mu_H - a) - (\mu_L - a)\right](w^B)^3 + \left[\frac{3b}{4}\left(1 - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}\right)\right](w^B)^4. \tag{50}$$

Equation (45) is a quartic inequality with a negative coefficient of the highest order term and has a discriminant given by,

$$\begin{aligned}\Delta = {} & 256g_1^3k_1^3 - 192g_1^2h_1j_1k_1^2 - 128g_1^2i_1^2k_1 + 144g_1^2i_1j_1^2k_1 - 27g_1^2j_1^4 + 144g_1h_1^2i_1k_1^2 \\ & - 6g_1h_1^2j_1^2k_1 - 80g_1h_1i_1^2j_1k_1 + 18g_1h_1i_1j_1^3 + 16g_1i_1^4k_1 - 4g_1i_1^3j_1^2 - 27h_1^4k_1^2 \\ & + 18h_1^3i_1j_1k_1 - 4h_1^3j_1^3 - 4h_1^2i_1^3k_1 + h_1^2i_1^2j_1^2.\end{aligned} \tag{51}$$

We assume $\Delta < 0$, which implies that the associated equation has two real roots and two complex conjugate roots. Thus, we have:

$$w_l^{thre} < w^A < w_u^{thre}, \tag{52}$$

where w_l^{thre} and w_u^{thre} are the two real roots. The linear relationship between w^A and m_L ensures that there exist m_l^{thre} and m_u^{thre} such that,

$$m_l^{thre} < m_L < m_u^{thre}. \tag{53}$$

E.2 Case 2: $w^B \leq w^A - 1$

In this case, the country with wealth of W^A only has high type children. Therefore, $N^{B,A}$ is given by,

$$\begin{aligned} N^{B,A} &= \frac{1}{dC_H\sigma_H^2}\int_{w^A-1}^{w^A} w^2(\mu_H - a - bw)\mathrm{d}w \\ &= \frac{1}{dC_H\sigma_H^2}\left[\frac{\mu_H - a}{3}((w^A)^3 - (w^A-1)^3) - \frac{b}{4}((w^A)^4 - (w^A-1)^4)\right]. \end{aligned} \tag{54}$$

Once again, using the linear relation between m_L and w^A and the condition $N^{A,thre} > N^{B,A}$, we obtain:

$$g_2(w^A)^3 + h_2(w^A)^2 + i_2(w^A) + j_2 > 0, \tag{55}$$

where

$$g_2 = -3b\left(1 - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}\right) - 3e < 0, \tag{56}$$

$$h_2 = 3\left[(\mu_L - a) - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}(\mu_H - a)\right] + \frac{9b}{2}\left(1 - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}\right) - 3(f - e), \tag{57}$$

$$i_2 = -3\left[(\mu_L - a) - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}(\mu_H - a)\right] - 3b\left(1 - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}\right) - (e - 3f), \tag{58}$$

$$j_2 = \left[(\mu_L - a) - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}(\mu_H - a)\right] - \frac{3b}{4}\left(1 - \frac{C_L\sigma_L^2}{C_H\sigma_H^2}\right) - f, \tag{59}$$

Equation (55) is a cubic inequality with a negative coefficient of the highest order term and has a discriminant given by,

$$\Delta = 18g_2h_2i_2j_2 - 4h_2^3j_2 + h_2^2i_2^2 - 4g_2i_2^3 - 27g_2^2j_2^2, \tag{60}$$

We assume $\Delta < 0$, which implies that the associated equation has one real roots and two complex conjugate roots. Thus, we obtain:

$$w^A < w^{thre}, \tag{61}$$

where w^{thre} is only one real root. The linear relation between w^A and m_L ensures that there exists m^{thre} such that

$$m_L < m^{thre}. \tag{62}$$

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