

# Uncertain Child Survival and Fertility: The Effects of Child Morbidity and Mortality

Anna-Maria Aksan<sup>1</sup>  
Department of Economics  
University of Oregon

September 2009

<sup>1</sup>Address: Department of Economics, 1285 University of Oregon, Eugene, OR 97403-1285. Email: aaksan@uoregon.edu. For our many helpful conversations I thank Shankha Chakraborty. For their helpful comments and suggestions, I also thank Bruce Blonigen, Christopher Ellis, Peter Lambert, Laura Leete and Jean Stockard. All errors are my own.

## **Abstract**

This paper analyzes the response of fertility and human capital investment to changes in childhood disease burden. Unlike previous studies, it focuses on disease-related mortality and distinguishes between disease morbidity and mortality. When child survival is uncertain, a fall in childhood disease burden may lower or raise total fertility in contrast to the unambiguously positive relationship in other studies. The fertility response depends on the type of disease (the severity of its long-term effects), the level of disease burden, and whether the disease burden falls due to lower infection or case fatality rates. Evidence from panel data on malaria and HIV/AIDS spanning 1985-2000 for 44 African countries supports a nonlinear response of fertility to disease prevalence. A negative response is more likely where child mortality rates are moderate and where the morbidity effect of the disease is milder. These results reconcile some of the conflicting results from multiple studies on the impact of HIV/AIDS on fertility in Africa.

Keywords: Child Mortality, Morbidity, Fertility, Demography, Population, Health Production, Productivity, Economic Development

JEL Classification Numbers: I12, J11, J13

# 1 Introduction

This paper studies the response of fertility and human capital investment to changes in childhood disease burden. It builds upon the work of Sah (1991) and Kalemli-Ozcan (2003, 2008, 2009), focusing on disease-caused child mortality and distinguishing between the contributions of disease prevalence and disease severity to overall disease burden.

If the long-term health consequences of a disease are severe, falling infection rates reduce morbidity, while falling case fatality rates amplify morbidity as more unhealthy individuals survive disease episodes. Unlike Kalemli-Ozcan's work and other mortality-fertility studies that find an unambiguously positive response of total fertility rates (TFR) to changes in child mortality, total fertility may rise or fall in response to changes in disease burden. The result depends on the type of disease (the severity of the disease's long-term effects), the level of disease burden (prevalence and case fatality), and whether the decline in child mortality is due to declines in infection rates or in case fatality rates.

Panel-based regression results using data from 44 countries in sub-Saharan Africa for 1985-2005 confirm the nonlinear response of fertility to disease prevalence. The overall predicted fertility response to disease prevalence tends to be positive for observations with the highest child mortality rates and for diseases with the most severe morbidity effects. For example, the predicted fertility response is positive for more observations in the case of HIV than malaria. Regressions of elementary education reinforce the results, and fewer children are educated in areas exposed to greater morbidity. Moreover, empirical results regarding HIV reconcile conflicting conclusions from previous studies of the impact of the AIDS epidemic on fertility.<sup>1</sup>

Historical population changes were triggered by declines in mortality rates, largely attributed to declines in disease burden and generally followed by declines in fertility. In England, whose experience represents that of many Western countries, an abrupt decline in deaths from infectious diseases began around 1872 and was followed approximately five years later with declines in total and net fertility rates. The net fertility rate began a period of recovery around 1950, when the decline in infectious disease mostly tapered off (Arora 2003).

Where child mortality rates are high, parents may intentionally have more children than they ultimately desire, because some children are likely not to survive to adulthood. They may have even more children than the average child mortality rate would suggest due to a strong aversion to ending up with too few. For those families where more

---

<sup>1</sup>When replicating Kalemli-Ozcan's specification, the negative and insignificant coefficient capturing the impact of HIV prevalence on TFR in that study becomes significant when the country set and time span of the data are expanded.

than an average number of children survive, resources for education and other human capital investment become strained, thwarting economic progress. I explicitly account for the uncertainty parents face in a high-disease environment by implementing the Delta method, which allows me to incorporate the variance of child survival outcomes into the parent's optimization problem. Models that incorporate uncertainty into the agent's utility maximization problem (Sah 1991, Kalemli-Ozcan 2003, 2008, Tamura 2006) show that higher child mortality increases fertility and reduces quality investment per child. By distinguishing between the contribution of infection and case fatality rates to overall child mortality, the model shows fertility can either increase or decrease in response to changes in child disease burden when child survival is uncertain.

This paper demonstrates how morbidity also affects fertility decisions. Acquiring and surviving a disease in early childhood (even if one is cured) may hamper cognitive or physical development or predispose one to other diseases later in life.<sup>2</sup> A new antibiotic reduces case fatality rates but not infection rates, and if the illness causes long-term cognitive or health problems, the result is a proportionally less healthy population.<sup>3</sup> On the other hand, a vaccine reduces infection rates, and the population becomes proportionally healthier.

Economically, children may be important as a future source of income for parents in old age. Children who suffer many diseases during childhood may grow up to be less productive and may have lower quality of life. If this situation affects most children, parents supplement the lower quality of their children by increasing their quantity. Thus if deaths from disease decline but morbidity, or disease prevalence, remains high, then high fertility rates persist and resources for human capital investment remain low. In the model below, I focus on the potential disease has to lower children's future productivity. There is heterogeneity in human capital investment, and parents invest more in the human capital of their healthier children.

Historically both total and net fertility rates have fallen, the former due to a decline in the precautionary motive for children and the latter as parents become less likely to overshoot their target number of children and also choose to have fewer children but invest

---

<sup>2</sup> The impact on health capital of surviving disease is highlighted in Arora (2003, 2005). Bronchitis, pneumonia and whooping cough before age 5 are linked to diminished respiratory function at ages 59-70 (Barker 1992, 1994). Acute rheumatic fever, syphilis, measles, typhoid and malaria can affect the functioning of the circulatory system (Khosla 1981). The human papillomavirus is the primary cause of cervical cancer. Infections that disrupt absorption, such as diarrhoea, deprive the body of nutrients necessary for optimal cellular growth (Martorell 1980, Martorell and Habicht 1986, Mata 1978).

<sup>3</sup>The antibiotic could technically reduce infection rates by reducing the time during which infected individuals are contagious, but this is a relatively unimportant effect since infection rates are generally tackled via prevention, through vaccines or behavioral changes, for example.

more in the quality of each child. In their seminal paper, Barro and Becker (1988) show that a decline in child mortality reduces the average fixed cost of raising a surviving child, so parents choose to have more children. This rise in demand for children is temporary and net fertility returns to its original level (unless mortality continues to decline), but because of lower mortality rates fewer births are required to achieve the target number of survivors. Birth rates may initially rise if the increase in demand for children is stronger than the decline in precautionary births. The Barro-Becker model generates an inverted U-shaped population path when mortality declines. Adaptations of the Barro-Becker model show an unambiguously positive response of TFR to mortality; any negative relationship resulting from the average cost effect is completely overshadowed by positive precautionary forces.<sup>4</sup> My model accounts for the Barro-Becker average cost effect and determines the circumstances in which it dominates the forces (precautionary demand for children and the quantity-quality tradeoff) contributing to a positive response of fertility to child mortality. By separating infection and case fatality rates, I introduce an additional but *positive* average cost effect that functions specifically through changes in morbidity and counteracts the negative average cost effect when infection rates decline but amplifies it when case fatality rates decline. As in Barro and Becker, my model also generates a U-shaped population path, but only when the decline in disease burden reduces child mortality sufficiently.<sup>5</sup>

Birchenall (2007) is an exception to previous mortality-fertility studies in that he distinguishes between infection and case fatality rates. Disease burden is endogenous, whereas here it is exogenous. Adult health depends on disease outcomes in childhood because the adult infection rate, which is equivalent to the proportion of adults who survived disease during childhood, determines labor productivity. This is a proxy for adult health and hence for the impact of childhood disease on adult health capital. In the model below, children's health capital depreciates explicitly if they survive a disease, and that depreciated health capital affects labor productivity in adulthood directly and also indirectly by affecting parental investment in the children's human capital. I study the consequences of changes in the degree of this depreciation and its impact on the nonlinear relationship between fertility and infection and case fatality rates.

According to UNICEF under-five mortality has declined globally by 50% between 1960 and 2002, and this has coincided with a decline in TFR. Yet the disease burden remains high, and many countries have yet to complete, or even begin, the transition to

---

<sup>4</sup>Among such studies are Boldrin and Jones (2002), Cervellati and Sunde (2007), Galor and Weil (1999), Kalemli-Ozcan (2003, 2008), Soares (2005), Tamura (2006).

<sup>5</sup>In Doepke (2005), Kalemli-Ozcan (2003, 2008) and Boldrin and Jones (2002), the reduction in TFR when mortality rates decline is initially weak, so net fertility rises before it declines from its original level.

low fertility. In industrialized countries women have between one and two children and child mortality rates average 0.6% and are due mostly to perinatal conditions, while in developing countries child mortality rates are as high as 26% in Sierra Leone, and women in Niger have up to eight children on average (CIA World Factbook, World Bank). As seen in Table 1, infectious diseases comprise 7 of the top 10 causes of child mortality in developing countries; pneumonia, diarrhoea, malaria, measles and AIDS account for half of all deaths among children under the age of five.

If reducing childhood disease leads to fertility decline and growing human capital, targeting the disease burden can stimulate economic development. A reduction in disease burden may be achieved by reducing disease prevalence, disease severity, or both. This paper shows that health initiatives in developing countries may have unintended consequences for fertility, depending on how diseases are combated. A declining disease burden will not necessarily reduce mortality and thus fertility unless it also reduces morbidity.

The AIDS epidemic has raised both child and adult mortality rates and may thus be reversing the general decline of TFR in African countries observed since 1960. Figures 3 and 4 depict TFR and HIV prevalence for Rwanda, South Africa, Tanzania and Zimbabwe for 1985-2005. An obvious relationship between HIV and fertility does not emerge from these data, and empirically, a consensus is lacking on the direction of the fertility response to AIDS. Young (2005) concludes that the fertility response to HIV is unambiguously negative, while Kalemli-Ozcan (2009) concludes that it is positive for other measures of the AIDS epidemic, although her regressions are less conclusive for the particular HIV prevalence measure employed here. Juhn, Kalemli-Ozcan and Turan (2009) conclude that there is no clear impact of local community HIV prevalence on TFR of HIV-negative women. In support of Young (2005), Boucekkine, Desbordes and Latzer (2008) compare the theoretical response of fertility to child and adult mortality rates and conclude that the AIDS epidemic is decreasing fertility in Africa because it affects adult rather than child mortality.<sup>6</sup> However, the AIDS burden among children in Africa is significant, as seen in Tables 1 and 2. This paper concludes that HIV tends to increase TFR where child mortality rates are higher and adult mortality rates are lower.

The paper proceeds as follows. I develop and solve the theoretical model in Section 2 and analyze the results in Section 3. In Section 4 I empirically test the model's main predictions. I discuss the results and policy implications in Section 5, and Section 6 concludes and considers possible extensions.

---

<sup>6</sup> The negative effect of adult mortality on TFR, which functions through wage effects resulting from a diminished labor supply, dominates in the long-run the positive effect of child mortality, which functions through the precautionary motive for children.

## 2 The Model

The two-period model is adapted from Kalemli-Ozcan (2003, 2008), where parents choose how many children to have and then invest in the human capital of children who survive infancy. Rather than an aggregate child mortality rate, the model here distinguishes between disease infection and case fatality rates.<sup>7</sup>

Young adults work and raise children, and in old age parents retire and consume a fraction of their adult children's income. Children are born with human capital endowment  $h_o$  and can get infectious disease in early childhood which they either survive or do not. On average  $i$  fraction of children get a disease, and of those  $(1 - d)$  fraction survive, where  $i, d \in (0, 1)$ . Of the  $n$  children born,  $N_1$  children avoid disease completely,  $N_2$  get a disease but survive, and  $N_3$  get a disease and die. Surviving a disease depreciates the human capital endowment,  $h_o$ , by a fraction  $\delta$ . (See Footnote 2.) Once children reach school age (5 years old), parents choose how much to invest in the quality of each child (education or health care investment, for example). Parents can invest differently in different types of children,  $h_1$  in healthy  $N_1$  type children and  $h_2$  in relatively unhealthy  $N_2$  type children. The production function for children's human capital is  $h_o^\alpha h_1^\theta$  for children who were never sick, and  $[(1 - \delta)h_o]^\alpha h_2^\theta$  for children who were sick, where  $\alpha, \theta \in (0, 1)$ . I normalize  $h_o$  to 1.

Young adults earn wage  $w$  per effective unit of labor, and they earn more if they are more productive. Labor productivity of a young adult is denoted by  $H$ , which depends on disease exposure during childhood:

$$H = \begin{cases} h_{o,t-1} \overline{h_1}^{-\theta} & \text{if not sick as child;} \\ (h_{o,t-1}(1 - \delta))^\alpha \overline{h_2}^{-\theta} & \text{if sick as child.} \end{cases}$$

$\overline{h_1}$  and  $\overline{h_2}$  are human capital investments made last period. Adults work and choose consumption,  $c$ , and the number of children they have,  $n$ . Once disease realizations of children are known, parents choose investment in each type of surviving child,  $h_1$  and  $h_2$ . In retirement they consume  $c'$ , which consists of  $\tau$  fraction of their adult children's earnings, where  $\tau$  is an exogenous parameter dictated by social norms, for example.<sup>8</sup> An adult parent maximizes

---

<sup>7</sup>Kalemli-Ozcan (2003, 2009) and Sah (1991) allow for a single child mortality rate, while Tamura (2006) separates infant and child mortality rates. In Kalemli-Ozcan parents invest in children after their survival outcomes are resolved, while in Tamura parents invest the same quantity in each child at birth. Here, parents invest in children after survival outcomes are known, and they can invest differently in children who survived disease and those who never contracted a disease. See below.

<sup>8</sup>While  $\tau$  is exogenous here, in Boldrin and Jones (2002) endogenous donations from children support parents in old age, but there is no human capital investment in children.

$$\beta \ln(c) + (1 - \beta) \ln(c')$$

subject to

$$\begin{aligned} c &\leq (1 - \gamma n)wH - N_1 h_1 - N_2 h_2 \\ c' &\leq \tau w' (N_1 h_1^\theta + N_2 (1 - \delta)^\alpha h_2^\theta) \end{aligned}$$

where  $\gamma \in (0, 1)$  is the fixed time cost of having a child, and the remainder of adults' time is spent working.  $\beta \in (0, 1)$  is the rate of time preference, that is, the weight adults place on consumption now versus consumption during retirement.  $w$  and  $w'$  are wage per effective unit of labor earned, respectively, by parents today and by adult children tomorrow.

Adults face uncertainty in the number of surviving children, but parental human capital investment in children occurs at age five after uncertainty about their survival is resolved. To incorporate the uncertainty of child survival outcomes, I use the Delta method, a second-order Taylor series approximation of the utility function around its mean that also converts the discrete fertility choice into a continuous representation. There are three, rather than two possible outcomes for each child born. The number and type of survivors is a random draw from a discrete multinomial distribution:

$$P(N_1 = n_1, N_2 = n_2, N_3 = n_3) = \frac{n!}{n_1! n_2! n_3!} P(n_1) P(n_2) P(n_3)$$

where  $N_1 + N_2 + N_3 = n$ . Of the  $n$  children born, on average  $N_1 = n(1 - i)$  children avoid disease,  $N_2 = ni(1 - d)$  children survive disease, and  $N_3 = nid$  children die from disease.

To see the importance of using the Delta method, I first solve the model without it and get results counter to historical trends.

## 2.1 Optimal Choices Under Certainty

When uncertainty about how many children will survive is not included in the model, parents make decisions assuming  $N_1 + N_2 = n(1 - id)$ , and they choose  $h_1, h_2, n$  to maximize

$$\beta \ln((1 - \gamma n)wH - n[(1 - i)h_1 + i(1 - d)h_2]) + (1 - \beta) \ln(\tau n w' [(1 - i)h_1^\theta + i(1 - d)(1 - \delta)^\alpha h_2^\theta])$$

and first order conditions yield:

$$\begin{aligned}
 n^* &= \frac{(1-\beta)(1-\theta)}{\gamma} \\
 c^* &= \beta w g(H) \\
 h_1^* &= \frac{\theta \gamma w g(H)}{(1-\theta)(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})} \\
 h_2^* &= (1-\delta)^{\frac{\alpha}{1-\theta}} h_1^*
 \end{aligned}$$

Under certainty, fertility is unaffected by the infection and case fatality rates, while human capital investment unambiguously *increases* with disease burden because investment is inversely related to number of survivors.<sup>9</sup> These results contradict historical data from demographic transitions in which declining mortality rates are associated with falling fertility and growing human capital investment. Furthermore, it can be shown that the net effect on total human capital investment when  $i$  or  $d$  change is exactly zero under certainty; the increase in survivors due to a decline in mortality exactly counteracts the decline in human capital investment per child. See Appendix B.1 for further details.

In order to explain a positive relationship between mortality and fertility and the ensuing negative relationship between both and child quality, Becker and Lewis (1973) show that the shadow price of an additional child (unit of child quality investment) must be an increasing function in quality (the number) of children. Here, however, this is not a sufficient condition.

## 2.2 Optimal Choices Under Uncertainty

Under uncertainty about child survival outcomes, fertility choice,  $n$ , does depend on childhood disease burden. Children are most susceptible to infectious disease during the first few years of life, and during the demographic transition the largest gains in longevity occur amongst infants and children.<sup>10</sup> Therefore, I assume that uncertainty about child survival is resolved after infancy and that human capital investment occurs after this time. As in Kalemli-Ozcan (2008), I use backwards induction to first solve for optimal human capital investment given  $(n, N_1, N_2)$  and then maximize expected utility, evaluated at

---

<sup>9</sup>Kalemli-Ozcan (2003, 2008) also obtains this result under certainty, and under constant relative risk aversion, TFR actually increases in response to lower mortality. In Boucekkine, Desbordes and Latzer (2008), who include a labor-leisure tradeoff, fertility declines with mortality despite constant relative risk aversion and certainty about child survival. TFR moves with mortality in Strulik (2008), despite certainty and logarithmic preferences; uniform investment in infants increases the probability of child survival and also enters parents' utility directly.

<sup>10</sup>Infants are particularly susceptible to infectious disease approximately 6-8 months after birth, when they begin to lose their mother's immunities but have yet to develop their own.

$c^*(n)$ ,  $h_1^*(n)$ , and  $h_2^*(n)$ , with respect to  $n$  under uncertainty.

Adults choose  $h_1$ ,  $h_2$  and  $c$  to maximize utility

$$\beta \ln((1 - \gamma n)wH - N_1 h_1 - N_2 h_2) + (1 - \beta) \ln(\tau w'(N_1 h_1^\theta + N_2 h_2^\theta (1 - \delta)^\alpha))$$

and the first order conditions yield:

$$\begin{aligned} h_1^*(n) &= \frac{\theta(1 - \beta)(1 - \gamma n)wg(H)}{(\beta + \theta(1 - \beta))(N_1 + N_2(1 - \delta)^{\frac{\alpha}{1-\theta}})} \\ h_2^*(n) &= (1 - \delta)^{\frac{\alpha}{1-\theta}} h_1 \\ c^*(n) &= \frac{\beta(1 - \gamma n)wg(H)}{\beta + \theta(1 - \beta)} \end{aligned}$$

Note that  $h_2 < h_1$  for  $\delta > 0$ .

Rewriting the utility function with  $h_1^*(n)$  and  $h_2^*(n)$  and  $c^*(n)$ :

$$\beta \ln\left(\frac{\beta(1 - \gamma n)wg(H)}{\beta + \theta(1 - \beta)}\right) + (1 - \beta) \ln\left((N_1 + N_2(1 - \delta)^{\frac{\alpha}{1-\theta}})^{1-\theta} \left(\frac{\theta(1 - \beta)(1 - \gamma n)wg(H)}{\beta + \theta(1 - \beta)}\right)^\theta w'\right)$$

and applying the Delta method (see Appendix B.2), the first order condition with respect to  $n$  implicitly defines fertility as a function of  $i$ ,  $d$ , and other parameters of the model.

$$n - \frac{\gamma(\beta + \theta(1 - \beta))n^2}{(1 - \beta)(1 - \theta)(1 - \gamma n)} = \frac{-i[1 - i + (1 - \delta)^{\frac{2\alpha}{1-\theta}}(1 - d)[1 - i(1 - d)]]}{2[1 - i + i(1 - \delta)^{\frac{\alpha}{1-\theta}}(1 - d)]^2}$$

### 3 Fertility, Human Capital Investment, and the Disease Burden

#### 3.1 Total Fertility Rate Responses

For the special case of no child mortality,  $i = 0$  or  $i = 1$  and  $d = 0$ , the first-order condition with respect to  $n$  solves to  $n = (1 - \beta)(1 - \theta)/\gamma$ , the same as under certainty. Adults have fewer children if they value own current consumption more ( $\beta$  higher) or if returns to human capital investment,  $\theta$ , in children are high.

Of interest is the response of  $n$  to changes in  $i$  and  $d$ , and for ease of interpretation I use implicit differentiation to analyze these relationships. See Appendix B.3 for details. For simplicity, assume  $\gamma = 1$ , which restricts  $n \in (0, 1)$  instead of  $n \in (0, 1/\gamma)$ . To allow for more decisive interpretation, the following analysis focuses on extreme values of  $\delta$ , which yields conclusions consistent with more general results.

Figure 1 depicts the solution for different parameter values. That fertility is higher when  $\theta$  is lower is confirmed for the general case, as can be seen in Figure 2. For  $\delta \rightarrow 1$ , fertility increases with  $i$ . Children who get a disease when  $\delta \rightarrow 1$  are worthless to selfish parents because they provide no financial support to parents during retirement regardless of whether they survive. For  $\delta \rightarrow 0$ , the response of fertility to infection rates is nonlinear: it is positive when disease prevalence is low or when both disease prevalence and case fatality rates are high; it is negative when infection is prevalent but case fatality rates are low. This result is empirically supported in Section 4 for HIV and malaria prevalence in sub-Saharan Africa. The net response of fertility to changes in  $d$  is generally subtle in Figure 1.

Theoretically, there are three primary forces determining the response of  $n$  to changes in  $i$  and  $d$ , the third of which is a key contribution of this paper and operates through morbidity effects. First, in the face of considerable child mortality, parents have more children than they ultimately desire in the anticipation that some will not survive, so a decline in mortality reduces births as more children survive. As mortality declines, uncertainty about the number of survivors declines, so parents are less likely to overshoot their desired number of surviving children. This precautionary motive generates a positive relationship between fertility and childhood disease.

Secondly, when child mortality declines, the average cost of raising a surviving child decreases, unless  $n$  rises to compensate. The total cost of child rearing is  $\gamma n$ , the fixed birth cost, plus the investment in each child after survival outcomes have been realized,  $N_1 h_1 + N_2 h_2$ . Investment,  $h_1$  and  $h_2$ , decreases with the number of survivors, so lower child mortality decreases  $h_1$  and  $h_2$  for a given level of  $n$ . The average cost per surviving child,  $(\gamma n + N_1 h_1 + N_2 h_2)/(N_1 + N_2)$ , decreases due to the increase in  $(N_1 + N_2)$ . The fixed birth cost drives the result in Barro and Becker (1988): demand for children rises in response to a decline in child mortality because survivors become cheaper to produce.

Thirdly, and unique to this model, parents substitute quantity and quality of children when the disease burden changes due to the morbidity effect. Suppose a vaccine becomes available and  $i$  declines. Survivors become more expensive on average, because  $N_1/(N_1 + N_2)$  increases and  $h_1 \geq h_2 = (1 - \delta)^{\alpha/(1-\theta)} h_1$ : hence parents have fewer children and invest more in the human capital of each. This quantity-quality tradeoff is stronger the larger is  $\delta$  and counteracts some of the negative average cost effect described above. For lower  $\delta$ , this force is weaker and the traditional (negative) average cost relationship is stronger;  $\partial n/\partial i < 0$  is more likely if  $\delta$  is low. In Figure 1  $\partial n/\partial i < 0$  occurs when  $\delta$  is low and infection is prevalent but case fatality rates are low; morbidity effects under  $\delta > 0$  are strongest where most of surviving children have experienced disease ( $N_2$  is biggest when  $i$

is high and  $d$  is low), so the largest contrast for the average cost effect between the cases  $\delta \rightarrow 1$  and  $\delta \rightarrow 0$  occurs for high  $i$ /low  $d$ . This is where we observe the most striking contrast in the fertility response when moving between the cases  $\delta \rightarrow 1$  and  $\delta \rightarrow 0$ .

Suppose that  $d$  declines because an antibiotic becomes available. For  $\delta > 0$ , reducing fatalities leaves proportionately more unhealthy children alive and  $N_1/(N_1+N_2)$  decreases. Since parents invest less in the human capital of unhealthy children, they have more children in order to substitute for their low quality. The range for optimal total fertility in Figure 1 is 0-0.3 for the case  $\delta \rightarrow 0$  and 0.9-0.4 for the case  $\delta \rightarrow 1$  where morbidity effects are stronger. In contrast to the vaccine scenario, the average cost effect is more negative when  $\delta$  is high. However, parents may have fewer children if the returns to having children (consumption during retirement) are so low.<sup>11</sup> These conflicting effects yield a very subtle response of fertility to  $d$  in Figure 1, for most cases.

When  $\delta$  is low, the  $i$ - and  $d$ -specific influences on morbidity are weak, and the response of  $n$  to falling disease burden depends on the net effect of a reduction in precautionary births ( $n$  decreases when child mortality decreases) and a decline in the (traditional) average fixed cost of survivors ( $n$  increases when child mortality decreases). The positive precautionary motive dominates the negative average cost effect when mortality changes are strongest; disease burden is high when  $i$  and  $d$  are both high, and it is moderate when  $i$  high/ $d$  low, or vice versa.  $dn/di > 0$  is more likely when  $d$  is high, and  $dn/dd > 0$  is more likely for higher  $i$ , since this is where mortality changes are strongest (see Appendix B.3 for calculation and analysis of the relevant thresholds).

If child disease-caused mortality rates are high, introducing a vaccine or antibiotic reduces fertility because either action considerably reduces mortality and uncertainty about child survival. A vaccine lowers mortality more for more fatal diseases, and it reduces morbidity more for high  $\delta$  diseases. The former reduces the average fixed cost of survivors and the latter causes parents to have fewer children but invest more in the human capital of each; lower  $\delta$  dampens only the latter effect. Thus  $\partial n/\partial i > 0$  unambiguously when  $\delta \rightarrow 1$ .

An antibiotic lowers mortality more for very prevalent and fatal diseases, and it increases morbidity more for high  $\delta$  diseases. Changes in case fatality rates only elicit a noticeable (and positive) fertility response in Figure 1 when morbidity effects are low ( $\delta$  low) and when the impact on child mortality is highest ( $i$  and  $d$  are both high). When both  $i$  and  $d$  are very low, fertility is already nearest its lower bound, as seen in Figure 1, so an increase in  $i$  or  $d$  cannot decrease  $n$  anyway.

---

<sup>11</sup>Also, a woman who knows she is HIV-positive, may choose to not have children, especially if she lacks access to medicine that reduces mother-to-child transmission.

**Proposition 1.** *Under uncertainty, fertility rises with disease prevalence if disease is very fatal or if disease is not very fatal but causes severe long-term morbidity.*<sup>12</sup>

### 3.2 Net Fertility Response

Kalemli-Ozcan (2003) and Galor (2005) stress the historically observed U-shaped pattern for net fertility; as child mortality declines and more children survive, total fertility does not initially decline enough to lower net fertility, but eventually the decline in total fertility dominates, bringing down net fertility. The model supports this as a possibility.

On average  $n(1 - id)$  children survive, and  $\partial n(1 - id)/\partial i = (\partial n/\partial i)(1 - id) - nd$  and  $\partial n(1 - id)/\partial d = (\partial n/\partial d)(1 - id) - ni$ . In the case where  $\partial n/\partial i > 0$  and  $\partial n/\partial d > 0$ , net fertility rises in response to lower  $i$  if  $\partial n/\partial i < (nd)/(1 - id)$ , and in response to lower  $d$  if  $\partial n/\partial d < (ni)/(1 - id)$ . These conditions are more likely to hold for high  $i$  and  $d$ . As disease prevalence and case fatality rates continue to decline, the conditions become more binding, and eventually net fertility also declines.

### 3.3 Human Capital Investment

Finally we turn to the impact of disease on human capital investment. See Appendix B.4 for calculations.

**Proposition 2.** *Under uncertainty, parental human capital investment in children rises when disease burden falls if the total fertility response to changing infection rates and case fatality rates is positive and sufficiently large.*

If TFR rises when child mortality falls, then fewer resources remain for human capital investment, and  $h_1$  and  $h_2$  decline. If TFR declines but not enough to counter the increase in survivors, then  $h_1$  and  $h_2$  still decline. In countries where the fertility response may be weak or even negative, health care initiatives should be coupled with human capital investment initiatives, such as school subsidies, because combating disease may actually raise net and even total fertility and thus reduce human capital investment.

## 4 Empirics

Previous empirical tests of the impact of disease on fertility focus on a linear relationship. In an attempt to replicate the results in Kalemli-Ozcan (2009), I show that the conclusions in that study change when the country set and time span of data are expanded. I compare

---

<sup>12</sup>Note that a low  $i$  here could be as high as  $i = 1/2$ . See Appendix B.3.

this linear specification to a nonlinear one, where the impact of disease on fertility depends on the level of child mortality. As predicted in the model, fertility tends to rise with infection rates when child mortality rates are highest and fall when child mortality rates are moderate. For the case of HIV, this reconciles conflicting conclusions in previous studies. In Kalemli-Ozcan (2009) the AIDS epidemic is shown to increase fertility in Africa, while Young (2005) concludes the opposite, and Juhn, Kalemli-Ozcan and Turan (2009) find no effect. In contrast to previous studies where fertility unambiguously rises with child mortality, I show there are both positive and negative components to the relationship between disease burden and fertility.

Fertility also responds differently to different types of diseases. Comparing fertility responses to HIV versus malaria, I show that fertility has more potential to decline with disease prevalence when the disease in question causes milder long-term morbidity.

Finally, I test the response of education to changes in disease burden. Education declines when fertility rises due to resource constraints. I confirm both the nonlinear relationship between disease burden and fertility and the negative relationship between education and fertility.

## 4.1 Regression Model

To properly capture the theoretical model, the regression model specification must account for disease prevalence, disease severity (fatality rates and long-term consequences), incomes and child-bearing costs. The primary model tested is

$$TFR = \beta_0 + \beta_1 Disease\ prevalence_{it} + \beta_2 Child\ mortality_{it} + \beta_3 Disease\ prevalence * Child\ mortality + \beta_4 Adult\ mortality_{it} + \beta_5 GDP\ per\ capita_{it} + \beta_6 Female\ Literacy_{it} + \epsilon_{it}$$

The interaction of disease prevalence and child mortality allows the disease-fertility relationship to vary according to child mortality levels. The model predicts that the fertility response to infection rates is most positive where mortality effects are strongest and muted or negative where mortality effects are moderate; I expect a positive coefficient on the interaction term.

To test the model's implications regarding long-term morbidity,  $\delta$ , I consider the impact of two very different infectious diseases that affect sub-Saharan Africa: HIV and malaria. Both are significant causes of child mortality, as seen in Tables 1 and 2. Child deaths due to HIV vary throughout sub-Saharan Africa from 0% in Mauritius to 57% of all child deaths in South Africa, while the corresponding figure for malaria is 0% in

South Africa to 29% in Gambia.<sup>13</sup> On average 84% of children born HIV-positive die by the age of five (World Health Organization). Malaria is a major cause of childhood anemia and is associated with maternal anemia during pregnancy and low birth weight, and cerebral malaria can cause persistent neurological deficits (Gollin and Zimmerman 2007). Yet exposure to malaria also endows survivors with partial immunity to further malarial infection.<sup>14</sup> Thus, although both HIV and malaria have severe consequences for health, I consider those of malaria relatively less so; HIV represents a high  $\delta$  disease and malaria represents a relatively low  $\delta$  disease in the regression analysis below.

The model predicts that fertility will generally rise with HIV prevalence, and fertility will rise with malaria prevalence if malaria case fatality rates are high but may fall where case fatality rates are low. Case fatality rates for malaria tend to be lower in high prevalence areas relative to low prevalence areas. Marsh and Snow (1999) find that the consequences of malarial infection are most severe in moderate transmission areas, with the severity maintaining a plateau or even falling for high transmission areas. A study of children in Tanzania by Reyburn *et al.* (2005) concludes that higher case fatality rates can be attributed to a higher occurrence of the more fatal cerebral malaria in low transmission areas.<sup>15</sup>

Control variables are female literacy, GDP per capita and adult mortality. More educated women tend to have fewer children because the opportunity cost of doing so is higher, since more skilled women can earn more by working. Female literacy is a proxy for the time cost of rearing children, or  $\gamma$  in the model.

Wealthier parents can devote more resources to protecting their children from disease, so more children survive and they tend to be healthier; parents have fewer children since the precautionary motive is weaker and because parents replace quantity of children with quality. Theoretically, surviving children are more expensive because  $h_1 > h_2$ .

An increase in adult mortality is equivalent to an increase in  $\beta$  in the model developed above; parents place more emphasis on current consumption and less on future consumption. Theoretically, both fertility and human capital investment in children decline, since

---

<sup>13</sup>90% of child HIV infection occurs vertically from the mother. In developing countries mother-to-child transmission of HIV is approximately 30% at birth and 3% with every month of breastfeeding, but less than 2% where appropriate treatment is available. According to the World Health Organization, the average proportion of HIV-positive pregnant women in sub-Saharan Africa receiving treatment to prevent mother-to-child transmission has been climbing but remains below 50%.

<sup>14</sup>Genetic adaptations have been discovered in groups of people living in malaria-intense regions; hemoglobin-related disorders and other blood cell dyscrasias are more prevalent in malaria endemic areas and are thought to provide protection from malarial disease. Acquired immunity from exposure is strain-specific and is lost if a person moves away from a malaria endemic area (Center for Disease Control).

<sup>15</sup>Lower transmission areas are those at higher altitudes where fewer mosquitoes live.

parents benefit from these only in retirement. However, children could also be modeled as consumption goods; parents gain utility from having a family, so fertility rises with adult mortality as parents consume more resources now and invest less for the future.

Country effects account for cultural, institutional, and other unobserved, time-invariant country characteristics that affect fertility rates, and year effects account for shocks affecting all countries simultaneously. As seen in Figure 3, TFR generally declines over the time span of the sample, so it is important to account for year effects or a time trend.

For the case of HIV, which affects adults more than children, I include an interaction term of adult mortality and HIV prevalence. Since malaria is mostly fatal in children, including a similar interaction between adult mortality and malaria is unnecessary.

## 4.2 Data

Table 3 summarizes the data collected on 44 African countries for 1985-2005. Data for each country and year is not always available, but panel regression analysis allows maximum exploitation of all variation across countries and years that are available. Data is interpolated to maximize observations, and Table 3 shows descriptive statistics for both original and interpolated variables. Appendix C contains a list of countries. Data on female literacy rates, adult, infant and child mortality rates, and GDP per capita are from the World Bank's World Development Indicators. Children's education statistics are from the World Health Organization (WHO). HIV prevalence data is collected from the US Census Bureau's HIV/AIDS Surveillance Database for 1985-2005 and is prevalence rates among pregnant women.<sup>16</sup> Reported malaria cases for 1982-1997 from the WHO's Weekly Epidemiological Record are supplemented with reported malaria cases for 1990-2007 from the WHO's Global Malaria Programme, and these proxy for malaria prevalence.

As seen in Table 3, HIV and malaria prevalence rates vary substantially both between countries and across time, with prevalence rates as high as 55% for HIV and incidence rates as high as 95% for malaria. Most of the variation in mortality rates and female literacy rates is between countries rather than across time. Mortality rates are as high as 19% for infants, 32% for children under five years of age, and 74% for adults, where adult mortality is defined as the probability of death between age 15 and 60. TFR varies from 1.91 to 8.35 births per woman.

---

<sup>16</sup>Pregnant women are the most commonly tested sub-population group, and seroprevalence studies of pregnant women are more reliable than aggregate statistics in Africa. Often multiple studies for a country and year are available, so I calculate a single, sample-size weighted average for each country-year pair. Studies also vary according to whether they test pregnant women for HIV1, HIV2 or either; regression results do not vary significantly whether I use just HIV1 or any HIV, and I use the latter.

There is some concern that using only HIV prevalence among pregnant women creates a biased picture of HIV burden. Women who engage in riskier sexual activity are both more likely to become pregnant and to contract HIV, so the measure would overestimate HIV prevalence in the general population. However, there is evidence that HIV-positive women are less fecund for physiological reasons (fewer pregnancies are carried to term) or because of social norms. For example, HIV-positive women are more likely to be widows (Glynn *et al.* 2000). Thus fewer HIV-positive women become pregnant relative to HIV-negative women, and the measure would then underestimate HIV prevalence in the general population.<sup>17</sup> Therefore, the measurement error does not necessarily go in one direction, and HIV prevalence among pregnant women remains the most consistent measure of HIV prevalence available for a large number of countries and years.

## 4.3 Regression Results

### 4.3.1 Total Fertility Rates

In an attempt to replicate the empirical results of Kalemli-Ozcan (2009), I run regressions for the full set of data available to me, specifically 44 countries for 1985-2005, but separately compare these results to those of the restricted time series (1985-2000) and the restricted country sample used in that paper (Tables 4 and 5).<sup>18</sup> The bottom right quadrant of Table 5 is the closest replication of that study given my data. The most reliable results are those with both country fixed effects and year fixed effects or a time trend, columns 1, 2, 5 and 6 in Tables 4 and 5. Focusing on these results, a negative and significant coefficient on HIV occurs for a larger country set and/or a longer (and more recent) time series. The negative and insignificant coefficient found in Kalemli-Ozcan (2009) using the same HIV prevalence measure as I do becomes significant when more data is included in the panel regressions.<sup>19</sup>

Remaining regressions use logs of all variables for ease of interpretation. Assuming a

---

<sup>17</sup>Juhn, Kalemli-Ozcan, and Turan (2008) differentiate between the impact of own HIV status on TFR versus community HIV prevalence on TFR of noninfected women. For the former they find a negative and significant effect, while for the latter they find no significant effect.

<sup>18</sup>The countries dropped in the restricted country sample are: Madagascar, Lesotho, Namibia, Zambia, Zimbabwe, Swaziland, Uganda, Benin, and the Central African Republic. Two sets of regressors appear in Tables 4 and 5; the bottom set are those used in Kalemli-Ozcan (2009), while for the other regressions in this paper, I instead use logs of mortality and female literacy rates. The coefficient on HIV does not differ substantially across the two sets of regressors, except that a negative and significant result occurs in columns 1 and 4 of Table 5 only for the Kalemli-Ozcan set of regressors, showing the importance of a longer time series.

<sup>19</sup>I also replicate her pooled regressions and, as in that study, find a positive coefficient on HIV prevalence in the absence of the interaction term of HIV prevalence and infant mortality, although pooled regressions do not account for country-specific factors. Results are available upon request.

linear relationship between fertility and HIV prevalence, columns 5-8 of Table 6, suggests a negative relationship between the two variables. When the interaction of HIV prevalence and child mortality is included, as in columns 1-4, the direct effect of HIV prevalence on fertility remains negative but is dampened where HIV prevalence coincides with higher child mortality rates. The net predicted values of the effect of HIV on TFR listed at the bottom of Table 6 are negative only for countries with lower (moderate) child mortality rates, theoretically those with high  $i$ /low  $d$  or low  $i$ /high  $d$ .<sup>20</sup> The negative coefficient on HIV prevalence is smaller in absolute value under the assumption of linearity, because it absorbs the counteracting positive effect of the interaction term.

The other regressors, particularly child mortality, are all insignificant under the assumption of linearity, while all but GDP per capita are significant when I allow for a non-linear response of fertility to HIV prevalence. Theoretically, income directly affects human capital investment but not fertility; GDP per capita is insignificant in most regressions of TFR, although it is negative where significant. Surprisingly, more educated women have more children. Unlike other mortality-fertility studies, the coefficient on child mortality is negative, although higher child mortality rates increase fertility indirectly through the interaction with disease prevalence.

HIV/AIDS is a unique disease in that its burden among *young* adults is so great. In Table 7 I examine the contribution of interacting adult mortality and HIV. The response of fertility to HIV tends toward negative where child mortality rates are lowest and adult mortality rates are highest. HIV kills young adults of child-bearing age, and the coefficient on the interaction of adult mortality and HIV is negative. Isolating this negative effect amplifies the positive coefficients on adult mortality and on the interaction of child mortality and HIV prevalence. Both interaction terms are significant, although there is no longer a direct effect of HIV on TFR; the relationship between HIV and TFR functions primarily through its high casualty rate, consistent with the theoretical predictions regarding very high  $\delta$  diseases.

Table 8 presents regression results using malaria instead of HIV prevalence, confirming the nonlinear relationship between fertility and disease prevalence; fertility declines when malaria prevalence declines where child mortality rates are highest. Under the assumption of linearity, the negative direct effect of malaria is smaller in absolute value, because it absorbs the positive effect of malaria on fertility functioning through child mortality.

---

<sup>20</sup>Results do not vary substantially if infant mortality is used instead of child mortality, although the proportion of the sample that has a negative predicted fertility response to each disease supports the predictions somewhat better when child mortality is used. Results are available upon request. Child mortality better represents the theoretical model developed above, because uncertainty about child survival outcomes is resolved when children reach school age.

Consistent with theoretical prediction, a larger proportion of observations have a negative predicted net response of fertility to moderate  $\delta$  malaria than to high  $\delta$  HIV, 97-98% for malaria versus 61% in comparable specifications for HIV. In the case of HIV, the specification that includes the interaction of HIV prevalence and adult mortality is even more supportive of this prediction, with only 31-37% of observations having a negative predicted response of fertility to HIV. If the interaction of disease prevalence and child mortality is omitted, 100% of observations have a negative predicted response of TFR to both malaria and HIV.

Over the sample period, TFR and child mortality rates generally decline while female literacy, adult mortality rates and GDP per capita generally follow an upward trend. Thus in regressions omitting year fixed effects or a time trend, the coefficient on child mortality is positive and those on female literacy, adult mortality rates and GDP per capita are negative. The coefficient on adult mortality is also negative unless its interaction with HIV prevalence is included, in which case the coefficient on adult mortality is insignificant. Also, the coefficient on the interaction of child mortality and disease prevalence is insignificant.

In countries with weak institutions, health and education infrastructure are less adequate, so disease prevalence and mortality rates may be higher while female literacy may be lower. With less effective dissemination of contraceptives and knowledge about family planning, TFR may be higher. Thus in regressions without country fixed effects, the coefficient on child mortality is positive and that on female literacy is negative, and the negative coefficient on adult mortality is surprising but returns to positive when its interaction with HIV prevalence is included. The negative, significant coefficient on disease prevalence disappears as it absorbs the positive relationship between TFR and disease prevalence that functions through institutions.

### 4.3.2 Education

If HIV generally raises fertility, then it should reduce children's education by diminishing the resources available per child. If malaria tends to lower fertility, then it should have a largely positive impact on children's education.

I regress children's education on HIV and malaria prevalence using three different measures of education: primary school completion rate and primary school gross and net enrollment rates. Gross enrollment rates measure the number of children in primary school as a proportion of total children of primary school age, so the gross enrollment rate could be above 100%. The net enrollment rate counts only children of primary school age, so the maximum net enrollment rate possible is 100%. Year fixed effects are included because,

for example, an increase in foreign aid one year could increase education expenditures and thereby increase school enrollment. Country fixed effects account for country differences such as institutions, which can affect the efficacy and influence of the education system.

Tables 9-14 present regression results. Regressions using net enrollment rates as the measure of parental human capital investment in children are most clear and supportive of the model. Net enrollment rates are lower where HIV is more prevalent, with this negative relationship amplified where child mortality is higher and dampened where adult mortality is higher. Where significant, net enrollment rates increase with malaria incidence but less so where child mortality is higher. In other words, the coefficient signs are the opposite of those in the TFR regressions above, as expected. The predicted values are less supportive of model predictions, as seen at the bottoms of the result tables. Recall, however, that if human capital investment is to rise when TFR declines, the change in TFR must be strong enough to also reduce net fertility.

Net enrollment rates are lower where child mortality is higher or where TFR is higher. When gross, rather than net, enrollment rates are used as the dependent variable, results are similar except for the positive coefficient on TFR; where TFR is greater, more children may begin school at a later age or have to repeat school years. Child mortality is not significant in explaining the variation in *gross* enrollment rates, but surprisingly, higher child mortality is associated with higher primary school completion rates in column 4 of Table 10.

## 5 Discussion and Policy Implications

By building on the mortality-fertility literature, this paper explores the consequences for fertility of reducing disease burden in developing countries. The results suggest that health initiatives can have different effects on fertility depending on the morbidity and mortality associated with the disease in question. Both empirically and theoretically, the strongest positive response of fertility to disease prevalence occurs where mortality rates are highest, while a negative or muted response occurs where mortality rates are moderate. That reducing disease burden may raise fertility rates does not contradict the existing consensus that fertility follows child mortality. Rather it highlights that health initiatives are most effective at reducing fertility when they also tackle morbidity.

Historically, in many Western countries both infection and case fatality rates were high, and it was primarily new knowledge about germs that stimulated strong declines in deaths from infectious diseases in the latter 19th century. Public sanitation reform and improvements in personal hygiene triggered an epidemiological transition, and mortality

and morbidity shrank together (McNeill 1976).<sup>21</sup> As infection rates declined, child mortality fell significantly, followed shortly by fertility rates. As fewer children got sick and survivors were healthier, parents had fewer children and invested more in the human capital of each child. The model generates such a quantity-quality tradeoff when morbidity declines. In many developing countries today, infectious disease burden and fertility rates remain high, and reductions in mortality function both through infection rates (improved sanitation in urban areas, vaccinations) and case fatality rates (antibiotics). If deaths but not morbidity from disease declines, high fertility rates will persist as parents continue to supplement the low quality of their children with greater quantity.

This paper demonstrates that, depending on the types of diseases that are most prominent and the manner in which they are combated, some developing countries may not necessarily be poised for the demographic transition and the resulting rise in human capital investment. Infectious diseases weaken individuals physically and may impair cognitive development, especially in developing countries where infections are particularly virulent and children are often under- or malnourished. Parasitic diseases are most prevalent in developing countries, and their treatment prevents deaths but cannot reverse damage already attained. For example, treatments exist for leishmaniasis, which damages the spleen and liver and can cause anaemia, and for schistosomiasis, a chronic disease that damages internal organs and impairs growth and cognitive development in children. Prevention is preferred to treatment in such cases. Since polio is an infectious disease that renders its survivors paralyzed (high  $\delta$ ) and for which there exists no cure, the model predicts its global eradication reduced fertility rates by reducing morbidity.<sup>22</sup>

Given the virulent nature of their disease burden,  $\delta$  is clearly substantial for developing countries, falling somewhere between the two extremes in Figure 1. Thus the model predicts that tackling the disease burden in developing countries will often reduce fertility but may have muted, rather than negative, effects on fertility in certain cases.

The result here that there are both positive and negative components to the relationship between disease and fertility helps reconcile conflicting conclusions in previous studies regarding the AIDS epidemic. HIV prevalence raises fertility rates through its impact on child mortality, in support of Kalemli-Ozcan (2009), and decreases fertility through its effect on adult mortality, in support of Young (2005). The latter may function through rising wages as labor supply shrinks. People may also reduce risky sexual behavior, and HIV infection tends to lower fecundity of HIV-positive women. The role of adult disease

---

<sup>21</sup>In 1853 vaccination was made compulsory and in 1871 legislation was introduced requiring all poor law unions to appoint vaccination officers and to set up a system of registration (Drake *et al.* 1997).

<sup>22</sup>Led by efforts of the WHO, polio has been eradicated in all but four countries: Afghanistan, India, Nigeria and Pakistan.

burden in fertility decisions remains a task for further research.

Debate surrounds the allocation of resources to battle the AIDS epidemic; is it worth diverting some resources from the search for a vaccine to treat those who are already infected? The model supports an affirmative answer, since for diseases that cause severe long-term morbidity ( $\delta \rightarrow 1$ ), fertility declines in response to both lower infection rates and lower case fatality rates. However, the case of AIDS is unique in that it is a large contributor to adult, not just child, mortality, and this particular case merits further research.

For malaria, the answer is case-specific. Recall that malaria tends to be more fatal in moderate transmission areas and less fatal in high transmission areas. The model predicts that reducing transmission will *increase* fertility in high transmission areas, and lower it in moderate transmission areas. The impact per infection averted on mortality rates will be higher in the moderate transmission setting where case fatality rates are high, so a positive fertility response dominates. Conversely, the reduction in mortality per fatality averted through treatment of malarial infections is higher in high transmission areas and lower in moderate transmission areas. More anaemic children survive, for example, but the average health quality of children declines less in high transmission areas where malarial infections are relatively milder ( $\delta$  is lower); parents have less incentive to increase quantity to replace quality loss in high transmission areas. From a population control standpoint, reducing transmission via dissemination of bed nets or insecticide to eradicate mosquito populations is preferred to treatment of existing infections in moderate transmission settings, and the opposite holds for high transmission settings. This allocation of resources will most effectively reduce fertility and improve human capital.

## 6 Conclusion and Extensions

The contribution here of morbidity to fertility choice is unique in the mortality-fertility literature. It generates a quantity-quality tradeoff that parents face regarding children; as morbidity lessens, parents have fewer but healthier children, and they invest more in their human capital. Morbidity can actually rise as the disease burden declines if the decline happens due to falling case fatality rates. The morbidity effect amplifies the positive fertility response to infection rates and dampens it in response to case fatality rates.

However, it is in the special case where I exclude morbidity effects that a negative fertility response is most likely, unlike the unambiguously positive response in previous mortality-fertility studies. This arises because I distinguish between the contribution of infection and case fatality rates to child mortality; the quantity-quality tradeoff plays less

of a role and changes in precautionary demand for children versus changes in the average fixed cost of surviving children determine the net response of total fertility. The quantity-quality tradeoff triggered by changing infection rates is strongest where infection rates are high, case fatality rates are low, and long-term morbidity consequences for survivors are severe; when this quantity-quality tradeoff disappears theoretically, the fertility response becomes most muted (even negative) for this case.

Regressions of fertility on HIV and malaria prevalence confirm the nonlinear disease-fertility relationship; fertility rises with disease prevalence for high child mortality rates but falls for moderate child mortality rates for diseases with less severe long-term health consequences. The predicted fertility response of fertility to HIV is mostly positive while that to malaria, whose long-term health consequences are less severe, is mostly negative. Regressions of primary school education on HIV and malaria prevalence further confirm the model predictions.

One could test the model's general predictions for diseases other than HIV and malaria, for example parasitic diseases that heavily burden the developing world. The case-specificity of malaria's impact on TFR could be further analyzed using micro level data on malaria eradication (pesticide), prevention (mosquito nets) and treatment programs, comparing responses to similar programs in regions with different malaria infection and case fatality rates. Further research could analyze the dynamic implication for the evolution of disease burden, fertility and human capital.

## 7 References

- Arora, S. (2003). ‘Disease, human disability and long-term economic growth’, Conference on Health and Economic Policy, CESifo Conference Centre, Munich, 27-28 (June).
- Arora, S. (2005). ‘On epidemiological and economic transitions: a historical view’ in *Health and Economic Growth: Findings and Policy Implications*. Edited by Lopez-Casasnovas, G., Rivera, B., and Currais, L. The MIT Press, Cambridge, MA, 197-238.
- Barker, D.J.P. (1992). ‘Fetal and Infant Origins of Adult Disease’, *British Medical Journal* London.
- Barker, D.J.P. (1994). ‘Mothers, babies, and disease in later life’, *British Medical Journal Publishing Group (BMJ)*.
- Barro, R. and Becker, G.S. (1988). ‘A Reformulation of the Economic Theory of Fertility’, *The Quarterly Journal of Economics* 103(1):1-25 (February).
- Becker, G.S. and Lewis, H.G. (1973). ‘On the Interaction between the Quantity and Quality of Children’, *The Journal of Political Economy* 81(2, Part 2):S279-S288.
- Birchenall, J. (2007). ‘Escaping high mortality’, *Journal of Economic Growth*, 12:351-387.
- Boldrin, M. and Jones, L.E. (2002). ‘Mortality, Fertility, and Saving in a Malthusian Economy’, *Review of Economic Dynamics* 5 (4):775-814.
- Boucekkine, R., Desbordes, R., and Latzer, H. (2008). ‘How do epidemics induce behavioral changes?’ CORE Discussion Paper.
- Cervellati, M. and Sunde, U. (2007). ‘Human capital, mortality, and fertility: a unified theory of demographic transition’, IZA Working Paper 2905.
- Doepke, M. (2005). ‘Child Mortality and Fertility Decline: Does the Barro-Becker Model Fit the Facts?’ *Journal of Population Economics* 18:337-366.
- Drake, M. *et al.* (1997). ‘Decline of Infant Mortality in England and Wales, 1871-1948 :

a Medical Conundrum (1997). Vaccination Registers, 1871-1913'.

Galor, O. and D. N. Weil. (1999). 'From Malthusian stagnation to modern growth', *American Economic Review* 89, 150-154.

Glynn, J.R. *et al.* (2000). 'Decreased Fertility Among HIV-1-Infected Women Attending Antenatal Clinics in Three African Cities', *Journal of Acquired Immune Deficiency Syndromes* 25(4):345-352.

Gollin, D. and C. Zimmerman (2007). 'Malaria: Disease Impacts and Long-Run Income Differences', Institute for the Study of Labor Discussion Paper.

Juhn, C., Kalemli-Ozcan, S. and Turan, B. (2009). 'HIV and Fertility in Africa: First Evidence from Population Based Surveys', NBER and University of Houston.

Kalemli-Ozcan, S. (2009). 'AIDS, 'Reversal' of the Demographic Transition and Economic Development: Evidence from Africa', NBER and University of Houston.

Kalemli-Ozcan, S. (2008). 'The Uncertain Lifetime and the Timing of Human Capital Investment', *Journal of Population Economics*, 21 (3).

Kalemli-Ozcan, S. (2003). 'A Stochastic Model of Mortality, Fertility, and Human Capital Investment', *Journal of Development Economics*, 70 (1).

Khosla, S.N. (1981). 'The heart in enteric (typhoid) fever', *Journal of Tropical Medicine and Hygiene* 84 (3):125-131.

Marsh and Snow (1999). 'Malaria Transmission and Morbidity', *Parassitologia* 41(1-3):241-6.

Martorell, R. and Habicht, J.P. (1986). 'Growth in early childhood in developing countries', In *Human Growth: A Comprehensive Treatise*, Vol.3, eds. F. Falkner and J.M. Tanner. New York and London: Plenum Press, 241-263.

Martorell, R. (1980). 'Inter-relationships between diet, infectious disease, and nutritional status', In *Social and Biological Predictors of Nutritional Status, Physical Growth*,

*and Neurological Development*, eds. L.S. Greene and F.E. Johnsston. New York: Academic Press.

Mata, L.J. (1978). *The Children of Santa Maria Cauque: A Prospective Field Study of Health and Growth*. Cambridge, MA, The MIT Press.

McNeill, W.H. (1976). *Plagues and People*. Anchor Books, New York.

Reyburn, H., Mbatia, R., Drakeley, C. *et al.* (2005) 'Association of Transmission Intensity and Age With Plasmodium falciparum Malaria Clinical Manifestations and Case Fatality of Severe', *The Journal of the American Medical Association* 293(12):1461-1470.

Sah, Raaj K. (1991). 'The effects of child mortality changes on fertility choice and parental welfare.' *The Journal of Political Economy* 99 (3):582-606 (June).

Soares, R. (2005). 'Mortality reduction, educational attainment, and fertility choice', *The American Economic Review* 95 (3):580-601 (June).

Strulik, H. (2008). 'Geography, health, and the pace of demo-economic development', *Journal of Development Economics* 86:61-75.

Tamura, R. (2006). 'Human capital and economic development', *Journal of Development Economics* 79:26-72.

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

## A Tables

Rank	Cause	Numbers (000)	% of all deaths
1	Perinatal conditions	2,375	23.1
2	Lower respiratory infections (pneumonia)	1,856	18.1
3	Diarrhoeal diseases	1,566	15.2
4	Malaria	1,098	10.7
5	Measles	551	5.4
6	Congenital anomalies	386	3.8
7	HIV/AIDS	370	3.6
8	Pertussis	301	2.9
9	Tetanus	185	1.8
10	Protein-energy malnutrition	138	1.3
	Other causes	1,437	14.0
	Total	10,263	100.0

Source: World Health Organization

Table 1: Top 10 causes of child mortality in developing countries

Table 2: Contribution of HIV and malaria to (under age five) child mortality, 2000

Country	Percentage of child deaths due to		
	HIV/AIDS	Malaria	Neonatal causes
Angola	2.2	8.3	22.2
Benin	2.2	27.2	25.0
Botswana	53.8	0	40.3
Burkina Faso	4.0	20.3	18.3
Burundi	8.0	8.4	23.3
Cameroon	7.2	22.8	24.8
Central African Republic	12.4	18.5	27.2
Chad	4.1	22.3	24.0
Comoros	3.7	19.4	37.3
Congo	9.3	25.7	30.9
Cote d'Ivoire	5.6	20.5	34.9
Dem. Rep. of the Congo	3.7	16.9	25.7
Equatorial Guinea	7.4	24.0	27.5
Eritrea	6.2	13.6	27.4
Ethiopia	3.8	6.1	30.2
Gabon	10.1	28.3	35.1
Gambia	1.3	29.4	36.6
Ghana	5.7	33.0	28.5
Guinea	2.3	24.5	28.8
Guinea-Bissau	2.6	21	24.1
Kenya	14.6	13.6	24.2
Lesotho	56.2	0	32.8
Liberia	3.6	18.9	29.1
Madagascar	1.3	20.1	25.6
Malawi	14.0	14.1	21.7
Mali	1.6	16.9	25.9
Mauritania	0.3	12.2	39.4
Mauritius	0	0	66.0
Mozambique	12.9	18.9	29.0
Namibia	53.0	0	38.5
Niger	0.6	14.3	16.7
Nigeria	5.0	24.1	26.1
Rwanda	5.0	4.6	21.7
Senegal	1.0	27.6	22.8
Seychelles	0	0	27.2
Sierra Leone	1.3	12.4	21.9
South Africa	57.1	0	35.1
Swaziland	47	0.2	26.8
Togo	5.8	25.3	29
Uganda	7.7	23.1	23.6
Tanzania	9.3	22.7	26.9
Zambia	16.1	19.4	22.9
Zimbabwe	40.6	0.2	28.1
Average	11.24	14.87	29.11

Source: World Health Organization

## B Solving the Model

### B.1 Net investment under certainty

$$\begin{aligned}\frac{\partial(N_1h_1 + N_2h_2)}{\partial i} &= \frac{\partial N_1}{\partial i}h_1 + \frac{\partial h_1}{\partial i}N_1 + \frac{\partial N_2}{\partial i}h_2 + \frac{\partial h_2}{\partial i}N_2 = (-) + (+) + (+) + (+) = 0 \\ \frac{\partial(N_1h_1 + N_2h_2)}{\partial d} &= \frac{\partial N_1}{\partial d}h_1 + \frac{\partial h_1}{\partial d}N_1 + \frac{\partial N_2}{\partial d}h_2 + \frac{\partial h_2}{\partial d}N_2 = (0) + (+) + (-) + (+) = 0\end{aligned}$$

When  $i$  decreases, fewer children are infected and so both fewer children die and children grow up healthier, while when  $d$  decreases fewer children die but more children grow up unhealthy for a given infection rate, if  $\delta \neq 0$ . Although the number of survivors,  $n(1 - id)$ , is decreasing in  $i$  and  $d$ ,  $\partial N_1/\partial i = \partial n(1 - i)/\partial i = -n < 0$  while  $\partial N_2/\partial i = \partial ni(1 - d)/\partial i = n(1 - d) > 0$ , and  $\partial N_1/\partial d = \partial n(1 - i)/\partial d = 0$  while  $\partial N_2/\partial d = \partial ni(1 - d)/\partial d = -ni < 0$ . Under uncertainty,  $\partial N_1/\partial d \neq 0$  because  $\partial n/\partial d \neq 0$ .

### B.2 Applying the Delta method

$$\begin{aligned}E(U(N_1, N_2, N_3)) &\cong U(E(N)) + (N_1 - E(N_1))U_{N_1}(E(N_1)) + \frac{(N_1 - E(N_1))^2}{2!}U_{N_1N_1}(E(N_1)) \\ &+ (N_2 - E(N_2))U_{N_2}(E(N_2)) + \frac{(N_2 - E(N_2))^2}{2!}U_{N_2N_2}(E(N_2)) \\ &+ (N_3 - E(N_3))U_{N_3}(E(N_3)) + \frac{(N_3 - E(N_3))^2}{2!}U_{N_3N_3}(E(N_3))\end{aligned}$$

$$\begin{aligned}E(U(N_1, N_2, N_3)) &\cong U(n(1 - id)) \\ &+ (N_1 - n(1 - i))U_{N_1}(n(1 - i)) + \frac{(N_1 - n(1 - i))^2}{2!}U_{N_1N_1}(n(1 - i)) \\ &+ (N_2 - ni(1 - d))U_{N_2}(ni(1 - d)) + \frac{(N_2 - ni(1 - d))^2}{2!}U_{N_2N_2}(ni(1 - d)) \\ &+ (N_3 - nid)U_{N_3}(nid) + \frac{(N_3 - nid)^2}{2!}U_{N_3N_3}(nid)\end{aligned}$$

or just

$$\begin{aligned}
E(U(N_1, N_2, N_3)) &\cong U + \frac{(N_1 - n(1-i))^2}{2!} U_{N_1 N_1}(n(1-i)) \\
&\quad + \frac{(N_2 - ni(1-d))^2}{2!} U_{N_2 N_2}(ni(1-d)) \\
&\quad + \frac{(N_3 - nid)^2}{2!} U_{N_3 N_3}(nid)
\end{aligned}$$

where

$$\begin{aligned}
U_{N_1} &= \frac{(1-\beta)(1-\theta)}{N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}}}, & U_{N_1 N_1} &= -\frac{(1-\beta)(1-\theta)}{(N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}})^2} \\
\rightarrow U_{N_1 N_1}(n(1-i)) &= -\frac{(1-\beta)(1-\theta)}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2} \\
E[N_1 - n(1-i)]^2 &= ni(1-i)
\end{aligned}$$

$$\begin{aligned}
U_{N_2} &= \frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{\alpha}{1-\theta}}}{N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}}}, & U_{N_2 N_2} &= -\frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{2\alpha}{1-\theta}}}{(N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}})^2} \\
\rightarrow U_{N_2 N_2}(ni(1-d)(1-i(1-d))) &= -\frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{2\alpha}{1-\theta}}}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2} \\
E[N_2 - ni(1-d)]^2 &= ni(1-d)(1-i(1-d))
\end{aligned}$$

$$U_{N_3} = 0$$

Making these substitutions yields:

$$\begin{aligned}
E(U) &= \beta \ln \left( \frac{\beta(1-\gamma n)wg(H)}{\beta + \theta(1-\beta)} \right) \\
&\quad + (1-\beta) \ln \left( wn^{1-\theta}(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^{1-\theta} \left( \frac{\theta(1-\beta)(1-\gamma n)wg(H)}{\beta + \theta(1-\beta)} \right)^\theta \right) \\
&\quad - \frac{(1-\beta)(1-\theta)i}{2n(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2} [1-i+(1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)(1-i(1-d))]
\end{aligned}$$

I then take the derivative with respect to  $n$ .

### B.3 Fertility response to $i$ and $d$

$$LHS(n) \equiv n - \frac{\gamma(\beta+\theta(1-\beta))n^2}{(1-\beta)(1-\theta)(1-\gamma n)} = \frac{-i[1-i+(1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)[1-i(1-d)]]}{2[1-i+i(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)]^2} \equiv RHS(i, d)$$

The equation is solved implicitly:  $dn/di = RHS_i/LHS_n$  and  $dn/dd = RHS_d/LHS_n$ .

The results for extreme values of  $\delta$  are consistent with the general results but allow for more decisive interpretation.

$RHS_i > 0$  if  $i > i_L$ , where

$$i_L = \frac{1 + (1 - \delta)^{\frac{2\alpha}{1-\theta}}(1 - d)}{1 + (1 - \delta)^{\frac{\alpha}{1-\theta}}(1 - d)[1 + (1 - \delta)^{\frac{\alpha}{1-\theta}}(1 - 2d + (1 - \delta)^{\frac{\alpha}{1-\theta}}(1 - d))]}$$

is increasing in  $\delta$  and  $d$ ;  $RHS_i > 0$  is less likely as  $d$  or  $\delta$  increase. If  $\delta = 1$ ,  $RHS_i < 0$  for all  $i$  and  $d$ . If  $\delta = 0$ ,  $RHS_i > 0$  if  $i > 1/2$  when  $d = 0$ , and  $RHS_i < 0$  when  $d = 1$ .

$RHS_d > 0$  if  $i < i_U$ , where

$$i_U = \frac{1}{4} \left( 2 + (1 - \delta)^{\frac{\alpha}{1-\theta}} - \sqrt{\frac{4 - (1 - \delta)^{\frac{\alpha}{1-\theta}}(4d + (1 - \delta)^{\frac{\alpha}{1-\theta}}(-5 + 4d - (1 - \delta)^{\frac{\alpha}{1-\theta}}(1 - d)))}{1 + (1 - \delta)^{\frac{\alpha}{1-\theta}}(1 - d)}} \right)$$

is decreasing in  $\delta$  and increasing in  $d$ ;  $RHS_d > 0$  is more likely as  $d$  increases and less likely as  $\delta$  increases. If  $\delta = 0$ ,  $RHS_d > 0$  when  $d = 0$  and if  $i < 1/2$  when  $d = 1$ . If  $\delta = 1$ ,  $RHS_d < 0$ .

Since  $RHS(i, d) < 0$ , it must be that  $n > ((1 - \beta)(1 - \theta))/\gamma \equiv \bar{n}$ .  $LHS_n > 0$  if  $n > \underline{n} \equiv (1/\gamma)(1 \pm \sqrt{(\beta + \theta(1 - \beta))})$ , but also  $n < 1/\gamma$  because of the budget constraint. Thus  $\underline{n} \equiv (1/\gamma)(1 - \sqrt{(\beta + \theta(1 - \beta))}) > \bar{n}$  so  $LHS_n < 0$  is not unambiguous, but given the response of  $n$  to  $i$  and  $d$  in 1, I conclude that  $LHS_n < 0$  for the assumptions about  $\beta$  and  $\theta$ . Note that  $\underline{n} = 0$  when  $\beta = 1$  or when  $\theta = 1$ .

## B.4 Human capital response to $i$ and $d$

$$h_1 = \frac{\theta(1-\beta)(1-\gamma n)w_t g(H_t)}{(\beta + \theta(1-\beta))(N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}})}$$

$$\frac{\partial h_1}{\partial i} = \frac{-\frac{dn}{di}}{n(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})} - \frac{(1-n)(n(-1+(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})+(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})\frac{dn}{di})}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}$$

which is negative if

$$\frac{dn}{di} > \frac{n(1-n)(1-(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})}{(2-n)(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})} > 0$$

And similarly for  $\partial h_1 / \partial d$ .

## C Data and Regression Results

Countries: Angola, Benin, Botswana, Burkina Faso, Burundi, Cameroon, Central African Republic, Chad, Comoros, Congo Democratic Republic, Congo Republic, Cote D'Ivoire,

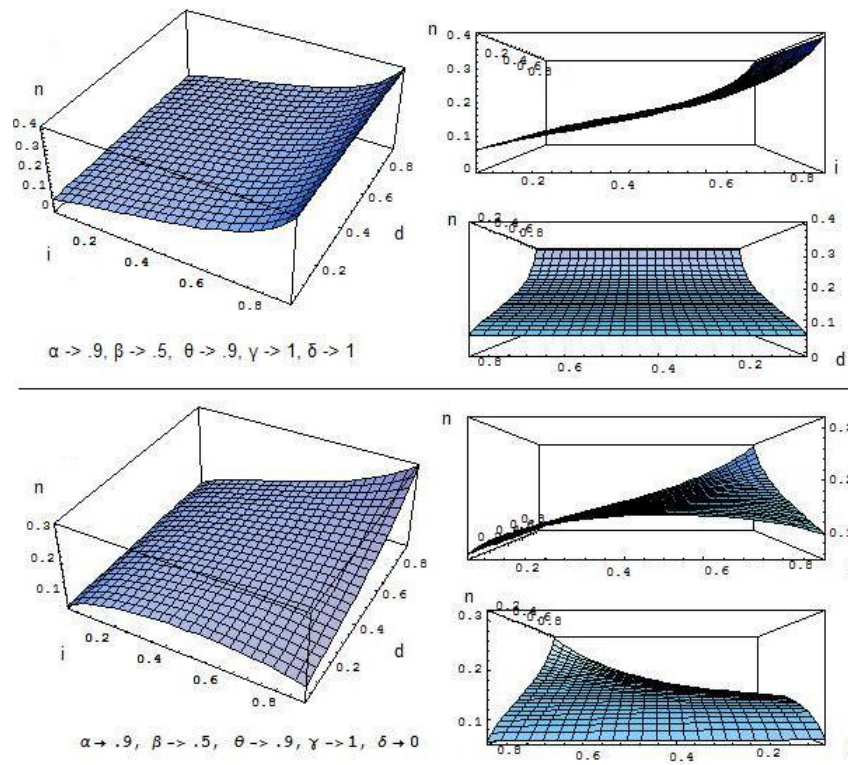


Figure 1: The optimal value of  $n$  for extreme values of  $\delta$

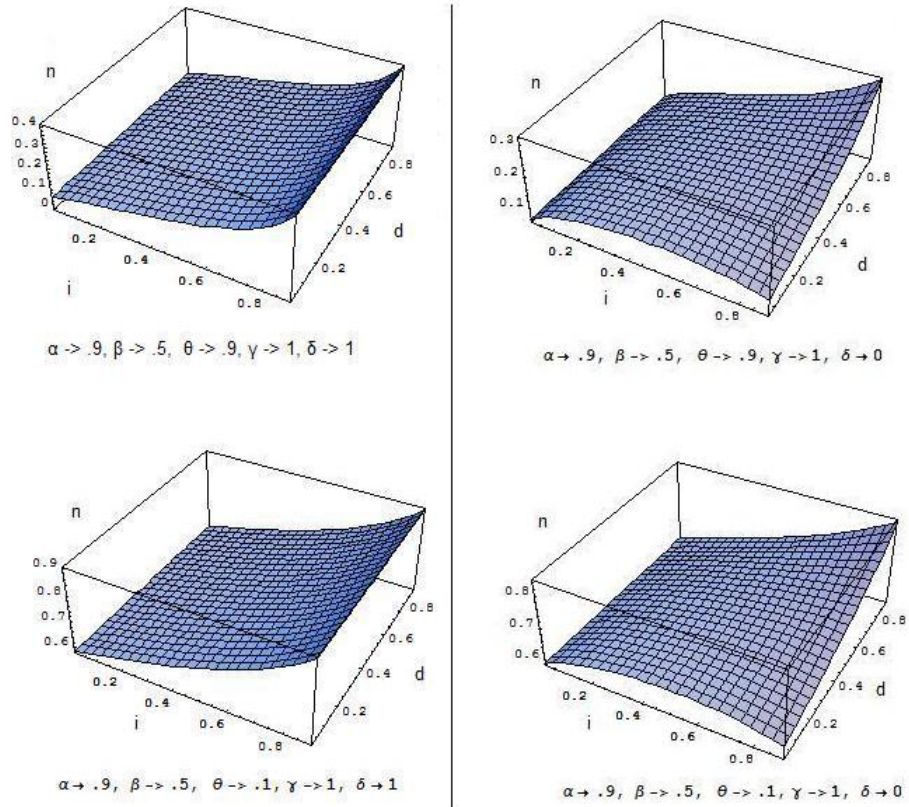


Figure 2: The optimal value of  $n$  for extreme values of  $\delta$  and  $\theta$

Equatorial Guinea, Eritrea, Ethiopia, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Kenya, Lesotho, Liberia, Madagascar, Malawi, Mali, Mauritania, Mauritius, Mozambique, Namibia, Niger, Nigeria, Rwanda, Senegal, Seychelles, Sierra Leone,<sup>23</sup> South Africa, Sudan, Swaziland, Tanzania, Togo, Uganda, Zambia, Zimbabwe.

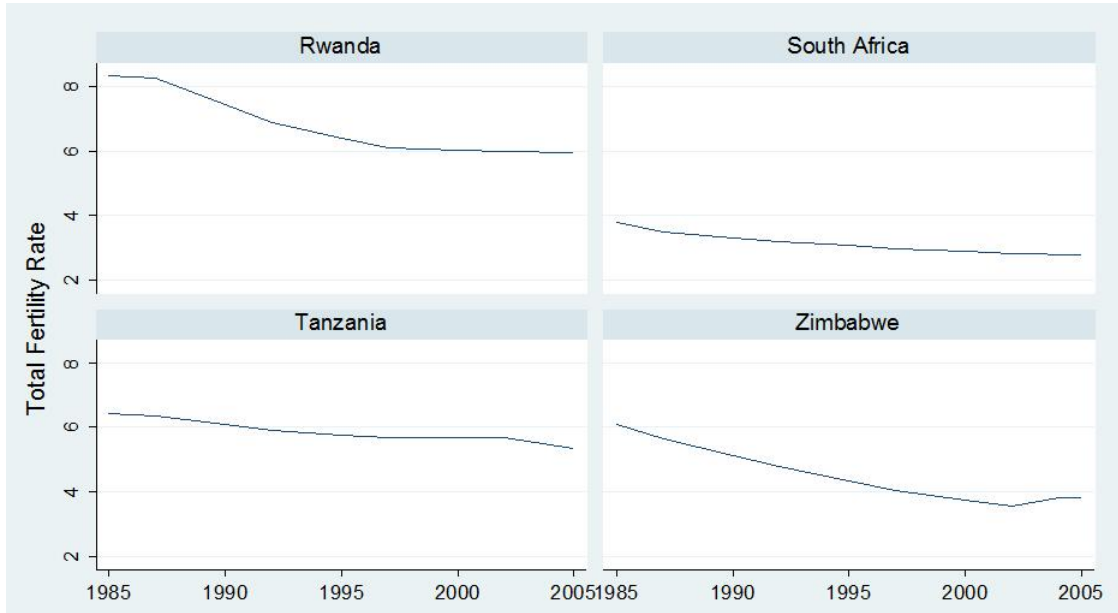


Figure 3: Changes in total fertility rates across time for select countries

<sup>23</sup>There is only one data point for HIV prevalence for Sierra Leone in 2003, so regressions limited to a time span of 1985-2000 omit Sierra Leone.

Table 3: Descriptive Statistics

Variable		Mean	Std. Dev.	Min	Max	Observations
HIV	overall	9.536	9.607	0	55.002	N=525
	between		7.355	0.005	26.379	n=44
	within		6.256	-16.843	49.562	T-bar=11.9318
iHIV	overall	8.963	9.405	0	55.002	N=705
	between		7.188	0.002	24.455	n=44
	within		6.120	-14.313	48.990	T-bar=16.0227
Malaria rate	overall	9.795	11.399	0.003	94.577	N=663
	between		9.232	0.006	37.635	n=42
	within		7.555	-15.516	78.485	T-bar=15.7857
iMalaria rate	overall	9.684	11.311	0.003	94.577	N=795
	between		8.693	0.006	35.714	n=42
	within		7.403	-15.942	79.554	T-bar=18.9286
Female literacy rate	overall	49.470	25.208	8.058	92.259	N=61
	between		23.956	12.024	90.300	n=40
	within		5.084	37.116	61.824	T-bar=1.525
iFemale literacy rate	overall	52.081	24.647	8.058	92.259	N=259
	between		23.995	11.087	90.300	n=40
	within		3.848	39.727	64.435	T-bar=6.475
GDP per capita	overall	919.713	1508.711	62.890	15549.690	N=903
	between		1322.649	154.942	6327.572	n=44
	within		731.657	-2902.108	13939.720	T-bar=20.5227
iGDP per capita	overall	912.300	1501.103	62.890	15549.690	N=914
	between		1322.573	154.942	6327.572	n=44
	within		727.244	-2909.521	13932.310	T-bar=20.7727
Infant mortality rate	overall	9.564	3.508	1.217	19.100	N=682
	between		3.457	1.422	16.828	n=43
	within		0.714	6.566	12.333	T=15.8605
iInfant mortality rate	overall	9.564	3.493	1.217	19.100	N=688
	between		3.457	1.422	16.828	n=43
	within		0.712	6.566	12.333	T=16
Under 5 mortality rate	overall	15.645	6.410	1.330	32.000	N=217
	between		6.204	1.756	29.220	n=44
	within		1.750	9.401	21.381	T-bar=4.93182
iUnder 5 mortality rate	overall	15.600	6.348	1.330	32.000	N=919
	between		6.243	1.726	29.300	n=44
	within		1.514	9.308	21.288	T-bar=20.8864
Adult mortality rate	overall	38.948	10.362	16.283	74.150	N=682
	between		9.154	17.784	56.266	n=43
	within		5.044	11.282	62.045	T=15.8605
iAdult mortality rate	overall	38.866	10.354	16.283	74.150	N=688
	between		9.154	17.784	56.266	n=43
	within		5.023	11.200	61.963	T=16
Total fertility rate	overall	5.526	1.468	1.910	8.350	N=422
	between		1.223	2.095	7.678	n=44
	within		0.517	4.028	7.477	T-bar=9.59091
iTotal fertility rate	overall	5.692	1.293	1.910	8.350	N=924
	between		1.211	2.095	7.678	n=44
	within		0.488	4.228	7.501	T=21
iPrimary school completion rate	overall	49.138	23.476	10.474	120.702	N=548
	between		23.784	18.511	114.980	n=42
	within		7.209	14.200	80.665	T-bar=13.0476
iPrimary school enrollment rate (gross)	overall	83.641	27.891	19.501	172.859	N=622
	between		25.757	34.268	150.614	n=44
	within		10.905	36.946	133.503	T-bar=14.1364
iPrimary school enrollment rate (net)	overall	60.517	19.571	14.710	99.401	N=510
	between		17.916	29.378	95.524	n=43
	within		7.899	26.867	93.038	T-bar=11.8605

Prefix "i" denotes interpolated variables.

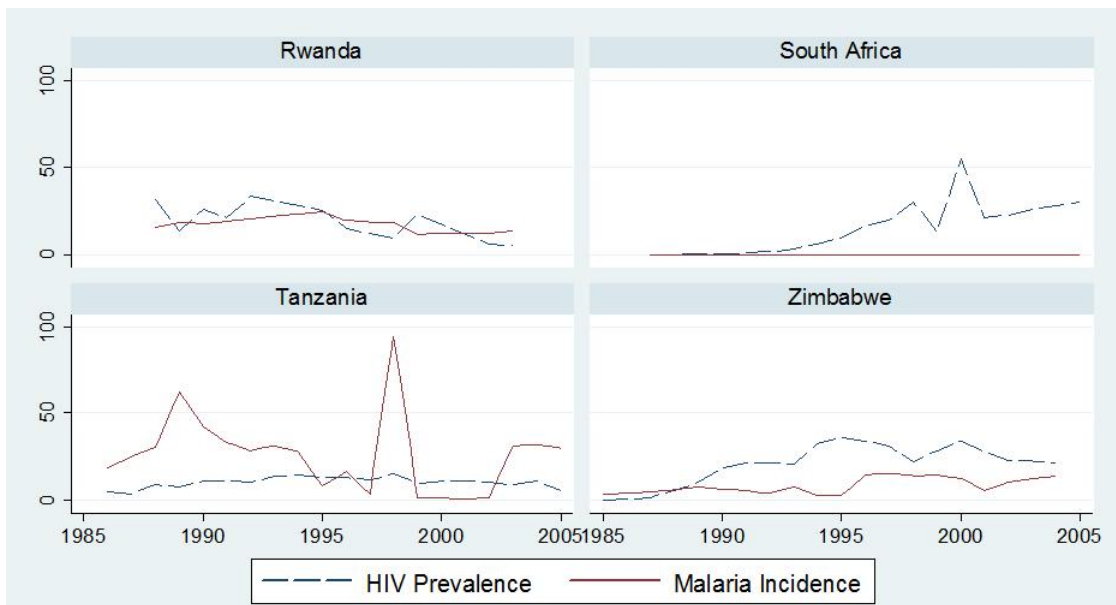


Figure 4: Changes in HIV prevalence and malaria incidence across time for select countries

Table 4: HIV: Kalemli-Ozcan specification panel regressions of total fertility rate with all countries

	1	2	3	4	5	6	7	8
	1985-2005				1985-2000			
Log(HIV)	-0.110***	-0.0964***	-0.307***	-0.150***	-0.0997**	-0.0855**	-0.304***	-0.0968***
Log(Infant Mortality)	-0.04	-0.0365	-0.0515	-0.0362	-0.0434	-0.0404	-0.0601	-0.0362
Log(Female Literacy)	-0.347*	-0.412**	-0.298	1.581***	-0.115	-0.19	0.00912	1.509***
Log(GDP per capita)	-0.2	-0.184	-0.324	-0.136	-0.264	-0.253	-0.427	-0.146
Constant	0.327	0.3	-1.707***	-0.205***	0.949***	0.907***	-1.618***	-0.239***
N	-0.249	-0.225	-0.175	-0.0531	-0.335	-0.339	-0.27	-0.0511
R <sup>2</sup>	0.035	0.00143	-0.200**	-0.499***	0.175**	0.124*	-0.168**	-0.477***
	-0.076	-0.0606	-0.085	-0.0543	-0.0738	-0.0651	-0.079	-0.0582
	4.139***	179.7***	14.46***	6.398***	0.799	205.6***	13.30***	6.476***
	-1.512	-14.55	-1.053	-0.597	-1.803	-18.09	-1.602	-0.645
	214	214	214	214	181	181	181	181
	0.991	0.991	0.979	0.863	0.992	0.992	0.982	0.867
Log(HIV)	-0.144***	-0.133***	-0.154***	0.0552	-0.146***	-0.130***	-0.235***	0.104**
Infant Mortality	-0.038	-0.0336	-0.0531	-0.0467	-0.0445	-0.0407	-0.0617	-0.0499
Female Literacy	-0.023	-0.0293	-0.0765***	0.168***	0.00888	0.000343	-0.00755	0.154***
Log(GDP per capita)	-0.024	-0.0226	-0.0288	-0.0284	-0.0339	-0.0311	-0.0445	-0.0361
Constant	0.031***	0.0308***	-0.0599***	-0.0124***	0.0336***	0.0329***	-0.0466***	-0.0134***
N	-0.008	-0.00797	-0.00861	-0.00241	-0.00859	-0.0085	-0.00981	-0.00274
R <sup>2</sup>	0.033	0.0107	-0.138	-0.529***	0.118*	0.0743	-0.0528	-0.516***
	-0.069	-0.0564	-0.0958	-0.0599	-0.0692	-0.0617	-0.091	-0.0697
	3.327***	220.0***	10.36***	7.813***	2.801***	220.9***	8.756***	7.837***
	-0.727	-16.42	-0.493	-0.598	-0.747	-18.8	-0.668	-0.739
	214	214	214	214	181	181	181	181
	0.992	0.992	0.98	0.834	0.993	0.992	0.982	0.832
Country FE	Yes	Yes	Yes	No	Yes	Yes	Yes	No
Year FE	Yes	No	No	Yes	Yes	No	No	Yes
Time trend	No	Yes	No	No	No	Yes	No	No

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 5: HIV: Kalemli-Ozcan specification panel regressions of total fertility rate with restricted country sample

	1	2	3	4	5	6	7	8
	1985-2005				1985-2000			
Log(HIV)	0.0159	0.0212	-0.234***	-0.258***	0.0828	0.0864	-0.146*	-0.212***
Log(Infant Mortality)	-0.0585	-0.0533	-0.063	-0.0415	-0.0639	-0.0581	-0.0816	-0.0403
Log(Female Literacy)	-0.686***	-0.762***	-0.589**	1.998***	-0.820***	-0.828***	-0.416	1.953***
Log(GDP per capita)	-0.14	-0.111	-0.26	-0.128	-0.19	-0.165	-0.358	-0.129
Constant	0.398	0.23	-1.640***	-0.262***	0.789*	0.725*	-1.715***	-0.311***
	-0.316	-0.252	-0.211	-0.0464	-0.416	-0.382	-0.311	-0.0457
	0.181*	0.0628	-0.246**	-0.363***	0.194*	0.146*	-0.274**	-0.332***
	-0.0997	-0.0796	-0.11	-0.0451	-0.108	-0.0839	-0.11	-0.0485
	3.431*	182.6***	14.75***	5.083***	2.438	211.7***	14.67***	5.140***
	-1.756	-17.68	-0.951	-0.515	-2.112	-22.04	-1.62	-0.541
N	133	133	133	133	110	110	110	110
R <sup>2</sup>	0.995	0.995	0.988	0.956	0.995	0.995	0.989	0.96
Log(HIV)	-0.124**	-0.0832***	0.0164	-0.0157	-0.0777	-0.0552	-0.0502	0.015
Infant Mortality	-0.0362	-0.029	-0.0745	-0.0439	-0.0496	-0.0435	-0.113	-0.0472
Female Literacy	-0.0327	-0.0476**	-0.108***	0.249***	-0.0211	-0.0273	-0.0304	0.251***
	-0.022	-0.0205	-0.034	-0.026	-0.0329	-0.0297	-0.0667	-0.0341
	0.0397***	0.0346***	-0.0562***	-0.0157***	0.0401***	0.0371***	-0.0432***	-0.0177***
	-0.0104	-0.00975	-0.0106	-0.00236	-0.0103	-0.00991	-0.0123	-0.00281
	0.248***	0.119*	-0.197	-0.357***	0.227**	0.154*	-0.122	-0.308***
	-0.0904	-0.0665	-0.144	-0.0535	-0.1	-0.0831	-0.132	-0.063
	1.630*	237.5***	10.26***	6.253***	2.007**	239.0***	8.641***	6.014***
	-0.917	-20.38	-0.623	-0.541	-0.986	-23.4	-1.085	-0.687
	133	133	133	133	110	110	110	110
R <sup>2</sup>	0.996	0.995	0.985	0.936	0.996	0.996	0.987	0.941
Country FE	Yes	Yes	Yes	No	Yes	Yes	Yes	No
Year FE	Yes	No	No	Yes	Yes	No	No	Yes
Time trend	No	Yes	No	No	No	Yes	No	No

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 6: Panel Regressions of Total fertility rate on HIV 1985-2005 with child mortality interaction

	1	2	3	4	5	6	7	8
Log(HIV)	-1.490***	-1.371***	-0.0453	0.053	-0.116***	-0.103***	-0.172***	0.0153
Log(HIV)Log(Child Mortality)	-0.33	-0.332	-0.314	-0.146	-0.0377	-0.0338	-0.0365	-0.0471
Log(Child Mortality)	0.520***	0.480***	-0.0469	-0.016				
	-0.119	-0.12	-0.112	-0.055				
Log(Adult Mortality)	-1.943***	-1.890***	0.607**	1.493***	-0.191	-0.291	0.480*	1.485***
	-0.389	-0.393	-0.262	-0.122	-0.261	-0.222	-0.278	-0.124
Log(Female Literacy)	0.645***	0.648***	-1.282***	-0.666**	-0.111	-0.0423	-1.259***	-0.661**
	-0.247	-0.238	-0.13	-0.294	-0.223	-0.194	-0.153	-0.292
Log(GDP per capita)	0.749***	0.729***	-1.223***	-0.219***	0.258	0.259	-1.232***	-0.220***
	-0.264	-0.264	-0.167	-0.048	-0.267	-0.254	-0.173	-0.047
Constant	0.072	0.037	-0.139*	-0.455***	0.0336	-0.00157	-0.140*	-0.448***
	-0.08	-0.0654	-0.071	-0.067	-0.0767	-0.0613	-0.0713	-0.0539
	4.059***	236.4***	14.43***	7.730***	4.631***	173.5***	14.73***	7.677***
	-1.491	-24.94	-0.77	-1.023	-1.641	-19.44	-0.766	-0.972
Country FE	Yes	Yes	Yes	No	Yes	Yes	Yes	No
Year FE	Yes	No	No	Yes	Yes	No	No	Yes
Time trend	No	Yes	No	No	No	Yes	No	No
N	214	214	214	214	214	214	214	214
R <sup>2</sup>	0.993	0.992	0.986	0.876	0.991	0.991	0.986	0.876
% of sample with negative predicted $\partial TFR / \partial HIV < 0$								
	61	60.6			100	100	100	

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 7: Panel Regressions of Total fertility rate on HIV 1985-2005 with child and adult mortality interactions

	1	2	3	4	5	6	7	8
Log(HIV)	-0.259	-0.17	0.929*	3.759***	-0.888	-0.817	0.702	2.255***
Log(HIV)Log(Child Mortality)	-0.457	-0.432	-0.556	-0.602	-0.594	-0.589	-0.598	-0.482
Log(HIV)Log(Adult Mortality)	0.778***	0.734***	0.149	0.318***				
Log(Child Mortality)	-0.179	-0.182	-0.15	-0.0731	0.218	0.202	-0.245	-0.655***
Log(Adult Mortality)	-0.542***	-0.528***	-0.422**	-1.315***	-0.166	-0.164	-0.165	-0.144
Log(Female Literacy)	-0.19	-0.182	-0.199	-0.217	-0.344	-0.417**	0.569**	0.983***
Log(GDP per capita)	-2.434***	-2.405***	0.227	0.32	-0.21	-0.184	-0.264	-0.186
Constant	-0.525	-0.541	-0.307	-0.235	-0.629	-0.531	-0.565	1.343**
Country FE	2.304***	2.292***	0.00647	3.471***	-0.515	-0.488	-0.563	-0.582
Year FE	-0.713	-0.694	-0.629	-0.804	0.375	0.368	-1.241***	-0.219***
Time trend	0.703***	0.692***	-1.276***	-0.230***	-0.295	-0.284	-0.176	-0.0492
N	-0.257	-0.255	-0.169	-0.047	0.042	0.00809	-0.140**	-0.486***
R <sup>2</sup>	0.0706	0.0321	-0.145**	-0.377***	-0.0796	-0.0639	-0.0698	-0.0562
% of sample with negative predicted $\partial TFR/\partial HIV < 0$	-0.0772	-0.0639	-0.0702	-0.056	6.278***	186.5***	12.10***	2.36
Robust standard errors in parentheses; *** p<0.01, ** p<0.05, * p<0.1	-0.309	235.6***	11.13***	-4.071*	-1.824	-23.27	-1.78	-1.618
	-2.167	-24.37	-1.944	-2.345	Yes	Yes	Yes	No
	Yes	Yes	Yes	No	Yes	No	No	Yes
	Yes	No	No	Yes	No	Yes	No	No
	No	Yes	No	No	No	Yes	No	No
	214	214	214	214	214	214	214	214
	0.993	0.993	0.986	0.893	0.991	0.991	0.986	0.885
	31	37.2	100	70.9				78.8

Table 8: Panel Regressions of Total fertility rate on Malaria 1985-2005

	1	2	3	4	5	6	7	8
Log(Malaria)	-0.246**	-0.239**	-0.0213	0.0175	-0.0497***	-0.0467***	-0.0620***	0.0624***
Log(Malaria)*Log(Child Mortality)	-0.109	-0.108	-0.13	-0.0524	-0.0172	-0.0154	-0.0192	-0.0204
Log(Child Mortality)	0.073*	0.0717*	-0.0151	0.0221				
	-0.038	-0.0386	-0.0468	-0.0231				
Log(Adult Mortality)	-0.504**	-0.536**	0.502**	1.306***	-0.424**	-0.458**	0.491*	1.243***
	-0.216	-0.209	-0.253	-0.157	-0.21	-0.196	-0.258	-0.133
Log(Female Literacy)	0.087	0.118	-1.445***	-0.502**	-0.0212	0.0129	-1.432***	-0.523***
	-0.2	-0.194	-0.141	-0.197	-0.196	-0.183	-0.138	-0.199
Log(GDP per capita)	0.405*	0.406*	-1.139***	-0.281***	0.379	0.383*	-1.144***	-0.285***
	-0.23	-0.217	-0.167	-0.042	-0.232	-0.22	-0.168	-0.0411
Constant	0.014	-0.00322	-0.139*	-0.479***	0.026	0.00489	-0.141**	-0.506***
	-0.072	-0.0557	-0.0706	-0.0569	-0.0727	-0.0561	-0.0693	-0.0446
	4.052***	187.3***	14.68***	7.849***	4.328***	182.2***	14.68***	8.313***
	-1.432	-17.51	-0.756	-0.864	-1.436	-17.21	-0.762	-0.66
Country FE	Yes	Yes	Yes	No	Yes	Yes	Yes	No
Year FE	Yes	No	No	Yes	Yes	No	No	Yes
Time trend	No	Yes	No	No	No	Yes	No	No
N	223	223	223	223	223	223	223	223
R <sup>2</sup>	0.993	0.993	0.987	0.896	0.993	0.993	0.987	0.895
% of sample with negative predicted $\partial TFR / \partial Malaria < 0$								
	98.3	97.3			100	100		

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 9: Panel regressions of primary school completion rate

	1	2	3	4	5	6	7
TFR	-10.963**	-10.55**	-9.062*	-6.71	-10.40*	-9.417*	-10.46*
Log(Child Mortality)	-4.661	-4.74	-5.318	-5.49	-5.283	-5.177	-5.317
	-5.389	-3.447	11.79	32.16	-5.572	-0.709	-3.546
Log(Female Literacy)	-12.321	-12.83	-26.64	-28.58	-12.13	-11.83	-12.51
	-10.609	-7.197	-11.68	-7.986	-11.57	-12.82	-7.319
	-11.927	-12.63	-15.08	-13.81	-12.64	-12.6	-13.52
Log(GDP per capita)	4.969	5.717	5.135	5.456	4.942	5.737	5.736
	-5.581	-5.618	-5.74	-5.153	-5.703	-5.628	-5.747
Log(Adult Mortality)	-13.376	-13.16	-18.88	-100.5**	-13.48	-19.49	-13.09
	-14.272	-14.65	-18.09	-46.45	-14.03	-13.58	-14.3
Log(HIV)		0.213	11.81	-52.64			0.202
		-1.866	-15.37	-32.71			-1.916
Log(HIV)*Log(Child Mortality)			-4.428	-15.92*			
Log(HIV)*Log(Adult Mortality)			-5.863	-8.629			
			27.12**				
				-13.04			
Log(Malaria)					0.153	9.653	0.0457
					-0.865	-6.571	-0.913
Log(Malaria)*Log(Child Mortality)						-3.517	
						-2.589	
Constant	175.644***	156.9**	154.3**	357.6**	176.4***	180.4***	156.7**
	-64.632	-78.35	-76.21	-137	-65.09	-65.06	-77.78
N	192	177	177	177	186	186	174
R <sup>2</sup>	0.96	0.955	0.956	0.959	0.955	0.956	0.954
% of sample with predicted $\partial Completion\ rate/\partial HIV < 0$							0

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 10: Panel regressions of primary school completion rate without total fertility rate

	1	2	3	4	5	6	7
Log(Child Mortality)	-2.599	-1.108	29.18	46.46*	-5.028	0.57	-3.175
Log(Female Literacy)	-12.17	-12.74	-23.77	-25.32	-12.13	-11.88	-12.59
	-16.17	-13.02	-20.47	-13.95	-18.01	-18.75	-14.33
Log(GDP per capita)	-11.92	-12.94	-13.73	-13.47	-12.08	-11.97	-13.14
	3.404	4.245	3.483	4.301	3.903	4.942	4.79
Log(Adult Mortality)	-5.364	-5.439	-5.251	-4.859	-5.656	-5.601	-5.729
	-10.53	-11.16	-23.36	-111.0***	-10.16	-17.52	-10.35
	-14.03	-14.43	-16.76	-42.29	-13.98	-13.5	-14.3
Log(HIV)		0.587	24.05*	-49.68			0.344
		-1.812	-13.33	-33.38			-1.89
Log(HIV)*Log(Child Mortality)			-8.997*	-20.23***			
Log(HIV)*Log(Adult Mortality)			-5.222	-7.479			
			29.56**				
			-12.31				
Log(Malaria)					0.888	11.86*	0.803
					-0.754	-6.491	-0.789
Log(Malaria)*Log(Child Mortality)						-4.091	
						-2.548	
Constant	147.4*	128	131.1*	359.3***	153.2*	165.0**	130.4
	-77.17	-81.26	-77.57	-131.1	-78.14	-77.35	-81.68
N	192	177	177	177	186	186	174
R <sup>2</sup>	0.958	0.953	0.954	0.958	0.953	0.954	0.952
% of sample with predicted $\partial \text{Completion rate} / \partial \text{Malaria} < 0$							
% of sample with predicted $\partial \text{Completion rate} / \partial \text{HIV} < 0$			46.1	0			34.9

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 11: Panel regressions of primary school net enrollment rate

	1	2	3	4	5	6	7
TFR	-9.420**	-11.57***	-11.32**	-9.979**	-10.91***	-10.10**	-12.52***
Log(Child Mortality)	-4.366	-4.404	-4.955	-4.611	-4.169	-3.97	-4.304
	-39.94***	-42.84***	-40.07*	-27.95	-38.17***	-33.76**	-41.63***
Log(Female Literacy)	-14.3	-13.9	-23.43	-20.35	-13.96	-13.18	-13.79
	5.845	-2.607	-3.38	-2.291	7.381	6.702	-0.925
	-6.22	-6.707	-9.131	-9.47	-5.93	-5.791	-6.415
Log(GDP per capita)	-0.623	-2.333	-2.374	-2.132	-0.883	-0.313	-2.418
	-3.022	-3.162	-3.185	-2.984	-3.019	-2.97	-3.128
Log(Adult Mortality)	14.26	17.54	16.07	-21.63	12.62	6.674	16.25
	-12.45	-12.5	-16.7	-25.83	-11.98	-11.65	-12.31
Log(HIV)		-2.466**	-0.33	-27.46			-2.196*
		-1.191	-11.57	-26.06			-1.197
Log(HIV)*Log(Child Mortality)			-0.779	-6.297			
Log(HIV)*Log(Adult Mortality)			-4.17	-5.395			
			11.83	11.83			
			-9.105	-9.105			
Log(Malaria)					-0.782	6.493	-0.496
					-0.51	-4.628	-0.498
Log(Malaria)*Log(Child Mortality)					-2.691	-1.82	
Constant	149.4***	203.7***	199.7***	288.4***	157.2***	162.4***	199.7***
	-46.46	-49.29	-50.31	-92.26	-45.98	-47.38	-49.14
N	177	164	164	164	173	173	162
R <sup>2</sup>	0.965	0.964	0.964	0.965	0.964	0.965	0.963
% of sample with predicted $\partial Net\ enrollment\ rate / \partial HIV < 0$							
		100	100	100	100	100	100

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 12: Panel regressions of primary school net enrollment rate without total fertility rate

	1	2	3	4	5	6	7
Log(Child Mortality)	-32.03**	-33.05***	-17.35	-4.106	-31.36**	-26.80**	-33.63***
Log(Female Literacy)	-13.25	-12.62	-20.44	-18.72	-13	-12.34	-12.75
Log(GDP per capita)	3.312	-4.787	-9.248	-6.738	3.45	2.999	-5.219
Log(Adult Mortality)	-6.724	-7.305	-9.2	-9.55	-6.62	-6.385	-7.237
Log(HIV)	-0.794	-2.281	-2.54	-2.172	-0.954	-0.282	-2.258
Log(HIV)*Log(Child Mortality)	-3.023	-3.161	-3.135	-2.89	-3.049	-2.992	-3.158
Log(HIV)*Log(Adult Mortality)	11.18	12.9	4.48	-46.59	10.73	3.948	13.38
Log(HIV)*Log(Child Mortality)	-12.15	-12.13	-15.84	-28.43	-11.88	-11.46	-12.24
Log(HIV)*Log(Adult Mortality)	-1.957	-1.187	11.07	-28.98	-2.047	-1.236	-1.236
Log(Malaria)							
Log(Malaria)*Log(Child Mortality)							
Constant	107.0**	142.3***	151.4***	284.2***	100.6**	113.5**	143.2***
N	-42.96	-45.09	-47.85	-93.71	-42.1	-43.46	-45.08
R <sup>2</sup>	177	164	164	164	173	173	162
% of sample with predicted $\partial Net enrollment rate / \partial Malaria < 0$	0.963	0.961	0.961	0.963	0.961	0.963	0.96
% of sample with predicted $\partial Net enrollment rate / \partial HIV < 0$							
							60.1

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 13: Panel regressions of primary school gross enrollment rate

	1	2	3	4	5	6	7
TFR	16.18***	15.57***	17.96***	17.77***	13.67*	15.10**	12.98*
Log(Child Mortality)	-6.051	-5.791	-6.264	-6.305	-7.025	-6.814	-6.825
	-4.794	-1.473	20.09	17.84	-13.89	-5.878	-10.42
Log(Female Literacy)	-11.78	-11.89	-22.64	-20.99	-12.67	-11.75	-12.67
	18.25	6.95	-0.0996	-0.102	15.45	13.07	5.636
Log(GDP per capita)	-11.78	-12.9	-15.62	-15.63	-11.82	-10.88	-12.92
	5.764	4.588	4.018	3.969	6.546	7.462*	5.812
Log(Adult Mortality)	-4.366	-4.669	-4.567	-4.507	-4.599	-4.451	-4.869
	-7.18	-12.34	-21.39	-14.68	-3.283	-13.26	-8.188
Log(HIV)	-11.82	-12.14	-15.17	-40.67	-12.21	-10.84	-12.49
		-2.732	13.96	18.59			-2.7
		-2.001	-13.42	-35.94			-1.933
Log(HIV)*Log(Child Mortality)			-6.186	-5.117			
Log(HIV)*Log(Adult Mortality)			-5.041	-7.08			
				-2.1			
				-13.31			
Log(Malaria)					1.328	18.75**	1.467
Log(Malaria)*Log(Child Mortality)					-1.006	-7.246	-1.027
						-6.473**	
						-2.696	
Constant	-77.98	-14.38	-23.85	-40.12	-51.9	-39.97	25.9
	-79.29	-81.36	-78.68	-136.9	-80.37	-77.54	-83.65
N	220	202	202	202	212	212	197
R <sup>2</sup>	0.949	0.937	0.937	0.937	0.949	0.951	0.935
% of sample with predicted $\partial Gross\ enrollment\ rate / \partial Malaria < 0$							35.4

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 14: Panel regressions of primary school gross enrollment rate without total fertility rate

	1	2	3	4	5	6	7
Log(Child Mortality)	-8.745	-3.583	-13.51	-22.64	-19.56	-13.06	-14.83
Log(Female Literacy)	-13.53	-13.4	-22.88	-23.7	-13.57	-12.44	-13.12
	25.54**	11.91	14.85	14.08	21.66*	20.15**	10.35
Log(GDP per capita)	-11	-11.9	-15.16	-15.34	-10.97	-10.14	-11.98
	7.42	6.046	6.208	5.864	7.845	8.768*	6.877
	-4.798	-5.107	-5.254	-5.154	-4.976	-4.892	-5.24
Log(Adult Mortality)	-10.84	-15.84	-11.36	20.37	-4.063	-12.88	-9.009
	-11.66	-12.21	-15.01	-44.61	-12.05	-10.54	-12.4
Log(HIV)		-4.551*	-12.22	11.31			-3.645*
		-2.35	-17.07	-39.5			-2.143
Log(HIV)*Log(Child Mortality)			2.891	7.57			
Log(HIV)*Log(Adult Mortality)			-6.147	-8.507			
			-10.09	-10.09			
Log(Malaria)				-14.38			
					0.74	15.96**	0.951
					-0.88	-6.662	-0.92
Log(Malaria)*Log(Child Mortality)						-5.674**	
						-2.5	
Constant	3.106	81.17	77.11	-5.977	17.14	36.48	80.8
	-70.01	-75.91	-80.63	-146.8	-70.55	-67.69	-74.53
N	220	202	202	202	212	212	197
R <sup>2</sup>	0.944	0.931	0.931	0.932	0.946	0.948	0.932
% of sample with predicted $\partial Gross\ enrollment\ rate/\partial Malaria < 0$							42.8
% of sample with predicted $\partial Gross\ enrollment\ rate/\partial HIV < 0$							100
							100

Robust standard errors in parentheses; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1