Childhood Disease and the Precautionary Demand for Children[1]

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Abstract

The childhood disease burden depends on the prevalence of infectious diseases, their case fatalities, and long-term morbidity. We propose a quantity-quality model of fertility choice under uncertainty that emphasizes morbidity and mortality from infectious disease. The fertility response to a decline in child mortality depends on the morbidity effect of the disease, the prevalence rate, and whether the prevalence or case fatality rate declines. Fertility follows mortality and morbidity but since mortality and morbidity do not always move in the same direction, the fertility response may be dampened or non-monotonic. Disease-specific evidence from sub-Saharan Africa supports these theoretical predictions.

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JEL Classification: I12, J11, O12

1 Introduction

A reduction in child mortality preceded the fertility transition in many early industrializers like England, Germany and Sweden. England's declining child mortality since 1870, for example, was followed about five years later by declining total and net fertility rates (Arora, 2005). Declining child mortality has also been instrumental in fertility transitions across many countries during 1960-2000 (Angeles, 2010).

Under-five mortality rates have dropped worldwide by more than half since 1960 and this has coincided with a global decline in the total fertility rate (Figure 1).[1] Yet this connection has been tenuous for sub-Saharan Africa (SSA) where the total fertility rate (TFR) has remained high and significantly lagged improvements in child survival: of the thirty nine countries with TFR exceeding 4 in the World Factbook 2012-13, all but three are from SSA. Figure 2 illustrates the case of several countries that have experienced high, sometimes increasing, fertility despite substantial decline in child mortality. Gambia's TFR, for instance, increased from 5.6 to 6.4 during 1960-1980 despite the child mortality rate falling from 360 to 214 per 1,000 live births. Even as Uganda's child mortality rate fell from 222 to 128 per 1,000 live births during 1960-2009, its TFR fell only slightly, from 7 to 6.25 (World Development Indicators).

Insert Figures 1 & 2 here

We identify infectious disease morbidity as the source of Africa's slow fertility transition. In our model parents are uncertain whether their children will contract infectious diseases in early childhood and whether infected children will succumb to them. Even when infected children survive, chronic illness during the early years leaves a permanent mark on their long-term health. This link between infectious disease morbidity and human capital rests on a large body of bio-medical evidence that finds infectious disease in early childhood to hamper cognitive and physical development and predispose people towards weaker health. The morbidity effect from infection lowers the marginal return from parental investment on unhealthy children. Survivors of infectious disease are, consequently, lower quality than children who never (significantly) contracted them in the early years after birth.

How a decline in child mortality affects fertility behavior depends on whether mortality falls due to improvement in treatment or in disease prevalence. Besides lowering uncertainty, the latter ensures that more surviving children are of higher quality and, consequently, strongly affects fertility choice. When mortality falls due to better survival from infectious disease, in contrast, a larger proportion of surviving children are of lower quality and the average cost

[1] See also `http://www.unicef.org/progressforchildren/2007n6/index_41802.htm`

of producing a surviving child falls. In this case a fall in mortality does not necessarily lower fertility.

Children continue to experience chronic illness from SSA's major infectious diseases. In the tropical and sub-tropical climate that make much of the region hospitable to a plethora of infections, cures or treatments for some diseases ensure that children survive from them only to suffer from others. Anthropometric evidence shows how persistent this disease burden has been. England and Wales' decline of infectious disease mortality which started in 1872 was followed 1890 onwards by a sharp upward trend in the height of 18-year-old males (Arora, 2005). Africa's mortality decline, in contrast, has not generated comparable gains in adult stature suggesting childhood morbidity has not improved much (Akachi and Canning, 2008).

The theoretical model presented in this paper includes both positive and negative fertility responses to the disease burden. In Barro and Becker (1988), a decline in child mortality reduces the average cost of raising a surviving child. The demand for children rises but temporarily: net fertility returns to its original level (unless mortality continues to decline) but fewer births are required to achieve that target. Hence the Barro-Becker model generates an inverted U-shaped population path when child mortality falls.

Adaptations of the Barro-Becker model in the literature feature an unambiguously positive response of the TFR to mortality.[2] The average cost effect is either not incorporated or, when it is, completely overshadowed by positive precautionary forces. The theory proposed here includes the Barro-Becker average cost effect and determines circumstances in which it dominates the forces (precautionary demand, quantity-quality tradeoff) driving a positive response of fertility to child mortality. By separating infection and case fatality rates, we introduce an additional but *positive* average cost effect that functions specifically through changes in morbidity: it counteracts the negative average cost effect when infection rates decline but amplifies it when case fatalities decline. As in Barro-Becker, the model generates an inverted U-shaped population path, but only when the decline in disease burden reduces child mortality sufficiently.[3]

Our partial equilibrium model has obvious general equilibrium implications. If reducing childhood disease lowers fertility and raises human capital investment, targeting the disease burden can stimulate economic development. The disease burden may be lowered by reducing disease prevalence or severity. Health initiatives in developing countries may have unintended consequences for fertility depending on how diseases are combated. A declining disease burden

[2]Among such studies are Boldrin and Jones (2002), Galor and Weil (1999), Kalemli-Ozcan (2008), Soares (2005) and Tamura (2006).

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

will not necessarily reduce mortality and fertility unless it concurrently lowers morbidity.

We present the theoretical model in Section 2. Fertility and human capital decisions are analyzed in Section 3 and empirical evidence presented in 4. The model's implications for specific diseases are discussed in Section 5. Section 6 concludes with policy implications.

2 The Model

An individual potentially lives for three periods – childhood, adulthood and old age. Children are born with a health endowment h_0 and are exposed to infectious disease in early childhood from which they may or may not survive. Infection can occur from a wide range of diseases depending on the disease ecology. Let $i \in (0,1)$ denote the infection rate across all such diseases among children. Of the fraction i of children contracting them a fraction $d \in (0,1)$ do not survive. The parameter d is the case fatality rate from a disease while the product id is the child mortality rate.[4]

What distinguishes our model from existing work on the precautionary demand for children is the idea that exposure to infectious disease has a morbidity effect even when a child survives. To be specific, infections depreciate a child's health by the fraction $\delta \in [0,1]$. This morbidity mechanism is motivated by extensive bio-medical evidence showing infectious diseases to hamper a child's cognitive and physical development and weaken his intrinsic health.

Diarrheal infections, for example, disrupt nutritional absorption, deprive the body of nutrients necessary for optimal cellular growth (Martorell, 1980; Martorell and Habicht, 1986; Mata, 1978), affect cognitive abilities, and cause stunting in the first two years of life (Berkman *et al.*, 2002; Eppig *et al.*, 2010; Mendez and Adair, 1999). Guerrant *et al.* (2003) report a high association between diarrhea in the first two years of life and cognitive function $4-7$ years later among Brazilian children. Growth impairment was observed even when enteric infections did not manifest in overt diarrhea. Diarrheal diseases which accounted for 16% of child deaths in SSA and 14.3% of DALYs in 2004 (WHO) have high recurrence rates. Median episodes per child per year reaches a maximum of $4.1-4.3$ among $6-11$ month olds, tapering off to $0.4-1.7$ by four years of age (Jamison *et al.*, 2006).

Another disease, malaria, accounted for 18% of SSA's child mortality in 2005 (WHO). In endemic regions most children are chronically infected with the *P. falciparum* parasite starting soon after birth. Prevalence can be as high as 80%, as among Kenyan and Tanzanian children. Repeated new infections offer immunity from the fatal consequences of infection but anti-parasitic immunity does not develop until early adulthood (Snow *et al.*, 1999). A significant

[4] In general i and d would depend on h_0 and on pre- and post-natal health inputs provided by the parent. Here both are treated as parameters.

proportion of children suffer from the disease throughout their childhood and, consequently, from anemia. Anemia prevalence is estimated at 75% in areas where malaria prevalence is at least 25% and is directly linked to poor school performance and lower earnings among working adults (Snow *et al.*, 2003). Also relevant for understanding malaria's impact is Bleakley's work (2007) on progressive iron and protein-deficiency anemia from hookworm infections. Bleakley finds that anemia was a leading cause of weak school performance and that remarkable improvements in children's scholastic performance were observed in the American South after hookworm (anemia) was eradicated in the early twentieth century.

In the model, infectious disease in childhood alters potential labor income in adulthood.[5] Children are most susceptible to infectious diseases during the first few years of life and, during the demographic transition, the largest gains in longevity occur among infants and children. Accordingly we assume that uncertainty about child survival is resolved after infancy and that parents invest in the surviving children's human capital (education, health) afterwards. Childhood health determines the productivity of such investment as long as $\delta > 0$.

Surviving children are of two types. Of the N children born to a household, those who were never (significantly) exposed to infectious diseases remain healthy (numbering N_1), those who survived diseases remain unhealthy (numbering N_2). Parents can invest differently in each type, h_1 on each healthy child, h_2 on each unhealthy one. A child's human capital as an adult reflects these investments and his health capital. Denoting by H' this human capital, we have

$$H' = \begin{cases} h_0^{\alpha} h_1^{\theta}, & \text{if healthy as a child} \\ \{(1-\delta)h_0\}^{\alpha} h_2^{\theta}, & \text{otherwise} \end{cases} \tag{1}$$

where child health investment and endowment are assumed to be complements and $\alpha, \theta \in (0,1)$. A child's consumption is not modeled explicitly and is instead subsumed in his parent's.

Adults work in youth and retire in old age. Young adults earn a wage w per effective unit of labor which, given their human capital H from past disease experience and human capital investment, yields income wH. Children are investment goods, valued because they financially support their elderly parents, an assumption relevant for developing societies with weak social safety nets. The social norm dictates that each adult contributes a $\tau \in (0,1)$ fraction of his labor earnings to his elderly parent and receives the same fraction of each child's earnings in old age.[6] Adults choose their consumption in youth c, the number of children they have n, and human capital investment in children (h_1, h_2), that supports old-age consumption needs. There are no alternative assets to save for old age and no incentive to leave bequests.

[5]While we do not have precise estimates of the overall effect on labor productivity from all types of infectious diseases, we will entertain different values of δ to get an idea how childhood morbidity affects fertility outcomes.

[6]Boldrin and Jones (2002) analyze endogenous donations from children that support parents in old age but do not include parental investment in children.

Assuming preferences over consumption are logarithmic and ignoring the additive term $\alpha(1-\beta)\ln h_0$, a young adult maximizes his expected lifetime utility

$$E\left[\beta \ln(c) + (1-\beta)\ln(\{N_1 h_1^{\theta} + (1-\delta)^{\alpha} N_2 h_2^{\theta}\}\tau w')\right] \tag{2}$$

subject to

$$\begin{aligned} c + N_1 h_1 + N_2 h_2 &\leq (1-\gamma n)(1-\tau) wH & (3)\\ N_1 + N_2 + N_3 &= n & (4) \end{aligned}$$

where the second term in (2) corresponds to utility from old-age consumption, $\beta \in (0,1)$ is the weight attached to consumption in youth versus old age, w' is the efficiency wage rate faced by children in their adulthood and $\gamma \in (0,1)$ is the fixed time cost of having a child. To conserve notation we define $z \equiv (1-\tau)wH$ and $x \equiv \tau w'$. As in much of this literature we ignore integer constraints and the restrictions $n \geq 1$ and $N_1 + N_2 \geq 1$ that ensure at least one surviving offspring. We do take into account that fertility is bounded above at $1/\gamma$.

Adults explicitly recognize the random nature of their children's survival from infections. Since human capital investment (h_1, h_2) occurs after this uncertainty is resolved, we will solve for optimal decisions using backward induction. That is, we identify optimal consumption and investment decisions conditional on n, N_1, and N_2, then use the Delta Method to solve for the fertility decision à la Kalemli-Ozcan (2008). The inclusion of two sources of uncertainty, in disease exposure and in survival from that exposure, amplifies the precautionary motive relative to Kalemli-Ozcan's work.

The number and type of survivors are random draws from the discrete multinomial distribution:

$$p(N_1 = n_1, N_2 = n_2, N_3 = n_3) = \frac{n!}{n_1! n_2! n_3!} p_1 p_2 p_3 \tag{5}$$

where $\sum_{j=1}^{n} n_j = n$ and $p_j \in [0,1]$ denotes the probability of outcome n_j out of n births. On average $\bar{N}_1 = n(1-i)$ children avoid disease and remain healthy, $\bar{N}_2 = ni(1-d)$ children survive disease but remain unhealthy, and $\bar{N}_3 = n - \bar{N}_1 - \bar{N}_2 = nid$ children succumb to disease. We refer to n as the total fertility rate (TFR) and $(1-id)n$ as the net fertility rate (NFR).

2.1 Decision under Certainty

It is instructive to first study the certainty version where the number of survivors of each type is simply taken to be its expected value. In this case the parent chooses h_1, h_2 and n to maximize

$$\beta \ln\left[(1-\gamma n)z - n\{(1-i)h_1 + i(1-d)h_2\}\right] + (1-\beta)\ln\left[nx\left\{(1-i)h_1^{\theta} + i(1-d)(1-\delta)^{\alpha} h_2^{\theta}\right\}\right].$$

The first-order conditions in an interior optimum yield

$$\begin{aligned} n &= \frac{(1-\theta)(1-\beta)}{\gamma} \\ c &= \beta z \\ h_1 &= \frac{\theta\gamma}{(1-\theta)\left[1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}}\right]} z \\ h_2 &= (1-\delta)^{\frac{\alpha}{1-\theta}} h_1. \end{aligned}$$

These results contradict historical demographic transitions where falling child mortality was associated with falling fertility and rising human capital investment.

While parental income has a positive effect on child investments, fertility does not depend on it. Parents have fewer children if they value their future consumption less (higher β) and if the return to investment in child quality is higher (higher θ). But fertility is unaffected by the infection and case fatality rates, while human capital investment is *increasing* in them.

That fertility is independent of child mortality is similar to Kalemli-Ozcan (2008): under logarithmic preference, parents essentially care about the number of childbirths not surviving children, and neither does child mortality affect the household's budget constraint.[7] Child investment, in contrast, is inversely related to the number of survivors which increases when either i or d decreases. Since the total fertility rate, n, does not adjust, an increase in either i or d decreases the number of surviving children so that investment per child rises.

Furthermore, total investment in surviving children is unaffected by i or d. The increase in survivors exactly counteracts the decline in human capital investment per child: the net effect on total investment, $N_1 h_1 + N_2 h_2$, is zero. When i decreases, fewer children die and more survivors are healthy. When d decreases, in contrast, fewer children die but more children are unhealthy for a given infection rate. Net fertility, $n(1-id)$, falls with i and d. But the effect of i and d on the number of survivors of each type differs: $\partial N_1/\partial i < 0$ while $\partial N_2/\partial i > 0$, and $\partial N_1/\partial d = 0$ while $\partial N_2/\partial d < 0$.

2.2 Decision under Uncertainty

When uncertainty about child survival outcomes is incorporated into the optimization problem, n depends on the disease experience. Parental decisions are solved sequentially. First, given the fertility decision (n) and the number and type (N_1 or N_2) of survivors, parents choose h_1,

[7] Under more general CRRA preferences, the TFR actually increases in response to lower mortality. In Boucekkine *et al.* (2009), the inclusion of a labor-leisure tradeoff ensures that fertility declines with mortality despite CRRA and certainty about child survival.

h_2, and c to maximize utility

$$\beta \ln\left[(1-\gamma n)z - N_1 h_1 - N_2 h_2\right] + (1-\beta)\ln\left[\left\{N_1 h_1^{\theta} + (1-\delta)^{\alpha} N_2 h_2^{\theta}\right\} x\right]$$

The first-order conditions yield

$$h_1(n) = \frac{\theta(1-\beta)(1-\gamma n)}{\{\beta + \theta(1-\beta)\}(N_1 + (1-\delta)^{\frac{\alpha}{1-\theta}} N_2)} z \tag{6}$$

$$h_2(n) = (1-\delta)^{\frac{\alpha}{1-\theta}} h_1(n) \tag{7}$$

and

$$c(n) = \frac{\beta(1-\gamma n)}{\beta + \theta(1-\beta)} z. \tag{8}$$

Quality investment in children now depends negatively on the number of survivors and on total fertility. This investment depends on child mortality even though investment decisions are made after child survival uncertainty is resolved. Moreover, even though parental investment can compensate for depreciated health capital among unhealthy children, it does so only partially ($h_1 > h_2$) unless $\delta = 0$.

Using these conditional choices $h_1(n)$, $h_2(n)$, and $c(n)$, rewrite the utility function as

$$\beta \ln\left[\frac{\beta(1-\gamma n)}{\beta + \theta(1-\beta)} z\right] + (1-\beta)\ln\left[(N_1 + (1-\delta)^{\frac{\alpha}{1-\theta}} N_2)^{1-\theta} \left\{\frac{\theta(1-\beta)(1-\gamma n)}{\beta + \theta(1-\beta)} z\right\}^{\theta} x\right] \tag{9}$$

which the parent then maximizes with respect to n, taking into account uncertainty regarding (N_1, N_2) as specified by (5) above. An online Appendix A (available as Electronic Supplementary Material on the journal's website) details how this optimization leads to the quadratic first-order condition

$$n - \frac{\gamma\{\beta + \theta(1-\beta)\} n^2}{(1-\beta)(1-\theta)(1-\gamma n)} = \frac{-i[1 - i + (1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)[1 - i(1-d)]]}{2[1 - i + i(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)]^2}. \tag{10}$$

which we analyze in depth in the following section.

3 Fertility, Quality Investment and the Disease Burden

A change in the childhood disease burden – a decline in disease prevalence and case fatality and the ensuing changes in morbidity – affects fertility in various ways. In order to understand these, consider the effect of (i, d, δ) on the TFR, the NFR, and quality investment.

3.1 The Total Fertility Rate

The first-order condition (10) is quadratic in n. Only one of its roots is positive and is the optimal fertility choice. For the special case $i = 0$ this optimal choice becomes $n = (1-\beta)(1-\theta)/\gamma$, the same as under certainty except here all children survive. More generally fertility is a highly non-linear function of the parameters, though, as expected, it is unambiguously higher than under certainty (online Appendix B).

Three forces determine the response of n to a decline in the child mortality rate id. First is the precautionary motive that leads to a positive relationship between fertility and child mortality. Faced with survival uncertainty, parents have more children than they ultimately need. A decline in child mortality will reduce total fertility since more children survive.

Secondly, similar to Barro and Becker (1988), when child mortality declines, survivors become cheaper to produce and fertility goes up. The total cost of child-rearing consists of the fixed birth cost (γn) and the variable cost of quality investment on surviving children ($N_1 h_1 + N_2 h_2$). Investments h_1 and h_2 decrease with the number of survivors for a given level of n from (6) and (7). Hence the average investment cost per surviving child,

$$\frac{N_1 h_1 + N_2 h_2}{N_1 + N_2} = \left[\frac{N_1 + (1-\delta)^{\alpha/(1-\theta)} N_2}{N_1 + N_2}\right] h_1, \tag{11}$$

decreases when $N_1 + N_2$ rises because quality investment in unhealthy children is lower and both types of quality investment fall. Hence, for a given n, the average birth cost of producing a surviving child decreases as the proportion of surviving children $(N_1 + N_2)/n$ rises from a decline in child mortality.

The third channel, a key contribution of this paper, operates through the morbidity effect and depends on whether child mortality falls from lower prevalence or from lower case fatality. Parents substitute between the quantity and quality of children depending on the disease burden. When child mortality falls due to lower disease prevalence, more of the survivors are healthy children. That is, $N_1/(N_1+N_2)$ increases. Since $h_1 > h_2$, this makes surviving children more expensive on average and pushes parents towards fewer but better quality children. The reverse is true when child mortality falls due to lower case fatality and parents have more children of worse quality.

Hence when child mortality id falls from lower i, the first and third effects work in the same direction to lower fertility while the second effect works in the opposite direction. The larger is δ, the stronger is substitution towards quality, amplifying the third effect. Moreover, the morbidity effect is stronger when most of the surviving children have experienced infectious disease, that is when i is high and d low.

When id falls instead due to lower d, only the first effect works towards lowering fertility. The second and third effects work towards higher fertility since more of the surviving children are of worse health. Hence, for a given reduction in child mortality, a reduction in i has a stronger effect on fertility behavior than a comparable reduction in d.

Some numerical results will better identify these tensions. For simplicity we set the time cost of child rearing to $\gamma = 1$ which restricts $n \in (0, 1)$. We set $\alpha = 0.3$, $\theta = 0.7$ and $\beta = 0.5$. Values for α and θ are restricted to satisfy CRS in health production. The value for θ falls between the values of 1 in Kalemli-Ozcan (2008) and 0.5 in Doepke (2005). In choosing these values we are being conservative about the magnitude of the quality gap which depends on $\alpha/(1-\theta)$. The literature does not offer clear guidance on the morbidity cost δ since it can differ across disease ecologies and, in the model, represents overall morbidity from all types of infection. We first establish results for extreme values of $\delta \in \{0, 1\}$. Then we consider whether or not our conclusions are robust to $\delta \in \{0.1, 0.3\}$. Values of δ closer to zero are more reasonable since they imply an empirically plausible quality gap between healthy and unhealthy children. For example, for $\delta = 0.1$, unhealthy children receive $(1-\delta)^{\alpha/(1-\theta)} = 90\%$ of the investment on healthy children. In any case, alternative parameter values do not change results qualitatively. Comparative statics results are available in the online Appendix.

Disease Prevalence and the TFR

Figures $3-6$ illustrate the household's fertility choice as a function of (i, d) for various values of δ.

Insert Figure 3 here

In the upper panel of Figure 3 the morbidity effect is exceptionally high at $\delta = 1$. The child mortality rate is effectively i. Since unhealthy children are completely unproductive as adults and unable to support their elderly parents, it is irrelevant for human capital investment whether or not an infected child survives. From the parent's perspective to get a disease is effectively to die and the model is isomorphic to Kalemli-Ozcan's (2008).[8]

Suppose the prevalence rate drops, say, from a new vaccine that lowers infection rates and overall prevalence among children. The fertility response from this lower child mortality rate is calculated for different values of d in Figure 3: the case fatality rate has no effect since the $n(i)$ functions for different values of d coincide when $\delta = 1$. Fertility is monotonically

[8]This isomorphism hinges on how preferences are modeled. If parents were altruistic in the Barro-Becker sense or derived warm glow from investment in each type of child, they would care about unhealthy children even if $\delta = 1$. Both of these complicate the household's decision problem without adding insight to the morbidity effect.

increasing in the disease prevalence rate, also the effective child mortality rate. A decline in i, in other words, unambiguously lowers the TFR similar to Kalemli-Ozcan (2008).

The lower panel of Figure 3 assumes that $\delta = 0$ which means all surviving children are equally healthy. But newborns are exposed to two sources of uncertainty, first whether or not they contract infectious disease, and secondly, whether or not they survive from it. The model does not simplify to the standard precautionary demand case because of the unique role d plays.[9]

The response of fertility to the prevalence rate can be non-monotonic for $\delta = 0$ depending on case fatalities (Figure 3, lower panel). The response is strongly positive when disease prevalence is relatively low. For relatively higher prevalence rates, the case fatality rate becomes important. At fairly high case fatalities ($d = 0.7,\ 0.9,\ 1$), the $n(i)$ relation is monotonically increasing as it was for $\delta = 1$. At low-to-medium case fatalities ($d = 0,\ 0.3,\ 0.5$) and relatively high infection rates, on the other hand, disease prevalence lowers fertility.

The sharpest contrast between the cases $\delta = 1$ and $\delta = 0$ in Figure 3 occurs for high i and low d. This is because the quantity-quality tradeoff due to the morbidity effect is absent for $\delta = 0$ and only the first two channels are at work. At high i and low d values, since most children get infected and few of them die, the precautionary motive is weak. Hence the average cost effect drives the negative relationship between fertility and disease prevalence in the lower panel of Figure 3 at high values of i and low values of d.

The two robustness checks for $\delta \in \{0.1, 0.3\}$ presented in Figure 4 are qualitatively similar to the $\delta = 0$ case in Figure 3. As δ keeps increasing towards 1 (not shown), the quality-quantity substitution effect gets stronger until the Barro-Becker average cost effect is eventually dominated.

Insert Figure 4 here

Case Fatality and the TFR

Now consider the relationship $n(d)$. Suppose that d declines because of the availability of an antibiotic. That could also reduce infection rates by reducing the duration when infected individuals are contagious but, in practice, would be a relatively unimportant channel. The response of the TFR to d is shown in Figures 5 and 6 for different values of the prevalence rate.

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,

[9] The standard precautionary demand model of Sah (1991) and Kalemli-Ozcan (2008) implicitly assumes that all children contract infectious diseases and uncertainty is with respect to survival alone.

they have more children in order to compensate for their lower quality. In contrast to the vaccine scenario above, the negative average cost effect is amplified by a decline in d when δ is high. A decline in d improves survival, thereby lowering the average fixed birth cost of surviving children, and with more unhealthy children surviving, the average surviving child is also cheaper in terms of human capital investment.

Insert Figure 5 here

An antibiotic lowers mortality more for widely prevalent and fatal diseases, and increases morbidity more for high δ diseases. The upper panel of Figure 5 echoes the $\delta = 1$ case in Figure 3 where case fatality had no effect on fertility. In the lower panel of Figure 5, $\delta = 0$ and lower case fatality rates elicit a pronounced positive fertility response when child mortality is high (i and d are both high). In contrast, when both i and d are low, fertility is already close to its lower bound: a decrease in i or d does not lower n by much. Hence, as $i \to 1$, we get a monotonic positive relation between n and d. Here the effective child mortality rate tends to d and the relation between n and d is again similar to Kalemli-Ozcan's model. Robustness checks for $\delta \in \{0.1, 0.3\}$ in Figure 6 show results to be qualitatively similar to the $\delta = 0$ scenario.

Insert Figure 6 here

We conclude that when δ 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: $\partial n/\partial i > 0$ occurs when d is high and $\partial n/\partial d > 0$ when i is high. These results are summarized in the following proposition with details provided in the online Appendix B.

Proposition 1 *The child mortality in this economy depends on the prevalence (i) and case fatality (d) rates. When the prevalence rate falls, the corresponding fall in child mortality lowers the fertility rate if the prevalence rate is relatively low, or at a given prevalence rate, when the disease is fatal (relatively high d) or causes serious long-term morbidity (high δ).*

When child mortality falls from lower case fatality, on the other hand, the fertility rate falls if the prevalence rate is relatively high, or at a given prevalence rate, when the disease is not too fatal (relatively low d) or causes serious long-term morbidity (relatively high δ). For exceptionally high morbidity ($\delta = 1$), the fertility rate is unaffected.

A simple example illustrates the differential effects of mortality and morbidity on the TFR. Suppose $i = 0.1$ and $d = 0.5$ which imply a child mortality rate of 0.05 or 5%. Suppose also that $\delta = 0.5$. A 20% reduction in either prevalence or case fatality rates will reduce the mortality rate from 5% to 4% but the effect on average child quality will not be identical.

Average child quality is $(1-i)h_1 + i(1-d)(1-\delta)h_2 = [1 - i + i(1-d)(1-\delta)^{\frac{1+\alpha-\theta}{1-\theta}}]h_1$. Holding constant h_1, when child mortality declines, improvements in average child quality will be greater if the mortality decline occurs via i. Specifically, the differential effect on average child quality when i declines versus when d declines is

$$\begin{aligned}&\left[1-(1-d)(1-\delta)^{\frac{1+\alpha-\theta}{1-\theta}}\right]h_1\Delta i - i(1-\delta)^{\frac{1+\alpha-\theta}{1-\theta}}h_1\Delta d \\ &\quad = 0.02\left[1-0.5^{\frac{1+\alpha-\theta}{1-\theta}}\right]h_1\end{aligned}$$

which is positive for $\alpha, \theta \in (0,1)$ and equal to $0.015h_1$ for the parameter values used above.

Finally, it is worth emphasizing what the morbidity channel adds to our understanding of the mortality-fertility relationship through precautionary incentives. Doepke (2005) shows that the precautionary motive, on its own, cannot generate a strong mortality-to-fertility response since much of the survival uncertainty is resolved by age five. When parents make sequential fertility choices, they can replace non-surviving children at relatively low cost and meet their target number of survivors with fewer births. Doepke's quantitative analysis establishes that the precautionary motive is far too weak to sustain a high TFR in this case.

The inclusion of two sources of uncertainty could amplify the precautionary motive. It remains to be seen if this bears more significantly upon fertility behavior than Doepke finds. Secondly, by not exclusively relying on survival uncertainty, our model extends the effect of the precautionary motive beyond the first five years of birth. Early and repeated exposure to infectious diseases have quality consequences for a child well into early adulthood. Hence there is an inherent link between child survival and child quality. Moreover, if children are exposed to a range of infectious disease well into early adulthood, an additional source of uncertainty not considered here is the productivity of ongoing quality investment.

3.2 The Net Fertility Rate

Galor and Weil (1999) stress a historically observed non-monotonic pattern for the NFR. As child mortality declines, initially total fertility does not decline sufficiently to reduce net fertility. As total fertility continues to decline, eventually net fertility falls. A similar effect is at work in this model.

On average $n(1-id)$ children survive, and

$$\frac{\partial\{n(1-id)\}}{\partial i} = (1-id)\frac{\partial n}{\partial i} - nd \tag{12}$$

and

$$\frac{\partial\{n(1-id)\}}{\partial d} = (1-id)\frac{\partial n}{\partial d} - ni. \tag{13}$$

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.

Investment in Human Capital

Finally we turn to the effect of childhood disease burden on human capital investment (see online Appendix C for details):

Proposition 2 *Under uncertainty, human capital investment in children rises when child mortality falls if and only if the total fertility response to decreasing prevalence and case fatality rates is positive and sufficiently large.*

If the TFR rises when child mortality falls, then fewer household resources are available for human capital investment: h_1 and h_2 decline. If the TFR declines but not enough to counter the effect of a larger number of survivors, then h_1 and h_2 will again fall. This suggests that where the fertility response is weak or even negative, health initiatives should be coupled with subsidies to human capital, in the form of education subsidies, for example. Else, combating the disease burden may raise net and even total fertility, thereby lowering overall human capital investment.

4 Some Empirical Evidence

This section empirically tests a few of the model's predictions about childhood disease burden and fertility.

We first present evidence from SSA on the precautionary motive: reductions in child mortality lower total fertility rates (TFR) by less at high rates of child mortality. We then show that lower infectious disease incidence has a stronger positive effect on the TFR while lower case fatality rate has a smaller, even negative, effect. The role of disease morbidity (δ) is also consistent with the theoretical predictions: higher δ strengthens the positive relationship between incidence and TFR and weakens that between case fatality and TFR.

4.1 Precautionary Motive

Central to our theory of Africa's slow fertility transition is the precautionary motive for fertility when child survival is uncertain. A testable implication of Sah (1991) and Kalemli-Ozcan (2008) is that the fertility response to child mortality is uniformly positive but weaker at higher levels of mortality. Abstracting from the morbidity channel, we begin by testing for this since, to the best of our knowledge, the literature has not offered direct empirical evidence on it. Specifically, instead of assuming a linear effect of the child mortality rate (CMR) on the TFR as is common in the literature, we incorporate a quadratic CMR term whose coefficient is expected to be negative to counteract the positive linear effect.

The dependent variable we use is the 2008 TFR from 45 countries in SSA. The independent variables are taken from 2004 to alleviate potential endogeneity problems. Similar results are obtained for the 2004 TFR and are available upon request.

Although the dependent variable is a country-wide average of TFR rather than a discrete count, a Poisson regression model is used for estimation because the data maintains characteristics appropriate for Poisson modeling. Specifically, the mean TFR is low (4.8 for 2008, 5.1 for 2004), all values are positive, and the distribution of the data is skewed rather than normal. Standard errors are robustly estimated. Results do not differ importantly when regressing ln(TFR) or TFR using OLS and are also available upon request.

Country effects are not included in the regression due to sample size limitations. We control for the following country characteristics that are thought to affect the TFR: GDP per capita, adult mortality, expected years of schooling, and access and quality of health care, as measured by the Health Worker's Reach Index. Data for the first three variables are taken from the World Bank, for expected years of schooling from the United Nations and for health care from the Save the Children project.[10] Descriptive statistics are presented at the top of Table 1.

Insert Tables 1 & 2 here

Regression results are reported in Table 2. Comparing Columns 1 and 2 we see that the statistically significant positive coefficient on CMR is larger when we allow for the nonlinear effect. The direct effect of CMR is positive, as expected, while the nonlinear (quadratic) effect is negative. The marginal, overall positive, effect of the CMR on the TFR is lower for higher values of the CMR, a result consistent with the presence of a precautionary motive. Moreover, the coefficients on the control variables have their expected signs although several are not statistically significant, likely due to collinearity.

[10] Available at
http://www.savethechildren.org.uk/resources/online-library/health-workers-reach-index

A caveat is in order. While this is a parsimonious way to test for the precautionary motive, Column 2 results are also consistent with alternative explanations. For example, since a decline in d dilutes average child quality, it would have a weaker effect on the TFR even without a precautionary motive. In that respect, a concave response of the TFR to CMR may be simply picking up the morbidity channel. While an important concern in general, it is less relevant for SSA where d is quite low (Table 1). SSA's high CMR is mainly due to its high i, and hence, the strong concave response of the TFR to CMR in Column 2 is unlikely to be driven by the morbidity channel alone.

4.2 Disease Morbidity

We next turn to the morbidity channel and its effect on fertility choice under survival uncertainty. To do so requires us to incorporate the severity of infectious disease morbidity. Since Africa suffers from a multitude of infectious diseases, instead of focusing on one particular disease we consider the top four infectious causes of death among children. These are lower respiratory infections (LRI) especially pneumonia, diarrheal infections, malaria, and HIV/AIDS.

We separate incidence and case fatality rates of these four diseases from all other causes of child mortality as described below. This data is available for 37 countries in SSA for the year 2004.[11] The dependent variable is again TFR in 2008 and disease data pertains to children under the age of five.

For $i \in \{\text{Malaria, HIV, Diarrheal infections, LRI}\}$, we construct for each country

$$\text{Incidence rate} = \frac{\sum_i \text{Incidents}_i}{\text{Child Population}}$$

$$\text{Weighted incidence rate} = \frac{\sum_i \delta_i \text{Incidents}_i}{\text{Child Population}}$$

$$\text{Case fatality rate} = \frac{\sum_i \text{Deaths}_i}{\sum_i \text{Incidents}_i}$$

$$\text{CMR other} = \text{CMR}_{\text{all causes}} - \sum_i \text{CMR}_i$$

where $\text{CMR}_i = \text{Incidence rate}_i \times \text{Case fatality rate}_i$.[12] For the morbidity effect of each disease we rely on disability weights from the Global Burden of Disease (GBD) project. This weight

[11] These 37 countries are: Angola, Benin, Botswana, Burkina Faso, Burundi, Cameroon, Central African Republic, Chad, Congo, Democratic Republic of Congo, Cote d'Ivoire, Equatorial Guinea, Eritrea, Ethiopia, The Gambia, Ghana, Kenya, Liberia, Malawi, Mali, Mauritania, Mauritius, Mozambique, Namibia, Niger, Nigeria, Senegal, Sierra Leone, Somalia, South Africa, Sudan, Swaziland, Tanzania, Togo, Uganda, Zambia, Zimbabwe. They accounted for 94% of SSA's 2008 population (World Bank).

[12] An alternative approach would be to consider the incidence and case fatality rates for each disease separately.

takes the values zero for ideal health and one for a state of health comparable to death. For Africa's four major infectious diseases, GBD assigns the values

$$\delta_{\text{Malaria}} = 0.2,\ \delta_{\text{AIDS}} = 0.5,\ \delta_{\text{Diarrheal infections}} = 0.1,\ \delta_{\text{LRI}} = 0.3.$$

As noted above, the overall weighted incidence rate is calculated as the weighted average of disease-specific incidence rates, with weights given by the corresponding δ. The differential effect of the two incidence measures will thus pick up the overall morbidity effect from the four infectious diseases. We use the disability weight for AIDS without anti-retroviral treatment (ART) (instead of that for HIV which is 0.1 or for AIDS with ART which is 0.2) because most cases of HIV in SSA progress to AIDS and treatment with ART is rare, in the single digits for many countries. The case fatality rates are not weighted by δ to construct the overall rate since death is an absolute outcome: whether a child dies from a high or a low δ disease is irrelevant.

The bottom half of Table 1 presents descriptive statistics for these data. For most infectious diseases afflicting African children, case fatality rates are quite low while incidence rates often exceed 100% of the child population since a disease can be contracted multiple times. HIV/AIDS is an exception in that incidence is relatively low and case fatality rates very high. In Table 1 disease incidence rates exceed one for some countries and the incidence rate of diarrheal diseases is five for all countries. Hence the aggregate disease incidence rate exceeds one.

The incidence and mortality data we use comes from the World Health Organization's National Burden of Disease (NBD) Toolkit. All available information on each country's relevant epidemiology was used by the GBD to estimate disease- and age-specific incidence rates. When the number of disease incidents was available directly from disease registers or epidemiological studies they were used, but often only prevalence data was available. In that case a software program called DISMOD was used to mathematically model incidence from estimates of prevalence, remission, and disease-specific mortality risk derived from population data or epidemiological studies. DISMOD was also used to ensure internal consistency of all estimates, for example, that mortality did not exceed incidence for any disease and to account for issues of co-morbidity. This was done by exploiting the fact that the various epidemiological variables are causally linked by a disease process (for details see Mathers *et al.*, 2003, 2006).

Given the paucity of data, instead of estimating a structural model, we use interaction and quadratic terms for a reduced form representation of the theoretical model. In Column

Sample size restrictions make it harder to implement this. Additional data is necessary to estimate and compare the fertility and morbidity effects from various diseases.

3 of Table 2 we regress TFR on the child disease incidence (i) and case fatality (d) rates aggregated across the four infectious diseases as discussed above, along with their interaction (id) which represents child mortality from these diseases. We control for child mortality from other causes and, as before, GDP per capita, adult mortality, expected years of schooling, and health care quality and access. In Column 5 we allow for a nonlinear effect of CMR from other causes, and the coefficient on CMR due to other causes becomes significant, supporting the result in Column 2. The coefficients on i and d are positive and that on their interaction, id, is negative. In other words, declines in child mortality reduce TFR less where mortality is high.

When i is weighted by disease severity, δ, the coefficients on i and id increase in magnitude while that on d falls. These changes are consistent with our theoretical predictions.[13] The disability weight for each disease except HIV/AIDS is relatively low but becomes sizable when aggregated (several diseases afflicting a child several times). Table 3 describes the net effect of a one percentage point reduction of i and d on the TFR with and without the disability weights. Accounting for the high δ nature of the disease burden in Africa, we see a stronger positive net effect of i on the TFR consistent with Figure 3 and a weaker net effect of d on the TFR consistent with Figure 5. That the mean net effect of d on the TFR is negative may indicate that SSA's prevalence rates, while high, are not as high as Proposition 1 requires. Note that while the coefficients are much larger in magnitude for d than for i, d is very low in all countries in the sample while i is high (Table 1).

Insert Table 3 here

To conclude, we evaluated three testable implications of the theoretical model. We showed that there is a nonlinear effect of child mortality on TFR in line with the precautionary motive for fertility. In particular, declines in child mortality reduce TFR less for higher levels of child mortality. We then disaggregated child mortality into incidence and case fatality rates to confirm this nonlinearity. Finally we showed that when the high cumulative morbidity burden of Africa's four major infectious diseases is taken into account, the positive effect of disease incidence on the TFR is strengthened while that of case fatality weakened. This role of morbidity is consistent with the theoretical results in Figures 3 and 5 when δ is high.

It is instructive to see how far these estimated effects can account for SSA's slow transition. Consider first a 68% drop in child mortality, similar to Gambia's experience during

[13] Since all the δs are less than one, the weighted i for each country would be a fraction of the unweighted i. This alone would inflate the coefficient estimates on the weighted-i regressors in Column 6 which differ from those in Column 5 by a factor of 4.6. But this is, at best, a partial explanation since the coefficient estimate on d changes too as do the net effects reported in Table 3. The ratio of unweighted to weighted i in the dataset is 8 on average and ranges from 6.6 to 9.9.

1960-2005 when the child mortality rate fell from 360 to 214 per 1,000 live births and the TFR from 5.57 to 5.28. Currently Gambia's $d = 0.002337$ and weighted $i = 0.7494$. Suppose the entire 68% decline in child mortality was due to lower case fatality which requires the 1960 fatality rate to have been 0.0073, so that $\Delta d = -0.00497$. Based on our regression results (Table 2, column 6), this would have changed ln(TFR) by

$$\Delta d \times (186.104 - 237.350 \times 0.7494) = -0.041,$$

that is, lowered the TFR by 4.02% (from 5.57 to 5.35). Suppose, instead, the entire decline in child mortality was due to lower disease prevalence spread uniformly across the four diseases. That imputes a value of 2.342 to Gambia's 1960 prevalence rate, a change in weighted i of -1.5925. This would have affected ln(TFR) by

$$\Delta i \times (1.768 - 237.350 \times .002337) = -1.932.$$

that is, reduced the TFR by 85.51% (from 5.57 to less than 1). A similar quantitative assessment can be done for Uganda where TFR fell by 6% and the CMR by 42% during 1960-2009. A purely case fatality driven CMR decline would have lowered its TFR by 1.07% compared to 45.4% from lower prevalence rates. For the "average" SSA country with mean d of 0.003 and weighted i of 0.789 (Table 1), a 20% decline in child mortality through lower d alone is predicted to have no impact on the TFR. A 20% decline in child mortality through lower i, in contrast, is predicted to lower the TFR by 15%.

One has to, of course, cautiously interpret these magnitudes since they are based on a parsimonious model without detailed attention to country-specific disease factors. But the message is consistent: sub-Saharan Africa would have made further progress towards lowering its TFR over the last half-century had it been more successful in eradicating the major infectious diseases afflicting its children. Drawing on historical data and epidemiology studies, we turn to this theme in the next section.

5 Discussion

Infection and case fatalities were historically high in many Western countries and it was primarily new knowledge about germs that triggered the fall of infectious disease mortality in the late nineteenth century. The new knowledge led to public sanitation reform and improvements in personal hygiene, and both mortality and morbidity fell during the epidemiological transition (McNeill, 1976). As infection rates and child mortality fell, fertility rates generally followed (Arora, 2005).

In contrast, despite a child mortality transition, sub-Saharan Africa's demographic transition remains stalled. This is not a recent phenomenon brought on by the AIDS epidemic or Africa's "lost decade" of economic decline. For example, child mortality steadily declined in Africa between $1960 - 1980$ while the TFR followed much slower than in other developing countries (World Development Indicators Database).

Our model suggests that Western Europe's mortality transition brought on by lower prevalence rates meant that fewer children got sick and survivors became healthier. Consequently fertility fell and human capital investment in children rose. The model generates such a quantity-quality tradeoff when morbidity falls too. Historical data on stature show that cohorts that experienced declining child mortality became taller (hence healthier) relative to their predecessors (Arora, 2005). The evidence thus suggests Western Europe's morbidity declined in tandem with mortality because improvements in public health and personal hygiene successfully controlled the spread of disease.

Worldwide reductions in child mortality over the past half century have occurred through both lower disease prevalence (improved sanitation, vaccination) and lower case fatalities (antibiotics, ORT). But there is reason to believe the latter has been more instrumental in Africa's mortality transition.

Despite the success achieved in reducing child mortality from acute diarrheal disease, for instance, Guerrant *et al.* (2003) lament its continuing high morbidity due to inadequate investment in sanitation and safe water. Macro-level evidence from SSA shows that the morbidity burden has remained high, perhaps even worsened (as it would in our model when a higher proportion of survivors are unhealthy). African children who have benefited from an ongoing mortality transition have not grown up to be much healthier as measured by adult height; in some cases height fell (Akachi and Canning, 2008). On top of inadequate public health infrastructure, this reflects the presence of diseases like malaria for which no effective vaccine exists and pneumonia for which the most effective vaccine is out of reach of the poor. Whatever the exact reason, our theory predicts persistently high fertility due to this continuing morbidity burden.

In the African continent where infectious diseases flourish and children are malnourished, repeated infections can significantly debilitate individuals and impair cognitive development. Some treatments, specifically for parasitic diseases, can prevent deaths but cannot reverse the damage. Effective treatments exist for diseases like leishmaniasis which damages the spleen and liver and can cause anaemia, and schistosomiasis (bilharzia), a chronic disease that damages internal organs and impairs growth and cognitive development in children. Prevention is preferred to treatment in such cases. Polio, which can render its survivors paralyzed (high δ) and for which there exists no cure, has declined in prevalence from 350,000

cases in 1988 to less than 2,000 in 2005 due to aggressive public health policy (Center for Disease Control).[14] The model predicts this global near-eradication would have had a significant effect on fertility rates, a hypothesis that deserves further attention.

To better understand the model's implications for fertility behavior and public policy, consider a few diseases that together account for much of Africa's health problems.

Malaria

Malaria tends to be more fatal in moderate prevalence areas (low i/high d) and relatively less so in high prevalence areas (high i/low d) (Marsh and Snow, 1999). 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.[15] Malaria's δ parameter tends to be higher in low i/high d areas and lower in high i/low d areas, and theoretically, the fertility response to malaria is stronger for the former and weaker for the latter. Conversely the fertility response to case fatality would be weaker in low i areas where the morbidity cost is higher.

In endemic areas malaria infections in children are more quickly recognized as such so that patients receive prompter treatment, avoiding fatalities. Delaying malaria treatment by 5-10 days raises case fatality by a factor of 5 and a delay of 10-20 days raises it by a factor of 20. Despite similar levels of nutrition and health care access, in Sri Lanka, case fatality is 0.01% in endemic areas versus 1% in non-endemic areas because of quicker diagnosis in endemic areas (Alles *et al.*, 1998). In endemic areas malaria fatalities are mostly restricted to children, while in areas that experience periodic outbreaks of malaria, malaria fatalities heavily affect all age groups.

Reducing malaria transmission will have a stronger effect on fertility in moderate transmission areas since that is where case fatality rates are higher. Conversely, the reduction in mortality per reduction in case fatality rate due to treatment is higher in high transmission areas and lower in moderate transmission areas. More anemic children survive, for example, but the average health quality of children declines less in high transmission areas where malarial infections are relatively milder (δ is lower); parents have less incentive to increase the quantity of children to replace quality loss in high transmission areas. From a population control standpoint, reducing transmission via dissemination of bed nets during the rainy season or insecticide to eradicate mosquito populations is preferred to treatment of existing

[14] Led by efforts of the WHO polio has been eradicated in all but three countries: Afghanistan, Nigeria and Pakistan.

[15] Lower transmission areas are those at higher altitudes where fewer mosquitoes survive and where sporogony is slower.

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.

HIV/AIDS

The HIV/AIDS epidemic ravaging Africa is an example of a high d disease, particularly among children, for whom the disease progresses rapidly relative to adults, and among African households, only a small portion of whom have access to antiretroviral drugs. The model predicts that the fertility response to the childhood HIV/AIDS burden is positive. At the same time, HIV/AIDS affects mainly adults by raising their mortality rate. In our model a rise in adult mortality can be interpreted as higher β which reduces fertility since children are investment goods and benefit parents only in old age.

Recent empirics on the demographic consequences of this epidemic offer mixed evidence. Kalemli-Ozcan (2012) finds a positive fertility response to the HIV/AIDS epidemic while Young (2005) finds a negative one.[16] Durevall and Lindskog (2011), in contrast, argue that the more meaningful effect of the epidemic is on the age-distribution of fertility rather than overall fertility. Our model provides two conflicting forces, a positive one functioning through child mortality and a negative one through adult mortality, a result that is consistent with the more general findings in Boucekkine *et al.* (2009).

Pneumonia and Diarrhea

Pneumonia accounted for 21% of deaths among African children in 2005, diarrhea for another 16% (WHO).

Research has linked severe pneumonia in early childhood to later chronic lung disease (Puchalski *et al.*, 2009). But since not a large proportion of children suffer from severe forms of the disease (except for HIV+ children), δ is relatively small for school-age children. Hence if pneumonia has an effect on fertility behavior it is more likely through survival uncertainty than through child quality.

The same cannot be said of diarrhea, the second most common cause of sub-Saharan African child mortality. Diarrheal morbidity has not abated unlike mortality (Jamison *et al.*, 2006). The disease is a high i, low d and, due to repeated episodes averaging 5 per year per child, high δ one in our model. Over this parameter space, the precautionary motive is heightened due to strong morbidity effects. Successful abatement of the disease would

[16] The wage effect that contributes to a negative response of fertility to adult mortality in Young (2005) is absent in our model.

substantially raise returns to quality investment and shift parental incentives away from high fertility while lowering case fatality, something that can be done relatively easily when there is knowledge of and access to ORT.

Hookworm

The interesting counterpoint to these diseases is hookworm which, on its own, is not a significant contributor to child mortality. But SSA has one of the world's highest prevalence rates, at 29%, and hookworm's morbidity effects persist due to recurrent infections within a few months after deparasitation. Of the 198 million cases of infections in the continent, about 30% are among children below 14 years (de Silva *et al.*, 2003) and the prevalence rate can reach as high as 80% among school-age children (Saathoff *et al.*, 2005).

Bleakley's work (2007) identifies hookworm to be a leading cause of weak school performance due to progressive iron and protein-deficiency anemia. Remarkable improvements in children's scholastic performance were observed in the American South after the disease was eradicated in the early twentieth century.[17]

Using randomized experiments, Miguel and Kremer (2004) study the educational effect of treatment for hookworm and other intestinal helminth infections on Kenyan primary school children. Reducing prevalence had large effects on school attendance (more effective learning) among the treated children, as well as among untreated children in treated schools (lower transmission due to externalities).

Besides this direct effect of hookworm on quality investment (moderate δ), one concern is the possibility of co-infection between malaria and hookworm due to geographical overlap in SSA. Since each disease has a distinct way of causing anemia, co-infection significantly raises the risk of chronic anemia in children $5-15$ years old. A quarter of African schoolchildren have been estimated to be at risk of such co-infection (Brooker *et al.*, 2006).

This is an instance of strong disease complementarities: while malaria and hookworm each moderately affect child quality, their cumulative effect is to substantially lower quality to a relatively high δ and heighten the quantity-quality tradeoff under survival uncertainty.

Returning to the theoretical predictions of section 3, the lower panels of Figures 3 and 5 (low δ case) show that declines in i reduce CMR and thus TFR more in a high d environment, while declines in d reduce CMR and thus TFR more in a high i environment. Based on the NBD data used in section 4, SSA is best described as a high i, low d environment. This would suggest health interventions should be aimed at reducing d. Yet, as the discussion above

[17] The Rockefeller Sanitary Commission began a hookworm eradication campaign in 1910 after discovering that 40% of school-aged children were infected with the parasite.

makes clear, SSA's cumulative disease burden, the average δ across childhood infections, is clearly substantial. Turning to the top panels of Figures 3 and 5 (high δ case), we see that while reducing d does indeed reduce CMR considerably given the high i in SSA, it also raises the prevalence of morbidity considerably (high i, high δ), and the two effects act to pull TFR in opposite directions. A decline in i, on the other hand, reduces CMR somewhat (low d) but especially reduces morbidity (high δ) so that both forces work together to reduce the TFR. Taking morbidity into account, efforts ought to be targeted towards reducing i, rather than d, in SSA. Besides reducing child mortality and morbidity, that would be more effective for lowering fertility and encouraging education.

6 Conclusion

By building on the mortality-fertility literature, we explored the consequences for fertility of reducing disease burden in developing countries. The analysis suggests that health initiatives can have different effects on fertility depending on the morbidity and mortality associated with the disease in question. The strongest positive response of fertility to disease prevalence occurs where both mortality and morbidity rates change in the same direction.

That reducing disease burden may raise fertility rates does not contradict the existing consensus that fertility follows child mortality. Rather it highlights the idea that health initiatives are most effective at reducing fertility when they also tackle morbidity. Nor do the results suggest that disease burdens should not be tackled if it risks raising fertility.

Health policies that increase fertility or cause a response so weak that population growth actually rises should be complemented with education subsidies, for instance, so that parents can afford to send their larger families to school. When facing limited resources, the model provides a method for prioritizing health interventions in order to stimulate a demographic transition and human capital accumulation. We highlighted this for the case of malaria.

In future work we expect to pursue the preliminary empirical evidence we have presented on morbidity and the demographic transition in sub-Saharan Africa. An analysis of health interventions in malarial regions or the eradication of polio, for example, can more concretely shed light on our theoretical predictions. Moreover, previous mortality-fertility models have had to assume a relatively large precautionary motive to obtain a positive relationship between child mortality and fertility. The morbidity channel present in our model may reduce the need for large precautionary incentives, another topic that deserves further exploration.

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7 Tables

Table 1: Descriptive Statistics

Variable	Observations	Mean	Std. Dev.	Min	Max
TFR 2004	45	5.10	1.16	1.87	7.31
TFR 2008	45	4.81	1.19	1.58	7.12
Log(GDP per capita)	45	7.03	0.96	5.10	9.14
Adult mortality rate	45	0.46	0.16	0.21	0.81
Expected years of schooling	45	8.28	2.54	1.80	13.40
Health worker's reach index	45	0.46	0.12	0.13	0.74
Child mortality rate	45	0.13	0.04	0.02	0.21
Disease-specific sample					
Malaria incidence rate	37	0.632	0.357	0	1.056
Malaria case fatality rate	36*	0.010	0.006	0.003	0.037
HIV incidence rate	37	0.004	0.004	0.0001	0.016
HIV case fatality rate	37	0.594	0.106	0.004	0.751
Diarrheal incidence rate	37	5	0	5	5
Diarrheal case fatality rate	37	0.001	0.001	0.00002	0.003
LRI incidence rate	37	0.535	0.360	0.014	1.481
LRI case fatality rate	37	0.014	0.001	0.012	0.015
Incidence rate	37	6.171	0.588	5.014	7.418
Weighted incidence rate	37	0.789	0.148	0.504	1.131
Case fatality rate	37	0.003	0.001	0.00005	0.006
TFR 2004	37	5.20	1.17	1.87	7.31
TFR 2008	37	4.91	1.18	1.58	7.12
Log(GDP per capita)	37	7.02	0.97	5.10	9.14
Adult mortality rate	37	0.48	0.15	0.21	0.82
Expected years of schooling	37	7.93	2.59	1.80	13.40
Health worker's reach index	37	0.45	0.12	0.13	0.74
Child mortality rate other	37	0.11	0.03	0.02	0.17

* Mauritius reported zero incidence of malaria, so a case fatality rate could not be calculated.

Table 2: Poisson regression of 2008 TFR on 2004 variables

	1	2	3	4	5	6
ln(GDP per capita)	-0.052**	-0.037*	-0.025	-0.022	-0.022	-0.018
	(0.040)	(0.061)	(0.238)	(0.315)	(0.307)	(0.399)
AMR	-0.140	-0.332**	-0.03	-0.021	-0.070	-0.060
	(0.438)	(0.048)	(0.849)	(0.9)	(0.650)	(0.706)
Expected years of	-0.012	-0.011	-0.022**	-0.021**	-0.019*	-0.019*
schooling	(0.279)	(0.280)	(0.033)	(0.035)	(0.055)	(0.064)
Health worker's	-0.549**	-0.372**	-0.314*	-0.275	-0.308*	-0.273
reach index	(0.013)	(0.028)	(0.073)	(0.125)	(0.076)	(0.129)
CMR	2.882***	13.163***				
	(0.000)	(0.000)				
CMR-squared		-37.739***				
		(0.000)				
CMR other			-0.073	-0.046	6.954*	7.268*
			(0.939)	(0.961)	(0.071)	(0.058)
CMR other-squared					-27.515*	-28.651*
					(0.087)	(0.070)
i			0.479***		0.371***	
			(0.000)		(0.003)	
Weighted i				2.216***		1.768***
				(0.000)		(0.002)
d			513.671***	286.833***	334.957*	186.104*
			(0.005)	(0.005)	(0.067)	(0.071)
$i \times d$			-77.664*		-51.456*	
			(0.004)		(0.057)	
Weighted $i \times d$				-343.215***		-237.350**
				(0.001)		(0.024)
Constant	1.956***	1.221***	-0.946	0.277	-0.677	0.217
	(0.000)	(0.000)	(0.243)	(0.589)	(0.396)	(0.674)
Observations	45	45	37	37	37	37

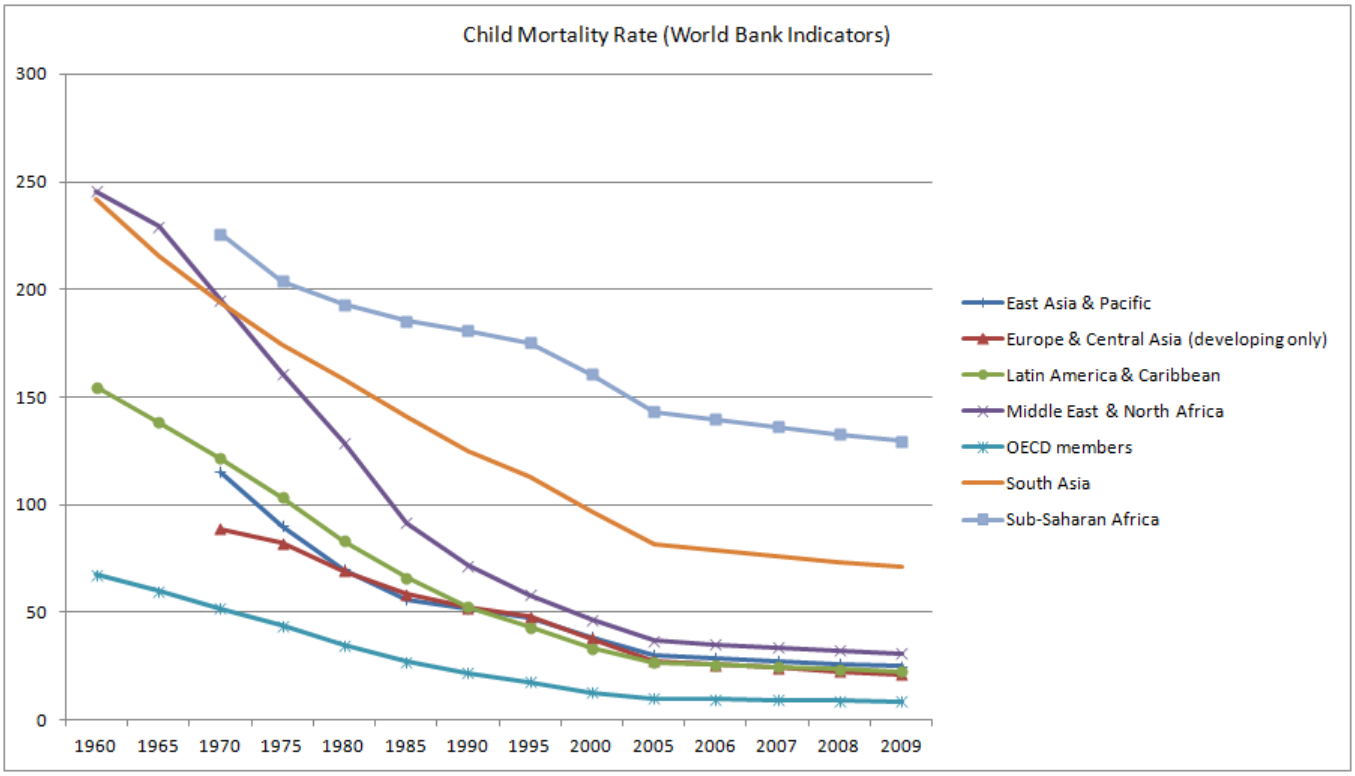
p-values in parentheses, standard errors robustly estimated.

***1%, **5%, and *10% levels of significance, respectively.

Table 3: Net effect on the TFR of a 1 percentage point decrease in i or d

Variable	Observations	Mean	Std. Dev.	Min	Max
Unweighted i (Regression 5)	37	-0.19%	0.07%	-0.37%	-0.04%
d for unweighted i (Regression 5)	37	6.14%	33.75%	-46.16%	99.38%
Weighted i (Regression 6)	37	-0.97%	0.30%	-1.74%	-0.32%
d for weighted i (Regression 6)	37	7.45%	39.75%	-48.54%	127.75%

Net effects are similar for regressions 3 and 4.



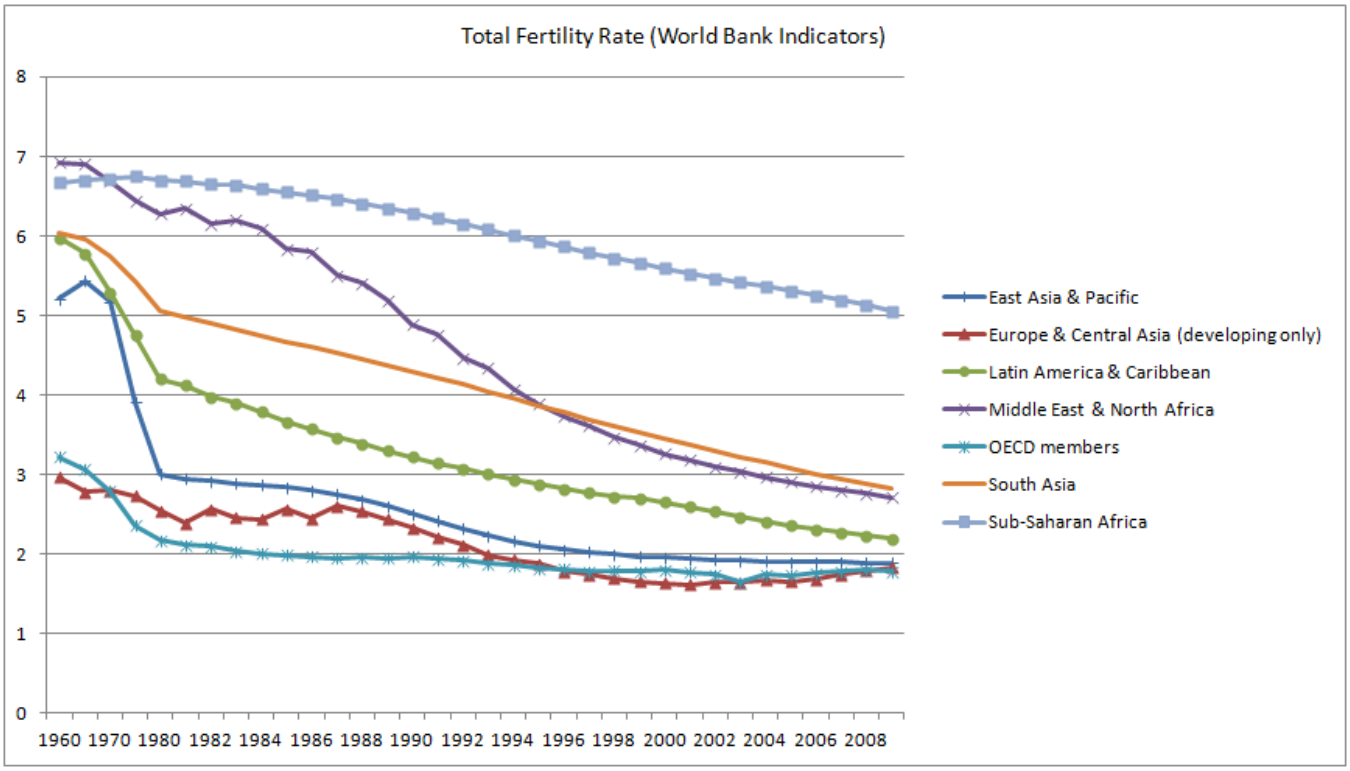


Figure 1: Demographic Transitions 1960-2009

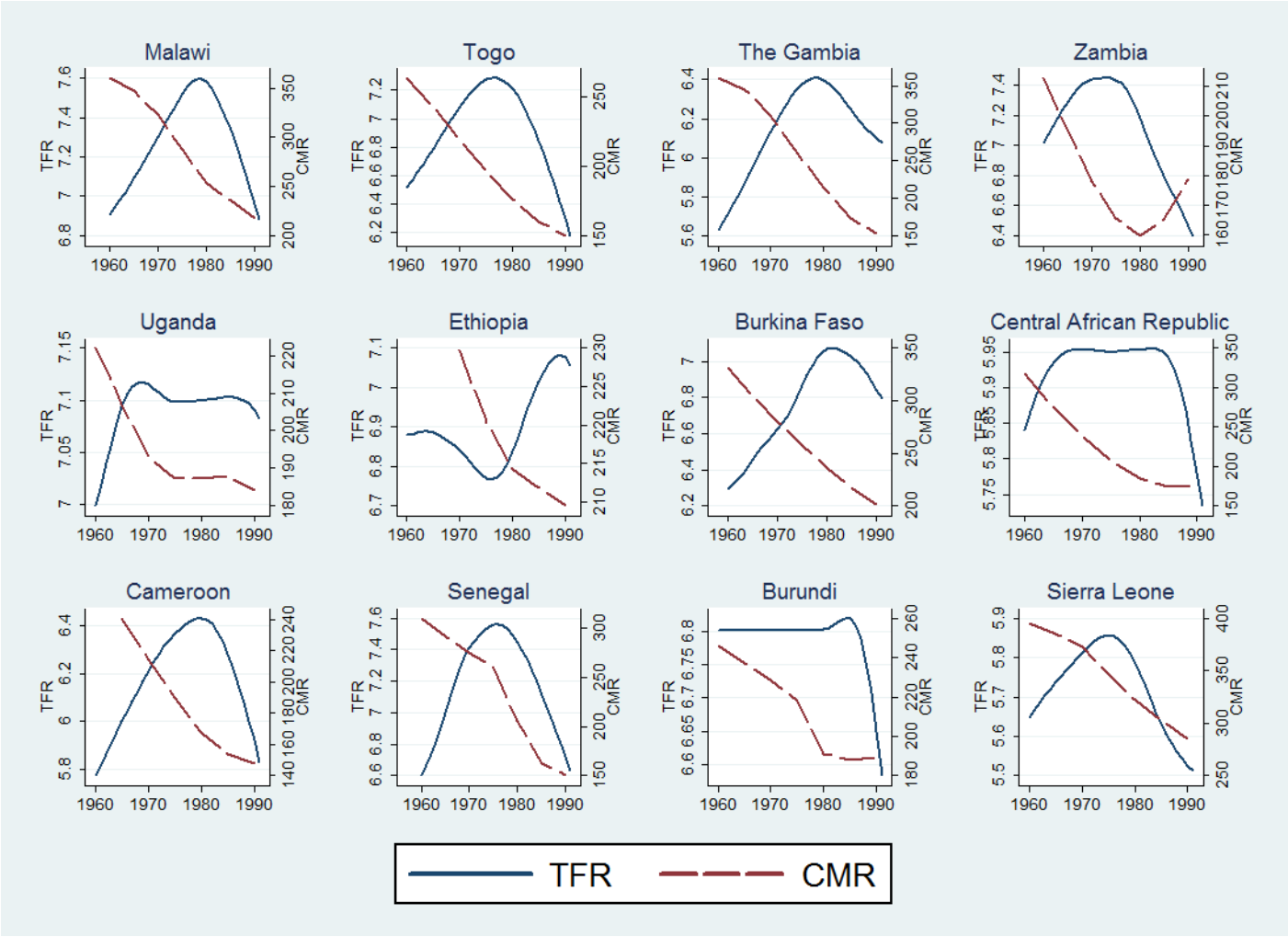


Figure 2: Total Fertility and Child Mortality Rates in Selected African Countries

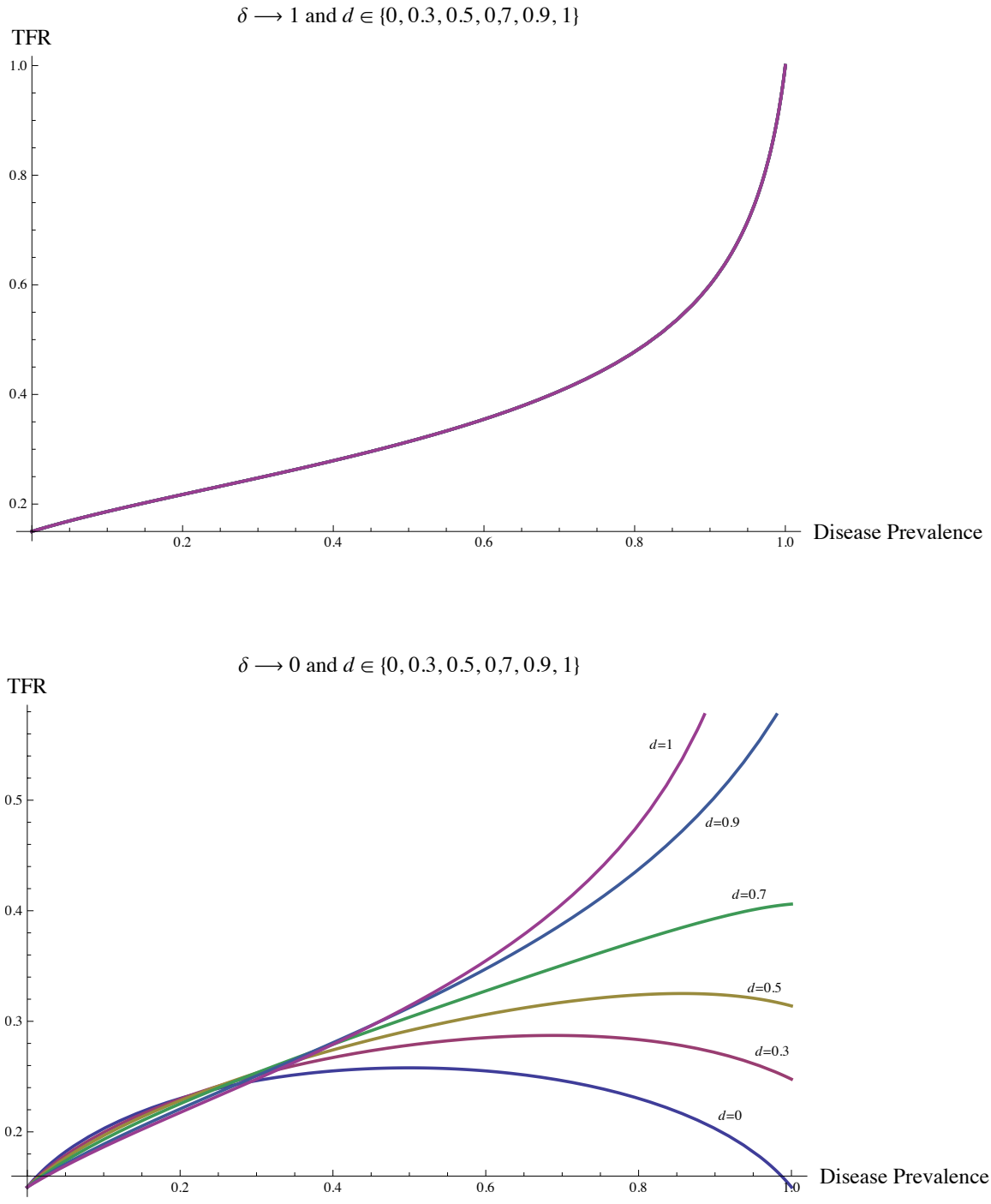


Figure 3: Fertility and Prevalence for δ = 1 and 0

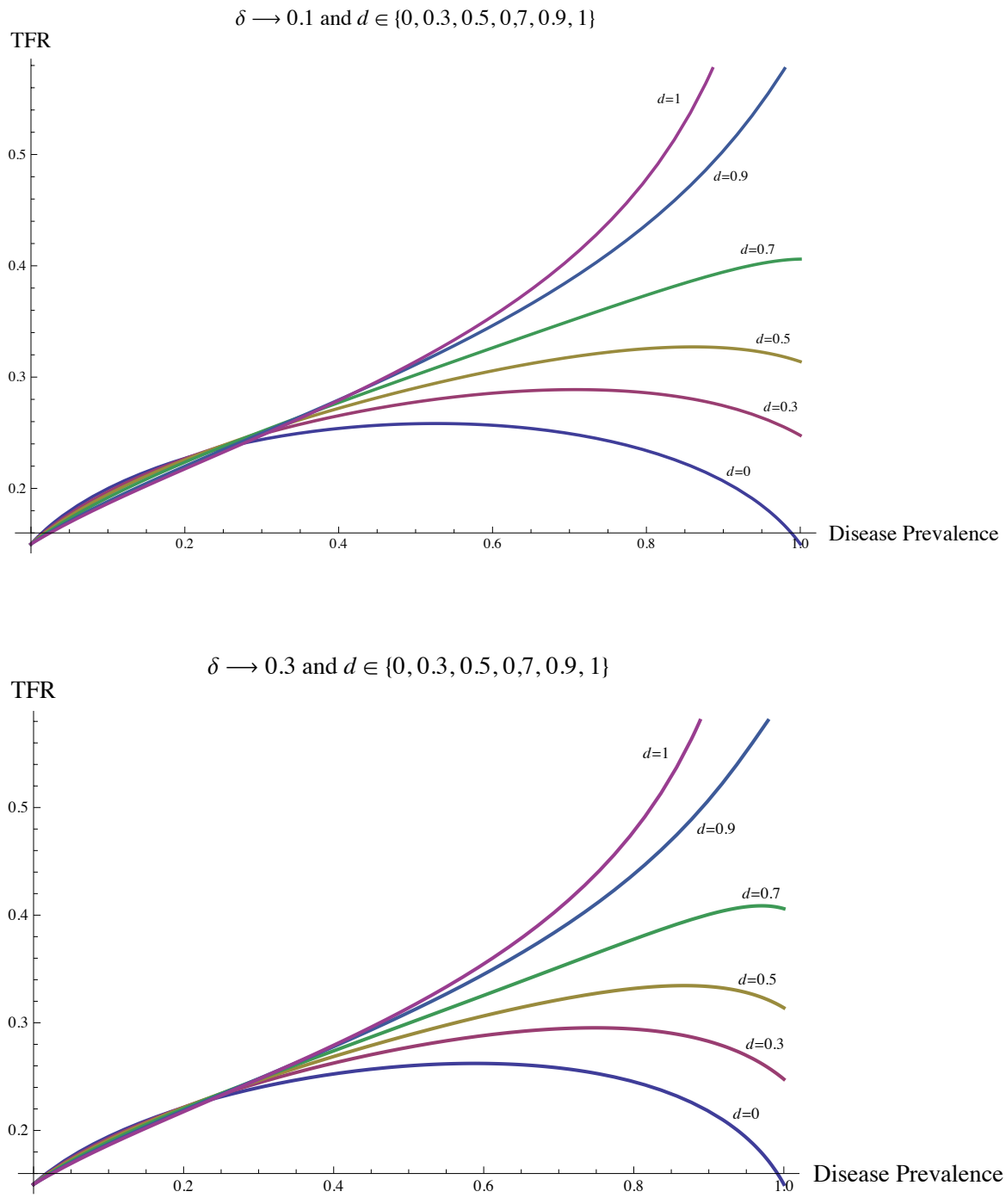


Figure 4: Fertility and Prevalence for δ = 0.1 and 0.3

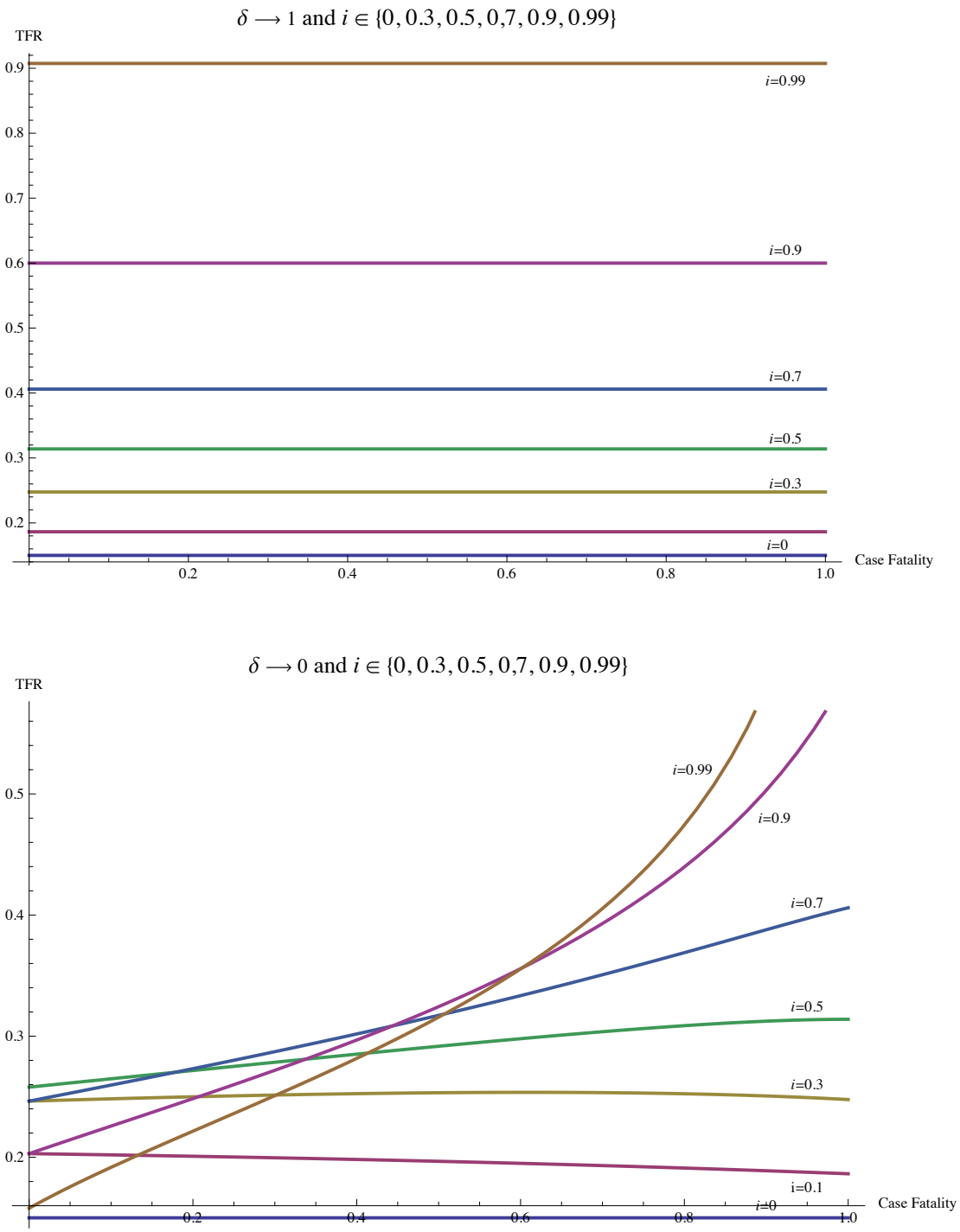


Figure 5: Fertility and Case Fatality for δ = 1 and 0

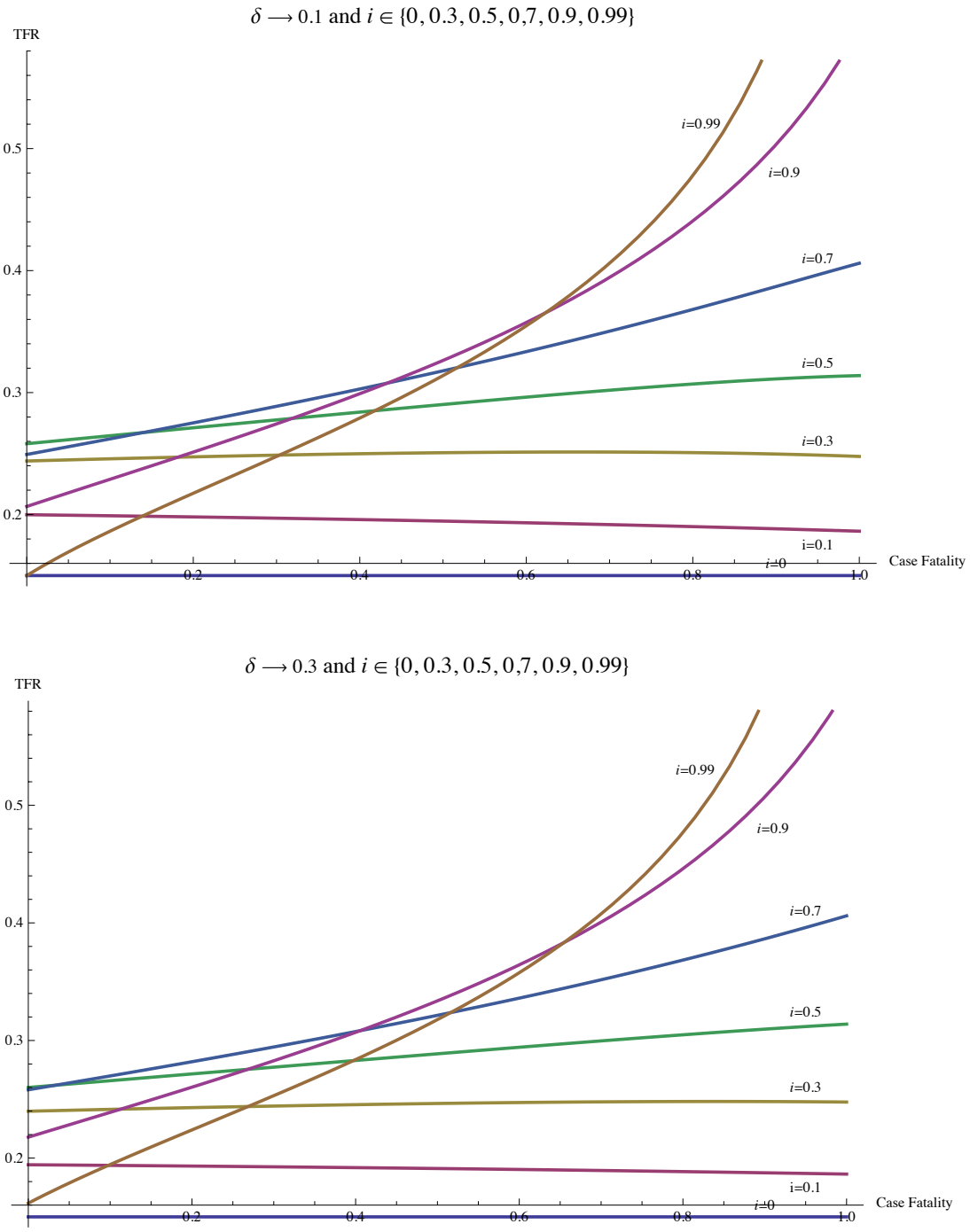


Figure 6: Fertility and Case Fatality for δ = 0.1 and 0.3

Electronic Supplementary Material for

Childhood Disease and the Precautionary Demand for Children

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Appendix

A Optimal Fertility using the Delta Method

Let $E(N_j) = \bar{N}_j$ for $j = 1, 2, 3$ and $\bar{\mathbf{N}} = (\bar{N}_1 \ \bar{N}_2 \ \bar{N}_3)$ where

$$\bar{N}_1 = n(1-i),\ \bar{N}_2 = ni(1-d),\ \bar{N}_3 = nid. \tag{1}$$

A second-order Taylor expansion around the means gives us

$$\begin{aligned} E(U(N_1, N_2, N_3)) \cong & U(\bar{\mathbf{N}}) + E(N_1 - \bar{N}_1)U_{N_1}(\bar{\mathbf{N}}) + \frac{E(N_1 - \bar{N}_1)^2}{2!}U_{N_1N_1}(\bar{\mathbf{N}}) \\ & + E(N_2 - \bar{N}_2)U_{N_2}(\bar{\mathbf{N}}) + \frac{E(N_2 - \bar{N}_2)^2}{2!}U_{N_2N_2}(\bar{\mathbf{N}}) \\ & + E(N_3 - \bar{N}_3)U_{N_3}(\bar{\mathbf{N}}) + \frac{E(N_3 - \bar{N}_3)^2}{2!}U_{N_3N_3}(\bar{\mathbf{N}}). \end{aligned} \tag{2}$$

Since $E(N_j - \bar{N}_j) = 0$ for $j = 1, 2, 3$, this simplifies to

$$\begin{aligned} E(U(N_1, N_2, N_3)) \cong & U(\bar{\mathbf{N}}) + \frac{E(N_1 - n(1-i))^2}{2!}U_{N_1N_1}(\bar{\mathbf{N}}) \\ & + \frac{E(N_2 - ni(1-d))^2}{2!}U_{N_2N_2}(\bar{\mathbf{N}}) + \frac{E(N_3 - nid)^2}{2!}U_{N_3N_3}(\bar{\mathbf{N}}). \end{aligned} \tag{3}$$

From the first and second derivatives of the utility function

$$
\begin{aligned}
U_{N_1} &= \frac{(1-\beta)(1-\theta)}{N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}}}, \qquad U_{N_1N_1} = -\frac{(1-\beta)(1-\theta)}{(N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}})^2} \\
U_{N_2} &= \frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{\alpha}{1-\theta}}}{N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}}}, \qquad U_{N_2N_2} = -\frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{2\alpha}{1-\theta}}}{(N^1 + N^2(1-\delta)^{\frac{\alpha}{1-\theta}})^2} \\
U_{N_3} &= 0
\end{aligned} \tag{4}
$$

we have

$$
U_{N_1N_1}(\bar{\mathbf{N}}) = -\frac{(1-\beta)(1-\theta)}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}, \tag{5}
$$

$$
U_{N_2N_2}(\bar{\mathbf{N}}) = -\frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{2\alpha}{1-\theta}}}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}. \tag{6}
$$

For the multinomial distribution, $V(N_j) = np_j(1-p_j)$ for $j = 1, 2, 3$, which implies

$$
E[N_1 - \bar{N}_1]^2 = ni(1-i), \tag{7}
$$

$$
E[N_2 - \bar{N}_2]^2 = ni(1-d)[1-i(1-d)]. \tag{8}
$$

Making these substitutions yields

$$
\begin{aligned}
E(U) \simeq & \beta \ln\left[\frac{\beta(1-\gamma n)z}{\beta+\theta(1-\beta)}\right] \\
& + (1-\beta)\ln\left[wn^{1-\theta}(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^{1-\theta}\left(\frac{\theta(1-\beta)(1-\gamma n)z}{\beta+\theta(1-\beta)}\right)^{\theta}\right. \\
& \left. -\frac{(1-\beta)(1-\theta)i}{2n(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}[1-i+(1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)(1-i(1-d))]\right]
\end{aligned} \tag{9}
$$

The optimality condition (10) in the paper is obtained by setting to zero the partial derivative of this expression with respect to n.

B Fertility Response to i and d

Rewrite equation (10) in the paper as

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

which implicitly solves for n as a function of i and d. Evidently $\partial n/\partial i = \Gamma_i/\Phi_n$ and $\partial n/\partial d = \Gamma_d/\Phi_n$.

Since $\Gamma(i,d) < 0$, it must be that $\Phi(n) < 0$ or, $n > [(1-\beta)(1-\theta)]/\gamma \equiv \overline{n}$, the fertility choice under certainty. In other words, the optimal fertility rate is higher because of the precautionary motive.

Next note that $\Phi_n < 0$ if $\Psi(n)[1+1/(1-\gamma n)] > 1$ where $\Psi(n) \equiv \gamma[\beta+\theta(1-\beta)]n/[(1-\beta)(1-\theta)(1-\gamma n)]$. Since n is restricted above by $1/\gamma$, it is easy to check that a sufficient condition for

$\Phi_n < 0$ is $n > \overline{n}$ which is true. Whether or not $\partial n/\partial i,\ \partial n/\partial d > 0$ therefore depends on whether or not $\Gamma_i,\ \Gamma_d < 0$.

Straightforward differentiation shows $\Gamma_i < 0$ if $i < i_L$. This threshold value

$$i_L \equiv \left[2[1+(1-\delta)^{2\alpha/(1-\theta)}]^2/[1+(1-\delta)^{2\alpha/(1-\theta)}] - [1-(1-\delta)^{\alpha/(1-\theta)}(1-d)]\right]^{-1}$$

is increasing in δ and d for empirically plausible values ($d < 0.54$). Hence $\partial n/\partial i > 0$ is more likely for relatively low values of i, or at a given prevalence rate, for relatively high values of δ and d.

Likewise $\Gamma_d < 0$ if $i > i_U$ and $\delta < 1$, where

$$i_U \equiv \frac{1}{2}\left[1+\frac{1}{2}(1-\delta)^{\alpha/(1-\theta)} - \left\{\left(1+\frac{1}{2}(1-\delta)^{\alpha/(1-\theta)}\right)^2 - \frac{2(1-\delta)^{\alpha/(1-\theta)}}{1+(1-d)(1-\delta)^{\alpha/(1-\theta)}}\right\}^{1/2}\right].$$

This threshold is decreasing in δ and increasing in d. We conclude that $\partial n/\partial d > 0$ is more likely for relatively high values of i, or at a given prevalence rate, for relatively low values of d and high values of δ. For $\delta = 1$, $\partial n/\partial d = 0$.

C Human Capital Response to Child Mortality

The child mortality rate id falls when i or d (or both) falls. Differentiating

$$h_1 = \frac{\theta(1-\beta)(1-\gamma n)z}{(\beta+\theta(1-\beta))\left(N_1+N_2(1-\delta)^{\frac{\alpha}{1-\theta}}\right)} \propto \frac{1-\gamma n}{n\left(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}}\right)}$$

with respect to i we get $\partial h_1/\partial i < 0$ *iff*

$$\frac{\partial n}{\partial i} > n(1-\gamma n)\left[\frac{1-(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}}}{1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}}}\right] \tag{10}$$

which is positive since $n < 1/\gamma$ in equilibrium. That is, a decrease in the prevalence rate increases quality investment in healthy children if and only if it lowers the TFR. The same condition is necessary for $\partial h_2/\partial d < 0$ since $h_2 = (1-\delta)^{\frac{\alpha}{1-\theta}} h_1$.