Childhood Disease and the Precautionary Demand for Children*

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Abstract

The prevalence of infectious diseases, their high case fatalities and long-term morbidity all contribute to the childhood disease burden. This paper proposes a quantity-quality model of fertility that emphasizes infectious disease morbidity and mortality. The fertility response to a decline in child mortality depends on the morbidity effect of the disease, the level of disease burden, and whether prevalence rates or case fatalities decline. Fertility rates follow 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. The theory is able to explain why fertility has not always followed child mortality declines in the developing world.

Keywords: Infectious Disease, Child Mortality, Morbidity, Fertility, Precautionary Demand, Quantity-Quality Tradeoff, Demographic Transition
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1 Introduction

The causes of the demographic transition are subject to much debate. The conventional view asserts that falling child mortality paves the way for a subsequent fertility transition. That view was challenged by the European Fertility Project: evidence that mortality did not always precede Western Europe's fertility transition in the nineteenth century has prompted researchers to identify other factors such as modernization and social upheaval (Caldwell, 2004), diffusion of ideas, and the emergent role of human capital in industrialization (Galor, 2005).[1]

This paper starts with the assumption that mortality leads fertility declines. We do so because, despite notable exceptions like France[2] and the US, child mortality declines preceded the fertility transition in many early industrializers like England, Germany and Sweden. In England, for example, a sharp decline in deaths from infectious disease began around 1872, followed about five years later by declines in the total and net fertility rates (Arora 2003, 2005). This connection between mortality and fertility has become tenuous in more recent time. Many developing countries continue to experience high fertility even after dramatic improvements in child mortality.

To interpret the evidence, we move beyond the effect of mortality on fertility by emphasizing infectious disease morbidity. We build on Sah (1991) and Kalemli-Ozcan's (2003, 2008) work on the precautionary demand for children. Our key innovation is separating the childhood disease burden into child mortality and morbidity. 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 morbidity effect lowers the marginal return from parental investment on unhealthy children. Survivors of infectious diseases are, consequently, lower quality than children who never (significantly) contracted them in the first few years after birth.

The desired fertility of risk-averse parents depends on the overall burden of infectious disease, its prevalence and its impact on child mortality and morbidity.[3] How a decline in

[1] De Bruijn (2006) offers a good overview of the broader literature and Doepke (2005) of economic demography.

[2] French life expectancy at birth began a steady rise around 1872, yet in many regions fertility began to decline much prior to 1872 (Bonneuil, 1997). Indeed France was a leader in Europe's fertility decline. Lesthaeghe and Neels (2002) argue that the early fertility decline in France was, at least partly, a response to declining wages due to recession when parents could not afford as many children.

[3] Uncertain parents in Sah (1991) and Kalemli-Ozcan (2003) overshoot their desired fertility to insure against the possibility of too few surviving children. Hence there is little investment in child quality. The inclusion of two sources of uncertainty, in disease exposure and in survival from that exposure, amplifies the precautionary

child mortality affects fertility depends on whether mortality falls from improvements in case fatality or disease prevalence. Besides lowering uncertainty, the latter ensures that more surviving children are of higher quality and, consequently, strongly impacts desired fertility.

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 of producing a surviving child falls. Fertility declines do not necessarily follow mortality declines, a scenario particularly relevant for developing countries that have experienced sharp reductions in child mortality without an ensuing fertility transition.

Under-five mortality has declined globally by 50% between 1960 and 2002 (UNICEF)[4] and this has coincided with a global decline in the total fertility rate (TFR). Yet many developing countries have not completed, or even begun, the fertility transition. Child mortality rates remain high – as high as 26% in Sierra Leone – as do fertility rates – Nigerian women have up to eight children on average (CIA World Factbook, World Bank). The prevalence of infectious disease is a natural explanation for both. Infectious diseases comprise seven of the top ten causes of child mortality in developing countries today (Table 1). Five of these, pneumonia, diarrhea, malaria, measles and AIDS, account for half of all deaths among children under the age of five.

A wealth of medical evidence shows that infectious disease in early childhood hampers cognitive or physical development and predisposes one to other diseases later in life. Bronchitis, pneumonia and whooping cough before age 5 have been linked to diminished respiratory function at ages 59-70 (Barker 1994). Acute rheumatic fever damages heart valves, while late-stage syphilis, measles, and malaria can affect the functioning of the circulatory system. Typhoid too shows cardiac involvement (Khosla 1981). Infections that disrupt absorption, such as diarrheal infections, deprive the body of nutrients necessary for optimal cellular growth (Martorell 1980, Martorell and Habicht 1986, Mata 1978), affecting cognitive abilities (Eppig *et al.*, 2010). In some cases, survivors of infection become susceptible to other health problems because "during infection, the biological system diverts its resources from cellular growth toward the synthesis of antibodies and repairing damaged tissue" (Arora 2005 p. 213). In other cases, an infectious disease is the direct cause of a non-infectious disease, as in the case of cervical cancer whose primary cause is the human papillomavirus.

That infectious diseases early in life have lifelong health consequences is also borne out by more indirect evidence, for instance, adult heights. Arora (2005) reports that infectious

motive.

Tamura (2006) distinguishes between infant and child mortality rates but parents invest the same amount on each child at birth. Here parents invest in children after survival outcomes are known, and they can invest differently in healthy and unhealthy children.

[4] http://www.unicef.org/mdg/childmortality.html

diseases started a downward decline in 1872 in England and Wales, followed from 1890 onwards by a sharp upward trend in the height of 18-year-old British males.[5] The cross-sectional evidence shows that adult height is associated with higher earning (Strauss and Thomas 1998), based on which it is not implausible to imagine that these secular health improvements generated significant productivity gains. There is also good evidence on the complementarity between health and returns from education during the early years (Behrman 1996). Hence the infectious disease burden bears upon childhood mortality as well as future productivity.

Explicitly recognizing the morbidity effect of infectious disease among children delivers both positive and negative fertility responses to disease. In Barro and Becker (1988), a decline in the child mortality rate reduces the average cost of raising a surviving child, prompting parents to have more children. The demand for children rises only 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 mortality declines.

Adaptations of the Barro-Becker model show an unambiguously positive response of the TFR to mortality.[6] Any negative relationship resulting from the average cost effect is completely overshadowed by positive precautionary forces. The theory proposed here accounts for the Barro-Becker average cost effect and determines circumstances in which it dominates the forces (precautionary demand for children, quantity-quality tradeoff) contributing to 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 and counteracts the negative average cost effect when infection rates decline but amplifies it when case fatality rates 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.[7]

The partial equilibrium model developed here has obvious general equilibrium consequences. 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

[5]Vaccination was made compulsory in 1853 and in 1871 legislation was introduced requiring all poor law unions to appoint vaccination officers and to set up a system of registration; this system, with only minor alterations, lasted until 1948 (Drake *et al.* 2001).

[6]Among such studies are Boldrin and Jones (2002), Cervellati and Sunde (2007), Galor and Weil (1999), Kalemli-Ozcan (2003, 2008), Soares (2005), Tamura (2006).

[7]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.

may have unintended consequences for fertility depending on how diseases are combated: a declining disease burden will not necessarily reduce mortality and fertility unless it concurrently lowers morbidity. This partly explains why the fertility transition in sub-Saharan Africa has lagged child mortality declines. As Akachi and Canning (2008) show, the reduction in infant mortality has been accompanied by persistent morbidity challenges.

We present the theoretical model in Section 2. Fertility and human capital decisions are analyzed in Section 3, while Section 4 offers further discussion and policy implications. Section 5 concludes.

2 The Model

An individual lives for three periods – childhood, adulthood and old age – though not all children survive their early years. Children are born with an innate health capital h_0 and are exposed to infectious diseases in early childhood from which they may or may not survive. Let i denote the infection rate among these children. A fraction i of children born contract infectious diseases of whom a fraction $1-d$ survive, where $i, d \in (0,1)$. The parameter d is the case fatality rate from a disease while the product id is the child mortality rate.[8] What distinguishes our model from existing ones is the idea that exposure to infectious diseases has a morbidity effect even when children survive. To be specific, infections depreciate a child's health by the fraction $\delta \in (0,1)$.

Uncertainty about child survival resolves after the first few years of a child's life. Subsequently parents invest in the human capital of these children, for example, in their education or health. A child's health capital determines the productivity of such investment.

Surviving children are of two types. 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 assume

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

where $\alpha, \theta \in (0,1)$. A child's consumption is not modeled explicitly and is instead subsumed in his parent's.

[8] 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.

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 (determined by their past disease experience and human capital investment), yields income wH. Children are valued because they financially support their elderly parents, an assumption that is 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 old parent and receives the same fraction of each child's earnings in old age.[9] 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.

Assuming preferences over consumption are logarithmic and normalizing $h_0 = 1$, a young adult maximizes his expected lifetime utility

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

subject to[10]

$$\begin{aligned} c + N_1 h_1 + N_2 h_2 &\leq (1-\gamma n)(1-\tau) wH, & (2)\\ N_1 + N_2 + N_3 &= n, & (3) \end{aligned}$$

where w' is the efficiency wage rate faced by children in their adulthood, $\gamma \in (0,1)$ is the fixed time cost of having a child, and $\beta \in (0,1)$ is the weight attached to consumption in youth versus old age. To conserve notation we define $z \equiv (1-\tau)wH$ and $x \equiv \tau w'$.

Adults face uncertainty in the number of surviving children and explicitly recognize the random nature of a child's survival from infectious disease. Since human capital investment (h_1, h_2) occurs after this uncertainty is resolved, we will solve the model using backwards induction. That is, we identify optimal consumption and investment decisions for the parent, conditional on n, N_1 and N_2. We then use the Delta Method to solve for the fertility decision, à la Kalemli-Ozcan (2008). The difference between our work and Kalemli-Ozcan's is that there are three rather than two possible outcomes for each child born.

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

$$p(N_1 = n_1, N_2 = n_2, N_3 = n_3) = \frac{n!}{n_1! n_2! n_3!} p_1 p_2 p_3 \tag{4}$$

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

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

[10] 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 $1/\gamma$.

survive disease but remain unhealthy, and $\bar{N}_3 = n - \bar{N}_1 - \bar{N}_2 = nid$ children succumb to disease. We will 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 consider the certainty version of the model. The number of survivors of each type is simply taken to be its expected value and the parent chooses h_1, h_2, 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:

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

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.[11]

These results contradict historical data from demographic transitions in which declining mortality rates are associated with declining fertility and increasing human capital investment. Under certainty, child investment is inversely related to the number of survivors, which increases when either i or d declines. Since total fertility, n, does not decline to compensate, an increase in either i or d raises the number of surviving children so that investment per child falls.

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

[11] Kalemli-Ozcan (2003, 2008) also obtains this result under certainty; under CRRA, the TFR actually increases in response to lower mortality. In Boucekkine *et al.* (2009), who include a labor-leisure tradeoff, fertility declines with mortality despite CRRA and certainty about child survival.

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 childhood disease experience. 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. Therefore, we assume that uncertainty about child survival is resolved after infancy and that human capital investment occurs after this time.

Parental decisions are solved sequentially. First, given the fertility decision (n) and the number and type (N_1 or N_2) of survivors, adults choose h_1, 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:

$$h_1(n) = \frac{\theta(1-\beta)(1-\gamma n)}{\{\beta+\theta(1-\beta)\}(N_1 + (1-\delta)^{\frac{\alpha}{1-\theta}} N_2)} z, \tag{5}$$

$$h_2(n) = (1-\delta)^{\frac{\alpha}{1-\theta}} h_1(n), \tag{6}$$

and

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

Quality investment in children now depends negatively on the number of survivors and on total fertility. Note especially that this investment depends on child mortality even though investment decisions are made after the uncertainty of number of survivors 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

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

which the parent then maximizes with respect to n, taking into account uncertainty regarding (N_1, N_2) as specified by (4) above. Appendix B details how this optimization leads to the quadratic first-order condition:

$$n - \frac{\gamma\{\beta+\theta(1-\beta)\}n^2}{(1-\beta)(1-\theta)(1-\gamma n)} = \frac{-i[1-i+(1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)[1-i(1-d)]]}{2[1-i+i(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)]^2}. \tag{7}$$

The following section analyzes this fertility choice in depth.

3 Fertility, Quality Investment and the Disease Burden

A change in the childhood disease burden – a decline in disease prevalence and case fatalities and the ensuing changes in morbidity – affects fertility decisions in various ways. In order to understand these, consider how the three parameters (i, d, δ) affect childhood mortality, morbidity and the quantity-quality tradeoff faced by parents. We discuss their effects on the total fertility rate, the net fertility rate and human capital investment.

3.1 The Total Fertility Rate

The first-order condition (7) is a 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.

Since fertility is a highly non-linear function of the parameters in the general case, it is best analyzed by numerically solving for n. For simplicity set $\gamma = 1$ which restricts us to $n \in (0, 1)$ instead of $n \in (0, 1/\gamma)$. For more decisive interpretations we focus on extreme values of δ which yield conclusions consistent with more general cases. Appendix C establishes these results formally.

Disease Prevalence and the TFR

Figures 1 and 2 depict the fertility decision as a function of (i, d) for $\delta = 1$ and $\delta = 0$ respectively. The two figures share the same set of values for the other parameters, $\alpha = 0.9$, $\theta = 0.9$, $\beta = 0.5$, besides $\gamma = 1$.

In Figure 1, $\delta = 1$ implies the child mortality rate is effectively i rather than id. Since unhealthy children are completely unproductive as adults and unable to support their parents in old-age, it is irrelevant for human capital investment if an infected child does or does not survive. In this case the structure is similar to Kalemli-Ozcan (2003, 2008).[12] Suppose now the prevalence rate drops, for example, from a new vaccine that lowers overall disease prevalence and infection rates among children. What effect does this have on the TFR?

Figure 1 identifies the relation between n and i for various values of d. The case fatality rate has no impact on fertility choice since the $n(i)$ functions for different values of d coincide.

[12] This isomorphism depends crucially on the way parental preferences are modeled. If parents were altruistic in the Barro-Becker sense or derived warm glow from investments in each type of child, they would care about unhealthy children even if $\delta = 1$. These alternative altruism specifications complicate the model considerably without adding much to our analysis of the morbidity effect.

Fertility is monotonically increasing in the disease prevalence rate which is also the effective child mortality rate. A decline in i, in other words, unambiguously lowers the TFR.

Turn now to Figure 2 where $\delta = 0$ and all surviving children are equally healthy. But newborns are exposed to two sources of uncertainty, first whether or not they contract infectious diseases, and secondly, whether or not they survive from it. Unlike in the case of $\delta = 1$, when $\delta = 0$ the model does not simplify to the standard case because d now plays a unique role.[13]

As the figure shows, the response of fertility to the prevalence rate can be nonlinear depending on case fatalities. 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 Figure 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.

Three forces determine the response of n to changes in i. First is the precautionary motive that points to a positive relationship between fertility and disease prevalence. Faced with child survival uncertainty, parents have more children than they ultimately desire: a decline in child mortality will reduce fertility as more children survive.

Secondly, when child mortality declines, survivors become cheaper to produce and desired fertility goes up (Barro and Becker 1988). The total cost of child rearing consists of the fixed birth cost (γn) and the cost of investing in child quality after survival outcomes are realized ($N_1 h_1 + N_2 h_2$). For a given n, the average birth cost of producing a surviving child decreases as $(N_1 + N_2)/n$ rises.

Investments h_1 and h_2 decrease with the number of survivors for a given level of n as (5) and (6) show. Hence the average investment cost per surviving child,

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

decreases when $N_1 + N_2$ rises because quality investment in unhealthy children is lower and because both types of quality investment fall. Hence the combined average cost per surviving child declines with lower disease prevalence.

The third channel, a key contribution of this paper, operates through the morbidity effect. Parents substitute between the quantity and quality of children depending on the disease

[13]The standard case implicitly assumes that all children contract infectious diseases and the uncertainty is with respect to survival alone. Alternatively, as in the case under discussion, one could interpret the standard case as (i) uncertainty with respect to contracting disease with survival from it known for sure, and (ii) whether or not the child survives is irrelevant for investment decisions since survivors and non-survivors alike do not support parents in old age.

burden. As disease prevalence falls, 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 first and third effects work in the same direction, the second one in the opposite. Larger is δ, the stronger is substitution towards quality. Moreover, the morbidity effect is stronger when most of the surviving children have experienced infectious disease, that is when i is high and d is low.

The sharpest contrast between the cases $\delta = 1$ and $\delta = 0$ in Figures 1 and 2 occurs for high i and low d. This is because in Figure 2, at high i and low d values, the quantity-quality tradeoff due to the morbidity effect disappears and only the first two channels are at work. Since most children born get infected, few of them die and all are equally healthy, the precautionary motive is weak. As a result, the average cost effect drives the negative relationship between fertility and disease prevalence in Figure 2 at high values of i and low values of d.

Case Fatality and the TFR

Now consider the relationship $n(d)$. Suppose that d declines because of the availability of an antibiotic. 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 (vaccines or behavioral changes). The response of the TFR to d is charted in Figure 3 for various 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, they have more children in order to substitute for their low 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 since more unhealthy children survive, the average surviving child is also cheaper in terms of human capital investment. The maximum TFR is 0.3 in Figure 2 ($\delta = 0$) but 0.4 in Figure 1 ($\delta = 1$) where the morbidity effect is stronger.[14]

An antibiotic lowers mortality more for very prevalent and fatal diseases, and increases morbidity more for high δ diseases. Changes in case fatality rates only elicit a noticeable (and positive) fertility response when morbidity effects are low (δ 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, so an increase in i or d cannot decrease n anyway.[15]

[14] As expected, fertility is higher in this model when θ, the returns to quality investment, is lower.

[15] In Figure 2, the special case of $i = 1$ yields a monotonic positive relation between n and d. Here the

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 (see Appendix C for calculation of the precise thresholds).[16]

Proposition 1 *Under uncertainty, fertility falls with declining disease prevalence (i) if the disease is fatal or (ii) if the disease is not fatal but causes severe long-term morbidity.*

In contrast, fertility rises with declining disease prevalence if the disease is not fatal and does not cause severe long-term morbidity.

To conclude the discussion, consider a simple example that illustrates the differential effect of mortality and morbidity on the TFR. Suppose $i = 0.1$, $d = 0.5$ and $\delta = 0.5$ which imply a child mortality rate of 0.05 or 5%. A 20% reduction in either prevalence or case fatality rates will reduce the mortality rate from 5% to 4%. However, the impact 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 fixed 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

$$\begin{aligned}
&\{1-(1-d)(1-\delta)^{\frac{1+\alpha-\theta}{1-\theta}}\}h_1\Delta i - i(1-\delta)^{\frac{1+\alpha-\theta}{1-\theta}}h_1\Delta d \\
&= 0.02\left[(1-0.5\times 0.5^{\frac{1+\alpha-\theta}{1-\theta}}) - 0.01\times 0.5^{\frac{1+\alpha-\theta}{1-\theta}}\right]h_1 \\
&= 0.02\left[(1-0.5^{\frac{1+\alpha-\theta}{1-\theta}})\right]h_1
\end{aligned}$$

which is positive for $\alpha, \theta \in (0,1)$.

3.2 The Net Fertility Rate

Kalemli-Ozcan (2003) and 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.

effective child mortality rate is d. Hence the relation $n(d)$ is again similar to Kalemli-Ozcan's model.

[16] A low i here could be as high as $i = 1/2$. See Appendix C.

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

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

and

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

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.

Investment in Human Capital

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

Proposition 2 *Under uncertainty, parental human capital investment in children rises when the disease burden falls if the total fertility response to changing infection 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 (e.g. education subsidies); otherwise combating the disease burden may actually raise net and even total fertility, thereby lowering overall human capital investment.

4 Discussion

Infection and case fatalities were historically high in many Western countries, and it was primarily new knowledge about germs that triggered sharp declines in 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 declined, child mortality fell significantly. Our model suggests that lower disease prevalence meant that fewer children got sick and survivors became healthier.

Consequently desired fertility fell and human capital investment in children rose. The model generates such a quantity-quality tradeoff especially when morbidity declines. Historical data on stature in Western countries show that cohorts of children who experienced declining mortality rates became taller (thus healthier) adults relative to their predecessors, suggesting morbidity declined in tandem with mortality (Arora 2005).

Worldwide reductions in child mortality over the past half century have occurred through both lower disease prevalence (improved sanitation in urban areas, vaccination) and lower case fatalities (antibiotics). But the fact that the infectious disease burden remains high in many developing countries suggests that much of the decline has come from averting deaths from diseases rather than from preventing those diseases in the first place. If infectious disease mortality but not morbidity declines, high fertility rates can persist as parents continue to supplement the low quality of their children with greater quantity. This is a particularly pressing problem in sub-Saharan Africa where infant mortality rates have fallen since 1960 but cohorts of children affected have not grown up to be much healthier as measured by adult height (Akachi and Canning 2008).

Depending on the more prevalent infectious diseases and the manner in which they are combated, countries may or may not be poised for the demographic transition and the resultant 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 widely prevalent in developing countries, and their treatment prevents deaths but cannot reverse the damage. Effective treatments exist, for example, for diseases like leishmaniasis, which damages the spleen and liver and can cause anaemia, and schistosomiasis, 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 renders its survivors paralyzed (high δ) and for which there exists no cure, is one infectious disease to have been eradicated globally.[17] The model predicts this global eradication would have had a significant impact on fertility rates, a hypothesis that deserves further attention.

Given the virulent nature of their disease burden, the average δ for childhood illnesses is clearly substantial for developing countries. Resources devoted towards lowering this burden will be more efficiently spent, in terms of lowering the TFR and raising education and its returns, when they are able to reduce both mortality and morbidity.

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

Malaria

Take the case of malaria which is prominent throughout much of sub-Saharan Africa. Malaria contributes significantly to child mortality (Table 2). It also generates substantial morbidity effects: it is the major cause of childhood anemia and maternal anemia during pregnancy, of low birth weight, and in the case of cerebral malaria, persistent neurological deficits (Gollin and Zimmerman 2007). The most common effect, anemia, is associated with poor school performance.

In terms of the theory, malaria should be viewed as a moderate δ disease. It causes long-term health problems that, while serious, do not render individuals completely unproductive. Malaria-induced anemia can impact child quality and returns to human capital significantly. When children in the southern United States were treated for hookworm, a disease whose primary morbidity consequence is also anemia, teachers reported a remarkable improvement in children's scholastic performance (Bleakley 2007).[18]

Malaria tends to be more fatal in moderate prevalence areas (high d/low i) and relatively less so in high prevalence areas (low d/high i) (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.[19] Thus 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 weakest for the latter.

In endemic areas adults have built up some immunity since childhood, and malaria infections in children are more quickly recognized as such, and therefore patients receive prompter treatment, avoiding fatalities.[20] Delaying treatment by 5-10 days raises case fatality by a factor of 5, and delay of 10-20 days raises case fatality 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 influxes of malaria, malaria fatalities heavily affect all age groups.

Accordingly, reducing malaria transmission will have a stronger impact on fertility in moderate transmission areas, since that is where case fatality rates are higher. Conversely, the

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

[19] Lower transmission areas are those at higher altitudes where fewer mosquitoes live.

[20] 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).

reduction in mortality per fatality averted through treatment of malarial infections 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 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. On the other hand, HIV/AIDS primarily affects adults by raising adult mortality rates. In our model a rise in adult mortality can be interpreted as higher β which reduces fertility since children are investment goods that benefit parents only in old age.

Recent empirics on the demographic consequences of this epidemic offer mixed evidence. Kalemli-Ozcan (2010) finds a positive fertility impact, while Young (2005) finds a negative one.[21] 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).

5 Conclusion

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. The strongest positive response of fertility to disease prevalence occurs where both mortality and morbidity rates change in the same direction.

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

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 promote human capital accumulation. We illustrated this for the case of malaria.

Future work will empirically assess the impact of morbidity on the demographic transition in developing countries today. An analysis of health interventions in malarial regions, for example, can more concretely shed light on this paper's 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 role of the precautionary motive, another topic that deserves further exploration.

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A Tables

Leading causes of death in children in developing countries, 2002

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 Ten Causes of Child Mortality in Developing Countries

Table 2: Contribution of Malaria to (under age five) Child Mortality (%), 2000

	Percentage of child deaths due to	
Country	Malaria	Neonatal causes
Angola	8.3	22.2
Benin	27.2	25.0
Botswana	0	40.3
Burkina Faso	20.3	18.3
Burundi	8.4	23.3
Cameroon	22.8	24.8
Central African Republic	18.5	27.2
Chad	22.3	24.0
Comoros	19.4	37.3
Congo	25.7	30.9
Cote d'Ivoire	20.5	34.9
Dem. Rep. of the Congo	16.9	25.7
Equatorial Guinea	24.0	27.5
Eritrea	13.6	27.4
Ethiopia	6.1	30.2
Gabon	28.3	35.1
Gambia	29.4	36.6
Ghana	33.0	28.5
Guinea	24.5	28.8
Guinea-Bissau	21	24.1
Kenya	13.6	24.2
Lesotho	0	32.8
Liberia	18.9	29.1
Madagascar	20.1	25.6
Malawi	14.1	21.7
Mali	16.9	25.9
Mauritania	12.2	39.4
Mauritius	0	66.0
Mozambique	18.9	29.0
Namibia	0	38.5
Niger	14.3	16.7
Nigeria	24.1	26.1
Rwanda	4.6	21.7
Senegal	27.6	22.8
Seychelles	0	27.2
Sierra Leone	12.4	21.9
South Africa	0	35.1
Swaziland	0.2	26.8
Togo	25.3	29
Uganda	23.1	23.6
Tanzania	22.7	26.9
Zambia	19.4	22.9
Zimbabwe	0.2	28.1
Average	14.87	29.11

Source: World Health Organization

B 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

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

A second-order Taylor expansion around the means gives us

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

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

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

From the first and second derivatives of the utility function

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

we have

$$\begin{aligned} U_{N_1N_1}(\bar{\mathbf{N}}) &= -\frac{(1-\beta)(1-\theta)}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}, \\ U_{N_2N_2}(\bar{\mathbf{N}}) \ &= \ -\frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{2\alpha}{1-\theta}}}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}. \end{aligned}$$

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

$$\begin{aligned} E[N_1 - \bar{N}_1]^2 &= ni(1-i), \\ E[N_2 - \bar{N}_2]^2 &= ni(1-d)[1-i(1-d)]. \end{aligned}$$

Making these substitutions yields

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

The optimality condition (7) is obtained by setting to zero the partial of this expression with respect to n.

C Fertility Response to i and d

Define

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

which implicitly solves for n as a function of i and d. Evidently $dn/di = \Gamma_i/\Phi_n$ and $dn/dd = \Gamma_d/\Phi_n$. The results for extreme values of δ are consistent with the general results.

$\Gamma_i > 0$ if $i > i_L$, where

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

is increasing in δ and d. As d or δ increase, $\Gamma_i > 0$ becomes less likely. If $\delta = 1$, $\Gamma_i < 0$ for all i and d. If $\delta = 0$, $\Gamma_i > 0$ if $i > 1/2$ when $d = 0$, and $\Gamma_i < 0$ when $d = 1$.

$\Gamma_d > 0$ if $i < i_U$, where

$$i_U \equiv \frac{1}{4}\left[2+(1-\delta)^{\frac{\alpha}{1-\theta}} - \left\{\frac{4-(1-\delta)^{\frac{\alpha}{1-\theta}}[4d+(1-\delta)^{\frac{\alpha}{1-\theta}}\{-5+4d-(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)\}]}{1+(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)}\right\}^{1/2}\right]$$

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

Since $\Gamma(i,d) < 0$, it must be that $n > [(1-\beta)(1-\theta)]/\gamma \equiv \overline{n}$, fertility choice under certainty. $\Phi_n > 0$ if $n > \underline{n} \equiv [1-\{\beta+\theta(1-\beta)\}^{1/2}]/\gamma$. Since we are restricted by $n < 1/\gamma$ we have $\underline{n} \equiv [1-\{\beta+\theta(1-\beta)\}^{1/2}]/\gamma > \overline{n}$. While this means it is not always the case that $\Phi_n < 0$, given the response of n to i and d in Figures 1 and 2, one can conclude that $\Phi_n < 0$ for the parametric assumptions we made about β and θ. Note that $\underline{n} = 0$ when $\beta = 1$ or $\theta = 1$.

D Human Capital Response to i and d

Differentiating

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

we get

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

which is negative if

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

Similarly for $\partial h_1/\partial d$.

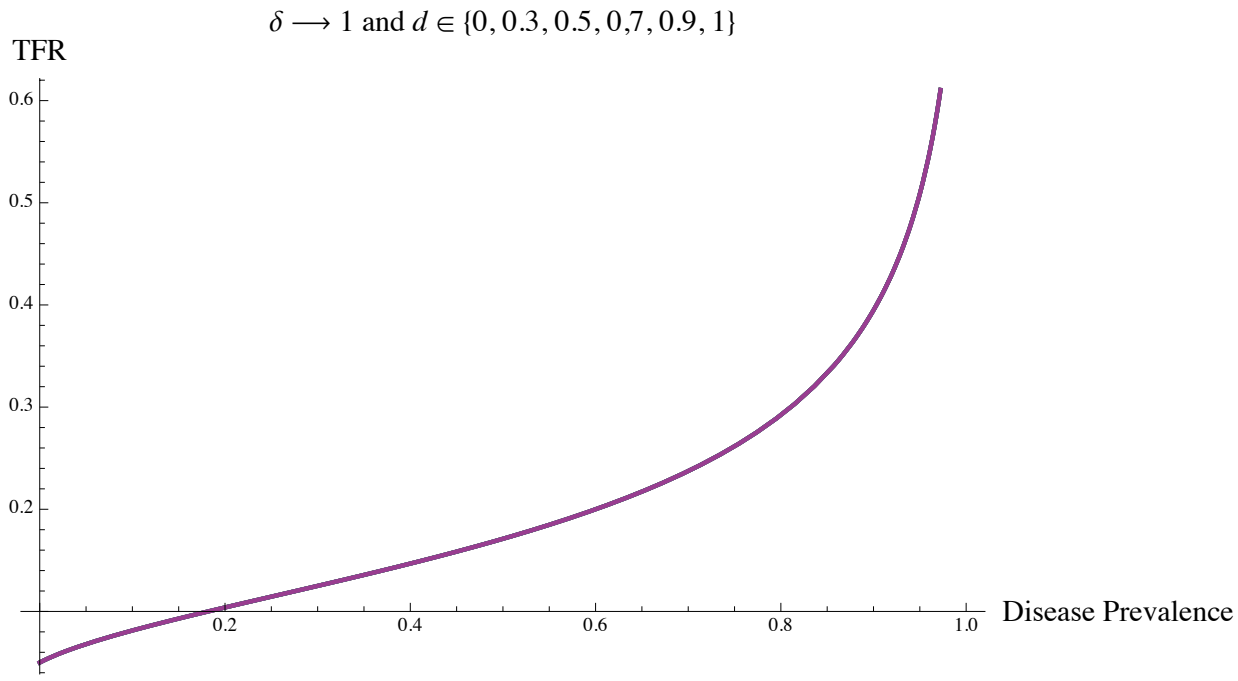


Figure 1: Fertility, Prevalence and Case Fatality when $\delta = 1$

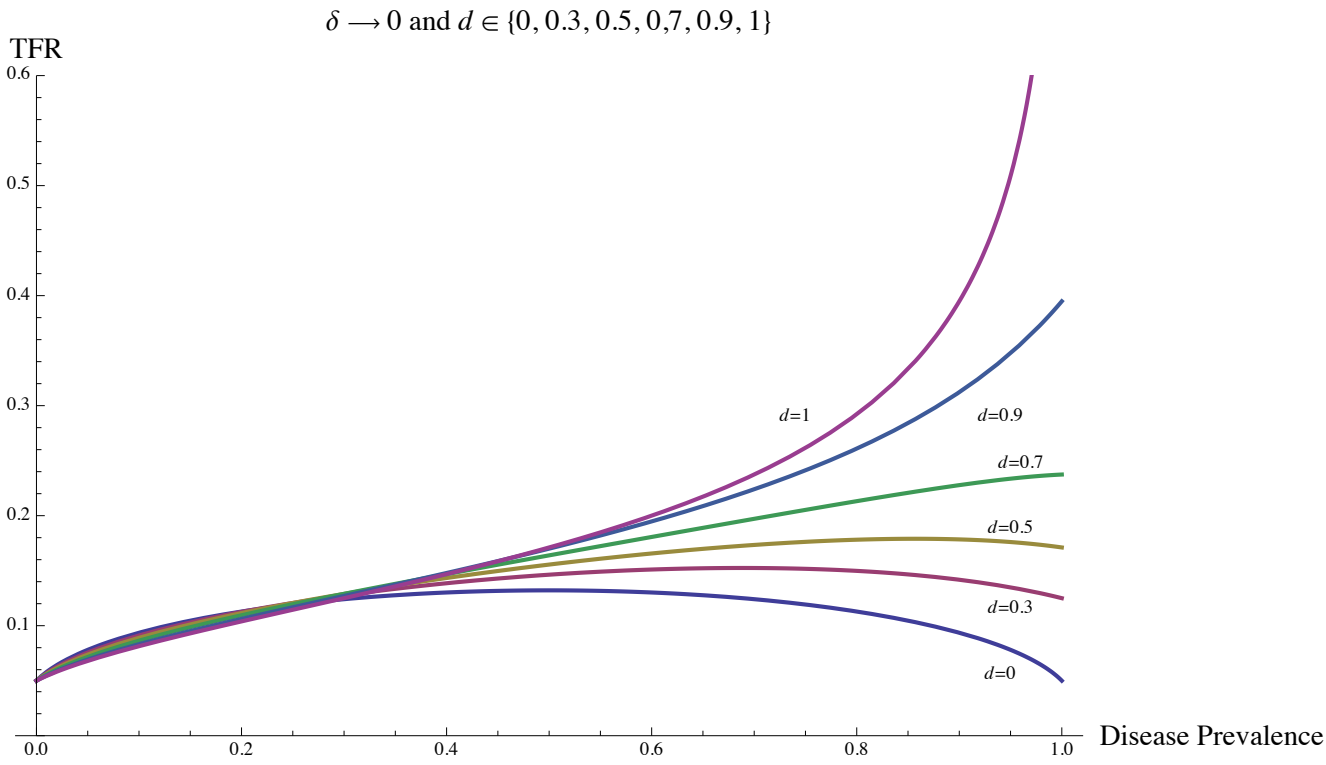
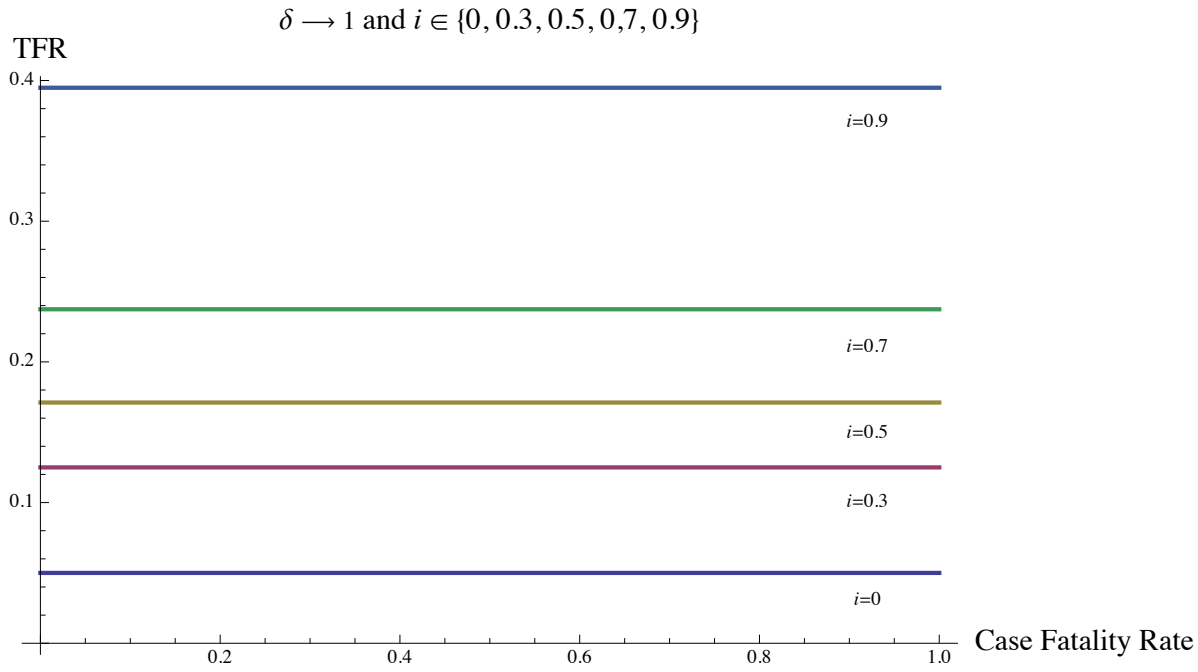


Figure 2: Fertility, Prevalence and Case Fatality when $\delta = 0$



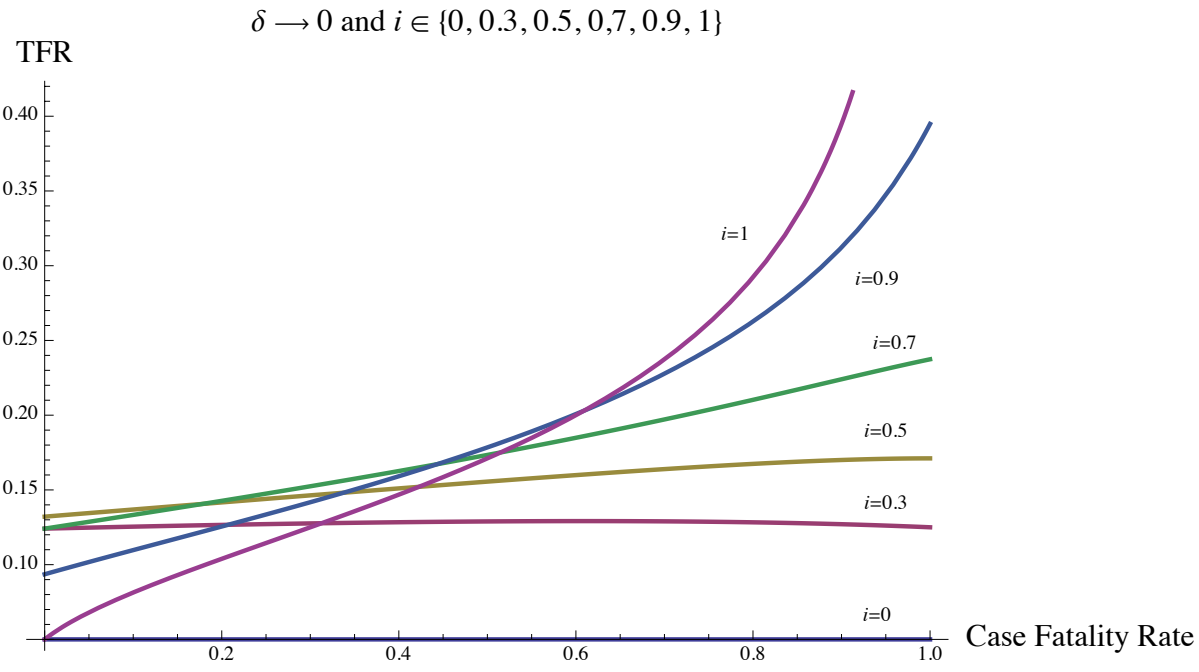


Figure 3: Fertility and Case Fatality