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ABSTRACT

Analyzing a variety of cross-national and sub-national data, we argue that high adult mortality reduces economic growth by shortening time horizons. Higher adult mortality is associated with increased levels of risky behavior, higher fertility, and lower investment in physical and human capital. Furthermore, the feedback effect from economic prosperity to better health care implies that mortality could be the source of a poverty trap. In our regressions, adult mortality explains almost all of Africa's growth tragedy. Our analysis also underscores grim forecasts of the long-run economic costs of the ongoing AIDS epidemic.

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1 Introduction: Mortality Matters

What causes a country to be trapped in poverty? The dismal numbers - over a billion people living on a less than $1 a day - do not lose, through familiarity, the capacity to shock. What could be weighty enough to explain why so many stay so poor? Development occurs only if people make provision for the future. If they see no future, there is no growth. We examine here a basic determinant of decision horizons: the risk of premature death.

The causal relationship between mortality and poverty is clearly bi-directional. On the one hand, in a poor country, unable to afford sanitation and medical care, people die young. Figure 1 displays the strongly negative relationship between income levels and adult mortality. On the other hand, where people have a short time horizon because they expect to die young, they have less reason to save and the economy fails to grow. Figure 2 shows that countries with high adult mortality also experience low rates of economic growth.

Both directions of causality, that poor countries have high mortality and that high mortality leads to low growth, seem straightforward. However, the magnitude of these effects remains unclear. This paper argues that the link from adult mortality to growth is substantial and significant, and that the feedback effect - poverty to high adult mortality to low growth - is part of the explanation of why large-scale poverty persists. Poverty leads to high mortality, and this in turn stifles the growth that would help countries escape poverty.

Does the adult mortality rate affect income and economic growth? Confirming our observation from Figure 1, a glance at the raw adult mortality data shows that, as expected, mortality varies with per capita income (Appendix 1). The safest country in the post-1960 period has been Sweden, where a fifteen-year-old's probability of dying before reaching sixty is only 13 percent. The worst has been Sierra Leone, where a fifteen-year-old's probability of dying before sixty is 57 percent. Yet per capita income is not a perfect predictor of mortality. Cyprus, Costa Rica, Cuba, and Turkey have adult mortality rates of 15 to 18 percent, better than France's 19 percent and the United States' 20 percent. Furthermore, the spread of health knowledge to less-developed countries has led to mortality rates converging more rapidly than income levels (Deaton, 2004, Becker et al., 2005). We find, nevertheless, that per capita income is significantly associated with the mortality rate, and that mortality is significantly associated with growth. When potential biases from endogeneity and measurement error are addressed with instrumental variables, our estimates of these effects become even stronger.

As a check on the robustness of the mortality-growth relationship, we also analyze data across states of India. Adult mortality (measured here as a twenty-year-old's probability of dying before reaching forty) varies from state to state: in West Bengal, it is 4 percent; in Assam, 7 percent. Across Indian states, we again find a significantly negative association between adult mortality and economic growth.

Our main argument is that people who expect to die young will fail to take actions, such as saving and getting educated, that generate long-term benefits at short-term costs. Figures 3 and 4 display strong negative relationships between adult mortality on the one hand, and rates of investment in physical and human capital on the other. Some corroboration of the influence of mortality is provided by the converse: people who expect to die young will take actions that involve short-term benefits at long-term costs. A high death rate from exogenous causes could lead to a high death rate from endogenous causes. Examples of such behaviors are unprotected sex and smoking. We document below that high adult mortality in 1960-1980 is associated with high rates of AIDS infection and death in 2002-2003. Similarly, we show that adult mortality is positively associated with a variety of measures of tobacco use. The association of high adult mortality rates with the decisions to smoke and to engage in activities leading to AIDS transmission arguably reflects the short time horizons held by adults living in societies where fewer citizens reach old age.

Previous empirical and theoretical work has often ignored the distinction between adult mortality, infant mortality, and life expectancy, taking them all to be proxies for a general concept of "health." In this research, we focus particularly on adult mortality rather than the more commonly used variables of infant mortality or life expectancy at birth. Although infant and adult mortality are strongly correlated, the channels by which each might influence economic growth are theoretically distinct, and so it is informative to separate them. The premature death of an adult means the total loss of any human capital investments and the inability of that adult to personally enjoy the fruits of other investments. The death of an infant, while tragic and costly in its own right, has less severe economic consequences.

Infant mortality, incidentally, varies more than adult mortality. Across countries, as noted, adult mortality ranges from 13 percent to 57 percent. Infant mortality, measured as the probability a child will die before the end of his or her first year, ranges from less than 1 percent (in Sweden) to 19 percent (in Sierra Leone). Nevertheless, we find that adult mortality has empirically distinguishable effects even controlling for infant mortality. Indeed, it is because much of the variation in total

mortality and in life expectancy is driven by infant mortality that we need to be careful to use the appropriate mortality measure. If the phenomenon in question relates specifically to adult mortality, then investigating it using other measures could yield misleading statistical results. Moreover, our understanding of the different economic consequences of mortality at different ages may influence how policymakers choose to allocate scarce health resources.

The estimated effects of high adult mortality on investment and growth, it turns out, are large enough to account for Africa's stagnation. Of the 40 countries with the highest adult mortality rates in our sample, all are in Africa except three (Afghanistan, Laos, and Cambodia). In our sample of 98 countries, Sub-Saharan African countries grew 1.65 percentage points more slowly than the rest of the world, from 1960 to 2000, meaning that over the forty-year period covered by our data, the gap in per capita incomes between Africa and the rest of the world doubled. In regressions controlling for the usual determinants of growth, there is typically an unexplained residual, the Africa dummy, roughly equal to 1 percentage point of annual growth (Collier and Gunning, 1999). Yet once we add adult mortality to the growth regression, the Africa dummy becomes statistically indistinguishable from zero.[1] Thus, not only is adult mortality a statistically significant predictor of growth, it is also economically large.

This paper also considers *how* mortality affects growth, examining several possible causal channels implied by theory. Estimating a system of equations, we find that school enrollment, investment rates, and fertility rates capture most if not all of the total effect of adult mortality on economic growth, with fertility and physical capital playing the major roles. While mortality appears to affect school enrollment rates, the further link between education and growth is tenuous in our empirical results.

Our findings stand in contrast to those of Young (2005), who uses a calibrated simulation for South Africa to forecast that survivors of the AIDS epidemic will be economically better off than they would have been without an epidemic. This comes about in Young's model because women become more cautious about having sex for fear of infection, and because as others die out of the workforce, female labor becomes more valuable. The consequent reduction in fertility leads to higher living standards for survivors. In contrast, we find empirically that higher adult mortality is associated with higher fertility, more risky behavior, and economic stagnation. While our analysis is retrospective, the forces identified in this paper suggest that the economic toll from AIDS may

[1] Note that the data are averaged over 1960 to 2000, so are not dominated by the AIDS epidemic.

greatly exceed the direct costs of health care and destroyed lives.[2]

By examining the mechanisms through which death rates affect growth, by considering various measures of mortality, and by acknowledging that the joint determination of income and mortality might drive a poverty trap, our paper attempts a comprehensive cross-national assessment of the effect of death on development.

In Section 2, we review conceptual arguments linking mortality to investment, human capital accumulation, fertility and ultimately economic growth. In Section 3, we describe and discuss the data on mortality used throughout this paper. In Section 4, we present simple cross-sectional regressions for the determination of growth, investment, human capital accumulation, fertility and several other variables, as a function of various mortality indicators. In Section 5 we try to identify the causal effect of adult mortality on these variables through the use of several instrumental-variables techniques. Section 6 concludes.

2 How Mortality Affects Growth

Our main hypothesis is that mortality affects growth by inducing short-sighted behavior. Among proximate determinants of growth, the accumulation of physical capital and of human capital, as well as the rate of fertility, may all be affected by the mortality rate.

2.1 Mortality and accumulation

Theory provides clear predictions on the effect of mortality on investment. The elementary logic is as follows: given an instantaneous utility function $u(c_t)$, a probability of survival of p, and a discount factor β, in a two-period model agents optimize $u(c_t) + p\beta u(c_{t+1})$. A reduction in the survival probability p, like a reduction in the discount factor β, brings lower savings and investment and thus lower growth. Since some of the gains of economic growth are in turn spent on improved sanitation and medical care, this can in theory be self-reinforcing, as we will argue below. Among various kinds of mortality, we would expect adult mortality rather than early childhood mortality to affect accumulation most, as decisions on physical and human capital accumulation are made in or close to adulthood on the basis of returns they will yield in adulthood.

[2] Kalemli-Ozcan (2005) finds that AIDS increased fertility and lowered school enrollment between 1985-2000 in many African countries.

While this framework applies most easily to physical capital investment, it can be applied quite readily to human capital accumulation as well: as the returns to human capital accrue over much of adult life, a high incidence of adult mortality will reduce incentives to obtain an education.[3] In fact, the theoretical link between mortality and human capital investment is arguably even stronger than that between mortality and physical capital investment: whereas parents with altruistic feelings towards their children will benefit indirectly from physical capital investments even if they are unable to enjoy their fruits personally, an early death destroys human capital investments before their full returns are realized.

The link between mortality and schooling is modeled by Kalemli-Ozcan et al. (2000). A reduction in mortality increases the individual's time horizon and thus increases the incentive to invest in human capital. Since lowered mortality affects growth in part via wages and interest rates, the effects should be smaller in small open economies where wages and interest are fixed by world markets. Calibrating their model using estimates of the return to schooling, Kalemli-Ozcan et al. find that a 1 percent reduction in mortality should lead to about a 1 percent increase in schooling.[4]

The empirical link between mortality and growth has previously been examined in several articles. Shastry and Weil (2003) find that adult mortality accounted for almost a third of the variation across countries in levels of per capita income not explained by factor accumulation. Turning to early modern Europe, Boucekkine et al. (2003) estimate that a steady decline in adult mortality (while child mortality stayed level) accounts for 70% of the growth acceleration from 1700-1820. A larger literature focuses on the relationship between health outcomes and economic growth. Bhargava et al. (2001) and Bloom, Canning and Sevilla (2004) provide strong evidence for a positive effect of health on economic growth. Bleakley (2003) finds that eliminating hookworm, a non-fatal condition afflicting children, boosted human capital accumulation in the American South. Sachs (2001) builds on findings like these to present a policy case for boosting investments in health, particularly malaria eradication, to promote economic development.

The issue of health and growth is related to, but distinct from, the issue of mortality and growth. The health of a population may affect growth because it reduces the time horizon of

[3] An early contribution on this topic is Ben Porath (1976), who argues that investments in education are a positive function of an individual's length of horizon.

[4] Zhang, Zhang, and Lee (2003) show theoretically that in highly developed societies, a growing population of retirees may choose to vote down public education funding, thus stifling growth. This mechanism is unlikely to be important in the period and countries we consider.

economic agents through increased mortality and therefore leads them to engage in short-sighted behavior. However, the debilitating effects of poorer health may also have a direct effect on the level and growth of productivity. This distinction is important to keep in mind when discussing the relationships between health, mortality and growth.

While each of the previous papers represents a step forward in our understanding of the mortality-growth linkage, each looks at only part of the picture. Much of the literature neglects the hypothesis that mortality affects growth, using mortality as an indicator of health, although as we just argued the effects of poor health are conceptually distinct from those of a short horizon. Furthermore, mortality affects growth through a variety of channels, but the relative magnitudes of the effects through each channel have not been systematically evaluated. In fact, most papers adopt a reduced form approach which fails to identify any channels at all. By examining these channels and other behavioral consequences of short time horizons we are able to partly disentangle the separate effects of health and mortality in a way that is impossible when attention is restricted to the raw mortality-growth relationship. While data limitations - the paucity of direct measures of health - prevent us from separately identifying the debilitating effects of poor health from those of a short horizon induced by high mortality, our empirical results overall are consistent with a major role for mortality.

The past studies do not generally distinguish between the effects of life expectancy at birth, infant mortality, and adult mortality. These various types of mortality bear a priori distinct relationships with economic variables. As we demonstrate below, adult mortality is a robust and economically significant predictor of economic growth, investment and fertility even when infant mortality is controlled for. Also, existing research often neglects the endogeneity of mortality. This endogeneity matters not only because it biases econometric estimates but also because the mortality-growth interaction is the possible source of a poverty trap, as we argue below.

2.2 Mortality and fertility

There is a strong cross-sectional relationship between (adult) mortality and the rate of fertility. As we document below, the relationship remains after controlling for various determinants of fertility, particularly income and human capital indicators, and after several attempts to control for the endogeneity of mortality. This is a consequence of the well-known demographic transition (Lee, 2003, Galor, 2004). In the demographic transition, mortality first falls as a result of the adoption

of better medical technologies, better hygiene, and better access to medical care (all of which are a function of per capita income). Subsequently, fertility declines (Bloom and Canning, 2003). Countries that have not started their demographic transitions feature high rates of mortality and fertility. Countries that have completed their demographic transitions feature low rates of both mortality and fertility. Thus there is a strong positive relationship between these two variables in the cross-section, with only relatively few countries in the transition itself (low mortality but high fertility), as displayed in Figure 5. Is any part of this correlation causal? The typical timing of the mortality and fertility declines suggests that, if anything, causality should run from mortality to fertility.

There is a lively theoretical debate on the link between mortality and fertility. Becker's (1960) quality-quantity theory stresses the role of income as a determinant of the fall in fertility. The basic argument is that an increase in female earnings raises the value of working relative to caring for children, shifting the terms of the trade-off between the quantity and the quality of children in favor of the latter. This theory has little role for mortality as a determinant of fertility. In Barro and Becker (1989), child mortality is introduced as a cost of having surviving children. A fall in child mortality is expected to lead to a lower marginal cost of offspring, raising parental demand for children. Thus, in this theory, child mortality and net fertility are *inversely* related.[5] Several recent papers have reinforced this theoretical finding, for instance Doepke (2004), Boldrin and Jones (2002), Fernandez-Villaverde (2001), in models that build on Barro and Becker's theory.[6]

A competing strand of models predicts that mortality and fertility are *positively* related, as we observe in the cross-national aggregate data. In a model in which parents gain both old age support and "companionship" utility from their adult children, Ehrlich and Lui (1991) find that an exogenous increase in the rate at which children survive to become productive adults can shift the economy from a low-growth high-fertility equilibrium to a high-growth, low-fertility equilibrium. In a model where parents only get utility for their integer number of surviving children, Sah (1991)

[5] In the demographic transition, net fertility also falls after the decline in mortality. The Barro-Becker model does imply that the gross rate of fertility (number of children born per woman) is positively related to mortality. We used a measure of net fertility instead of fertility in our empirical work and results obtained using either measure were extremely similar. For a variety of reasons linked to its definition, net fertility is difficult to capture and any measure is likely to be more noisy than total fertility rates. Thus we retained the latter in our regressions.

[6] Hazan and Zoabi (2004) go further in a model where changes in adult longevity affects neither fertility nor human capital investment.

shows that the number of births increases in the child mortality rate. The need for additional births in order to avoid an undesirably low number of children is known as the "hoarding" or precautionary motive, whereas births that occur after the loss of one child are referred to as replacement births (Ben-Porath, 1976).

A compelling story linking exogenous mortality declines to a fall in fertility is provided in Kalemli-Ozcan (2003). This model integrates a precautionary motive with education choices, showing that hoarding will not occur when the mortality rate is identical within each family, but will occur when uncertainty is taken into account. She states: "uncertainty about child survival gives rise to a precautionary demand for children. Thus, exogenous reductions in mortality lead to a decline in fertility and eventually in population growth". Where child mortality is high, parents have a large number of children to try to ensure some of them survive. Moreover, the children's lowered mortality increases the return from investing in their education, and the reduction in fertility frees up resources to make this investment: lower adult mortality tips the scales in the trade-off between the quantity and quality of children in favor of the latter. A reduction in child mortality will thus lead to lower fertility and higher human capital investment. The reduction in fertility can be expected to affect not only human-capital investment but also per capita income growth directly, by reducing population growth. Kalemli-Ozcan's results require that marginal utility be convex in surviving children, an assumption for which there is no direct evidence. While such assumptions are controversial (Galor, 2004), they are common in the context of consumption theory (see for example Carroll and Kimball, 1996).

Most of these models link *child* mortality to fertility, although it is not clear that their predictions are limited to the effects of child mortality as opposed to other concepts of mortality. The models generally make the reasonable simplifying assumption that children either die young or live out their lives. It is important to be careful when bringing such models to the data, however. If parents derive utility not simply from the number and quality of their children who reach adulthood, but also from their children's lifetime utilities, reproductive success, or ability to support the parents in old age, adult mortality rates will also affect parental fertility choices.

In most cases, it is sufficient when evaluating these models to keep in mind that what is referred to in the model as child mortality should probably include adult mortality as well. In other models, such as Doepke (2004), the theoretical results are driven by the assumption that deceased children can mostly be replaced. This is plausible only if young children are the only ones lost. A look at the

mortality data makes it clear that even without taking AIDS into account, adult mortality cannot be neglected: for example, in Ecuador in 2000, about 4% of children born died before the age of 5, but 7% of 15-year-olds did not survive to age 40. Fernandez-Villaverde (2001), Doepke (2004) and Galor (2004) make much of the fact that in England, the major drop in infant mortality occurred after the drop in fertility, arguing that this single departure for the general pattern invalidates any causal link between mortality and fertility. This argument neglects adult mortality: in Europe from the 16th to the mid-19th century "most gains in life expectancy are concentrated among adults rather than children" (Boldrin and Jones, 2002). Taking that fact into consideration, the English case looks less anomalous.

Soares (2005) presents one of the few models in which fertility is affected not only by child mortality but also by adult mortality. Altruistic parental utility is determined not just by the number and quality of children, but also by the lifespans over which adult children will enjoy their consumption. In this model, longevity increases can transform a Malthusian trap in which increased fertility destroys any welfare gains from technological development or capital accumulation can be transformed into an equilibrium with long-run growth.

Both theoretically and empirically, the relationship between mortality rates at different ages and fertility remains poorly understood. In our empirical work, we are careful to distinguish between mortality rates at various ages. The strong results we obtain linking adult mortality and fertility suggest that there would be a high return to further theoretical investigations of this link.

Our main interest is in the link between mortality and growth. However, as the literature suggests and as we will confirm empirically, a key channel linking mortality and growth is fertility. One of the most straightforward consequences of most growth models is that high population growth should reduce income levels. In the canonical Solow model a higher rate of population growth reduces the capital-labor ratio in the steady-state and thus the steady-state level of income. Thus, if mortality has a positive causal impact on fertility, fertility might be an important channel whereby mortality affects growth. Of course, population growth is decreasing in mortality by definition, so the relationship depends on how tightly linked mortality and fertility changes are in time, and on the rate at which changes in the death rate translate into changes in the birth rate. In addition to the raw population effects of fertility, parents with more children will have fewer resources to devote to each child, the quality-quantity trade-off first introduced by Becker (1960).

2.3 Mortality and poverty traps

Can the relationship between mortality and growth be the source of a poverty trap? In theory, it can. A family of overlapping-generations models has established theoretically that a reduction in mortality should be good for economic growth. Conversely, higher incomes may lead to lower mortality. If the magnitudes of both of these effects is sufficiently large, poverty traps can emerge.[7] Models constructed by Ehrlich and Lui (1991), Meltzer (1992), Kalemli-Ozcan (2002), Chakraborty (2004, 2005), and Soares (2005) explore this relationship in different ways, but come to some common conclusions: for certain parameter values, there may be multiple steady-states, creating the possibility that a country gets trapped in a low-level equilibrium. Even if the steady-state is unique, higher mortality countries invest less and grow more slowly. Putting mortality into a growth model has the effect of slowing the rate at which poorer countries' capital-labor ratios and per capita incomes converge to those of richer countries, possibly preventing convergence altogether.

Other models focus on the demographic transition, including fertility as a choice variable along with human capital investment.[8] A mortality drop causes a switch from a Malthusian equilibrium in which population keeps up with production shifts to a pro-growth equilibrium with smaller families and more human capital investment. These models are consistent with the basic facts of the demographic transition around the world (Lee, 2003). Furthermore, as argued in Dasgupta (2004), poor health might generate a poverty trap through the mechanism of poor health making workers unproductive, and the resulting low incomes reinforcing poor health.[9]

In a recent survey, Azariadis and Stachurski (2004) note that while it is clear from the data that poverty and wealth tend to persist, "poverty trap models tend to be lacking in testable quantitative implications". Any poverty trap model will (by definition) predict a bimodal income distribution, but it requires more effort to prove that poverty traps rather than differences in initial conditions

[7] Various other mechanisms leading to poverty traps have been proposed (see the survey by Azariadis, 2001 and Azariadis and Stachurski, 2004).

[8] Including Ehrlich and Lui (1991), Meltzer (1992), Kalemli-Ozcan (2002), Soares (2005).

[9] See in particular Dasgupta and Ray (1986) for an early example of poverty traps based on "metabolic pathways". These theories "are based on physiological links connecting nutritional status and work capacity among adults, and those connecting nutritional status and physical and mental development among children" (Dasgupta, 2004). This is essentially an efficiency wage argument centered on nutrition. Some people will be locked in unemployment because it is better for employers to hire workers at a wage that ensures they are well-fed and productive, rather than lowering the wage so that everyone could get a job at their (now lower) marginal productivity

are to blame. They find two empirical strategies promising. The first assesses whether, controlling for exogenous variables, countries conditionally converge to a single regime or to multiple regimes. Bloom, Canning, and Sevilla (2003a), using this approach, find that a likelihood ratio test rejects the single-regime hypothesis where only geographical variables are considered to be exogenous. However, while this supports the hypothesis that there is some kind of poverty trap, it provides no evidence about the channels or mechanisms through which the trap works. The second approach is that of Graham and Temple (2004), who run a calibrated numerical simulation of a specific poverty trap model and find that it explains 40 to 50 percent of the observed variation in incomes.

Whether a particular mechanism, in our case mortality, is a plausible *empirical* candidate to generate a poverty trap depends on the magnitude of the relationships linking income and mortality. We offer evidence that the effect of mortality on growth is economically large, suggesting that mortality is a plausible candidate as a source of poverty traps.

3 Sources and Definitions of Mortality Data

3.1 Mortality and fertility measures

3.1.1 Mortality

Mortality can be measured in various ways. The most straightforward measure is the crude death rate. This simply equals the number of deaths in a year divided by total population. However, this variable is greatly influenced by the age structure of the population. Countries that have experienced declining birth rates (such as the most developed economies) will have relatively top-heavy age distributions. Since older people die at higher rates than the young, this will increase the crude death rate. Similarly, holding the age-specific death rates constant, an economy with a population bulge of young adults will have lower death rates because fewer of its members are in the high-risk zones of childhood and old age. This population structure will occur when child mortality drops without a corresponding drop in fertility, as occurs at the beginning of the demographic transition. Thus, poor countries may look healthier by this measure than they would in a fair assessment. For example, in our dataset, Sweden has a crude death rate of 10.62 per thousand, while the Bahamas has a crude death rate of 6.32 per thousand.

Life expectancy at birth is the most commonly used summary measure of mortality. While its name appears self-explanatory, the qualifier "at birth" is important. Infant mortality, defined as the

fraction of children who die before their first year, is a major source of variation in life expectancy at birth. For instance, among American males in 1999, there were as many deaths before age one as there were between the ages of one and nineteen combined (Bell and Miller, 2002). Note also that life expectancy is not an expectation as commonly understood by statisticians and economists. It is instead the expected lifetime of a child born in that period if all mortality rates remain constant through the remainder of the child's life.

Infant mortality is conventionally defined as the fraction of children who die before their first birthday. Many of the major initial advances in health care worldwide have had their greatest effect through infant and child mortality, as basic sanitary practices were introduced, thus cheaply and drastically lowering deaths due to infectious disease at these vulnerable ages (Bloom et al., 2003b, p.26).

In this paper, we focus our attention mostly on the adult mortality rate.[10] This is the probability that a fifteen-year old will survive until age sixty, given current age-specific mortality rates.[11] Where a indicates age and m_a is the probability of dying at that age, the adult mortality rate is calculated as:

$$AMR = 1 - \prod_{a=15}^{59} (1 - m_a)$$

As an illustration, assume that the probability of an adult dying in a given year is a constant 1%. The probability of surviving that year is then 99%. The probability of surviving 45 such years in a row is $(0.99)^{45} = 63.6\%$, implying an adult mortality rate of 36.4%. In reality, the probability of surviving each year generally declines steadily from age fifteen on, making the fuller calculation above necessary.

While the adult mortality rate between ages 15 and 60 is the most widely available, it is sometimes more appropriate to consider mortality over younger age ranges. As we show below, in the Indian cross-state dataset, adult mortality computed over the 20-40 age range is a better predictor of cross-state growth. Those years are the beginning of productive adult life, coming after most of the educational and other investments have been made in raising a child, but before the economic returns to the family unit are realized. Thus deaths in this age-range can cause

[10] Fertility may affect female adult mortality directly through a greater incidence of deaths in childbirth. As a consequence, we focus on *male* adult mortality throughout this paper. The two series are very highly correlated (in our sample, the correlations between male and female adult mortality averaged over 1960-2000 is 97.4%).

[11] Demographers refer to this mortality rate as 45q15, the probability of surviving 45 more years from age 15.

the maximum economic loss. This may be particularly true in societies where physical labor is important, such as India, since the capacity for physical labor decreases after this age.

3.1.2 Fertility

Fertility measures suffer from some of the same concerns as mortality measures. The crude birth rate is simply the number of births per person per year. Along with the crude death rate it determines (by definition) the population growth rate. However, like the crude death rate, it is dependent on the age and gender structure of the population: populations with more young women will have higher birth rates, all else equal. The total fertility rate, which we use, is thus the preferred measure. The total fertility rate for a given year is the number of children that a typical female would have over the course of her lifetime, assuming she survived through menopause and at each age had children at the same rate as women of that age did during the year in question. Thus, like life expectancy and adult mortality, it is a snapshot of behaviors of all the age groups in a population at one time, not a forecast.

3.2 Data sources and limitations

The cross-country data for this study come from the World Bank's World Development Indicators (WDI).[12] This data are assembled by the World Bank's demographers based on based on life tables from either the World Health Organization or the UN Population Division. Adult mortality rates have been collected for 1960, 1970, 1980, 1990, 1995, and 2000 for 163 countries, with an additional 25 joining the sample from 1990.[13]

The most reliable data come from countries with a complete vital registration system, where every birth and death is recorded, generally with the age and the cause of death. Collecting such data requires both that the state bureaucratic capacity be fairly well-developed and that the state have the economic resources to allocate to the task. Many developing countries lack either the motivation or the capabilities to gather these data reliably. Of the 155 economies included in the 2004 edition of the World Bank's World Development Indicators, fewer than half were assessed as having complete vital registrations for that year. Historical data are of course even more limited.[14]

[12] The sources and descriptions of all the variables used in this study appear in Appendix 2.

[13] Annual data are only provided consistently for six countries, all of which are highly developed.

[14] We also constructed our own mortality dataset based on the World Health Organization's Mortality Database.

Where vital registration data are unavailable or incomplete, demographers use a variety of techniques to estimate mortality. One common approach is to interview samples of the population about the number, ages, and deaths of their children, their siblings, and their parents, allowing projection to the larger population. In general, data on fertility, infant mortality and child mortality (deaths prior to age 5) are considered to be reliable, because parents are able to provide accurate birth histories and account for any deaths of their children. The quality of data on adult mortality gathered by this method is lower, as adults can move away from and lose touch with family members. Comparisons of the sizes of age cohorts between censuses provide another way to estimate mortality, although this is highly sensitive to migration and changes in the completeness of census coverage (Hill, 2003).

These data are then compared against tables relating mortality rates across different age groups. These model life tables were originally constructed based on the relative mortality rates of countries with high-quality vital registration systems. Different tables are available to represent different regions of the world. For instance, the widely-used Coale-Demeny "North" tables were based on Scandinavian countries, where infant mortality tended to be lower, child mortality higher, and old age mortality lower than elsewhere. A demographer then uses the model table that most closely fits the available data to complete the mortality estimates by age for that region (Murray et al., 2000).

For some countries, chiefly in Africa, data from sampling methods are limited and the data on adult mortality are sometimes obtained by imputation, based on other mortality data such as infant mortality. Thus, the quality of the data for Sub-Saharan Africa is the least satisfactory. Recent data incorporate corrections for the impact of the AIDS pandemic on adult mortality in African countries, and these corrections can also be questioned since age-specific AIDS mortality is rarely observed directly in these countries. To the extent that adult mortality is estimated mainly from

This database includes absolute numbers of deaths and population by age groups as provided by participating countries. A typical entry would be the number of reported deaths of men aged 20-24 in the United States for 1975. From this data we calculated age-specific mortality rates for reach age grouping as the number of deaths divided by population and then calculated adult mortality from this. Adult mortality rates for 1960, calculated in this fashion, have a 67% correlation with those provided in the WDI. The correlation between the two measures rises to 95% for 1990. These rates are not perfectly correlated because the life tables used for the WDI may involve some subjective judgment and smoothing by the demographer. The WHO database only includes countries that choose to submit data to the WHO, which excludes most of Africa. We conducted our empirical analysis using this dataset and derived qualitatively similar results to those obtained using the WDI dataset.

infant mortality, in possibly nonlinear ways, without any additional input from other data sources, adult mortality estimates for poor and/or African countries might largely be a function of infant mortality.[15] This could affect our estimates in two ways. First, it will lead to higher measurement error, since true mortality will be estimated with noise. Second, it will make the separate identification of the adult mortality and infant mortality effects more difficult in specifications that control for both, since by construction these variables will be (possibly nonlinear) functions of each other for a subsample of the data: identification will be obtained largely off the variation in the richer countries.

These largely inescapable drawbacks of the available mortality data can be addressed in several ways. First, we show below that our results hold up when we exclude from our sample Sub-Saharan African countries, for which the data problems are most acute. Second, the African adult mortality data are still informative, even when they are largely based on projections from survey-based estimates of infant and child mortality. In a recent paper given to a UN workshop, Kenneth Hill, the Director of the Johns Hopkins Population Center concluded that the UN's "model life tables generally fit the age patterns of mortality reasonably well, though they tend to underestimate young adult male mortality in most populations... and cannot represent the age patterns associated with the HIV/AIDS epidemic".[16] The underestimation of young adult male mortality may partly explain why fairly small increases in mortality in our data can have substantial effects on long-run growth.

As noted by the World Bank's specialists, the "adequacy of mortality estimates also depends on what they are being used for... to document short-term fluctuations, and even more so to link them to a changing socioeconomic environment, requires far greater detail than can often be obtained" (Bos et al., 1992). We believe that this makes higher frequency econometric techniques (such as panel data methods) an inappropriate use of these data, so we focus long-run averages.

[15] Surprisingly, for the subsample of Sub-Saharan African countries in our dataset, the raw correlation between adult mortality and infant mortality averaged over 1960-2000 is 0.53, which is actually lower than the full sample correlation of 0.87. The correlation for the 1960 African data is only 0.38 (0.80 in the full sample). Similar correlations of infant mortality with life expectancy are much higher (on the order of -0.85 for Sub-Saharan Africa and -0.95 for the whole sample, for both 1960 and 1960-2000 averages). We are grateful to Angus Deaton for pointing out this fact.

[16] This last point is not crucial for the purposes of our paper. We are primarily concerned with the effects of young adult mortality on growth in the period prior to the 1990s, and HIV-related increases in mortality rates are primarily a phenomenon of the 1990s.

Such averages also reduce the incidence of measurement error.

In addition to the cross-country sample, we also collected cross-state data from India. While India is not considered to have comprehensive vital statistics coverage by developed-country standards, since 1970 it has had in place a well-regarded system called the Sample Registration System. In this system, vital statistics are gathered and double-checked each year in a random sample of several thousand villages and urban blocks around the country. From these data overall birth and death rates can be estimated. Mortality rates for five-year age ranges were compiled based on these data for each state and union territory by India's Registrar General for the years 1971-1997. We then used these death rates to calculate adult mortality directly.

The following sections will show that our findings are robust to the use of a variety of subsamples and empirical approaches, giving us confidence that these results are not simply driven by flaws in the collection of the data.

4 Mortality and economic outcomes: empirical linkages

4.1 Descriptive statistics

Tables 1a and 2a present summary statistics and correlations for the main variables of interest in the cross-country dataset. Table 1a provides the means and standard deviations of these variables, which can be used to assess the magnitude of the effects estimated below. The demographic variables, averaged over the period 1960-2000, display substantial variation. Not surprisingly, Table 2a shows that the correlations among the various measures of mortality are high: the correlations between infant mortality, adult mortality and the crude death rate range from 0.87 to 0.89, suggesting that multicollinearity might be an issue in the regressions. Perhaps more surprisingly, the fertility rate is highly correlated with our various measures of mortality. Below, we investigate the empirical relationship between fertility and mortality in detail.

The corresponding summary statistics for the India dataset are presented in Tables 1b and 2b. Similar observations hold in this alternative dataset: correlations are quite high among our various measure of mortality, but lower than in the cross-country dataset. The lower extent of multicollinearity suggests that the inclusion of these measures jointly in cross-state regressions may lead to more consistent results across specifications (as we indeed find below). For the India dataset our measure of mortality refers to the probability of dying by age 40 conditional on reach age 20.

This is actually a somewhat better measure of prime-age mortality, as it avoids measuring the age-related maladies that become significant causes of death by age 50.[17] Obviously, the average probability of dying is much lower for the shorter 20-40 age range than it is for the 15-60 range.

4.2 OLS growth estimates

4.2.1 Economic growth in a cross-section of countries

The first step in our empirical analysis is to examine the partial correlations between growth of income per capita and the various mortality measures. We start with the simplest possible approach: OLS regressions of economic growth on log initial income per capita and an increasingly large set of controls, with each variable averaged over the 1960-2000 time period in order to reduce bias due to measurement error.[18] We explore several specifications, adding control variables progressively to assess the robustness of the mortality variables, as well as their relative importance. Table 3 displays the least squares results.

The first lesson from this exercise is that adult mortality rate is a very significant predictor of growth when entered alone with the log of initial per capita income (column 1). The coefficient is negative and significant at the 1% level. The adjusted R-squared from a simple regression of income growth on log initial income is 0.04. The adjusted R-squared rises to 0.46 simply by adding the adult mortality rate, suggesting that a large portion of the cross-country variation in economic growth might be attributable to this variable. Of course, it is critical to control for other measures of mortality which may be highly correlated with adult mortality and whose effects may be picked up by the latter. Importantly, column 2 shows that despite the high collinearity between the measures of mortality, the impact of adult mortality holds up when we control for other mortality rates, i.e. infant mortality and the crude death rate.[19] Crude mortality is only weakly related to growth,

[17] Using data from the World Health Organization, we constructed a similar variable for the cross-country sample, with results similar to those discussed below. However, we chose to rely primarily on the World Bank data described earlier as it is available for a much larger set of countries. This dataset does not include the 20-40 adult mortality rate.

[18] See Hauk and Wacziarg (2004), for a discussion of the virtues of simple OLS estimators, in terms of limiting the incidence of classical measurement error bias in the cross-country context.

[19] The coefficient on adult mortality is also robust to the inclusion of the child mortality rate in the regression. These results are available upon request. Measures of child and infant mortality bear a correlation coefficient of 0.99 in the cross-section of countries, so using one or the other makes very little difference.

and its sign changes depending on the controls used. Despite high collinearity between adult and infant mortality, we are able to identify the effects of each variable separately: both bear a negative relationship with growth.

One variable that does sharply reduce the significance of adult mortality is the fertility rate. Out of a large set of the control variables that we included in the regression, fertility was the only one that consistently made mortality insignificant. Indeed, column 3 shows that the coefficient on adult mortality is sensitive to the inclusion of the fertility rate in the regression: it remains negative but its magnitude and statistical significance fall. This result, which remains when we include additional controls in the regression (column 5), suggests that the interplay between fertility and adult mortality may be an important channel whereby adult mortality could indirectly affect economic growth. Thus, we will reexamine the empirical relationship between adult mortality and fertility when we explore these channels in Section 5.[20] In interpreting these results, one should remember that all our demographic variables are highly collinear. The correlation between fertility and adult mortality averaged over the 1960-2000, for instance, is 0.80. If these variables are measured with some error, as they surely are, these high correlations make it difficult to tell which one dominates statistically in any given specification. As discussed above, the measurement error for adult mortality is likely to be higher than that for fertility in developing countries, probably increasing the estimated coefficient of fertility at the expense of adult mortality.

Next, we include a series of other control variables besides the log of initial per capita income. We follow the baseline growth specifications in Barro and Sala-i-Martin (1996) and Alesina, Spolaore and Wacziarg (2000), namely we control for government consumption as a share of GDP, the rate of investment, the secondary school gross enrollment ratio, openness (measured by the trade to GDP ratio), the log of population and its interaction with openness. The estimates of the coefficients on

[20] We also tried to control for life expectancy (results are available upon request). This variable, averaged over 1960-2000, bears a 96% correlation with the adult mortality variable that we use. When including life expectancy at birth instead of adult mortality, this variable came out positive and highly significant (except when fertility was controlled for). When both variables were included, both lost significance due to the very high level of multicollinearity. This finding is interesting since results in Barro and Sala-i-Martin (1996) show life expectancy to be one of the most consistently significant correlates of growth across countries. Adult mortality and life expectancy capture roughly similar concepts, i.e. the first measures the probability of surviving to reap the returns to various forms of investment and the second captures the number of years over which this return can be accumulated and enjoyed. However, life expectancy is also strongly determined by infant and early childhood mortality, making adult mortality the more appropriate variable for testing our theory.

these control variables all have the expected signs. The coefficient on adult mortality is reduced slightly in magnitude, but remains significant at the 1% level (column 4). When we remove the mortality variables from this regression, its adjusted R-squared falls from 0.68 to 0.58, suggesting that measures of mortality (chief among them the adult mortality rate) can explain roughly an additional 10% of cross-country variation in growth when other controls are included. Again, when we add fertility to the equation (in column 5), the estimated coefficient of adult mortality drops below conventional significance levels. The high collinearity between fertility and adult mortality, which generates sensitivity in the coefficient on adult mortality, is again consistent with the view that fertility might be a channel through which mortality operates. We will explore this hypothesis in a more structured model in Section 5, where we will attempt to quantify the importance of different channels.

The magnitude of the partial correlation between adult mortality and growth is substantial. Using estimates in the baseline regression of column 4, a one standard deviation in adult mortality (equal to 0.136) is associated with a 0.72 percentage point difference in growth. Moving from the 75^{th} percentile of adult mortality (Cambodia) to the 25^{th} percentile (the USA) brings an extra 1.39 percentage points of growth holding all the included determinants of growth constant.

4.2.2 Extensions and robustness checks

In Table 4, we consider extensions to the basic OLS specification. First, we consider whether high adult mortality might account for Africa's growth tragedy. As we pointed out above, most of the world's high-mortality countries are in Sub-Saharan Africa. In column 1, we run the baseline regression without the mortality measures, but with standard growth controls, plus a dummy variable for Sub-Saharan Africa. The dummy variable bears an estimated coefficient of about minus one percentage point, Africa's "missing growth".[21] Running the same regression with adult mortality included (column 2), we find that the Africa effect becomes positive and statistically

[21] See Collier and Gunning (1999) for more on Africa's growth tragedy. See also Easterly and Levine (1997) who find a significant negative effect of the Sub-Saharan Africa dummy, even after controlling for a set of growth determinants (somewhat different from ours) and a measure of ethnic fractionalization. The latter reduces but does not eliminate the Sub-Saharan Africa dummy, while in our regressions adult mortality eliminates the effect entirely. The coefficient on adult mortality is insensitive to the inclusion of a measure of ethnic fractionalization in our specification.

indistinguishable from zero.[22] Thus, in a statistical sense, adult mortality can account for all of the growth shortfall experienced in Sub-Saharan Africa between 1960 and 2000.

A major source of collinearity between adult mortality and fertility is the demographic transition. Countries that have not started their demographic transitions display both high mortality and high fertility. Countries that have completed their demographic transitions display both low mortality and low fertility. These countries drive the high correlation between these variables in our dataset. Figure 5 shows this graphically. For the intermediate countries, the correlation between adult mortality and fertility is much lower. In a subsample where the collinearity between fertility and adult mortality is lower because countries are caught during their demographic transitions (rather than mostly before or after it takes place), adult mortality remains a strong predictor of economic growth even after controlling for the fertility rate. Column 3 of Table 4 isolates 40 mid-transition countries in which adult mortality falls between 0.2 and 0.5. In this regression, the effect of mortality is negative, large in magnitude and statistically significant, even when we control for fertility.

The confirmation of our result in this subsample, which excludes both most African countries and the highly developed nations, also suggests that these results are not driven by systematic mismeasurement in Africa or solely by the stark distinction between the richest and poorest nations. As a further check of this, we reran our baseline regressions excluding Sub-Saharan Africa but including the rest of the developing world (most of which has higher-quality data). The coefficient estimates on the mortality variables were very close to those derived without the Africa data, although their significance levels were lower, due to the much smaller sample size.[23]

Next, we examine the role of war and war casualties (columns 4 and 5 of Table 4). Countries that have experienced the turmoil of war may experience low growth because of damaged political, social, and economic institutions. At the same time, their mortality rates will be higher, leading to possibly wrong inferences that mortality accounts for low growth. We consider four variables to capture the effects of wars, keeping intra-state (civil) wars and inter-state wars separate because their disruptive effects are not necessarily comparable.[24] One measure of the impact of war is total

[22] Note also that Sub-Saharan Africa is not driving the estimated coefficient of adult mortality. Similar regressions excluding African countries yield the same result, as we discuss below.

[23] These regressions are available upon request.

[24] Consider how the deaths of a thousand US soldiers in a foreign theater would differ in economic impact from the

battle deaths from 1960-1997, divided by average population. Battle deaths from actual combat are a fairly minor source of mortality over the entire period, but we take them to be a reasonable proxy for the total disruption caused by wars. Comparing the regressions in Table 4 with column 4 of Table 3, it appears that including war deaths slightly reduces the coefficient and significance of adult mortality, but this is entirely due to sample selection (we lose 5 somewhat influential observations by using the battle deaths data). As the battle death numbers are imperfect, we also use war duration in months, a proxy that can be more reliably measured. Including all four war variables further reduces the coefficient on adult mortality.

Surprisingly, casualties from and time spent in inter-state wars appear to bear a somewhat positive relationship with growth, while intrastate wars bear a somewhat negative relationship with growth. Of the four variables, only interstate battle deaths and months of intrastate war are statistically significant at the 10% level, but an F-test for the joint significance of the four variables gives a p-value of 0.049. A variety of other war-related controls that we tried did not have any significant effect, nor did they substantially reduce the estimated coefficient of adult mortality. While these results are interesting, they are not very informative with respect to our hypothesis. It would be surprising if war were not both somewhat collinear with adult mortality and also bad for growth. In addition, theory does not require people to fear premature death for any particular reason: war, starvation, or disease would all have the same effect of shortening time horizons.

Next, to assess the possibility that adult mortality can capture the effect of institutional quality, we control for two measures of institutions (columns 6 and 7). Expropriation risk, a survey-based measurement of institutional quality, was previously used in Acemoglu et al. (2001). Acemoglu and Johnson (2005) argue that an index of constraints on the executive is a better measure of institutions because it is more objective and thus less likely to be conflated with wealth. In column 6 of Table 4, using our baseline specification, controlling for expropriation risk in 1990 has almost no effect on the coefficient or significance level of adult mortality, although expropriation risk does come up as significant in its own right. In column 7, including the second measure of institutional quality (constraints on the executive, average of 1970 and 1990 values), actually *raises* the estimated magnitude and coefficient of adult mortality, while the estimated coefficient on the institutions variable is statistically indistinguishable from zero.[25] These results suggest that if institutions

same thousand deaths occurring in an intra-US secession war.

[25] Measures of institutional quality, such as indices of democracy, often come out insignificant in cross-country

matter for growth rates, their effects are in addition to the effects of mortality, rather than being the sole explanatory variable. It may well be that good health-care institutions tend to go along with good political and economic institutions, but each has separate effects.

Finally, we carried out an outlier analysis. Only one outlying observation is notably influential: South Africa. Excluding it from the sample used in column 5 of Table 3 *increases* the coefficient estimate of adult mortality from −2.94 to −4.64, and increases the t-statistic from 1.47 to 2.58. None of the other coefficients change substantially from this exclusion. Excluding one-by-one the other thirteen observations for which the growth rate differs from the predicted value by more than 1 percentage point (1.38 standard deviations) changes the coefficient on adult mortality by 0.57 at most. The model in Table 3, column 5 predicts a growth rate of 0.11 for South Africa, as opposed to its actual growth of 1.05.[26]

To summarize, adult mortality is a consistently significant predictor of economic growth across countries. The magnitude of the effect is large and the variable accounts for a substantial portion of cross-country variation in growth, as well as for Africa's growth tragedy. However, the effect is reduced and its statistical significance eliminated if we include the fertility rate in the regression. Of course, OLS regressions simply reflect partial correlations, not causal effects, an issue we address in Section 5.

4.2.3 Mortality and growth across Indian States

As an additional check on the robustness of our findings, we attempt to approximate the same growth specifications using a dataset we have gathered for Indian states. Our demographic variables are directly comparable to those used in the cross-country dataset in terms of definitions and units. Many control variables, in contrast, differ due to data availability issues. Given the small number of Indian states with available data, we ran both cross-sectional regressions (using the between estimator) and panel random-effects regressions exploiting the availability of data at the decade level.[27] The latter are likely to deliver more reliable results given that all regressions involving

growth specifications. This stands in contrast with their estimated effect on income levels. See Acemoglu, Johnson and Robinson (2001) for compelling evidence on the latter.

[26] South Africa's status as an outlier should also be kept in mind when evaluating the relevance of Young's (2005) study of South Africa to the AIDS crisis elsewhere.

[27] This requires giving up the reduction in measurement error bias resulting from time averaging, but is made necessary by the paucity of observations.

our mortality measures cover at most 19 states (we are able to obtain up to 47 observations when exploiting the panel dimension). Our regressions cover the period 1970-2000, with one observation per decade.

Results from the Indian dataset, displayed in Table 5, closely resemble those obtained using the cross-sectional dataset.[28] One noteworthy aspect of growth across Indian states is divergence in per capita income, as shown by the significantly positive coefficient on the log of initial per capita income in column 1 of Table 5.[29] While we cannot account for divergence in per capita income by conditioning on adult mortality alone, this variable is by far the most robust partial correlate of cross-state growth in India. Given the data limitations and the small number of observations, this is a strong result. The coefficient is robust to the inclusion of a broad range of controls, including other death rates (infant mortality, child mortality and the crude death rate), as well as other potential determinants of cross-state growth such as the literacy rate, the urbanization rate, religious fractionalization, and federal development assistance. In contrast to our findings using the cross-country data, the effect of adult mortality is also robust to the inclusion of the fertility rate in the cross-state growth specification (columns 3 and 7).

In terms of magnitudes, focusing on the random effects specification in column 4 of Table 5, which includes a wide range of controls, a one standard deviation difference in adult mortality (equal to 0.013) is associated with a 1.04 percentage point difference in growth of per capita net state domestic product (this is to be compared to the standard deviation in 1970-2000 economic growth across states in India, 1.78 percentage points). This effect is slightly larger but roughly in line with that obtained in the cross-country regressions. Adult mortality accounts for 1.67 percentage points of the growth difference between a state at the 75^{th} percentile of adult mortality, such as Kerala (with a male adult mortality rate at ages 20-40 of 4.5% in 1991), and a state at the 25^{th} percentile, such as Madhya Pradesh (with a male adult mortality rate of 6.7% in 1991). This is virtually all of the growth difference between Kerala and Madhya Pradesh in the 1990s. Again, we see that the economic magnitude of growth differences associated with difference in adult mortality is very large.

[28] We ran many more specifications for cross-state growth in India than are shown in Table 5. In all these specifications, adult mortality remained significant. The results are available upon request.

[29] This has been observed previously by Ghosh, Marjit and Neogi (1998), among others

4.3 Behavioral consequences of adult mortality

Adult mortality is likely to impact growth by reducing incentives to engage in behavior that yield long-term benefits at short-term costs. Examples of such behavior are investment in physical capital (and more generally entrepreneurship) and investment in human capital. Adult mortality also raises incentives to engage in behavior with short-term benefits but long-term costs, such as smoking or engaging in activities associated with contracting the virus that causes AIDS. Finally, as discussed earlier, higher fertility bears a robust empirical link with higher mortality.

In this section, we investigate these relationships empirically. We examine how adult mortality relates to investment, human capital accumulation, fertility, the death rate from AIDS and several measures of tobacco use and consumption. Since the required data are not available across Indian states, we look only at the cross-country dataset. We have two goals. First, we are primarily concerned with partial correlations, to assess whether adult mortality has any potential to explain vast cross-sectional growth differences. The results here paint a picture consistent with the theory discussed in Section 2, without necessarily implying causality. Second, the estimates of the channels through which mortality affects growth provide a reference point for the systems estimates presented in Section 5.

4.3.1 Channel variables

Adult Mortality and Physical Capital Investment. Columns 1 and 2 of Table 6 show that high adult mortality is associated with a reduction in the investment rate. Column 2 controls for the crude death rate, the infant mortality rate, and other controls based on the specification for the investment rate equation in Barro and Sala-i-Martin (1996). In that specification, a one standard deviation increase in the male adult mortality rate is associated with a 2.81 percentage point reduction in the investment rate. This is a sizable effect, considering that the mean of the investment rate in our sample is 15.12%. As theory would predict, infant mortality does not bear a significant relationship with the investment rate.

Adult Mortality and Human Capital Accumulation. Columns 3 and 4 of Table 6 display the correlates of human-capital accumulation. Following Mankiw, Romer and Weil (1992), we use the enrollment rate in secondary education as a measure of human capital investment.[30] The

[30] Our measure of enrollment differs slightly from Mankiw, Romer and Weil's (1992), who used the gross enrollment ratio in secondary education multiplied by the fraction of the working age population aged 15 to 19. We use the gross

human-capital augmented Solow model implies that this flow rate is theoretically more appropriate than a stock measure of human capital. However, the secondary enrollment rate is highly correlated with commonly used stock measures, themselves constructed from enrollment data (see Barro and Lee, 2000): the correlation between the secondary school enrollment rate and the number of years of primary, secondary and higher schooling in the adult population is 90.5%, and results obtained using these alternative measures are close.[31]

Adult mortality is negatively associated with human capital accumulation, and this effect is statistically significant. It remains significant when infant mortality and the crude death rate are added to the equation, and when other controls are included. Again, the magnitude of the effect is sizable: using the estimates in column 4, which includes standard controls, a one standard deviation increase in adult mortality is associated with a 9 percentage point decrease in the enrollment rate (this is about a third of the average country's enrollment).

The significant negative coefficient estimate for infant mortality is somewhat surprising, from a theoretical point of view. There is no obvious reason why infant mortality should affect the secondary school enrollment rate. This probably comes about because infant mortality is correlated with childhood mortality in the secondary-school years and with the general health of the population, both of which should affect enrollment.

Adult Mortality and Fertility. Columns 5 and 6 address the determinants of the total fertility rate. As we suggested above, the relationship between adult mortality and fertility appears to be central in accounting for the relationship between adult mortality and economic growth. Fertility is significantly positively associated with adult mortality and with infant mortality, and both variables have separately significant effects. The partial correlation between adult mortality and fertility is again very large in magnitude: using the specification in column 6, controlling for several other determinants of fertility, a standard deviation increase in adult mortality is associated with a 0.56 point increase in the fertility rate. This strongly supports the idea that fertility decisions are not simply determined by the number of children expected to survive early childhood, but rather reflect a more sophisticated set of preferences affected by the risks the child will face throughout life.

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enrollment ratio since it is more widely available for a broad panel of countries. The correlation between our gross enrollment ratio and Mankiw, Romer and Weil's schooling variable for the overlapping sample and period (1960-1985) cross-sectional average is 95.4%, so the difference should be immaterial in practice.

[31] For further discussion of the measurement of human capital, see Bils and Klenow (2000).

4.3.2 Corroborative evidence

Adult Mortality and the AIDS pandemic. The next set of regressions seeks to predict the prevalence of AIDS in 2001 as a function of variables observed before the AIDS pandemic emerged. If high adult mortality leads to increases in behavior with short-term benefits but long-term costs, we would expect it to be associated with the spread of AIDS. The results of OLS regressions for the determination of the AIDS death rate are displayed in the first three columns of Table 7. We find statistically significant evidence that adult mortality over 1960-1980 is positively associated with the death rate from AIDS, across all specifications. The finding is robust even to the inclusion of the fertility rate, despite its high collinearity with adult mortality. The estimated effect is large: using the estimates in column 3 of Table 7, a one standard deviation increase in 1960-1980 adult mortality (equal to 0.09) is associated with a 1.40 death per thousand increase in the AIDS death rate. For comparison, the mean of the AIDS death rate in 2001 was 1.39 per thousand, so the effect is slightly more than the mean of the dependent variable.

There are two possible interpretations of this finding, not mutually exclusive. The first is behavioral, along the lines described above: people who are already likely to die of other causes will be more prone to engage in risky behavior yielding short-term benefits at longer term costs. An alternative explanation is medical: in locations where adults are at greater risk of dying, for instance due to a pre-existing prevalence of communicable diseases and limited medical care, a further weakening of their immune systems through the virus that causes AIDS will result in a larger number of deaths classified as AIDS related. To try to discriminate between these two stories, we used the proportion of adults living with AIDS as an alternative dependent variable. Relative to mortality rates from AIDS, any association between this variable and (pre-AIDS) adult mortality is more likely to reflect the behavioral interpretation rather than the medical interpretation.

The results are in the last three columns of Table 7. The statistical significance of adult mortality is even stronger than before: adult mortality in 1960-1980 is consistently negatively related to the prevalence of AIDS in the adult population in 2003. Using the estimates in column 6, a one standard deviation increase in adult mortality is associated with a 4.40 percentage point increase in the share of the adult population living with AIDS. For comparison, the mean of this variable is 2.72, so the effect is large. These regressions provide further evidence that high adult mortality is associated with behavior characterized by short-term benefits and long-term costs.

Smoking and Adult Mortality. The final set of OLS regressions we report relates to the relationship between tobacco use and adult mortality. Smoking is a quintessential activity with short-term benefits and long-term costs, so the propensity to smoke should be related to agent's time horizons. To examine this hypothesis, we consider three different dependent variables: the proportion of the male population that was smoking, the proportion of the total population that was smoking, and the average number of cigarettes consumed per person. These variables are observed as of 2002.

An important caveat is in order when it comes to these regressions: reverse causality is a priori more serious here than in some of our previous regressions, because smoking has been prevalent throughout the period and it directly causes adult mortality. Ideally, we would like to run a regression using adult mortality net of smoking-related deaths as the regressor. Unfortunately, the required data are not available for our sample. According to Mackay and Ericksen (2002), in 2002 4.2 million people died worldwide of tobacco-related causes, broadly defined. With an overall world death rate of 8.93 per thousand in 2002, this implies that 7.5% of deaths worldwide were directly or indirectly attributable to tobacco. While this is not a very large share of overall deaths, the regressions should be interpreted cautiously.

Table 8 displays our results. Across dependent variables, we find evidence that adult mortality is positively related to the prevalence of smoking and cigarette consumption, after controlling for various other variables. The results are strongest when smoking is measured by the share of male smokers, smoking being mostly a male phenomenon. A one standard deviation increase in adult mortality is associated with an 11 percentage point increase in the proportion of male smokers (the mean of this variable in our sample is 40.25%). The estimates are smaller in magnitude when the dependent variable is the share of the total population that smokes, though adult mortality remains statistically significant. The smaller magnitude needs to be compared to the correspondingly smaller mean of the dependent variable (27.40%), reflecting the fact that fewer women smoke than men. Finally, adult mortality averaged over 1960-2000 is a moderately significant predictor of the number of cigarettes consumed per person in 2002 (column 6). The magnitude indicates that a one standard deviation increase in adult mortality is associated with an increase in cigarettes consumed per person of 311.15 (the mean number of cigarettes consumed per person in 2002 in our sample is 1211).

4.4 Summary

In this section, we have sought to characterize the partial correlations between adult mortality and a variety of dependent variables: growth, investment, human capital accumulation, fertility, and the propensity to smoke and to become infected with HIV/AIDS. We have found considerable evidence that adult mortality is negatively associated with growth, investment and human capital accumulation, and positively associated with fertility, smoking and the prevalence of AIDS. We also found that the link between fertility and adult mortality seems central to the relationship between death and development.

Together, these partial correlations paint a picture consistent with the conceptual framework introduced in Section 2: a high rate of adult mortality is conducive to behavior that yields short-term private benefits at a long-term social cost, and is detrimental to behavior that yields long-term social benefits at a short-term private cost. We have explicitly refrained from interpreting these partial correlations causally, recognizing that causality might run both ways and thereby biasing the OLS coefficient on mortality away from zero. We have instead examined whether the partial correlations yielded magnitudes big enough for our story to have any potential to account for a large portion of cross-country differences in economic performance. We have found this to be the case, and turn to issues of causality in the following section.

5 A structural approach to mortality

In this section, we pursue an explicitly structural econometric approach to explore the partial correlations uncovered in the previous section. This has two benefits. First, it helps us deal with the possibility of endogeneity in the mortality-growth relationship. Contrary to what would happen if reverse causality were a major source of bias, we find that instrumenting for adult mortality actually *increases* the estimated effect on growth. Second, using a system of equations allows us to explore the relative importance of the channels through which mortality affects growth. We find that fertility and physical-capital investment indeed account for most of the mortality-growth connection. Surprisingly, human-capital investments, as measured by enrollment levels, do not seem to play a substantial role.

5.1 The problem of reverse causality

Causality between mortality and development is likely to run both ways, as mentioned already. In fact, reverse causality between income levels and mortality is a necessary condition for adult mortality to generate a poverty trap. Countries with low income have high adult mortality, which in turn makes it hard to grow out of poverty. If adult mortality is an important enough determinant of growth, as we argue in this paper, then the vicious cycle between death and development might explain a significant portion of cross-country income differences.

Problems of reverse causality would be most pronounced had we run regressions of income levels on adult mortality, since the *level* of income is clearly a strong determinant of mortality.[32] Rich countries typically have completed their demographic transitions, devote substantial resources to health care and are thus characterized by lower mortality rates across the board. This is why we focused on regressions of growth on mortality in Section 3: reverse causality is likely to be less consequential in growth regressions, since the initial level of income appears as a control on the right-hand side of the growth equation. Moreover, OLS coefficients are useful to establish whether adult mortality is a plausible candidate as a major explanation for economic performance, since endogeneity bias would a priori increase the magnitude of the coefficient on adult mortality. We found that the magnitude of the partial correlation was indeed large.

However, it is still conceivable that growth specifications could be vulnerable to reverse causality. Persistently slow-growing countries, for instance in Africa, may not be able to devote incremental resources to fighting diseases and improving medical infrastructure, thereby reducing mortality. Hence, while controlling for income on the right-hand side mitigates the problem of reverse causality, it may not eliminate it altogether. Reverse causality is also a potential concern in the investment, school enrollment and fertility regressions presented above (although again, we controlled for initial income in all of these regressions). In this section, we confront head-on the potential for reverse causality in the growth, investment, human capital and fertility equations.

[32] However, several findings should lead to caution when advocating the view that there is a strong effect of income levels on mortality measures. Becker et al. (2005) show that the worldwide convergence in mortality rates has been dramatic, despite the lack of convergence in income levels. Relatedly, Deaton (2004) argues that a variety of historical and econometric evidence indicates that "the transmission of health knowledge and technology is as important as changes in income" in determining current levels of mortality.

5.2 Specification of the structural model

In order to deal with these issues, we formulate a structural model making explicit the causal links between growth, the channels linking it to mortality, and the mortality variables themselves. The channel variables we examine are those already discussed in Sections 2 and 4, namely investment in physical capital, school enrollment and the rate of fertility. We explicitly relate the mortality variables to a set of exogenous variables to be used as instruments for mortality, now treated as an endogenous regressor. These exogenous variables, to be further described below, relate to the natural conditions for the prevalence of malaria ("malaria ecology"), climatic factors and geographic characteristics of the countries in the sample.[33] Our structural system for the simultaneous determination of the variables of interest is the following:

$$\left.\begin{array}{c}\text{Malaria ecology}\\ \text{Climatic factors}\\ \text{Geographic features}\end{array}\right\} \Rightarrow \left.\begin{array}{c}\text{Adult mortality}\\ \text{Crude death rate}\\ \text{Infant mortality}\end{array}\right\} \Rightarrow \left.\begin{array}{c}\text{Investment}\\ \text{Secondary enrollment}\\ \text{Fertility}\end{array}\right\} \Rightarrow \text{Growth}$$

This structural system entails two main assumptions. The first is that the total effect of the mortality variables on economic growth is exhausted by the channel variables that we specified. In other words, there is no direct effect of mortality on growth, so that the sum of the effects of mortality on growth through investment, enrollment and fertility should be commensurate with the total effect of mortality estimated from a specification of growth on mortality and other controls (without controlling for the channel variables).[34] The second assumption is that the only way that malaria ecology, climatic factors and geographic features affect growth is through their effects on the mortality variables. We will provide statistical tests of both of these critical assumptions below.

The specification for the equations in the model follows closely those of Section 4. Specifically, the specification for the growth regression is that of column (5) of Table 3, with the mortality

[33] In a previous version of this paper, we used initial values and lagged values of the regressors as instruments. This yielded results broadly consistent with those we report here. However, since this procedure requires assuming that the endogenous regressors are predetermined, and since this assumption is not easily justifiable, we do not pursue this approach further. The corresponding results are available upon request.

[34] In Table 3, when all channel variables were controlled for, adult mortality was no longer significant in the growth regression. This is consistent with the view that adult mortality has no direct effect on growth. We further evaluate this statement statistically below.

variables excluded:

$$\begin{aligned} \text{growth}_i &= \alpha_1 + \alpha_2(\text{log initial income per capita})_i + \alpha_3(\text{fertility})_i + \alpha_4(\text{investment rate})_i \\ &\quad + \alpha_5(\text{secondary enrollment})_i + \alpha_6(\text{government consumption})_i + \alpha_7(\text{openness})_i \\ &\quad + \alpha_8(\text{log of population})_i + \alpha_9(\text{openness} * \text{log of population})_i + \varepsilon_i \end{aligned} \tag{1}$$

where growth is measured in annual terms from 1960 to 2000, initial income is measured in 1960, and the other regressors are time averages over 1960-2000.[35] The functional forms for the channel equations are exactly those of columns (2), (4) and (6) of Table 6, which include the three mortality variables.

5.3 Instrumental variables

5.3.1 Choice of variables

To address the endogeneity problem, we use three categories of variables as instruments for the three mortality indicators: malaria ecology, climatic variables, and geographic features of countries. We require several instruments because all three mortality variables are possibly endogenous and we need at least one instrument per endogenous regressor. Moreover, additional instruments might result in a better first-stage fit and allow for tests of overidentifying restrictions.

The malaria ecology index (ME), developed by Sachs et al. (2004), measures the exogenous portion of malaria incidence. One drawback of using malaria incidence directly is that it is affected by human actions, and may thus depend on income (richer countries are better equipped to eradicate the malaria vector).[36] In contrast, the malaria ecology index combines "climatic factors, the presence of different mosquito vector types and the human biting rate of the different mosquito

[35] This specification corresponds quite closely to the one found in the cross-country growth literature, derived from an augmented Solow model. It contains, on the right hand side, flow measures of accumulation (investment, enrollment) and depreciation of per capita quantities (fertility) - our channel variables. In addition, following the findings in Alesina, Spolaore and Wacziarg (2001), we include "extent of the market" controls: openness, the log of population, and the interaction of these two variables. Following Barro and Sala-i-Martin (1996), the specification includes the government consumption share of GDP. The latter two sets of variables are controls and are not central to our analysis.

[36] This would also be a concern with any attempt to use the prevalence or mortality rates of other diseases as instruments. Poor or poorly-run countries are more likely to suffer from a variety of diseases, especially prior to the rapid diffusion of health knowledge that occurred in the post-World War II period.

vectors" (Sachs et al., 2004) to generate a measure of *potential* malaria prevalence independent of human activity. It is therefore plausibly exogenous in the sense of being unaffected causally by growth and our channel variables, and yet correlated with malaria incidence and other tropical diseases related to mortality. In fact, the raw correlation between ME and our measure of adult mortality is 0.66 in a sample of 153 countries for which both variables are available.

To supplement the malaria ecology index, we use a collection of climate variables. Many diseases require specific ranges of temperature, precipitation, and humidity to survive and spread. Mosquito-born diseases such as malaria, dengue fever, and yellow fever require warm weather and standing water. Influenza epidemics generally occur during cooler weather. Meningitis is more common in dry environments (National Research Council, 2001). Cholera outbreaks are associated with temperature and tidal fluctuations (Lobitz et al., 2000). In addition, the climate will also affect mortality through its effects on the variability of agricultural outcomes and even directly through instances of extreme heat and cold. As a rough summary of climate, we use a set of variables measuring the percentage of a country's land located in each of the twelve climate zones.[37] To these variables we add a variable measuring the proportion of land with more than five days of frost per month in winter, from Masters and McMillan (2001). Climate is strongly linked to mortality rates: in the sample of 144 countries for which all these variables are available, a regression of adult mortality on the climate variables together yields a joint F-test of 24.73 (with a p-value of 0.000) and an adjusted R^2 of 0.38.[38] In addition, climate is unaffected causally by investment, mortality, or income growth.

Finally, our set of instruments includes measures of a country's geographic features: the distance of a country's centroid from the equator, the mean distance to the nearest coastline, the average elevation, and the log of land area. Again, these variables are causally unaffected by the variables they are meant to instrument, but are related to climatic and possibly historical factors affecting

[37] The 12 Koeppen-Geiger climate zones are: tropical rainforest climate (Af), monsoon variety of Af (Am), tropical savannah climate (Aw), steppe climate (BS), desert climate(BW), mild humid climate with no dry season (Cf), mild humid climate with a dry summer (Cs), mild humid climate with a dry winter (Cw), snowy-forest climate with a dry winter (Dw), snowy-forest climate with a moist winter (Ds), tundra/polar ice climate (E) and highland climate (H). Category E was eliminated from our list of instruments to avoid linear dependence.

[38] We discuss formal first-stage F-tests for our instruments below. These involve a smaller sample (that used in the IV regressions), additional exogenous controls not used as instruments, and varying sets of instruments, as described below.

mortality levels. In a regression of adult mortality on these geographic indicators alone, in a sample of 123 countries, the F-statistic for their joint significance has a value of 87.59 (with a p-value of 0.000) and an adjusted R^2 of 0.52. These variables are valid instruments under the assumption that they affect the outcome of interest only through the regressors that are treated as endogenous, a hypothesis we test statistically below.

In total we have 17 instruments, organized in three sets. We use various subsets of these variables as instruments for adult mortality, the crude death rate and infant mortality. We do so in order to examine the robustness of our estimated coefficients to using different sets of instruments, to address the concern that some variables may not be excludable from the estimating equations. Specifically, we present estimates using all sets of variables, and all three possible combinations of two sets.[39] In addition, in order to control for the possible endogeneity of openness and the interaction term between openness and the log of population, we add two commonly used instruments in some of our IV regressions: the gravity-based measure of exogenous openness developed by Frankel and Romer (1999) and its interaction with the log of population.

The first concern we have is whether the instruments might be weak, which would bias IV estimates towards OLS (see Stock, Wright and Yogo, 2002 and Staiger and Stock, 1997). Table 9 presents F-statistics and Shea's R^2 statistics from first stage regressions of each mortality variables on the various instrument sets. The first-stage relationships are generally quite strong, except when the climate variables are excluded from the list of instruments.[40] In other cases, Shea's R^2 statistic takes values of up to 0.59 (for the first stage of adult mortality when using all instruments). The weak first-stage relationship when only ME and geography are used as instruments suggests the corresponding IV results might be unreliable.

[39] We also ran IV regressions using only malaria ecology to instrument for adult mortality, finding coefficients that are of magnitudes similar to those we report below. This amounts to treating the other mortality indicators as exogenous, an undesirable assumption.

[40] Note that the Stock and Staiger rule of thumb for assessing the weakness of instruments states that instruments are weak when the first-stage F-test is smaller than 10. However, this rule of thumb only applies to the case of one endogenous regressor. In our application, we have three. For this reason, we rely mostly on Shea's R^2 as a measure of first-stage fit.

5.3.2 IV estimates of the total effect of mortality

In our first pass at IV estimation of our structural model, we seek to characterize the total effect of the mortality variables, particularly adult mortality, on economic growth. To do so, we substitute the channel equations into the growth equation. Given our chosen specifications for the channel equations, the resulting "reduced form" growth specification is as follows:[41]

$$\begin{aligned}\text{growth}_i &= \beta_1 + \beta_2(\text{log initial income per capita})_i + \beta_3(\text{adult mortality})_i + \beta_4(\text{crude death rate})_i \\ &\quad +\beta_5(\text{infant mortality})_i + \beta_6(\text{government consumption})_i + \beta_7(\text{population density})_i \\ &\quad +\beta_8(\text{urbanization rate})_i + \beta_9(\text{democracy index})_i + \beta_{10}(\text{log of population})_i \\ &\quad +\beta_{11}(\text{openness})_i + \beta_{12}(\text{openness} * \text{log of population})_i + \nu_i \qquad (2)\end{aligned}$$

To estimate equation (2), we treat the three mortality indicators as endogenous, and the rest of the control variables as exogenous, though in some specifications we allow openness and its interaction with the log of population to be endogenous. When we do so we add to our list of instruments the Frankel and Romer gravity-based measure of openness. The results are presented in Table 10. Column (1) displays results using all three sets of instruments, treating openness and its interaction with population as exogenous. The estimates of the mortality variables are all statistically significant, and in magnitude *larger* than those obtained with OLS. Running the OLS equivalent of the specification in column (1) yields an effect of adult mortality equal to -6.32, whereas the IV coefficient is equal to -8.56. IV estimates are larger than OLS estimates in magnitude, despite theoretical priors to the contrary. This a common finding in this type of application.[42] It may suggest a reduced incidence of attenuation bias due to measurement error under IV.

The estimated effects of the mortality variables are quite robust to the use of alternative sets of instruments, and to treating openness and its interaction with log population as endogenous (columns 2-8). One exception is when the list of instruments excludes the climate variables (columns 3 and 7). In this case, the estimated effect of adult mortality, equal to -21.303 (column 3), is perhaps unreasonably large, and the estimates on the other mortality variables are also sensitive to

[41] Reduced form is a slight misuse of language here: the mortality variables, which are treated as endogenous, still appear on the right hand side. What we mean by "reduced form" is that we have substituted away the channel variables in order to estimate the total effect of mortality on growth. These are still IV estimates.

[42] See Acemoglu, Johnson and Robinson (2001) and Frankel and Romer (2001), for instance, for applications where this is the case.

this choice of instruments. This is not surprising, as Table 9 suggests that the instruments in this particular specification are weak, as indicated by the small value of Shea's R^2. In no case is the estimate on adult mortality smaller in magnitude than that of column (1), so to be conservative we can use these estimates as a baseline for the total effect of mortality. In terms of magnitudes, a one-standard deviation increase in adult mortality in that specification, is associated with a 1.16 percentage point reduction in economic growth, a large effect.[43]

As a final diagnostic test, we report Hansen J statistics to conduct tests of overidentifying restrictions. This statistic, basically an extension of the Sargan statistic, is consistent in the presence of heteroskedasticity and autocorrelation (the standard errors we present throughout this paper are robust to both). The null hypothesis is a joint hypothesis that the error term is uncorrelated with the instruments, and that the instruments are correctly excluded from the regression. In our baseline specification of column (1), the $\chi^2(14)$-distributed test statistic takes on a value of 13.55, with an associated p-value of 0.48. Thus, we fail to reject the null of valid overidentifying restrictions. While the power of this type of test may be low in the presence of other sources of misspecification, we can be heartened by the results: they do suggest that the only way our instruments affect economic growth is through the mortality variables jointly. This is a critical assumption to identify their effects.

These estimates, obtained by substituting away the channel variables in our structural system, provide a notion of the total effect of the mortality variables on growth. We now turn to decomposing this total effect into our three postulated channels of influence.

5.4 System estimates of the mortality-growth relationship

We argued above that the effect of adult mortality is likely to work through investment in human and physical capital, as well as fertility. We now quantify the relative importance of these channels. To do so, we estimate directly the simultaneous-equations system described in Section 5.2. To facilitate the readability and interpretation of our results, we depart slightly from the specifications we have shown so far and use a single summary measure of mortality in the system: the adult

[43] We also explored specifications that include measures of institutional quality as a control. We instrumented for institutions using Acemoglu et al.'s (2001) log of settler mortality variable. In the specification of column 5, including either expropriation risk or executive constraints as an additional regressor instrumented for with log settler mortality had almost no effect on the magnitude or significance of adult mortality, estimated on the same (much smaller) subsample for which settler mortality data was available. Results are available upon request.

mortality rate, which we have argued is both the most theoretically appropriate and the most consistently robust predictor of the channel variables and growth. We do this so that the growth effect of a single mortality variable can be traced out through the channel variables in an easy-to-interpret way.[44] Our baseline specification of the growth and channel equations is otherwise identical to what has been presented so far. The full specification of our baseline model, along with the estimates for each equation, are presented in Appendix 3, Table A3.

Our econometric methodology, relying on three-stage least squares estimation (3SLS), follows that in Tavares and Wacziarg (2001) and Wacziarg (2001). As instruments, we use the three sets of exogenous variables described above (malaria ecology, climate variables and geographic features). In addition, the 3SLS methodology implies that the exogenous variables in the system that are excluded from a given equation are used as instruments for the included endogenous variable(s) in that equation. Joint estimation of the growth and channel equations allows us to take advantage of possible cross-equation error correlations, resulting in gains in efficiency. An additional advantage of this method is that we can compute a single covariance matrix for all the estimates in the system, allowing for possibly complex inferences on functions of the parameters, even if they belong to different equations. For instance, we are interested in the effect of adult mortality on growth through each channel variable, which is the effect of mortality on the channel multiplied by the effect of the channel on growth. We are also interested in inference on the sum of these channel effects. Below, we present Wald tests for these hypotheses based on nonlinear functions of the system estimates.

The results of our baseline system estimation are in Table 11. The total effect of adult mortality on growth through the three channels is equal to -6.25, implying that a one standard deviation increase in adult mortality is associated with a 0.85 percentage point decrease in growth. If our model is well specified, the sum of the channel effects should be commensurate with IV estimates of the total effect of adult mortality from Table 10. The total effect we estimate here is slightly smaller than the total effect estimated in column (1) of Table 10, where the estimate was -8.56, but it is in the 95% confidence interval of that estimate. This suggests that our three channels capture most if not all of the total effect of adult mortality on economic growth. Further evidence of the

[44] The estimated effect of adult mortality is not very sensitive to the inclusion of the other mortality variables in the system. System estimates that include these variables are available upon request. These estimates are harder to interpret since the scales of our three mortality variables are different, so we would have to present 9 channel effects, and 3 total effects (adult mortality, infant mortality and the crude death rate, respectively).

exhaustiveness of the channels can be obtained by running a simple OLS regression of the residuals from the growth equation on adult mortality. The resulting estimate on the adult mortality variable is equal to -0.23, and is statistically indistinguishable from zero (the t-statistic is equal to -0.24). Thus, we can be quite confident that our three channels exhaustively capture the effect of adult mortality on economic growth.

Turning to the channels themselves, we note that, consistent with the observations based on OLS estimates in Section 4, adult mortality is negatively related to the investment rate and secondary enrollment, but positively related to the fertility rate. The investment effect (-20.85) is very close in magnitude to the OLS estimate in column (2) of Table 6 (-20.64). Similarly, the enrollment and fertility effects are close to those we reported earlier (a little smaller for enrollment, a little bigger for fertility), and all of these effects are statistically significant at the 7% level or better. As for the effects of the channels on growth, physical capital investment bears a positive effect and fertility a negative one, in line with the predictions of the Solow model. However, the enrollment effect comes out negative and statistically insignificant (the effect was also insignificant in the OLS estimates of Table 3, although it was positive). This is consistent with the general difficulty economists have had in pinning down a robust relationship between human-capital variables and economic growth (Pritchett, 1996; Bils and Klenow, 2001, Benhabib and Spiegel, 1994).

These results suggest that the main channels through which adult mortality affects growth are physical-capital investment and fertility: the effect of adult mortality on economic growth through physical-capital investment is equal to -3.82, and is statistically significant at the 6% level. The fertility effect is -3.87, and is significant at the 0.4% level. The effect through enrollment, which bears the opposite sign from that expected, is small ($+1.44$) and statistically insignificant even at the 10% level.[45]

To conclude, the effect of adult mortality on economic growth seems predominantly due to the effects on fertility and on investment, in roughly equal proportions. Secondary enrollment does not seem to be an important channel, though adult mortality does affect enrollment negatively, in line

[45] If human capital investment and fertility are jointly determined by parents, and the quantity-quality tradeoff operates as suggested in Kalemli-Ozcan (2002) and Soares (2004), then it may be that fertility (which is well-measured) could be proxying for human capital investment (which is imperfectly measured). That is, parents in high fertility countries would be under-investing not only in the measured portion of education (secondary enrollment) but in other unmeasured aspects of childcare and education quality. Without a more comprehensive and accurate cross-national measure of human capital investment, however, this remains conjecture.

with theory.

5.5 Summary

A consistent picture emerges from our attempts to account for endogeneity in the growth-adult mortality relationship. The overall effect of adult mortality on growth comes out negative and statistically significant. The magnitudes vary somewhat, but a reasonable estimate of the total effect of adult mortality on growth, from Tables 10 and 11 seems to be in the range of -6 to -10. With such a range of estimates, a one standard deviation increase in mortality is associated with a reduction in the annual economic growth rate of between 0.8 and 1.4 percentage points, slightly larger than the corresponding magnitude from the benchmark OLS estimate in column 4 of Table 3. Channel estimates suggest that fertility and investment are important mediating channels linking adult mortality to growth. Consistent with our theoretical priors, adult mortality also reduces secondary school enrollment. However, in line with past findings in the literature on human capital and growth, the effect of the schooling on growth is not robust.

6 Conclusion

We opened this paper with a straightforward observation: the short time horizon induced by high mortality causes people to take actions that yield short-term benefits at long-term costs. We find evidence of this effect across a range of data using multiple econometric approaches.

Mortality matters: adult mortality alone can account for all of Africa's growth shortfall over the 1960-2000 period. Furthermore, adult mortality is a significantly negative predictor of physical capital investment rates, enrollment rates in secondary education, and growth of per capita GDP. These effects are economically large. In addition, mortality is a significantly positive predictor of fertility rates as well as a variety of measures of risky behavior, such as the prevalence of smoking and AIDS infection rates.

We explored three channels whereby adult mortality may affect growth: investment, human-capital accumulation and the fertility rate. Each of the channels operates in the expected direction, but the strongest linkage is through fertility. The demographic transition accounts for much of the high correlation between fertility and adult mortality: countries with high fertility and high mortality, that are in the early stages of their transitions, and countries with low mortality and

low fertility, that have completed it, dominate the variation. The demographic transition is characterized by a fall in mortality followed by a fall in fertility. This timing suggests that causality runs mostly from mortality to fertility, rather than the reverse. In light of the importance of the fertility channel in our empirical results, further theoretical and empirical research on the impact of falling mortality on fertility rates seems called for.

Overall, the results of this paper are consistent with the hypothesis that short horizons are a first order problem of development: high adult mortality induces economic agents to invest less, accumulate less human capital, have a large number of children rather than fewer, high quality ones. This, in turns, lowers economic growth. Low growth means that countries, especially in Africa, are unable to devote resources to fighting diseases and reducing mortality. At a minimum, high adult mortality has hindered developing countries' economic growth. At its worst, the negative link between death and development may lead to self-perpetuating poverty.

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Appendix 1 – Data on Mortality

Table A1 - Income, Growth and Mortality: Data (cross-section of countries) – 1960-2000 averages

country	PPP Income per capita	Growth 1960-2000	Infant mortality	Total death rate	Adult mortality (age 15-60)
Sweden	17004	2.108	0.009	0.011	0.126
Iceland	16479	2.758	0.009	0.007	0.129
Netherlands	16077	2.417	0.010	0.008	0.130
Israel	11402	2.795	0.018	0.007	0.132
Greece	10214	3.137	0.025	0.009	0.138
Norway	16635	2.973	0.011	0.010	0.140
Switzerland	21766	1.418	0.011	0.009	0.141
Japan	15591	4.230	0.012	0.007	0.146
Cyprus	7930	.	0.019	0.009	0.146
United Kingdom	15016	2.075	0.013	0.011	0.151
Denmark	18661	2.211	0.011	0.011	0.151
Spain	11476	3.397	0.020	0.008	0.152
Italy	14719	2.878	0.021	0.010	0.152
Ireland	11118	4.091	0.015	0.010	0.155
Canada	18238	2.380	0.014	0.007	0.155
Malta	13106	.	0.018	0.008	0.157
Australia	17338	2.177	0.012	0.008	0.162
Puerto Rico	9709	.	0.023	0.007	0.165
New Zealand	15118	1.222	0.014	0.008	0.165
Belgium	15631	2.794	0.016	0.011	0.166
Germany	17494	.	0.016	.	0.168
Costa Rica	4667	1.310	0.037	0.005	0.170
Yugoslavia	.	.	0.044	0.010	0.170
Cuba	6167	.	0.023	0.007	0.172
Barbados	9587	3.938	0.032	0.009	0.173
Turkey	4657	2.332	0.104	0.011	0.175
Kuwait	23386	.	0.038	0.005	0.175
Portugal	9025	3.839	0.036	0.010	0.177
Hong Kong	13709	5.391	0.017	0.005	0.177
Brunei	.	.	0.031	0.006	0.178
United Arab Emirates	.	.	0.051	0.008	0.179
Austria	15232	2.929	0.018	0.012	0.180
Uruguay	7103	1.234	0.034	0.010	0.182
France	15547	2.625	0.014	0.010	0.186
Luxembourg	21526	3.310	0.015	0.011	0.188
Jamaica	3602	0.741	0.033	0.007	0.192
Panama	4616	2.398	0.037	0.007	0.192
U.S.A.	21500	2.495	0.015	0.009	0.197
Paraguay	3903	1.645	0.045	0.008	0.197
Bulgaria	6164	.	0.025	0.011	0.197
Taiwan	6765	.	.	.	0.198

country	PPP Income per capita	Growth 1960-2000	Infant mortality	Total death rate	Adult mortality (age 15-60)
Singapore	10649	.	0.015	0.005	0.198
Albania	2771	.	0.065	0.007	0.200
Iran	4436	2.024	0.094	0.012	0.201
Argentina	9249	1.002	0.039	0.008	0.201
Armenia	2399	.	0.027	0.007	0.202
Bahrain	13261	.	0.043	0.007	0.205
Sri Lanka	2059	2.266	0.044	0.007	0.209
Finland	14996	2.889	0.011	0.009	0.209
Czech Republic	12962	.	0.015	0.012	0.211
Slovenia	13181	.	0.018	0.010	0.217
Venezuela	8063	-0.500	0.036	0.006	0.218
Romania	3389	3.544	0.038	0.010	0.219
Trinidad & Tobago	8208	2.347	0.037	0.007	0.220
Qatar	19844	.	0.039	0.009	0.222
Tajikistan	1198	.	.	0.009	0.224
Mexico	6557	1.973	0.058	0.008	0.227
Lithuania	6755	.	0.026	0.010	0.229
Poland	7032	.	0.028	0.009	0.231
Dominican Republic	2867	2.836	0.072	0.010	0.232
Chile	5715	2.366	0.049	0.008	0.233
Algeria	4174	1.521	0.099	0.012	0.235
Georgia	4971	.	0.034	.	0.236
Tunisia	4078	.	0.088	0.011	0.238
Ecuador	3295	1.371	0.065	0.010	0.241
Lebanon	4705	.	0.042	0.010	0.243
Brazil	5199	2.773	0.072	0.009	0.243
Hungary	8357	.	0.027	0.013	0.247
Suriname	.	.	0.045	0.008	0.248
Fiji	4116	.	0.040	0.007	0.249
Uzbekistan	2652	.	.	.	0.250
Bahamas	16527	.	0.031	0.006	0.251
Colombia	4083	1.888	0.049	0.008	0.252
Egypt	2544	2.602	0.115	0.014	0.258
Azerbaijan	2368	.	0.092	0.007	0.258
Mauritius	6920	3.711	0.040	0.007	0.260
Belarus	7174	.	0.023	0.010	0.268
Ukraine	6453	.	0.024	0.011	0.269
Malaysia	5071	3.859	0.035	0.008	0.270
Iraq	.	.	0.082	0.013	0.273
Latvia	7029	.	0.022	0.012	0.274
Morocco	2837	2.611	0.091	0.013	0.274
Estonia	7943	.	0.023	0.012	0.276
Turkmenistan	4533	.	0.087	0.009	0.282
Saudi Arabia	12246	.	0.082	0.012	0.284
Guyana	2497	.	0.073	0.009	0.287

country	PPP Income per capita	Growth 1960-2000	Infant mortality	Total death rate	Adult mortality (age 15-60)
China	1484	4.261	0.071	0.011	0.289
Libya	.	.	0.074	0.011	0.290
Thailand	3331	4.595	0.056	0.009	0.291
Korea, Rep. of	6485	5.906	0.032	0.008	0.291
Peru	4438	0.879	0.089	0.011	0.292
Vietnam	1379	.	0.046	0.012	0.292
CapeVerde	2015	3.496	0.067	0.011	0.293
India	1365	2.684	0.108	0.015	0.294
Korea, Dem. Rep.	.	.	0.044	0.009	0.295
Honduras	2063	0.468	0.081	0.011	0.296
Seychelles	6587	3.049	.	.	0.296
Pakistan	1283	2.885	0.107	0.016	0.299
Nicaragua	3065	-1.218	0.083	0.011	0.300
Moldova	2211	.	0.042	.	0.310
El Salvador	3999	0.732	0.081	0.010	0.313
Russian Federation	7780	.	0.029	.	0.314
Kyrgyzstan	2787	.	0.092	.	0.315
Philippines	2791	1.327	0.054	0.009	0.334
Kazakhstan	6199	.	.	.	0.334
Oman	16668	.	0.080	0.013	0.336
Mongolia	1268	.	0.093	0.012	0.344
Bolivia	2664	0.365	0.111	0.015	0.366
Guatemala	3371	1.282	0.090	0.012	0.370
Liberia	.	.	0.168	0.021	0.378
Madagascar	1045	-0.985	0.103	0.017	0.380
Indonesia	2050	3.397	0.081	0.014	0.392
Myanmar (Burma)	.	.	0.111	0.016	0.397
Bangladesh	1171	1.163	0.115	0.017	0.398
Yemen	901	.	0.149	0.019	0.402
Nepal	978	1.570	0.129	0.018	0.410
Ghana	1171	1.111	0.095	0.014	0.417
Haiti	1077	.	0.126	0.016	0.418
Swaziland	5268	.	0.112	0.016	0.452
Papua New Guinea	3104	.	0.093	0.015	0.456
Cambodia	1220	.	0.117	0.019	0.461
Gabon	7454	2.552	0.104	0.019	0.462
Congo, Republic Of	1443	3.250	0.099	0.018	0.470
Benin	1067	0.324	0.132	0.019	0.471
Kenya	1118	1.117	0.086	0.015	0.473
Lesotho	1186	2.060	0.114	0.016	0.475
Togo	1156	-0.073	0.115	0.019	0.476
Zimbabwe	2329	1.756	0.076	0.014	0.477
Mauritania	1524	.	0.138	0.020	0.486
Cote d'Ivoire	2237	0.345	0.137	0.018	0.489
Equatorial Guinea	2646	0.246	0.144	0.022	0.490

country	PPP Income per capita	Growth 1960-2000	Infant mortality	Total death rate	Adult mortality (age 15-60)
Ethiopia	581	0.466	0.146	0.023	0.498
Namibia	4607	.	0.088	0.015	0.505
Mali	840	-0.034	0.190	0.023	0.506
Laos	1367	.	0.129	0.019	0.509
Botswana	3362	.	0.080	0.013	0.509
Cameroon	2018	0.487	0.113	0.017	0.511
Burkina Faso	781	0.594	0.142	0.022	0.513
Sudan	1159	.	0.090	0.018	0.514
Senegal	1567	-0.285	0.127	0.020	0.514
Tanzania	531	0.584	0.117	0.017	0.516
Afghanistan	.	.	0.186	0.025	0.519
SouthAfrica	7124	1.047	0.066	0.012	0.520
Chad	1092	-0.721	0.141	0.023	0.520
Guinea-Bissau	546	1.193	0.169	0.025	0.520
Uganda	659	1.295	0.104	0.019	0.520
Malawi	601	1.568	0.163	0.024	0.522
Nigeria	1024	-0.948	0.126	0.018	0.522
Mozambique	1290	-1.051	0.153	0.021	0.530
Burundi	715	-0.059	0.125	0.020	0.539
Niger	1196	-1.546	0.190	0.025	0.541
Angola	2262	.	0.177	0.024	0.541
Djibouti	2103	.	0.141	0.020	0.542
Rwanda	933	-0.116	0.119	0.022	0.542
Guinea	2555	0.066	0.169	0.024	0.542
Gambia, The	1157	0.748	0.146	0.024	0.544
Central African Republic	1727	.	0.137	0.021	0.547
Zambia	1175	-0.756	0.106	0.018	0.559
Somalia	.	.	0.147	0.024	0.561
Sierra Leone	1241	.	0.192	0.029	0.573

Table A2 - Income, Growth and Mortality: Data
(Indian states dataset) – 1991 data only

State	NSDP per capita, 1991	Growth 1991-2000	Infant mortality	Total Death rate	Adult mortality (ages 20-40)	Adult Mortality (Ages 15-60)
Nagaland	1925	.	0.020	.	0.031	0.160
A_&_N_Islands	2505	.	0.030	0.006	0.033	0.221
Tripura	1602	4.85	0.052	0.008	0.040	0.233
West_Bengal	2102	4.25	0.063	0.008	0.043	0.236
Delhi	5192	.	0.031	0.006	0.044	0.229
Kerala	1791	4.52	0.015	0.006	0.045	0.197
Gujarat	2603	7.73	0.065	0.008	0.052	0.266
Rajasthan	1901	4.48	0.082	0.009	0.053	0.254
Maharashtra	3410	6.26	0.054	0.008	0.054	0.236
Sikkim	3298	.	0.052	0.007	0.055	0.247
Himachal_Pradesh	2202	.	0.069	0.009	0.058	0.226
Haryana	3428	2.42	0.068	0.008	0.059	0.223
Pondicherry	3166	.	0.018	0.007	0.059	0.310
Goa	4803	4.71	0.023	0.007	0.060	0.314
Karnataka	2007	2.85	0.065	0.008	0.061	0.273
Uttar_Pradesh	1620	1.64	0.091	0.011	0.061	0.259
Bihar	1171	-1.13	0.070	0.010	0.062	0.268
Andhra_Pradesh	1744	3.21	0.069	0.009	0.065	0.288
Tamil_Nadu	2208	4.15	0.055	0.008	0.066	0.274
Madhya_Pradesh	1658	3.64	0.107	0.012	0.066	0.286
Punjab	3659	2.54	0.052	0.007	0.066	0.232
Orissa	1358	1.01	0.110	0.011	0.067	0.299
Assam	1509	0.91	0.078	0.010	0.069	0.336
Manipur	1696	.	0.025	0.006	0.070	0.239
Arunachal_Pradesh	2636	0.69	0.052	0.009	0.074	0.274
Meghalaya	1681	1.10	0.052	0.009	0.075	0.285

Correlation between two last columns is 0.7.

Appendix 2 – Description of variables and data sources

A . Cross-national dataset

Variable	Source and definition
Log income per capita	Log of PPP real income per capita, Chain index Source: Penn World Tables, v. 6.1
Growth of income per capita	Annual growth of PPP real income per capita, Chain index Source: Penn World Tables, v. 6.1
Investment share of GDP	Investment in constant prices/GDP in constant prices, % Source: Penn World Tables, v. 6.1
Government consumption share of GDP	Government consumption in constant prices/GDP in constant prices, %. Source: Penn World Tables, v. 6.1
Imports plus exports over GDP (openness)	(Imports+Exports in constant prices)/GDP in constant prices, % Source: Penn World Tables, v. 6.1
Log of population	Log of population in 1000s. Source: Penn World Tables, v. 6.1
Adult mortality rate	Probability of a male surviving to age 60, conditional on surviving to age 15. Source: World Bank WDI
Infant mortality rate	Probability of an infant dying before age one. Source: World Bank WDI
Crude death rate	Proportion of the total population dying in a given year. Source: World Bank WDI
Total fertility rate	Expected number of births per woman, based on age-specific fertility rates. Source: World Bank WDI
Urbanization rate, %	Urban population / total population, % Source: World Bank WDI
Secondary school gross enrollment ratio	Rate of enrollment in secondary school Source: Barro-Lee dataset
Interstate battle deaths/population	Total estimated battle deaths summed over interstate wars 1960-1997, divided by average population over 1960-2000. Countries where a war occurred but no reliable battle death estimates are available are coded as missing data. Source: COW dataset.
Intrastate battle deaths/population	Total estimated battle deaths summed over intrastate wars 1960-1997, divided by average population over 1960-2000. Countries where a war occurred but no reliable battle death estimates are available are coded as missing data. Source: COW dataset
Months of interstate war	Total months between 1960 and 1997 in which the country was involved in interstate war, with double-counting for multiple wars. Source: COW dataset
Months of intrastate war	Total months between 1960 and 1997 in which the country was involved in intrastate war, with double-counting for multiple wars. Source: COW dataset
Democracy Index	Freedom House index of political rights. Source: Freedom House
Revolutions	Revolutions per year, attempted or successful, 1980-1999 average Source: Arthur Banks CNTS dataset

Variable	Source and definition
Sub-Saharan Africa dummy	Source: CIA World Factbook
Dummy for landlocked country	Source: CIA World Factbook
Dummy for island	Source: CIA World Factbook
Log of Land area in square km	Source: CIA World Factbook
Distance from equator	Absolute value of latitude/90. Source: CIA World Factbook
Dummy for Catholic majority	Source: Alesina et al.
Dummy for Muslim majority	Source: Alesina et al.
Ethnic fractionalization	Source: Alesina et al.
Death rate from AIDS per 1000, 2001	Death rate from AIDS in adults and children, 2001, per 1000. Source: UNAIDS
% adults (15-49) living with AIDS, end 2003	AIDS prevalence rate among adults aged 15-49, end 2003. Source: UNAIDS
Adults smoking, %, male	% of males smoking, 2000 or latest available data. Source: Mackay and Ericksen
Adults smoking, %, total	% of adults smoking, 2000 or latest available data. Source: Mackay and Ericksen
Cigarette consumption per capita, 1000s, 2002	Per capita cigarette consumption figures constructed from production, trade (import and export) and population data. Source: Mackay and Ericksen
Distance from coast	Mean distance to nearest coastline (km). Source: Gallup et al.
Elevation	Mean elevation in meters above sea level. Source: Gallup et al.
Koeppen-Geiger climate zones	11 variables measuring the fraction of land area in each of the Koeppen-Geiger climate zones, polar and tundra zones omitted. Source: Gallup et al.
ME	Malaria ecology index. This index combines “climatic factors, the presence of different mosquito vector types and the human biting rate of the different mosquito vectors” to generate a measure of potential malaria prevalence independent of human activity. Source: Sachs malaria dataset
Frost	Proportion of land with more than five frost days per months in winter. Source: Masters-McMillan dataset
Frankel-Romer instrument [FR]	Portion of a country’s total trade volume (as a percentage of GDP) constructed by summing the geography-determined portion of bilateral trade shares. Source: Frankel and Romer.

B. India dataset

Log of per Capita Income in Period Initial Year	Indian National Accounts
Mortality rate, male, ages 20-40	Source: Registrar General, Govt. of India.
Death rate, all ages	Source: Registrar General, Govt. of India.
Infant mortality rate, per 1000 live births	Source: Registrar General, Govt. of India.
Total fertility rate, avg. of first 5 years of each decade	Source: Registrar General, Govt. of India.
Urbanization Rate, %	Source: Census of India
Population density, inhab/sq km	Source: Census of India
Religious Fractionalization	1-Herfindahl index of religion shares. Constructed from data on religions by states. Source: Census of India
Share of scheduled castes and tribes, %	Source: Census of India
Literacy rate, %	Source: Census of India
Share of development expenditures in NSDP, %	Source: Reserve Bank of India.

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Appendix 3 – System Estimates

Table A3 - 3SLS estimates of the baseline system specification

	(1)	(2)	(3)	(4)
	Growth	**Investment**	**Human capital**	**Fertility**
Adult mortality rate		**-20.848**	**-0.509**	**5.681**
		(1.97)**	**(2.84)****	**(4.72)****
Investment share of GDP, %	**0.183**			
	(5.59)**			
Secondary school gross enrollment ratio	**-2.840**	-6.930		-3.463
	(1.82)*	(0.11)		(4.25)**
Total fertility rate	**-0.681**			
	(3.36)**			
Log income per capita, 1960	-0.947	-3.007	0.037	-0.252
	(4.24)**	(2.00)**	(1.41)	(1.47)
Government consumption share of GDP, %	-0.025			
	(1.75)*			
(Imports+Exports)/GDP, %	0.043			
	(1.94)*			
Log of population	0.308			
	(2.11)**			
Openness*log of population	-0.004			
	(1.53)			
Log per capita income * Enrollment		3.403		
		(0.56)		
Democracy index			-0.141	
			(1.98)**	
Urbanization rate, %			0.002	0.008
			(1.55)	(1.21)
Population density			0.138	-1.570
			(1.29)	(2.23)**
Constant	6.800	40.045	0.097	5.053
	(2.01)**	(2.71)**	(0.46)	(3.75)**
Observations	81	81	81	81
R-squared	0.45	0.53	0.79	0.84

Absolute value of t statistics in parentheses; * significant at 10%; ** significant at 5%.
Instruments for adult mortality in the channel regressions: Malaria ecology, climate variables, geography variables, as defined in text.

Table 1. Summary statistics for the main variables of interest

Variable	Obs.	Mean	Std. Dev.	Min	Max
a. Cross-Country Dataset					
Log income per capita, 1960-2000 avg.	168	8.300	1.005	6.274	10.060
Income per capita, 1960-2000 avg.*	168	6321	5779	531	23386
Growth of income per capita (annual, 1960-2000)	98	1.785	1.519	-1.546	5.906
Adult mortality rate (age 15-60)	160	0.311	0.136	0.126	0.573
Infant mortality rate	169	0.071	0.049	0.009	0.192
Crude death rate	163	0.013	0.005	0.004	0.029
Total fertility rate	162	4.183	1.628	1.633	7.260
Investment share of GDP	168	15.116	7.526	2.066	41.200
Secondary school gross enrollment ratio	109	0.253	0.188	0.013	0.685
b. Indian States Dataset**					
Log net state product per capita (1970-2000 average)	96	7.413	0.394	6.357	8.555
Net state product per capita (1970-2000 average)***	96	1802	828	577	5192
Growth of net state product per capita (annual, 1970-2000)	85	2.123	1.794	-1.247	7.726
Adult mortality rate (age 20-40)	54	0.062	0.013	0.031	0.091
Infant mortality rate	57	0.080	0.036	0.015	0.173
Total death rate	53	0.010	0.003	0.006	0.019
Fertility rate	49	3.865	1.228	1.460	6.520

* 1996 PPP US dollars; ** Pooled panel dataset, 1971-2000; *** 1981 constant rupees.

Table 2 - Correlation matrix among the main variables of interest
(Number of observations in parentheses; all correlations significant at the 5% level)

a. Cross-country Dataset (1960-2000 averages unless otherwise specified)

	Log income per capita	Growth income per capita	Adult mortality rate	Infant mortality	Crude death rate	Total fertility rate	Investment share of GDP
Income per capita growth	0.52 (98)	1.00 (98)					
Adult mortality rate	-0.78 (149)	-0.61 (95)	1 (160)				
Infant mortality	-0.83 (153)	-0.62 (97)	0.87 (155)	1 (169)			
Crude death rate	-0.71 (145)	-0.60 (95)	0.88 (151)	0.89 (155)	1 (163)		
Total Fertility Rate	-0.77 (146)	-0.66 (94)	0.80 (150)	0.87 (154)	0.70 (158)	1 (162)	
Investment share of GDP	0.53 (168)	0.64 (98)	-0.53 (149)	-0.57 (153)	-0.52 (145)	-0.61 (146)	1 (168)
Secondary school gross enroll. ratio	0.84 (109)	0.51 (89)	-0.82 (105)	-0.86 (108)	-0.69 (106)	-0.87 (105)	0.66 (109)

b. Indian States Dataset (pooled decade panel, 1971-2000)

	Log per capita NSDP	Growth of NSDP	Adult mortality rate	Infant mortality	Crude death rate
Growth of NSDP Per capita	0.270 (85)	1 (85)			
Adult mortality rate (age 20-40)	-0.407 (54)	-0.577 (47)	1 (54)		
Infant mortality	-0.625 (57)	-0.422 (49)	0.608 (52)	1 (57)	
Crude death rate	-0.672 (53)	-0.509 (47)	0.658 (53)	0.951 (51)	1 (53)
Fertility rate	-0.614 (49)	-0.406 (43)	0.408 (48)	0.847 (49)	0.796 (47)

Table 3 - OLS estimates of the growth equation, cross-country data.
Dependent Variable: Growth of per capita income, annual, 1960-2000

	(1)	(2)	(3)	(4)	(5)
Log income per capita, 1960	-0.762 (4.06)**	-1.132 (5.61)**	-1.227 (6.38)**	-1.313 (5.32)**	-1.379 (6.28)**
Adult mortality rate	**-10.054 (8.08)****	**-6.587 (3.65)****	**-1.783 (1.00)**	**-5.268 (2.38)****	**-2.941 (1.47)**
Crude death rate		85.530 (1.65)	-17.296 (0.38)	49.228 (0.89)	1.408 (0.03)
Infant mortality rate		-26.788 (5.00)**	-7.273 (1.25)	-16.612 (3.28)**	-6.677 (1.10)
Total fertility rate			-0.793 (5.04)**		-0.587 (3.83)**
Government consumption/GDP, %				-0.035 (2.21)**	-0.029 (1.82)*
Investment share of GDP, %				0.046 (2.32)**	0.032 (1.62)
Secondary school gross enrollment ratio				1.211 (1.04)	0.383 (0.32)
Imports plus exports over GDP, %				0.043 (3.45)**	0.028 (2.41)**
Log of population				0.359 (4.03)**	0.202 (2.20)**
Openness*log of population				-0.004 (2.64)**	-0.002 (1.52)
Constant	10.861 (6.00)**	13.483 (7.32)**	15.994 (8.80)**	9.899 (3.77)**	13.879 (5.51)**
Observations	95	94	93	86	85
Adjusted R-squared	0.46	0.56	0.67	0.68	0.72

Robust t-statistics in parentheses. * significant at 10%; ** significant at 5%.
All regressors appear as averages of available years over 1960-2000 except log per capita income (1960).

Table 4 - OLS growth estimates: extensions and robustness.
Dependent Variable: Growth of per capita income, annual, 1960-2000

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Africa Dummy	**Africa Dummy**	**Mid-Transition**	**War Deaths**	**War Variables**	**Institu-tions**	**Institu-tions**
Log income per capita, 1960	-1.014 (4.65)**	-1.179 (4.54)**	-1.605 (4.44)**	-1.258 (4.89)**	-1.297 (5.17)**	-1.364 (5.21)**	-1.080 (4.12)**
Adult mortality rate		**-6.531 (2.82)****	**-6.664 (2.48)****	**-4.543 (2.10)****	**-3.654 (1.47)**	**-4.880 (2.12)****	**-6.858 (3.18)****
Sub-Saharan Africa Dummy	**-1.015 (2.83)****	**0.224 (0.39)**					
Interstate battle deaths/population				1.972 (1.59)	2.129 (1.81)*		
Intrastate battle deaths/population				-0.034 (0.71)	-0.008 (0.15)		
Months of interstate War					0.005 (1.02)		
Months of intrastate War					-0.001 (1.87)*		
Risk of expropriation, 1990						0.259 (2.02)**	
Constraints on executive, 1970/1990 avg.							0.032 (0.34)
Crude death rate			66.373 (0.60)	43.296 (0.75)	47.197 (0.78)	60.663 (1.04)	142.022 (2.50)**
Infant mortality rate			-2.339 (0.18)	-15.731 (2.81)**	-19.073 (3.11)**	-15.205 (2.36)**	-23.381 (3.74)**
Total fertility rate			-0.720 (2.96)**				
Government Consumption / GDP, %	-0.034 (2.27)**	-0.035 (2.16)**	-0.064 (2.27)**	-0.042 (2.45)**	-0.047 (2.75)**	-0.029 (1.65)	-0.030 (1.72)*
Investment share of GDP, %	0.067 (3.36)**	0.056 (2.70)**	0.085 (3.26)**	0.046 (2.01)**	0.060 (3.29)**	0.042 (2.04)**	0.028 (1.49)
Secondary school gross enrollment ratio	3.763 (3.67)**	2.601 (2.68)**	-0.698 (0.44)	1.156 (0.93)	0.494 (0.42)	0.769 (0.66)	-0.322 (0.22)
(Imports+Exports)/, GDP %	0.044 (3.14)**	0.043 (3.75)**	0.049 (1.95)*	0.043 (3.25)**	0.043 (3.16)**	0.029 (3.37)**	0.074 (2.83)**
Log of population	0.331 (3.27)**	0.375 (4.02)**	0.233 (1.40)	0.369 (4.17)**	0.361 (4.01)**	0.271 (3.28)**	0.531 (3.61)**
Openness*log of Population	-0.004 (2.25)**	-0.003 (2.54)**	-0.003 (1.10)	-0.004 (2.41)**	-0.004 (2.50)**	-0.002 (2.25)**	-0.007 (2.44)**
Constant	4.825 (2.14)**	7.798 (2.81)**	15.533 (3.48)**	9.261 (3.50)**	9.683 (3.50)**	8.775 (3.35)**	6.533 (2.45)**
Observations	89	86	40	81	81	79	73
Adjusted R-squared	0.63	0.66	0.71	0.69	0.71	0.68	0.61

Robust t statistics in parentheses; * significant at 10%; ** significant at 5%
All regressors appear as averages of available years over 1960-2000 except log per capita income (1960).

Table 5 - Estimates of the growth regression, India Data, decade panel.
Dependent Variable: Growth of Net State Domestic Product per Capita, %, annual, 1970-2000

	Random Effects Estimator				Between Estimator			
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**
Log of per Capita Income in Period Initial Year	1.289 (2.21)**	1.750 (2.27)**	1.621 (2.09)**	0.282 (0.26)	0.792 (0.93)	1.914 (2.67)**	1.803 (2.58)**	0.613 (0.70)
Mortality rate, male, ages 20-40		**-67.917 (3.17)****	**-90.169 (3.55)****	**-82.327 (2.75)****		**-80.463 (3.16)****	**-107.691 (3.69)****	**-87.869 (2.21)***
Death rate, all ages		-0.055 (0.23)	0.059 (0.25)	0.281 (1.10)		-0.011 (0.03)	0.022 (0.07)	0.143 (0.39)
Infant mortality rate, per 1000 live births		0.006 (0.32)	0.015 (0.76)	-0.025 (0.97)		0.012 (0.50)	0.032 (1.39)	0.009 (0.21)
Total fertility rate, avg. of first 5 years of each decade			-0.567 (1.64)	0.041 (0.08)			-0.698 (2.01)*	0.023 (0.04)
Urbanization Rate, %				0.081 (1.87)*				0.076 (2.04)*
Population density, inhab/sq km				-0.002 (1.17)				-0.002 (1.00)
Religious Fractionalization				-2.911 (1.47)				-1.790 (1.03)
Share of scheduled castes and tribes, %				0.037 (1.13)				0.003 (0.09)
Literacy rate, %				0.041 (0.92)				0.046 (1.09)
Share of development expenditures in NSDP, %				-0.042 (0.50)				0.043 (0.44)
Constant	-7.384 (1.71)*	-6.218 (0.97)	-3.567 (0.54)	2.326 (0.27)	-3.666 (0.58)	-7.650 (1.26)	-4.315 (0.71)	-2.020 (0.28)
Observations	85	45	42	41	85	45	42	41
Number of States	27	19	18	18	27	19	18	18
R-squared					0.03	0.65	0.75	0.94

Absolute value of t statistics in parentheses. * significant at 10%; ** significant at 5%

Table 6 - OLS estimates of the channel equations.
(1990 values for all dependent variables)

	(1)	(2)	(3)	(4)	(5)	(6)
	Investment	**Investment**	**Enrollment**	**Enrollment**	**Fertility**	**Fertility**
Log income per capita, 1960	-0.105 (0.11)	-7.544 (4.13)**	0.121 (3.95)**	0.023 (0.62)	-0.349 (2.15)**	-0.196 (0.92)
Adult mortality rate	**-36.661** **(6.77)****	**-20.641** **(1.86)***	**-1.306** **(7.02)****	**-0.664** **(1.95)***	**9.894** **(9.81)****	**4.152** **(2.36)****
Crude death rate		-603.999 (1.37)		18.628 (1.98)*		51.721 (1.16)
Infant mortality rate		-16.396 (0.37)		-4.716 (4.69)**		10.821 (1.91)*
Secondary school gross enrollment ratio		-242.888 (4.64)**				-2.196 (2.11)**
Log per capita income * Enrollment		27.627 (4.84)**				
Democracy index				-0.008 (0.10)		
Urbanization rate, %				0.002 (1.54)		0.009 (1.44)
Population density				-0.054 (2.41)**		-0.330 (3.57)**
Constant	27.258 (2.96)**	87.996 (5.21)**	-0.005 (0.02)	0.571 (2.15)**	3.497 (2.22)**	2.838 (1.58)
Observations	106	92	94	93	104	91
Adjusted R-squared	0.37	0.56	0.74	0.81	0.80	0.86

Robust t statistics in parentheses; * significant at 10%; ** significant at 5%.
All regressors appear as averages of available years over 1960-2000 except log per capita income (1960).

Table 7 - OLS estimates of the AIDS equation, cross-country data.

	(1)	(2)	(3)	(4)	(5)	(6)
	Death Rate from AIDS, per 1,000, 2001			**% Adults (15-49) living with AIDS, end 2003**		
Log per capita income, 1990	0.252 (0.50)	0.177 (0.22)	0.920 (0.97)	1.357 (1.27)	1.104 (0.82)	2.663 (1.62)
Adult mortality rate, male, 1960-1980 avg.	**11.013 (2.45)****	**14.123 (2.25)****	**15.597 (2.12)****	**28.898 (3.09)****	**37.112 (2.70)****	**38.597 (2.15)****
Death rate, 1960-1980 avg.		-113.889 (0.70)	-334.768 (1.22)		-296.948 (1.00)	-824.568 (1.36)
Infant mortality rate, 1960-1980 avg.		-24.255 (2.31)**	1.588 (0.08)		-29.891 (1.76)*	41.562 (0.99)
Fertility rate, total 1990		0.714 (1.53)	0.504 (0.99)		0.953 (1.19)	0.762 (0.77)
Index of political Rights, 1990			1.303 (0.59)			2.576 (0.52)
Revolutions, 1980-1999 average			-1.502 (1.53)			-3.238 (1.60)
Population density, 1990			-0.723 (1.88)*			-1.488 (2.24)**
Dummy for Landlocked country			3.436 (2.06)**			6.075 (1.99)*
Dummy for island			-0.310 (0.51)			0.354 (0.29)
Log land area, square km			-0.078 (0.53)			-0.133 (0.43)
Dummy for Catholic Majority			-1.095 (1.11)			-2.036 (1.18)
Dummy for Muslim Majority			-2.392 (2.50)**			-6.294 (3.47)**
Distance from Equator			2.614 (0.88)			8.708 (1.23)
Ethnic Fractionalization			0.341 (0.21)			0.402 (0.11)
Number of years of schooling, 1990			-0.226 (0.87)			-0.546 (1.09)
Constant	-4.427 (0.79)	-3.940 (0.47)	-7.941 (1.05)	-17.989 (1.54)	-15.479 (1.13)	-25.305 (1.71)*
Observations	97	95	79	110	108	85
Adjusted R-squared	0.19	0.24	0.38	0.22	0.24	0.35

Robust t statistics in parentheses; * significant at 10%; ** significant at 5%.

Table 8 - OLS estimates of the Tobacco Consumption equation.
Dependent Variable: See below

	Adults smoking, %, male		Adults smoking, %, total		Cigarette consumption per capita, 1000s, 2002	
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Log income per capita, 1960	-0.034 (1.38)	-0.039 (1.08)	0.025 (1.36)	0.018 (0.59)	0.271 (2.72)**	-0.084 (0.62)
Adult mortality rate	**-0.000 (0.00)**	**0.805 (2.67)****	**0.103 (0.68)**	**0.330 (1.74)***	**-2.752 (3.97)****	**2.288 (1.87)***
Infant mortality rate		2.037 (1.67)*		1.541 (1.47)		0.857 (0.21)
Crude death rate		-24.894 (2.87)**		-8.856 (1.38)		-46.224 (1.42)
Urbanization rate, %		-0.000 (0.13)		0.000 (0.14)		0.004 (0.81)
Population density		-0.037 (1.54)		-0.028 (1.38)		-0.034 (0.30)
Total fertility rate		-0.083 (4.00)**		-0.043 (2.40)**		-0.389 (3.46)**
Distance from Equator		-0.015 (0.11)		0.012 (0.12)		1.477 (2.26)**
Dummy for Muslim Majority		0.082 (1.75)*		0.004 (0.11)		0.178 (1.21)
Dummy for Catholic Majority		0.039 (1.34)		0.041 (1.72)*		0.114 (0.85)
Constant	0.651 (2.69)**	0.931 (3.47)**	0.036 (0.19)	0.177 (0.78)	-0.190 (0.19)	2.530 (2.13)**
Observations	80	77	86	83	92	90
Adjusted R-squared	0.03	0.19	0.01	0.04	0.48	0.61

Robust t statistics in parentheses; * significant at 10%; ** significant at 5%.
All regressors except initial income 1960 appear as averages of available years over 1960-2000.

Table 9 – First Stage F-tests for alternative sets of instrumental variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	ME + Geography + Climate	**ME+ Climate**	**ME + Geography**	**Geogra-phy + Climate**	**ME + Geography + Climate + FR**	**ME + Climate + FR**	**ME + Geography + FR**	**Geography + Climate + FR**
F-test Degrees of Freedom	(17, 63)	(13, 68)	(5, 76)	(16, 64)	(19, 63)	(15, 67)	(7, 76)	(18, 64)
Adult Mortality								
Shea R^2	0.59	0.37	0.06	0.58	0.57	0.38	0.06	0.57
1st stage F-statistic	7.07	6.13	7.75	4.39	7.04	6.08	5.84	5.66
(p-value)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Crude Death Rate								
Shea R^2	0.53	0.45	0.21	0.52	0.56	0.49	0.20	0.56
1st stage F-statistic	5.10	6.29	7.75	3.53	6.80	7.31	7.77	5.10
(p-value)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Infant Mortality								
Shea R^2	0.48	0.43	0.03	0.42	0.46	0.41	0.03	0.40
1st stage F-statistic	3.81	4.27	4.30	2.26	3.81	4.80	3.84	2.45
(p-value)	(0.00)	(0.00)	(0.00)	(0.01)	(0.00)	(0.00)	(0.00)	(0.00)

(columns list alternative sets of instruments; rows refer to the endogenous variable being instrumented for).

Table 10 - IV Estimates of the Growth Regression

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	IV=ME + Geography + Climate	**IV=ME+ Climate**	**IV=ME + Geography**	**IV=Geogra-phy + Climate**	**IV=ME + Geography + Climate + FR**	**IV=ME + Climate + FR**	**IV=ME + Geography + FR**	**IV=Geogra-phy + Climate + FR**
Log income per capita, 1960	-1.129 (3.72)**	-1.183 (4.37)**	-0.889 (1.56)	-1.137 (3.24)**	-0.925 (3.14)**	-0.960 (3.11)**	-0.881 (1.36)	-0.944 (2.90)**
Adult mortality rate, male	**-8.564 (3.84)****	**-10.531 (4.04)****	**-21.303 (2.00)****	**-9.196 (3.86)****	**-9.283 (4.16)****	**-10.009 (3.78)****	**-23.161 (1.92)***	**-9.626 (4.19)****
Crude death rate	145.765 (2.25)**	193.941 (2.62)**	76.898 (0.44)	122.667 (1.65)*	132.038 (1.87)*	151.085 (1.96)*	65.042 (0.33)	127.926 (1.68)*
Infant mortality rate	-31.644 (4.57)**	-34.837 (4.05)**	28.666 (0.59)	-39.021 (3.99)**	-27.298 (3.83)**	-30.091 (3.65)**	32.725 (0.61)	-33.807 (3.60)**
Democracy index	1.071 (1.37)	1.347 (1.79)*	0.033 (0.02)	1.902 (1.74)*	1.336 (1.60)	1.531 (1.75)*	0.221 (0.16)	1.901 (1.83)*
Urbanization rate, %	0.002 (0.17)	-0.001 (0.07)	0.007 (0.35)	-0.009 (0.77)	-0.003 (0.29)	-0.005 (0.45)	0.007 (0.33)	-0.010 (0.89)
Population density	-0.155 (0.13)	-0.320 (0.25)	0.417 (0.21)	-0.136 (0.11)	-0.499 (0.38)	-0.513 (0.38)	1.594 (0.57)	-0.572 (0.45)
(Imports+Exports) /GDP, %	0.058 (2.35)**	0.057 (3.53)**	0.025 (1.36)	0.062 (2.14)**	0.116 (3.69)**	0.113 (3.58)**	0.037 (3.08)**	0.118 (3.37)**
Log of population	0.482 (3.48)**	0.472 (4.05)**	0.337 (2.15)**	0.441 (2.62)**	0.842 (3.96)**	0.810 (3.83)**	0.244 (1.44)	0.819 (3.50)**
Openness*log of population	-0.005 (2.00)**	-0.005 (2.91)**	0.0001 (0.04)	-0.006 (1.97)**	-0.010 (3.20)**	-0.010 (3.14)**	-0.004 (1.36)	-0.011 (2.98)**
Constant	7.937 (2.27)**	8.672 (3.04)**	7.218 (1.80)*	9.704 (2.39)**	2.553 (0.65)	3.400 (0.85)	9.415 (2.23)**	3.707 (0.85)
Observations	88	89	89	88	88	88	89	88
Hansen J statistic (p-value)	13.551 (0.48)	12.018 (0.28)	0.921 (0.63)	10.046 (0.69)	12.764 (0.55)	10.887 (0.37)	0.164 (0.92)	9.821 (0.71)

Robust t statistics in parentheses; * significant at 10%; ** significant at 5%

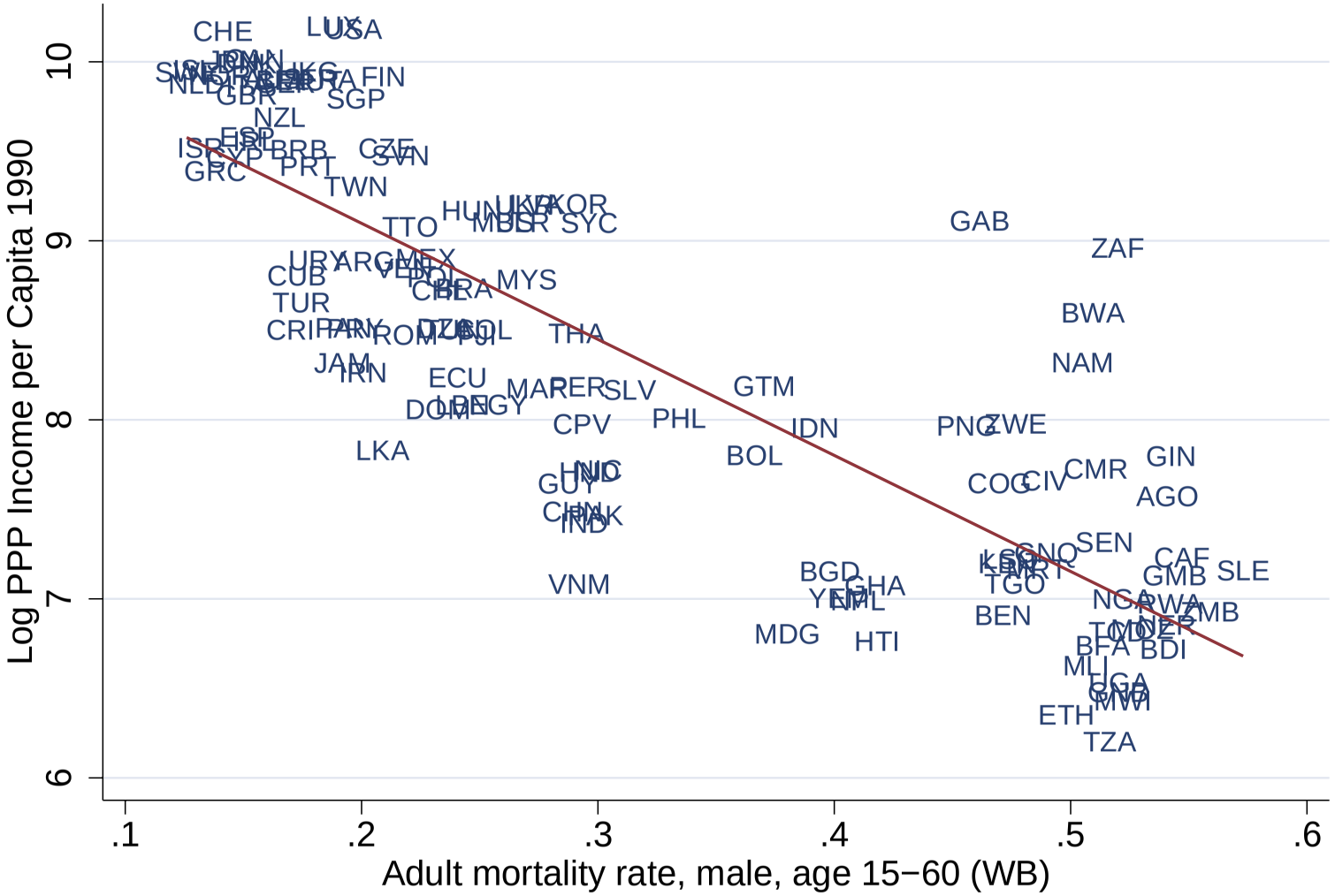
All regressors except initial income 1960 appear as averages of available years over 1960-2000.

Table 11 – System estimates of the Adult Mortality Effect (3SLS)

	3SLS Baseline Model		
	Effect of channel on growth	**Effect of adult mortality on channel**	**Effect of adult mortality on growth**
Investment Effect	0.183	-20.848	-3.823
t/Wald test statistic	5.59	1.97	3.62
p-value	(0.000)	(0.049)	(0.058)
Enrollment Effect	-2.840	-0.509	1.444
t/Wald test statistic	1.82	2.84	2.35
p-value	(0.070)	(0.005)	(0.126)
Fertility Effect	-0.681	5.681	-3.868
t/Wald test statistic	3.36	4.72	8.20
p-value	(0.001)	(0.000)	(0.004)
Total Effect			**-6.247**
Total Effect (1 s.d)			**-0.850**
Wald test statistic			7.08
p-value			(0.008)

Columns 1, 2: Wald test statistic has (1, 372) degrees of freedom.
Columns 3, 4: Wald test statistics has (1, 352) degrees of freedom.
t-statistics appear for the effect of the channels on growth and the effect of adult mortality on the channels.
Wald statistics appear for the effect of adult mortality on growth.
Instruments for adult mortality in the channel regressions: Malaria ecology, climate variables, geography variables, as defined in text.

Figure 1 − Log Income Per Capita 1990 and Adult Mortality



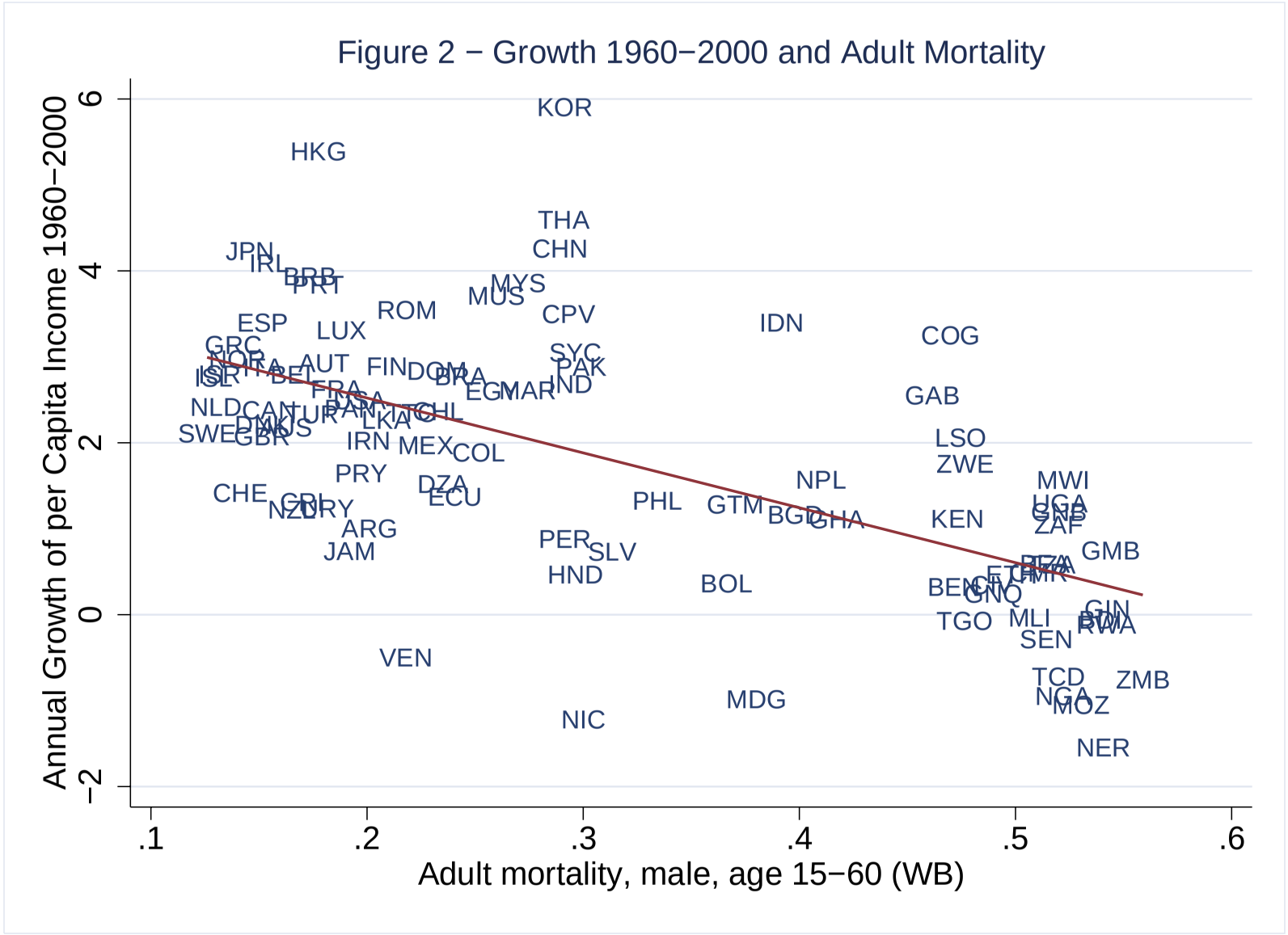


Figure 2 − Growth 1960−2000 and Adult Mortality
Annual Growth of per Capita Income 1960−2000
6
4
2
0
−2
.1
.2
.3
.4
.5
.6
Adult mortality, male, age 15−60 (WB)
KOR
HKG
THA
CHN
JPN
IRL
PRT
MYS
MUS
ROM
CPV
IDN
COG
ESP
LUX
SYC
AUT
FIN
PAK
IND
BRA
EGY
MAR
GAB
NLD
CAN
CHL
LKA
LSO
ZWE
SWE
GBR
IRN
MEX
COL
NPL
MWI
PRY
DZA
ECU
CHE
PHL
GTM
BGD
GHA
KEN
ZAF
GMB
ARG
JAM
PER
SLV
HND
BOL
GNQ
TGO
MLI
SEN
VEN
TCD
ZMB
MDG
NGA
MOZ
NIC
NER

Figure 3 − Investment Rate 1990 and Adult Mortality

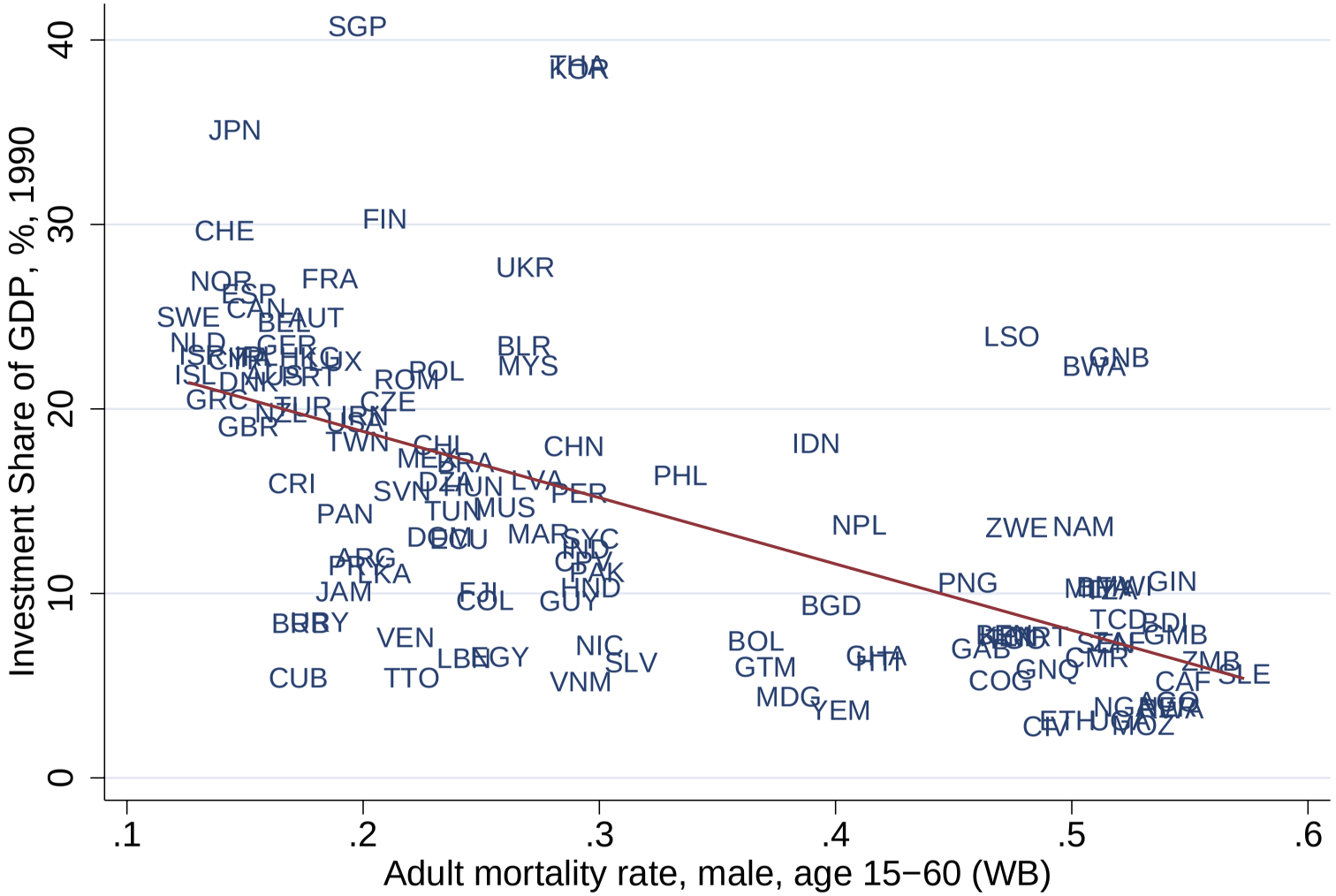
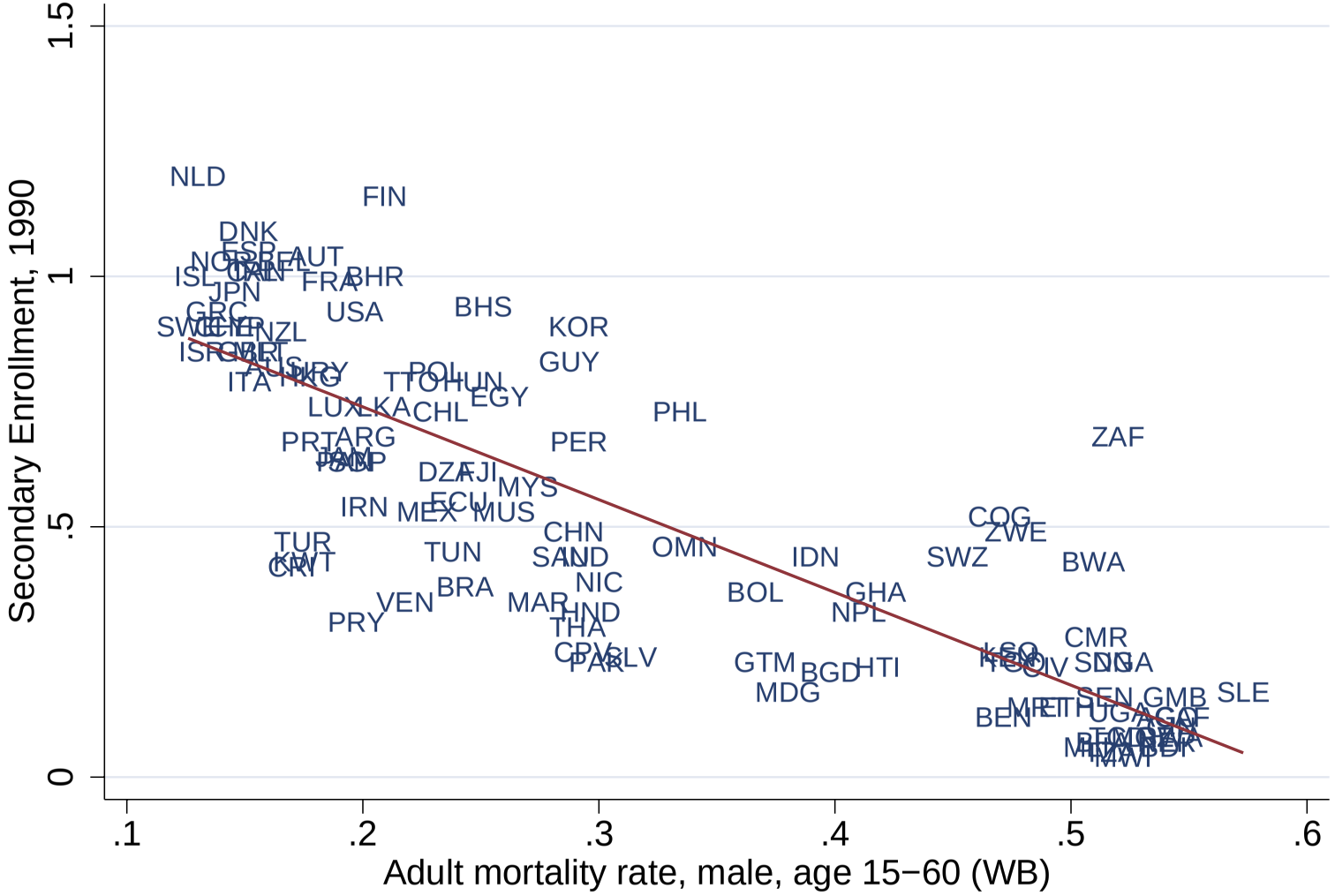


Figure 4 − Enrollment Rate 1990 and Adult Mortality



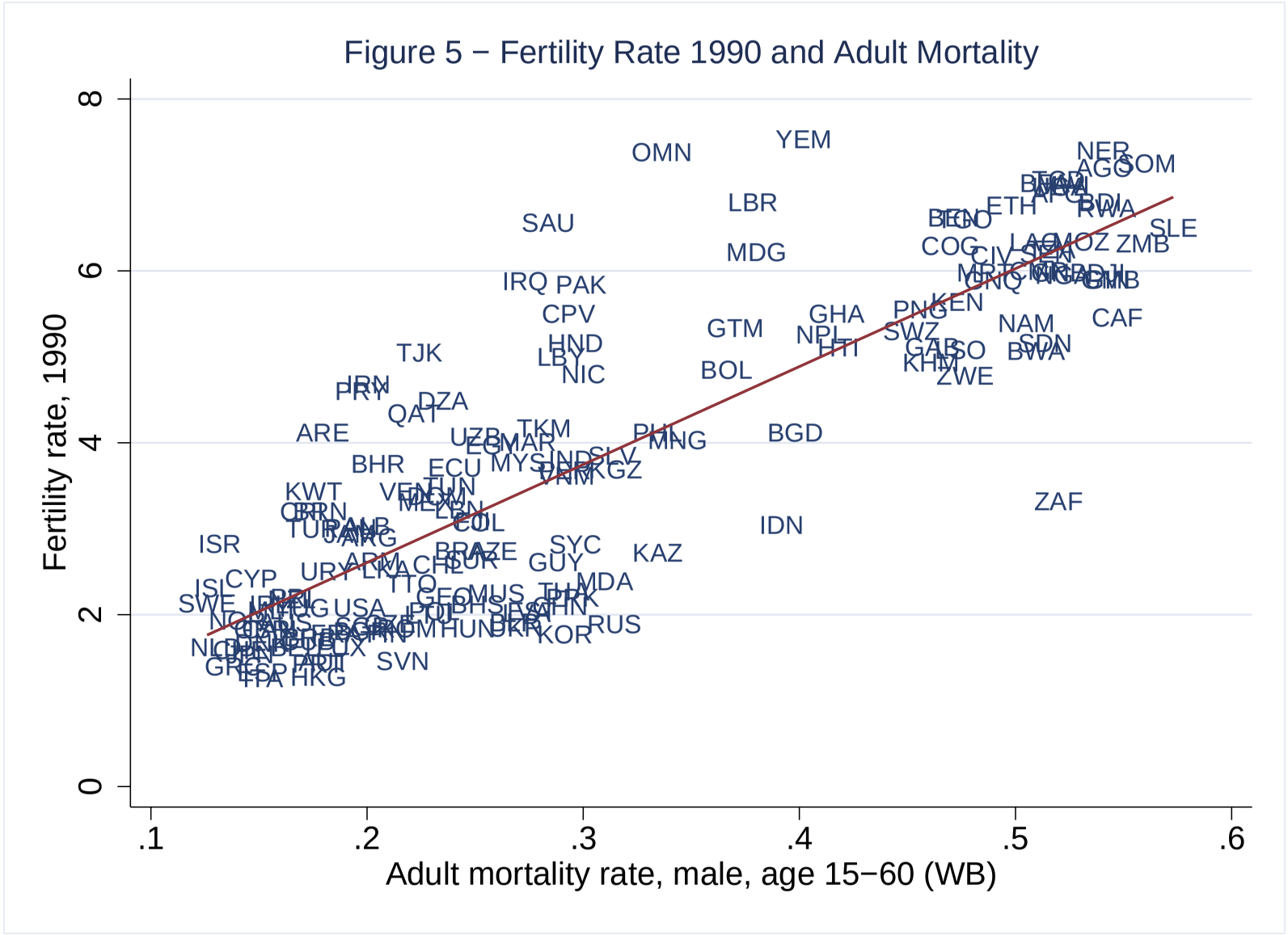


Figure 5 − Fertility Rate 1990 and Adult Mortality
Fertility rate, 1990
0
2
4
6
8
.1
.2
.3
.4
.5
.6
Adult mortality rate, male, age 15−60 (WB)
OMN
YEM
NER
SOM
AGO
LBR
ETH
RWA
SLE
SAU
MDG
COG
ZMB
IRQ
PAK
CPV
GHA
PNG
KEN
CAF
GTM
NPL
SWZ
NAM
SDN
TJK
HND
LBY
HTI
BWA
NIC
BOL
ZWE
DZA
QAT
ARE
TKM
BGD
MNG
BHR
ECU
KWT
ZAF
IDN
ISR
SYC
KAZ
GUY
CYP
MDA
USA
RUS
KOR
SVN
HKG