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Death and development

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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. Paying careful attention to the age pattern of mortality and to endogeneity issues, we find that a greater risk of death during the prime productive years is associated with higher levels of risky behavior, higher fertility, and lower investment in physical capital, even when controlling for infant mortality. In our regressions, adult mortality explains almost all of Africa's growth tragedy. This analysis underscores grim forecasts of the long-run economic costs of the ongoing AIDS epidemic.

Keywords Fertility · Growth · Human capital · Investment · Mortality

JEL Classifications I10 · J10 · O10

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

Most of the work for this paper was completed prior to the tragic passing away of our dear friend and colleague John McMillan in March of 2007.

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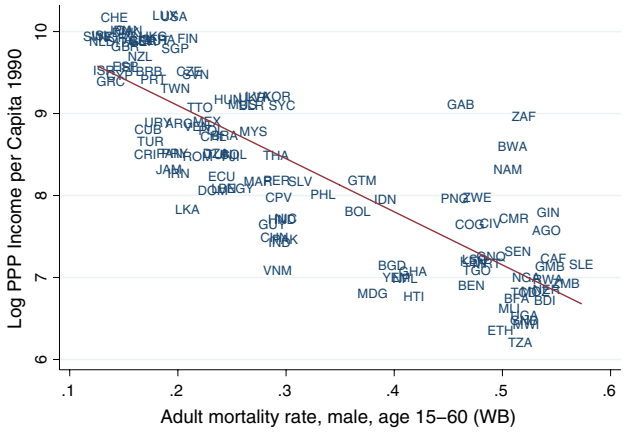

Fig. 1 Log income per capita 1990 and adult mortality

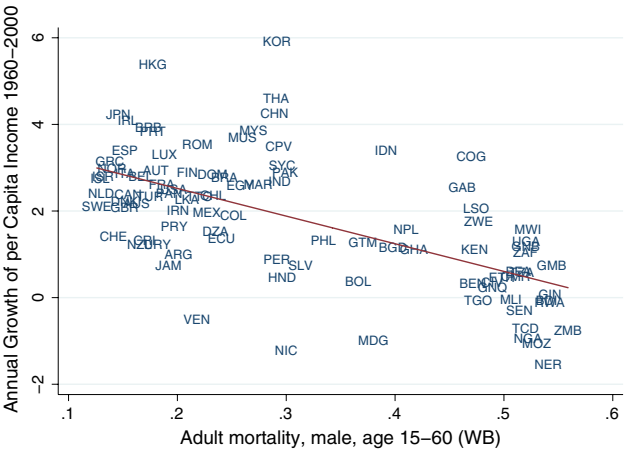

Fig. 2 Growth 1960–2000 and adult mortality

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 bidirectional. 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. Indeed, 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 inhibits the growth that would help countries escape poverty.

Does adult mortality affect income and economic growth? Confirming our observation from Fig. 1, a glance at the raw adult mortality data shows that, as expected, mortality varies with per capita income (Appendix 1, Table A1). The safest country in the post-1960 period has been Sweden, where a 15-year-old's probability of dying before reaching sixty is only 13 percent. The worst has been Sierra Leone, where a 15-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 15–18 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.

The estimated effects of high adult mortality on 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 40-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.

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 20-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.

This paper moves beyond these basic findings to consider *how* mortality affects growth, examining several possible causal channels implied by theory. People who expect to die young will fail to take actions, such as saving and educating themselves or their children, that generate an uncertain long-term benefit at a short-term cost. 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. Figure 5 displays a strong positive relationship between the total fertility rate and adult mortality. Estimating a system of equations, we find that adult mortality's effect on economic growth appears to act primarily through the channels of physical capital investment and fertility.

Mortality is of course highly correlated with non-fatal illness which can reduce total factor productivity: illness may affect growth directly by reducing people's ability to work effectively, even if they do not die from a particular disease. Like the rest of the literature on this topic, this paper does not precisely quantify the productivity-reducing effect of poor health as distinct from the horizon effect of mortality. What it does provide is evidence that incentive effects stemming from the horizon effect are substantial and have a major impact on long-run growth.

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

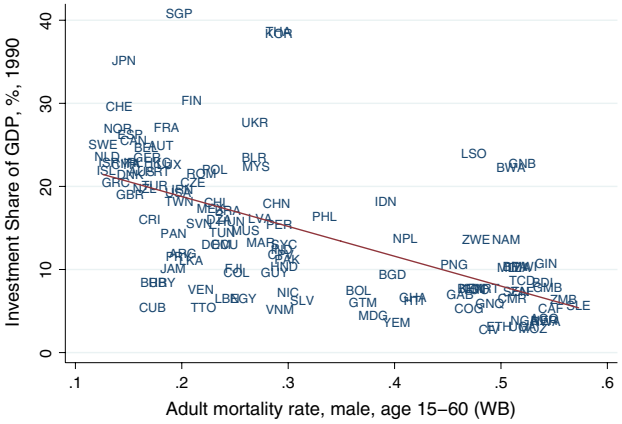

Fig. 3 Investment rate 1990 and adult mortality

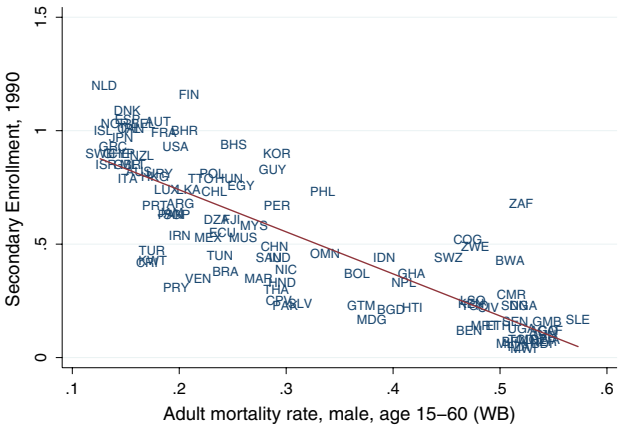

Fig. 4 Enrollment rate 1990 and adult mortality

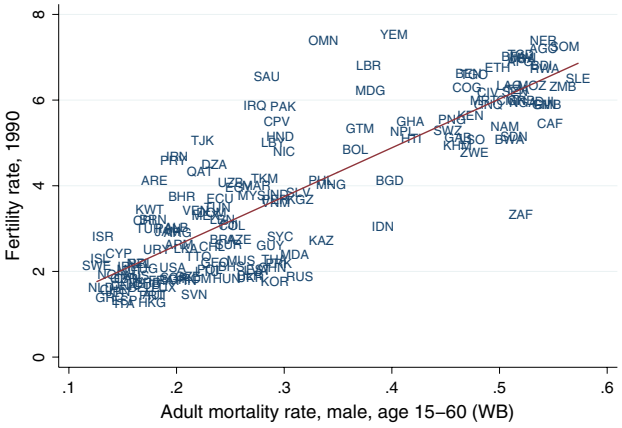

Fig. 5 Fertility rate 1990 and adult mortality

Empirical research on mortality and growth has typically ignored the distinction between adult mortality, infant mortality and life expectancy, treating these measures as interchangeable.[2] In this paper we focus particularly on the effects of adult mortality, the probability of a young adult living long enough to reap the gains of long-term investments. Although infant and adult mortality are strongly correlated, the channels by which each might influence economic growth are theoretically distinct, making it informative to distinguish between 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 when 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, a better understanding of the economic consequences of mortality at different ages may influence how policymakers choose to allocate scarce health resources.

By examining the mechanisms through which death rates affect growth and by considering the age pattern of mortality, our paper attempts a comprehensive cross-national assessment of the effect of death on development. In Sect. 2, we provide further motivation for the incentive effects of adult mortality. In Sect. 3, we review conceptual arguments linking mortality to investment, human capital accumulation, fertility and ultimately economic growth. In Sect. 4, we briefly present simple cross-sectional regressions for the determination of growth as a function of various mortality indicators, for both the world and India samples. In Sect. 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 Behavioral consequences of adult mortality

We begin, by way of motivation, with a digression. This paper argues that overall mortality affects aggregate economic growth because people who expect to die young fail to take actions with short-term costs and long-term benefits. Some corroborative evidence, however, comes from the converse: high mortality induces actions with short-term benefits at long-term costs. A high death rate from exogenous causes could lead to a high death rate from endogenous causes. The prospect of early death brings shortsighted behavior.

Anecdotal evidence comes from the aftermath of the Chernobyl nuclear accident. The initial predictions that those living near Chernobyl would die in large numbers from radiation-induced cancer have turned out to be overstated. The dire predictions of high death rates have nevertheless affected behavior, according to a United Nations report. Perceiving themselves as "helpless, weak and lacking control over their future," people have engaged

[2] See Bloom et al. (2004) for a useful summary of much of this literature as well as further results and Weil (2007) for an innovative recent contribution.

in "reckless conduct, such as consumption of mushrooms, berries and game from areas still designated as highly contaminated, overuse of alcohol and tobacco, and unprotected promiscuous sexual activity" (WHO 2005).

Further evidence of this relationship between high mortality and short-term-oriented behavior comes from the cross-country patterns of AIDS. If high adult mortality leads to shortsighted behavior, we would expect it to be associated with the spread of AIDS. Is the current prevalence of AIDS partly a function of pre-AIDS mortality rates? The results of OLS regressions for the determination of the AIDS death rate, displayed in the first two columns of Table 1, suggest this is the case. We find statistically significant evidence that adult mortality over 1960–1980 is positively associated with the death rate from AIDS in 2001 across all specifications. The estimated effect is large: using the estimates in column 2 of Table 1, a one standard deviation increase in 1960–1980 adult mortality (equal to 0.09) is associated with a 0.81 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 half 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 two columns of Table 1. The statistical significance of adult mortality is even stronger than before: adult mortality in 1960–1980 is consistently positively related to the prevalence of AIDS in the adult population in 2003. Using the estimates in column 4, a one standard deviation increase in adult mortality is associated with a 2.03 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 economically large. These regressions provide preliminary evidence that high adult mortality is associated with behavior characterized by short-term benefits and long-term costs.

Finally, in the working paper version of this study (Lorentzen et al. 2005), we presented regressions linking the prevalence of smoking to adult mortality. Smoking is quintessentially an activity with short-term benefits and long-term costs, so the propensity to smoke should be related to agent's time horizons. We indeed uncovered a positive partial correlation between the prevalence of smoking in 2002 (measured either by the share of adults smoking or cigarette consumption per capita) and adult mortality, controlling for a wide array of determinants of smoking. This was suggestive of a horizon effect, but since reverse causality is a priori more serious here than in some of our other regressions (according to Mackay and Eriksen (2002), in 2002 4.2 million people died worldwide of tobacco-related causes) we do not dwell upon these results here.[3]

[3] The empirical estimates are available upon request.

Table 1 OLS estimates of the AIDS equation (Dependent variable: As in second row)

	Death rate from AIDS per 1,000, 2001 (1)	Death rate from AIDS per 1,000, 2001 (2)	Adults living with AIDS, end 2003 (3)	Adults living with AIDS, end 2003 (4)
Log per capita income, 1990	0.252 (0.50)	0.977 (0.97)	1.357 (1.27)	2.779 (1.53)
Adult mortality rate, male, 1960–1980 avg.	11.013 (2.45)**	8.966 (2.30)**	28.898 (3.09)**	22.555 (2.24)**
Infant mortality rate, 1960–1980 avg.		−14.687 (0.89)		−2.375 (0.08)
Fertility rate, total, 1990		0.282 (0.68)		0.304 (0.35)
Freedom House index of political rights, 0-1, 1990		1.577 (0.75)		3.473 (0.73)
Revolutions, attempted or successful, 1980–1999 average		−1.375 (1.52)		−3.058 (1.56)
Population density, 1000 inhabitants per sq km, 1990		−0.715 (1.84)*		−1.512 (2.20)**
Dummy = 1 if country is landlocked		3.248 (1.99)*		5.409 (1.86)*
Dummy = 1 if the country is an island or on an island		−0.302 (0.50)		0.245 (0.22)
Log of area in Km2		−0.001 (0.01)		0.014 (0.04)
Dummy for Catholic majority		−1.155 (1.10)		−2.043 (1.15)
Dummy for Muslim majority		−2.795 (2.77)**		−7.008 (3.48)**
Distance from Equator		−0.834 (0.27)		0.220 (0.04)
Ethnic fractionalization		−0.327 (0.21)		−1.386 (0.40)
Number of years of primary, secondary, higher schooling,1990		−0.247 (0.91)		−0.548 (1.09)
Constant	−4.427 (0.79)	−7.785 (1.00)	−17.989 (1.54)	−24.909 (1.59)
Observations	97	79	110	85
Adjusted R^2	0.19	0.37	0.22	0.33

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

3 How mortality affects growth

3.1 Physical investment and human capital accumulation

Our main hypothesis is that mortality affects growth by diminishing incentives for behavior with short-run costs and long-run payoffs. 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.

While this framework applies most easily to physical capital investment, it can be applied quite readily to human capital accumulation as well, an insight that goes back at least to Ben-Porath (1967). As the returns to human capital accrue over much of adult life, a high incidence of adult mortality may reduce incentives to obtain an education or accumulate other skills. 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. In either case, we would expect adult mortality rather than early childhood mortality to affect accumulation most, as decisions about physical and human capital accumulation are made primarily on the basis of returns they will yield in adulthood. The robustness of this theoretical relationship has been reaffirmed over the years in models with a variety of different specifications and assumptions (Ehrlich and Lui 1991, Kalemli-Ozcan et al. 2000, Kalemli-Ozcan 2002, 2003, Cervellati and Sunde 2005, Soares 2005, Chakraborty 2004, Chakraborty et al. 2007).

3.2 Fertility behavior

Mortality might also affect growth through fertility. The first order effect of mortality is on total fertility, the number of children born: As noted in Galor and Weil (1999), "If households care about their number of surviving children, and if they have a target number of survivors, then a reduction in mortality will mechanically lead to a corresponding reduction in fertility". If this were the only factor, then changes in mortality would only affect total fertility, not net fertility—the number of surviving children. However, such a logic does not explain the strong correlation between mortality rates and net fertility that has also been documented.

To understand the relationship between mortality and fertility, two further effects must be considered, each of which will ultimately affect growth. First, if fertility and mortality are stochastic from the individual's perspective and offspring are difficult or impossible to replace, there may be a precautionary demand for children (Kalemli-Ozcan 2003). That is, in an environment of high uncertainty, parents will bear more children than they would prefer in order to minimize the risk of ending up with too few surviving descendants. This precautionary or "hoarding" motive should not have a strong effect, since deceased children can be replaced after the fact (Doepke 2005, Galor 2005). However, adult children are difficult or impossible for their parents to replace, so a precuationary motive stemming from high adult mortality is much more plausible. This results in higher net fertility and therefore higher population growth. The further link to growth comes about because a higher rate of population growth reduces the capital-labor ratio and the growth rate, as in the canonical Solow model.

A second channel from mortality to fertility to growth is related to human capital accumulation. Fertility decisions are inextricably linked to human capital investments, through the quantity-quality trade-off first introduced by Becker (1960). As noted above, higher adult mortality reduces the expected payoff from investing in human capital. This shifts the balance from quantity toward quality (Kalemli-Ozcan 2003, Soares 2005). In contrast, infant and child mortality happens when children can still be replaced but before most human capital investment occurs. Higher child mortality might actually favor greater investments in education because it makes it more costly to raise a surviving child to the age at which education begins (Doepke 2005, Azarnert 2006).

In sum, the recent theoretical literature has clarified the links between mortality and fertility. The literature highlights the crucial distinction between infant (or child) mortality and adult mortality, which directly supports the empirical distinction made in the present paper.

3.3 Alternative views

The negative relationship between mortality and income has not gone unchallenged. Despite the dramatic absolute cross-country convergence in life expectancy, absolute convergence in per capita income has not occurred (as highlighted in Becker et al. 2005). This is inconsistent with a simplistic view that life expectancy is the sole determinant of economic growth, but lifetime horizons could still have an important effect on growth and income if some other factors are responsible for divergence in income. Two recent studies have gone further to argue that increased mortality has had no impact on economic growth or that this effect may even be positive.

Acemoglu and Johnson (2006) examine the relationship between life expectancy and per capita income by exploiting within-country variation in post-1940 data. Instrumenting for changes in life expectancy with the shocks to mortality resulting from the introduction of new health technologies, they find that increases in life expectancy had either an insignificant or a small negative effect on per capita GDP. A major difference with our approach is that they focused on life expectancy, which is driven largely by infant mortality, rather than distinguishing between infant and adult mortality as we do. Theoretically, adult mortality should be expected to bear a stronger effect on investment and human capital accumulation than infant mortality, a hypothesis confirmed by our findings.[4]

Young (2005) comes to an even stronger conclusion: a mortality shock can improve per capita GDP. Using a calibrated simulation for South Africa, he predicts 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 study 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. However, empirical research by Kalemli-Ozcan (2007) has found that AIDS led to higher fertility and lower school enrollments in a panel of African countries from 1985–2000.

[4] There are two further sources for the differences between their findings and ours. First, using fixed effects in the presence of measurement error tends to bias estimates toward zero (Barro 1997, Hauk and Wacziarg 2004). Life expectancy data is subject to measurement error, resulting in likely downward bias in OLS estimates. Second, Acemoglu and Johnson (2006), were unable to include Africa in their baseline analysis due to lack of data. Although our results do not hinge on the inclusion of Africa in the sample, Africa is an important source of variation in the data and its inclusion in the Acemoglu-Johnson sample might have led them to different conclusions.

Table 2 Summary statistics for the main variables of interest

Variable	Obs.	Mean	Std. Dev.	Min	Max
a. Cross-country dataset					
Log income per capita, 1960	110	7.730	0.889	5.944	9.614
Income per capita, 1960	110	3359	3177	382	14978
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

4 Mortality and growth: exploratory correlations

4.1 Descriptive statistics

In this subsection, we discuss summary statistics that can be used to assess the magnitude of estimated effects presented below. Our data sources and further details on the construction of mortality series are described in Appendix 1. Table 2a provides the means and standard deviations of the main variables of interest in the cross-country dataset. As noted in the introduction, the demographic variables, averaged over 1960–2000, display substantial variation. Table 3a shows that the correlations among the various measures of mortality are high: the correlation between infant mortality and adult mortality, for instance, is 0.87, suggesting the potential for multicollinearity. The fertility rate is also highly correlated with various measures of mortality, an issue we reexamine below.

The corresponding summary statistics for the India dataset are presented in Tables 2b and 3b. Correlations are again quite high among 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. In the Indian dataset our measure of adult mortality refers to the probability of dying by age 40 conditional on reaching 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.[5] Obviously, the average probability of dying is much lower for the shorter 20–40 age range than it is for the 15–60 range—life expectancy at birth in 2000 in India was 62 years.

[5] 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.

Table 3 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 1960	Growth income per capita	Adult mortality rate	Infant mortality	Crude death rate	Total fertility rate	Investment share of GDP
Income per capita growth	0.23 (98)	1 (98)					
Adult mortality rate	−0.70 (106)	−0.61 (95)	1 (160)				
Infant mortality	−0.72 (108)	−0.62 (97)	0.87 (155)	1 (169)			
Crude death rate	−0.59 (106)	−0.60 (95)	0.88 (151)	0.89 (155)	1 (163)		
Total fertility rate	−0.74 (105)	−0.66 (94)	0.80 (150)	0.87 (154)	0.70 (158)	1 (162)	
Investment share of GDP	0.48 (110)	0.64 (98)	−0.53 (149)	−0.57 (153)	−0.52 (145)	−0.61 (146)	1 (168)
Secondary school gross enroll. ratio	0.78 (97)	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)

4.2 OLS growth estimates in the world cross-section

The first step in our empirical analysis is to examine the partial correlations between growth of income per capita and various mortality measures. We will refrain from interpreting these partial correlations causally, recognizing that causality might run both ways, biasing the OLS coefficient on mortality away from zero. Instead, we focus on whether the partial correlations yield magnitudes big enough for our story to have any potential to account for a large portion of cross-country differences in economic performance. 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.[6] Table 4 displays the results.

The first lesson from this exercise is that adult mortality is a very significant predictor of growth when entered alone with the log of initial per capita income and the infant mortality rate (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 0.55 simply by adding the mortality rates, suggesting that a large portion of the cross-country variation in economic growth might be attributable to these variables. The R-squared rises to 0.46 when introducing adult mortality alone in the regression. Furthermore, despite the 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.[7]

In column 2, we add a number of other control variables. We follow the baseline growth specifications in Barro and Sala-i-Martin (1996) and Alesina et al. (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 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. When we remove the mortality variables from this regression, its adjusted R-squared falls from 0.69 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.

The magnitude of the partial correlation between adult mortality and growth is substantial. Using estimates in the regression of column 2, a one standard deviation in adult mortality (equal to 0.136) is associated with a 0.58 percentage point difference in growth. Moving from the 75th percentile of adult mortality (Cambodia) to the 25th percentile (the USA) brings an extra 1.12 percentage points of growth holding constant the included determinants of growth.

One variable that does reduce the significance of adult mortality is the fertility rate. 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—its p-value is now 5.1%. This result suggests that the interplay between fertility and adult mortality may be an important channel whereby adult mortality could indirectly affect

[6] 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.

[7] The coefficient on adult mortality is also robust to the inclusion of the crude death rate in the regression. Similar results are also obtained when infant mortality is replaced with the child mortality rate. These results are available upon request.

Table 4 OLS estimates of the reduced form growth equation, cross-country data (Dependent variable): Growth of per capita income, annual, 1960–2000

	Without controls (1)	With controls (2)	Fertility control (3)	Africa dummy (4)	Africa dummy (5)	Africa dummy (6)
Log income per capita, 1960	−1.030 (5.30)**	−1.289 (5.41)**	−1.378 (6.31)**	−1.014 (4.65)**	−1.179 (4.54)**	−1.293 (5.39)**
Adult mortality rate	−5.060 (3.67)**	−4.248 (2.68)**	−2.910 (1.99)*		−6.531 (2.82)**	−4.791 (2.05)**
Infant mortality rate	−20.850 (4.58)**	−11.865 (3.01)**	−6.524 (1.55)			−11.765 (2.96)**
Government consumption share of GDP, %		−0.036 (2.26)**	−0.029 (1.83)*	−0.034 (2.27)**	−0.035 (2.16)**	−0.035 (2.26)**
Investment share of GDP, %		0.048 (2.43)**	0.032 (1.63)	0.067 (3.36)**	0.056 (2.70)**	0.047 (2.33)**
Secondary school gross enrollment ratio		1.810 (1.78)*	0.398 (0.38)	3.763 (3.67)**	2.601 (2.68)**	1.761 (1.71)*
(Imports + Exports)/GDP, %		0.044 (3.81)**	0.028 (2.45)**	0.044 (3.14)**	0.043 (3.75)**	0.044 (3.86)**
Log of population		0.355 (4.27)**	0.202 (2.28)**	0.331 (3.27)**	0.375 (4.02)**	0.362 (4.10)**
Openness * log of population		−0.004 (2.90)**	−0.002 (1.54)	−0.004 (2.25)**	−0.003 (2.54)**	−0.004 (2.93)**
Total fertility rate			−0.588 (4.01)**			
Sub-Saharan Africa dummy				−1.015 (2.83)**	0.224 (0.39)	0.152 (0.29)
Constant	12.889 (7.08)**	9.540 (3.79)**	13.876 (5.53)**	4.825 (2.14)**	7.798 (2.81)**	9.631 (3.81)**
Observations	94	86	85	89	86	86
Adjusted R^2	0.55	0.69	0.73	0.63	0.66	0.68

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)

economic growth. We will return to the empirical relationship between adult mortality and fertility when we explore these channels in Sect. 5.6.[8]

As a final assessment of the magnitude of the effect of adult mortality, we examine to what extent it can account for Africa's growth shortfall. Most of the world's high-mortality countries are in Sub-Saharan Africa. Column 4 excludes the mortality variables but adds a dummy variable for Sub-Saharan Africa to the specification with controls. The coefficient on this dummy takes on the conventional value of −1, Africa's "missing growth".[9] Adding adult mortality, the estimate for the Africa dummy switches signs and becomes statistically indistinguishable from zero (column 5). The same conclusion holds when both mortality measures are included (column 6). Thus, variation in adult mortality has the potential to account quantitatively for all of the growth shortfall experienced in Sub-Saharan Africa between 1960 and 2000.

4.3 Mortality and growth across states of India

As a check on robustness, we attempt to approximate the same growth specifications using a data on Indian states. Within-country data may exhibit less extraneous variation (such as that due to institutions or culture) than cross-country data. We use Indian data rather than, say, US state data or European regional data because the effect of mortality can be expected to be stronger where death is a more common occurrence. For India, 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 regressions exploiting the availability of data at the decade level. The latter are likely to deliver more reliable results given that all regressions involving our mortality measures cover at most 19 states, and we are able to obtain up to 45 observations when exploiting the panel dimension. The data cover the period 1970–2000, with one observation per decade.

Results across Indian states, displayed in Table 5, closely mirror those obtained using the world cross-section.[10] 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.[11] 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

[8] 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 error, as they surely are, these high correlations make it difficult to tell which one dominates statistically. As discussed in Appendix 1, measurement error for adult mortality is likely to be worse than that for fertility in developing countries, likely increasing the estimated coefficient of fertility at the expense of adult mortality.

[9] 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.

[10] 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.

[11] This has been observed previously by Ghosh et al. (1998), among others.

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	Fixed effects estimator
	(1)	(2)	(3)	(4)	(5)	(6)
Log of per capita income in period initial year	1.289 (2.21)**	1.812 (2.54)**	1.561 (2.14)**	−0.001 (0.00)	0.663 (0.81)	−9.139 (1.75)
Mortality rate, male, ages 20–40		−69.883 (3.59)**	−87.455 (3.88)**	−68.820 (2.51)**	−84.099 (2.32)*	−88.054 (1.91)*
Infant mortality rate, per 1000 live births		2.057 (0.25)	19.098 (1.38)	−4.441 (0.25)	22.887 (1.12)	−35.046 (1.21)
Total fertility rate, avg. of first 5 years of each decade			−0.550 (1.65)*	0.146 (0.29)	−0.018 (0.03)	0.582 (0.31)
Urbanization rate, %				0.081 (1.87)*	0.075 (2.15)*	0.177 (0.54)
Population density, inhab/sq km				−0.002 (1.15)	−0.001 (0.99)	−0.011 (0.79)
Religious fractionalization				−2.312 (1.21)	−1.619 (1.02)	−33.319 (0.83)
Share of scheduled castes and tribes, %				0.029 (0.91)	−0.006 (0.24)	0.076 (0.25)
Literacy rate, %				0.046 (1.05)	0.043 (1.10)	0.513 (1.54)
Share of development expenditures in NSDP, %				−0.030 (0.35)	0.069 (1.02)	−0.754 (2.58)**
Constant	−7.384 (1.71)*	−6.819 (1.18)	−3.035 (0.50)	4.004 (0.47)	−2.260 (0.33)	73.289 (1.57)
Observations	85	45	42	41	41	41
Number of States	27	19	18	18	18	18
R^2					0.93	0.72

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

of a broad range of controls, including other death rates (infant mortality as in column 2, but also 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 (column 4). The effect of adult mortality is also robust to the inclusion of the fertility rate in the cross-state growth specification (columns 3 and 4), the two variables being much less strongly correlated across Indian states ($\rho = 0.4$) than they are in the world cross-section ($\rho = 0.8$). Finally, the estimated magnitude of the adult mortality effect is increased when using the between estimator and fixed effects, though the level of statistical significance falls to 10% (columns 5 and 6).

In terms of magnitudes, focusing on the random effects specification in column 4 of Table 5, with a broad range of controls, a one standard deviation difference in adult mortality (equal to 0.013) is associated with a 0.89 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, 1.79 percentage points). This effect is slightly larger but roughly in line with that obtained in the cross-country regressions. Adult mortality accounts for 1.44 percentage points of the growth difference between a state at the 75th 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 25th percentile, such as Madhya Pradesh (with a male adult mortality rate of 6.7% in 1991). Again, the economic magnitude of growth differences associated with difference in adult mortality is very large.

5 A structured approach to mortality

5.1 The problem of reverse causality

Causality between mortality and development is likely to run both ways, as mentioned earlier.[12] If adult mortality is an important enough determinant of growth, then the vicious cycle between death and development might explain a significant portion of cross-country income differences. In this section, we pursue a more explicitly structured econometric approach to address causality issues. This has two benefits. First, it helps us deal with endogeneity in the mortality-growth relationship. Second, using a system of equations allows us to explore the relative importance of the channels through which mortality affects growth.

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.[13] 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 Sect. 4: reverse causality is likely to be less consequential in growth regressions, where the initial level of income appears as a control on the right-hand side. Moreover, OLS coefficients are useful to

[12] At the same time, recent work by Galor and Moav (2007) suggests that an important component of contemporary variation in life expectancy is genetic, and was determined as far back in history as the Neolithic Revolution. This suggests a substantial portion of the cross-sectional variation in mortality rates may be historically determined.

[13] The view that there is a strong effect of income levels on mortality measures is subject to caveats. 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.

establish whether adult mortality is a plausible candidate as a major explanation for economic performance, since reverse causality would a priori increase the magnitude of the OLS coefficient on adult mortality. We found that the magnitude of the partial correlation was indeed large.

While controlling for initial income on the right-hand side mitigates the problem of reverse causality, it may not eliminate it altogether. Persistently slow-growing countries may not be able to devote incremental resources to fighting diseases and improving medical infrastructure, thereby reducing mortality. Reverse causality is also a potential concern when estimating regressions for investment, school enrollment and fertility. In this section, we confront head-on the potential for reverse causality in the growth, investment, human capital and fertility equations.

5.2 Specification of the structural model

We formulate a structural model making explicit the causal links between growth, the channels linking it to mortality, and the mortality variables. The channel variables we examine are those already discussed in Sects. 3 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.[14] Our structural system for the simultaneous determination of the variables of interest is the following:

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

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 reduced form specification of growth on mortality and other controls (without controlling for the channel variables). 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 provide statistical tests of both of these assumptions below.

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

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

[14] 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. These results are available upon request.

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.[15]

The channel equations all include infant and adult mortality as well as a common set of controls often used in regressions of this type in the literature: log per capita initial income, an index of democracy, population density and the urbanization rate. These controls are included in every channel equation to avoid making arbitrary exclusion restrictions. [16] The investment measure is simply the ratio of investment in physical capital to GDP, and investment is allowed to depend on secondary enrollment and its interaction with initial per capita GDP (as in Barro and Sala-i-Martin 1996, Chap. 12). As a measure of human capital investment, we follow Mankiw et al. (1992) and use the enrollment rate in secondary education.[17] Finally, fertility is measured by the gross fertility rate. Fertility and enrollment enter each other's equations to reflect the impact of fertility on schooling through the quantity-quality trade-off, and to capture the impact of education on fertility behavior.

5.3 Choice of instrumental variables

To address the endogeneity problem, we use three categories of variables as instruments for the two mortality indicators: malaria ecology, climatic variables, and geographic features of countries. We require several instruments because both 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).[18] 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 vectors" (Sachs et al. 2004) to generate a measure of *potential* malaria

[15] 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 et al. (2000), 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.

[16] Very close results are obtained if each channel equation is allowed to contain different controls, based on specifications for the determinants of these variables gleaned from the literature.

[17] We note two features of this choice: (1) Our measure of enrollment differs slightly from Mankiw et al. (1992), who used the gross enrollment ratio in secondary education multiplied by the fraction of the working age population aged 15–19. We use the gross 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. (2) The human-capital augmented Solow model implies that a 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 stock measures are close to the ones we report. For a further discussion of the measurement of human capital, see Bils and Klenow (2000).

[18] 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.

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 may also affect mortality 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.[19] 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 simple 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.[20] 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 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.

In total we have 17 instruments, organized in three sets. We use various subsets of these variables as instruments for adult mortality and infant mortality. We do so in order to examine the robustness of our estimated coefficients to using different sets of instruments and to address the concern that some variables may not be excludable from the estimating equations: we present estimates using all sets of variables, and all three possible combinations of two sets.[21] 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 and its interaction with the log of population.[22]

[19] 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.

[20] 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.

[21] 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 infant mortality as exogenous, an undesirable assumption.

[22] See Frankel and Romer (1999) for discussion of the first instrument, and Alesina et al. (2000) for discussion of the second instrument.

These variables are valid instruments under the assumption that they affect the outcome of interest only through the regressors that are treated as endogenous. Since geographic variables have been used in the past to instrument for institutions in regressions for the level of income, an important issue is whether they allow the identification of the effects of mortality in growth regressions (see for instance Acemoglu et al. 2001). As described above, we argue there is a direct theoretical link between current geography and current mortality. In our empirical analysis, we will include measures of institutional quality in our regression to assess if the effect of institutions and mortality can be estimated separately. Both sets of regressors can be treated as endogenous thanks to the fact that our model is overidentified. We also examine the issue of the validity of instruments through tests of overidentifying restrictions.

Another important concern is whether the instruments might be weak, which would bias IV estimates towards OLS (see Stock et al. 2002, Staiger and Stock 1997). Table 6 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.[23] In other cases, Shea's R^2 statistic takes values of up to 0.51 (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.

5.4 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:[24]

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

To estimate Eq. 2, we treat the 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 7.

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 both statistically significant at the 1% level, and in magnitude *larger* than those obtained with OLS. Running the OLS equivalent of the specification in column (1) on the same sample yields an

[23] 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 two. For this reason, we rely mostly on Shea's R^2 as a measure of first-stage fit.

[24] 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 remain IV estimates.

Table 6 First Stage F-tests for alternative sets of instrumental variables

	ME + Geography + Climate (1)	ME + Climate (2)	ME + Geography (3)	Geography + Climate (4)	ME + Geography + Climate + FR (5)	ME + Climate + FR (6)	ME + Geography + FR (7)	Geography + Climate + FR (8)
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	0.51	0.35	0.07	0.50	0.50	0.35	0.06	0.50
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)
Infant mortality								
Shea R^2	0.51	0.42	0.06	0.37	0.49	0.43	0.06	0.38
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 7 IV Estimates of the growth regression

	IV = ME +Geography +Climate (1)	IV = ME +Climate (2)	IV = ME +Geography (3)	IV = Geography +Climate (4)	IV = ME +Geography +Climate+FR (5)	IV = ME +Climate+FR (6)	IV = ME +Geography+FR (7)	IV = Geography +Climate+FR (8)
Adult mortality rate	−6.699 (3.19)**	−6.955 (3.18)**	−24.073 (2.23)**	−7.734 (3.54)**	−7.465 (3.33)**	−7.358 (3.11)**	−25.623 (2.22)**	−7.877 (3.47)**
Infant mortality rate	−20.299 (3.24)**	−20.912 (2.85)**	47.073 (1.37)	−30.533 (3.25)**	−16.416 (2.32)**	−17.880 (2.25)**	48.546 (1.38)	−23.485 (2.44)**
Log income per capita, 1960	−0.985 (3.25)**	−0.987 (3.83)**	−0.751 (1.46)	−1.020 (2.94)**	−0.769 (2.64)**	−0.777 (2.62)**	−0.764 (1.36)	−0.794 (2.51)**
Democracy index	0.926 (1.12)	1.027 (1.38)	−0.078 (0.05)	1.879 (1.64)	1.175 (1.32)	1.251 (1.36)	0.136 (0.08)	1.764 (1.63)
Urbanization rate, %	−0.001 (0.08)	−0.002 (0.20)	0.005 (0.23)	−0.012 (1.05)	−0.005 (0.50)	−0.006 (0.57)	0.006 (0.24)	−0.012 (1.09)
Population density	0.319 (0.28)	0.316 (0.28)	0.758 (0.34)	0.256 (0.23)	−0.179 (0.14)	−0.204 (0.16)	1.922 (0.66)	−0.264 (0.22)
(Imports + Exports)/ GDP, %	0.058 (2.17)**	0.058 (3.58)**	0.024 (1.15)	0.062 (1.97)**	0.123 (3.56)**	0.122 (3.52)**	0.037 (2.71)**	0.124 (3.26)**
Log of population	0.442 (3.06)**	0.438 (3.80)**	0.312 (1.74)*	0.403 (2.28)**	0.860 (3.83)**	0.853 (3.84)**	0.220 (1.18)	0.835 (3.41)**
Openness*log of population	−0.005 (1.89)*	−0.005 (3.12)**	0.001 (0.20)	−0.006 (1.84)*	−0.011 (3.10)**	−0.011 (3.03)**	−0.003 (1.22)	−0.011 (2.88)**
Constant	7.837 (2.13)**	8.034 (2.72)**	6.818 (1.53)	9.826 (2.32)**	1.538 (0.38)	1.752 (0.43)	9.134 (2.05)**	2.762 (0.62)
Observations	88	89	89	88	88	88	89	88
Hansen J-statistic (p-value)	16.601 (0.34)	15.949 (0.14)	0.823 (0.84)	13.639 (0.48)	13.821 (0.54)	12.438 (0.33)	0.209 (0.98)	11.770 (0.62)

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 initial income (1960)

effect of adult mortality equal to −4.495, whereas the IV coefficient is equal to −6.699 (the OLS coefficient on infant mortality is −16.414 whereas the IV estimate is −20.299). IV estimates are larger in magnitude than OLS estimates, despite theoretical priors to the contrary. This a common finding in this type of application, and may suggest a reduced incidence of attenuation bias due to measurement error under IV.[25]

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 −24.073 (column 3), is unreasonably large, and the estimates on infant mortality is also sensitive to this choice of instruments. This is not surprising: Table 6 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 0.91 percentage point reduction in economic growth, a large effect. These estimates provides a notion of the total effect of adult mortality on growth.

As a final diagnostic test, we report Hansen J statistics to conduct tests of overidentifying restrictions. This statistic, 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 $\chi^2(15)$-distributed test statistic takes on a value of 16.601, with an associated p-value of 0.34. Thus, we fail to reject the null of valid overidentifying restrictions. Similar results are obtained for the other sets of instruments. 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.[26] This is a critical assumption to identify their effects.

5.5 Extensions and robustness checks

In Table 8, we consider extensions to the baseline IV specification. To facilitate comparisons, column 1 reports the same estimates as the baseline regression from Table 7. The first robustness test we consider is to use initial values of the mortality measures rather than their averages over 1960–2000 (column 2). While averaging has advantages from the viewpoint of measurement error, using initial values may be less susceptible to bias from reverse causality. While the coefficient on adult mortality becomes about 14% smaller when using initial mortality rather than the 1960–2000 average, the effect of a one standard deviation change in 1960 mortality (equal to 0.153) is 0.88 points of growth, very close to the 0.91 effect found in column (1) of Table 7.

In column 3, we exclude countries in Sub-Saharan Africa from our sample. As discussed in Appendix 1B, the mortality data for this region is worse than for the rest of the world, so it

[25] See Acemoglu et al. (2001) and Frankel and Romer (1999), for instance, for applications where this is the case.

[26] To be more precise, since there are 2 endogenous variables in Eq. (2) and 17 instruments, the proper interpretation of the overidentification test is that, assuming that 2 of the instruments are uncorrelated with the error term in Eq. (2), the other 15 instruments are uncorrelated with the error term in (2) as well (as is well-known, there is no empirical way of testing whether all instruments are excludable).

Table 8 IV Growth regressions: Extensions and Robustness Dependent variable: Growth of per capita income, annual, 1960–2000

	Baseline (1)	Initial mortality (2)	Excl. Sub-Saharan Africa (3)	Peaceful states (4)	War effects (5)	Institutions (6)	Institutions (7)
Adult mortality rate	−6.699 (3.19)**		−8.620 (2.19)**	−8.239 (1.91)*	−6.054 (2.72)**	−4.373 (1.90)*	−7.227 (3.03)**
Infant mortality rate	−20.299 (3.24)**		−35.276 (3.74)**	−17.185 (1.77)*	−21.626 (3.77)**	−20.278 (2.73)**	−18.126 (3.41)**
Adult mortality rate 1960		−5.774 (2.55)**					
Infant mortality rate 1960		−10.254 (2.58)**					
Months of inter-state war 1960–1997					0.008 (1.61)		
Months of intra-state war 1960–1997					−0.001 (0.92)		
Risk of expropriation, 1990						0.520 (2.46)**	
Constraints on executive, 1970/1990 avg.							−0.186 (1.03)
Log income per capita, 1960	−0.985 (3.25)**	−1.061 (3.89)**	−1.710 (5.19)**	−0.669 (1.66)*	−1.030 (3.58)**	−1.200 (3.74)**	−0.719 (2.55)**
Constant	7.837 (2.13)**	4.636 (1.36)	17.841 (4.27)**	4.938 (0.93)	8.480 (2.41)**	8.302 (2.36)**	5.717 (1.70)*
Observations	88	88	59	46	88	81	78
Hansen J-statistic (p-value)	16.60 (0.34)	18.39 (0.24)	12.38 (0.65)	17.31 (0.24)	18.39 (0.24)	15.49 (0.35)	13.54 (0.41)

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

All regressions include the following controls (estimates not reported): Democracy index, urbanization rate (%), population density, (Imports+Exports)/GDP (%), log of population, openness*log of population

Instruments for adult and infant mortality in all columns: Malaria ecology, climate variables and geography variables, as in column (1) of Table 7 and as defined in text

is important to examine the robustness of our results when focusing on the sample with better mortality data. Our result confirmed in this sample—the coefficient on adult mortality is negative and has a p-value of 2.8%. Since the standard deviation of adult mortality is smaller in the sample that excludes Sub-Saharan Africa, the effect of a one standard deviation change in adult mortality falls to 0.644 points of annual per capita income growth—still a large effect quantitatively. This suggests that our results are not driven by systematic mismeasurement in Africa or solely by the stark distinction between the richest and poorest nations.

Next, we examine the role of war (columns 4 and 5). Countries that have experienced the turmoil of inter-state war or civil war may experience slower long-term growth because of damaged political, social, and economic institutions. At the same time, their mortality rates might be directly or indirectly increased, in which case the direct connection between mortality and low growth would be spurious. However, the evidence suggests this is not the case. In column 4, we run our baseline IV regression on the subset of 46 countries that have engaged in neither war nor civil war during the period. Contrary to the hypothesis that war might be an important omitted variable, we see that in fact the estimated coefficient of adult mortality increases in magnitude to −8.239 (with a p-value of 5.6%), and the effect of a one standard deviation change in adult mortality on growth in this subsample rises to 1.309. If anything, this suggests that reductions in adult mortality matter even more in stable, peaceful countries than in ones where other sources of turmoil and uncertainty predominate. In column 5, we run our baseline regression with additional controls for the number of months spent in inter-state wars and for the number of months spent in intra-state (civil) wars. We do see a 10% reduction in the estimated coefficient of adult mortality, suggesting that if war is in fact an omitted variable in our previous analysis, it is a minor one. Neither interstate nor intrastate war has any significant impact on growth.[27]

Next, to address the possibility that adult mortality captures the effect of institutional quality, we use two different measures of the quality of institutions. Expropriation risk, a survey-based measure, 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 institutional quality because it is more objective and thus less likely to be conflated with wealth. When entering each of these variables in our regression, we treat them as endogenous and instrument for them using our set of geographic instruments.

In column 6, controlling for expropriation risk in 1990 reduced the magnitude of the coefficient on adult mortality by roughly one third, to a level comparable to that of the OLS estimate, while its p-value falls to 5.7%. Expropriation risk does come up as positive and significant in its own right (the index is decreasing in the risk of expropriation). In column 7, adding constraints on the executive (averaged over the 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.[28] Thus, we find little evidence that adult mortality captures the effect of institutions on growth.

[27] We also used different measures of the impact of wars and civil wars: total battle deaths from 1960-1997 (in either itra- or inter-state wars), divided by average population. These measures led to a positive relationship between inter-state war and growth, and a negative relationship between intra-state war and growth, but these effects were not significant at conventional levels. The use of these variables rather than the duration variables to measure war has a negligible impact on the estimated coefficient on adult mortality.

[28] Measures of institutional quality, such as indices of democracy, often come out insignificant in cross-country growth specifications (Tavares and Wacziarg 2001). This stands in contrast with their estimated effect on income levels. See Acemoglu et al. (2001) for compelling evidence on the latter.

5.6 Channel estimates

We now turn to estimating the channel equations. As we have argued earlier, adult mortality impacts growth by reducing incentives to engage in behavior that yields 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. In addition, as discussed earlier, competing theories posit strong links between fertility and mortality as agents take into account not only their horizons but also the horizon of their offsprings when making fertility choices. In this section, we investigate these relationships empirically. We examine how adult mortality relates to investment, human capital accumulation, and fertility. We have two goals. First, these relationships are interesting in their own right as evidence for the horizon effect of mortality. The results here paint a picture consistent with the theory discussed in Sect. 2. Second, they are a first step toward decomposing the total effect of mortality on growth into its various channels.

For each of the three channels, we estimate two different specifications: first, a barebones specification that includes no other regressors than log per capita income and the two measures of mortality; second, the baseline specification with many more controls as described in Sect. 5.2. As before, the mortality variables are instrumented for using malaria ecology, geography and climate variables. The results are presented in Table 9.

5.6.1 Adult mortality and physical capital investment

A high rate of adult mortality is associated with a reduction in the investment rate. In column 2, adult mortality affects investment negatively. The coefficient estimate has a p-value of 9.5%. In that specification, a one standard deviation increase in the male adult mortality rate is associated with a 2.61 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.

5.6.2 Adult mortality and human capital accumulation

Columns 3 and 4 of Table 9 display the correlates of human-capital accumulation. Adult mortality is negatively associated with human capital accumulation in the barebones specification (column 3), though this effect is not statistically significant (the p-value is 12.7%). The effect switches signs and remains insignificantly different from zero when controls are introduced (column 4). The main reason the coefficient changes signs is the introduction of the fertility rate in the enrollment equation. As predicted by a simple model of the quantity-quality trade-off, we find that fertility is negatively associated with enrollment. Working with different specifications and different measures of human capital as dependent variables, we consistently found fragile and insignificant relationships between schooling and adult mortality. Similarly, infant mortality is not robustly associated with enrollment rates.

5.6.3 Adult mortality and fertility

Columns 5 and 6 address the determinants of the total fertility rate. As 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 infant mortality, and both variables have separately

Table 9 IV estimates of the channel equations (1960–2000 averages for all dependent variables)

	Investment (1)	Investment (2)	Enrollment (3)	Enrollment (4)	Fertility (5)	Fertility (6)
Adult mortality rate	−12.935 (1.26)	−19.203 (1.67)*	−0.247 (1.53)	0.168 (1.05)	4.579 (3.71)**	3.373 (2.70)**
Infant mortality rate	−90.348 (2.83)**	−49.966 (1.15)	−2.105 (3.64)**	−0.214 (0.28)	19.598 (5.10)**	14.909 (2.69)**
Log income per capita, 1960	−0.978 (0.89)	−5.254 (2.86)**	0.053 (2.70)**	0.011 (0.41)	0.002 (0.01)	−0.097 (0.51)
Total fertility rate				−0.078 (3.96)**		
Secondary school gross enrollment ratio		−56.647 (0.68)				−3.784 (4.15)**
Log per capita income* Enrollment		7.917 (0.99)				
Democracy index		−0.830 (0.26)		−0.087 (1.68)*		−0.495 (0.92)
Urbanization rate, %		0.040 (0.77)		0.002 (1.80)*		0.012 (1.86)*
Population density		−8.517 (1.84)*		0.031 (0.37)		−0.837 (1.66)*
Constant	34.372 (3.41)**	62.120 (3.62)**	0.064 (0.35)	0.397 (1.85)*	1.352 (0.86)	3.496 (2.22)**
Observations	95	86	86	85	94	85

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

Instruments for adult and infant mortality in all columns: Malaria ecology, climate variables and geography variables, as in column (1) of Table 7 and as defined in text. In column (2). Fertility, secondary school gross enrollment and its interaction with per capita income are treated as endogenous whenever they appear on the right-hand side of channel equations

significant effects, as suggested by theory. The magnitude of the relationship between adult mortality and fertility is again very large: using the specification in column 6, controlling for several other determinants of fertility, a standard deviation increase in adult mortality is associated with a 0.459 point increase in the fertility rate (the mean of fertility in our sample is 4.183 births per woman). 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.

5.7 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 Sect. 5.2. Our baseline specification of the growth and channel equations is 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 2, Table A4.

The 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 appear in Table 10. The total effect of adult mortality on growth through the three channels is equal to −8.307, implying that a one standard deviation increase in adult mortality is associated with a 1.130 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 7. In fact, the total effect we estimate here is slightly larger than the total effect estimated in column (1) of Table 7, where the estimate was −6.699. This suggests that our three channels capture well the total effect of adult mortality on economic growth. Further evidence of the 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 −0.280, and is statistically indistinguishable from zero (the t-statistic is equal to −0.33). A similar result holds for infant mortality. Thus, we can be quite confident that our three channels exhaustively capture the effect of mortality on economic growth.

Turning to the channels themselves, we note that, consistent with the observations based on IV estimates in Sect. 5.6, adult mortality is negatively related to the investment rate and secondary enrollment, but positively related to the fertility rate. The investment and fertility effects of adult mortality are very close in magnitude to the IV estimate in Table 9, and both are statistically significant at least at the 8% level. The effect on enrollment is still positive, and

Table 10 System estimates of the adult and infant mortality effects (3SLS)

	3SLS Baseline Model				
	Effect of channel on growth	Effect of adult mortality on channel	Effect of adult mortality on growth	Effect of infant mortality on channel	Effect of infant mortality on growth
Investment effect	0.180	−19.281	−3.472	−33.932	−6.111
t/Wald test statistic	5.51	1.77	2.91	(0.92)	0.83
p-value	(0.000)	(0.079)	(0.089)	(0.359)	(0.363)
Enrollment effect	−3.862	0.410	−1.585	−0.722	2.787
t/Wald test statistic	2.49	2.09	2.66	(1.12)	1.03
p-value	(0.013)	(0.037)	(0.104)	(0.263)	(0.311)
Fertility effect	−0.747	4.348	−3.250	2.891	−2.161
t/Wald test statistic	3.70	3.22	6.26	(0.61)	0.37
p-value	(0.000)	(0.001)	(0.013)	(0.544)	(0.545)
Total effect			−8.307		−5.484
Total effect (1 s.d)			−1.130		−0.269
Wald test statistic			8.34		0.35
p-value			(0.004)		(0.553)

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

larger here (as in Table 9, the estimate is negative if fertility is excluded from the enrollment equation). 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. This is consistent with the general difficulty economists have had in pinning down a robust relationship between human-capital variables and economic growth (Pritchett 2001, Bils and Klenow 2000, Benhabib and Spiegel 1994). Using alternative specifications for the system of equations, we consistently found a small and fragile relationship between adult mortality and growth through the enrollment channel—both because of the fragile effect of mortality on enrollment, and because of the fragile effect of enrollment on growth.

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 −3.472, and is statistically significant at the 9% level. The fertility effect is −3.25, with a p-value of 1.3%. The effect through enrollment is small (a one standard deviation increase in adult mortality reduces growth by 0.216 points through this channel alone) and statistically insignificant at the 10% level.[29] In sum, 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, as the corresponding estimates are close to zero and sensitive to specification choices.

Finally, our system estimates allow us to quantify the effect of infant mortality. We find evidence that infant mortality reduces growth, but the effect is modest in size and statistically insignificant. Results in Table 10 suggest that a one standard deviation increase in infant mortality reduces growth by 0.269 points, much less than the standardized effect of an increase in adult mortality.

5.8 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 7, 8 and 10 seems to be in the range of −6 to −8. With such a range of estimates, a one standard deviation increase in mortality is associated with a reduction in annual economic growth of between 0.8 and 1.1 percentage points, larger than the corresponding magnitude from the OLS estimates in Table 4. Channel estimates suggest that fertility and investment are important mediating channels linking adult mortality to growth.

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 found evidence of this effect across a range of data using multiple empirical approaches.

[29] 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 (2005), 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.

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 and the growth rate 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 AIDS infection rates.

We explored three channels whereby adult mortality may affect growth: investment, human capital accumulation and the fertility rate. Investment and fertility are the strongest channels. The demographic transition accounts for much of the high correlation between fertility and adult mortality: countries with high fertility and high mortality, in the early stages of their transitions, and countries with low mortality and low fertility, that have completed their transitions, 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.

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 and have a larger number of children rather than fewer, high quality ones. This, in turn, 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: sources and definitions of mortality data

A. Mortality and fertility measures

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 stages 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 (Table A1).

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,

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
USA	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
Singapore	10649	–	0.015	0.005	0.198

Table A1 Continued

Country	PPP Income per capita	Growth 1960–2000	Infant mortality	Total death rate	Adult mortality (age 15–60)
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

Table A1 Continued

Country	PPP Income per capita	Growth 1960–2000	Infant mortality	Total death rate	Adult mortality (age 15–60)
Saudi Arabia	12246	–	0.082	0.012	0.284
Guyana	2497	–	0.073	0.009	0.287
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

Table A1 Continued

Country	PPP Income per capita	Growth 1960–2000	Infant mortality	Total death rate	Adult mortality (age 15–60)
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
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

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 the commonly-used life expectancy data do not capture forward-looking expectations as commonly understood by statisticians and economists. A life expectancy statistic is instead a snapshot of mortality in a given year, summarized as the

expected lifetime of a child born in that year if all mortality rates were to remain constant through the remainder of the child's life.

Infant mortality is usually 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. 2003, p 26).

In this paper, we focus our attention mostly on the adult mortality rate. [30] This is the probability that a 15-year old will die before age sixty, given current age-specific mortality rates.[31] 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. In the Indian cross-state dataset, adult mortality computed over the 20–40 age range is a better predictor of cross-state growth (Table A2). 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 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.

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.

[30] 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%).

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

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

B. Data sources and limitations

The data series and sources used in this study are listed in Table A3. The cross-country demographic data for this study come from the World Bank's World Development Indicators (WDI). 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.[32]

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

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

Table A3 Description of variables and data sources

Variable	Source and definition
A. Cross-national dataset	
Log income per capita	Log of PPP real income per capita, Chain index. Source: Heston et al. (2002)
Growth of income per capita	Annual growth of PPP real income per capita, Chain index. Source: Heston et al. (2002)
Investment share of GDP	Investment in constant prices/GDP in constant prices, %. Source: Heston et al. (2002)
Government consumption share of GDP	Government consumption in constant prices/GDP in constant prices, %. Source: Heston et al. (2002)
Imports plus exports over GDP (openness)	(Imports + Exports in constant prices)/GDP in constant prices, %. Source: Heston et al. (2002)
Log of population	Log of population in 1000s. Source: Heston et al. (2002)
Adult mortality rate	Probability of a male surviving to age 60, conditional on surviving to age 15. Source: World Bank (2004)
Infant mortality rate	Probability of an infant dying before age one. Source: World Bank (2004)
Crude death rate	Proportion of the total population dying in a given year. Source: World Bank (2004)
Total fertility rate	Expected number of births per woman, based on age-specific fertility rates. Source: World Bank (2004)
Urbanization rate, %	Urban population/total population, %. Source: World Bank (2004)
Secondary school gross enrollment ratio	Rate of enrollment in secondary school. Source: Barro and Lee (2000)
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: Lacina and Gleditsch (2005)
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: Lacina and Gleditsch (2005)
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: Lacina and Gleditsch (2005)
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: Lacina and Gleditsch (2005)
Democracy index	Freedom House index of political rights. Source: Freedom House (2004)
Sub-Saharan Africa dummy	Source: Central Intelligence Agency (2004)
Log of Land area in square km	Source: Central Intelligence Agency (2004)
Distance from equator	Absolute value of latitude/90. Source: Central Intelligence Agency (2004)

Table A3 Continued

Variable	Source and definition
Death rate from AIDS per 1,000, 2001	Death rate from AIDS in adults and children, 2001, per 1,000. Source: UNAIDS (2004)
% adults (15–49) living with AIDS, end 2003	AIDS prevalence rate among adults aged 15–49, end 2003. Source: UNAIDS (2004)
Distance from coast	Mean distance to nearest coastline (km). Source: Gallup et al. (2001)
Elevation	Mean elevation in meters above sea level. Source: Gallup et al. (2001)
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. (2001)
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 et al. (2004)
Frost	Proportion of land with more than five frost days per months in winter. Source: Masters and McMillan (2001)
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 (1999)
B. India dataset	
Log of per Capita Income in Period Initial Year	Reserve Bank of India
Mortality rate, male, ages 20–40	Source: Census of India
Death rate, all ages	Source: Census of India
Infant mortality rate, per 1,000 live births	Source: Census of India
Total fertility rate, avg. of first 5 years of each decade	Source: Census 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

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.[33]

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

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 for 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, only limited data from sampling methods is available and data on adult mortality is 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 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.[34] 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 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

Footnote 33 continued
United States for 1975. From this data we calculated age-specific mortality rates for each 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.

[34] 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.

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" (Hill 2003).[35] 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 experts, 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 on long-run averages. Such averages also reduce the incidence of measurement error.

In addition to the cross-country sample, we also collected cross-state data from India (Table A3, Panel B). 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 census services for the years 1971–1997. We then used these death rates to calculate adult mortality directly.

The findings presented in this paper are robust to the use of a variety of subsamples and empirical approaches, giving us confidence that they are not simply driven by flaws in the collection of the data.

Appendix 2 - system estimates

Table A4 3SLS estimates of the baseline system specification

	Growth of per capita income, annual, 1960–2000 (1)	Investment share of GDP, % (2)	Secondary school gross enrollment ratio (3)	Total fertility rate (4)
Adult mortality rate		−19.281 (1.77)*	0.410 (2.09)**	4.348 (3.22)**
Infant mortality rate		−33.932 (0.92)	−0.722 (1.12)	2.891 (0.61)
Total fertility rate	−0.747 (3.70)**		−0.111 (6.25)**	
Investment share of GDP, %	0.180 (5.51)**			
Secondary school gross enrollment ratio	−3.862 (2.49)**	−47.173 (0.72)		−5.439 (6.49)**

[35] 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.

Table A4 continued

	Growth of per capita income, annual, 1960–2000 (1)	Investment share of GDP, % (2)	Secondary school gross enrollment ratio (3)	Total fertility rate (4)
Government consumption share of GDP, %	−0.026 (1.88)*			
(Imports + Exports)/ GDP, %	0.045 (2.06)**			
Log of population	0.320 (2.26)**			
Openness*log of population	−0.004 (1.61)			
Log per capita income* Enrollment		7.684 (1.20)		
Log income per capita, 1960	−0.856 (3.84)**	−5.297 (2.89)**	−0.007 (0.30)	−0.108 (0.59)
Democracy index		2.253 (0.62)	−0.063 (0.95)	−0.420 (0.84)
Urbanization rate, %		0.055 (1.11)	0.001 (1.54)	0.010 (1.42)
Population density		−7.086 (1.35)	−0.073 (0.74)	−1.007 (1.38)
Constant	6.556 (1.96)*	57.255 (3.37)**	0.670 (3.32)**	4.698 (3.10)**
Observations	81	81	81	81
R^2	0.45	0.57	0.80	0.84

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

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