Wealthier is Healthier
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ABSTRACT

We estimate the effect of income on health using cross-country, time-series data on health (infant and child mortality and life expectancy) and income per capita. We use instrumental variables estimates using exogenous determinants of income growth to identify the pure income effect on health, isolated from reverse causation or incidental association. The long-run income elasticity of infant and child mortality in developing countries lies between −0.2 and −0.4. Using these estimates, we calculate that over a half a million child deaths in the developing world in 1990 alone can be attributed to the poor economic performance in the 1980s.

I. Introduction

Wealthier nations are healthier nations. Figure 1 displays the simple association between per capita income[1] and two measures of a country's health performance, infant mortality and life expectancy. Both improve sharply

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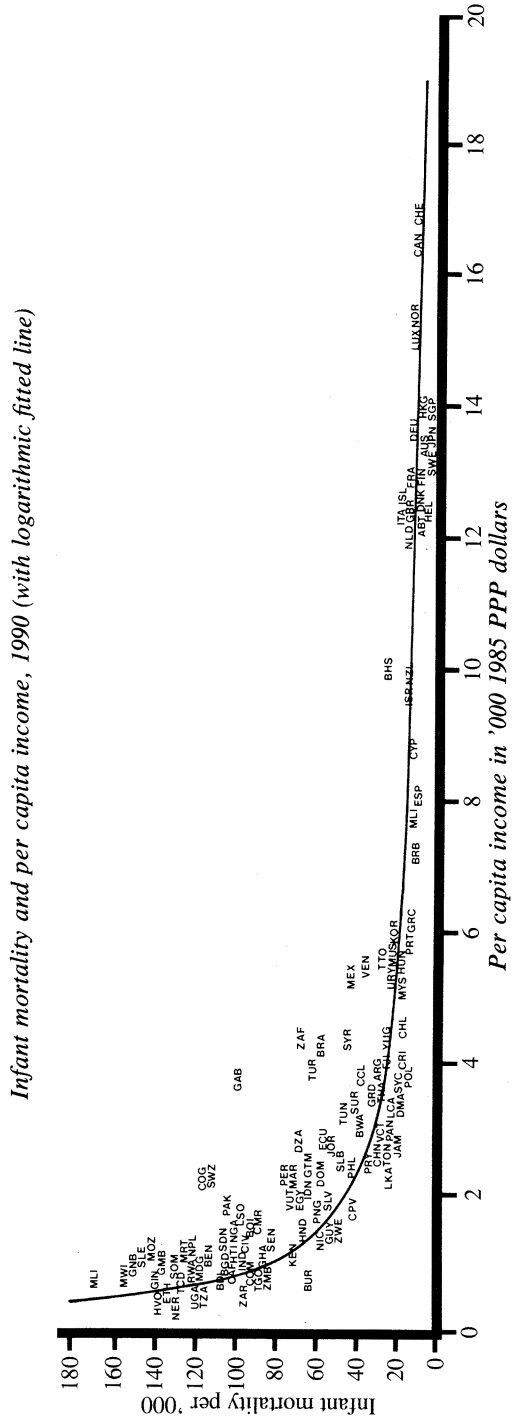
1. We use GDP per capita at 1985 real purchasing power (PPP) adjusted dollars (RGDPCH) from the Penn World Tables 5 (PWT5) of Summers and Heston (1991) throughout this paper, unless noted otherwise. These figures, the result of the International Comparisons Project (ICP), adjust for price level differences across countries. The fact that nontradables are generally cheaper in poor countries implies that comparisons of GDP per capita at official exchange rates tend to overstate differences in real income across countries. For the poorer countries the PPP incomes are higher than World Bank Atlas GDP per capita estimates by a factor of 2 to 3.



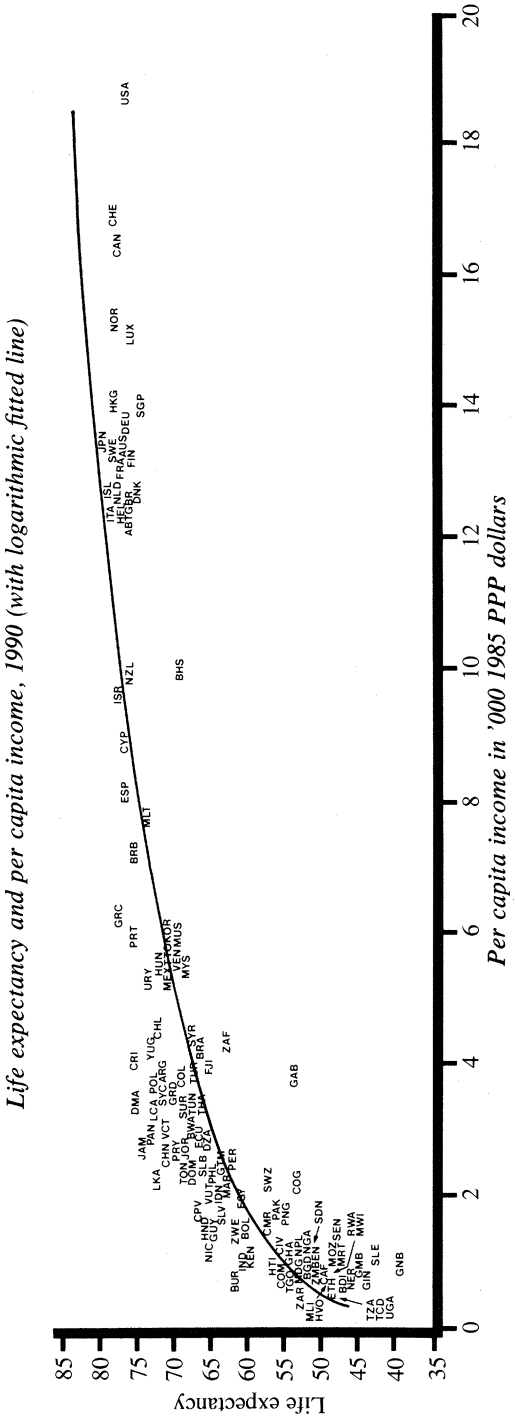


Figure 1

with rising income, especially at low income levels. There are three plausible explanations for the existence of this health-wealth relationship: (i) increased income causes better health; (ii) healthier workers are more productive and hence wealthier (reverse causation); or (iii) some other factor may cause both better health and higher wealth (incidental association). Using instrumental variables estimation and data across countries and over time, we find strong evidence that the positive relationship between income and health is not merely associative but is causal and structural. The estimated income elasticity of infant and child mortality is between -0.2 and -0.4. Country differences in income growth rates over the last three decades explain roughly 40 percent of the cross-country differences in mortality improvements. Since rising income causes improved health (most likely through increased public and private spending on goods that directly or indirectly improve health), raising per capita incomes will be an important component of any country's health strategy. The estimates imply that if income were 1 percent higher in the developing countries, as many as 33,000 infant and 53,000 child deaths would be averted annually.

This article has five sections following this introduction. The second section discusses the data, specification, and estimation technique. The third section presents the results of the estimation. The fourth section explores the relationship of our results to the previous literature. The fifth section examines the health implications of economic performance in light of a causative income-health link. A brief concluding section ends the article.

II. Health and Wealth

Table 1 shows GDP per capita, infant mortality rate (IMR), and life expectancy by income quartiles in both 1960 and 1990.[2] This table illustrates two sources of variations in health status: income levels and trends over time. First, in each year IMR falls by roughly half as average income rises across each quartile. In 1990, IMR falls from 114 deaths per 1,000 live births at US$660 in the poorest quartile, to 66 at $1,727, to 34 at $3,795, and to only 9 per 1,000 at $11,422. Similarly, life expectancy is 76 years in the richest country quartile and falls monotonically to only 50 years in the poorest countries. Second, there has been substantial progress across years in lowering mortality and raising life expectancy. Life expectancy has increased by at least nine years within each income quartile, and infant mortality fell by more than 50 percent for countries in the top three quartiles (although the gains were much smaller for those countries which ended the period in the bottom quartile).

In addition to income and time trends there is a third source of variation, country-specific factors, that influences health status. The large spread of health outcomes within each income quartile illustrates the extent to which factors other than income and secular trends influence health status. As seen in Figure 2, the country with the best IMR in the second income quartile (Sri Lanka, at 19) does

2. The income quartiles in 1960 and 1990 are defined by a country's place in that year; therefore quartiles do not contain the same set of countries in each period.

Table 1
Average Infant Mortality and Life Expectancy, by Per Capita Income Quartiles

	Poorest 25%	Second Quartile	Third Quartile	Richest 25%
Infant mortality rate				
1960	167	153	95	41
1990	114	66	34	9
Percent change	−32%	−57%	−64%	−78%
Life expectancy				
1960	41	43	60	67
1990	50	61	69	76
Percent change	22%	42%	15%	13%
Income per capita				
1960	540	1,069	1,803	5,172
1990	660	1,727	3,795	11,422
Percent change	22%	62%	110%	120%

Notes: GDP per capita is from Penn World Tables, Mark 5, Summers and Heston (1991). The income quartiles are based on distributions of income in 1960 and 1990; therefore the sets of countries are not the same in the two periods.

much better than the median country in the third quartile (at 28) and even better than the worst country in the top income quartile (Trinidad and Tobago, at 25). Jamaica has income of only $2,555, but an IMR of 16, while Portugal's IMR is 13 in spite of income twice as high. Brazil's ($4,130) life expectancy is 66 years, while Costa Rica, with similar income, has a life expectancy of 75 years. Other country characteristics besides income clearly play a large role in determining health, and many have been suggested in the literature: these include effective public programs (Dreze and Sen 1991), equitable distribution of income (Anand and Ravallion 1993), and higher status of women (Caldwell 1986).

A. Specification

This paper disentangles these three sources of variation in health status. In order to isolate the effect of income from country-specific factors and time trends, the basic relation estimated is Equation (1):

$$(1) \quad H_{it} = \beta^* YPC_{it} + \gamma^* X_{it} + \alpha_i + \delta_t + \varepsilon_{it}.$$

The (log of) each indicator of health status H_{it} (namely, infant mortality, child mortality, or life expectancy) in the ith country in the tth period is assumed to be a linear function of its (log) GDP per capita (YPC) in that period, other variables (X, such a schooling), a country-specific effect (α_i), and a time-specific effect (δ_t). The presence of country-specific effects allows for the presence of any number of unspecified country-specific, time-invariant variables that improve the

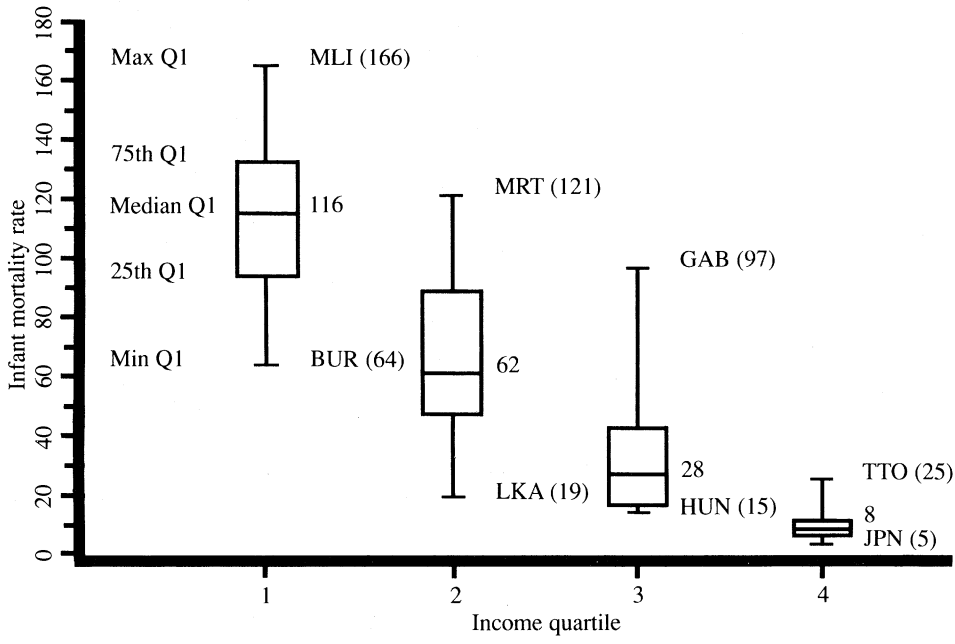


Figure 2
Infant mortality by income quartile, 1990 (Box plots with; median, min, max, 25th and 75th percentile)

NOTE: MLI (Mali), BUR (Burma), MRT (Mauritania), LKA (Sri Lanka), GAB (Gabon), HUN (Hungary), TTO (Trinidad and Tobago), JPN (Japan)

level of health for any given level of income. The inclusion of a separate intercept for each period also allows overall mortality to shift downward over time, perhaps due to exogenous improvements in, or the diffusion of, health technology.

There are three econometric issues in the specification and interpretation of the estimated relationship: estimating total versus partial effects, functional form, and the identification problem. First, we are estimating the total effect of income on health, not the partial effect. Increases in income lead *pari passu* to increased per capita expenditures, both public and private, on goods that improve health: food, safe water, basic sanitation, and shelter, items that absorb large fractions of the poor's expenditures. Some estimates of mortality include as proximate determinants of health outcomes either expenditures on health-promoting goods (for instance, food spending and public health spending) or other physical variables (for example, daily caloric intake, number of health workers per person, availability of clean water) that are principally determined by income.[3] The esti-

3. The R^2 in a cross-country regression on GDP per capita of per capita doctors is 0.76 and daily caloric intake 0.69 (Ingram 1992).

mated coefficient on income in an equation of this type is the *partial* effect of income, holding all else constant, including those variables that would normally vary with income. This partial effect reflects only that effect of income on health that is not already captured by the variables included. Of course the total effect of income on health is the partial effect, plus the effect of income mediated through the other variables. One possibility for recovering the total effect is to estimate a structural multi-equation model of the many paths whereby income affects health, specifying and estimating an equation for each potential proximate determinant of health (for instance, daily caloric intake, sanitation, health workers per capita). This, however, requires the (correct) specification of the proximate determinants of health (analogous to a direct "production function" for health status) as well as the estimation of a large number of equations to explain the proximate determinants themselves. We instead estimate directly a (semi)-reduced-form equation relating income to health, excluding those variables primarily determined by income.

The second specification issue is choosing a functional form. We use the double log form, which imposes a constant elasticity. This has the convenience of easy interpretation of the estimated coefficients. Health gains are effectively bounded, however, which suggests increasing effort to achieve equal absolute gains in health status as mortality approaches its lower bound. Other researchers have used a logistic transformation or a semi-log form to capture this effect. Only developing countries are included in our sample, however, and these countries were not approaching the achievable minimum of mortality where the relationship may become highly nonlinear. Empirical experimentation showed that the basic results presented below were invariant and the estimated elasticities roughly constant across the range of incomes considered.[4]

The third major issue in specification is identifying a causative as opposed to a merely associative relationship between income and health. There are two well-known difficulties. First, there is the danger that both good health and high incomes are associated with some other country-level factor, such as "cultural values" or "good government," that is either intrinsically unobservable or for some other reason excluded from the estimation. With excluded variables a statistical relationship between health and income may be merely an incidental association. The second possibility is that good health could directly raise living standards (Fogel 1994), in which case one could not interpret a statistical relationship between health and income to imply that income causes better health (reverse causation).

We deal with these difficulties in two ways. First, the estimation of Equation (1) using only (log) time differences (or country fixed effects) eliminates any

4. A logistic specification, that is, transforming y to $\log(y/(1 - y))$, produces nearly identical results but is much less convenient for simulations. We also experimented (excessively) with semi-log forms and linear splines, again finding elasticities roughly invariant. Using fixed effects estimates and including schooling, the elasticity at the mean is -0.35 for level, -0.33 for semi-log, and -0.42 for double log form (without education). Similarly, Kakwani (1993) finds roughly constant income elasticity across income levels, as his estimates vary only from -0.56 at the 1980 \$500 level to -0.51 at \$5,000. We didn't consider the issue of functional form in the context of the present work to be sufficiently important to merit formal specification tests.

impact on the income estimates of any country-specific, time-invariant variable (for example, climate or culture) that happened to be related both to income and health. Since most hypothesized excluded variables that could cause a merely associative relationship are time persistent, especially relative to income changes, first-difference estimation should reduce the effect on the income estimates of excluded variables.

Second, and this is the central innovation of this research, we marshal an important piece of evidence that the relationship is causal by using instrumental variables estimation. If the statistical association between income and infant mortality was due to incomes and mortality being caused jointly by some other factor or due to reverse causation, then one would expect that the estimated relationship would change when different variables, related to income but not directly related either to health or to the lurking "third" variable, were used to instrument for income changes in estimating their association with mortality changes. By using a set of plausibly exogenous growth determinant instruments singly, we have a number of potentially consistent IV estimates, $\tilde{\beta}_{IV_j}$, where j indexes the set of possible instruments. To the extent that the $\hat{\beta}_{IV_j}$ estimates are similar to each other, this is evidence in favor of a stable, causal relationship between income and health. To the extent that these IV estimates are in turn similar to β_{OLS}, this is some evidence that the positive association is not primarily created by reverse causation. The use of the instrumental variables approach is of course dependent on the availability of valid instruments, a point we address and test below.

B. Data

Before turning to the estimates, the data deserve some discussion. Estimates of infant mortality and life expectancy are available with broad country and time coverage, but that is only because the international organizations that produce the data strive for comprehensive coverage, not because reliable foundations for such estimates actually exist. As reported in notes to the World Bank publications (see, for instance, World Bank 1990), the figures reported for infant mortality are often based on interpolations, extrapolations, or simply on comparisons with other countries. While these estimated figures will tend to be reasonably accurate for their intended purpose of comparing *levels* across countries at a point in time, much of the variation over time in the reported series is completely artificial. The figures for the most recent years tend to be the least reliable (Bos, Vu, and Stephens 1992).[5] A recent United Nations publication, *Child Mortality Since the 1960s* (1992), presents a compilation of data on infant and child mortality that give an indication of the potential magnitude of the problems. The IMR in Bangladesh in 1973 is estimated variously at 157, 118, and 148 in three different household surveys.[6] The IMR in Brazil in 1975 was 92, or 74, or 84—depending on the

5. Particularly worrisome is the fact that the extrapolation procedure *assumes* a downward trend that is larger the higher the initial level. Comparisons of recent trends using this extrapolated mortality data are therefore worse than worthless.

6. The Bangladesh Fertility Survey, December 1975 to March 1976, Bangladesh Contraceptive Prevalence Survey 1979–80, or Bangladesh Contraceptive Prevalence Survey 1983–84 (see United Nations 1992). The World Bank data report 139.

source.[7] Despite these discrepancies, however, the gross magnitude of changes is captured. In the UN publication (1992), all sources show Thailand falling from near 100 in the early 1960s to near 30 in the late 1980s, while again most sources show Bangladesh falling only from around 150 in the 1960s to near 100 currently.

Since the mortality data from the standard sources have excessive and unreliable coverage, we use only those estimates that were judged to be based on reliable, recent surveys by King and Rosenzweig (1991).[8] We also avoid annual data and use only data at five-year intervals over the period from 1960 up to 1985, for a maximum of five observations per country.[9] Using only data likely to be of high quality, using country differences over time, and using estimation strategies robust to measurement error (IV) assures that the results are unlikely to be merely an artifact of poor mortality data.

III. Results

We first report the OLS results on infant mortality, then verify the robustness of the OLS estimates with respect to variations of timing of observation, data quality, and income definition. We then report the various IV estimates for infant mortality for a single specification and sample. We then report similar OLS and IV estimates for total child (under 5) mortality and life expectancy.

A. OLS for Infant Mortality

Table 2 reports the results of estimating Equation (1) in five-year log differences for countries with GDP per capita below $6,000 using observations for the years 1960 to 1985.[10,11] Infant mortality falls with income with an elasticity of −0.19

7. The estimates of a national household survey of 1977, a census of 1980 (see United Nations 1992), or our World Bank data.

8. Elizabeth King, as part of the background for the WDR '91, used evidence on the quality of the infant mortality data to create a dummy indicator if the country's observation was "good." See Bos, Vu, and Stephens (1992) for a description of the derivation of the current World Bank estimates.

9. While the data are available at annual frequencies in the World Bank databases, the annual data for infant mortality are simply extrapolations between estimates at five-year (or longer) frequencies.

10. The cutoff point excludes 33 countries in 1985. Greece's 1985 *YPC* was $5,712, while Ireland's was $6,008. We excluded the richer countries primarily because our research interest was the developing countries. We experimented with their inclusion with a variety of functional forms, in particular forms that involved splines at various points. We found that the estimated elasticities were often actually *higher* for the richer countries, but we also found that the presence or absence of the richer countries did not appreciably affect the results for the developing countries.

11. Truncating the sample used in the estimation on per capita income when per capita income is potentially endogenous with respect to health status raises the possibility of biased and inconsistent estimates. However, we don't feel that this has a significant impact on our estimates for four reasons. First, using the predicted value of the level of income based using lagged values to predict current incomes (or leads for 1960) to purge the contemporaneous correlation of health status and current income has almost no impact on the estimates. The estimated IMR elasticity changes only from −0.191 (Table 2, column 2) to −0.197. Second, as reported below, we find no evidence of reverse causation from health status to income, and the impact on the estimates of the sample selection problem in the presence of reverse causation should be much smaller than the impact of reverse causation itself. Third, the

Table 2
The effect of per capita income on infant mortality and Per Capita Income, OLS Estimates, 1960–85

Column	1	2	3	4	5	6	7	8	9
	Five-year first differences		Levels		Just difference 1960–85	Five-year first differences			Five-year levels
	without education	with education	without education	with education	with education	Using all data	Weighted by income per capita	Weighted by population	Country fixed effects
GDP per capita	−.24	−.19	−.713	−.42	−.23	−.121	−.18	−.18	−.313
	(5.03)	(4.34)	(16.71)	(8.13)	(2.99)	(4.33)	(4.21)	(4.55)	(6.51)
		‹4.28›							
Years of school		−.019		−.136	−.076	−.016	−.019	−.018	−.010
(Level)		(6.22)		(8.02)	(4.96)	(8.87)	(6.41)	(6.48)	(.51)
		‹5.47›							
1965	−.097	−.030	9.66	8.03	—	.046	−.030	−.02	−.09
1970	−.078	−.016	9.70	8.07	—	−.041	−.017	−.01	−.17
1975	−.124	−.048	9.67	8.03	—	−.066	−.048	−.066	−.28
1980	−.158	−.074	9.69	8.05	—	−.077	−.071	−.132	−.42
1985	−.192	−.091	9.45	7.89	—	−.088	−.088	−.101	−.61
Constant	—	—	—	—	−.206	—	—	—	—
R^2	.177	.325	.634	.731	.445	.273	NA	NA	.795
# Countries	58	58	58	58	66	111	58	58	64
# Observations	184	184	184	184	66	368	184	184	248

Note: Absolute values of *t*-statistics are in parenthesis (angled brackets indicate heteroskedasticity consistent *t*-statistics).
Source: The World Bank.

when controlling for education (column 2), implying that a country at the sample mean would avert one death per 1,000 births if income were higher by 1 percent. The point estimate is quite precise, as the standard error is 0.044 (t of 4.34), and hence a two standard error interval runs from 0.11 to 0.29.

The third and fourth columns of Table 2 show that OLS estimates that do not control for country-specific effects are much too large. The income elasticity when estimated in levels, instead of time differences, without education (−0.71) is nearly three times the differenced estimate (−0.24) and more than twice the fixed effects estimate (−0.31, column 9).[12] This instability of the income coefficients between levels and first differences (or deviations from means) suggests serious upward bias in estimates using levels of exactly the type that would result from excluding variables positively correlated with the level of per capita income.

For example, education has often been cited for its strong effect on reducing infant mortality, and levels of education are highly correlated with levels of income. This suggests that the exclusion of education might create misleading estimates of the effect of income on mortality. Column 4 shows the results of including the (log) level of educational attainment in a regression in levels without country-specific effects.[13] The inclusion of education in the levels regression has the expected impact, lowering the income estimates substantially (from −0.71 to −0.42), and education itself is strongly significant (−0.136, t of 8.0). Reassuringly, adding educational attainment in the differenced equations has a minimal impact on the estimated coefficient on income (−0.24 versus −0.19).[14]

Given concerns about the quality of the data, we tried three variations in the estimation. First, using all the available data instead of only the "good" data produces broadly similar results, but with a lower income elasticity of −0.121 (column 6 of Table 2). Given that pure measurement error in the dependent variable should not induce biases, this result is a little surprising, although it is possibly the result of systematic biases induced by the extrapolation procedure used in producing the World Bank data.[15] Second, although we have made some

results are quite robust to the particular income cutoff used, so there is no evidence of sharply differing parameter values of those selected into and out of the sample. Fourth, although the sample is chosen based on the level of income, the results are estimated in five-year differences.

12. Others report similar differences comparing level and first-difference estimates. Bhalla and Gill (1993) report estimates of the elasticity of infant mortality with respect to private income one-third as large when using both time and country effects as those from simple cross-sections. Kakwani (1993) finds effects for GDP less than half as large using differences. King and Rosenzweig (1991) similarly report results using country dummy variables and find that a 10 percent increase in income is associated with a 1.1 percent fall in infant mortality, a result inconsistent with the cross-section estimates.

13. We use a recently created estimate of the mean years of schooling of the workforce-aged (over 25) population (Barro and Lee 1992).

14. The coefficient on education is not our primary investigation. Hill and King (1992) find strong effects of lagged female enrollment reducing infant mortality and also find strong effects of a large gap in enrollment rates between males and females. A more detailed specification of infant mortality determination would include gender differences in education. Bhalla and Gill (1993) find an effect twice as large for the stock of female versus male education.

15. In extrapolating from past estimates to create up-to-date estimates, the procedure used for World Bank data builds in a downward trend. Moreover, this downward trend is nonlinear in levels, such that countries with higher initial mortality are assumed to have larger percentage reductions over time. If countries with very low growth also tend not to produce up-to-date mortality estimates (not a bad

attempts to control for the quality of the data, it is almost certain that substantial variation in the quality of the data remains, implying a heteroskedastic error term. The heteroskedasticity consistent t-statistic estimates (presented in angle brackets in column 2) suggest that this potential inconsistency is not affecting inferences. The most worrisome aspect of data quality is that the coefficient estimates may be influenced by a few outlying observations. Our fears on this score were calmed by two facts. Weighted least squares estimates, weighted using either per capita income or population as weights (columns 7 and 8), are very similar to OLS.[16] Moreover, dropping each country sequentially from the regression gives estimates ranging only from -0.174 (dropping Portugal) to -0.218 (dropping Rwanda).

Relating current infant mortality to current (as opposed to lagged) income changes and using five-year (as opposed to longer) intervals is likely to underestimate the long-run effect of income changes. This understatement could be caused either because some of the effects of income increases are relatively long-lived (like long-term investments in sanitation) or because the measurement error attenuation bias is more severe in shorter-run data (Grilliches and Hausman 1986). A simple cross-country regression using the percentage changes between 1960 and 1985 gives a much larger point estimate (column 5 of Table 2). Table 3 shows that in five-year differences (using the entire sample, including the questionable data, and dropping education to generate sufficient observations), the income elasticity estimate is -0.15.[17] Using longer time differences we get consistently higher estimates: -0.24 (one decade), -0.35 (two decades), and -0.43 (three decades).[18] This suggests that our base case income elasticity estimate using five-year differences, -0.19, is likely to understate substantially the long-run income impact.

As is typical for a regression explaining differences, the R^2 is quite low. In the OLS results in column 2 of Table 2 only 33 percent of the total five-year differences of infant mortality is explained by income changes, initial education, and time dummies. The partial R^2 of income after controlling for education and time effects is just 10 percent. These R^2s at five years again sharply understate the explanatory power of income changes alone over longer periods. The R^2 of income over the 1960 to 1985 period is 0.4.

The time dummies estimate the exogenous improvements in health. The explanatory power of the set of time dummies, after controlling for income and education, is also around 10 percent. The estimates suggest that, controlling for income,

guess since the median growth rate during 1980–93 of low-income countries was *negative*), then this extrapolation procedure will produce estimated mortality declines, but attributing mortality declines to countries with slow income growth will obviously create a huge downward bias in the income coefficient.

16. The benefit of weighted OLS in this case is principally heuristic. First, under the assumptions that make OLS consistent, weighted least squares (WLS) for any arbitrary vector of weights should also be consistent; it is (mildly) reassuring that this is true. Second, if one or some few observations have a large influence on the results, this should be detected by shifting the weights that individual observations receive.

17. Since the education data end in 1985, this limits the time periods available, but the point estimates increase substantially with the duration of the period with the inclusion of education.

18. Bhalla and Gill (1993) use *annual* data and not surprisingly find even lower elasticities for private income of around -0.05.

Table 3
Income Elasticity of Infant Mortality Estimated Using Various Length Differences

Period	Without Education	With Education
Five-year differences	−0.15	−0.12
Decades	0	0
60 to 70	−0.25	−0.21
65 to 75	−0.22	−0.17
70 to 80	−0.23	−0.11
75 to 85	−0.21	−0.17
80 to 90	−0.27	—
Average	−0.24	−0.165
Two decades	0	0
60 to 80	−0.32	−0.201
70 to 90	−0.37	—
Average	−0.345	
Three decades	0	
60 to 90	−0.43	—

Note: To have sufficient observations, all available countries' data were used so that the "five year" estimate with education corresponds to column 6 of Table 2. Education data are available only through 1985.

education, and country-specific effects, infant mortality has fallen by about 5 percent in each five-year period.

B. Instrumental Variables for Infant Mortality

Our estimation strategy for identification of the causal effect of income on health depends on the availability of adequate instruments. These need to be variables that are determinants of income growth but exogenous with respect to health, and they also need to be variables that are not driven by whatever unobserved "third" variable we suspect might be causing both growth and health improvement. Fortunately, a huge and rapidly expanding body of empirical literature on the cross-country determinants of growth provides a large number of variables that influence country growth rates. From this literature we chose four instruments.

Easterly, Kremer, Pritchett, and Summers (1993) have shown that growth rates of income over five-year periods are explained in part by terms-of-trade shocks. This finding suggests the use of terms of trade as an instrument, because five-year changes in terms of trade are convincingly exogenous, both in the sense of not having a direct relationship to infant mortality and of not being determined by any other country-level variable that would jointly affect income growth rates and mortality improvements (such as "good government" or "cultural values").

We also use three other growth instruments. Levine and Renelt (1992) show that the ratio of investment to GDP is robustly related to growth. In addition, several recent papers (Fischer 1993) have also shown that a large black market premium for foreign exchange is negatively related to growth. Dollar (1990, 1992) has shown that the deviation of the official exchange rate from its purchasing power parity level, after adjusting for the systematic effect of per capita income and other factors, which he proposes as a proxy for outward orientation, is an important determinant of growth. These latter three instruments are plausibly not affected by health status, but are somewhat suspect on the incidental association front.

Table 4 presents the results of IV estimation of Equation (1) using the various instruments. Column 2 uses terms-of-trade shocks as the instrument. Compared to the OLS base results (repeated for convenience in column 1 of Table 4), the elasticity is much higher (-0.98 versus -0.19), but the estimates are very imprecise (t of 1.28). In column 3 we see that the estimated impact of income using investment ratios as an instrument for income growth (-0.35) is also substantially larger than the OLS estimate and in this case is statistically significant (t of 3.4). Using the black market premium as an instrument produces results that are slightly higher than OLS (-0.23 versus -0.19), but are again quite imprecise (t of 1.25). The distortion in price levels produces a high (-0.75) but extremely imprecise estimate. Each of the β_{IV} estimates using a single instrument is larger than OLS. The final two columns report the results when all instruments are used together and when all but the black market premium are used. Both of these IV estimates are higher than OLS (-0.28 and -0.29), and each is statistically significant. Pure measurement error in the five-year differences in income could account for why the IV estimates are consistently larger than OLS.[19]

These IV results tell us that using only that component of income variations related to any of a set of other variables (either singly or as a group) produces estimates of the impact on health similar to the estimates produced by using income itself. Since under the null hypothesis that the mortality relationship is causal, $\tilde{\beta}_{IV}$ should converge to each other (as well as to the "true" β), this is evidence in favor of causation from income to health. There are three aspects of the IV estimates that strengthen this interpretation.

First, the lower the correlation among the instruments, the more impressive the result that each $\tilde{\beta}_{IV_j}$ is roughly the same, as each new instrument represents independent information. In the extreme case with perfectly correlated instruments, the different IV estimates would provide no new information. The highest correlation between any of the four instruments used is 0.126. That a number of nearly orthogonal components give nearly the same (and uniformly higher) estimates as OLS strengthens the plausibility of the IV evidence.

Second, our instruments are individually relatively weak, as the first-stage regressions (of five-year income changes on the instruments) have R^2 values be-

19. The higher point estimates from IV could stem from a measurement error problem in five-year differences of income. If the IV estimates are fully consistent, then measurement error as a fraction of the total variance in five-year growth rates would be about 35 percent (namely, $1 - 0.19/0.29$), a pretty reasonable figure.

Table 4
Infant Mortality and Per Capita Income, Instrumental Variable Estimates, Five-Year Differences, 1960–85

	1 (OLS)	2 IV	3 IV	4 IV	5 IV	6 IV	7 IV
GDP per capita	−0.19	−0.98	−0.350	−0.234	−0.755	−0.28	−0.29
	(4.34)	(1.28)	(3.37)	(1.25)	(0.67)	(2.15)	(2.51)
Years of school	−0.019	−0.018	0.017	−0.019	−0.012	−0.019	−0.021
(Level)	(6.22)	(2.64)	(5.05)	(4.53)	(0.821)	(4.70)	(5.32)
1965	−0.030	−0.127	−0.015	−0.022	−0.024	−0.030	−0.007
1970	−0.016	−0.118	−0.004	−0.015	−0.057	−0.011	−0.005
1975	−0.048	−0.065	−0.036	−0.046	−0.002	−0.041	−0.028
1980	−0.074	−0.035	−0.061	−0.069	−0.027	−0.065	−0.055
1985	−0.091	−0.213	−0.109	−0.094	−0.153	−0.095	−0.087
Instrumental variable	—	Terms of trade	Investment ratio	Black market premium (BMP)	Price level distortion	All four	Without BMP
First stage R^2	—	0.243	0.353	0.285	0.175	0.436	0.412
Hausman (p-value)	—	0.102	0.077	0.979	0.488	0.573	0.319
Hausman-Taylor (p-value)	—	—	0.147	0.045	0.815	0.058	0.151
Sargan (p-value)	—	—	—	—	—	0.051	0.343
Number of countries	58	51	58	50	58	48	51
Number of observations	184	143	184	150	184	134	143

Notes: Absolute value of t-statistics in parentheses. "First Stage" R^2 is the R^2 from regressing the first differences of GDP on the instrument set (including the time dummies). The Hausman statistic tests the equality of the IV and OLS estimates. The Hausman-Taylor is a test of each instrument set on the maintained hypothesis that the terms of trade are exogenous. The Sargan is a test of the over-identifying restrictions.

tween 0.17 and 0.35 (which includes the effect on income of the year dummies and education).[20] On the bad side, the low first-stage R^2s imply that large standard errors from IV estimation are inevitable because explaining five-year growth rates is difficult. On the good side, this low correlation in the first-stage results lends credibility to the exogeneity of the instrument, as extremely high correlated instruments would simply reproduce OLS.[21]

Third, until now we have relied on arguments that our instruments are plausibly exogenous, but we can do some statistical tests. One can test (Hausman 1978) whether the coefficients estimated using all income variation (OLS) and the using only the income variation related to another variable (IV) are equal. The tenth row of Table 4 shows the *p*-value of the Hausman test for each of the instrument sets.[22] The test never rejects that the OLS and IV estimates are equal at the 5 percent significance level (although it does reject at the 7 percent level when using the investment share as an instrument). Of course to the extent that the original instruments are weak, the statistical power of the test will be low.

A second test for our instrumental variables estimates is to test that the instrument set satisfies the orthogonality restrictions assumed and imposed in IV estimation. We use a test proposed by Hausman and Taylor (1981) that allows us to test a subset of the over-identifying restrictions. If it is accepted that one of our instruments, in this case terms of trade, is truly exogenous, we can test whether or not an additional instrument is also exogenous by comparing the IV estimate with and without the instrument being tested. The eleventh row of Table 4 reports the results of testing the exogeneity of each instrument, assuming the terms-of-trade shock is truly exogenous, the just identifying assumption in which we have the most confidence. In this case, somewhat surprisingly, we can actually reject the black market premium on foreign exchange as an adequate instrument. In the final two columns, we see this as well. Testing the exogeneity of all of the instruments against the terms of trade, we reject the exogeneity of the instrument set at roughly the 5 percent level when the premium is included and fail to reject it when the premium is not used as an instrument.

We even have a story (admittedly ex post) for why the BMP is a bad instrument. Since the BMP is high in times of social unrest (for example, the value of BMP in our data was 4,800 percent in Mozambique in 1986, and 917 percent in Uganda in 1980) and social unrest is bad for health independently of its effect on growth (public services disrupted, etc.), it is not legitimately excluded from having a direct effect on infant mortality and hence is an unacceptable instrument.

The final test is a test of all of the over-identifying restrictions imposed on the estimation. As IV estimation assumes that the instruments are unrelated to the error term, this is tested by regressing the residuals on the instruments.[23] The

20. The variance-covariance of an IV estimate is $\sigma^2 (x'P_z x)^{-1}$, where P is the projection matrix, while the R^2 of x on z is $x'P_z x/x'x$.

21. The consistency of an IV estimate of the model $y = x\beta + \varepsilon$ depends on the assumptions that $z'\varepsilon = 0$ even though $x'\varepsilon \neq 0$. Obviously if z is perfectly correlated with x this is impossible.

22. The *p*-value is the probability of a test statistic of the magnitude observed if the null were true. If the *p*-value is less than some predetermined significance level, one rejects the null hypothesis.

23. The form of the test used is due to Sargan (1988). The critical assumption for IV is that in the model $y = x\beta + \varepsilon$, plim $z'\varepsilon = 0$. The Sargan test regresses the IV residuals on the instrument set to test those orthogonality restrictions.

test is reported only in the final two columns, as of course with a single instrument there are no over-identifying restrictions and hence no degrees of freedom for the test. When the black market premium is included, the test rejects the instrument set at roughly the 5 percent level (the p-value is 0.051), while when the premium is dropped, the test is far from rejecting (p-value of 0.34). Given that the major drawback of this test of over-identifying restrictions is low power, the fact that we reject one instrument lends some credibility to the empirical resolution of the test in this case.

Even though we have these formal statistical tests of the assumptions used in the IV estimation, the fact that they mostly fail to reject the appropriate null is not wildly reassuring, as we knew going into the tests that the instruments were weak. The strength of the IV results lies more in the rough consensus across a number of reasonably uncorrelated instruments that the income elasticity of infant mortality is -0.2 or larger (for the five-year data).

This evidence from IV estimation eliminates whole classes of objections to the existence of a causal relationship between income and health. In order to sustain the argument that the income-health relationship was only incidentally associative, it would have to be the case that the unobservable variable causing the bias in the income–infant mortality relationship was related to each of the instruments used. As the variables represent quite different economic phenomena and are nearly uncorrelated among themselves, this would make a good story indeed.

C. Other Health-Status Indicators

We have used infant mortality as in indicator of health status for two reasons: it is available for a large number of years and countries, and it avoids the potentially more severe reverse causation problems associated with the relationship between adult health and income growth. Neither of these is critical, and the basic result—a strong link between income and health—will hold true with regard to other health measures. We examine two that are of particular interest: mortality of children under 5, and life expectancy.

UNICEF (1991, 1992) has chosen the under-5 mortality rate as its principal indicator of child well-being. We compare the data on child mortality in 1960, 1980, and 1990 from UNICEF's most recent report with our data on infant mortality. Not surprisingly, the infant and child mortality rates have an extremely high correlation, both across countries (0.975 in 1990) as well as for changes over time (the correlation of the percentage changes from 1960 to 1990 is 0.932). Given this high correlation, it is not surprising that the regression results are substantially similar. Table 5 shows the OLS and IV regression coefficients using the percentage changes in child mortality and per capita income from 1960 to 1980 (we only use changes up to 1980 to be able to include a level of education term).[24] As can be seen, both the OLS and the instrumental variables estimates for child mortality are very similar to those for infant mortality.

There are two basic reasons to expect that the results for life expectancy will

24. We do not use terms of trade, as terms of trade affects only short-run, not long-run growth.

Table 5
The Effect of Income Per Capita on Child (under 5) Mortality Using Differences, 1960–80

Estimation Technique	Instrument	Elasticity Estimate
OLS	—	−0.17
		(3.14)
IV	Investment rate	−0.76
		(2.66)
IV	Black market premium	−0.43
		(1.85)
IV	Price level distortions	−0.55
		(1.05)
OLS (for infant mortality, same sample)	—	−0.19
		(3.61)

Notes: Absolute values of t-statistics in parentheses. The terms of trade is dropped as an instrument in the 20-year horizon. The data are available for 88 countries.

be more tenuous than for infant mortality. First, a recent review of adult health (Feachem et al. 1992) shows that, generally, the causes of death in adults are much less likely to decrease with income, and in fact may increase. Table 6 compares causes of death among children with those among nonchildren. Fewer of the nonchild causes of death are due to communicable diseases (31 versus 71 percent) of the type that can be prevented by improved sanitation and nutrition. Moreover, the incidence of many of the adult causes of death may increase with income. Among male adults (15–45), motor vehicle fatalities account for more deaths of adults than all communicable diseases combined.[25] Also, many argue that the use of tobacco and alcohol, as well as the consumption of foods related to heart disease, tends to rise with income.

Second, as a pure measurement issue, the data on life expectancy are much more tenuous than those on infant or child mortality. In fact, life expectancy figures are generally derived from model life tables rather than observed directly from death registrations. The life expectancy estimates reported in the *World Development Report* (World Bank 1990) for instance are simply updated using an infant (or child) mortality figure applied to the model life table (that provides mortality rates at each age). Given the infant (or child) mortality, the synthetic figure for the life expectancy is derived from these *assumed* mortality rates. Hence in nearly all cases for developing countries, the changes in life expectancy will contain no new information beyond that contained on the changes in infant (or child) mortality

In spite of these reservations, we present regression results for overall life

25. Table 2–10 of Feachem et al. (1992), using data from 56 countries, shows that male adult mortality ($45q$ 15) from communicable disease is 0.79 versus 1.03 for "unintentional motor vehicle injuries."

Table 6
Causes of Death in Under Fives versus Over Fives in Developing Countries

	Under 5	Over 5
Infectious and parasitic diseases	71%	31%
Diarrheal	25%	4%
Respiratory (for example, pneumonia)	33%	9%
Tuberculosis	2%	11%
Perinatal	23%	—
Neoplasms	—	11%
Heart and lung disorders	—	35%
External (for example, accidents)	2%	9%
Other and unknown	4%	14%

Source: WHO (1992), Table i.

expectancy and income per capita. As anticipated, life expectancy is less sensitive to income than child mortality. Table 7 reports the OLS estimate of the elasticity of 0.015, implying that a 10 percent rise in income would raise life expectancy at the mean by one month. The estimates of the effect on life expectancy are quite imprecise, and the coefficient is not quite significant at the 5 percent level (t is 1.77). The instrumental variables analysis produces broadly similar results, as the point estimates under most instruments are higher (with the exception of the BMP), in some cases much too high (using the price distortion measure, the estimate is 0.71). Using all instruments but the BMP, the point estimate is 0.024, roughly 50 percent higher than the OLS result, but again the higher standard errors that result from the relatively low correlation of the instrument set with income produce low power tests, and we cannot reject that the coefficient is zero.

IV. Comparisons with Other Evidence

Our evidence adds to a large literature that has estimated an income-health relationship using cross-national data. The results of previous studies are generally similar to our OLS results, but the researchers who conducted those studies were unable to address issues of causality. Hill and King (1992) estimated a semi-log specification in levels for infant mortality, controlling for physicians per capita and access to safe water and purging the effects of education on income, and found a strongly significant *partial* income elasticity of −0.161 (at the 1985 mean of infant mortality). Subbarao and Raney (1995) estimated a linear equation for IMR, controlling for male and female secondary enrollment, physicians per capita, access to water, and urbanization, and found a significant *partial* income elasticity of −0.21 at the means. Flegg (1982) found a partial income elasticity of −0.19. Parpel and Pillai (1986) found a partial elasticity of −0.27

Table 7
Life Expectancy and Per Capita Income, Instrumental Variables Estimates, Five-Year Differences

	1 (OLS)	2 IV	3 IV	4 IV	5 IV	6 IV	7 IV
GDP per capita	0.015	0.075	0.007	−0.018	0.709	0.012	0.24
	(1.77)	(.660)	(0.36)	(0.50)	(0.39)	(0.55)	(1.18)
Years of school (level)	−0.0015	−0.002	0.002	0.007	−0.046	0.004	0.001
	(0.64)	(0.27)	(0.60)	(1.51)	(0.38)	(1.05)	(0.27)
1965	0.037	0.016	0.038	−0.038	−0.058	0.026	0.026
1970	0.037	0.027	0.038	−0.042	−0.070	0.037	0.035
1975	0.035	0.028	0.036	−0.038	−0.036	0.035	0.034
1980	0.036	0.029	0.037	−0.038	−0.039	0.035	0.035
1985	0.033	0.036	0.032	−0.029	−0.085	0.031	0.035
Instrumental variable	—	Terms of trade	Investment ratio	Black market premium (BMP)	Price level distortion	All four	Without BMP
First stage R^2	—	0.243	0.353	0.285	0.175	0.478	0.466
Hausman (*p*-value)	—	0.618	0.66	0.288	0.021	0.34	0.933
Hausman-Taylor (*p*-value)	—	—	0.49	0.558	0.377	0.589	0.561
Sargan (*p*-value)	—	—	—	—	—	0.219	0.153
Number of countries	58	51	58	50	58	48	51
Number of observations	184	143	184	150	184	134	143

Notes: Absolute value of *t*-statistics in parentheses. "First Stage" R^2 is the R^2 from regressing the first differences of GDP on the instrument set. The Hausman statistic tests the equality of the IV and OLS estimates. The Hausman-Taylor is a test of each instrument set on the maintained hypothesis that the terms of trade are exogenous. The Sargan is a test of the over-identifying restrictions.

among developed countries from 1950 to 1975. Interestingly, the *partial* elasticities estimated with income levels are similar to our shorter period differenced *total* elasticities (around 0.2) but are, as expected, significantly lower than our long-term results. Kakwani (1993) used a functional form for which income elasticities varied across levels of income and, using levels of income in estimation, found total income elasticities of infant mortality between −0.5 to −0.6. This is somewhat higher than our long-run estimates, but the discrepancy stems from Kakwani's use of levels rather than differences in his estimation.

For life expectancy Preston (1980) used data on life expectancy in developing countries from 1940 to 1970 to attribute about half of the 50 percent rise in life expectancy (17 years) to increases in standards of living; during the 1940–70 period, developing country income rose by roughly 250 percent. Hill and King's (1992) estimate of the effect of the partial income elasticity of life expectancy (again controlling for physicians, safe water, and education) is 0.028 for males and for females (though estimated separately). Lutter and Morrall (1992), on the other hand, using fixed effects in estimation between 1965 to 1985, found a total income elasticity of life expectancy substantially higher than our estimate.

Evidence on the causal impact of household income on health indicators is difficult to come by for three reasons. First, at the household level the causation in the relationship between health (especially adult health) and income certainly runs strongly in both directions. Second, the impact of increased household income will understate the impact of increasing community average income, as many factors that affect a household's health outcomes (for instance, exposure to infectious diseases, access to safe water) depend on average community income. Third, there is generally little match between demographic surveys (which tend to be retrospective) and household income surveys. This leaves two sources of household evidence: the relation of infant mortality to variables related to income, and those few demographic studies that also attempt to measure household income.

A survey of the results of the correlates of infant and child mortality from household World Fertility Surveys in 24 countries shows that mortality for children at various ages falls with changes in factors related to household income, such as the "husband's occupation" or "husband's education" (Hobcraft, McDonald, and Rutstein 1984).[26] Table 8 reports the median across countries of the ratio of mortality at each of three stages—neonatal, post neonatal, and childhood—of children in households with a husband whose occupational category is "Professional or Clerical" versus "Skilled and Unskilled Labor" and where the husband has seven or more years of schooling compared with between one and three years. Although the variation of the education effect is surprisingly wide (in Pakistan the under-5 mortality rate among children of more-educated husbands is 92 percent that among children of uneducated husbands, while for Mexico it

26. We compare across levels husbands' education, even though the literature has shown mothers' education to have a stronger impact (Summers 1992), because the effect of husbands' education on mortality is more likely to be directly related to income, as opposed to quality of care. Of course, to the extent that husbands' and wives' education levels are correlated in marriages, the husband's education may overstate the income effect.

Table 8
Median Ratio of Children's Mortality Rates at Various Ages for Different Categories of Husband's Education and Occupation from Household Surveys in 24 Countries

	Neonatal (0–1 month)	Post Neonatal (1 month–1 year)	Childhood (1–5 years)
Husband's education 7 or more years versus 1–3 years	0.63	0.60	0.49
Husband's occupation "Professional or Clerical" versus "Skilled and Unskilled Labor"	0.68	0.69	0.55

Source: Hobcraft, McDonald, and Rutstein (1984), Tables 4, 5, and 6, pp. 202–204.

is only 13 percent), mortality for groups with higher educational status is, on average, 40 to 50 percent lower. Martin et al. (1983) show percentage reductions of mortality across these same classes of father's education of 25 percent in Pakistan, 67 percent in the Philippines, and 68 percent in Indonesia.[27] If a plausible guess of the income differentials across these educational/occupational groups is between 200 to 300 percent and if one assumes that all of the father's education effect is due to income, this implies a (very crude) elasticity between −0.15 and −0.25.[28] A review of 14 household surveys (United Nations 1985) suggests that one additional year of husband's schooling reduces infant mortality by about 5 percent, again consistent with an income elasticity around −0.2 or higher.[29]

The same UN review (1985) found, for the three countries for which husband's income was available in household surveys, elasticities of −0.16 (Nigeria), −0.20 (Thailand), and −0.10 (Sri Lanka). A recent study by Benefo and Schultz (1994)

27. Some evidence that the father's education effect is mainly an income effect is the fact that as more control variables correlated with income (such as household assets) are added to an equation, the effect of father's education becomes lower. For instance in the Philippines the univariate effect was 67 percent while the multivariate effect from a regression that included controls for electricity and toilet facilities lowered the estimate to 19 percent, which is what one would expect if the husband's education effect is principally through income, not due to application of knowledge to health care.

28. If one assumes that the average schooling of those with seven or more years of schooling is ten years as opposed to two years for the 1–3 years category, and if one assumes that the wage increment to each year of schooling is 15 percent (a plausible value for LDCs), then on average the incomes of more-educated husbands will be around 300 percent higher. Evidence from Tanzania and Kenya suggests that wages are roughly twice as high in the "professional and clerical" occupation than in the "skilled and unskilled labor" occupations (Hazelwood et al. 1989).

29. In this study (United Nations 1985) two findings suggest that "husband's education" is primarily an income effect: (i) the impact of husband's education is reduced by the inclusion of income terms (or characteristics related to household income); and (ii) the effect is stronger in urban than in rural areas, suggesting it is not primarily a quality-of-household-care effect.

using household data found income elasticities of infant mortality of −0.4 in Cote d'Ivoire and −0.3 to −0.8 in Ghana. Palloni (1981) estimates income elasticities of infant mortality for a variety of Latin American countries and finds elasticities between −0.15 and −0.25.[30]

V. Implications for Growth

The gains from rapid economic growth flow into health gains. Infant mortality in developing countries fell by 50 percent on average from 1960 to 1990. Table 9 shows the differences in reductions in infant mortality between the rapidly growing and stagnating countries between 1960 and 1990. Korea's quintupling of GDP accounts for the 80 percent fall in infant mortality, while Argentina, with only 12 percent per capita growth over the entire period, saw infant mortality fall only in half. There are, of course, exceptions, such as Brazil and Pakistan, which had rapid growth with less-than-average improvements in IMR, and Sri Lanka and Jamaica, which enjoyed above-average improvements in IMR in spite of relatively modest growth.

Table 10 shows the global health consequences of various growth paths of income. The 1980s was a decade of mixed performance for many developing countries; per capita growth in many regions slowed to a crawl, or turned negative. Table 10 shows that over 450,000 infant deaths, and possibly over a million child deaths, in 1990 alone would have been averted had countries been able to maintain the same rate of growth in the 1980s as in the period 1960–80 (and this despite the fact that China and India, the world's two largest countries, grew more rapidly in the 1980s and hence are excluded from the figures in panel C). In Africa and Latin America the effects are especially large, as growth was lower by 2.5 percent on average. Had these two regions enjoyed the same growth in the 1980s as they did in the period from 1960 to 1980, over 400,000 child deaths would have been averted in 1990 alone. Suggestions that, for whatever reason, per capita growth in the developing world should be curtailed must confront the fact that absent some fairly dramatic compensating action, a reduction in growth rates will substantially slow the improvement of child (and adult) health.

We are examining the impact of increased income on health *pari passu*. The oft-made statement that there is no *necessary* connection between income growth and improved health is true in the sense that there are conceivable ways in which measured GDP per capita could rise without producing health benefits (for example, an increase in income accruing entirely to the top 1 percent of the income distribution) and ways in which health could improve without increased income. On one level, this is simply saying that average income is not the only determinant of health and that simultaneous changes in other variables that coincide with a change in income (such as a change in the distribution of income) could offset the incipient increase from income. That is true, but not at variance with the

30. While we focus on overall household income, there is some evidence that the distribution of income within the household matters. Duncan Thomas (1990) finds that unearned income in the hands of a mother has a bigger effect on the family's health than income under the control of the father.

Table 9
Income Growth and Infant Mortality, Percentage Changes in Selected Countries, 1960 to 1990

	Income Growth					
	Above Average			Below Average		
Fall in Infant Mortality	Country	Income Growth (%)	IMR Fall (%)	Country	Income Growth (%)	IMR Fall (%)
Above average	Korea	500	80	Jamaica	39	74
	China	240	78	Sri Lanka	51	72
	Turkey	127	70			
Below average	Pakistan	97	36	Ghana	−14	35
	Brazil	190	50	Somalia	−18	28

Note: Mean income growth over the countries in the sample over the entire period was 71 percent. The mean fall in infant mortality was 50 percent. The reported countries are those chosen as either above or below the means in each category.

statement that a rise in income, holding factors unrelated to income constant, will produce improved health. For instance, Anand and Ravallion (1993) examine the mechanisms whereby income changes produce mortality changes and suggest that all of the average income effect is due either to its effect on poverty reduction or to increased public spending on health.

The focus in our econometric estimation on income terms to the exclusion of other factors is not intended to convey that other factors are not at work in improving child mortality and health more generally. The wide variation of infant mortality rates by income shows that other factors besides income are at work. A recent, and rapidly growing, literature shows the importance of the education of mothers in promoting child health, even after controlling for household income (Summers 1992). Caldwell (1986), drawing on the experience of Sri Lanka, Costa Rica, and the Indian state of Kerala, has argued more broadly that the economic status of women raises child health for a given level of income. UNICEF (1992) details the experience of the Brazilian state of Ceara, where coordinated public action reduced infant death rates by a third over a three-year period. A range of low-cost interventions exists for reducing infant mortality, and the widespread adoption of these is likely responsible for much of the improvement in child health not attributable to income changes.

There are two points related to these excluded variables, one statistical and one interpretive. First, the excluded effects that are exogenous with respect to income are not likely to have affected the estimates of the income term, both because the income term was estimated with time-series variation, which is

Table 10
Simulation of Deaths Averted in Developing Countries (in thousands)

	Infant	Child (<5)	Infant	Child (<5)
Assumed Income Elasticity	0.2		0.4	
(A) If the level of income were 1% higher				
Developing	16.5	26.3	33.0	52.6
Africa	4.9	8.2	9.9	16.5
Latin America	1.2	1.7	2.3	3.4
(B) If income growth were 1% higher in the 1980s				
Developing	171	275	338	551
Africa	51.7	86.1	102.4	172.2
Latin America	12.1	17.9	23.9	35.8
(C) If income growth in the 1980s were the same as in 1960–80				
Developing (w/o India, China)	457	656	914	1,311
Africa	206.4	334.4	412	688
Latin America	77.7	113.1	155	266

Notes: These simulations are based on summing the number of deaths averted country by country using 1990 values for infant mortality, child mortality, crude birth rates, and populations. In scenario C India and China are excluded as their growth rates were higher in the 1980s than in the period 1960–80.

mostly lacking in these series, and because IV estimates were used.[31] Second, by estimating income effects, we are not attempting to compare the efficiency of investment in overall income growth with investments in child mortality judged solely on the basis of the improvement in child mortality. Improved child mortality is not the only benefit of economic growth, so obviously investments specific to child health improvements are expected to be more "cost effective" in producing health gains than economic growth.

VI. Conclusion

This paper confirms that increases in a country's income will tend to raise health status. Our estimate of the income elasticity of infant and child

31. Suppose IMR is a function of income and of women's status, but that the higher a woman's status, the higher the income level with which she is associated. Then excluding women's status from the estimates will bias the estimate of the effect of income upward. To the extent that women's status across countries changes only slowly over time, however, the use of first differences will "sweep out" the effect of the exclusion of women's status from the estimate of the income term.

health with respect to infant mortality is −0.2 for five-year intervals, while the estimate over 30-year periods is −0.4. Our use of instrumental variables allows us to comfortably assert that the income-mortality relationship is not an artifact of reverse causation or incidental association. The fact that using different, nearly orthogonal, components of income to estimate the elasticity produces generally higher (and tolerably similar) results provides strong evidence in favor of a causal and structural relationship running from income to mortality.

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