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Background paper for World Development Report 1993

Wealthier is Healthier

Lant Pritchett
and
Lawrence H. Summers

In 1990 alone, more than half a million child deaths in the developing world could be attributed to poor economic performance in the 1980s. Wealthier nations are healthier nations.

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With cross-country, time series data on health (infant and child mortality, and life expectancy) and per capita income, Pritchett and Summers estimate the effect of income on health.

They use instrumental variables estimation to identify the effect of income on health that is structural and causal, isolated from reverse causation (healthier workers are more productive and hence wealthier) or incidental association (some other factor may cause both better health and greater wealth).

The long-run income elasticity of infant and child mortality in developing countries lies between 0.2 and 0.4.

Using those estimates, they calculate that in 1990 alone, more than half a million child deaths in the developing world could be attributed to poor economic performance in the 1980s.

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Introduction

Wealthier nations are healthier nations. Figure 1 displays the association between per capita income[1] and two measures of a country's health, infant mortality and life expectancy. Both improve sharply with rising income, especially at low income levels. If rising income causes improved health through increased spending on goods that directly or indirectly improve health, raising per capita incomes will be an important component of a country's health strategy. There are, however, two other plausible explanations for the existence of a health-wealth relationship: (1) healthier workers are more productive and hence wealthier (reverse causation) or (2) some other factor may cause both better health and higher wealth (incidental association).

Using instrumental variables estimation techniques and data across countries and over time, we find strong evidence that the relationship between income and health is not merely associative but causal and structural. The income elasticity of infant and child mortality is between .2 and .4 and differences in income growth rates over the last three decades explain roughly 40% of the cross country differences in mortality improvements. If income were one percent higher in the developing countries, up to 33,000 infant and 53,000 child deaths would be averted annually.

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[1] We use GDP per capita at purchasing power adjusted dollars 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 non-tradables are generally cheaper in poor countries implies that comparisons of GDP per capita at official exchange rates tends to overstate differences in real income across countries. For the poorer countries the PPP incomes are higher than World Bank Atlas numbers by a factor of 2 to 3.

Our work has three major advantages over the previous cross country literature on income and health. First, we estimate the total, not the partial, effect of income. Second, the time series nature of the data is exploited to eliminate the potential bias from the exclusion of unobservable country-specific time-invariant variables. Third, instrumental variables estimation is used to identify income effect and eliminate any potential bias from either reverse causation or incidental association.

The paper has three sections. The first section discusses the data and the specification and estimation technique and presents the results of the estimation. The second section explores the relationship of our results to the previous literature. The final section examines the implications of a causative income-health link.

I) Health and wealth

Table 1 shows GDP per capita, infant mortality and life expectancy by income quartiles in 1960 and 1990. As the average income rises across each quartile in 1990, the infant mortality rate (IMR) falls by roughly half: from 114 at US$660, to 66 at $1,727, to 34 at $3,795, to 9 at $11,422. Similarly, life expectancy is 76 years in the rich countries and falls monotonically across income quartiles to only 50 years in the poorest countries.

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

	Poorest 25%				Second Quartile			
	Mean	Min	Max	St Dev.	Mean	Min	Max	St Dev.
Infant Mortality Rate								
1960	167	118	214	28	153	79	218	40
1990	114	64	166	25	66	19	121	28
% Change	-32%				-57%			
Life Expectancy								
1960	41	32	48	4	43	32	64	7
1990	50	39	61	5	61	47	71	6
% Change	22%				42%			
Income Per Capita								
1960	540	262	854	170	1,069	871	1368	142
1990	660	320	930	174	1,727	931	2448	451
% Change	22%				62%			

	Third Quartile				Richest 25%			
	Mean	Min	Max	St Dev.	Mean	Min	Max	St Dev.
Infant Mortality Rate								
1960	95	30	193	46	41	13	134	29
1990	34	15	97	20	9	5	25	5
% Change	-64%				-78%			
Life Expectancy								
1960	60	41	69	46	67	48	73	7
1990	69	53	75	4	76	69	79	2
% Change	15%				13%			
Income Per Capita								
1960	1,803	1,373	2,409	317	5,172	2,701	9,983	1,904
1990	3,795	2,545	5,382	894	11,422	5,513	18,671	3,484
% Change	110%				120%			

The last three decades have also seen substantial progress in lowering mortality and raising life expectancy as incomes rose. Life expectancy has increased by at least nine years in each quartile. Infant mortality fell by more than 50% in the top three quartiles, although the gains were much smaller in the bottom quartile[2].

The large spread of health outcomes within each income quartile illustrates the extent to which factors other than GDP per capita influence health status. The country with the best IMR in the second income quartile (Sri Lanka with 19) does much better than the average country in the third quartile and better than the worst country in the top income quartile (Trinidad and Tobago with 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 factors besides income clearly play a large role in determining health and many have been suggested: efficient public programs, equitable distribution of income, higher status of women. These factors are undoubtedly important, but our interest in this paper is to estimate the income effect and our principal concern with their inclusion is statistical, that the exclusion of these factors do not impede this estimation.

[2] The income quartiles in 1960 or 1990 are defined by a country's place in that year, therefore it is not the same set of countries in each period.

A) Data and specification

The basic relation to be estimated is equation 1. The indicator of health status (infant mortality, child mortality, or life expectancy) in the i^{th} country in the t^{th} period is assumed to be a linear function of its (log) income per capita in that period, other variables (included in X), a country specific effect, α_i, a time specific effect δ_t. The presence of a country specific effect allows for the presence of any number of country specific, time invariant variables that improve the level of health for any given level of income. The inclusion of a separate intercept for each period allows overall mortality to shift downward over time by exogenous improvements in health technology.

1) $H_{it} = \beta * YPC_{it} + \delta * X_{it} + \alpha_i + \delta_t + \epsilon_{it}$

There are three important issues in the specification and interpretation of the relationship, estimating total versus partial effects, the identification problem and functional form. First, we are estimating the total effect of income on health, not the partial effect. Increases in income lead to increased per capita expenditures, both public and private, on goods that improve health: food, safe water, basic sanitation, and shelter, items which absorb large fractions of the poor's income. Many estimates of countries' mortality determinants include as direct determinants of health outcomes these increased expenditures or variables which are themselves principally determined by income (e.g. daily caloric intake, number of health workers per person, availability of clean water)[3]. The estimated coefficient on income in an equation of this type

[3] The R^2 in a cross section regression on GDP per capita of per capita doctors is .76 and daily caloric intake .69 (Ingram, 1992).

is the partial effect of income, holding all else constant all variables, including those that would normally vary with income. This partial term reflects only that effect of income on health that is not captured by the factors already included. The total effect of income is partial effect, plus the effect of income through the additional variables.

For example, if $H_i = \beta YPC_i + \alpha HPC_i$ and $HPC_i = \delta\ YPC_i$ where YPC is income per capita and HPC is health workers per capita, then the partial effect of income is β, while the total effect of income is $\beta + \delta * \alpha$. The estimate of β from estimating the first equation would be an unbiased estimate of the partial effect of income but is not an estimate of the total income effect at all (even calling it a biased estimate would be too kind).

One possibility for recovering the total effect is to estimate a multi-equation model of the many paths of influence whereby income affects health, specifying and estimating an equation for each potential determinant of health (e.g. estimating both equations in the example above and calculating $\hat{\beta} + \hat{\delta}\hat{\alpha}$). This has the drawback of complexity, as it requires the specification and estimation of a large number of equations. We take a different approach and estimate directly an equation relating income to health, excluding those variables primarily determined by income. The risk of this approach is the usual bias induced by excluding variables[4], although our estimation strategy minimizes this risk, as will be seen.

[4] It is somewhat more complex in this case as it is really only the bias of the component of the excluded variables not due to income that matters.

The second major issue in specification is identifying a structural and causative as opposed to merely associative relationship between income and health. Here there are two possible difficulties. First, there is the danger that both good health and high incomes are associated with some other factor, such as "cultural values" or "good government", which are unobservable or otherwise excluded from estimation. In this case a statistical relationship may be merely associative. Second, good health could actually raise living standards in which case one could not interpret such a statistical relationship as implying the direction of cause and effect.

We deal with these difficulties in two ways. First, equation 1 is estimated in (log) differences across five-year periods (e.g. the percentage change in mortality on percentage change in income). This eliminates any impact on the income estimates of any country specific, time invariant variable that happened to be related both to income and health.

As an example, if the true equation were $H_{it} = \beta * YPC_{it} + \delta * C_i + \epsilon_{it}$, where health depends positively on both income and some country specific, time invariant variable C_i (call it climate). Say there was positive covariance between *YPC* and climate. Then estimation of the model in levels excluding climate would produce a coefficient on income that would be biased upward due to the positive covariance of income and climate. Even if the true model were that $\beta = 0$, exclusion of climate could result in a positive estimated coefficient. However, if the country specific effects are swept out, either using country specific dummy variables (fixed effects) or first differences, then the correlation between the variation in the included variables and the excluded variables is identically zero and hence the income term is unbiased[5].

Second, we marshal an important piece of evidence that the relationship is structural by using instrumental variables estimation. If the relationship between income and infant mortality were not structural and the statistical association were an artifact of the fact that per capita GDP is driven by some other variable that also influences mortality, 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 unknown variable, were used as instruments for income. To the extent that the IV estimates using only the component of income related to the instruments to estimate the impact of income on infant mortality gives the same results as OLS, this gives credence to the claim that a structural direct relationship exists between income and health.

For example, say the true model were:

$$IMR_{it} = \alpha * GG_{it} + \beta * YPC_{it} + \epsilon_{it}$$
$$YPC_{it} = \delta * GG_{it} + \gamma * TOT_{it} + \eta_{it}$$

where GG is good government and TOT is terms of trade shocks. To sharpen the point assume that $\beta = 0$, so that YPC has no independent role in determining infant mortality. However if we were to estimate the equation:

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[5] Algebraically, if $H_{i\cdot}$ represents the average of H_{it} across time for country i, then in deviations form means: $(H_{it} - H_{i\cdot}) = \beta * (YPC_{it} - YPC_{i\cdot}) + \delta * (C_i - C_{i\cdot}) + (\epsilon_{it} - \epsilon_{i\cdot})$ but since C is time invariant $C_i - C_{i\cdot} \equiv 0$.

$IMR^{it} = \beta ypc^{it}$ one would find (on average) $\beta > 0$ as βOLS would converge to α/γ (if TOT and GG were uncorrelated). However, the IV estimate of β using TOT as an instrument, β_{tt} would converge to zero, as

$$\text{plim } \hat{\beta} = \text{plim } cov(IM,ToT)/cov(YPC,ToT) \rightarrow 0.$$

There are a large number of unobservable variables that one could reasonably believe cause both income growth and independently cause health improvements (e.g. good government, culture). One strategy for obtaining a structural estimate of the link between income growth and health in spite of the exclusion of these factors is to identify a set of instrumental variables, correlated with income but plausibly uncorrelated with the "third variable" and uncorrelated with health improvements (except through income). Using these instruments singly, we have a number of consistent estimates $\tilde{\beta}_{IV_j}$, where j indexes the set of possible instruments. To the extent the $\hat{\beta}_{IV_j}$ estimates are similar, this is evidence in favor of a structural relationship.

The last specification question is of functional form. We use the double log form, which imposes that the elasticity is constant. This has the convenience of having an easy interpretation of the parameters. Others dealing with infant mortality have used a logistic transformation, or a semi-log form, but experimentation showed the basic results were invariant and the estimated elasticities roughly constant across the range of incomes considered[6].

[6] 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.

Linear versus logs. While the appropriate functional form is not known a priori, there are pragmatic concerns in choosing the functional form. Infant mortality is effectively bounded below, making a simple linear model unattractive as the form implies equal absolute reductions in infant mortality from equal absolute income changes (or percentage changes in semi-log form). Japan's infant mortality of 4.8 in 1990 was the lowest recorded, but was an absolute fall only 5.2 since 1975, compared to very much larger absolute falls in countries that began from much higher levels, such as Bangladesh, where infant mortality fell from 138 to 117 per thousand. This suggests the use of a logarithmic or logistic specification. In any case, other functional forms give similar results for the income elasticity at the means.[7]

We focus first on estimating the income impact on infant mortality primarily because the data are available for more periods and are marginally more reliable. Later sections will address child mortality and life expectancy. Infant mortality estimates are available with broad country and time coverage, but this is principally because the international organizations which produce the data strive for comprehensive coverage. As reported in notes to the World Bank publications (e.g. WDR, 1990) many times the figures reported for infant mortality are 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, much of the variation over time in the reported series is artificial. The figures for the most recent years tend to be the least reliable (Bos, Vu and Stephens, 1992).

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[7] Using fixed effects estimates, including schooling the elasticity at the mean is -.35 for level, -.33 for semi-log and -.42 for double log form (without education).

Hence we only use data at five year intervals over the period from 1960 up to 1985, for a maximum of five observations per country[8].

A recent UN publication (1992), Child Mortality Since the 1960s, presents a compilation of data on infant and child mortality and gives an indication of the potential magnitude of problems with the data. The infant mortality rate in Bangladesh 1973 is estimated variously at 157, 118 and 148 in three different household surveys.[9] The IMR in 1975 in Brazil was 92, or 74 or 84 - depending on the source.[10] Nevertheless, the gross magnitude of changes is captured. All sources show Thailand falling from near 100 in the early '60's to near 30 in the late '80s while again most sources show Bangladesh falling only from around 150 in the 1960s to near 100 currently. To address this problem, we use in our base estimates only those data that were judged to be based on reliable estimates in an earlier work by King (King and Rosenzweig, 1991)[11]. We feel that using only data likely to be of high quality, using country differences over time, and using estimation strategies robust to observational error assures the results are unlikely to be merely an artifact of poor mortality data.

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[8] While the data are available at annual frequencies in the World Bank databases, much of the annual data for infant mortality is simply an extrapolation between estimates at five year frequencies. In addition, one could expect that the effects at business cycle frequencies of a few years might be quite different.

[9] According to the Bangladesh Fertility Survey, December 1975 to March 1976 (1973-6), Bangladesh Contraceptive Prevalence Survey 1979-80, or Bangladesh Contraceptive Prevalence Survey 1983-84.

[10] The estimates of a national household survey of 1977, a census of 1980 or our World Bank data.

[11] Beth King, as part of the background for the WDR '91 used evidence on the quality of the infant mortality 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.

B) Results

We first report the OLS results and verify the robustness of the OLS results with respect to variations of timing of observation, data quality, and income definition. We then report the IV estimates for a single specification and sample.

i) Basic results

Table 2 reports the results of estimating equation 1 in five year log differences for countries with YPC below $6,000[12], using observations for the years 1960 to 1985. Infant mortality falls with income with an elasticity of .2 (column 2), implying that a five percent increase in incomes would lead to a one percent fall in infant mortality. A country at the sample mean of 100 deaths per thousand births would avert one death per thousand births if income were higher by one percent. The point estimate is quite precise, as the standard error is .044 (t of 4.34), and a two standard error confidence interval runs from .11 to .29.

[12] We felt that the relationship may well be sufficiently different in the richer countries. The cutoff point excludes 33 countries in 1985. Greece's 1985 YPC was $5,712, while Ireland's was $6,008.

Table 2: Infant Mortality and Per Capita Income
OLS Estimates, 1960-1985, five year differences

Variable:	1	2	3 Levels	4	5 Just 1960-85	6 Using All Data	7 Weighted by GDP pc	8 Regression: by Population	9 Deviations from Means
GDP per capita	-.24 (5.03)	-.19 (4.34) <4.28>	-.713 (16.71)	-.42 (8.13)	-.23 (2.99)	-.121 (4.33)	-.18 (4.21)	-.18 (4.55)	-.313 (6.51)
Years of Schooling (level)		-.019 (6.22) <5.47>		-.136 (8.02)	-.076 (4.96)	-.016 (8.87)	-.019 (6.41)	-.018 (6.48)	-.010 (.51)
1965	-.097	-.016	9.66	8.03	--	.046	-.030	-.02	-.09
1970	-.078	-.048	9.70	8.07	--	-.041	-.017	-.01	-.17
1975	-.124	-.074	9.67	8.03	--	-.066	-.048	-.066	-.28
1980	-.158	-.091	9.69	8.05	--	-.077	-.071	-.132	-.42
1985	-.192	-.019	9.45	7.89	--	-.088	-.088	-.101	-.61
Constant	--	--	--	--	-.206	--	--	--	--
R2	.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

Notes: Absolute values of statistics are in parenthesis, angled brackets are heteroskedasticity consistent t-statistics.

ii) Reliability of OLS Results

Country effects. The third and fourth columns of Table 2 show that estimates which do not control for country specific effects are much too large. The income elasticity in levels of -.71 is nearly three times the differenced estimate (-.24) and more than twice the fixed effects estimate (-.31, column 9)[13]. This instability of the income coefficients between levels and first differences, or deviations from means, suggests serious upward bias in estimates using levels of 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 affect on reducing infant mortality and levels of education are highly correlated with levels of income. This suggests 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[14] in a regression without country specific effects. The inclusion of education has the expected impact on the regression in levels, lowering the estimates substantially (-.71 to -.42) and education itself is strongly significant (-.136, t of 8.0). Reassuringly, following the inclusion of country effects, adding educational attainment has a minimal impact on the estimated coefficient on income (-.24 vs -.19)[15].

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[13] Others report similar differences comparing levels and first differences. Bhalla (1991) reports estimates of the elasticity of infant mortality with respect to private income one fifth as large when using both time and country effects as those from simple cross sections. King and Rosenzweig (1991) similarly report results using country dummy variables and find that a ten percent increase in income is associated with a 1.1% fall in infant mortality, a result inconsistent with the cross section estimates.

[14] We use a recently created estimate of the mean years of schooling of the work force aged (over 25) population (Barro and Lee, 1992).

[15] The coefficient on education is not our primary concern, Hill and King, 1992, find strong effects of lagged female enrollment reducing infant mortality and also find strong positive effect 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.

Data quality, heteroskedasticity, outliers. Given concerns about the quality of the data, we tried three variations. Using all the available data instead of only the "good" data produces broadly similar results, but with a lower income elasticity of -.128 (column 6 of table 2). Given that pure observational error in the dependent variables should not induce biases, this is a little surprising.

Second, while we have made some 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. This is a concern for two reasons -- estimation of the standard errors and the presence of influential observations. The minor concern is the potentially inconsistent standard error estimates, but the heteroskedasticity correct t-statistic estimates in angle brackets in column 2 suggest this bias is not affecting inferences.

A more worrisome aspect of varying data quality is that the coefficient estimates may be influenced by a few outlying observations. Fears on this score were calmed by two facts. The weighted least squares estimates, weighting observations either by per capita income or population[16](columns 7 and 8), are very similar to the OLS results. Moreover, dropping each country sequentially from the regression gives estimates ranging only from -.174 (dropping Portugal) to -.218 (dropping Rwanda).

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[16] The benefit of weighted OLS in this case is principally heuristic for two reasons. Since under the assumptions that make OLS consistent, weighed least squares (WLS) for any arbitrary vector of weights should also be consistent, it is (mildly) reassuring 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 individual observations receive.

Long versus short differences. Relating current infant mortality to current (as opposed to lagged) income changes and using five year (as opposed to longer intervals) may underestimate the long-run effect. A simple cross country regression of the percentage changes between 1960 and 1985 gives a larger point estimate (column 5 of table 2). Table 3 shows that in five year differences (using the entire sample), the income elasticity estimates are -.15 (-.12) without (with) education. For longer time differences of one, two or three decades, we get consistently higher estimates, around -.25, -.35 and -.4 respectively (without education). The education data limits the time periods but the point estimates again increase substantially with the duration of the period. This suggests our estimate with five year differences likely understates the long-run income impact.

Table 3: Estimates of income elasticity with various length differences

Period	Without Education	With Education
Five Year	-.15	-.12
Decades		
60 to 70	-.25	-.208
65 to 75	-.22	-.171
70 to 80	-.234	-.107
75 to 85	-.213	-.174
80 to 90	-.27	--
Two Decades		
60 to 80	-.32	(-.201)
70 to 90	-.37	--
Three Decades		
60 to 90	-.43	--

Note: To have sufficient observations, we've used all available countries' data, corresponding to column 6 of table 2.

Typically for a regression using differences as opposed to levels, the R-squared is quite low. In the OLS, only 33% of the total five year differences of infant mortality are explained by income, education and time dummies. The partial R^2 of income after controlling for education and time effects is just 10%. The time dummies give the exogenous improvements in health. The explanatory power of the set of time dummies, after controlling for income and education is also around 10%.

These R^2s sharply understate the explanatory power of income changes alone over longer periods - the R^2 over the 1960 to 1985 period is .4, so that 40% of the differences in mortality improvements are associated with income improvements[17].

iii) Identification of the income effect, instrumental variables

Our strategy for identification of the effect of income alone on health and eliminating suspicions of incidental association depends on the availability of adequate instruments. These need to be variables which are determinants of growth but are exogenous and also not driven by whatever "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.

Recently, Easterly, Kremer, Pritchett and Summers, 1992, have shown that growth rates of income over five-year periods are explained in part by terms of trade shocks. This suggests an appropriate instrument, as five-year changes in terms of trade are convincingly exogenous,

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[17] This is using all countries data and excluding education.

both in the sense of not having a direct relationship to infant mortality as well as not being determined by any other variable that would affect both income growth rates and mortality improvements (such as good government or cultural values).

We use four other instruments. In a recent series of papers, Dollar (1991, 1992) has suggested that the deviation of the official exchange rate from its income level adjusted purchasing power parity level is a proxy for policies that reduce outward orientation and is an important determinant of growth. Levine and Renelt (1992) show that the ratio of investment to GDP is robustly related to growth and hence we will use this as an instrument, although this is somewhat suspect. Several recent papers (Fischer, 1992, Easterly, et al, 1992) have also shown that a large black market premium for foreign exchange is negatively related to growth.

As our final instrument, if other countries are subjected to similar shocks from the external environment, then the correlation between changes in country i's growth rate and country j's growth rate over time could be used as an indicator of the effect of the external environment on country i's growth rates. After constructing a vector of "growth in a similar country", we use that as an instrument.[18] This is (by construction) correlated with country i's growth rate but, by using only the component of i's growth rate related to changes in some other country's growth we hope to produce a good instrument by purging country i's income changes of any component associated directly with infant mortality changes or with variables like "good government."

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[18] We define "similar country" mechanically by searching for each country i over all possible bivariate correlations j.

Table 3 presents the results of IV estimation of equation 1 using the various instruments. Column 2 uses terms of trade shocks as an instrument. Compared to the OLS base results (repeated from table 2 in column 1) the elasticity is much higher (-.98 vs. -.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 is also larger than the OLS estimate (-.35) and is statistically significant (t of 3.4) . Using the black market premium as an instrument produces results that are slightly higher than does OLS (-.23 vs. -.19), but are again quite imprecise (t of 1.25). The distortion in price levels produces a high (-.75) but extremely imprecise estimate. Finally, the use of similar country growth rates produces results nearly identical to the OLS (-.24), but very imprecise (t of .94).

The final two columns report the results first 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 (-.28 and -.32) and are statistically significant.

Intuitively, these IV results tell us that using only that component of income variations related to any of a group of other variables (either singly or as a group) produces estimates of the impact on health similar to using income itself. Since under the null hypothesis that the relationship is structural, $\tilde{\beta}_{IV}$ should converge to $\hat{\beta}OLS$ and to the true β , this is evidence in favor of the structural nature of the estimate. There are three aspects of the IV estimates that strengthen this interpretation.

The lower the correlation of the instruments, the more impressive the result as the $\tilde{\beta}_{IV_j}$ are more independent. For example, in the extreme case with perfectly correlated instruments, different IV estimates would provide no new information. The highest correlation between any of the five instruments used is .126, implying each instrument is capturing and using a different component of income in the estimation. That a number of nearly orthogonal components of income give nearly the same (and uniformly higher) estimates than OLS strengthens the evidence.

Second, our instruments are weak, which in this case is bad and good. A good instrument for our purposes is a variable whose association with income is just strong enough that the standard errors don't blow up[19] but not so strongly related that it essentially reproduces the OLS result. The first stage regressions (of five year income changes on the instruments) have R-squared values between .17 and .35, which includes the effect on income of the year dummies and education. These low first stage R^2s imply that large standard errors from IV estimation are inevitable. On the plus side, this low correlation in the first stage results lends credibility to the exogeneity of the instrument[20].

Third, until now we have relied on arguments that our instruments are plausibly exogenous, but we do have some statistical tests. Our IV estimates use only the variation of

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[19] The variance-covariance of an IV estimate is $\hat{\alpha}_2(x^1P_zX)^{-1}$ the projection matrix, while the R^2 of x on z is $x'P_zx/x'x$.

[20] The consistency of an IV estimate of the model $y = x\beta + \epsilon$ depends on the assumptions that $Z'\epsilon = 0$ even though $X'\epsilon = 0$, obviously if z is perfectly correlated with x this is impossible.

income growth that is related to the instrument to identify the income effect. One can test, Hausman (1978), whether the coefficient estimated using all income variation (OLS), and the restricted variation (IV) are equal.[21] As we illustrated with the earlier example, one reason (among many) that the OLS and IV estimates would be different is if income and infant mortality were both driven by some other variable, excluded from the estimation. To that extent the test is a test of the specification.

The sixth row of table 3 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% significance level, although it does reject at the 7% level when using the investment share as an instrument. Of course to the extent the original instruments are weak, the statistical power of the test (the probability one rejects the null hypothesis when it is false) will be low.

A second test for our instrumental variable estimates is to test that the instrument set that they use satisfies the orthogonality restrictions assumed and imposed in IV estimation using a test proposed in Hausman-Taylor (1981) which allows us to test a subset of the over-identifying restrictions. Given that one of our instruments is truly exogenous, we can test whether or not an additional instrument is also exogenous by comparing the IV estimate with and without the

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[21] As the IV estimation process decomposes the x variable into a component lying in the space of z (the instrument) and a component orthogonal to z, v. Since $x = \hat{x}+v$, the OLS regression is $y = \beta^1(\hat{x}+\beta^2)+\epsilon$ and constrains $\beta^1=\beta^2$. In our earlier hypothetical example, $\hat{x}$ would be the component of income in the terms of trade space and $\beta^1 = 0$ while v would be related to "good government" and hence $\beta^2 > 0$.

[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 pre-determined significance level, one rejects the null hypothesis.

instrument being tested. The seventh row of table 3 reports the results of testing the exogeneity of each instrument, assuming the terms of trade shock is truly exogenous, an assumption in which we have reasonable confidence. In this case, 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% level when the BMP is included and fail to reject when BMP is not used as an instrument.

The final test is the Sargan (1984) 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 testing by regressing the residuals on the instruments[23]. The Sargan test is reported only in the final two columns as with a single instrument there are no over-identifying restrictions and hence no degrees of freedom for the test. We see that when the black market premium is included, the test marginally rejects the instrument set (e.g. the p-value is less than .1) while when the BMP is dropped, the test is far from rejecting (p-value of .35). Given that the major drawback of the Sargan test is low power, the fact that we reject in one case lends some credibility to the resolution of the test. Moreover, 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 (e.g. the BMP was 4,800% in Mozambique in 1986, 917% in Uganda in 1980) and social unrest is bad for health (public services disrupted, etc.), it is not legitimately excluded from having a direct effect on infant mortality and hence is a poor instrument.

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[23] The critical assumption for IV is that in the model $y = x\beta + \epsilon$, $plim\ Z'\epsilon = 0$. The Sargan test regresses the IV residuals $U = Y - x\hat{\beta}_{IV}$ on the instruments to test those orthogonality restrictions.

Table 4: Infant Mortality and Per Capita Income, Instrumental Variable Estimates, Five Year Differences

	1 OLS	2 IV	3 IV	4 IV	5 IV	6 IV	7 IV	8 IV
GDP per capita <$6000	-.19 (4.34)	-.98 (1.28)	-.350 (3.37)	-.234 (1.25)	-.755 (.67)	-.239 (.944)	-.28 (2.21)	-.32 (2.72)
Years of School (level)	-.019 (6.22)	-.018 (2.64)	.017 (5.05)	-.019 (4.53)	-.012 (.821)	-.019 (4.53)	-.019 (4.23)	-.020 (5.26)
1965	-.030	.127	-.015	-.022	-.024	-.026	-.029	-.002
1970	-.016	.118	-.004	-.015	-.057	-.011	-.010	-.008
1975	-.048	.065	-.036	-.046	-.002	-.045	-.040	-.025
1980	-.074	.035	-.061	-.069	-.027	-.070	-.064	-.052
1985	-.091	-.213	-.109	-.094	-.153	-.097	-.095	-.089
Instrumental Variable	--	Terms of Trade	Investment Ratio	Black Market Premium (BMP)	Price [illegible] Distortion	Similar Countries Growth	All	w/o BMP
First Stage R2	--	.243	.353	.285	.175	.197	.286	.296
Hausman (p-value)	--	.102	.077	.979	.488	.848	.543	.215
Hausman-Taylor (p-value)	--	--	.147	.045	.815	.303	.051	.143
Sargan (p-value)	--	--	--	--	--	--	.087	.347
# Country # Observations	58 184	51 143	58 184	50 150	58 184	58 184	48 134	51 143

Notes: Absolute value of t-statistics in parenthesis.

1. "First Stage" R2 is the R2 from regressing the first differences of GDP on the instrument.
2. The Hausman statistic tests the equality of the IV and OLS estimates.
3. The Sargan is a test of over-identifying restrictions.
4. The Hausman-Taylor is a test of each instrument set on the maintained hypothesis that the terms of trade are exogenous.

Even though we have these formal statistical tests, the fact that they all fail to reject the appropriate null is not wildly reassuring, as we knew going in that the instruments were weak. The strength of the results lies more in the rough consensus across a fairly large number of reasonably uncorrelated instruments that the income elasticity is .2 or higher.

The evidence from IV estimation eliminates whole classes of objections to the existence of a structural relationship between health and income. In order to maintain the belief 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 interrelated amongst themselves, would make a good story indeed.

C. Other Indicators of Health Status

We have used infant mortality as an 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 are critical and the basic result - a strong link between income and health - will hold true of other health measures. We examine two that are of particular interest: under 5 (or child) mortality and life expectancy.

i) Child mortality

UNICEF (1991, 1992) has chosen the under 5 mortality rate as its principal indicator of child well-being. It is important to verify that whatever impact income has on infant mortality also be true of child (under 5) mortality. We use the data on child mortality in 1960, 1980, and 1990 from UNICEF's most recent report to compare with our data on infant mortality. Not surprisingly, the infant and child mortality have an extremely high correlation, both across countries (.975 in 1990) as well as for changes over time, correlation of .932 of the percentage changes 1960 to 1990. 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 use to 1980 to include a level of education term). As can be seen, both the OLS as well as the instrumental variables are very similar.

Table 5: Child (under 5) Mortality and Income, Estimates 1960 - 1980, Eighty-Eight Countries

Estimation Technique	Instrument	Elasticity Estimate
OLS	—	-.172 (3.14)
IV	Investment Rate	-.756 (2.66)
IV	Black Market Premium	-.433 (1.85)
IV	Price Level Deviations	-.55 (1.05)
OLS (for infant mortality, same sample)	--	-.192 (3.61)

ii) Life expectancy

There are two basic reasons to expect that the results for life expectancy will 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 to that of adults. Fewer of the adult causes of death are due to communicable diseases of the type that can be prevented by improved sanitation and nutrition. The incidence of many of the adult causes of death may increase with income. Motor vehicle fatalities account for more deaths than all communicable diseases combined. Use of tobacco, alcohol and consumption of foods related to heart disease also tend to rise with income.

Table 6: Causes of Death in Under Fives vs. Over Fives in Developing Countries

	Under 5	> 5
Infectious and parasitic disease	71%	31%
o/w: diarrheal	25%	4%
Respiratory (e.g.pneumonia)	33%	9%
Tuberculosis	2%	11%
Perinatal	23%	--
Neoplasms	--	11%
Heart and Lung disorders	--	35%
External (e.g. accidents)	2%	9%
Other and unknown	4%	14%

Source: WHO, Global health situation and projections, 1992, table i.

Second, the data on life expectancy are much more tenuous than those on infant or child mortality. Life expectancy figures are generally derived from model life tables rather than observed. For instance, to update the life expectancy number the general procedure is to use an updated infant (or child) mortality figure then apply a model life table that provides mortality rates at each age, given the infant (or child) mortality and the synthetic figure for the life expectancy is derived from these model based mortality rates. Hence in many cases the changes in life expectancy will contain no new information beyond that contained in the changes in infant (or child) mortality.

In spite of these reservations, we present regression results for overall life 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 .015, implying a 10% rise in income would raise life expectancy at the mean by a month. The estimates of the effect on life expectancy are quite imprecise and the coefficient is not significant at the 5% 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 .71). Using all instruments but the BMP the point estimate is .024, roughly 50% 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.

Table 7: Life Expectancy and Per Capita Income, Instrumental Variable Estimates, Five Year Differences

	1 OLS	2 IV	3 IV	4 IV	5 IV	6 IV	7 IV	8 IV
GDP per capita <$6000	.015 (1.77)	.075 (.660)	.007 (.36)	-.018 (.50)	.709 (.39)	.054 (1.06)	.012 (.55)	.024 (1.18)
Years of School (level)	-.0015 (.64)	-.002 (.27)	.002 (.60)	.007 (1.51)	-.046 (.38)	-.001 (.223)	.004 (1.05)	.001 (.27)
1965	.037	.016	.038	-.038	-.058	.031	.026	.026
1970	.037	.027	.038	-.042	-.070	.031	.037	.035
1975	.035	.028	.036	-.038	-.036	.031	.035	.034
1980	.036	.029	.037	-.038	-.039	.031	.035	.035
1985	.033	.036	.032	-.029	-.085	.036	.031	.035
Instrumental Variable	--	Terms of Trade	Investment Ratio	Black Market Premium (BMP)	Price Level Distortion	Similar Countries Growth	All	w/o BMP
First Stage R2	--	.243	.353	.285	.175	--	.478	.466
Hausman (p-value)	--	.618	.66	.288	.021	.418	.34	.933
Hausman-Taylor (p-value)	--	--	.49	.558	.377	.612	.589	.561
Sargan (p-value)	--	--	--	--	--	--	.219	.153
# Country # Observations	58 184	51 143	58 184	50 150	58 184	58 184	48 134	51 143

Notes: Absolute value of t-statistics in parenthesis.

1. "First Stage" R2 is the R2 from regressing the first differences of GDP on the instrument.
2. The Hausman statistic tests the equality of the IV and OLS estimates.
3. The Sargan is a test of over-identifying restrictions.
4. The Hausman-Taylor is a test of each instrument set on the maintained hypothesis that the terms of trade are exogenous.

II) Comparisons with other evidence

1) Historical and cross-national studies

The magnitude and strength of the relationship between income and mortality is consistent with other historical cross-national studies even though most previous studies focus on the partial, not total effect of income changes.

Infant mortality rates in the currently developed countries were roughly at or above the current levels for developing countries a century or so ago. McKowen and Record (1962) attribute half of the increase in overall (not just infant) mortality in Britain between 1851 and 1911 to increases in standards of living. In this period British per capita income roughly doubled while mortality fell by forty percent. Watterson (1988) using data from the 1911 British Census of Fertility finds income to be an important determinant of the fall in infant mortality. Fogel (1990) reviews the historical evidence of declining mortality in Europe and America from the eighteenth to early twentieth centuries and concludes that rising living standards, especially the elimination of chronic malnutrition, were an important explanatory component, along with improvements in medical knowledge and technology of the decline.

Hill and King (1992) estimate 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 find a strongly significant partial income elasticity of .161 (at the 1985 mean of infant mortality). Subbarao and Raney (1992) estimate a linear equation for IMR and, controlling for male and female secondary enrollment, physicians per capita, access to water, and urbanization, find a significant partial income elasticity of -.21 at the means[24].

Preston (1980) uses data on life expectancy in developing countries in 1940 to 1970 and attributes about half of the 50% rise in life expectancy (17 years) in developing countries between 1940 and 1970 to increases in standards of living, a period in which LDC income rose by roughly 250%. Hill and King's (1992) estimates of the effect of income on life expectancy (again controlling for physicians, safe water and education) is an elasticity of .028 for male and female (though estimated separately).

2) Micro studies of income-health outcomes

Evidence on the 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 poor health and low income certainly runs both ways. Secondly, 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 (e.g. exposure to infectious diseases, access to safe water) may 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 evidence: the relation of infant mortality to variables related to income and those few studies which also attempt to measure household income.

[24] Other earlier studies find similar results. Flegg (1982) in finds a partial income elasticity of -.19. Parpel and Pillai (1986) find a partial elasticity of -.27 among developed countries from 1950 to 1975.

A survey of the results of the correlates of infant and child mortality from household World Fertility Surveys in twenty-four countries shows that mortality for children at all ages fall with changes in factors related to household income, such as the "husband's occupation" or "husband's education" (Hobcraft, McDonald, and Rutstein, 1984)[25]. Table 8 reports the median ratio of mortality at each of three stages neo-natal, post neo-natal and childhood, of children in households with a husband "Professional or clerical" versus "Skilled and Unskilled Labor" and where the husband has 7+ compared to 1-3 years of schooling. Although the variation of the effect is surprisingly wide (in Pakistan the under five mortality for more educated husbands is 92% that of uneducated husbands, while for Mexico it is 13%) mortality for higher educational groups is, on average, forty to fifty percent lower. A plausible guess is that income differentials across these groups would be 200 to 300 percent[26] which implies an elasticity between .15 and .25. Similar results for the percentage reduction in child mortality for father's education are given by Martin, et. al., (1983) from household surveys in showing univariate estimates of the reduction across these same education classes for fathers of 25% in Pakistan, 67% in Philippines, and 68% in Indonesia[27]. A review of fourteen household surveys

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[25] 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.

[26] If one assumes that the average schooling of those with 7+ years is 10 years as opposed to 2 years for the 1-3 years category then is the rate of return to a year of schooling is 15% (a plausible value for LDCs) then on average incomes of more educated husbands will be 300% higher. Evidence from Tanzania and Kenya suggests that wages are roughly double in the "professional and clerical" versus "skilled and unskilled labor" (Hazelwood, et al, 1989).

[27] Some evidence that the father's education effect is mainly an income effect is that as more control variables that were added related to income the lower the effect of father income. For instance in the Philippines the univariate effect was 67% while the multivariate effect from a regression that included controls for electricity and toilet facilities lowered the estimate to 19%, 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.

(UN, 1985) suggests that an additional year of husband's schooling reduces infant mortality by about 5%, again consistent with an elasticity around -2[28].

TABLE 8: Median Ratio of Child Mortality Rates at Various Ages for Different Categories of Husbands' Education and Occupation from Household Surveys from 24 Countries

	Neo Natal (0 - 1 month)	Post Neo Natal (1 month - 1 year)	Child (1 - 5 years)
Husband's Education 7+ Years vs. 1-3 Years	.63	.60	.49
Husband's Occupation "Professional or Clerical" vs. "Skilled and Unskilled Labor"	.68	.69	.55

Source: Hobcraft et al, 1984, Tables 4, 5 and 6, pp. 202-204.

The UN study (1985) found, for the three countries for which husband's income was available in household surveys, elasticities of -.16 (Nigeria), -.20 (Thailand) and -.10 (Sri Lanka). A recent study by Beneto and Schultz (1992) using household data found income elasticities of infant mortality of -.4 in Cote d'Ivoire and -.3 to -.8 in Ghana. Palloni (1981) estimates income elasticities of infant mortality for a variety of Latin American countries and finds an elasticities between .15 and -.25.[29]

[28] In this study two findings suggest that "husband's education" is primarily an income effect, a) the impact of husband's education is reduced by the inclusion of income terms (or characteristics related to household income), b) the effect is stronger in urban versus rural areas, suggesting it is not primarily a quality of household care effect.

[29] There is some evidence that the distribution of income within the household matters. Duncan Thomas (1990) finds 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.

4) The importance of other factors

The focus in the 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)[30]. 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 exist 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.

The link between household income and child and adult health status is not straightforward. The intuitive notion is that at low levels of income there will be a high marginal propensity to spend on health goods. For example, links between household income and nutrient intake and weight or height are quite complex.[31,32] More research is needed.

[30] There have been studies cross nationally (Hill and King, 1992, Subbarao and Raney, 1992) as well as with household data, although the studies are not unanimous about the relative importance of education versus income effects. Thomas, 1992, shows with data from rural Brazil that after controlling for mother's education, household income is not an important determinant while Benefo and Schultz, 1992, show with data from Cote D'Ivoire and Ghana that mother's education is not important after controlling for household income.

[31] Behrman and Deolalikor (1990) argued for a very low elasticity of caloric intake with respect to household income. Ravallion (1990) has argued that the finding of very low income elasticities is principally due to the sharp non-linearity of the relationship between nutrients and income at low income levels, a finding reinforced by Thomas, Strauss and Henriques (1990).

There are three points related to the excluded variables, one statistical and two 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 mostly lacking in these series and because IV estimates were used[33].

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. Moreover, the question is not growth or direct efforts at reducing mortality, as both are valuable.

Third, we are examining the impact of increased income on health ceteris paribus. The oft made statement that there is no <u>necessary</u> 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 (e.g. an increase in income accruing entirely to the top 1% 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 which coincide with a change in income (such as a change in the distribution of income) could offset the incipient increase from income,

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[32] The relationship between nutrient intake and health outcomes is also not simple and straightforward.

[33] Suppose IMR is a function of income and women's status that higher's women's status increases income. Then excluding women's status from the estimates will bias the estimate of the effect of income upward. However, to the extent that women's status across countries changes only slowly over time, the use of first differences will "sweep out" the effect of the exclusion of women's status from the estimate of income term.

this is true, but not at all variance with the statement that a rise in income, all else constant, will produce improved health. Anand and Ravallion (1993) examine the mechanisms whereby income changes produce mortality changes and find all of the effect is due to poverty reduction or increased public spending on health.

III) The health toll of life in the slow growth lane

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

TABLE: 9
Income Growth and Falling Infant Mortality
Percentage Changes 1960 to 1990

Fall in Infant Mortality	Income Growth	
	High	Low
High	Korea (500%) 80% China (240%) 78% Turkey (127%) 70%	Jamaica (39%) 74% Sri Lanka (51%) 72%
Low	Pakistan (97%) 36% Brazil (190%) 50%	Ghana (-14%) 35% Somalia (-18%) 28%

Note: Mean income growth in the period was 71% and the mean fall in infant mortality was 50%.

Table 10 shows the global health consequences of various growth paths for income. The decade of the 1980's 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 450,000 infant 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 the world's two largest countries grew more rapidly in the 1980s and hence are excluded from the total. In Africa and Latin America the effects are especially large as growth was lower by 2.5% on average. Had these two regions enjoyed the same growth in the 1980's 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 dramatic compensating action a reduction in growth rates will slow the improvement of child (and adult) health.

Table 10: Deaths Averted ('000) if Income:

	Infant	<5	Infant	<5
Assumed Elasticity	.2		.4	
A) 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) Growth Was 1% Higher in 1980's				
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) Growth in 1980s was Same as 1960-80				
Developing (w/o India, China)	457	656	914	1,311
Africa (1.5% lower)	206.4	334.4	412	688
Latin America (4.6% lower)	77.7	113.1	155	266

Conclusion

On one level this paper confirmed what must be a relatively unobjectionable statement: that increases in income tend to raise health status. Our estimate of the income elasticity of infant and child health with respect to infant mortality is between .2 and .4. Income is more important for infant and child health than for overall life expectancy. The regression of income levels on levels of health status across countries tends to impart substantial upward bias and the resulting estimates are likely to be unreliable.

Our use of instrumental variables allows us to comfortably assert that the income mortality relationship is not an artifact of reverse causation or simple association. The fact that using different, nearly orthogonal, components of income to estimate the elasticity produced generally higher (and tolerably similar) results provides strong evidence in favor of a causal and structural relationship running from income to mortality.

We again wish to stress that the exclusion of other variables from the equation does not represent a claim that these are unimportant; but their exclusion does not particularly affect our estimates. Overall, income changes explain less about mortality changes than economists might have supposed (or than comparisons of levels suggest) but substantially more than would be consistent with a suggestion that income *per se* is unimportant.

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