Disease and Development: The Effect of Life Expectancy on Economic Growth

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We exploit the major international health improvements from the 1940s to estimate the effect of life expectancy on economic performance. We construct predicted mortality using preintervention mortality rates from various diseases and dates of global interventions. Predicted mortality has a large impact on changes in life expectancy starting in 1940 but no effect before 1940. Using predicted mortality as an instrument, we find that a 1 percent increase in life expectancy leads to a 1.7–2 percent increase in population. Life expectancy has a much smaller effect on total GDP, however. Consequently, there is no evidence that the large increase in life expectancy raised income per capita.

I. Introduction

Improving health around the world today is an important social objective, which has obvious direct payoffs in terms of longer and better lives for millions. There is also a growing consensus that improving health can have equally large indirect payoffs through accelerating economic

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growth (see, e.g., Bloom and Sachs 1998; Gallup and Sachs 2001; WHO 2001; Alleyne and Cohen 2002; Bloom and Canning 2005; Lorentzen, McMillan, and Wacziarg 2005). For example, Gallup and Sachs (2001, 91) argue that wiping out malaria in sub-Saharan Africa could increase that continent's per capita growth rate by as much as 2.6 percent a year, and a recent report by the World Health Organization states that "in today's world, poor health has particularly pernicious effects on economic development in sub-Saharan Africa, South Asia, and pockets of high disease and intense poverty elsewhere" (WHO 2001, 24) and "extending the coverage of crucial health services . . . to the world's poor could save millions of lives each year, reduce poverty, spur economic development and promote global security" (i).

The evidence supporting this recent consensus is not yet conclusive, however. Although cross-country regression studies show a strong correlation between measures of health (e.g., life expectancy) and both the level of economic development and recent economic growth, these studies have not established a causal effect of health and disease on economic growth. Since countries suffering from short life expectancy and ill health are also disadvantaged in other ways (and often this is the reason for their poor health outcomes), such macro studies may be capturing the negative effects of these other, often omitted, disadvantages. While a range of micro studies demonstrate the importance of health for individual productivity,[1] these studies do not resolve the question of whether health differences are at the root of the large income differences we observe because they do not incorporate general equilibrium effects. The most important general equilibrium effect arises because of diminishing returns to effective units of labor, for example, because land and/or physical capital are supplied inelastically. In the presence of such diminishing returns, micro estimates may exaggerate the aggregate productivity benefits from improved health, particularly when health improvements are accompanied by population increases.

This article investigates the effect of general health conditions, proxied by life expectancy at birth, on economic growth. We exploit the large improvements in life expectancy driven by international health interventions, more effective public health measures, and the introduction of new chemicals and drugs starting in the 1940s. This episode, which we refer to as the *international epidemiological transition*, led to an unprecedented improvement in life expectancy in a large number of

[1] See Strauss and Thomas (1998) for an excellent survey of the research through the late 1990s. For some of the more recent research, see Schultz (2002), Bleakley (2003, 2007), Behrman and Rosenzweig (2004), and Miguel and Kremer (2004).

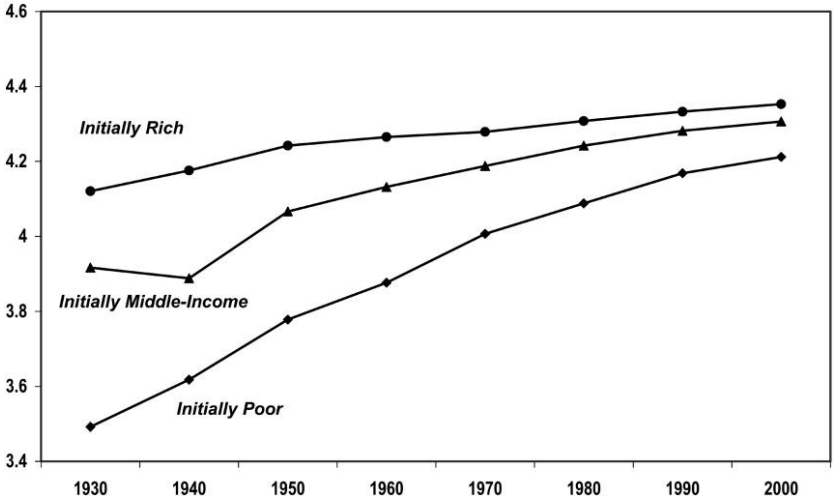

FIG. 1.—Log life expectancy at birth for initially rich, middle-income, and poor countries in the base sample.

countries.[2] Figure 1 shows this by plotting life expectancy in countries that were initially (circa 1940) poor, middle-income, and rich. It illustrates that while in the 1930s life expectancy was low in many poor and middle-income countries, this transition brought their levels of life expectancy close to those prevailing in richer parts of the world.[3] As a consequence, health conditions in many poor countries today, though still in dire need of improvement, are significantly better than the cor-

[2] The term "epidemiological transition" was coined by demographers and refers to the process of falling mortality rates after about 1850, associated with the switch from infectious to degenerative disease as the major cause of death (Omran 1971). Some authors prefer the term "health transition," since this includes the changing nature of ill health more generally (e.g., Riley 2001). We focus on the rapid decline in mortality (and improvement in health) in poorer countries after 1940, most of which was driven by the fast spread of new technologies and practices around the world (hence the adjective "international"). The seminal works on this episode include Stolnitz (1955), Omran (1971), and Preston (1975).

[3] This figure is for illustration purposes and should be interpreted with caution, since convergence is not generally invariant to nonlinear transformations. Our empirical strategy below does not exploit this convergence pattern; instead, it relies on potentially exogenous changes in life expectancy. In this figure and throughout the article, rich countries are those with income per capita in 1940 above the level of Argentina (the richest Latin American country at that time, according to Maddison's [2003] data, in our base sample). See App. table A1 for a list of initially rich, middle-income, and poor countries.

responding health conditions were in the West at the same stage of development.[4]

The international epidemiological transition provides us with an empirical strategy to isolate potentially exogenous changes in health conditions. The effects of the international epidemiological transition on a country's life expectancy were related to the extent to which its population was initially (circa 1940) affected by various specific diseases, for example, tuberculosis, malaria, and pneumonia, and to the timing of the various health interventions.

The early data on mortality by disease are available from standard international sources, though they have not been widely used in the economics literature. These data allow us to create an instrument for changes in life expectancy based on the preintervention distribution of mortality from various diseases around the world and the dates of global intervention (e.g., discovery and mass production of penicillin and streptomycin, or the discovery and widespread use of DDT against mosquito vectors). The only source of variation in this instrument, which we refer to as *predicted mortality*, comes from the interaction of baseline cross-country disease prevalence with global intervention dates for specific diseases. We document that there were large declines in disease-specific mortality following these global interventions. More important, we show that the predicted mortality instrument has a large and robust effect on changes in life expectancy starting in 1940, but has no effect on changes in life expectancy prior to this date (i.e., before the key interventions).

The instrumented changes in life expectancy have a fairly large effect on population: a 1 percent increase in life expectancy is related to an approximately 1.7–2 percent increase in population over a 40–60-year horizon. The magnitude of this estimate indicates that the decline in fertility rates was insufficient to compensate for increased life expectancy, a result that we directly confirm by looking at the relationship between life expectancy and total births.

However, we find no statistically significant effect on total GDP (though our two standard error confidence intervals do include economically significant effects). More important, GDP per capita and GDP per working age population show relative declines in countries experiencing large increases in life expectancy. In fact, our estimates exclude any positive effects of life expectancy on GDP per capita within 40- or 60-year horizons. This is consistent with the overall pattern in figure 2,

[4] For example, life expectancy at birth in India in 1999 was 60 compared to 40 in Britain in 1820, when income per capita was approximately the same level as in India today (Maddison 2001, 30). According to Maddison (264), income per capita in Britain in 1820 was $1,707, whereas it stood at $1,746 in India in 1998 (all figures in 1990 international dollars).

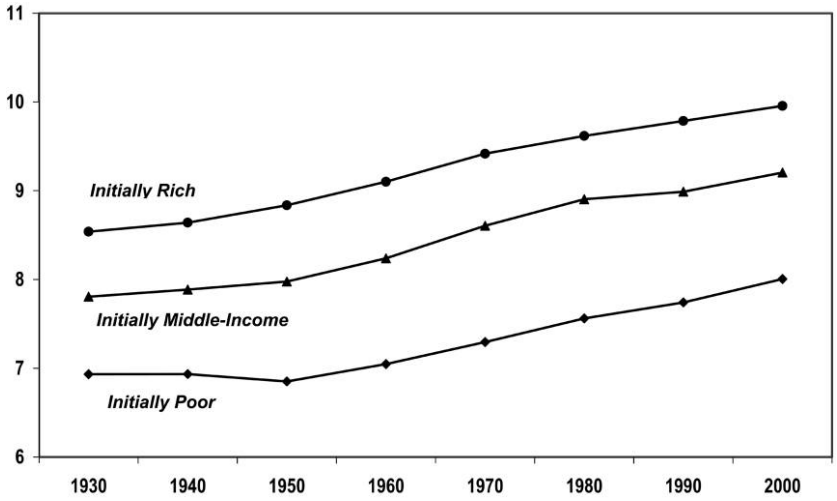

FIG. 2.—Log GDP per capita for initially rich, middle-income, and poor countries in the base sample.

which, in contrast to figure 1, shows no convergence in income per capita between initially poor, middle-income, and rich countries. We document that these results are robust to a range of specification checks and to the inclusion of various controls. We also document that our results are not driven by life expectancy at very early ages. The predicted mortality instrument has a large, statistically significant and robust effect on life expectancy at 20 (and at other ages), and using life expectancy at 20 instead of life expectancy at birth as our measure of general health conditions leads to very similar results.

The most natural interpretation of our results comes from neoclassical growth theory. Increased life expectancy raises population, which initially reduces capital-to-labor and land-to-labor ratios, thus depressing income per capita. This initial decline is later compensated by higher output as more people enter the labor force and as more capital is accumulated. This compensation can be complete and may even exceed the initial level of income per capita if there are significant productivity benefits from longer life expectancy. Yet, the compensation may also be incomplete if the benefits from higher life expectancy are limited and if some factors of production, for example, land, are supplied inelastically.

Our findings do not imply that improved health has not been a great benefit to less developed nations during the postwar era. The accounting approach of Becker, Philipson, and Soares (2005), which incorporates information on longevity and health as well as standards of living, would

suggest that these interventions have considerably improved "overall welfare" in these countries. What these interventions have not done, and in fact were not intended to do, is to increase output per capita in these countries.

Our article is most closely related to two recent contributions: Weil (2007) and Young (2005). Weil calibrates the effects of health using a range of micro estimates and finds that these effects could be quite important in the aggregate.[5] The major difference between Weil's approach and ours is that the conceptual exercise in his paper is concerned with the effects of improved health when population is held constant. In contrast, our estimates look at the general equilibrium effects of improved health from the most important health transition of the twentieth century, which takes the form of both improved health and increased life expectancy (and thus population). Young evaluates the effect of the recent HIV/AIDS epidemic in Africa. Using micro estimates and calibration of the neoclassical growth model, he shows that the decline in population resulting from HIV/AIDS may increase income per capita despite significant disruptions and human suffering caused by the disease.[6]

In addition, our work is related to the literature on the demographic transition both in the West and in the rest of the world, including the seminal contribution of McKeown (1976) and the studies by Arriaga and Davis (1969), Preston (1975, 1980), Caldwell (1986), Fogel (1986, 2004), Kelley (1988), and Deaton (2003, 2004). More recent work by Cutler and Miller (2005, 2006) finds that the introduction of clean water accounts for about half of the decline in U.S. mortality in the early twentieth century.

The rest of this article is organized as follows. In Section II, we present a simple model to frame the empirical investigation. Section III describes the health interventions and the data on disease mortality rates and life expectancy, which we constructed from a variety of primary sources. Section IV presents the ordinary least squares (OLS) relationships between life expectancy and a range of outcomes. Section V discusses the construction of our instrument and shows the first-stage relationships, robustness checks, falsification exercises, and other supporting evidence. Section VI presents our main results. Section VII presents a number of robustness checks and additional results, and Section VIII

[5] Weil's baseline estimate uses the return to the age of menarche from Knaul's (2000) work on Mexico as a general indicator of "overall return to health." Using Behrman and Rosenzweig's (2004) estimates from returns to birth weight differences in monozygotic twins, he finds smaller effects.

[6] For more pessimistic views on the economic consequences of HIV/AIDS, see Arndt and Lewis (2000), Bell, Devarajan, and Gersbach (2003), Forston (2006), and Kalemli-Ozcan (2006).

presents conclusions. Appendix A provides further information on data sources and data construction. Appendices B and C, which provide further details on data and historical sources, are available in the online edition and on request.

II. Motivating Theory and Estimating Framework

A. *Motivating Theory*

To frame the empirical analysis, we first derive the medium-run and long-run implications of increased life expectancy in the closed-economy neoclassical (Solow) growth model. Labor and land are supplied inelastically. We proxy all variables related to health in terms of life expectancy at birth (see below for more on this). Economy i has the constant returns to scale aggregate production function

$$Y_{it} = (A_{it}H_{it})^{\alpha}K_{it}^{\beta}L_{it}^{1-\alpha-\beta}, \tag{1}$$

where $\alpha + \beta \leq 1$, K_{it} denotes capital, L_{it} denotes the supply of land, and H_{it} is the effective units of labor given by $H_{it} = h_{it}N_{it}$, where N_{it} is total population (and employment) and h_{it} is human capital per person.

Without loss of any generality, we normalize $L_{it} = L_i = 1$ for all i and t. Let us also assume that life expectancy (or more generally health conditions) may increase output (per capita) through a variety of channels, including more rapid human capital accumulation or direct positive effects on total factor productivity (TFP).[7]

To capture these effects in a reduced-form manner, we assume the following isoelastic relationships:

$$A_{it} = \bar{A}_i X_{it}^{\gamma},$$

$$h_{it} = \bar{h}_i X_{it}^{\eta}, \tag{2}$$

where X_{it} is life expectancy in country i at time t, and $\bar{A}_i$ and $\bar{h}_i$ designate the baseline differences across countries. Finally, greater life expectancy also naturally leads to greater population (both directly and also potentially indirectly by increasing total births as more women live to childbearing age), so we posit

$$N_{it} = \bar{N}_i X_{it}^{\lambda}. \tag{3}$$

Now imagine the effect of a change in life expectancy from some

[7] On the potential effects of life expectancy and health on productivity, see Bloom and Sachs (1998). On their effects on human capital accumulation, see, e.g., Kalemli-Ozcan, Ryder, and Weil (2000), Kalemli-Ozcan (2002), or Soares (2005), which point out that when people live longer, they will have greater incentives to invest in human capital.

baseline value X_{it_0} at t_0 to a new value X_{it_1} at time t_1. First, suppose that while life expectancy changes (and, as a result, population, productivity, and human capital per worker change), the total capital stock remains fixed at some $\bar{K}_{it_0}$. In this case, substituting (2) and (3) into (1) and taking logs, we obtain the following log-linear relationship between log life expectancy, $x_{it} \equiv \log X_{it}$, and log income per capita, $y_{it} \equiv \log (Y_{it}/N_{it})$:

$$y_{it} = \beta \log \bar{K}_{it_0} + \alpha \log \bar{A}_i + \alpha \log \bar{h}_i - (1 - \alpha) \log \bar{N}_i + [\alpha(\gamma + \eta) - (1 - \alpha)\lambda] x_{it}, \quad (4)$$

for $t = t_0, t_1$. This equation shows that the increase in log life expectancy will raise income per capita if the positive effects of health on TFP and human capital, measured by $\alpha(\gamma + \eta)$, exceed the potential negative effects arising from the increase in population because of fixed land and capital supply, $(1 - \alpha)\lambda$.

Although land may be inelastically supplied even in the long run, the supply of capital will adjust as life expectancy, population, and productivity of the factors of production change. Equation (4) gives one extreme without such adjustment. The other extreme is the full adjustment of population and the capital stock to the change in life expectancy (which can in practice take longer than 40–60 years; see Ashraf, Lester, and Weil 2007). To model this possibility in the simplest possible way, suppose that country i has a constant saving rate equal to $s_i \in (0, 1)$ and capital depreciates at the rate $\delta \in (0, 1)$, so that the evolution of the capital stock in country i at time t is given by $K_{it+1} = s_i Y_{it} + (1 - \delta)K_{it}$. Suppose also that life expectancy changes from X_{it_0} to a new value X_{it_1} and remains at this level thereafter. After population and the capital stock have adjusted, the steady-state capital stock level will be $K_i = s_i Y_i/\delta$. Using this value of the capital stock together with (1), (2), and (3), we obtain the long-run relationship between log life expectancy and log income per capita as

$$y_{it} = \frac{\alpha}{1 - \beta} \log \bar{A}_i + \frac{\alpha}{1 - \beta} \log \bar{h}_i + \frac{\beta}{1 - \beta} \log s_i - \frac{\beta}{1 - \beta} \log \delta - \frac{1 - \alpha - \beta}{1 - \beta} \log \bar{N}_i + \frac{1}{1 - \beta} [\alpha(\gamma + \eta) - (1 - \alpha - \beta)\lambda] x_{it}, \quad (5)$$

again for $t = t_0, t_1$. This equation is similar to (4), except that it features the saving rate of country i, s_i, instead of its capital stock, and as a result of this adjustment, the effect of life expectancy on income is greater ("more positive"). Intuitively, capital now adjusts to the increase in population and productivity resulting from improvements in life expectancy. In fact, for industrialized economies in which land plays a small role in

production (because only a small fraction of output is produced in agriculture), $1 - \alpha - \beta \simeq 0$ would be a good approximation to reality. In this case, the potential negative effect of population disappears and the impact of log life expectancy on log income per capita is given by $\alpha(\gamma + \eta)/(1 - \beta) \geq 0$. However, for less developed economies in which a significant fraction of production is in the agricultural sector, the effect is still ambiguous and depends on the size of the externalities as measured by γ and η versus the negative effects of population, which are captured by the share of land in GDP, $1 - \alpha - \beta$, as well as the size of the population response, λ.[8]

B. *Estimating Framework*

Our estimating equation follows directly from (4) and (5). In particular, when an error term and potential covariates are added, these equations yield

$$y_{it} = \pi x_{it} + \zeta_i + \mu_t + \mathbf{Z}'_{it}\boldsymbol{\beta} + \varepsilon_{it}, \tag{6}$$

where y is log income per capita; x is log life expectancy (at birth);[9] the ζ_i's denote a full set of fixed effects that are functions of the parameters $\bar{A}_i$, $\bar{h}_i$, $\bar{N}_i$, and $\bar{K}_i$ (or s_i) in equations (4) and (5); the μ_t's incorporate time-varying factors common across all countries; and $\mathbf{Z}_{it}$ denotes a vector of other controls. The coefficient π is the parameter of interest, equal to $\alpha(\gamma + \eta) - (1 - \alpha)\lambda$ when (4) applies or to $[\alpha(\gamma + \eta) - (1 - \alpha - \beta)\lambda]/(1 - \beta)$ when (5) applies. Including a full set of country fixed effects, the ζ_i's, is important, since the country characteristics, $\bar{A}_i$, $\bar{h}_i$, $\bar{N}_i$, $\bar{K}_{it_0}$, and s_i, will be naturally correlated with life expectancy (or health) and thus with the error term ε_{it}. In addition, many other country-specific factors will simultaneously affect health and economic outcomes. Fixed effects at least remove the time-invariant components of these factors.

Motivated by equations (4) and (5), and since we do not expect the yearly or decadal changes in life expectancy to have their full effect on income per capita or on other economic variables, we will estimate (6) in *long differences*, that is, in a panel including only two dates, t_0 and

[8] See Galor and Weil (2000), Hansen and Prescott (2002), and Galor (2005) for models in which at different stages of development the relationship between population and income may change because of a change in the composition of output or technology. In these models, during an early Malthusian phase, land plays an important role as a factor of production and there are strong diminishing returns to capital. Later in the development process, the role of land diminishes, allowing per capita income growth. Hansen and Prescott, e.g., assume a Cobb-Douglas production function during the Malthusian phase with a share of land equal to 0.3.

[9] In view of eqq. (4) and (5) and the regression models used in the existing literature, we use log life expectancy on the right-hand side throughout. All the results reported in this article are very similar if we use the level of life expectancy instead (results available on request).

t_1 (in practice, 1940 and 1980 or 1940 and 2000). These long-difference regressions also make interpretation easier because they directly measure the effect of change in life expectancy between two dates on the change in economic variables between the same two dates. Since in the long-difference specification we have only two dates, (6) is also (algebraically) equivalent to estimating the first-differenced specification,

$$\Delta y_i = \pi \Delta x_i + \Delta \mu + \Delta \mathbf{Z}_i' \boldsymbol{\beta} + \Delta \varepsilon_i,$$

where the $\Delta y_i \equiv y_{it_1} - y_{it_0}$, and Δx_i, $\Delta \mu$, $\Delta \mathbf{Z}_i'$, and $\Delta \varepsilon_i$ are defined similarly.

Throughout, in addition to log income per capita, we look at a number of other outcome variables. They include log population, log births, and the age composition of the population, which will be informative to show the impact of the increase in life expectancy on population, fertility behavior, and also changes in age composition (which are important for interpreting the results related to GDP). They also include total GDP and GDP per working age population. The last variable is particularly important, since GDP per capita might be affected by changes in the "dependency ratio," defined as the ratio of nonactive population to total population (however, we will see that over 40- or 60-year horizons, there is little change in dependency ratios).

Finally, despite the presence of fixed effects controlling for fixed country characteristics such as $\bar{A}_i$, $\bar{h}_i$, $\bar{N}_i$, $\bar{K}_{it_0}$, and s_i, OLS estimates of (6) will not yield the causal effects of life expectancy (or health) on economic outcomes, because of the presence of potentially time-varying factors simultaneously affecting health and economic outcomes. For example, countries that increased their relative growth rates between 1940 and 1980 may have also invested more in health during this period, increasing life expectancy. More generally, societies that are able to solve their economic problems are also more likely to have solved their disease control problems. These considerations imply that the (population) covariance term Cov $(x_{it}, \varepsilon_{it})$ in (6) is not equal to zero, because even conditional on fixed effects, health is endogenous to economics. For this reason, our main focus will be on the instrumental variables (IV) estimates using the cross-country variation induced by the international epidemiological transition described in Section I. We next provide more details on this episode, on the data used in our study, and on our IV strategy.

III. Background and Data

A. *International Epidemiological Transition*

Despite early improvements in public health in western Europe, the United States, and a few other places from the mid-nineteenth century,

until 1940 there were limited improvements in health conditions in most of the Americas, Africa, and Asia and even in southern and eastern Europe.[10] In part, the reason was that there were few effective drugs against the major diseases in these areas, so most of the measures were relatively expensive public works (e.g., to drain swamps). Colonial authorities showed little enthusiasm for such expenditures.

The situation changed dramatically from around 1940 mainly because of three factors (see, e.g., Stolnitz 1955; Davis 1956; Preston 1975). First, there was a wave of global drug and chemical innovations. Many of these products offered cures effective against major killers in developing countries. The most important was the discovery and subsequent mass production of penicillin, which provided an effective treatment against a range of bacterial infections (National Academy of Sciences 1970; Easterlin 1999). Penicillin, which was used only in small quantities even in the most developed countries through the mid-1940s (Conybeare 1948, 66), became widely available by the early 1950s (see, e.g., Valentine and Shooter 1954).[11] Further antibiotic development quickly followed, most notably with the discovery of streptomycin, which was effective against tuberculosis. Between 1940 and 1950, the major bacterial killers became treatable and, in most cases, curable. Diseases that could now be treated, for most people without serious side effects, included pneumonia, dysentery, cholera, and venereal diseases. Antibiotics also reduced deaths indirectly caused by (and attributed to) viruses, such as influenza, which often kill by weakening the immune system and allowing secondary bacterial infections to develop.

Also important during the same period was the development of new vaccines, for example, against yellow fever.[12] The major chemical in-

[10] During the 1920s and 1930s, there were measures to reduce mortality from smallpox and cholera in Indonesia, smallpox and plague in the Philippines, malaria in India, and malaria and respiratory and diarrheal diseases in British Guiana (see, e.g., Mandle 1970; Preston 1980). Gwatkin (1980, 616) states that "such increases [in life expectancy] were modest compared with those that came later, for soon after World War II annual gains in life expectancy averaging over a year were recorded for periods of up to a decade in such diverse places as Taiwan, Malaysia, Sri Lanka, Mauritius, Jamaica, and Mexico." On public health improvements in western Europe and the United States, see, e.g., Cutler, Deaton, and Lleras-Muney (2006).

[11] Alexander Fleming isolated penicillin in the 1930s but could not produce it in any significant quantity; Howard Florey and Ernst Chain made the breakthroughs essential for the use of penicillin as a drug, and they shared the Nobel Prize with Fleming in 1945 (see, e.g., Chain 1980). The first large-scale use of penicillin was in 1943, by Allied armies in North Africa. Andrew Moyer's patent in 1948 is often regarded as a major step in its mass production. The invention of penicillin led to a wave of discovery of other antibiotics, including streptomycin, chloromycetin, aureomycin, and terramycin (National Academy of Sciences 1970, 147). Selman Waksman discovered streptomycin in 1944 and was awarded the Nobel Prize in 1952 (see Keers 1978).

[12] The yellow fever vaccine was invented by Max Theiler in 1930 and became widely available in the 1940s. Theiler was awarded a Nobel Prize in 1951. More vaccine inventions followed in the 1950s and 1960s (e.g., against smallpox and measles), but antibiotics already provided usually effective treatment against those diseases.

novation of this era was the discovery of DDT (dichlorodiphenyl trichloroethylene), which allowed a breakthrough in attempts to control one of the major killers of children in less developed regions of the world, malaria.[13] Aggressive use of inexpensive DDT led to the rapid eradication of malaria in Taiwan, much of the Caribbean, the Balkans, parts of northern Africa, northern Australia, and large parts of the South Pacific and all but eradicated malaria in Sri Lanka and India (see, e.g., Davis 1956).

The second pillar of the improvements in public health was the establishment of the World Health Organization, which greatly facilitated the spread of medical and public health technology to poorer countries. From the 1950s, the WHO, together with other United Nations–related bodies, most significantly, the United Nations International Children's Emergency Fund (UNICEF), was the driving force behind the public health (e.g., antimalaria campaigns) and immunization drives (e.g., against smallpox).[14]

The third factor was a change in international values. As Preston (1975) emphasizes, after the 1930s, "Universal values assured that health breakthroughs in any country would spread rapidly to all others where the means for implementation existed" (243).

These three factors combined caused a dramatic improvement in life expectancy in much of the world, especially in the lesser-developed parts of the globe, starting in the 1940s. Most new drugs, chemicals, and public health knowledge were available in almost all countries by 1950. As a result, by the late 1940s and early 1950s, there were significant improve-

[13] DDT was first synthesized in 1874, but the discovery of its insecticide properties occurred much later—in 1939, by Paul H. Müller; he received a patent for the insecticide in 1940 and was awarded a Nobel Prize in 1948 (Alilio, Bygbjerg, and Breman 2004, 270). Desowitz (1991), for example, describes the impact of DDT as follows: "There was nothing quite like [DDT] before and has been nothing quite like it since. Here was a chemical that could be sprayed on the walls of a house and for up to six months later any insect that alighted or rested on that wall would die. It was virtually without toxicity to humans. And, for the icing on the chemical cake, it was dirt-cheap to manufacture" (62–63).

[14] It is notable that Brazil and China, both poor countries at the time, took the initiative in pushing for the formation of the WHO (WHO 1998). A central goal of the organization was to diffuse medical practices and technology to poorer countries. Between the world wars, the League of Nations was responsible for international disease interventions and worked with other European organizations, e.g., against typhus in eastern Europe (see also Office International d'Hygiene Publique 1933). However, in contrast with the WHO, the League of Nations showed less interest in and had few resources for combating diseases in less developed countries, limiting itself to monitoring epidemics that might spread to the West.

On UNICEF, Lee et al. (1996, 303) report that "[created in 1946] Unicef was given the task of utilising its resources 'for child health purposes generally.' When the WHO came on to the scene two years later it was accepted that coordination on health matters was needed. This led to the creation of the WHO/Unicef joint committee on health policy, with the WHO, importantly, designated as the lead health organisation." The U.S. military also played a significant role in developing treatments for diseases such as cholera and in spreading the use of DDT and penicillin (Bhattacharya 1994).

ments in health conditions and life expectancy in Central America, South Asia, and parts of eastern and southern Europe compared to richer countries.

B. Coding Diseases

We collected comparable data on 15 of the most important infectious diseases across a wide range of countries and constructed cross-country mortality rates for these diseases before the 1940s. These 15 diseases are tuberculosis, malaria, pneumonia, influenza, cholera, typhoid, smallpox, whooping cough, measles, diphtheria, scarlet fever, yellow fever, plague, typhus fever, and dysentery/diarrhea-related diseases (see online App. B for more details). In all cases, the primary data source is national health statistics, as collected and republished by the League of Nations (until 1940) and the WHO and the United Nations (after 1945). We tried several different ways of constructing these data, all of which produce similar results.

In addition, we confirmed these quantitative assessments of geographic disease incidence with data and qualitative evidence in Lancaster (1990, esp. chap. 48), the maps and discussion of Cliff, Haggett, and Smallman-Raynor (2004), and the maps of disease incidence published by the American Geographical Society (1951*a*, 1951*b*, 1951*c*, 1951*d*) immediately after World War II. Appendices A and C provide details on sources and construction. Information on the etiology and epidemiology of each disease is obtained from the comprehensive recent surveys in Kiple (1993) and other sources (see App. B). We also checked that our data are comparable with those reported in Preston and Nelson (1974).

The other building block for our approach is global intervention dates for each specific disease, that is, dates of significant events potentially reducing mortality around the world from the disease in question. These events are described below (and in App. B), and the relevant dates were obtained from WHO Epidemiological Reports, as well as National Academy of Sciences (1970), Preston (1975), Kiple (1993), Easterlin (1999), and Hoff and Smith (2000).

Among the 15 diseases (in fact among all diseases), tuberculosis was the largest single cause of death around the world in 1940. It is primarily caused by *Mycobacterium tuberculosis*, transmitted through the air. Vaccination had been available from the 1920s, but the breakthrough cure was the 1944 invention of streptomycin.[15] This drug spread quickly and

[15] Previously tuberculosis could be treated by surgery, but even in the United Kingsom, resources for this were limited and not available to many patients (Conybeare 1948, 61). One discussant of Conybeare's paper made the point, based on data from the United Kingdom's Statistical Reviews, that when 1939 was compared with 1931–35, "in the general

has remained important. Following the above discussion of the invention and introduction of penicillin and streptomycin, we code the intervention against tuberculosis in the 1940s.

The other major cause of death was pneumonia, which results from a variety of infectious agents and toxins, including various bacterial and viral pathogens. Frequently, it appears as a secondary bacterial infection that causes death. The primary causes are often tuberculosis, influenza, and more recently AIDS. Antibiotics, for example, penicillin, proved highly effective against bacterial pneumonia in the 1940s (though by now resistant strains have developed).[16] Also, from the 1940s there were partially effective vaccines against pneumonia. In our baseline instrument, the intervention against pneumonia takes place in the 1940s.

The third most major disease at this time was malaria, which is caused by four types of parasites, transmitted by the bite of an infected female *Anopheles* mosquito. Control of mosquito vectors had been under way since the late nineteenth century but became much more effective with the discovery that DDT was an effective insecticide (see Expert Committee on Malaria 1947, 26–28). The use of DDT became widespread in the late 1940s (particularly following a successful demonstration in Greece) and was intensified following the 1955–57 WHO decision to campaign systematically to eradicate malaria (see Bradley 1992; WHO 2004).[17] In our baseline instrument, the intervention against malaria is taken to be the extensive use of DDT during the 1940s (chloroquine was also invented during the 1940s and quickly replaced mepacrine as the antimalarial drug of choice, until chloroquine-resistant parasites developed).[18]

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population tuberculosis had not recently been a decreasing risk at all" (81). This was on the eve of the dramatic impact of streptomycin (Keers 1978).

[16] Sulfonamides were also used against pneumonia but were soon superseded by penicillin (Conybeare 1948, 65; National Academy of Sciences 1970, 144–46). In any case, according to Conybeare's paper, these drugs were not widely available, even in the United Kingdom, until the very end of the 1930s.

[17] While it is generally accepted that DDT played a major role in the dramatic declines in the prevalence of malaria, there is some controversy in the demography literature about whether broader public health interventions of the 1940s were also essential (see, e.g., Langford 1996). Following the WHO campaign, it became apparent that some mosquitoes could develop resistance to insecticides. However, the view from the WHO was that, if used properly, spraying with DDT remained effective. E. J. Pampana (1954), chief of the Malaria Section of the WHO, called for a change in strategy, but this strategy still centered around insecticide spraying.

[18] Alternatively, one might take the major intervention against malaria to be the WHO campaign and thus code the date of global intervention as the 1950s. Our working paper (Acemoglu and Johnson 2006) shows that all the results reported here are robust to this alternate coding.

C. *Life Expectancy, Population, and GDP Data*

Other key variables for our investigation include life expectancy at birth, life expectancy at different ages, and total births, which are all obtained from historical UN data (various issues of the *Demographic Yearbook*) and League of Nations reports.[19] Since we need population and GDP data before World War II, we use the data from Maddison (2003). Postwar demographic data are taken from UN data sources (see App. A). We also constructed life expectancy at different ages for a subset of our base sample using these same UN sources. Results using life expectancy at age 20 are reported in Section VII.

Our full sample contains 75 countries from western Europe, Oceania, the Americas, and Asia, though when we restrict the sample to countries that have the relevant data for predicted mortality, life expectancy, and second-stage variables in 1940 and 1980 (or 2000) and when we exclude eastern Europe and Russia, our base sample consists of 47 countries.[20] Eastern Europe and Russia are excluded from the base sample because of concerns about the quality of their GDP data.[21] Because of a lack of reliable data on life expectancy in 1940, Africa is not in our base sample, though in Section VII we briefly discuss the robustness of our main results to including data from Africa.

We focus on 1940 and 1980 as our base sample. Post-1980 is excluded from our base sample because the emergence of AIDS appears to have led to a divergence in life expectancy between some poor countries and the richer nations.[22] In order to approximate the longer-run effects of health on economic outcomes, we also look at the changes between 1940 and 2000. In addition, we look at pre-1940 changes in our falsification exercises.

Table 1 provides basic descriptive statistics on the key variables (see also the raw data in App. table A1). Column 1 refers to the whole world,

[19] These data are often based on rough estimates. For example, life expectancy is calculated by combining data on age-specific death rates at a point in time, but often approximations are made using standard life tables (Lancaster 1990, chap. 3; Kiple 1993, 4: 4). Preston (1975) previously used some of the prewar data for the 1930s; see App. C.

[20] The 47 countries in our base sample are listed in App. table A1. In addition, we have data from 1950 onward (but not for 1940) on Algeria, Bolivia, Egypt, Iran, Iraq, Lebanon, Morocco, Singapore, South Africa, Tunisia, Turkey, and Vietnam. These countries are included in our panel regressions, e.g., in panel B of table 5 and table 6, but not in the long-difference regressions of tables 2 and 3, panel A of table 5, and tables 7–10. For two-stage least-squares (2SLS) results including these countries, see Acemoglu and Johnson (2006).

[21] The only communist country in our sample is China. Excluding China or including eastern European countries has no effect on any of our results (see Acemoglu and Johnson 2006).

[22] In addition, malaria reappeared in the 1970s and 1980s because of reduced international efforts, the international ban on the use of DDT, and the emergence of insecticide-resistant mosquitoes and drug-resistant strains of malaria. Tuberculosis has also returned as a secondary infection associated with AIDS.

TABLE 1
DESCRIPTIVE STATISTICS

	Whole World (1)	Base Sample (2)	Initially Rich Countries (3)	Initially Middle-Income Countries (4)	Initially Poor Countries (5)	Above-Median Change in Predicted Mortality 1940–80 (6)	Below-Median Change in Predicted Mortality 1940–80 (7)
Life expectancy at birth in 1900	30.90	37.59	49.36	36.92	28.77	31.50	43.95
	(8.83)	(10.31)	(3.67)	(8.13)	(5.42)	(5.71)	(10.26)
Life expectancy at birth in 1940	46.70	49.30	65.13	50.93	40.63	39.66	59.35
	(11.59)	(12.67)	(1.86)	(9.37)	(8.39)	(7.99)	(7.90)
Life expectancy at birth in 1980	61.13	67.60	74.30	69.66	61.92	62.91	72.49
	(11.02)	(7.41)	(1.13)	(4.57)	(7.18)	(7.28)	(3.24)
Life expectancy at age 20 in 1940		63.61	70.41	64.51	56.96	59.32	67.70
		(6.20)	(1.08)	(3.91)	(4.36)	(5.34)	(3.73)
Life expectancy at age 20 in 1980		73.08	75.73	73.59	70.27	71.40	74.69
		(2.89)	(.87)	(2.42)	(2.05)	(2.77)	(1.95)

Predicted mortality in 1940		.47	.17	.48	.53	.70	.23
		(.27)	(.05)	(.21)	(.32)	(.18)	(.08)
Log population in 1940	8.94	9.10	9.34	8.82	9.14	8.99	9.22
	(1.54)	(1.53)	(1.34)	(1.40)	(1.79)	(1.59)	(1.49)
Log population in 1980	8.88	9.81	9.76	9.44	10.00	9.93	9.68
	(1.62)	(1.47)	(1.29)	(1.25)	(1.75)	(1.48)	(1.48)
Log GDP in 1940	9.78	9.94	11.08	9.75	9.19	9.39	10.51
	(1.67)	(1.58)	(1.39)	(1.49)	(1.71)	(1.51)	(1.49)
Log GDP in 1980	9.99	11.59	12.47	11.41	10.88	11.09	11.98
	(1.98)	(1.48)	(1.33)	(1.35)	(1.52)	(1.43)	(1.43)
Log GDP per capita in 1940	7.64	7.73	8.64	7.84	6.95	7.30	8.19
	(.69)	(.72)	(.15)	(.33)	(.32)	(.51)	(.63)
Log GDP per capita in 1980	7.98	8.62	9.61	8.88	7.79	8.06	9.20
	(1.07)	(.95)	(.13)	(.44)	(.73)	(.82)	(.70)
Log GDP per working age population in 1940	8.19	8.27	9.03	8.36	7.51	7.86	8.71
	(.63)	(.63)	(.14)	(.30)	(.30)	(.50)	(.45)
Log GDP per working age population in 1980	9.13	9.18	10.04	9.40	8.36	8.65	9.75
	(.80)	(.85)	(.11)	(.39)	(.71)	(.79)	(.46)

NOTE.—The table reports the mean values of variables in the samples described in the column heading, with their standard deviations in parentheses. The base sample is 47 countries. Initially rich countries had log GDP per capita over 8.4 in 1940, middle-income countries had log GDP per capita betweeen 7.37 and 8.4, and low-income countries had log GDP per capita below 7.37 in 1940. Predicted mortality is measured per 100 per year. Columns 6 and 7 report descriptive statistics for subsamples in which change in predicted mortality between 1940 and 1980 was above or below median value in the base sample (−0.409). See the text and App. A for details and definitions.

and column 2 refers to our base sample. A comparison of these two columns indicates that, despite the absence of Africa from our base sample, averages of life expectancy, population, GDP, and GDP per capita are broadly similar between the whole world and our sample. Columns 3–5 show numbers separately for the three groups of countries used in figures 1 and 2—initially rich, middle-income, and poor countries (measured in terms of GDP per capita in 1940). These columns show the same patterns as figures 1 and 2: there is a large convergence in life expectancy among the three groups of countries between 1940 and 1980, but no convergence in GDP per capita. These columns also give information on predicted mortality, which will be our instrument for life expectancy. Columns 6 and 7 of this table will be discussed below.

IV. OLS Estimates

Tables 2 and 3 report OLS regressions of equation (6) for the main variables of interest listed at the end of Section II. These results are useful both to show the (conditional) correlations in the data and for comparison to the IV estimates reported below. All regressions in these tables and throughout the article (except some first-stage estimates) pertain to the long-difference specification as described in Section II.*B* above, with data for 1940 and 1980 or for 1940 and 2000.

Table 2 focuses on population-related outcomes. Panel A pertains to log population, panel B pertains to log births (we do not have the data necessary to compute fertility rates), and panel C pertains to the age composition of the population measured by the percentage of the population under the age of 20. Column 1 includes all countries for which we have the relevant data. The remaining columns focus on our base sample, consisting of countries for which we can construct predicted mortality rates.

A number of features are notable in table 2. First, the "whole-world" sample gives results very similar to those of our base sample for 1960–2000. Second, the results in our base sample for 1960–2000 are also similar to the results for 1940–80. For example, in panel A the effect of log life expectancy on log population in column 1 is 1.6 (standard error 0.30), whereas in our base sample over the same time period, the same coefficient is estimated as 1.75 (0.40). In column 3, when we focus on our main sample period, 1940–80, the estimate is 1.62 (0.19). The magnitudes of these estimates are reasonable. They suggest that a 1 percent increase in life expectancy is associated with a 1.6–1.75 percent increase in population. If births are held constant, a 1 percent increase in life expectancy would be associated with a 1 percent increase in population (since each individual would live for 1 percent longer). Naturally, an increase in life expectancy is also associated with an increase

TABLE 2
LIFE EXPECTANCY, POPULATION, BIRTHS, AND PERCENTAGE OF POPULATION UNDER 20: OLS ESTIMATES

	Whole World	Base Sample		Low- and Middle-Income Countries Only	Base Sample	Low- and Middle-Income Countries Only
	(1)	(2)	(3)	(4)	(5)	(6)
	A. Dependent Variable: Log Population					
	Just 1960 and 2000	Just 1960 and 2000	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 2000	Just 1940 and 2000
Log life expectancy	1.60	1.75	1.62	1.86	2.01	2.25
	(.30)	(.40)	(.19)	(.26)	(.22)	(.32)
Number of countries	120	59	47	36	47	36
	B. Dependent Variable: Log Number of Births					
	Just 1960 and 1990	Just 1960 and 1990	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1990	Just 1940 and 1990
Log life expectancy	2.09	2.01	2.35	2.57	2.19	2.66
	(.37)	(.40)	(.27)	(.40)	(.27)	(.42)
Number of countries	115	47	45	34	45	34
	C. Dependent Variable: Percentage of Population under Age 20					
	Just 1960 and 2000	Just 1960 and 2000	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 2000	Just 1940 and 2000
Log life expectancy	.045	.045	.094	.124	.053	.132
	(.087)	(.087)	(.029)	(.042)	(.038)	(.058)
Number of countries	40	40	40	29	40	29

NOTE.—OLS regressions with a full set of year and country fixed effects. Robust standard errors are reported in parentheses. Long-difference specifications with two observations per country, one for the intial date and one for the final date. In all regressions the independent variable is the log of life expectancy at birth. "Whole world" is the set of countries for which we have data on the variables in the regression shown. The base sample is the set of countries for which we can estimate 2SLS regressions. The assignment of countries to the low-, middle-, and high-income categories is based on income per capita levels for 1940. See the text and App. A for definitions and details.

in births, since more women survive to childbearing age, so we should expect a somewhat larger effect than 1 percent. The results in panel B, which show a significant increase in total number of births associated with the increase in life expectancy, confirm this interpretation. In particular, a 1 percent increase in life expectancy is associated with a 2–2.7 percent increase in total births.

Column 4 reports estimates for the sample of initially low- and middle-income countries (as defined in App. table A1). This subsample is useful for verifying that our results are not driven by a comparison of initially rich to initially low- and middle-income countries. The association between life expectancy and population (and life expectancy and births) is slightly stronger in this sample than in the base sample.

Columns 5 and 6 look at 1940 and 2000 rather than 1940 and 1980 as in our baseline specification. The longer window is useful to gauge whether longer-run effects are different from those that can be detected in a 40-year period. In panel A, there is a slightly stronger association

TABLE 3
LIFE EXPECTANCY, GDP, GDP PER CAPITA, AND GDP PER WORKING AGE POPULATION: OLS ESTIMATES

	WHOLE WORLD: Just 1960 and 2000 (1)	BASE SAMPLE: Just 1960 and 2000 (2)	BASE SAMPLE: Just 1940 and 1980 (3)	LOW- AND MIDDLE-INCOME COUNTRIES ONLY: Just 1940 and 1980 (4)	BASE SAMPLE: Just 1940 and 2000 (5)	LOW- AND MIDDLE-INCOME COUNTRIES ONLY: Just 1940 and 2000 (6)
	A. Dependent Variable: Log GDP					
Log life expectancy	1.17	1.55	.78	.65	.85	.43
	(.56)	(.35)	(.33)	(.42)	(.28)	(.38)
Number of countries	120	59	47	36	47	36
	B. Dependent Variable: Log GDP per Capita					
Log life expectancy	−.42	−.19	−.81	−1.17	−1.14	−1.79
	(.58)	(.54)	(.26)	(.38)	(.27)	(.41)
Number of countries	120	59	47	36	47	36
	C. Dependent Variable: Log GDP per Working Age Population					
Log life expectancy	−1.01	−1.03	−.78	−1.10	−1.26	−1.78
	(.60)	(.60)	(.26)	(.38)	(.24)	(.38)
Number of countries	51	47	46	35	46	35

NOTE.—OLS regressions with a full set of year and country fixed effects. Robust standard errors are reported in parentheses. Long-difference specifications with two observations per country, one for the intial date and one for the final date. In all regressions the independent variable is the log of life expectancy at birth. "Whole world" is the set of countries for which we have data on the variables in the regression shown. The base sample is the set of countries for which we can estimate 2SLS regressions. The assignment of countries to the low-, middle-, and high-income categories is based on income per capita levels for 1940. See the text and App. A for definitions and details.

between life expectancy and population from 1940 to 2000 than from 1940 to 1980 (e.g., the base sample estimate now increases to 2.01, with a standard error of 0.22).

Panel B shows the estimates for the log number of births. The various specifications show a robust and statistically significant 2–2.6 percent increase in total births in response to a 1 percent increase in life expectancy.

Finally, panel C shows that in our base sample increases in life expectancy are associated with an increase in the ratio of the population that is under the age of 20, though the magnitude of the effect is not very large. For example, the estimate in column 3 (0.094) indicates that a 10 percent increase in life expectancy will be associated with a one-percentage-point increase in the fraction of the population that is under the age of 20. This implies that the relationship between life expectancy and working age population is very similar to that between life expectancy and total population.

Table 3 presents results that parallel those in table 2, but now the dependent variables are log GDP, log GDP per capita, and log GDP per

working age population.[23] The structure of the table is identical to that of table 2. Panel A shows a positive relationship between log life expectancy and log GDP. For example, the results in columns 1 and 2 indicate that a 1 percent increase in life expectancy is associated with a 1.2–1.5 percent increase in GDP. Notably, the effect of life expectancy on GDP is much reduced when we focus on our base sample for 1940–80 (col. 3). This is exactly what one would expect if a larger fraction of changes in life expectancy is driven by exogenous factors in this sample than in the samples for columns 1 and 2.[24]

While panel A shows a positive relationship between life expectancy and total income, panels B and C show that this increase in total GDP is insufficient to compensate for the increase in total population and working age population. As a result, there is a negative (sometimes significant) relationship between GDP per capita and GDP per working age population and life expectancy. There is no evidence of a positive effect of life expectancy on GDP per capita in table 3. Nevertheless, since these estimates are not necessarily causal, the true effect of life expectancy on income per capita might be larger or smaller than those shown in table 3. The rest of the article investigates this question.

V. Predicted Mortality and First Stages

Because of reverse causality and omitted variable problems, OLS estimates of (6) are unlikely to uncover the causal effect of life expectancy on economic variables. We now outline a source of exogenous variation in life expectancy that may help us estimate these causal effects.

A. The Predicted Mortality Instrument

Prior to the international epidemiological transition, there was considerable variation in the prevalence of diseases across the world. For example, during the 1940s, while malaria was endemic in parts of South Asia and Central America, it was relatively rare in much of western Europe and in the Southern Cone of Latin America. We therefore ex-

[23] We define working age population as population between the ages of 15 and 60. Estimates of the age distribution of the population and hence of the working age population for this time period are often rough.

[24] In particular, OLS estimates of the effect of log life expectancy on log GDP (or log GDP per capita or log GDP per working age population) will be typically biased upward because of reverse causality and common shocks to income and health. If much of the change in life expectancy in our base sample between 1940 and 1980 comes from exogenous variation due to the international epidemiological transition, then this upward bias will be reduced. The reduction of the coefficient on log life expectancy from 1.55 to 0.78 between cols. 2 and 3 in table 3 likely reflects this change in the composition of the source of variation in life expectancy between these two samples.

pect variation in the effects of global interventions on life expectancy in different countries depending on the baseline distribution of diseases. For example, DDT should reduce malarial infections and mortality and increase life expectancy in Central America and South Asia relative to western Europe or the Southern Cone of Latin America.

Motivated by this reasoning, our instrument, *predicted mortality*, is constructed as

$$M_{it}^{I} = \sum_{d \in \mathcal{D}} [(1 - I_{dt})M_{di40} + I_{dt}M_{dFt}], \tag{7}$$

where M_{dit} denotes mortality in country i from disease d at time t, I_{dt} is a dummy for intervention for disease d at time t (it is equal to one for all dates after the intervention), and $\mathcal{D}$ denotes the set of the 15 diseases listed above. It is measured as the number of deaths per 100 individuals per year. The term M_{di40} refers to the preintervention mortality from disease d in the same units, and M_{dFt} is the mortality rate from disease d at the health frontier of the world at time t. In our baseline instrument, we take M_{dFt} to be equal to zero.[25] Predicted mortality, M_{it}^{I}, thus uses a country's initial mortality rate from the 15 diseases until there is a global intervention; after the global intervention, the mortality rate from the disease in question declines to the frontier mortality rate.

We then use our measure of predicted mortality, M_{it}^{I}, as an instrument for life expectancy in the estimation of (6). In particular, we posit the following first-stage relationship between log life expectancy and predicted mortality:

$$x_{it} = \psi M_{it}^{I} + \tilde{\zeta}_i + \tilde{\mu}_t + \mathbf{Z}_{it}'\tilde{\boldsymbol{\beta}} + u_{it}. \tag{8}$$

The key exclusion restriction for our IV strategy is $\mathrm{Cov}(M_{it}^{I}, \varepsilon_{it}) = 0$, where recall that ε_{it} is the error term in the second-stage equation, (6).

Equation (7) makes it clear that the only source of variation in predicted mortality comes from the interaction of the baseline distribution of diseases with global interventions (in particular, note that M_{di40} applies until the time of the relevant global intervention). Whether a country has successfully eradicated a disease or has been quick at adopting international technologies will have no effect on M_{it}^{I}; the dummy I_{dt} turns on for all countries at the same time. This makes our exclusion restriction $\mathrm{Cov}(M_{it}^{I}, \varepsilon_{it}) = 0$ plausible. Since variations in M_{it}^{I} are unrelated to any actions or economic events in the country, there is no obvious reason for it to be correlated with economic or population shocks in the country in question.

[25] We also calculated an alternative measure of predicted mortality using the average mortality rate from disease d at time t among the richest countries, but since these rates are close to zero, this alternative measure is very similar to our baseline predicted mortality series and yields identical results.

The only potential threat to the exclusion restriction would be that the baseline mortality rates, the M_{di40}'s, are correlated with future changes in population or income. To show that this is unlikely to be the case, we will show the robustness of our IV results to the inclusion of differential trends that are parameterized as functions of various baseline characteristics (see eqq. [11] and [13] below). In addition, we will report a range of falsification exercises illustrating that the variable M^I_{it} has no predictive power for life expectancy or other economic variables before the international epidemiological transition.

B. *Alternative Instruments*

We also constructed a number of alternative instruments to investigate the robustness of our results. The first is the *global mortality instrument*,

$$M^I_{it} = \sum_{d \in \mathcal{D}} \frac{M_{dt}}{M_{d40}} M_{di40}, \tag{9}$$

where M_{di40} denotes mortality in country i from disease d in 1940, and M_{dt} (M_{d40}) is global mortality from disease d in year t (1940), calculated as the unweighted average across countries in our sample. The advantage of this instrument is that it does not use any information on global intervention dates, instead relying on aggregate changes in worldwide disease-specific mortality rates.[26] The estimates using the global mortality instrument therefore show that none of our results depend on the coding of intervention dates.

We also constructed alternative instruments using different (reasonable) timings of interventions, especially whenever there was any potential doubt about the exact dates. In addition, we experimented with an instrument constructed using only the three big killers: malaria, tuberculosis, and pneumonia. The results with these alternative instruments are very similar to the baseline estimates and are not reported to save space (see Acemoglu and Johnson 2006).

C. *Zeroth-Stage Estimates*

Our approach is predicated on the notion that global interventions reduce mortality from various diseases. Therefore, before documenting the first-stage relationship between our predicted mortality measure and log life expectancy, we show the effect of various global interventions

[26] Constructing this instrument requires us to track all diseases through changes in the classification of death over time. As explained further in App. A, this is not possible for dysentery/diarrhea-related diseases or yellow fever, which are therefore excluded from the global mortality instrument.

on mortality from specific diseases. In this exercise, in addition to the available data on the infectious diseases listed above, we also use deaths from cancers and malignant tumors as a control disease, since these were not affected by the global interventions.[27]

Table 4 reports the estimates from the following "zeroth-stage regression":

$$M_{idt} = \theta I_{dt} + \mu_t + \pi_d + \delta_i + v_{it}. \tag{10}$$

The dependent variable is mortality in country i from disease d at time t, and the regression includes a full set of time, disease, and country dummies. The coefficient of interest, θ, measures whether there is a decline in mortality from a specific disease associated with an intervention.

Table 4 reports estimates of equation (10). In all cases, as expected, the estimate of θ is negative and significant. For example, in column 1, θ is estimated to be -24.15 (standard error 5.67), which indicates an average reduction of 24 deaths per 100,000 population due to the interventions. In column 2, when we add lagged intervention, the coefficient on the intervention dummy is largely unchanged (-24.47), and the lagged intervention itself is also significant, likely reflecting the gradual diffusion of global interventions within our sample (recall that the intervention date corresponds to the time of the major global breakthrough).

More challenging is the specification in column 3, which includes contemporaneous and lead interventions. This specification investigates whether it is the interventions or preexisting trends that are responsible for the declines in mortality. It is reassuring that the estimate of the negative coefficient on contemporaneous intervention, θ, is unaffected, and lead intervention has an insignificant coefficient, with the opposite (positive) sign of about a third of the magnitude of the effect of contemporaneous intervention. These results therefore show that mortality from specific diseases around the world fell sharply following the global health interventions, but not before.

Columns 4–7 investigate whether one of the main diseases is responsible for the results in columns 1–3, by excluding tuberculosis, pneumonia, malaria, and influenza one at a time. Without tuberculosis or pneumonia, which were the most major diseases of this era, the coefficient estimates are somewhat smaller but still highly significant (-17.72

[27] The zeroth-stage regressions are estimated on an unbalanced panel going back to 1930. The 1930 data enable us to look for potential lead effects. For the reasons noted in n. 26, we do not have sufficient data to include yellow fever and dysentery/diarrhea-related diseases in table 4 (see App. A for details).

TABLE 4

EFFECT OF INTERVENTIONS ON DISEASE MORTALITY, ZEROTH STAGE: PANEL REGRESSIONS 1930–60

Dependent Variable: Mortality per 100,000 from Disease *i* in Country *j* at Period *t*

	BASE SAMPLE			WITHOUT TUBERCULOSIS	WITHOUT PNEUMONIA	WITHOUT MALARIA	WITHOUT INFLUENZA
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Intervention	−24.15	−24.47	−22.78	−17.72	−18.59	−26.41	−25.16
	(5.67)	(5.19)	(6.11)	(5.14)	(5.25)	(5.58)	(5.78)
Lagged intervention		−18.81					
		(4.25)					
Lead intervention			7.27				
			(4.14)				
R^2	.47	.48	.47	.49	.49	.49	.48
Observations	1,723	1,723	1,723	1,577	1,613	1,610	1,578

NOTE.—OLS regressions with a full set of disease, year, and country fixed effects. Robust standard errors, adjusted for clustering by country-disease pair, are in parentheses. Unbalanced panels with data for 1930, 1940, 1950, and 1960. Dependent variable is deaths per 100,000 from disease *i* in country *j* at year *t*. Base sample is 13 infectious diseases plus cancer and malignant tumors for which data are available (this excludes dysentery/diarrhea and yellow fever). Independent variables: dummy for intervention (e.g., for tuberculosis equals one for 1950 and 1960, zero otherwise), dummy for lead intervention (e.g., for tuberculosis equals one for 1940, 1950, and 1960), and dummy for lagged intervention (e.g., for tuberculosis equals one for 1960).

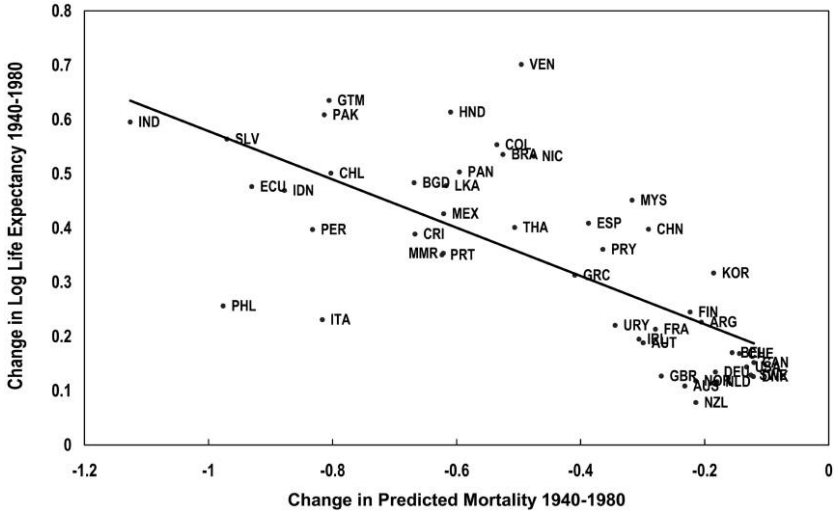

FIG. 3.—Change in log life expectancy and change in predicted mortality, 1940–80, base sample.

and −18.59, with standard errors of 5.14 and 5.25, respectively).[28] Without malaria or influenza, the coefficient estimates are very similar to the baseline estimates.

D. First-Stage Estimates

We next turn to the first-stage relationship between life expectancy and predicted mortality. While the zeroth-stage regression in equation (10) is at the disease-country-time level, the structural relationships of interest, captured in (6), and thus our first-stage relationships are at the country-time level.

Figure 3 shows the first-stage relationship visually. The horizontal axis depicts the change in predicted mortality between 1940 and 1980, and the vertical axis shows the change in log life expectancy during the same time period. A strong negative relationship is clearly visible in figure 3.

[28] Tuberculosis and pneumonia were much more important than the other diseases as major causes of death at this time and also accounted for a very large fraction of the decline in mortality during this episode. For example, in our base sample the (unweighted) cross-country average of deaths per 100,000 due to tuberculosis was 177.24 in 1940 and declined to 26.90 in 1960 (a decline of over 150 deaths per 100,000). The same numbers for pneumonia were 208.14 in 1940 and 62.07 in 1960 (a decline of 146 deaths per 100,000). Both the death rates in 1940 and the declines are much smaller for other diseases. For example, the decline between 1940 and 1960 was just under 20 deaths per 100,000 for malaria; just over six deaths per 100,000 for typhoid; approximately four deaths per 100,000 for influenza, smallpox, and cholera; and much smaller for the remaining diseases.

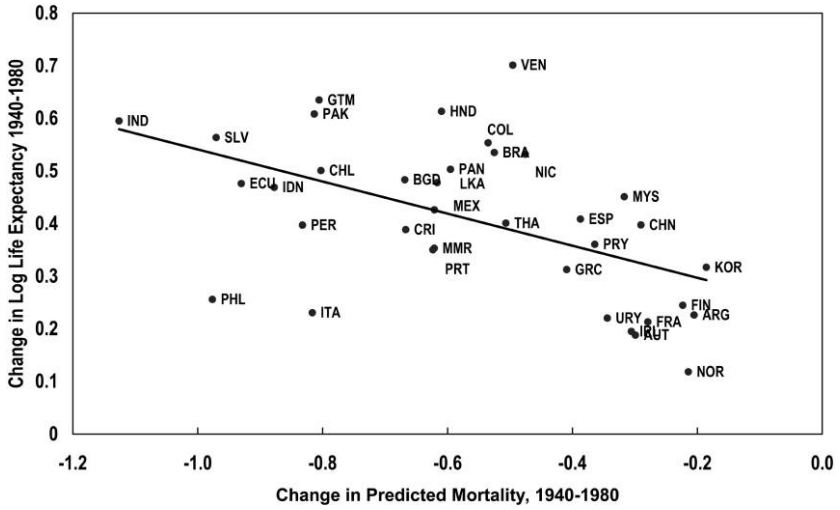

FIG. 4.—Change in log life expectancy and change in predicted mortality, 1940–80, low- and middle-income countries.

Predicted mortality declined by a large amount in India, the Philippines, Indonesia, and parts of Central America, while remaining largely unchanged in parts of western Europe, Uruguay, Argentina, Korea, Australia, and New Zealand. Life expectancy, in turn, increased by a large amount in the first group of countries and much less in the second group. The pattern shown in figure 3 can also be seen in table 1, columns 6 and 7. These columns show the descriptive statistics for countries with above- and below-median changes in predicted mortality between 1940 and 1980. The second and the third rows show that there is a much larger increase in life expectancy at birth (over 22 years) for countries with above-median changes in predicted mortality than for those with below-median changes (a change of 13 years).

Figure 4 depicts the same relationship without the richest countries. It shows that the first-stage relationship is not driven by the comparison of initially rich countries to initially low- and middle-income countries.[29]

[29] Predicted mortality has a similar effect on life expectancy at different ages (see table 10 below for life expectancy at 20). It also has an impact on infant mortality, though this relationship is somewhat less robust. In particular, change in predicted mortality between 1940 and 1980 reduces infant mortality between 1940 and 1980, but this effect becomes statistically significant only when we look at infant mortality between 1940 and 2000. Moreover, if we look at log infant mortality rather than the level of infant mortality, the sign of the relationship is reversed, largely because there are some countries with relatively large increases in life expectancy that had relatively small falls in infant mortality and also because many rich economies experienced large proportional declines in infant mortality (though much smaller changes in life expectancy); see, e.g., Lancaster (1990, chap. 32). This pattern is not entirely surprising in view of the fact that the main killers of this era,

Table 5 shows the first-stage relationship in regression form by estimating equation (8). Panel A reports long-difference specifications, which are similar to the OLS regressions reported in tables 2 and 3. For completeness and comparison, panel B reports panel regressions, with each observation corresponding to a decade. These regressions always include country and year dummies, and we report standard errors that are fully robust against serial correlation at the country level (e.g., Wooldridge 2002, 275).

Column 1 includes all countries for which we have life expectancy and predicted mortality data. It shows an estimate of ψ equal to -0.39 with a standard error of 0.07, which is significant at less than 1 percent. Column 2 pertains to our base sample and will be the first stage corresponding to our main 2SLS regressions in tables 8 and 9. The estimate of ψ is now -0.45 (0.06), which is again significant at less than 1 percent.[30] This estimate implies that an improvement in predicted mortality of 0.47 (per 100 or 470 per 100,000, which is the mean improvement between 1940 and 1980 in our base sample) leads approximately to a 21 percent increase in life expectancy (mean life expectancy in our sample in 1940 was 49.30, so this is an increase of about 10.5 years, whereas the actual mean improvement in life expectancy between 1940 and 1980 was 17 years). This implies that changes in predicted mortality account for almost two-thirds of the increase in life expectancy between 1940 and 1980. Perhaps more important, 10.5 years is approximately equal to the decline in the gap between initially rich versus initially poor and middle-income countries, so that the closing of the health gap during this time period appears to be almost entirely accounted for by the variation driven by the international epidemiological transition.

Column 3 repeats the same regression for 1940 and 2000. Now the estimate of ψ is slightly larger, -0.56 (0.07). Column 4 looks at only low- and middle-income countries. The estimate of ψ is slightly smaller and less precise than in column 2, but still significant at less than 1 percent (-0.31, with a standard error of 0.08).

Panel B repeats the same regressions using a panel with decadal observations. The results are still highly significant but slightly smaller, which is reasonable since these regressions exploit shorter-run responses to changes in predicted mortality.

As noted above, a major concern regarding the validity of our instrument is its potential correlation with baseline country characteristics.

tuberculosis, pneumonia, and malaria, affected mainly adults and children above the age of 1.

[30] Since the *t*-statistics in the basic first-stage relationships are above five, there is no issue of weak instruments; in the 2SLS regressions below we use the standard Wald confidence intervals (see, e.g., Stock, Wright, and Yogo 2002).

Whether this explains the first-stage relationship is investigated in columns 6–8. These columns report regressions of the form

$$x_{it} = \psi M^I_{it} + \tilde{\zeta}_i + \tilde{\mu}_t + \sum_{t=1940}^{1980} \mathbf{c}_i' \tilde{\boldsymbol{\omega}}_t + u_{it}, \tag{11}$$

where $\mathbf{c}_i$ denotes "time-invariant" characteristics of country i, in particular, either a measure of average quality of institutions (computed as the average of the constraints on the executive from the Polity IV data set over 1950–70) in column 5, the 1930 value of GDP per capita in column 6, or a vector of continent dummies in column 7. Since equation (11) includes a full set of time interactions with $\mathbf{c}_i$, differential trends related to these characteristics are taken out. In long-difference regressions reported in panel A, this specification is equivalent to including an interaction between the 1980 (or the 2000) dummy and the baseline characteristics.

The results in both panels of table 5 show that controlling for these characteristics has little effect on our results. For example, the coefficient estimate in column 5, panel A, is −0.35 (0.07), which is slightly smaller than the baseline in column 2 but still significant at less than 1 percent. The coefficient estimates in columns 6 and 7 are −0.25 and −0.30 and are both statistically significant at less than 1 percent. The results in panel B are similar.

Finally, columns 8 and 9 report results using the global mortality instrument defined in (9). Once again, the results are similar. For example, the estimate of ψ for the base sample in column 8 of panel A is −0.46 (0.10), and the estimate for low- and middle-income countries is −0.31 (0.13), both of which are very close to the results in columns 2 and 4.

Overall, the results in table 5 show a large and robust effect of the predicted mortality instrument on life expectancy. We next investigate the robustness of these results further.

E. Mean Reversion, Lags, and Leads

The specifications in table 5 do not allow for mean reversion in life expectancy and also assume that it is contemporaneous predicted mortality that affects life expectancy. In more general specifications we may find that it is the lags or leads of predicted mortality that affect life expectancy. In particular, if it is the leads of (future changes in) predicted mortality that affect life expectancy, this would cast doubt on our interpretation of the first-stage relationship. Table 6 investigates these issues using the specifications with decadal observations from panel B of table 5. Column 1 repeats our baseline specification (from col. 2 of

TABLE 5
First-Stage Estimates: Predicted Mortality and Life Expectancy
Dependent Variable: Log Life Expectancy

	Baseline Predicted Mortality							Using Global Mortality Rate	
	All Countries (1)	Base Sample (2)	Base Sample (3)	Low- and Middle-Income Countries Only (4)	Base Sample: Interaction with Institutions (5)	Base Sample: Interaction with Initial (1930) log GDP per Capita (6)	Base Sample: Interaction with Continent Dummies (7)	Base Sample (8)	Low- and Middle-Income Countries Only (9)
	A. Long Differences								
	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 2000	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980
Predicted mortality	−.39	−.45	−.56	−.31	−.35	−.25	−.30	−.46	−.31
	(.07)	(.06)	(.07)	(.08)	(.07)	(.09)	(.07)	(.10)	(.13)
R^2	.93	.95	.95	.95	.96	.96	.96	.95	.95
Number of observations	150	94	94	72	94	94	94	94	72
Number of countries	75	47	47	36	47	47	47	47	36

	B. Panel Regressions								
	1940–80	1940–80	1940–2000	1940–80	1940–80	1940–80	1940–80	1940–80	1940–80
Predicted mortality	−.29	−.33	−.41	−.23	−.27	−.24	−.25	−.41	−.26
	(.06)	(.06)	(.06)	(.07)	(.06)	(.09)	(.06)	(.07)	(.09)
R^2	.93	.93	.91	.93	.94	.95	.96	.93	.93
Number of observations	405	283	401	228	271	243	283	263	208
Number of countries	84	59	59	48	56	49	59	59	48

NOTE.—OLS regressions with a full set of year and country fixed effects. Robust standard errors, adjusted for clustering, are in parentheses. Panel A contains long-difference specifications with two observations per country, one for the initial date and one for the final date. Panel B contains unbalanced panel regressions with one observation per country and per decade. The dependent variable is the log of life expectancy at birth. "All countries" are those for which we have disease data; base sample countries are those not missing data on second-stage outcome variables. Columns 1–7 use baseline predicted mortality as the independent variable, and cols. 8 and 9 use the predicted global mortality. See the text and App. A for the construction of the predicted mortality instrument, definitions, and data sources. Countries are assigned to the low- and middle-income categories on the basis of 1940 income per capita. Regressions in cols. 5–7 also include year dummies with the following interactions: in col. 5, institutions measured as constraints on the executive in 1950, 1960, and 1970 from Polity IV; in col. 6, the log of GDP per capita in 1930; and in col. 7, a full set of continent dummies (Africa, Asia, Americas, and Europe; Oceania is the omitted category).

TABLE 6
First-Stage Estimates: Mean Reversion and Robustness (Panel Regressions, 1940–80)
Dependent Variable: Log of Life Expectancy, in Regressions on the Base Sample

	OLS		Lagged Life Expectancy Instrumented by Second Lag of Life Expectancy	GMM (Arellano-Bond)	OLS				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Predicted mortality	−.33	−.18	−.27	−.19	−.20	−.33	−.20	−.31	−.14
	(.06)	(.08)	(.14)	(.06)	(.06)	(.08)	(.07)	(.06)	(.08)
Lagged log life expectancy		.44	.32	.71					.45
		(.09)	(.39)	(.06)					(.09)
Lagged predicted mortality					−.17		−.17		
					(.03)		(.03)		
Lead predicted mortality						.19	.14		
						(1.04)	(1.04)		
Lagged log GDP per capita								−.06	−.07
								(.04)	(.02)
p-value of test for second-order autocorrelation				.83					
Hansen *J*-test (*p*-value)				.014					
R^2	.93	.95	.95		.94	.93	.95	.93	.95
Number of observations	283	267	231	248	283	283	283	273	257
Number of countries	59	59	57	59	59	59	59	59	59

Note.—OLS (cols. 1–2 and 5–9) and 2SLS (cols. 3–4) regressions with a full set of year and country fixed effects. Robust standard errors, adjusted for clustering by country, are in parentheses. All columns are unbalanced panels with one observation per decade, per country, using base sample countries. The dependent variable is the log of life expectancy at birth. Lagged values are 10 years earlier and lead predicted mortality is 10 years ahead. Assignment of countries to low-, middle-, and high-income categories is based on 1940 income per capita. In col. 3, the second lag of log life expectancy is used as an instrument for lagged log life expectancy. In col. 4, GMM (Arellano-Bond) uses all available lags of log life expectancy as instruments.

panel B in table 5). Column 2 reports OLS estimates from the following model:

$$x_{it} = \nu x_{it-1} + \psi M^I_{it} + \delta'_i + \mu'_t + u_{it}, \tag{12}$$

which allows lagged log life expectancy to affect current log life expectancy. There is indeed evidence for mean reversion; the coefficient ν in the top panel is estimated to be 0.44 (0.09). Nevertheless, the negative relationship between predicted mortality and life expectancy remains. The parameter of interest, ψ, is now estimated at -0.18 (0.08) and implies a long-run impact similar to that in our baseline specification (the long-run impact in this case is $-0.18/[1 - 0.44] \approx -0.32$).

Because we have a relatively short panel, OLS estimation of (12) will lead to inconsistent estimates. To deal with this problem, in column 3 we follow the method of Anderson and Hsiao (1982). This involves first-differencing (12), so that $\Delta x_{it} = \nu \Delta x_{it-1} + \psi \Delta M^I_{it} + \Delta \mu'_t + \Delta u_{it}$, where the fixed country effects are removed by differencing. Although this equation cannot be estimated consistently by OLS either, in the absence of serial correlation in the original residual, u_{it}, there will be no second-order serial correlation in Δu_{it}, so x_{it-2} will be uncorrelated with Δu_{it} and can be used as instrument for Δx_{it-1} to obtain consistent estimates. Similarly, M^I_{it-1} is used as an instrument for ΔM^I_{it}. This procedure leads to results very similar to those of the OLS estimates. The estimate of ψ is -0.27 (0.14).

Although the IV estimator of Anderson and Hsiao (1982) leads to consistent estimates, it is not efficient since, under the assumption of no further serial correlation in u_{it}, not only x_{it-2} but all earlier lags of x_{it} in the sample are also uncorrelated with Δu_{it} and can also be used as additional instruments. Arellano and Bond (1991) develop a generalized method-of-moments (GMM) estimator using all these moment conditions. When all these moment conditions are valid, this GMM estimator is more efficient than Anderson and Hsiao's estimator. The GMM estimation, which we use in column 4, leads to similar but more precisely estimated coefficients. The estimate of ψ in the full sample is now -0.19 (0.06). Tests for second-order autocorrelation in the residuals, reported at the bottom of the column, show that there is no evidence of additional serial correlation. However, the Hansen *J*-test shows that the overidentification restrictions are rejected, presumably because different lags of life expectancy lead to different estimates of the mean reversion coefficient. This rejection is not a major concern for our empirical strategy since the exact magnitude of the mean reversion coefficient, ν, is not of direct interest to us (because the models in [8] and [12] are the first stages in our 2SLS regressions, all we need is for M^I_{it-1} not to have a direct effect on the second-stage outcomes).

Columns 5–7 investigate the effect of lagged and lead mortality. In

column 5, contemporaneous and lagged mortality are included together. Not surprisingly, both of these are significant, since in many countries global health interventions were implemented gradually over time.

The more important challenge for our approach is the inclusion of lead predicted mortality. Because global interventions did not start before 1940, lead mortality should have no effect on life expectancy. Column 6 investigates this by including contemporaneous and lead mortality together. In this case, the estimate of the effect of contemporaneous predicted mortality is −0.33 (0.06), whereas lead mortality is not significant and has the wrong sign. Column 7 includes contemporaneous, lag, and lead predicted mortality together; in this case both contemporaneous and lag mortality are statistically significant, whereas lead mortality remains highly insignificant. These results suggest that, consistent with our hypothesis, it was indeed the global interventions of the 1940s onward that led to the increase in life expectancy in countries previously affected by these diseases rather than some preexisting trends in life expectancy. The issue of preexisting trends will be investigated more directly in the next subsection and in table 7 below.

Finally, columns 8 and 9 show that controlling for the effect of income per capita has little impact on the relationship between predicted mortality and life expectancy.

F. Preexisting Trends and Falsification

Table 6 already showed that life expectancy responds to contemporaneous changes in predicted mortality and does not respond to future changes. This suggests that our first stage is unlikely to be driven by preexisting trends. Nevertheless, the exercise in table 6 uses data only from 1940 onward. An alternative falsification exercise is to look at changes in life expectancy during the preperiod, 1900–1940, and see whether they correlate with future (post-1940) changes in predicted mortality. This is done in figures 5 and 6 and in table 7.

Figure 5 shows the change in log life expectancy 1900–1940 against the change in predicted mortality 1940–80 (see also cols. 6 and 7 in table 1). There is no evidence of a negative relationship similar to those in figures 3 and 4. In fact, there is a slight positive slope (though col. 1 of table 7 shows that this relationship is not significant). Figure 6 further substantiates the lack of preexisting trends. It shows changes in log life expectancy just before the international epidemiological transition, between 1930 and 1940, against the predicted mortality instrument. Once again, there is no evidence of a significant negative relationship. These figures therefore suggest that our measure of predicted

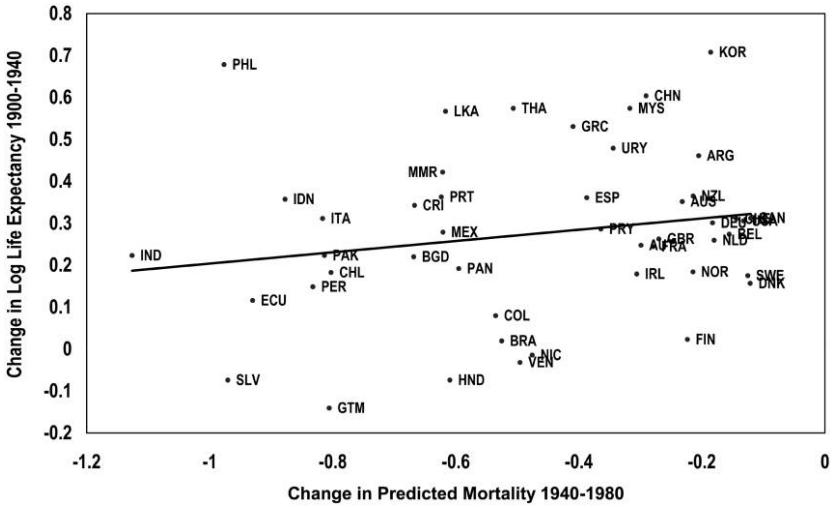

FIG. 5.—Change in log life expectancy 1900–1940 and change in predicted mortality, 1940–80, base sample.

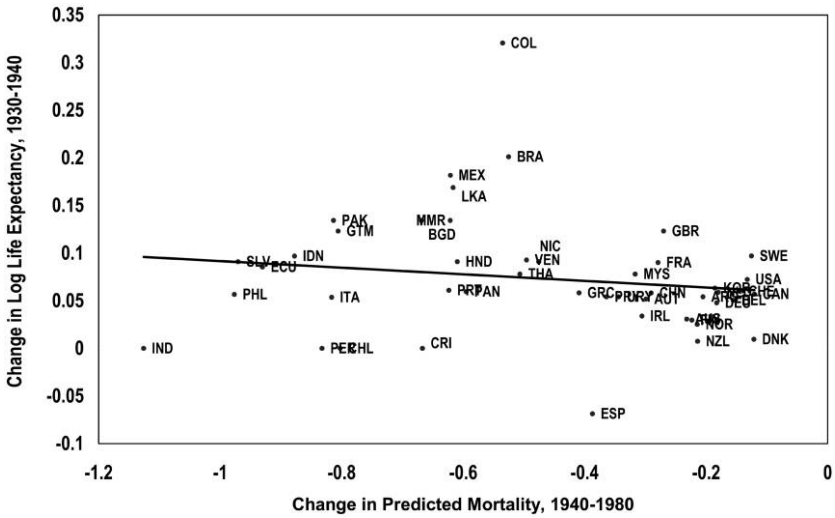

FIG. 6.—Change in log life expectancy, 1930–40, and change in predicted mortality, 1940–80, base sample.

TABLE 7
FALSIFICATION EXERCISE AND REDUCED FORMS: OLS REGRESSIONS

	Base Sample (1)	Low- and Middle-Income Countries (2)	Base Sample (3)	Low- and Middle-Income Countries (4)	Base Sample (5)	Low- and Middle-Income Countries (6)	Base Sample (7)	Low- and Middle-Income Countries (8)
	A. Falsification Exercise							
	Dependent Variable: Change in Life Expectancy from 1900 to 1940		Dependent Variable: Change in Log Population from 1900 to 1940		Dependent Variable: Change in Log GDP from 1900 to 1940		Dependent Variable: Change in Log GDP per Capita from 1900 to 1940	
Change in predicted mortality from 1940 to 1980	.13	.21	−.17	−.13	.009	.05	.02	.04
	(.11)	(.16)	(.15)	(.24)	(.24)	(.36)	(.17)	(.23)
R^2	.04	.06	.03	.01	.0001	.0008	.0005	.0008
Number of countries	47	36	45	34	31	20	31	20
	B. Reduced Forms							
	Dependent Variable: Change in Life Expectancy from 1940 to 1980		Dependent Variable: Change in Log Population from 1940 to 1980		Dependent Variable: Change in Log GDP from 1940 to 1980		Dependent Variable: Change in Log GDP per Capita from 1940 to 1980	
Change in predicted mortality from 1940 to 1980	−.44	−.30	−.74	−.62	−.14	.11	.58	.71
	(.06)	(.08)	(.15)	(.21)	(.22)	(.28)	(.15)	(.20)
R^2	.5	.27	.29	.17	.008	.004	.18	.18
Number of countries	47	36	47	36	47	36	47	36

NOTE.—Robust standard errors are in parentheses. Both panels regress change in the variable indicated from the start to the end date on the change in predicted mortality from 1940 to 1980. Predicted mortality is measured in deaths per 100 population. Panel A uses the subset of the base sample for which data on all outcome variables are available.

mortality explains changes in life expectancy after 1940 but not before 1940.

Panel A of table 7 confirms these results using regression analysis and also shows that there is no preexisting trend when we look at the sample of low- and middle-income countries. Table 7 also looks for potential preexisting trends in our outcome measures (to save space, we focus on population, GDP, and GDP per capita). Columns 3 and 4 (panel A) show that there is no differential preexisting trend in log population between 1900 and 1940 either for the entire sample or for the sample excluding the initially richest countries. Columns 5–8 show similar results for log GDP and log GDP per capita.

These results therefore indicate that there were no preexisting trends related to changes in predicted mortality either in life expectancy or in our key outcome variables.[31] This gives us greater confidence in using predicted mortality as an instrument to investigate the effect of life expectancy on a range of economic outcomes.

VI. Main Results

We now present our main results, which are the 2SLS estimates of the effect of log life expectancy on six outcome variables: log population, log total births, the fraction of the population under the age of 20, log GDP, log GDP per capita, and log GDP per working age population. For each outcome, we report long-difference regressions for 1940 and 1980 (see Acemoglu and Johnson [2006] for similar results using decadal observations as in panel B of table 5 and in table 6). We also report regressions for 1940 and 2000, which may better approximate "longer-run" changes.

A. Population

Figure 7 shows a strong negative reduced-form relationship between the change in log population 1940–80 and the change in predicted mortality over the same period. This pattern can also be seen in reduced-form regressions in panel B of table 7 both for the entire sample and for low- and middle-income countries. It implies that countries with a larger decline in predicted mortality experienced a larger increase in log population, that is, more population growth. Given the negative relationship between predicted mortality and life expectancy in figure 4, this translates into a positive effect of life expectancy on population. This is

[31] For a more qualitative confirmation that there were no preexisting trends before 1940, see Carr-Saunders (1936). In this comprehensive review of population trends, there is no hint of the remarkable increases in life expectancy and population that were to occur shortly.

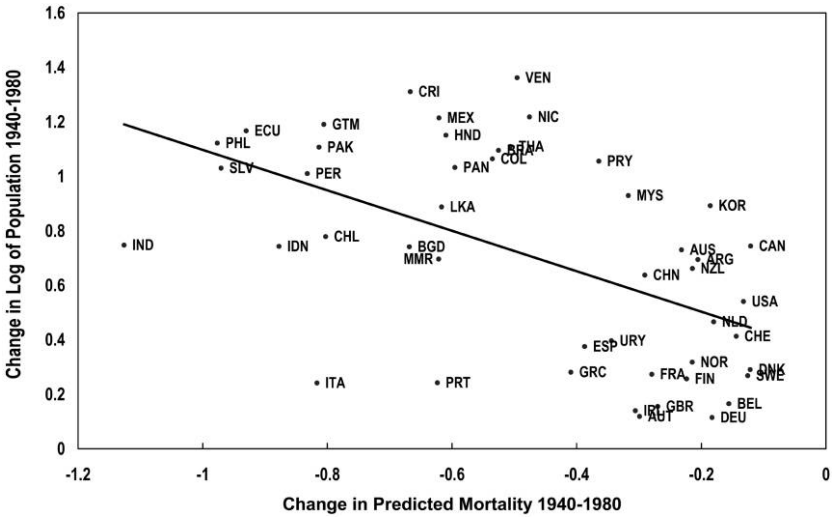

FIG. 7.—Change in log of population and change in predicted mortality, 1940–80, base sample.

confirmed in panel A of table 8, which reports 2SLS regressions of log population on log life expectancy.

In column 1 we look at long differences between 1940 and 1980. The coefficient estimate is 1.67 (standard error 0.50), which is statistically significant at 1 percent. This estimate is very similar to the OLS estimate in column 3 of panel A of table 2. This coefficient increases to 1.96 when we look at the longer horizon, 1940–2000. This suggests that in countries that benefited from the international epidemiological transition, population continued to increase in the 1980s, most likely because the increase in population until the 1980s led to an increase in total number of births (which is confirmed in panel B of table 8).

The coefficient estimates are also larger for low- and middle-income countries. For 1940–80, the coefficient is now 2.04 (1.01), and for 1940–2000, it is 2.18 (0.93). Both of these coefficients are significant at 5 percent.

Columns 5 and 6 estimate specifications that include controls for preexisting trends. In particular, as in equation (11), the second-stage equation in these columns takes the form

$$y_{it} = \pi x_{it} + \zeta_i + \mu_t + \sum_{t=1940}^{1980} \mathbf{c}_i' \boldsymbol{\omega}_t + \varepsilon_{it}, \tag{13}$$

where $\mathbf{c}_i$ includes average institutions (measured as in Sec. V and table 5) or initial (1930) log population. Remarkably, in both cases this has

little effect on the the estimate of π. In column 5, this estimate is 1.63, and in column 6, the estimate of π is 1.68; in both cases the estimate is statistically significant at less than 1 percent.[32]

Finally, column 7 shows that using the global mortality instrument leads to very similar results (a coefficient of 1.70, with a standard error of 0.48).

Overall, we conclude that there is a large, relatively precise and robust effect of life expectancy on population. The elasticity of population in response to life expectancy at birth is estimated consistently to lie between 1.65 and 2.15, which is similar to the OLS estimates.

B. Births and Age Composition

Panel B of table 8 presents 2SLS estimates for the effect of log life expectancy on log total births. The structure is identical to that of panel A, except that because we lack data for 2000, the longer-term specification uses 1940 and 1990. Consistent with the magnitude of the response of population to life expectancy, these results show relatively large effects of life expectancy on total births. The coefficient estimates vary between 2.15 and 2.9 and are typically significant at less than 1 percent (except in col. 3, where the estimate is significant at 5 percent). The estimates are also remarkably robust across different samples and are also robust to controlling for preexisting trends and to the use of the alternative instrument.

There is also some evidence that the effect on total number of births is declining (the estimates for 1940–90 are smaller than those for 1940–80). In Acemoglu and Johnson (2006), we used decadal observations to show that this is a consistent pattern. Therefore, the fertility response to the decline in mortality appears to be slightly delayed. This is consistent with the results in Kelley (1988) and Bleakley and Lange (forthcoming).

Panel C shows that the increase in life expectancy is associated with an increase in the fraction of the population under the age of 20 between 1940 and 1980. However, this effect goes away when we look at 1940–2000 or even in the 1940–80 sample when we look at different specifications. Our interpretation of these results is that there is a slight effect on the age composition immediately following the international epidemiological transition, both because antibiotics, DDT, and public health measures saved the lives of children and because those surviving to childbearing age contributed to the increase in births. However, this

[32] Note that in col. 6, the interaction with initial population is also significant. In addition, results including the interaction with initial log GDP per capita or continent dummies are also very similar and are not reported to economize on space.

TABLE 8
EFFECT OF LIFE EXPECTANCY ON POPULATION, TOTAL BIRTHS, AND POPULATION UNDER AGE 20: 2SLS ESTIMATES

	Baseline Predicted Mortality Instrument						Global Mortality Instrument
	Base Sample (1)	Base Sample (2)	Low- and Middle-Income Countries Only (3)	Low- and Middle-Income Countries Only (4)	Base Sample: Interaction with Institutions (5)	Base Sample: Interaction with Initial (1930) Value of Dependent Variable (6)	Base Sample (7)
	A. Dependent Variable: Log Population						
	Just 1940 and 1980	Just 1940 and 2000	Just 1940 and 1980	Just 1940 and 2000	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980
Log life expectancy	1.67	1.96	2.04	2.18	1.63	1.68	1.70
	(.50)	(.53)	(1.01)	(.93)	(.73)	(.44)	(.48)
Postyear dummy × institutions or initial log population					−.006	−.055	
					(.05)	(.03)	
Number of countries	47	47	36	36	47	47	47
	B. Dependent Variable: Log Total Births						
	Just 1940 and 1980	Just 1940 and 1990	Just 1940 and 1980	Just 1940 and 1990	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980
Log life expectancy	2.53	2.15	2.92	2.67	2.40	2.53	2.52
	(.70)	(.64)	(1.36)	(1.20)	(1.09)	(.70)	(.72)
Postyear dummy × institutions or initial log of total births					−.018	−.056	
					(.09)	(.05)	
Number of countries	45	45	34	34	45	44	45

	C. Dependent Variable: Fraction of Population under Age 20						
	Just 1940 and 1980	Just 1940 and 2000	Just 1940 and 1980	Just 1940 and 2000	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980
Log life expectancy	.12	.05	.18	.16	.15	.26	.12
	(.06)	(.08)	(.14)	(.17)	(.08)	(.31)	(.057)
Postyear dummy × institutions or initial fraction of young population					.005	−.30	
					(.01)	(.52)	
Number of countries	40	40	29	29	40	40	40

NOTE.—2SLS regressions with a full set of year and country fixed effects. Robust standard errors are in parentheses. All regressions in all panels are long-difference specifications, with two observations per country: one for the initial date and one for the final date. Dependent variables in panel A are the log of population; in panel B, the log of total births; and in panel C, the fraction of the total population that is 20 years old or younger. In all panels, the independent variable is the log of life expectancy at birth, which is instrumented by the baseline predicted mortality in cols. 1–6 and by the predicted global mortality in col. 7. First stages are reported in table 5. In col. 5, regressions include interactions of year dummies with institutions, measured by the average of constraints on the executive in 1950, 1960, and 1970 from Polity IV. In col. 6, regressions include interactions of year dummies with the initial (1930) log of population in panel A, the initial (1930) log of total births in panel B, and the initial (1940) percentage of population aged 20 or younger in panel C.

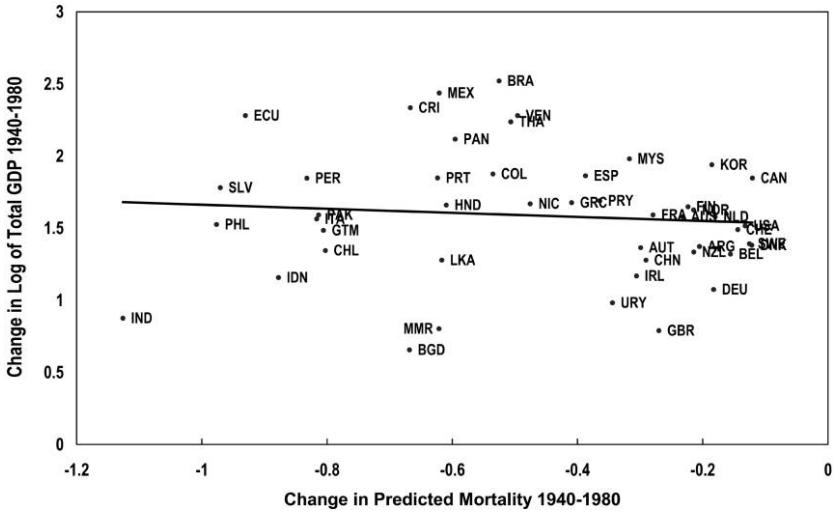

FIG. 8.—Change in log of total GDP and change in predicted mortality, 1940–80, base sample.

effect largely abates by 2000. We have also verified that the results are essentially identical with the dependency ratio (the ratio of nonactive to total population) and that the effect of life expectancy at birth on working age population is very similar to its effect on total population (results available on request). This is also consistent with the patterns reported in panel C.

C. GDP, GDP per Capita, and GDP per Working Age Population

Figure 8 shows the reduced-form relationship between change in log (total) GDP and change in predicted mortality during 1940–80. As also shown in panel B of table 7 (both for the base sample and for low- and middle-income countries), there is a slight (but not statistically significant) downward slope, which indicates that countries with larger declines in predicted mortality experienced somewhat higher GDP growth between 1940 and 1980.

Panel A of table 9 presents the corresponding 2SLS estimates. In column 1, the estimate of the key parameter is 0.32 (0.84), and the estimate using 1940 and 2000 in column 2 is 0.42 (0.52). Both of these estimates suggest that there is a slight positive effect on GDP, though it is imprecisely estimated and thus is not statistically significant. In both cases, the standard errors are large enough that economically significant positive effects on total GDP cannot be ruled out. For example, the two standard error bands always include a response of GDP to life expectancy

with an elasticity that could be as high as 1.5. It is also interesting that the estimate for 1940–2000 is somewhat larger than that for 1940–80, which may correspond to a delayed response of GDP to the increase in population and health conditions. This is consistent with the neoclassical growth model outlined in Section II.[33]

The remaining columns show that the effect of life expectancy on GDP is somewhat smaller or even negative when we focus on low- and middle-income countries or when we include baseline interactions. We interpret these estimates as suggesting that the increase in life expectancy and the associated increase in population had a relatively small effect on total GDP, perhaps with a somewhat larger effect over 60 years than in the first few decades after the decline in mortality. Although the relatively large standard errors make it impossible for us to pin down the exact magnitude or the timing of the impact of life expectancy on GDP, we view the lack of a somewhat larger positive effect on total GDP as a potential puzzle.

The response of total GDP already reveals that the effect of the increase in life expectancy on GDP per capita was negative. Panel B of table 9 confirms this pattern by presenting the 2SLS estimates of the effect of log life expectancy on GDP per capita. There is a significant negative effect of life expectancy on GDP per capita in columns 1 and 2. For example, in column 1, the estimate of π in equation (6) is -1.32 (0.56). The estimates are somewhat more negative when we focus on low- and middle-income countries in columns 3 and 4.

Columns 5, 6, and 7 show that the estimates are very similar when we include the interaction between the postyear dummy and average institutions or the initial value of GDP per capita, or when we use the global mortality instrument.

One concern with these results is that, to the extent that the increase in population occurs largely at young ages, GDP per capita may be low precisely because the denominator has increased, whereas the working age population has not. The results in panel C of table 8, which show only limited changes in age composition, already suggest that this is unlikely to be the case. Panel C of table 9 investigates this issue directly by estimating models with log of GDP per working age population on the left-hand side. The results are very similar to those in panel B and indicate that the effect of life expectancy on GDP per working age population is also negative.

Overall, the 2SLS estimates show no evidence that the large increase

[33] In Acemoglu and Johnson (2006), we reported additional findings consistent with a somewhat delayed response of GDP to life expectancy. The recent paper by Ashraf et al. (2007) shows that even when health has positive effects on long-run income per capita, population dynamics will lead to considerable delays before any increase in income per capita is observed.

TABLE 9
EFFECT OF LIFE EXPECTANCY ON GDP, PER CAPITA GDP, AND GDP PER WORKING AGE POPULATION: 2SLS ESTIMATES

	Baseline Predicted Mortality Instrument						Global Mortality Instrument
	Base Sample: Just 1940 and 1980 (1)	Base Sample: Just 1940 and 2000 (2)	Low- and Middle-Income Countries Only: Just 1940 and 1980 (3)	Low- and Middle-Income Countries Only: Just 1940 and 2000 (4)	Base Sample: Interaction with Institutions: Just 1940 and 1980 (5)	Base Sample: Interaction with Initial (1930) Value of Dependent Variable: Just 1940 and 1980 (6)	Base Sample Just 1940 and 1980 (7)
	A. Dependent Variable: Log GDP						
Log life expectancy	.32	.42	−.39	−.58	−.11	−.069	.46
	(.84)	(.52)	(1.44)	(1.09)	(.99)	(.73)	(.73)
Postyear dummy × institutions or initial log GDP					−.063	−.109	
					(.055)	(.059)	
Number of countries	47	47	36	36	47	47	47
	B. Dependent Variable: Log per Capita GDP						
Log life expectancy	−1.32	−1.51	−2.35	−2.70	−1.64	−1.59	−1.21
	(.56)	(.57)	(1.13)	(1.40)	(.77)	(1.22)	(.52)
Postyear dummy × institutions or initial log per capita GDP					−.049	−.073	
					(.060)	(.278)	
Number of countries	47	47	36	36	47	47	47

	C. Dependent Variable: Log GDP per Working Age Population						
Log life expectancy	−1.35	−1.62	−2.43	−2.63	−1.82	−1.87	−1.23
	(.63)	(.54)	(1.30)	(1.31)	(.88)	(1.39)	(.57)
Postyear dummy × institutions or initial log GDP per working age population					−.068	−.158	
					(.065)	(.369)	
Number of countries	46	46	35	35	46	46	46

NOTE.—2SLS regressions that include a full set of year and country fixed effects. Robust standard errors are in parentheses. All regressions in all panels are long-difference specifications, with two observations per country: one for the beginning date and one for the end date. Dependent variables in panel A are the log of GDP; in panel B, the log of GDP per capita; and in panel C, the log of GDP per working age population. In all panels, the independent variable is the log of life expectancy at birth, which is instrumented by the baseline predicted mortality in cols. 1–6 and by the predicted global mortality in col. 7. First stages are reported in table 5. In col. 5, regressions include interactions of year dummies with institutions, measured by the average of constraints on the executive in 1950, 1960, and 1970 from Polity IV. In col. 6, regressions include interactions of year dummies with the initial (1930) log of GDP in panel A, the initial (1930) log of GDP per capita in panel B, and the initial (1930) log of GDP per working age population in panel C.

in life expectancy in many parts of the world starting in the 1940s led to a significant increase in GDP per capita. Instead, the increase in life expectancy was associated with a significant increase in population and a considerably smaller increase in total GDP.[34]

We can also evaluate these estimates in terms of the neoclassical growth model presented in Section II. First, suppose that the results for 1940–80 correspond to the impact of life expectancy on income per capita with the capital stock held constant. From equation (4) in Section II, the coefficient of interest in this case is $\pi = \alpha(\gamma + \eta) - (1 - \alpha)\lambda$. Recall that λ is the response of population to changes in life expectancy, so according to the estimates for the base sample in panel A, table 8, we have $\lambda \approx 1.7$. The coefficient α corresponds to the share of labor. Since the countries that benefited most from increases in life expectancy include many low-income countries in which land is an important factor of production, we take the share of land as one-third, that is, $1 - \alpha - \beta \approx 1/3$ (see n. 8), and thus set $\alpha \approx 1/3$ and $\beta \approx 1/3$. This would imply that our estimate of $\pi = \alpha(\gamma + \eta) - (1 - \alpha)\lambda \approx -1.3$ is consistent with $\gamma + \eta$ close to zero or even slightly negative. If, on the other hand, we were to take λ to be around two as suggested by the high-end estimates from low- and middle-income countries in table 8, $\gamma + \eta$ would be small but positive. Similar and somewhat less positive results follow if we take the estimates for 1940–2000 to correspond to the long-run effects in equation (5). Recall that in this case $\pi = [\alpha(\gamma + \eta) - (1 - \alpha - \beta)\lambda]/(1 - \beta)$. From column 2 in panel A of table 8, $\lambda \approx 2$, and from panel B of table 9, $\pi \approx -1.5$. Again if we take $\alpha \approx 1/3$ and $\beta \approx 1/3$, the estimate for π can be rationalized by having negative values for $\gamma + \eta$. These computations suggest that the results reported here could be reconciled with the simple neoclassical growth model presented in Section II, but only if the share of land in GDP is about one-third and the positive effects of health on TFP and education are limited. Since a share of land in GDP of about one-third is quite large,[35] there may be other factors, beyond those captured by the neoclassical growth model, that are important for understanding the effects of life expectancy on income per capita.

[34] The comparison of these results to the OLS estimates in table 3 (together with the pattern discussed in n. 24) also suggests that the zero OLS relationship between life expectancy and GDP per capita is likely to be a combination of a short-run negative effect of life expectancy on GDP per capita and a positive effect of income on life expectancy. See also Pritchett and Summers (1996) for estimates of income per capita on life expectancy.

[35] For example, Hansen and Prescott (2002) suggest a value of 0.3 for $1 - \alpha - \beta$, 0.1 for β, and 0.6 for α in preindustrial societies.

VII. Further Results

A. *Robustness*

We verified that our results are not affected by the fact that we are combining data on causes of death (individual diseases) from two sources. In particular, using only the 32 countries for which we have disease data from one source, Federal Security Agency (1947), has little effect on our first-stage, reduced-form, or 2SLS results. We also checked the robustness of our results to dropping all data for which we had to use information on life expectancy from neighboring countries. The first-stage, reduced-form, or 2SLS estimates in this smaller sample of 39 countries are again very similar to the baseline results.

In addition, in Acemoglu and Johnson (2006) we showed that the results reported in tables 8 and 9 are robust to a variety of additional specifications. First, in panel specifications with decadal observations, we can include data from sub-Saharan African countries.[36] The inclusion of African data leads to estimates very similar to the baseline results. We also showed that the results are robust to excluding countries that were demographically most affected by World War II.[37] We also estimated regressions dropping countries that were involved in developing the new "miracle" drugs and chemicals of the 1940s and 1950s: the United Kingdom, the United States, Germany, and Switzerland. The exclusion of these countries again has no effect on the baseline results. Finally, we estimated specifications that control for mean reversion in the second stage, again with little effect on the main results.

B. *Further Results*

A potential concern, already discussed above, is whether the international epidemiological transition mainly affected life expectancy at birth, with little effect on adult mortality. This is not the case. In particular, tuberculosis and pneumonia, two of the main killers in our sample, affected the entire age distribution. As a result, our predicted mortality

[36] There are no life expectancy data for sub-Saharan Africa before 1950, and post-1950 data may be less reliable for this region than for the rest of the world. Nevertheless, in general terms, we know that health in Africa improved, at least for a while after World War II. For example, Cutler et al. (2006, 17) write that "life expectancy [in Africa] rose by more than 13 years from the early 1950s to the late 1980s, before declining in the face of HIV/AIDS." Estimates in Gwatkin (1980, e.g., fig. 2) also suggest that increases in life expectancy were at least as dramatic in Africa as in other developing countries, but only until average life expectancy for these societies reached 40; at that point the rate of increase slowed sharply. This could point to a failure to sustain health improvements or some other factor and needs further investigation.

[37] The countries most affected by World War II in our base sample are Germany, Italy, Finland, Austria, and China (see Urlanis 2003). Excluding these countries has little effect on the first- or second-stage estimates (see Acemoglu and Johnson 2006).

TABLE 10
EFFECT OF LIFE EXPECTANCY AT AGE 20 ON POPULATION, TOTAL BIRTHS, GDP, AND PER CAPITA GDP: 2SLS ESTIMATES AND FIRST STAGES

	Baseline Predicted Mortality Instrument				Global Mortality Instrument
	Base Sample: Just 1940 and 1980 (1)	Low- and Middle-Income Countries Only: Just 1940 and 1980 (2)	Base Sample: Just 1940 and 2000* (3)	Low- and Middle-Income Countries Only: Just 1940 and 2000* (4)	Base Sample Just 1940 and 1980 (5)
	A. Dependent Variable: Log Population				
Log life expectancy at 20	4.54	5.04	6.54	7.16	4.75
	(2.11)	(3.64)	(2.45)	(4.22)	(2.02)
Number of countries	45	34	46	35	45
	B. Dependent Variable: Log Total Births				
Log life expectancy at 20	6.60	6.98	7.33	9.21	6.73
	(2.64)	(4.40)	(3.78)	(7.10)	(2.67)
Number of countries	43	32	40	29	43

	C. Dependent Variable: Log GDP				
Log life expectancy at 20	1.17	−.39	1.53	−1.71	1.64
	(2.55)	(3.45)	(1.84)	(3.51)	(2.30)
Number of countries	45	34	46	35	45
	D. Dependent Variable: Log per Capita GDP				
Log life expectancy at 20	−3.27	−5.24	−4.91	−8.68	−3.05
	(1.45)	(2.95)	(2.36)	(5.75)	(1.47)
Number of countries	45	34	46	35	45
	E. First Stages of IV Estimations; Dependent Variable: Log Life Expectancy at 20				
Predicted mortality	−.17	−.13	−.17	−.14	−.17
	(.039)	(.049)	(.032)	(.041)	(.06)
R^2	.93	.92	.96	.96	.92
Number of countries	45	34	46	35	45

NOTE.—In panels A–D, 2SLS regressions with a full set of year and country fixed effects; corresponding first stages are in panel E. Robust standard errors are in parentheses. All regressions are long-difference specifications, with two observations per country: one for the initial date and one for the final date. Dependent variables in panel A are the log of population; in panel B, the log of total births; in panel C, the log of GDP; in panel D, the log of GDP per capita; and in panel E, the log of life expectancy at age 20. The log of life expectancy at 20 is also the independent variable for panels A–D. It is instrumented by baseline predicted mortality in cols. 1–4 and by global predicted mortality in col. 5.

* 1990 for panel B.

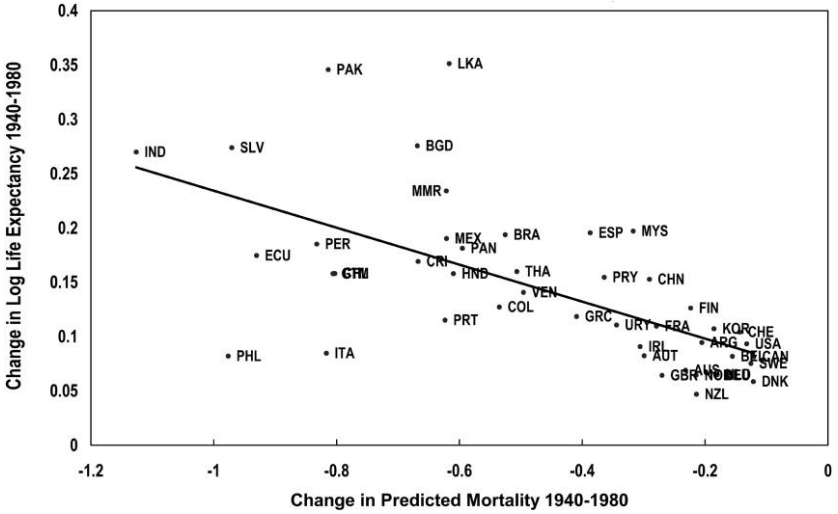

FIG. 9.—Change in log life expectancy at age 20 and change in predicted mortality, 1940–80, base sample.

instrument has a strong effect on life expectancy at various ages. In table 10 we focus on life expectancy at 20 (defined as total life expectancy conditional on having reached the age of 20) and present results using this variable as the proxy for health rather than life expectancy at birth. Panels A–D report results for the outcome variables of tables 8 and 9. Panel E shows the corresponding first stages and documents the impact of predicted mortality on life expectancy at 20.

Panel E shows a strong relationship between life expectancy at 20 and predicted mortality. For example, in the base sample for 1940–80, which now includes 45 countries, the coefficient estimate of predicted mortality in a regression of log life expectancy at 20 is −0.17 (standard error 0.039). This first-stage relationship is also shown in figure 9. The first stage is similar in the other columns, which focus on low- and middle-income countries, on longer-term changes (1940–2000), and on results using the global mortality instrument. As noted in note 29, the effects of predicted mortality on life expectancy at 5, 10, 15, and 30 are also similar, though its impact on infant mortality is somewhat weaker.

The 2SLS results in panels A–D are also similar to those in tables 8 and 9. There is a positive effect on population and births, a positive and insignificant effect on total GDP, and a negative effect on GDP per capita. Results for GDP per working age population are once again similar to those for GDP per capita.

VIII. Concluding Remarks

A recent consensus in academic and policy circles holds that differences in disease environments and health conditions lie at the root of large income differences across countries today and argues that improving health not only will improve lives but will by itself spur rapid economic growth.

This article investigated these claims by estimating the effect of life expectancy on economic growth. The innovation in our approach is to exploit the international epidemiological transition, which led to potentially exogenous differential changes in mortality from a number of major diseases across the world. As a result of new chemicals, drugs, and international health campaigns, mortality from tuberculosis, pneumonia, malaria, and various other diseases declined sharply in many parts of the world, whereas other countries that were largely unaffected by these diseases did not experience similar improvements in health and mortality. Exploiting these differential changes in predicted mortality as an instrument for life expectancy, we estimated the effect of life expectancy on a range of economic variables, most important, population and GDP.

Our results indicate that the increase in life expectancy led to a significant increase in population; birth rates did not decline sufficiently to compensate for the increase in life expectancy. We find a small positive effect of life expectancy on total GDP over the first 40 years, and this effect grows somewhat over the next 20 years, but not enough to compensate for the increase in population. Overall, the increases in life expectancy (and the associated increases in population) appear to have reduced income per capita. There is no evidence that the increase in life expectancy led to faster growth of income per capita or output per worker. This evidence casts doubt on the view that health has a first-order impact on economic growth.

Considerable caution is necessary in interpreting our results for at least two reasons. The most important limitation is that because our approach exploits the international epidemiological transition around the 1940s, the results may not be directly applicable to today's world; the international epidemiological transition was a unique event, and perhaps similar changes in life expectancy today would not lead to an increase in population and the impact on GDP per capita may be more positive. Second, the diseases that take many lives in the poorer parts of the world today are not the same ones as those 60 years ago; most notably HIV/AIDS is a major killer today but was not so in 1940. Many of the diseases we focus on had serious impacts on children (with the notable exception of tuberculosis), whereas HIV/AIDS affects individuals at the peak of their labor productivity and could have a larger

negative impact on growth. Further study of the effects of the HIV/AIDS epidemic on economic outcomes as well as more detailed analysis of different measures of health on human capital investments and economic outcomes are major areas for future research.

Appendix A

Data Sources and Construction

Key data are shown in table A1. Population, GDP, and GDP per capita data are taken from Maddison (2003), specifically the downloadable data available to purchasers of his 2003 book. Working age population is defined as population between the ages of 15 and 60 and is obtained from the online UN demographic database from 1950 (http://esa.un.org/unpp). Population structure for 1940 is taken from the 1948 UN *Demographic Yearbook* (United Nations 1949, 108–58, table 4). We use data for 1940 or the closest available year or range of years.

Life expectancies in 1940 and earlier are taken from various UN *Demographic Yearbook*s. Key yearbooks are the original 1948 edition (United Nations 1949) and subsequent issues for 1949–50 (United Nations 1950), for 1951 (United Nations 1951), and particularly the retrospective section of the 1967 *Demographic Yearbook* (United Nations 1967). We use the most recently revised UN data available to calculate the unweighted averages of male and female life expectancy for 1940 (we also check these data against United Nations [2000], but the coverage of this generally begins no earlier than 1948). When there are no data for 1940, but such data exist for neighboring years, for example, 1938 and 1942, we use linear interpolation to obtain an estimate for 1940. In a few cases, we use information from neighboring countries when they have similar crude death rates (from the UN *Demographic Yearbook*s). Appendix C provides further details and gives the specifics for each country.

Life expectancy from 1950 onward was downloaded from the online UN demographic database; these data are given in five-year intervals, so we use 1950–55 for 1950, 1960–65 for 1960, and so forth. Life expectancy in 1900, used in the falsification tests, is taken from Maddison (2001, 30, table 1-5a). These estimates for life expectancy in 1900 for Europe, Latin America, and Asia are consistent with the numbers in Arriaga and Davis (1969), Riley (2001), and Bengtsson et al. (2004).

To classify the cause of death, we use the Abridged List of the 1938 revision of the International Classification of Disease. This list is comprehensive and has 44 categories. We omit any diseases that are not infectious or could be degenerative, for example, "diseases of the heart" (Abridged List no. 24), and residual categories, such as "other infectious or parasitic diseases" (no. 14). Syphilis (no. 9) and puerperal fever/infection (no. 35), which results from an infection after childbirth, are omitted because their prevalence depends on sexual and fertility behavior, which fall outside our focus here. Finally, we further omit diseases that were never major causes of death, even though they may have had serious effects on health (e.g., acute poliomyelitis). In all, there are 15 infectious diseases for which we can obtain comparable cross-country data on deaths per 100,000 in 1940 (or 1939 or a close year). Of these 15, three are reviewed in more detail in the text and 12 are covered in online Appendix B. We have checked that the data we use in or around 1940 are not significantly affected by the impact of World War II; this is generally possible since in most cases some combination of UN sources yields numbers for at least two early years. For European countries

affected by the war, we prefer data from 1937 or 1938, where available. Also, in our robustness checks, we drop all data from countries in which Urlanis (2003) deemed that war had a major demographic impact.

The classification of death rates by cause changed in 1948, and some of our data for 1950 and after are available only according to the Abbreviated List, 1948 Revision of the International Classification of Disease. For example, the 1954 UN *Demographic Yearbook* reports cause of death in and around 1950 for some countries using the 1938 classification and for others using the 1948 classification. The terminology of the Abridged List for the 1938 classification and the Abbreviated List for the 1948 classification is as used in the *Demographic Yearbook*. Most of our 15 diseases can be tracked through this reclassification, but dysentery/diarrhea-related diseases cannot. Consequently, we have information for this category only for 1940, which is what we need to construct the predicted mortality instrument, but is not sufficient for the zeroth-stage regressions in table 4 or for the global mortality instrument. In addition, there are not enough data on yellow fever to track it over time, so this disease is also not included in table 4 or in the global mortality instrument.

For our data on cause of death in 1940, we start with the Summary of International Vital Statistics, 1937–44, published by the Federal Security Agency (1947) of the U.S. government immediately after World War II. This source provides comparable comprehensive data on cause of death around 1940, as well as longer time series on the more important diseases (i.e., death rates by country), primarily from League of Nations sources; however, it did not use all the available data (2). For this reason, we fill gaps for 1940 using the original sources, which are national health statistics collected, cleaned, and republished between the wars by the League of Nations Health Organization (1–3); we also use information from the league and its direct postwar successors for earlier and later data as discussed in online Appendix C. A key issue is the area covered by the registration of deaths in various countries. Apart from the very richest countries in 1940, there was seldom universal registration of death, with a death certificate signed by a doctor. Consequently, some of the data pertain to major cities, whereas others pertain to all towns or to the entire population. Unfortunately, our sources do not always document clearly the precise coverage of the underlying data (for lower-income countries, the data almost certainly overweigh towns relative to rural areas, and diseases related to urban overcrowding are likely to be overrepresented). Nevertheless, our results are robust to using only the more reliable data.

The League of Nations established comparable international health statistics for a large number of countries but never to our knowledge published a comprehensive retrospective of the data. Their first relevant publication was issue 7 of the *Annual Epidemiological Report*, which appeared in October 1923. But only from 1929 (covering the year 1927) did this publication include death rates from specific causes (League of Nations 1929). Early issues of this publication are also referred to as *Statistics of Notifiable Diseases*. The first six issues focused on eastern Europe, particularly typhus and malaria epidemics in Russia. For a comprehensive list of publications by the League of Nations on health, see Aufricht (1951, esp. 176–77). For an explanation of the structure and purpose of the League of Nations Health Organization, see League of Nations (1931). For more on the early development of internationally comparable health statistics, see Stocks (1950).

We use the death rates by disease for 1930 from League of Nations (1933). For 1940 we supplement the information discussed above with WHO (1951),

which provided data for 1939–46, based on the League of Nations' work. For cholera, yellow fever, plague, and typhus, we have comparable data for 1940 but not for 1930. For malaria in 1930, we use data from the League of Nations' Malaria Commission (League of Nations 1932). We also checked our data against information on location of malaria in the 1940s from American Geographical Society (1951*b*). Data on deaths by disease for 1950 and 1960 are taken from the UN *Demographic Yearbook* for 1954, 1962, and 1966. Data for 1970 are taken from the 1974 UN *Demographic Yearbook* and data for 1980 are taken from the 1985 UN *Demographic Yearbook*.

We further confirmed that our data do not miss major epidemics by reviewing every available interwar issue of the League of Nations' *Weekly Epidemiological Record* (WER). For example, for the distribution of cholera in 1938, see WER, March 3, 1938. For the distribution of smallpox in 1930, see WER, August 21, 1930; for 1938, see WER, March 3, 1938; for the early 1940s, see WER, January 3, 1946. For the prewar distribution of diphtheria, with a focus on Europe, see WER, December 21, 1939. For the distribution of plague in 1938, see WER, March 3, 1938. For more detail on the pre-1940 distribution of typhus, see WER, September 14, 1939. For the endemic yellow fever zone in 1951, see the supplement to the WER, September 25, 1952. We also confirm that our numbers are consistent with contemporary qualitative assessments, in particular in the League of Nations and WHO's annual reports. Further details on these checks and data sources are provided in our working paper (Acemoglu and Johnson 2006).

Predicted mortality in 1940 is calculated by adding deaths per 100,000 from the 15 component diseases (for ease of exposition, we then convert this number to per 100 of population). Preston (1980) points out that data on precise cause of death should be handled with care; for example, it is notoriously difficult to determine how many deaths are due directly and indirectly to malaria. While this is an important warning in general, our analysis is about changes in total predicted mortality from infectious disease, and because most of the global interventions were clustered in the late 1940s and early 1950s, this issue is less of a concern here.

TABLE A1
KEY DATA FOR BASE SAMPLE

Country	Initial Income	Year	Predicted Mortality	Life Expectancy	Population	GDP	GDP per Capita
Argentina	Middle	1940	.205	55.50	14,169	58,963	4,161
		1980	.000	69.59	28,370	232,802	8,206
Australia	Rich	1940	.232	66.80	7,042	43,422	6,166
		1980	.000	74.44	14,616	210,642	14,412
Austria	Middle	1940	.299	60.20	6,705	26,547	3,959
		1980	.000	72.65	7,549	103,874	13,759
Bangladesh	Poor	1940	.668	29.90	41,966	25,044	597
		1980	.000	48.47	88,077	48,239	548
Belgium	Rich	1940	.156	61.80	8,346	38,072	4,562
		1980	.000	73.25	9,847	142,458	14,467
Brazil	Poor	1940	.525	36.70	41,114	51,381	1,250
		1980	.000	62.67	122,958	639,093	5,198
Canada	Rich	1940	.121	64.20	11,688	62,744	5,368
		1980	.000	74.72	24,593	397,814	16,176
Chile	Middle	1940	.803	42.00	5,093	16,596	3,259
		1980	.000	69.30	11,094	63,654	5,738
China	Poor	1940	.291	43.90	518,770	291,603	562
		1980	.000	65.31	981,235	1,046,781	1,067
Colombia	Middle	1940	.535	37.90	9,174	17,386	1,895
		1980	.000	65.91	26,583	113,375	4,265
Costa Rica	Middle	1940	.667	49.30	620	1,093	1,763
		1980	.000	72.70	2,299	11,290	4,911
Denmark	Rich	1940	.121	65.50	3,832	19,606	5,116
		1980	.000	74.29	5,123	78,010	15,227
Ecuador	Poor	1940	.930	39.30	2,466	3,344	1,546
		1980	.000	63.26	7,920	32,706	4,129
El Salvador	Poor	1940	.970	32.50	1,630	1,811	1,111
		1980	.000	57.10	4,566	10,748	2,354
Finland	Middle	1940	.223	57.30	3,698	11,909	3,220
		1980	.000	73.19	4,780	61,890	12,949
France	Middle	1940	.279	60.00	41,000	165,729	4,042
		1980	.000	74.25	53,870	813,763	15,106
Germany	Rich	1940	.183	63.50	69,835	377,284	5,403
		1980	.000	72.63	78,298	1,105,099	14,114
Greece	Middle	1940	.409	54.40	7,280	16,183	2,223
		1980	.000	74.36	9,643	86,505	8,971
Guatemala	Middle	1940	.806	30.40	2,200	6,033	2,742
		1980	.000	57.35	7,235	26,632	3,681
Honduras	Poor	1940	.610	32.50	1,150	1,334	1,160
		1980	.000	60.01	3,635	7,014	1,930
India	Poor	1940	1.126	30.00	321,565	265,455	686
		1980	.000	54.39	679,000	637,202	938
Indonesia	Poor	1940	.878	34.30	70,175	86,682	1,235
		1980	.000	54.81	147,490	275,805	1,870
Ireland	Middle	1940	.306	59.80	2,958	9,028	3,052
		1980	.000	72.67	3,401	29,047	8,541
Italy	Middle	1940	.816	58.70	44,341	155,424	3,505
		1980	.000	73.92	56,451	742,299	13,149
Korea, Republic	Poor	1940	.186	48.70	15,627	22,536	1,442
		1980	.000	66.84	38,124	156,846	4,114
Malaysia	Poor	1940	.317	42.60	5,434	6,945	1,278
		1980	.000	66.87	13,764	50,333	3,657
Mexico	Middle	1940	.621	43.60	20,393	37,767	1,852

TABLE A1
(*Continued*)

Country	Initial Income	Year	Predicted Mortality	Life Expectancy	Population	GDP	GDP per Capita
		1980	.000	66.76	68,686	431,983	6,289
Myanmar	Poor	1940	.621	36.60	16,594	12,274	740
		1980	.000	52.10	33,283	27,381	823
Netherlands	Rich	1940	.180	67.40	8,879	42,898	4,831
		1980	.000	75.72	14,144	207,979	14,705
New Zealand	Rich	1940	.214	67.70	1,636	10,308	6,300
		1980	.000	73.20	3,170	39,141	12,347
Nicaragua	Poor	1940	.476	34.50	830	1,139	1,372
		1980	.000	58.72	2,804	6,043	2,155
Norway	Middle	1940	.214	67.30	2,973	12,152	4,088
		1980	.000	75.74	4,086	61,811	15,129
Pakistan	Poor	1940	.813	30.00	28,169	20,137	715
		1980	.000	55.12	85,219	98,907	1,161
Panama	Middle	1940	.595	42.40	697	1,199	1,721
		1980	.000	70.12	1,956	9,961	5,091
Paraguay	Middle	1940	.364	46.60	1,111	1,947	1,752
		1980	.000	66.83	3,193	10,549	3,304
Peru	Middle	1940	.832	40.60	6,298	11,483	1,823
		1980	.000	60.38	17,295	72,723	4,205
Philippines	Poor	1940	.976	47.30	16,585	26,326	1,587
		1980	.000	61.09	50,940	121,012	2,376
Portugal	Middle	1940	.623	50.30	7,675	12,396	1,615
		1980	.000	71.39	9,778	78,655	8,044
Spain	Middle	1940	.387	50.20	25,757	53,585	2,080
		1980	.000	75.53	37,488	344,987	9,203
Sri Lanka	Poor	1940	.617	42.30	6,134	7,673	1,251
		1980	.000	68.20	14,900	27,550	1,849
Sweden	Rich	1940	.125	66.70	6,356	30,873	4,857
		1980	.000	75.86	8,310	124,130	14,937
Switzerland	Rich	1940	.144	64.10	4,226	27,032	6,397
		1980	.000	75.85	6,385	119,909	18,779
Thailand	Poor	1940	.506	42.60	15,513	12,820	826
		1980	.000	63.60	47,026	120,116	2,554
United Kingdom	Rich	1940	.270	65.00	48,226	330,638	6,856
		1980	.000	73.78	56,314	728,224	12,931
United States	Rich	1940	.132	63.80	132,637	929,737	7,010
		1980	.000	73.66	227,726	4,230,558	18,577
Uruguay	Middle	1940	.344	56.50	1,965	7,193	3,661
		1980	.000	70.43	2,920	19,205	6,577
Venezuela	Middle	1940	.496	33.90	3,784	15,307	4,045
		1980	.000	68.34	14,768	149,735	10,139

NOTE.—Life expectancy is at birth, population is in thousands, and GDP is in millions (1990 international Geary-Khamis dollars). Predicted mortality is as defined in the text; units are per 100 per year.

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