



Health and Economic Growth

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Abstract

This chapter examines the relationship between health and economic growth. Across countries, income per capita is highly correlated with health, as measured by life expectancy or a number of other indicators. Within countries, there is also a correlation between people's health and income. Finally, over time, the historical evolution of cross-country health differences has largely paralleled the evolution of income differences, with the exception that in the last half century the convergence of health has been much faster than the convergence of income. How are health and income related? Theoretically, there is good reason to believe that causality runs in both directions. Healthier individuals are more productive, learn more in school, and, because they live longer, face enhanced incentives to accumulate human capital. Similarly, higher income for individuals or countries improves health in a variety of ways, ranging from better nutrition to construction of public health infrastructure. Empirically, there is evidence for both of these causal channels being operative, but the magnitude of the effects is limited, at least as they apply to cross-sectional differences among countries or individuals. Apparently, other factors that simultaneously raise income and improve health outcomes, such as institutional quality (for countries) and human capital (for individuals), are responsible for a good deal of the observed health-income correlation. The final section of the chapter discusses measures of aggregate welfare that combine consumption and life expectancy.

Keywords

Economic Growth, Health, Mortality, Productivity, Disease

JEL Classification Codes

I10, J17, N30, O11, O40



3.1. INTRODUCTION

The largest part of this literature, and the part on which I focus most extensively, examines the effect of health on economic growth. Does making a population healthier make it richer? Over what time horizon and through which channels? What is the magnitude of health's impact on income, and how much of income variation among countries is explained by variation in health?

The second topic on which I focus is causality running in the other direction, from income to health. Humanity has experienced great improvements in health over the last two centuries, roughly contemporaneously with the period of steady income growth

that followed the Industrial Revolution. But the causes of this health improvement are not transparent, particularly the extent to which better health is attributable to income growth per se, to changes in health technology, and to changes in the institutions that deliver health services. Most notably, over the past century, the cross-sectional relationship between income and health has changed significantly, indicating that the “technology” of health, and perhaps the price of health, have changed. I discuss the nature of this health technology.

The final large topic I address is how to comprehend health improvements in a growth framework focused on utility, rather than income. An important difference between health and many of the other determinants of income that are considered in the growth literature is that health is primarily valued in its own right, rather than for its effects on output. This has led to a certain politicization of the health–growth literature, in which the view that health is an important determinant of economic growth sometimes seems to be embraced in part because the widespread acceptance of such a view would lead to greater spending on health, which is viewed as a good thing in and of itself. For example, the WHO Commission on Macroeconomics and Health (Sachs, 2001) writes

Improving the health and longevity of the poor is an end in itself, a fundamental goal of economic development. But it is also a means to achieving the other development goals relating to poverty reduction. The linkages of health to poverty reduction and to long-term economic growth are powerful, much stronger than is generally understood. The burden of disease in some low-income regions, especially sub-Saharan Africa, stands as a stark barrier to economic growth and therefore must be addressed frontally and centrally in any comprehensive development strategy.

The rest of this article is organized as follows. [Section 3.2](#) presents the facts regarding the relationship between income and health, both between and within countries. [Section 3.3](#) presents a very simple theoretical framework for thinking about the simultaneous determination of health and income, and then uses this framework to highlight some of the key issues that will inform the rest of the article. [Section 3.4](#) looks at the role of income and other factors in explaining improvements in health, taking both a historical approach (focused on the currently wealthy countries), and looking at differences between rich and poor countries today. [Section 3.5](#) focuses on causality running from health improvements to income growth. I lay out several channels that theoretically could lead to such causality, discuss available evidence, and address the problem of quantifying the overall effect. In this section, I also discuss empirical work that has assessed the overall effect of particular episodes of health improvement historically. [Section 3.6](#) presents a framework in which one can assess health as an aspect of economic growth, in practice producing an income-equivalent measure of the value of health improvements. I also discuss how this framework can be parameterized using data on the revealed value of living vs. dying, and some of the problems this approach raises. [Section 3.7](#) concludes.



3.2. FACTS

I start by laying out the facts regarding the relationship between income and health, cross-sectionally among countries, cross-sectionally within countries, and over time. I use a number of indicators of health, because health is by its nature a multidimensional concept. One natural and widely used measure of health is the probability of death, as captured by life expectancy or the infant mortality rate. But variations in death probabilities are far from fully informative about the health status of the living. Some conditions that cause premature death may leave little health impact on those who survive, and may even raise the health of the living via selection. Other conditions that cause high mortality (for example, smallpox) also leave a great deal of physical damage among survivors. Similarly, “improvements in health” can take the form of reduced probabilities of dying, better health among those who are alive, or both. Even within the category of health of the living, there are many different dimensions. Some conditions may impact a person’s physical but not mental functioning, or vice versa. Similarly, some conditions may have a larger relative effect on quality of life or utility on the one hand, compared to economic productivity, on the other. And of course, the economic impact of a specific condition will vary with the structure of the economy: the relative wage of brawn relative to brains has declined as countries have developed, meaning that the relative productivity decrement from physical vs. mental disability has declined as well (Galor and Weil, 1996).

3.2.1 Cross-Section

3.2.1.1 Cross-Country Data

Life Expectancy

Life expectancy at birth is the number of years that a newborn baby would be expected to live, using current age-specific survival rates. Life expectancy is thus a scalar summary measure of the underlying vector of age-specific survival rates, which demographers call the life table. (Age-specific survival is not actually measured in many instances, and the full set of life table values is imputed from observation of only a few elements, such as infant mortality). In principle, a given life expectancy at birth is consistent with many different possible shapes of the survival function; in practice, there are empirical regularities regarding how the survival function changes shape as life expectancy rises. Demographers construct model life tables that embody these regularities (sometimes with adjustments for the constellations of diseases found in different locations or historical eras). Figure 3.1 shows the probability of survival to different ages for a family of model life tables for a variety of life expectancies.¹ The figure shows that infant and child survival is the most important component of increased survival associated with increases in life expectancy

¹ Li and Gerland (2011). This is the general table. Data are for females.

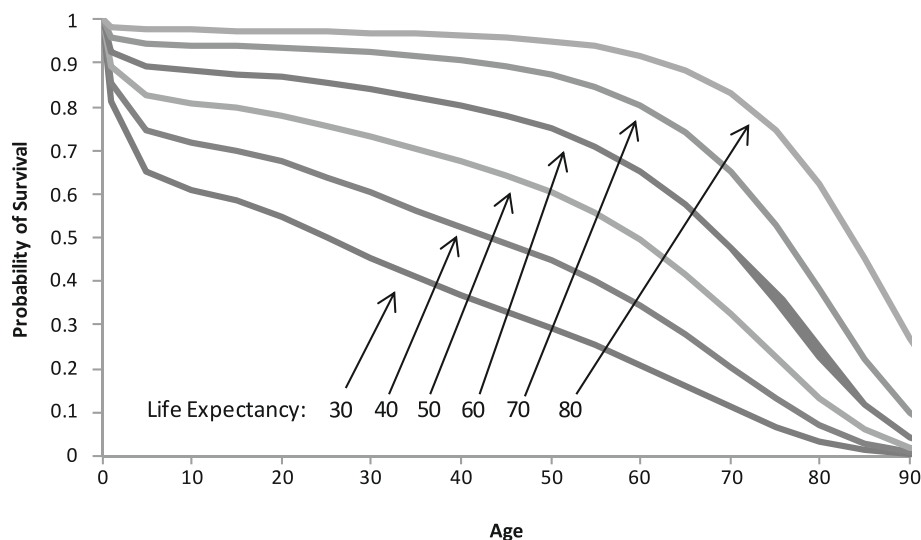


Figure 3.1 Model life tables.

(from a low level). This pattern is close to universal in examining both cross-sectional differences and time trends in life expectancy (with the effect of HIV in Africa today being an exception). An implication of this regularity is that differences in life expectancy at ages other than birth tend to be far smaller than differences in life expectancy at birth. (Another implication of the typical pattern of change as pointed out by [Peltzman \(2009\)](#), is that as life expectancy rises, inequality in experienced lifetimes declines. In the US, the Gini coefficient for lifetimes declined from roughly 0.50 to 0.12 between 1850 and 2000.)

[Figure 3.2](#) shows the cross-sectional relationship between the log of income per capita and life expectancy, using data from 2009. There is obviously a very strong relationship between the two. The R-squared from a simple regression of life expectancy on the log of income per capita is 0.67. There are no major outliers lying above the regression line, and those lying below are characterized by high rates of HIV (South Africa and Botswana), war (Afghanistan), or are oil producers that only recently experienced enormous increases in income (Gabon and Equatorial Guinea).

Years Lost to Disability

Life expectancy is often used as a measure of the health impact of the disease environment because premature death is the most significant (and certainly the most observable) impact of disease. But death is not the only impact of disease. In the scheme of the World Health Organization, Disability Adjusted Life Years (DALYs) lost as a result of a disease or injury are the sum of years lost to premature death (Years of Life Lost, YLLs), and healthy life

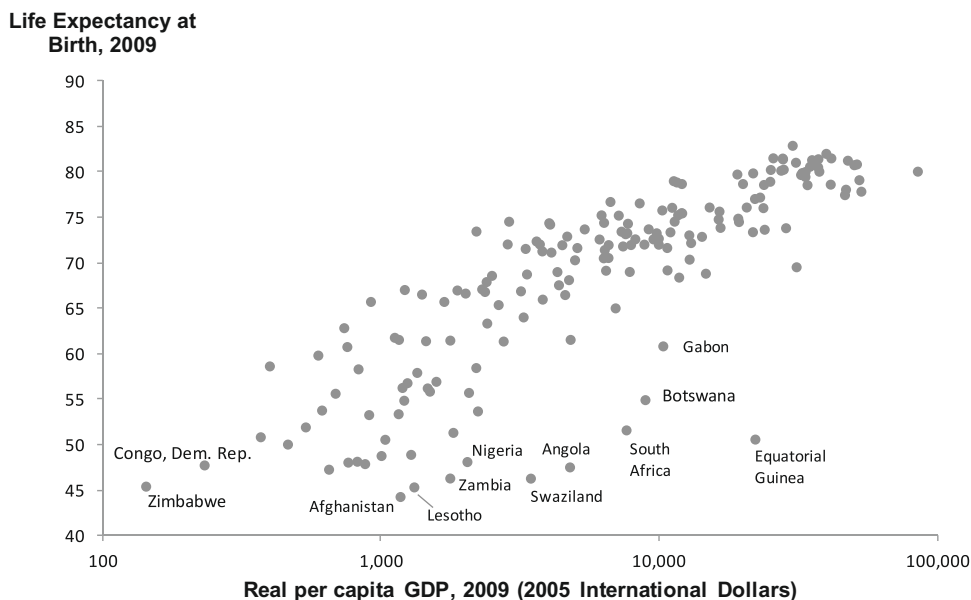


Figure 3.2 Income and life expectancy across countries.

year equivalents lost as a result of being in a state of poor health or disability.² The latter are called Years lost to Disability (YLDs) and can thus be thought of as a measure of the non-death costs of disease. According to [Mathers et al. \(2008\)](#), 60% of DALYs lost in 2004 were due to premature mortality, with the other 40% due to non-fatal health outcomes.

[Figure 3.3](#) shows the cross-sectional relationship between life expectancy at birth and YLDs, looking across WHO country groupings. There is clearly a very tight fit, establishing that health as measured by deaths and health as measured by sickness among the living, vary in tandem. However, it is worth noting that, at least in this data, the gap in life expectancy understates the gap in the health of the living: the poorest regions in the world have roughly twice the rate of YLDs as the richest, while the gap in life expectancy at birth is closer to a factor of 1.7.

Other Health Measures

Beyond summary measures such as life expectancy and years lost to disability, one can look at individual indicators of health. [Figure 3.4](#) shows data from [Shastri and Weil \(2003\)](#) on the cross-country relationship between income and the fraction of women

² Equivalence between healthy life years and years under different states of poor health or disability is established using a person trade-off in which experts compare the utility of living with different conditions to the utility of living fewer years disability-free.

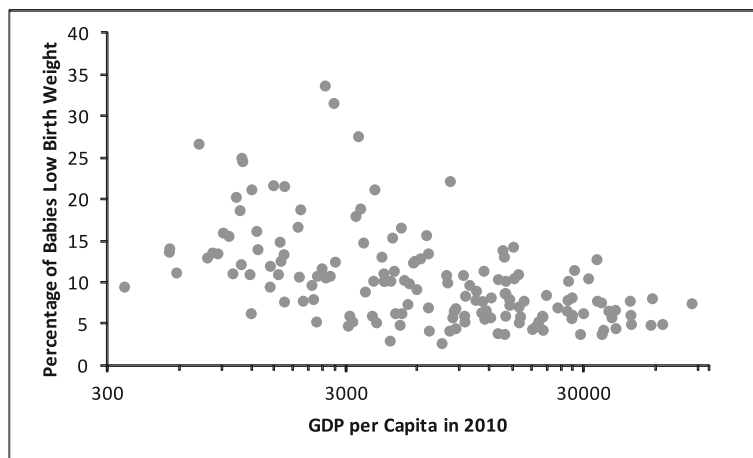


Figure 3.5 GDP and low birth weight.

blood cells) and helminth infections (which lead to intestinal bleeding). Anemia has negative effects on fetal and child growth as well as cognitive function of students, and increases morbidity and mortality among people of all ages. Anemia also affects a person's stamina, making him or her tire more easily, thus causing workers to be less productive (Thomas and Frankenberg, 2002). Although anemia is clearly only one dimension of health (and is far more prevalent among women than among men), it is of particular interest because there exist direct measures of its effect on productivity, which are discussed in Section 3.5.

Figure 3.5 shows the fraction of babies that are classified as low birth weight for a cross-section of countries.³ As discussed further below, birth weight is a useful summary measure of health and nutritional insults in utero, a crucial period for human development. Low birth weight is correlated with high blood pressure and many other health conditions, as well as reduced cognitive development.⁴ Behrman and Rosenzweig (2004) and Black et al. (2007) show that differences in birth weight among identical twins translate into differences in education and wages among adults.

Other indicators of health that one can look at in cross-section include age of menarche (the onset of menstruation, Weil (2007)), height (Subramanian et al. 2011), and body mass index (BMI).

³ Data on GDP and low birth weight are both from the WDI database. Low birth weight is for the most recent year available in the range 2000–2010.

⁴ Almond et al. (2005) point out that it may not be low birth weight per se that causes poor health outcomes, but rather other inputs to health that cause both low birth weight and poor health outcomes. Thus, policies that directly target a reduction in low birth weight will not necessarily have the impact on other health measures that would be predicted by the correlation between health outcomes and low birth weight.

3.2.1.2 Within-Country Covariation of Health with Income

The relationship between income and health or life expectancy that is observed across countries is echoed in within-country data. [Deaton \(2003\)](#), using US data, calculates that the probability of a 50-year-old man dying within the next 9 years was more than twice as high for men in families with income below \$10,000 as in households with income above \$60,000 (1980 dollars). Income has more effect on health outcomes at the lower end of the income distribution. [Deaton and Paxson \(2001\)](#) find that in general, higher income and education both reduce mortality within the US, although there is evidence that short-run increases in income may raise mortality for males. More specifically, using data from the National Longitudinal Mortality Study (Table 4.4), they find that the elasticity of mortality risk with respect to income per adult equivalent is -0.35 for men and -0.26 for women (when education is not controlled for), and the semi-elasticity of mortality with respect to years of education is -0.037 for men and -0.038 for women (when income is not controlled for). The effects when both income and education are controlled for are inconsistent between men and women, and more generally Deaton and Paxson argue that it is hard to see in their data whether the effect of education operates solely through income or has an independent effect as well. [Case et al. \(2002\)](#) show that there is a significant gradient of child health with respect to income in the United States, and that the gradient grows steeper as children age, reflecting the accumulation of adverse health impacts over children's lives.

[Gwatkin et al. \(2007\)](#) present data on a large number of health indicators broken down by quintile of wealth (rather than income) for 56 developing countries. The underlying data come from the Demographic and Health Surveys (DHS). [Table 3.1](#) shows several representative indicators.

Turning to measures of adult health beyond mortality, [Floud et al. \(2011\)](#) show a strong relationship between economic outcomes, on the one hand, and markers of nutritional status, on the other. Earnings increase with height for both Union Army veterans in the 19th century and for modern American males. Similarly, in both time periods, risk of poverty and non-labor force participation rise as body mass index falls below a cutoff of approximately 24. In developing countries, the relationship between height and income is more steeply sloped than in rich countries. In five different samples from the United States and United Kingdom, [Case and Paxson \(2010\)](#) estimate semi-elasticities of wages

Table 3.1 Health indicators by wealth quintile in developing countries

Wealth quintile	1	2	3	4	5
Infant Mortality	85.0	80.1	75.6	65.1	50.1
Under-5 Mortality	135.4	129.0	120.2	102.5	73.5
Prevalence of Diarrhea in Children	19.0	18.2	17.4	16.5	13.9
Moderate Stunting in Children	21.8	19.6	18.3	16.2	12.1
Moderate Underweight in Children	20.5	18.9	17.0	14.8	11.1

with respect to adult height (controlling only for ethnicity) of between 0.48 and 1.1% per centimeter for men and 0.26 and 1.1% for women. In Mexican data, [Vogl \(2012\)](#) finds a semi-elasticity of wage with respect to height of 2.5% per centimeter. In the Indonesian data for 1997, [Thomas and Frankenberg \(2002\)](#) find that a 1% increase in height is associated with a 5% increase in wages (implying a semi-elasticity of roughly 3.1% per centimeter). In the same data, the elasticity of wage with respect to BMI, not conditioning on other factors, is 2. Unlike the US data, the relationship between BMI and log wage is linear throughout the range of observed BMI.⁵

Conditioning on other determinants of wages does not eliminate the effect of height. [Schultz \(2002\)](#) regresses log wages on height, controlling for education, experience, and rural residence. An extra centimeter raises wages by 1.5% for men and 1.7% for women in Ghana; 1.4% for men and 1.7% for women in Brazil; but only 0.45% for men and 0.31% for women in the United States. Height is believed to be related to economic outcomes through one or more channels: because taller people are healthier and stronger, and these characteristics are rewarded in the labor market; because adult height is affected by childhood inputs that also contribute to cognitive ability, which is rewarded; or because height affects self-esteem or social status, which in turn affect wages ([Currie and Vogl, 2013](#)).⁶

3.2.2 Historical

Economists studying long-run growth have established a set of rough facts describing the historical evolution of income per capita and the world distribution of income. See [Lucas \(2000\)](#) for a summary. Although health data are just as imprecise as those for income, and health is itself, as mentioned above, a fundamentally multidimensional concept, it is nonetheless the case that in rough terms the evolution of health looks very similar to that for income. In particular:

1. In the period prior to the Industrial Revolution, there was little or no long-run change in countries' levels of health, though with considerable short-run variations.
2. Prior to the Industrial Revolution, cross-sectional differences among countries were relatively small.
3. The 19th century saw a takeoff of health status in Europe and its offshoots, with little change elsewhere, leading to growing health inequality among countries.
4. Starting in roughly the middle of the 20th century, health improvements in trailing countries began to exceed those in the leaders, resulting in a narrowing of the cross-sectional health variance.

⁵ Thomas and Frankenberg find that a good deal of the predictive power of BMI for wages goes away once they control for height and education. This shows that much of the predictive effect of BMI on wages results from the endogeneity of BMI, rather than a direct effect of health on income.

⁶ [Baten et al. \(forthcoming\)](#) show a negative correlation between height and innumeracy, as measured by age-heaping in survey data, for a variety of historical data sets.

It is with respect to the last of these four points that the analogy between the evolution of health and that of income breaks down most significantly. While growth economists question why the convergence of income per capita has been so slow (to the extent that it has happened at all), convergence in health measures has been quite rapid.

3.2.2.1 *Life Expectancy*

At the end of the 18th century, Malthus wrote “With regard to the duration of human life, there does not appear to have existed from the earliest ages of the world to the present moment the smallest permanent symptom or indication of increasing prolongation.” He was basically right. Prior to the 19th century, data on life expectancy come from diverse and sometimes inconsistent sources, including family reconstructions, census records, and temple and parish records. Though imprecise, the picture painted by these data is of little or no improvement in life expectancy over a span of millenia, as well as some cross-sectional variation, with Europe (and Japan) being slightly healthier than the rest of the world. [Maddison \(2001\)](#) reports life expectancy in Roman Egypt at 24 years, the same as the value for England in the 14th century. By the middle of the 18th century, life expectancy was 35 years in England, but still 25 years in France. In Japan at the same time, life expectancy was in the early 30s.⁷

[De la Croix and Licandro \(2012\)](#) conduct an examination of long-run mortality trends looking at biographies of 300,000 famous individuals born starting in the 24th century BCE. By construction, their data focus only on adults (who lived long enough to become prominent) and on regions that were sufficiently developed that written biographical records survive, primarily Europe. De la Croix and Licandro date the beginning of mortality improvements to the cohort born 1640–1649, more than a century earlier than most other sources. The mean lifespan of famous people was 60 years in the four millenia prior to that; by the time of the cohort born in 1869, it had risen to 69.

Around 1800, life expectancy started to increase, first in Europe and its offshoots, spreading to the rest of the world by the middle of the 20th century. Average life

⁷ Historical data for regions outside of Europe are extremely sparse. [Acemoglu and Johnson \(2007, Appendix C\)](#) provide an extensive and well-documented compilation of estimates for developing countries in the first half of the 20th century (these data underlie [Figures 3.11](#) and [3.12](#) below). [Riley \(2005\)](#) estimates that prior to the “health transition” (he uses a different definition than Acemoglu and Johnson) that began in Africa in the 1920s, life expectancy at birth averaged 26.4 years. In Asia, life expectancy prior to the health transition, which started there between 1870 and 1890, was 27.4. In Europe, the transition started in the 1770s, and prior to it life expectancy was 34.3. Riley comments that available estimates of African mortality prior to the health transition all come from European colonies in Africa. There is a reasonable basis for thinking that life expectancy may have been higher prior to colonization, the arrival of Arabic speaking merchants, and the dislocations produced by the slave trade. Unfortunately, almost no information for this period is available. [Steyn \(2003\)](#) examines mortality in the pre-colonial period in northern South Africa through an examination of skeletal remains. She estimates life expectancy in the period 1000–1300 AD at 23.2. Remains for the post-1830 period show a slight decline in life expectancy after the expansion of European influence.

expectancy in Western Europe rose from 36 in 1820 to 47 in 1900, 67 in 1950, and 78 in 1999. In the analysis of [Oeppen and Vaupel \(2002\)](#), life expectancy in the “best practice” countries (those with the highest life expectancy in the world) has increased linearly since 1840 at a pace of 3 months per annum, with no sign of a slowdown.

In the last half of the 20th century there were rapid gains in life expectancy associated with the international epidemiological transition in which modern health technologies were quickly diffused to the developing world ([Acemoglu and Johnson, 2007](#)). Between 1950 and 1999, life expectancy rose by 22 years in both Brazil and Mexico, 28 years in India, and 30 years in China. The pattern of widening and subsequent narrowing of the world health distribution can be seen in the difference between life expectancy in the United States vs. the world average. In 1820, this gap stood at 13 years (39 vs. 26). By 1900 it had risen to 16 years (47 vs. 31), and by 1950, 19 years (68 vs. 49). By 1999, however, the gap had narrowed to only 11 years (77 vs. 66) ([Maddison, 2001](#)).

Another way to see this same phenomenon is to look at the speed with which different countries traversed a particular set of life expectancies. For example, in India, life expectancy increased from 26.9 years in 1930 to 55.6 years in 1980. In France, a roughly comparable change took more than three times as long: Life expectancy at birth was 27.9 years in 1755 and reached 56.7 years only in 1930 ([Livi-Bacci, 1997](#); [Kalemli-Ozcan, 2002](#)).

Since 1960, the cross-country standard deviation of the infant mortality rate has fallen by almost 40%. However, the cross-sectional standard deviation of life expectancy fell from 1960 to 1990 before turning upward due to the effects of HIV. By 2004, it had returned to its 1960 level. Similarly, [Soares \(2007\)](#) shows that there was “ β convergence” in cross-country life expectancy (lower life expectancy predicting faster growth in life expectancy) from 1960 to 1990, but not thereafter.

3.2.2.2 Other Health Indicators

Data on other health indicators show improvements that parallel the increase in life expectancy. [Figure 3.6](#) shows data from [Weil \(2007\)](#) on the adult height and the adult survival rate (the probability of living from 15 to 60 years of age, using the current life table) for 10 countries covering different time intervals, up to 180 years. In a regression with country and year fixed effects, an increase in 10 percentage points in the adult survival rate is associated with a rise in adult height of 1.6 cm. Over the period 1775–1995, average height in Great Britain rose by 9.1 cm.^{8,9}

⁸ Although height is a useful measure of long-run growth within countries, it does not perform well in cross-section as a measure of the standard of living. [Deaton \(2007\)](#) examining data for women in Demographic and Health Surveys for 43 developing countries, finds no consistent relationship between adult height on the one hand and mortality rates or living standards from the period when those women were children, on the other.

⁹ [Fogel \(1994\)](#).

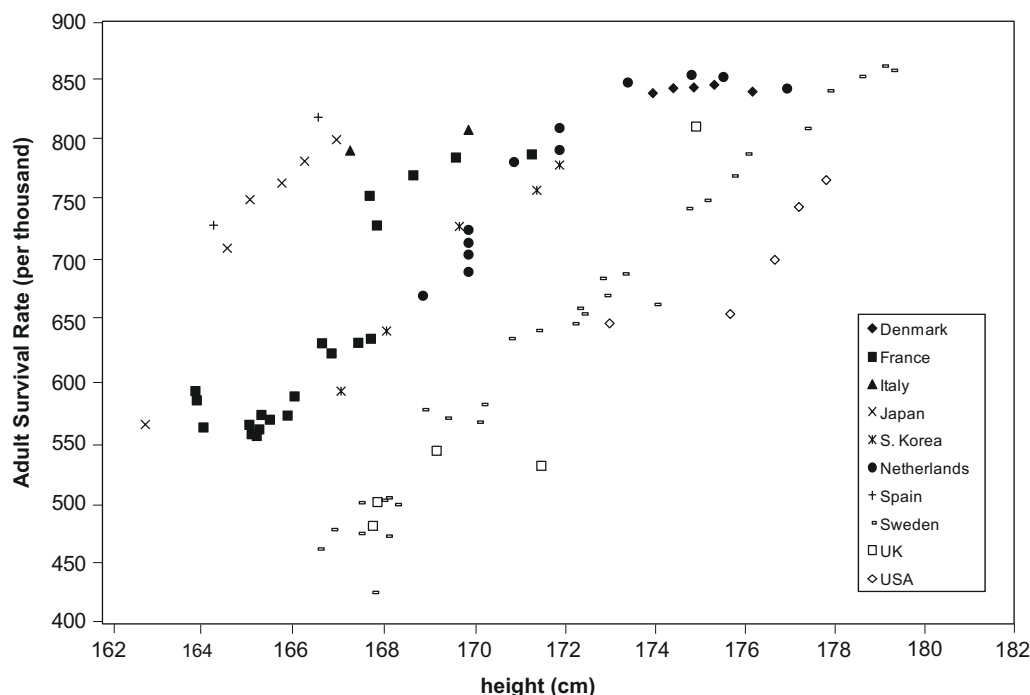


Figure 3.6 Height and adult survival.

The pattern of rapid catch-up during the second half of the 20th century that is observed in the case of life expectancy is repeated for other health measures. [Figure 3.6](#) shows that the relationship between height and adult survival is roughly linear. But what one cannot see in that figure is that the time scale over which these measures grew is not the same for all countries. In Sweden, whose experience is typical for Europe, height increased by 5.5 cm between 1820 and 1900 and a further 6.8 cm between 1900 and 1965. By contrast, in South Korea, the height of adult males rose by 4.8 cm over the 33-year period, 1962–1995, and in Indonesia, adult height attainment as a function of birth year rose by 1.5 cm per decade between 1925 and 1955.¹⁰ [Schultz \(2010\)](#) reports differences in adult female height for birth cohorts separated by 30 years (25–29 years old vs. 55–59 year olds, as measured in roughly 1990) of 3.10 cm in Brazil and 3.43 cm in Vietnam, but smaller jumps of 1.60 in Ghana and 1.54 in Côte d’Ivoire.¹¹ Similarly, among industrialized countries in Europe, there was a roughly linear decline in age at

¹⁰ [Sohn \(2000\)](#) and [Thomas and Frankenberg \(2002\)](#).

¹¹ [Currie and Vogl \(2013\)](#) suggest that the slow rate of increase in height in some developing countries may be explained by decreased selection into mortality of unhealthy children.

menarche of 0.2–0.3 years per decade over the period 1860–1980. By contrast, in South Korea, age of menarche fell from 16.8 to 12.7 between 1958 and 1998, a decline of more than 1 year per decade.¹²

A final measure of health is intelligence. Intelligence is a combination of biological aspects of human development and education, whether formal or informal. Thus it falls on the border of health and more conventionally measured human capital from schooling. Nonetheless, there is a good deal of evidence that the aspect of intelligence that is related to biological health has risen over time in developed countries. The so-called Flynn effect refers to the rise in measured IQ that has been observed in many developed countries over the last half century or more (Flynn, 1987). Test scores have been rising at a rate in the neighborhood of three points (out of 100) per decade. The specific IQ tests on which researchers focus (predominantly the Raven's progressive matrices) are designed to measure fluid intelligence, which in theory should not reflect skills acquired in schooling. A number of the health insults and nutritional deficiencies both in utero and very early in life are known to affect intelligence. Thus, the Flynn effect is often taken as resulting from improved nutrition and health over time (Lynn, 1998). Martorell (1998) reports on a number of studies that estimate the impact of low birth weight in currently developed countries as six IQ points among early school age children, and speculates that in more impoverished environments the effect is larger. He also reports that severe, clinical malnutrition is associated with an IQ deficit of 15 points. Within populations, the correlation between body size and IQ tends to be higher in environments where food intake is limited (Sigman and Whalley, 1998). Eppig et al. (2010) find a very strong statistical relationship between the prevalence of intestinal parasites and average IQ, looking across countries, even when controlling for GDP per capita and average years of education. They theorize that parasites compete for nutrients that are needed for proper development of the brain. They suggest that the Flynn effect may be due to reduced pressure from infectious diseases.¹³ While most studies of the Flynn Effect use data from developed countries, Daley et al. (2003) examine data on children in rural Kenya roughly 4 months after school entry at two points in time (1984 and 1998). They find improvements in IQ commensurate with trend growth observed in industrial countries. The fraction of children with insufficient nutrition in their samples fell from 56% in the first cohort to 36% in the second, and the fraction with hookworm fell from 36% to 18%.

¹² Hwang et al. (2003) and Eveleth and Tanner (1990).

¹³ There is also a strong relationship between IQ and income per capita, looking across countries. Jones (2011) reports the correlation as 0.7, and that the increase in GDP per capita associated with a one point increase in IQ is far higher than the within-country increase in individual wages associated with the same change in IQ (6–7% for the former vs. roughly 1% for the latter). He argues that there is an effect of IQ on national income that goes through channels outside individual productivity, such as higher patience and ability to solve public goods problems.

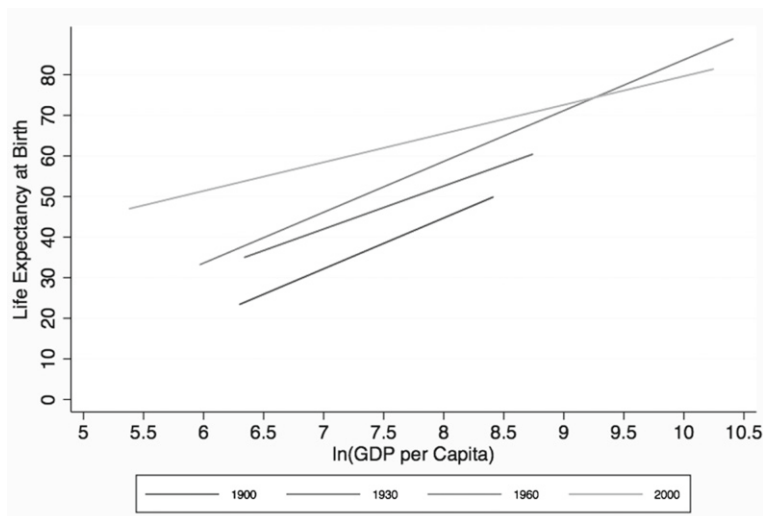


Figure 3.7 Preston curves by year.

3.2.3 Changing Relationship Between Income and Health

Changes over time in the relationship between income and health were most famously pointed out by [Preston \(1975\)](#). Preston noted that the curve representing the relationship between income and life expectancy had shifted upward over time. He estimated that at most, 1/3 of the increase in life expectancy observed between 1930 and 1960 could have been a result of increasing income, with the rest due to the shift in the curve. [Figure 3.7](#) shows a family of estimated Preston curves for cross-country data for the years 1900, 1930, 1960, and 2000.¹⁴

Another way to look at the relationship between health and income is to examine short-run changes in the two measures. From the cross-sectional relationship, it is clear that over very long periods of time (for example, since the beginning of the 19th century, at which time both income and health differences among countries were small) there must be a positive correlation between the growth of income and the growth of life expectancy. However, as discussed below, there are many reasons why such a relationship might not hold at high frequencies. [Figure 3.8](#) shows the relationship between 40-year changes in the two measures. Weighting country observations by population, the R-squared of a regression of change in life expectancy on change in $\ln(\text{GDP/capita})$ is 0.50, and the coefficient implies that a change of 1% in GDP per capita is associated with an increase in life expectancy of 0.13 years.

¹⁴ Data are from [Acemoglu and Johnson \(2007\)](#). Each curve is graphed for the range of income values found in the data. Observations are weighted by country population.

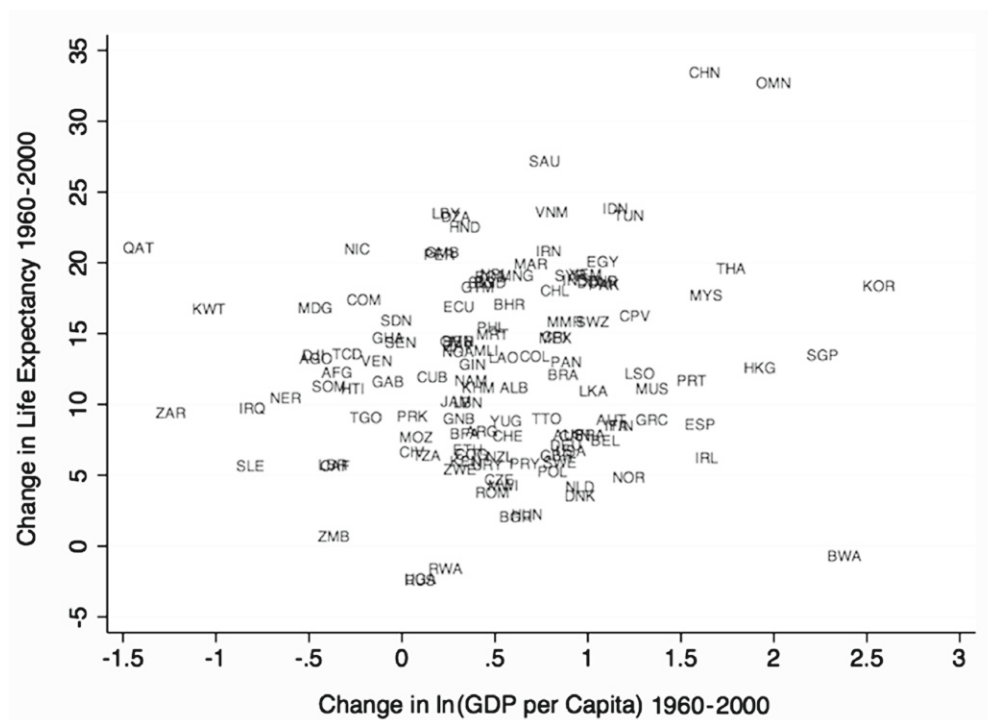


Figure 3.8 Changes in GDP and life expectancy.



3.3. INTERACTION OF HEALTH AND INCOME: A THEORETICAL FRAMEWORK

The various pieces of evidence presented above establish that, in a statistical sense, income and health are strongly related. The exact nature of the correlation varies with the setting (cross-section, time series, country vs. individual), but it is clearly strong. As is usual in economics, the real debate is over what structural relationships underlie these observed data. What causes what, how much variance does this causality explain, and at what time horizon?

As a starting point, one can think of health and income being determined simultaneously. Figure 3.9 presents a simplified framework in which y represents income per capita and ν (for vitality) represents health. The effect of higher income in improving health is represented by the $\nu(y)$ curve. The effect of better health in raising income is represented by the $\gamma(\nu)$ curve. Equilibrium health and income are given by the intersection of the two curves. In this abstract form, the model can be thought of as applying equally well to either individuals or countries.

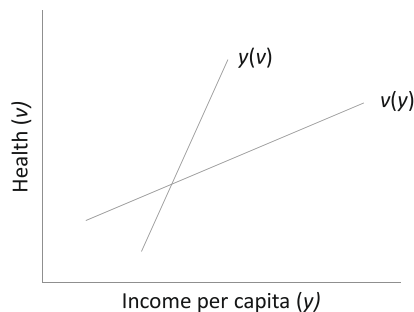


Figure 3.9 Interaction of health and income.

In this simple model, a positive correlation between health and income (looking across countries or individuals or over time) can be induced by three forces:

1. Variation in the $y(v)$ curve, holding the $v(y)$ curve fixed. This would be due to factors other than health that affect income. Examples in cross-country data could be availability of natural resources, differences in institutions or productive technology, etc. Among individuals, shifts in the $y(v)$ curve could be caused by variation in non-health aspects of human capital. Such variation would trace out the $v(y)$ curve, and so in order to match the observed positive correlation between v and y in the data, it would have to be the case that the $v(y)$ curve was upward sloping. In other words, it would have to be the case that raising income improved health.
2. Variation in the $v(y)$ curve, holding the $y(v)$ curve fixed. This would be due to factors other than income that affected health, such as variation in the “disease environment” across countries, or variation in idiosyncratic health outcomes across individuals. Such variations would trace out the $y(v)$ curve, and so for the observed data to fall on a upward sloping line it would have to be the case that the $y(v)$ curve had a non-zero slope (when viewed in a rotated fashion). In other words, it would have to be the case that improving health actually did raise income.
3. Correlated shifts in both curves. This would be the case if some factor shifted both health and income. Looking over time, a natural candidate to produce such correlated shifts is technology, which allows for higher output (given a set of factor inputs) and for better health, holding income constant. Looking across countries, one might think that differences in institutional quality would produce correlated shifts of the two curves. Finally, looking among individuals, a natural candidate for producing such correlated shifts would be education, which raises wages and imparts knowledge that improves health at any given wage level. Correlated shifts in the $v(y)$ and $y(v)$ curves would produce an upward sloping relationship between y and v even if both of these curves had zero slope (in other words, even if there were no causal link from health to income or vice versa).

The empirically observed pattern of health and growth in any particular setting will depend on the slopes of these curves, the relative variances of shocks to them, and the covariance of such shocks.

As in any model where the two curves describing structural effects slope in the same direction, there will be multiplier effects in this simple setup. For example, some exogenous change that affects the level of income, holding health constant, will shift the $\gamma(\nu)$ curve to the right, raising income directly, but also improving health and resulting in a second round of health-induced increases in income. There will be similar multiplier effects to exogenous shocks to health. These multiplier effects will be larger, the larger are the responses of income-health and health-income. Similarly, it is not hard to introduce nonlinearities in one or both of these relationships that could produce multiple equilibria.

To a large extent, debates about the importance of health in economic growth can be boiled down to claims about the slopes of, as well as the relative variances and correlations of shocks to, the $\gamma(\nu)$ and $\nu(\gamma)$ curves. [Sachs \(2001\)](#) stresses the variability of the underlying health environment across countries, arguing that even if tropical countries were rich, they would still be unhealthy. Implicitly, he views the variance of the $\gamma(\nu)$ curve to be small, and so the observed data on γ and ν will trace out the $\gamma(\nu)$ curve—and thus we learn from the data that the $\gamma(\nu)$ curve is steeply sloped—health has a big effect on income. By contrast, [Acemoglu and Johnson \(2007\)](#) interpret their results (discussed below) as showing that the $\gamma(\nu)$ curve is flat, and so the correlation between health and income observed in the data results from a combination of correlated shocks to the two and causality running from income to health. [Pritchett and Summers \(1996\)](#) use an instrumental variables approach to argue for a positive effect of income on health—that is, that the $\nu(\gamma)$ curve is not flat.

Looking at the within-country covariation of health and income, [Cutler et al. \(2006\)](#) argue that relatively little is due to causation running from income to health—in other words, that the $\nu(\gamma)$ curve is relatively flat. Rather, they view the two most important sources of the observed correlation to be causation from health to income (in particular, the effect of disability on wages) and the effect of education in producing correlated shocks to both curves.

An important observation is that the degree to which different causal channels shape the relationship between income and health need not be the same in all contexts, that is, across countries, historically within individual countries, or cross-sectionally within a country. Indeed, as discussed below, there is good reason to think that this is not the case.

3.3.1 Timing

The framework presented above abstracts from the dynamics of adjustment in health and income. Such an approach is reasonable if one is thinking about differences among countries that vary greatly in their levels of health and income, or alternatively, if one is thinking about changes over very long time spans, like centuries. In considering shorter

timespans—for example, in thinking about a country undergoing rapid economic growth or a rapid improvement in health—the dynamics of the process become important.

3.3.1.1 Delays in Health Improvements Due to Human Physiology

One important source of dynamics in the health-growth relationship results from delays inherent in the process of human development. Adult health, and thus the labor input of adult workers, is strongly affected by health conditions early in life, or even prior to birth. Thus, the health of adults does not immediately respond to changes in the health environment.

The most obvious manifestation of this phenomenon is height. In many countries that have experienced rapid income growth, there is a striking age gradient in height among adults: young adults tower over their elderly grandparents. The crucial periods for determining adult height are in utero, during childhood up to the age of four, and then during the adolescent growth spurt. After the early 20s, at the latest, nutrition does not affect adult height. [Fogel and Engerman \(1992\)](#) find that slaves who were fed abundantly only after they entered the labor force (after age 10) remained stunted by at least four inches in adulthood.

The 9 months of gestation are a particularly important period for determining adult health outcomes. Nutritional deficiencies, either in terms of total calories or in terms of specific micronutrients, as well as other health insults, can all have major impacts on adult health. For example, [Bleichrodt and Born \(1994\)](#) estimate that iodine deficiency in utero causes reductions in adult IQ averaging 13.5 points. The Barker Hypothesis holds that fetal malnutrition is associated with ill health, particularly in the form of chronic diseases such as diabetes and coronary artery disease, in the adult years that follow reproductive age (see [Almond and Currie \(2011\)](#) for a review). The hypothesized channel is “fetal programming” via the mechanisms of epigenetics. Nutritional deficiency in utero is often reflected in low birth weight, but this need not be the case. Fetuses exposed to malnutrition only early in gestation may attain normal birth weight but still suffer long-term damage to health.

Beyond malnutrition, there are also disease insults. [Almond \(2006\)](#) shows that in the United States, cohorts exposed to Spanish Influenza in utero had lower educational attainment and higher rates of disability than surrounding birth cohorts. Wages of men were 5–9% lower because of in utero exposure to the infection. Other examples of health insults in utero that produce lifelong health impairments include congenital rubella syndrome, fetal alcohol syndrome, and the effects of maternal smoking. [Case et al. \(2002\)](#) find that maternal smoking affects health outcomes in middle age, even controlling for an individual’s education as well as health and social status earlier in adult life.

Nutrition and disease in childhood can also have lifelong effects. For example, in areas where malaria is endemic, most people have developed substantial immunity by age five. However, cases of cerebral malaria in young children leave lifelong scars. In sub-Saharan

Africa, the prevalence of malaria episodes among adults (that is, the fraction suffering at any point in time) is only half of the prevalence among adults of neurological sequelae from childhood episodes of cerebral malaria (Ashraf et al. 2009). In addition to its direct effects on adult health (and thus adult productivity), ill health in childhood can also impact adult outcomes by reducing human capital accumulation. For example, Bleakley (2007) shows lifelong effects on education and wages from exposure to hookworm during childhood. Bleakley (2010), Cutler et al. (2010), and Lucas (2010) all use national anti-malaria campaigns in the middle of the 20th century as quasi-experiments in order to study the effect of childhood exposure to the disease on human capital accumulation and adult economic outcomes, though with varying findings. Cutler et al. find no effect of malaria eradication on schooling or literacy, no effect on adult female income, and a modest effect on adult male income. At the other end of the spectrum, Bleakley's estimate is that persistent childhood malaria reduces adult income (through a combination of adult health and human capital accumulation) by 50%. Lucas finds that a 10% reduction in malaria incidence raises completed schooling by 0.1 years.

Several studies have examined the long-run effects of childhood nutrition, using a variety of exogenous sources of variation in nutrition, including randomized controlled trials of nutritional supplementation as well as shocks to income during childhood, such as rainfall, war, and famine. These studies generally find that better nutrition leads to improvements in school completion, IQ, height, and wages.¹⁵ Case et al. (2005) suggest that the positive effect of parental income on child health, along with the positive effect of child health on adult economic status, may be an important pathway leading to the observed inter-generational correlation of economic outcomes.

While much of the literature looking at the relationship between child health and adult outcomes focuses on developed countries, where the data are better, Currie and Vogl (2013) suggest that the effect is probably larger in poor countries, where health insults are more frequent and are likely to positively reinforce each other. In order to achieve clean identification, most of the studies economists have conducted on the long-term effects of childhood health have examined one particular health insult (a nutritional deprivation or disease exposure) at a time. (Although Currie and Vogl also point out that in developing countries, the negative effects of early-life health insults on adult outcomes may be blunted by positive selection into survival.)

The extent to which adult health is a function of the adult health environment vs. the health environment that prevailed when current adults were young will be one of the factors determining the speed with which improvements in the overall health environment are translated into improvement in adult health (which is the aspect of health that matters for output). Ashraf et al. (2009), in their simulation of the output effects of health improvements, introduce a parameter that measures the relative importance of

¹⁵ See Currie and Vogl (2013) for an extensive review.

child vs. adult health inputs and show that the dynamic economic effects of health improvements are very sensitive to it.

3.3.1.2 Negative Short-Run Effects of Income Growth on Health

A second important dimension of timing has to do with the impact of income on health via inputs at the national level. As discussed below, an important contributor to health gains is improvements in public health infrastructure, most notably clean water and sanitation. Public health expenditures rise with national income, but in many cases there are appreciable lags in the implementation. Further, many of the short-term effects of economic growth can be deleterious to health.

Inter-regional or international migration, often associated with economic growth, can have negative health consequences, particularly in the short run. The most stark example of this phenomenon is the spread of old-world diseases to the Americas that followed the voyage of Columbus, which resulted in tens of millions of deaths. Smaller expansions of settlement have also produced similar results. In the early 20th century, the spread of Chinese settlement into Manchuria brought a new population into contact with rodents that harbored the bacteria causing plague, leading to a local outbreak that almost became a worldwide pandemic (McNeill, 1998). As described in McGuire and Coelho (2011), increases in the speed of transport in the centuries following Columbus allowed for ever more pathogens to make the leap between continents.

Another important source of increased disease exposure from economic growth is urbanization, both because it brings people into contact with new infectious agents, and because the collections of food and waste in cities make the spread of disease far more likely. Until the early 20th century, even in the most developed countries, cities were far less healthy than the countryside.

Costa and Steckel (1997) find that the average height of native-born residents of the United States declined by 4 cm between the cohort born in 1830 and that born in 1880. Life expectancy at age 10 also fell from the cohort born in 1790 to the cohort born in 1850.¹⁶ They suggest that the decline in health was due to greater exposure to infection resulting from inter-regional trade and migration, as well as to less healthy working conditions that accompanied the move away from farming and home manufacturing. (They do not view urbanization itself as a major cause of the health decline; as of 1860, only 10% of the population lived in cities with a population greater than 50,000.)

3.3.1.3 Health Improvements and Population Growth

A final dimension in which timing is important in considering the relationship between health and growth is in the entanglement of health with population growth. Health is related to population growth via infant and child mortality. One of the reasons that fertility was high in undeveloped countries was to compensate for the fact that so many

¹⁶ Floud et al. (2011), Figure 6.1.

newborns would not reach adulthood. A standard idea in demographic transition theory is that when mortality falls, there is a delay in the response of fertility, and that as a result of this delay there is a spurt of population growth. [Acemoglu and Johnson \(2007\)](#) attribute their finding (discussed below) that mortality reductions in developing countries led to a decline in income per capita to exactly this channel. Their IV estimate of the effect of a change in log life expectancy on the change in the log population size between 1940 and 1980 is 1.67. This implies that an increase in life expectancy from 40 to 60 years would raise population size by a factor of 1.97 over this 40-year period, which is an increase in the annual growth rate of slightly less than 2%. Acemoglu and Johnson claim that the negative economic effects of rapid population growth more than compensated for direct economic benefits from better health, and so income per capita fell.

While the approach of Acemoglu and Johnson is purely econometric, [Ashraf et al. \(2009\)](#) pursue the question of how much a reduction in mortality would be expected to affect population growth, and in turn economic growth, using a simulation model. The demographic side of the model is set up to roughly match the international epidemiological transition studied by Acemoglu and Johnson. Ashraf et al. consider a stylized economy in which age-specific mortality and fertility rates have been constant for sufficiently long that the age structure of the population is unchanging—what demographers call a stable population. The stable population is constructed with life expectancy at birth of 40 years and a total fertility rate of 5.2, yielding population growth of 1.5% per year. The authors then consider an instantaneous shock to health that raises life expectancy at birth to 60 years. To represent the effect of mortality reduction on fertility, they allow age-specific fertility to fall linearly so that after a fixed number of years the net rate of reproduction has returned to its pre-shock level. They trace through the effect of this change on population growth. They find that if fertility adjusts over a period of 50 years, the maximum increase in the population growth rate is 1 percentage point, and that at a horizon of 40 years, population is 1.36 times as large as it would have been without the reduction in mortality. This is significantly smaller than Acemoglu and Johnson's estimate of 1.97. Ashraf et al. also find that in their economic model (discussed below), the rise in life expectancy from 40 to 60 produces an increase in income of 2%. By contrast, the coefficient estimated by Acemoglu and Johnson, applied to this change in life expectancy, implies a decline in income per capita of 41%.

Ashraf et al. then experiment with altering their model so that it produces population dynamics similar to those estimated by Acemoglu and Johnson. This requires having fertility rise in response to a decline in mortality. In this case, they find that at a horizon of 40 years, income per capita would fall by 20% in response to the rise in life expectancy from 40 to 60. In other words, more than half of the gap between the findings of the two studies can be explained by the divergent conclusions regarding the response of fertility, and thus population growth, to a decline in mortality.



3.4. IMPACT OF INCOME ON HEALTH

The improvement in health and extension of life that has taken place around the world in the last two centuries, as described in [Section 3.2](#) of this chapter, is one of humanity's greatest accomplishments. As such, it has been the subject of a voluminous literature. In this section, I take a very selective tour through this literature, focusing in particular on the question of how much of the improvement in health can be attributed, either directly or indirectly, to increasing income. In assessing this question, of course, it is inevitable that one has to address the question of what else, if not rising income, is responsible for health improvements over time and health differences among countries.

The improvement in health that has taken place over the last two centuries resulted from three sets of forces: first, improvements in the standard of living, in particular, better nutrition; second, changes in the public health environment, including sanitation and the supply of clean water; and finally, improvements in medical technology, including antibiotics and other medical treatments. The extent to which credit for improved health should be divided among these sources is a matter of debate. Further, there are not only interactions among the different forces, but also cases where a particular health problem could be remedied by more than one channel (for example, both sanitation improvements and treatment with antibiotics will reduce mortality from infectious diseases).

In the countries that developed first, and in which health improvements began earliest, the three channels just mentioned had their primary effects on health in the order just discussed. In countries that experienced health improvements later, the time pattern has been more heterogeneous. Thus, for example, in some developing countries, the state of medical treatment today exceeds what was available in rich countries in the middle of the 20th century, while nutrition and public health lag further behind.

Improvements in health, as measured by mortality, can be linked to specific changes in both the ages at which people die and to the diseases that they die from. [Cutler and Meara \(2004\)](#) estimate that in the United States, 80% of life expectancy improvements in the first four decades of the 20th century were due to reductions in death before age 45, with two-thirds of that coming before age 15. In the last four decades of the century, by contrast, two-thirds of life expectancy gains came from mortality reductions at ages greater than 45. This change in the distribution of mortality improvement reflected in part the distribution of mortality itself: by the latter part of the 20th century, the infant mortality rate was low enough that even though the rate of mortality decline in this age group was the same as earlier in the century, the contribution to increased life expectancy was only a quarter as large. But it also reflected changes in the rate of progress at different ages. Mortality among the 65+ age group declined at a rate of 0.3% per year in the first 40 years of the century, vs. 1.1% per year in the last 40 years.

Reduced death from infectious diseases accounted for three quarters of the reduction in mortality in the first four decades of the 20th century. The rate at which mortality from

infectious diseases declined sped up appreciably in the period 1940–1960 with the deployment of antibiotics, but because mortality from these conditions was already lower than it had been in 1900, the contribution of infectious diseases to overall mortality decline was only half of what it had been in the century's first four decades. By 1960, infectious diseases accounted for only 5% of mortality. On an age-adjusted basis, death rates from both cardiovascular disease and cancer increased over the first 60 years of the century. Cardiovascular disease accounted for 22% of mortality in 1900 but 59% in 1960, while deaths from cancer rose from 5% to 15% over the same period. Between 1960 and 1990, declining death from cardiovascular disease was equal to 98% of entire decline in death rates, and it was for this reason that the decline in mortality was concentrated in ages above 45.¹⁷

3.4.1 The Standard of Living and Health Improvements

3.4.1.1 *Positive Effects of Economic Growth on Health*

McKeown (1976) famously argued that much of the reduction in mortality that took place over the last centuries was due to improvements in nutrition, rather than explicit interventions, either public health or medicine. Most significantly, McKeown showed that declines in mortality from a number of infectious diseases took place prior to any such intervention. For example, the death rate from tuberculosis declined by 80% from when his data begin in 1848 to the advent of effective treatment in the mid-1940s. Similarly, Cutler and Meara (2004) show that great reductions in death from infectious diseases that took place in the United States in the first four decades of the 20th century occurred before the availability of medical treatments such as sulfa drugs (invented in 1935) and widespread vaccination.¹⁸ McKeown's argument has been carried forward by Robert Fogel and co-authors in a series of articles and books. Fogel cites both direct evidence on caloric intake as well as data on the resulting changes in body sizes.

Caloric intake is the simplest measure of an input into health. As described in Floud et al. (2011), economic historians have put enormous effort into measuring this input. The majority of work has focused on Britain and France over the last two centuries. Data sources include estimates of total food production and imports; household surveys; and institutional records. In assessing average caloric intake, it is important to adjust for the demographic structure of the population, since children eat less than adults. Their estimate for France in 1785 is 2413 calories per standardized consuming unit (male age 20–39). In England in 1800, the equivalent was 3271 calories. Floud et al.'s estimate is that calories per consuming unit in England rose 20% between 1800 and 1913 and by a further 10% by 1960.¹⁹ In France, calories rose by 65% between 1800 and 1960. The rise in calorie consumption somewhat understates the degree to which nutrition improved,

¹⁷ Cutler and Meara (2004), Table 9.3.

¹⁸ See Deaton (2006) and Cutler et al. (2006) for extensive discussion of this argument.

¹⁹ Tables 4.13 and 5.5.

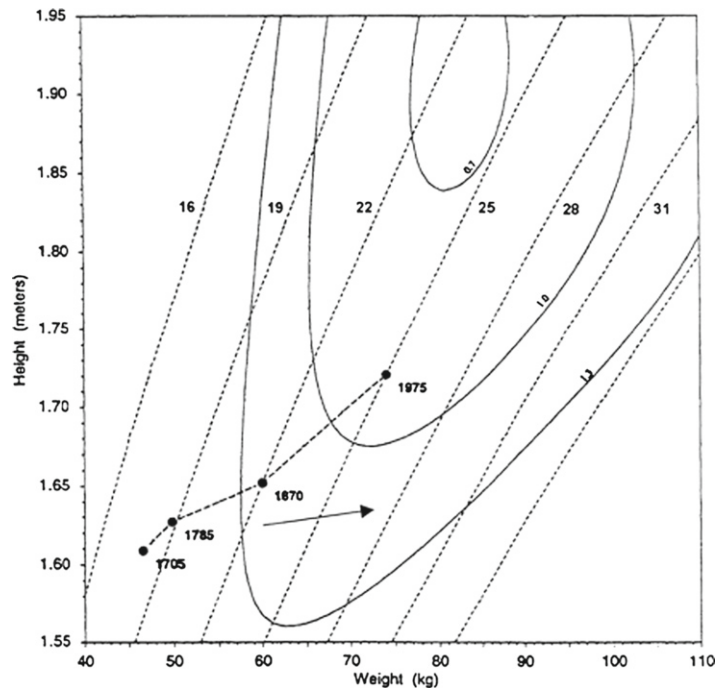


Figure 3.10 A Waaler surface.

in that the caloric demands of labor done by most adults have declined over time, so that more calories are left over for bodily maintenance.

Better nutrition translated into bigger bodies. As discussed above, both adult height and body mass index increased in leading countries over the last two centuries. The final piece of Fogel's argument that increases in living standards have been a major source of health improvement is the observed relationship between anthropomorphic measures, on the one hand, and health outcomes, on the other. [Figure 3.10](#) is an example of a Waaler surface, which shows the relationship between weight, height, and mortality risk. The oval-shaped curves are iso-mortality risk contours for men aged 50–64, labeled to show relative mortality hazards, based on data from Norwegian men. A man with weight/height on the outermost curve had almost twice the mortality risk of a man with weight/height on the innermost curve. The upward sloping lines are iso-BMI curves. Finally, the figure shows estimates of average weight and height for French men at four points in time. Assuming that the relationship between body size and mortality embodied in the Waaler surface has remained stable over time, the change in height and weight shown in this data would have contributed to a significant reduction in mortality. [Fogel \(1997\)](#) shows that changes in height and weight alone explain 90% of the reduction in French crude death rates between 1785 and 1870 and a further 50% of the reduction between 1870 and 1975.

The Fogel/McKeown view that living standards played a major role in improving health has attracted a good deal of criticism. One important argument against the view that nutrition is of paramount importance is the shifting upward of the Preston curve, as discussed above. In the period during which this phenomenon is observed, the vast majority of improvement in life expectancy is due to such shifts, rather than to movements of countries along a fixed curve as income rises. Going further, [Soares \(2007\)](#) shows that between 1940 and 1970, life expectancy rose, holding not only income but also average daily calorie consumption constant. However, the evidence on the Preston curve comes only from the 20th century (particularly after 1930), while much of Fogel's argument applies to an earlier period. Looking at this earlier period, [Smith \(2013\)](#) argues that changes in mortality in the 18th and 19th centuries were well synchronized among countries at different levels of economic development within Europe and North America, and similarly synchronized among different parts of the social spectrum within England. He interprets this finding as evidence that income cannot have played an important role, instead attributing the mortality changes to variation over time in the epidemiological environment.

Another line of argument against the Fogel/McKeown view is that the bigger bodies we observe (and thus the movement over the Waaler surface) do not result solely from a better standard of living (i.e. nutrition). Infection both increases the body's need for nutrition and interferes with the absorption of nutrients from food consumed. Thus, the increase in height and BMI observed historically is not necessarily due solely to more food intake, but may also have resulted from decreased rates of infection (due in the first instance to improved public health, and later to antibiotics).

3.4.1.2 Negative Effects of Economic Growth on Health

Although there is debate about the fraction of increased health that can be attributed to better nutrition, there is little doubt that until recently, with the rise of obesity, diabetes, and other diseases of overconsumption, better nutrition has been a net contributor to better health. However, other behaviors associated with economic growth worked in the opposite direction. Most significant among these has been urbanization. Historically, cities were notably unhealthy, both because they put people into contact with many other potential disease carriers, and because in a large population the risk of contamination of food and water by human waste is greatly increased. For example, in 1900 in the United States, the rural-urban gap in life expectancy among white males was 10 years (54 vs. 44 years). Cities were particularly hard on children. Mortality at ages 1–4 was twice as high in cities as in the countryside. The urban penalty in US mortality disappeared in the early decades of the 20th century ([Haines, 2001](#)). In currently developing countries, the dynamics of the rural-urban mortality gap have been different, evidently because superior access to medical care more than made up for the inherent unhealthiness of cities. In South Asia (with the exception of Sri Lanka), infant mortality has been lower in cities than

rural areas since the 1970s, with the gap narrowing over time. For example, India's infant mortality rate over the period 1978–1983 was 68 for urban and 111 for rural areas while during the period 1994–1999 the values were 47 and 73, respectively. In sub-Saharan Africa the picture is more mixed, although on average cities also have lower mortality²⁰

A second channel through which growth can negatively affect health is pollution. In the presence of an environmental Kuznets curve, growth in a poor country will worsen pollution. The experience of China over the last several decades is a case in point.

Finally, economic growth may not automatically produce health improvements because the extra consumption spending afforded by rising incomes is not always directed in a manner that improves health outcomes. Consumption of tobacco is an obvious example. Further, the translation from higher income to better nutrition is not automatic. [Deaton and Dreze \(2009\)](#) note that although the Indian economy has been growing rapidly since the 1980s, average intake of calories, protein, and other nutrients has declined. Although in cross-section there is a strong, positive relationship between household expenditure and household calorie intake, the intercept of this Engel curve has been shifting down over time. Part of the decline may have been due to reduced calorie demands from infectious disease, physical labor, and fertility, all of which were reduced over this period. However, indicators of nutrition such as children's weight-for-age and height have improved only modestly over this period, and undernutrition remains widespread. Deaton and Dreze suggest that one explanatory factor may have been changes in preferences away from coarse grains and in the direction of more processed foods, due to advertising or emulation of the more affluent classes. See [Easterly \(1999\)](#) for a more general intellectual history of the idea that income growth does not translate into health improvements.

3.4.1.3 Econometric Evidence

[Cutler et al. \(2006\)](#) point out that almost all of China's remarkable improvement in infant mortality took place before economic growth took off in 1980, and similarly that the acceleration in economic growth in India following economic reforms in the early 1990s was accompanied by a slowdown in the rate of decline in infant mortality. Similarly, in Bolivia, Honduras, and Nicaragua, gains in life expectancy on the order of 20 years took place during periods of modest or even negative income growth ([Soares, 2007](#)).

[Caldwell \(1986\)](#), looking cross-sectionally at the relationship between income and health outcomes, focuses on the outliers, that is, countries with unusually good or bad health outcomes relative to their income levels. Among the poor health achievers he notes that most have large Muslim populations (leading to limited female autonomy and schooling). He attributes health differences beyond those explained by income to schooling, local health service provision, and possibly family planning (as well as being

²⁰ Data from Demographic and Health Survey summary reports (various issues) as well as [Sahn and Stifel \(2003\)](#).

a former British colony). Studying episodes of “mortality breakthroughs” such as Sri Lanka, where life expectancy rose by 12 years between 1946 and 1953, his conclusion is that such episodes are more a matter of the political and social will to address health issues than the availability of economic resources.

In terms of the framework described above, such examples of large changes in health in the absence of shifts in income are evidence of there being a large variance of shocks to the $\nu(y)$ curve. Further, to the extent that these mechanisms are at work, there is no need for the $\nu(y)$ curve to have a positive slope in order to explain the observed correlation between health and income. At the same time, there is nothing inconsistent with viewing the $\nu(y)$ curve as having both a lot of variance and a positive slope.

Attempts to estimate the structural effect of income on health, that is, the slope of the $\nu(y)$ curve, run into obvious identification issues. They also suffer from the difficulty that feasibly identified estimates may only pick up a short-run effect. Easterly (1999) uses cross-country data for the period 1960–1990 on income per capita and a number of health indicators. In decadal data, income growth is linked to *lower* life expectancy, while the relationship between income growth and infant mortality has the expected sign. Income growth is also positively related, though only sometimes statistically significant, with observable inputs into health such as calorie intake, physicians per capita, and access to clean drinking water. Of course, all of these correlations are not well identified. In an attempt to achieve identification, Easterly estimates IV regressions (in changes), using twice-lagged income as well as “policy” measures (black market premium, financial depth, and inflation) as instruments. Here, he finds mixed results with income growth significantly reducing infant mortality but having no significant effect on life expectancy. He concludes that there are “long and variable lags” in the translation of higher income growth into better health. This is also consistent with the observation made above that many of the outliers in the cross-sectional income–growth relationship are countries where income has recently grown very quickly but health has not improved.

Unfortunately, the identification in both Easterly (1999) and Pritchett and Summers (1996) papers is far from perfect. The policy measures used in both papers may easily be correlated with the types of effective institutions that affect health through channels other than income. Combined with the fact that such approaches are really only suited to looking at short-run effects of income changes on health, one is left with little hope of learning much about the slope of the $\nu(y)$ curve through this approach.

3.4.2 Public Health, Medicine, and Economic Growth

As mentioned above, to the extent that the improvements in health are not direct results of economic growth, via changes in nutrition and other aspects of the standard of living, then such improvements are due to two other channels: improved public health and direct application of medicine. A natural question is to what extent these forces are, in turn, linked to economic growth.

Cutler et al. (2006) organize their narrative of the sources of reduced mortality around the themes of knowledge, science, and technology. Knowledge of the causes of ill health, most importantly the germ theory of disease (empirically validated in the 1880s and beginning to displace previous theories around the turn of the century), allowed for the introduction of effective public health infrastructure, particularly clean water. Accumulation and dissemination of knowledge also allowed for improvements in private health behaviors, ranging from washing hands and boiling water to the reduction in smoking in the United States over the last half century. And of course, new science and technology have been driving forces in medical improvements since the middle of the 20th century. This focus on the role of knowledge has the implication that the explanation for the time series relationship between income and health, on the one hand, is different than the explanation for the cross-country or within-country relationships between these same variables, on the other. The reason that the explanations differ is that at any point in time, at least in the world today, gaps between or within countries in applicable health knowledge must be very small. Cutler et al. (2006) seem to view the cross-country relationship between health and income as resulting from correlated shocks to the $\nu(y)$ and $\gamma(\nu)$ curves, particularly in the form of differences among countries in the quality of institutions that impact both income and health. For an explanation of the within-country correlation of health and income, they focus on both causality running from health to income, in particular the effects of disability on earnings, and on the role of education in raising wages and allowing for better application of existing health knowledge.

Soares (2007) similarly stresses the diffusion of ideas (new technologies, personal health practices, and public goods) from rich to poor countries as the driving force shifting the Preston curve upward in the post-war period. However, unlike the diffusion of ideas used in producing output more generally, the ideas that are relevant for health often have significant dimensions of public goods (such as sanitation and clean water), externalities (quarantine, vaccination), or principal-agent problems. Even private health practices that are not reliant on public infrastructure, such as hand-washing, often require public information campaigns to put in place the relevant information. For these reasons, there is a strong complementarity between health ideas and the strength of institutions, particularly government. There is similarly a strong complementarity between health ideas and human capital of those in a position to apply them. Preston and Haines (1991) find that in the late 19th century, prior to the widespread acceptance of the germ theory of disease, the children of doctors and teachers had only slightly lower mortality rates than average. By 1925, such children had mortality rates that were one-third below average. Similarly, at the time of the Surgeon General's report on the health hazards of smoking, there was little variation in smoking rates by educational group. By 1987, smoking among

male college graduates had fallen to 17%, vs. 41% for high-school dropouts (Preston, 1996).²¹

This focus on the role of knowledge, science, and technology, rather than economic growth per se, leaves open two questions. First, whether it is possible to really separate the growth of knowledge from the process of economic growth more generally. And second, whether, even with adequate knowledge, there remains a role for income in determining the application of this knowledge.

The ideas about health on which Cutler et al. focus have to be put into practice in order to impact health. Some actions can be taken at the individual level. In the historical context these have included hand-washing, boiling milk, appropriate food storage, and breastfeeding. In currently developing countries, other examples of actions that are taken at the individual level and rely on individuals understanding and valuing their health benefits are the use of chlorine drops in water, sleeping under a bed net, and use of condoms to prevent transmission of HIV. However, much of the benefit from improved knowledge required public action, ranging from quarantine of the sick to food inspection and regulation to construction of public works.

Public health measures, broadly viewed, were probably responsible for most of the improvement in health in the later 19th and early 20th centuries. In the United States, the infant mortality rate declined from 229 per thousand births in 1850 to 69 per thousand in 1930. Cutler and Miller (2005) use a difference-in-difference approach to estimate the effects of water filtration and chlorination on mortality in a sample of 13 large US cities. Their finding is that these water quality improvements reduced mortality by 13% over the period 1900–1936, which was 43% of the total decline in mortality over these years. In addition to clean water, the introduction of refrigeration (especially for milk) played an important role.

Cutler et al. (2006) are certainly correct that from the perspective of a single developing country, economic growth can be considered to be divorced from the advancement of medical knowledge. This same argument is less germane when one considers the *application* of knowledge, either in the form of medical treatments or public health measures. Trained medical personnel, equipment for treatment, and public health infrastructure all cost money. It is certainly true that there are cases where efficiently organized medical establishments have produced great leaps in health using relatively few resources. Cuba has long served as a model of medical care, even as its economy has collapsed. Similarly, in China, massive advances in life expectancy took place prior to economic liberalization (and for this reason, data for 1980 shows China as a notable outlier above the Preston Curve). But these examples do not disprove the claim that economic growth, by allowing

²¹ The United States in the late 19th and early 20th centuries is often the subject of studies on the causes of health improvements. However, it is worth noting that the McKeown/Fogel theory is at a severe disadvantage in this context, because the country had unusually good nutrition. Consumption per adult equivalent was 3700 calories in 1900 (Preston (1996)).

for increased spending on public health and medical treatment, will usually pay at least some dividend in terms of health outcomes.

Writing of long-term improvements in health, [Cutler et al. \(2006\)](#) say “Perhaps controversially, we tend to downplay the role of income. Over the broad sweep of history, improvements in health and income are both the consequence of new ideas and new technology, and one might or might not cause the other.” Such a view only makes sense if we imagine that scientific and technological progress of the type that has taken place since the Industrial Revolution as being possible in a context in which economic output did not grow—or did not grow at the speed actually observed. In other words, we are to imagine that science could have advanced from its level of 1860 (when the germ theory of disease was developed) until 1960s (beta blockers) in the absence of the massive increases in both income per capita and total output observed in the richest countries during this period. Such a scenario is hard to swallow, for several reasons. First, in standard models of technological progress such as the Schumpeterian model of [Aghion and Howitt \(1992\)](#), effort devoted to R&D is a function of the size of market. In the absence of income growth, incentives for R&D would have been much smaller. Second, as stressed by [Jones \(1995\)](#), maintaining a relatively constant pace of technological progress over the last century has required vastly increased resources to be devoted to R&D. The science that produced the germ theory was low budget. More recent medical advances have required enormous spending. The channeling of such resources (through both the public and private sectors) would have been inconceivable in the absence of robust income growth.

The statement of [Cutler et al. \(2006\)](#) that economic growth was not essential for observed health improvements in the most advanced countries is probably meant to be more rhetorical than serious. Underlying it, however, are two serious observations. The first is that, over periods shorter than the “very long run,” there indeed may be very little relationship between income growth and health improvement. Even in the most advanced countries, the stock of usable but non-applied health knowledge is so large that many decades of health improvement could take place without any new discoveries being made. Second, when one considers developing countries, the assumption that income growth will automatically lead to health improvement is unwarranted; and the assumption that income growth is the best way to achieve health improvement is even more unwarranted. As [Deaton \(2006\)](#) writes, “Economic growth frequently needs help to guarantee an improvement in population health.”



3.5. IMPACT OF HEALTH ON ECONOMIC GROWTH

3.5.1 Direct Productivity Effects

The simplest channel of causality running from health to economic growth is via the productivity of workers. Individuals who are healthier are able to work more effectively,

both physically and mentally. Further, adults who were healthier as children will have acquired more human capital in the form of education. [Weil \(2007\)](#) refers to this as the “proximate effect” of health on the level of income.

To pursue this issue, I start with a simple production framework in which health is explicitly incorporated. Assume that aggregate output is given by a Cobb-Douglas production function taking as its arguments physical capital and a composite labor input,

$$Y_i = A_i K_i^\alpha H_i^{1-\alpha}, \quad (3.1)$$

where Y is output, K is physical capital, A is a measure of productivity, and i indexes countries. The labor composite is in turn composed of raw labor L , average human capital in the form of education h , and average human capital in the form of health v :

$$H_i = h_i v_i L_i \quad (3.2)$$

A setup like this has been used in the development accounting literature to assess the contributions of productivity, physical capital, and human capital in the form of education to variation in income among countries (see [Caselli \(2005\)](#) for a review). To implement such a calculation, one has to be able to measure the average level of human capital in the form of education at the country level. The approach taken in the literature has been to combine data on the average number of years of education among adults with an estimate of the return to education (Mincer coefficient) that converts years of education into a measure of human capital. The rate of return estimate plays a key role here, because the units in which the data are measured (years of schooling) are not proportional to the amount of human capital. For example, using a standard estimate of 10% per year of schooling, a person with 4 years of schooling does not have twice as much human capital as someone with 2 years, but rather only 1.21 times as much.

To proceed analogously in the case of health, we need a consistent measure of health across countries and a measure of “return to health” that can be used to convert such a measure into units of human capital in the form of health. Compared to the case of human capital in the form of education, there are two additional complications. First, unlike human capital in the form of education, where years of schooling seems like a reasonable summary measure, health has many different dimensions that might be relevant for productivity. Second, in the case of health there is not as long a tradition of measuring rates of return as there is in the case of education.

3.5.1.1 *Estimates of the Return to Health Characteristics*

Define w_i as the wage of the labor composite in country i (this could be its marginal product, although this is not necessary). The wage of worker j will depend on his own health and education, as well as this aggregate wage:

$$\ln(w_{i,j}) = \ln(w_i) + \ln(h_{i,j}) + \ln(v_{i,j}) + \eta_{i,j}, \quad (3.3)$$

where the last term is an individual-specific error.

As a simplification, I take the approach that the many different aspects of health that we observe (height, survival, etc.) are all functions of a single, underlying (latent) health status which is scalar. This is obviously an extreme approach—Weil (2007) discusses some of the biases that it introduces. Modeling underlying health as being a scalar does not mean that individual aspects of health will all move together, however. Instead, I allow for outcome-specific shocks at the individual level that can reflect luck, genetics, and so on. For example, consider the relationship between underlying health (z_j) and height:

$$\text{height}_j = \text{constant} + \gamma_{\text{height}} z_j + \epsilon_{\text{height},j}. \quad (3.4)$$

Latent health is never observed directly, so the coefficient that relates health to height is not observable either. However, the assumption of latent health being scalar allows for the calculation of a useful measure of health's impact. Assume that the relationship between latent health and v (the aspect of health that determines wages) is determined by a similar equation (the use of log here follows the existing literature).

$$\ln(v_j) = \text{constant} + \gamma_v z_j + \epsilon_{v,j}. \quad (3.5)$$

The ratio of the coefficients $\gamma_v / \gamma_{\text{height}}$ is defined as the “return to height.” It tells by how much a change in underlying health that raises height by one unit will raise log wages. The return to height is not the same as the observed relationship between height and wages, both because observed height contains a component that is unrelated to underlying health ($\epsilon_{\text{height},j}$), and because in general latent health (and thus height) will be correlated with other factors that affect wages.

Calculations of the return to health characteristics like this can be done for any health outcome. They will be most informative, however, to the extent that the health outcome is representative of the totality of health—in other words, in cases where the assumption of latent health being scalar does the least violence to reality. It is for this reason that I focus on height, which is often viewed as a useful summary measure of nutrition and health insults through early adulthood.

The question then becomes, how to estimate the return to height? Simply regressing log wages on height is clearly problematic. People with higher incomes can afford better health inputs, and unobserved characteristics (such as being from a wealthy family) may affect both income and height. A series of papers has estimated the return to height using a IV approach (see Schultz (2002), Ribero and Nunez (2000)). Data are from Ghana, Brazil, and Mexico, and the instruments are inputs into health in childhood such as distance to local health facilities and the relative price of food in the worker's area of origin. Education is included as a control. The estimated return to height ranges from 7.8% to 9.4% per centimeter.²² Unfortunately, the instruments used in these analyses

²² Knaul (2000) does a similar analysis using age at Menarche as a measure of health, while Schultz (2005) presents IV regressions in which health is measured by both height and BMI.

have potential problems. To the extent that good inputs into child health reflect family characteristics that also lead to high wages, these estimates of the return to health will be upwardly biased.

As an alternative, I identify the return to height using exogenous variation in uterine nutrition among monozygotic twins, taking advantage of estimates from [Behrman and Rosenzweig \(2004\)](#). Within pairs of monozygotic twins there are significant variations in birth weight, reflecting differences in intrauterine nutrition due to the location of fetuses within the womb. In their sample, which covers female monozygotic twins from the United States, the average absolute gap in birth weight is 10.5 oz, compared to a mean birth weight of 90.2 oz. Behrman and Rosenzweig regress within-pair difference in log wages, adult height, and education on the difference in fetal growth (measured in ounces per week of gestation). Their estimate is that a one-unit difference in fetal growth leads to a gap of 0.190 (standard error of 0.077) in log wages, 3.76 (0.43) centimeters in adult height, and 0.657 (0.211) years of schooling. Dividing the estimated effect of fetal growth on log wages by the estimated effect of fetal growth on height gives a TSLS estimate of the return to height of 5.1% per centimeter. This return includes the effect of improved health in raising education. Making an adjustment to eliminate this channel (see [Weil \(2007\)](#) for details) yields an estimated effect of health as proxied by height on wages, holding education constant, of 3.3% per centimeter. A similar calculation using data on Norwegian twins from [Black et al. \(2007\)](#) yields an estimate of the same effect of 3.5% per centimeter. In the calculations that follow, I use the average of these two estimates—3.4% per centimeter.

This estimate of the return to height can be applied to the historical data discussed above. In the typical developed country, the rise in adult height over the last 200 years has been roughly 10 cm. My estimate of the return to height thus implies that labor input per worker went up by a factor of 1.4 (in the steady state of a standard growth model, this will also be the effect on output per worker). Thus, while higher labor productivity due to health has been an important factor, it is certainly not nearly the dominant factor in income growth. To put some quantitative flesh on this statement, consider a country in which income has risen by a factor of 15 over this period. The fraction of this rise due to improved labor productivity from better health can be calculated as $\ln(1.4)/\ln(15)$, which comes to 12.4%.

One important benchmark against which to compare my estimate of the increase in labor input over time comes from [Fogel \(1997\)](#). Looking at data on the distributions of caloric intake and basal metabolic needs in the UK over the period 1780–1980, he calculates that improved nutrition raised labor input per working age adult by a factor of 1.96.

3.5.1.2 Health's Overall Contribution of Cross-Country Income Variance

In addition to asking how health has contributed to growth over time, we would like to ask how much of cross-country variation in income it explains. However, there are two obstacles to using the estimate of the return to height just derived for this purpose.

First, there do not exist consistent cross-country data on adult height. Second, one might worry that genetic factors affecting height but not health vary across countries. For these reasons, [Weil \(2007\)](#) creates a mapping from changes over time in height to changes over time in the adult survival rate (ASR), using the data on both variables presented in [Figure 3.6](#). His estimate is that a change in the ASR of 0.1 is associated with a change in height of 1.92 cm. This implies a “return to ASR” of 0.653, which in turn says that an increase in ASR by 0.1 will raise labor input per worker by 6.7%. In cross-country data, ASR ranges from 0.214 (Botswana) to 0.904 (Iceland). The implied increase in labor input per worker moving over this range would be a factor of 1.59.

Using this estimate, Weil then asks how much of the variance in cross-country income can be explained by health. Following [Caselli \(2005\)](#), variance in log output per worker is decomposed into pieces attributable to physical capital, human capital in the form of education, human capital in the form of health, and a productivity residual. The variance in log output per worker is equal to the sum of the variances of these component terms, along with a full set of covariances. One can then calculate the reduction in the variance of the log of output per worker that would result from eliminating health gaps among countries; this is just the variance in $\ln(\nu)$ plus all of the covariance terms that involve ν . Setting these to zero reduces the variance of log output per worker by 9.9%. As an additional measure, Weil calculates the contribution of health to the 90/10 income ratio. In the raw data, the ratio is 20.5. Eliminating health gaps, the ratio would fall to 17.9, with the large majority of that reduction coming from a fall in the 50/10 income ratio.

These results say that health is a significant contributor to cross-country income differences, but that it is not of overwhelming importance. For comparison, the effect of health estimated here is a little more than one-third as large as the contribution of human capital in the form of education to cross-country income variance. It is also of interest to note that the fraction of cross-sectional income variance explained by health (9.9%) is quite similar to the back-the-envelope calculation in the last section of the fraction of long-term income growth explained by health (12.4%).

3.5.2 Other Channels

The analysis in [Section 3.5.1](#) focuses on worker productivity. However, there are several other channels by which changes in health may impact economic growth.

3.5.2.1 Longevity and Human Capital Accumulation

The idea that reducing mortality will raise the return on human capital investments, and thus the level of schooling, has a long pedigree in economics. Discussions of the literature can be found in [Kalemli-Ozcan et al. \(2000\)](#) and [Hazan \(2009\)](#); the latter traces the mechanism to [Ben-Porath \(1967\)](#).

To assess the potential size of this effect, I consider a simple model in which individual earnings are proportional to human capital h , which is in turn a function of years of

schooling: $h = f(s)$. I abstract for trend growth in wages and the method by which schooling is financed as well as the risk associated with mortality, and simply assume that schooling is chosen to maximize the expected present discounted value of lifetime earnings. Further, for simplicity, I assume that the only cost of schooling is the opportunity cost of foregone wages. The value of s is chosen to maximize:

$$\int_s^\infty S(a)f(s)e^{-ra} da, \quad (3.6)$$

where $S(a)$ is the probability of survival to age a . For the $f(s)$ function, I use the specification from [Bils and Klenow \(2000\)](#):

$$f(s) = \frac{\Theta}{1 - \Psi} s^{1-\Psi}.$$

Based on cross-country data on the Mincerian return to schooling, they estimate values of $\Theta = 0.32$ and $\Psi = 0.58$. I take age zero (the first age at which schooling is possible) to be five. To match the example studied by Hazan, I start by using data on survival (the $S(a)$ function) from age five for the cohort of males born in the United States in 1850, when life expectancy at age 5 was 52.5 years.²³ Hazan reports that this cohort received an average of 8.7 years of schooling. I choose the real interest rate so that optimal schooling matches this value.²⁴

To assess the effect of declining mortality, I hold the other parameters constant and change the $S(a)$ function to match that of the cohort born in 1930, for which life expectancy at age five was 66.7 years. Optimal schooling rises to 9.6 years. In fact, average years of schooling for this cohort was 13.3. Thus, the pure mortality effect explains roughly one-fifth of the actual increase in schooling that took place over this period. This exercise shows that reduced mortality over the range found in cross-country or historical data should have some effect on schooling, but that we would not expect it to be the dominant explanation.

Some empirical evidence also supports the model of decreasing mortality raising schooling. Of course, estimation of the effect is made difficult by the fact that mortality is correlated with other determinants of schooling, and is itself endogenous. The solution is to look for cases in which there is plausibly exogenous and sharp variation in mortality. [Oster et al. \(forthcoming\)](#) examine the effect in US data on individuals at risk for Huntington Disease, which onsets during adulthood, reducing life expectancy by roughly 20 years and healthy life expectancy by 35 years. Individuals with one parent who suffered from the disease have a 50% chance of developing it themselves. They can find out their fate either by taking a genetic test or with the appearance of early symptoms.

²³ I am grateful to Moshe Hazan for sharing this data.

²⁴ The implied value of r is 8.7%, which might be viewed as high. However, given both that human capital investment carries risk, and that the discount rate applied may reflect credit market imperfections, I don't think of this value as unreasonable.

Oster et al. find that the information that he/she will suffer from Huntington Disease lowers the probability of an individual's completing college by 30–33% points. Their estimate, extrapolated to cross-country data, implies that differences in mortality explain about 10% of the observed variation in college enrollment. Consistent with the calculation above, they conclude that the time-horizon effect exists as predicted by economic theory, but that it is not the major determinant of schooling variation. [Jayachandran and Lleras-Muney \(2009\)](#) examine the effects of a rapid reduction in maternal mortality in Sri Lanka over the period 1946–1953, which raised female life expectancy at age 15 (censored at 65) by 1.5 years, or 4%. Their estimates, based on regional variation in maternal mortality as well as male–female differences, are that every extra year of life expectancy raised literacy by 0.7% points and education by 0.11 years. Once again, the 0.17 years of increased female schooling due to mortality reduction is small compared to the total increase of 1.5 years comparing women in the treated and untreated cohorts.

The most trenchant critique of the view that time horizon influences schooling has come from [Hazan \(2009\)](#). He argues that the essence of the Ben-Porath mechanism is that an increase in survival that induces a rise in schooling must also induce a rise in lifetime labor supply. In his paper, Hazan measures expected total working hours (ETWH) over the lifetime for cohorts of American men born between 1850 and 1970. In addition to mortality, ETWH is affected by labor supply along both the extensive margin (working or not working) and the intensive margin (hours per week). He shows that declines in weekly hours, along with earlier retirement, have more than offset the decline in mortality. For example, ETWH at age 20 fell from 112,199 for men born in 1850 to 81,411 for men born in 1930.

Hazan's observation that ETWH has fallen over time is indeed well taken, but it is worth noting that the paper does not actually show that changing mortality did not affect schooling. Rather, it shows that even though falling mortality worked to increase ETWH, other factors more than undid this effect. We can still believe that the Ben-Porath mechanism works, which is to say that ETWH positively affects schooling. In that case, some other factors must have raised schooling even though the effect of ETWH would be to reduce it. Thus, if mortality had not fallen, ETWH would have fallen more than what we observe, and schooling would have risen less. Even if one knew with certainty that the effect of mortality on schooling took the form described above, it would have to be the case that other factors also affected schooling. Hazan shows that the net effect of lifetime hours on schooling should have been negative, because the working week and retirement age have fallen. However, if mortality had not fallen as well, the decline in ETWH would have been larger, and so schooling would have risen less.²⁵ Another critique of Hazan is that, as pointed out by [Cervellati and Sunde \(forthcoming\)](#), reduced

²⁵ Lonstrup (2013) points out that even in the framework of Hazan, there is an important interaction by which increased longevity raises the impact of other factors, such as the return to human capital, on optimal years of schooling.

labor supply on the intensive margin (i.e. fewer hours worked per year) decreases the opportunity cost of schooling as well as the benefits to additional years of education.

3.5.2.2 *Mortality, Fertility, and Human Capital Investment*

In the model of Soares (2005) reductions in both child and adult mortality lead parents to increase investment in their children's human capital (beyond a zero level that holds in Malthusian equilibrium) and to lower fertility. The reduction in fertility goes beyond the amount that would be induced by lower mortality, if parents were aiming to hold the expected number of survivors fixed. Somewhat similarly, in the model of Kalemli-Ozcan (2002), reduced mortality, by reducing variance of realized survival outcomes, allows parents to reduce their precautionary child-bearing, and thus the average number of surviving children, and this in turn allows for higher human capital investment.²⁶ However, Hazan and Zoabi (2006) argue that the effect of longevity on human capital investment is not clear in the presence of quality-quantity tradeoffs because increased longevity positively affects quantity as well as quality.

3.5.2.3 *Other Theoretical Channels*

Lorentzen et al. (2008) stress a set of effects of short time horizons due to high mortality that go beyond investment in human capital. Specifically, they see high mortality as negatively affecting physical capital accumulation (because individuals see lower probability of using their savings) as well as raising fertility. "The prospect of early death," they write "brings shortsighted behavior." This can include not only failure to put aside resources for the future, but risky activities such as unprotected sex and smoking that further raise the hazard of mortality. (Consistent with this view, Oster (2012) finds that reductions in risky sexual behavior in response to the HIV epidemic in Africa were smallest in areas with high non-HIV mortality.)

Bloom et al. (2003) show in the context of a life cycle model that increased longevity will raise saving rates at every age, even allowing for endogenous changes in retirement age. This in turn will raise capital accumulation and output. In their empirical work they find that higher life expectancy raises national saving rates, controlling for the age structure of the population.

Change in health is also related to population aging. Although the largest contribution to aging in developed countries is the decline in fertility that has taken place over the last half-century or more, a secondary contributor has been the decline in mortality at older ages. Population aging, in turn, puts great strain on government transfer schemes, potentially leading to tax increases that will sharply reduce growth (see Weil, 2008 for a review). Of course, lower old-age mortality has been accompanied by lower morbidity,

²⁶ Lorentzen et al. also stress the importance of adult mortality for the net rate of reproduction: since deaths beyond childhood are nearly impossible for a parent to "replace," mortality in this period should lead to precautionary childbearing and a higher expected number of survivors.

and so the economic problems due to this aspect of population aging are relatively easy to fix by increases in the retirement age (the same cannot be said for population aging due to lower fertility in the past). For political reasons, retirement ages do not seem to rise as quickly as would be warranted by better health of the elderly. [Kalemli-Ozcan and Weil \(2010\)](#) present a model in which falling uncertainty about mortality in old age can actually lower retirement ages.

3.5.3 Econometric Analyses of Health's Effect on Economic Growth

Given the numerous theoretical channels by which health improvements can affect economic growth, one strategy is to look at the reduced-form effect of actual health differences (or improvements over time) on growth. Of course, the endogeneity of health, and the possibility of omitted factors that affect both health and growth mean that any econometric approach must carefully deal with the issue of identification.

[Gallup and Sachs \(2001\)](#) set the pattern for much of the literature that was to follow, putting disease in the framework of a standard growth regression. In their framework, the steady-state level of income per capita in a country is determined by the level of disease as well as some other covariates X :

$$\ln(y_{ss,i}) = \beta_0 + \beta_1 \text{disease}_i + \sum_j \beta_j X_{j,i}. \quad (3.7)$$

Following [Mankiw et al. \(1992\)](#), the growth rate of income per capita is taken to be a function of the gap between the current level of income and the steady state:

$$\frac{\dot{y}}{y} = \lambda(\ln(y_{ss,i}) - \ln(y_i)). \quad (3.8)$$

Substituting (3.7) into (3.8) gives an equation relating growth with the current level of income and the determinants of the steady state. This allows for the interpretation of the parameters in a standard growth regression of the form:

$$\text{growth}_i = \gamma_0 + \gamma_1 \text{disease}_i + \gamma_2 \ln(y_i) + \sum_j \gamma_j X_{j,i} + \epsilon_i. \quad (3.9)$$

Specifically, $\gamma_1 = \lambda\beta_1$ and $\gamma_2 = -\lambda$.

The disease measure that [Gallup and Sachs \(2001\)](#) use is the fraction of the population at risk for falciparum malaria. The dependent variable is income growth between 1965 and 1990, while the controls include measures of geography, institutions, schooling, and life expectancy (to control for other diseases). Their estimated value of γ_1 is -1.3 , leading to their widely cited conclusion that eliminating malaria in a country where it was endemic (index of 1.0) would raise growth by 1.3% per year. Another way to interpret the Gallup-Sachs finding is to look at the implied effect of malaria on the steady-state level of income per capita. Their estimate of γ_2 is -2.6 . Dividing γ_1 by γ_2 and reversing

the sign gives an estimate β_1 , the effect of the disease on steady-state output per capita. In this case, the value is -0.5 , implying that going from a malaria index of one to an index of zero would raise steady-state output by 65%.

Threats to identification in the Gallup-Sachs approach arise if malaria is endogenous and/or if the disease environment that generates malaria is correlated with geographic factors that independently affect output (and are not properly controlled for). The former problem can be dealt with by instrumenting for malaria prevalence with a measure of “malaria ecology” created by Kiszewski et al. (2004). This measure is based on the biological characteristics of vector mosquitoes as well as climate data on a 0.5-degree grid.²⁷ Sachs (2003) runs regressions in which the dependent variable is the log of GDP per capita. He controls for the quality of institutions but not for schooling or life expectancy (in order to measure the overall effect of malaria). The coefficient on malaria, instrumented with malaria ecology, is close to one, implying that in high prevalence regions, eliminating the disease would raise output per capita by a factor of 2.7.

While the above papers focus on malaria, Bloom et al. (2004) use a more general measure of health, specifically life expectancy. Using lagged values of the endogenous variables as instruments, they estimate that an increase in life expectancy by one year raises steady-state output per capita by 4%. (The paper controls for accumulation of physical capital and human capital in the form of schooling, and so any health effect that runs through these channels is not included in the estimated effect.) Bloom et al. (2004) also summarize the results of 13 other studies that run similar regressions of GDP per capita or productivity on life expectancy, which get broadly similar results. Unfortunately, the use of lagged dependent variables as instruments is very questionable in this case.

Lorentzen et al. (2008) follow a IV approach similar to Sachs (2003), but using health measures that go beyond malaria. They include both infant mortality and adult mortality on the right-hand side of a regression in which the growth rate of income per capita from 1960 to 2000 is the dependent variable, also including a relatively standard set of controls for institutions. The instruments are measures of climate and geography, along with the malaria ecology measure. Their IV estimates of the effect of mortality are extremely large—even larger than the OLS relationship between mortality and income.²⁸ For example, moving from the values from India (infant mortality of 0.108, adult mortality of 0.294) to the values for the United States (0.015 and 0.197) implies that steady-state income would rise by a factor of 13.1—an enormous amount. In terms of the framework discussed above, they view the slope of the $\gamma(\nu)$ curve as large. While

²⁷ Alsan (2012) creates a similar index of suitability for TseTse fly, and finds that this predicts patterns of pre-modern development in Africa.

²⁸ I focus on the coefficients in column (1) of Table 7 of their paper. These are -6.699 on adult mortality, -20.299 on infant mortality, and -0.985 on the log of initial income per capita.

the specification “passes” an overidentifying test, there is good reason to be suspicious that climate and geography cannot in fact be excluded from the second stage of their regression.

3.5.3.1 Using Changes in Health as Instruments

The studies discussed above rely on cross-sectional variation in the health environment in order to identify the effect of health on income. This approach suffers from the inevitable problem that determinants of health are correlated with aspects of geography and climate that may have independent effects on income, and which are difficult to properly control for. The other way to achieve identification of the effect of health is to look in the time domain, in particular to examine rapid changes in health status that differentially affect different groups or regions. The papers by [Bleakley \(2010\)](#), [Cutler et al. \(2010\)](#), and [Lucas \(2010\)](#) discussed above all used this approach in studying the effect of malaria on human capital accumulation. In order to estimate the effect on GDP at the national level, rather than in data on individuals, the health shocks examined must be large enough to produce an effect that can be distinguished from background noise. Two papers have taken this approach.

[Ahuja et al. \(2007\)](#) examine the economic impact of HIV/AIDS in sub-Saharan Africa. The disease has reduced life expectancy by up to two decades in a number of countries. Because it is primarily productive adults who are dying, one would expect the economic impact of the disease to be particularly strong. As an instrument for the spread of HIV, Ahuja et al. use variation in the male circumcision rate, which differs significantly among countries for cultural reasons. The authors show that a low circumcision rate is a good predictor of the extent to which HIV spread in the population, and that it is uncorrelated with other factors likely to have affected growth. In the second stage of their analysis, they show that HIV, as predicted by circumcision, has no effect on the level of GDP per capita, although it is correlated with a slowdown in educational gains and an increase in poverty as measured by malnutrition.

[Acemoglu and Johnson \(2007\)](#) (AJ) look at cross-country variation in health improvements during the international epidemiological transition, starting in the 1940s, in which modern health technologies were rapidly transferred to the developing world. Their analysis proceeds in three steps. They begin by looking at cross-country data on disease-specific death rates prior to the transition. They combine these data with information on the rates at which death rates from different diseases declined, based either on the dates of discovery of disease-specific treatments or worldwide declines in disease-specific mortality, in order to construct a measure of predicted mortality change for every country in their sample. This measure of predicted mortality change should not be related to the component of actual mortality change in each country that results from economic growth or institutional improvements. In the second stage, they show that the predicted change in mortality that they construct is a very good predictor of actual change in life

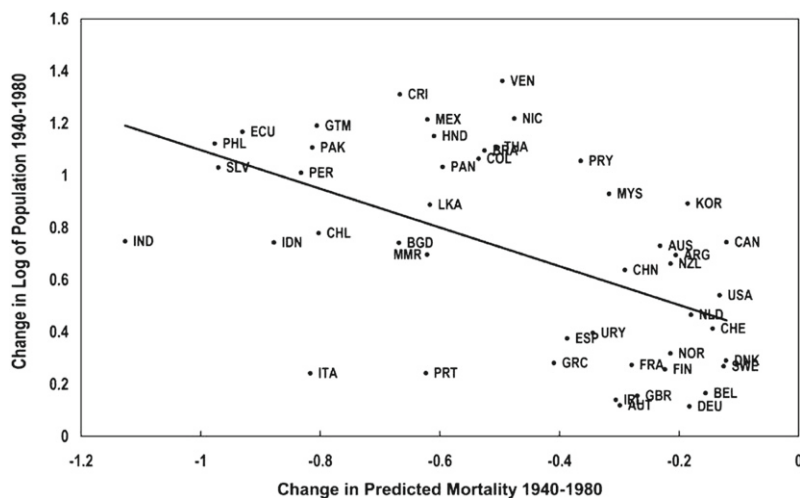


Figure 3.11 Effect of health improvements on population.

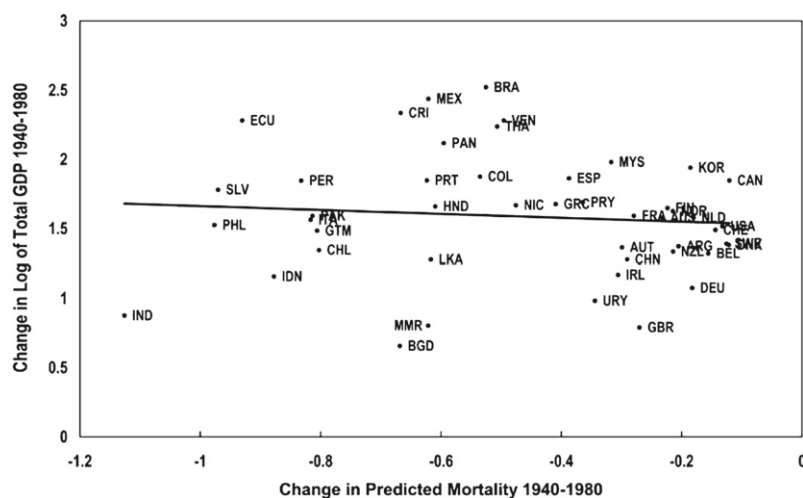


Figure 3.12 Effect of health improvements on total GDP.

expectancy over the period 1940–1980. Finally, they regress a series of outcome variables (population size, total GDP, GDP per capita, GDP per working age adult) on the change in life expectancy, instrumented with changes in predicted mortality.

Figures 3.11 and 3.12 show the key result in the paper. Figure 3.11 shows that reductions in mortality led to higher population growth. Their point estimate from a regression of log population change from 1940 to 1980 on the (instrumented) change in log life

expectancy over the same period is 1.67. By contrast, [Figure 3.12](#) shows no statistically significant relationship between the change in predicted mortality and the change in total GDP. In other words, countries with larger reductions in predicted mortality did not see total income rise faster. When AJ regress the change in log GDP per capita on log life expectancy, instrumented with predicted mortality, the coefficient is -1.32 and is statically significant. The coefficient implies that a country in which life expectancy rose from 40 to 60 would experience a 41% decline in income per capita, holding other factors constant. AJ attribute the negative effect they find to the entanglement of health with population growth discussed above: rapid declines in mortality unleashed a population explosion which through the mechanisms of Solow and Malthus reduced income per capita.

The findings of AJ are not completely comparable to those of the cross-sectional studies discussed above, since the cross-sectional studies are implicitly looking at the very long-run effects of health, while AJ are looking only over a period of 4–6 decades. Nonetheless, it is clear that AJ results are contrary to the drift in much of the cross-country literature, which finds a large, positive effect of health.

[Bloom et al. \(forthcoming\)](#) note that in the data studied by AJ, declines in mortality were not randomly distributed among countries. Rather, as a consequence of the narrowing of cross-country health gaps discussed above, the largest gains in life expectancy were in the countries where life expectancy was lowest. The correlation between initial life expectancy and the subsequent change life expectancy is -0.97 . Initial life expectancy is also correlated with subsequent growth of income per capita (correlation of 0.50). The latter correlation, say Bloom et al., is to be expected: In a model of conditional convergence such as that presented in [Section 3.5.3](#), any factor that affects steady-state income per capita will also affect growth, conditioning on initial income. They argue that life expectancy falls into this category, since there is abundant evidence that health raises individual productivity.

[Bloom et al.](#) argue that for these reasons, the initial level of health cannot be excluded from a regression in which income growth is the dependent variable. When they re-run the AJ analysis, including both initial life expectancy and initial income on the right-hand side (the latter to control for convergence dynamics), the AJ result goes away. In their reply to this critique ([Acemoglu and Johnson, 2013](#)), AJ start by pointing out that simply controlling for initial income does not make their result go away. Regarding the effect of controlling for initial life expectancy, they concur with [Bloom et al.](#) that this is very highly correlated with the change in life expectancy, and so in a mechanical sense there is no surprise that putting both on the right-hand side of a regression kills the statistical significance of the change in life expectancy. However, they argue that theory imposes limits on how large the effect of life expectancy on subsequent growth should be. When they impose these limits (either using the approach of [Ashraf et al. \(2009\)](#), discussed in the next section), they find that their result survives. They find the same thing when they control for the potential effect of initial life expectancy using a panel data approach.

My own work in this area ([Ashraf et al. 2009](#)) focuses on a different potential problem with the AJ result. Ashraf et al. question AJ's finding regarding the effect of reduced mortality on population growth. Although falling mortality should indeed increase population growth, Ashraf et al. in their simulation model, are unable to match the size of the increase that AJ find. This suggests that there is a negative correlation between life expectancy in 1940 and some unobserved factor(s) (that is, something other than the decline in mortality) that affected population growth over the period 1940–1980. As discussed above, this unexplained population growth explains about half of the difference between AJ's finding and the conclusion of [Ashraf et al. \(2009\)](#) that increases in life expectancy should have a modestly positive effect on income growth. A potential explanation for the remainder of the gap is that the same unobserved factor(s) that predicted rapid population growth in countries with low life expectancy in 1940 also predicted slow income growth (for reasons unrelated to population or health) in such countries. Countries that had low life expectancy in 1940 differed in a systematic fashion from those that had high life expectancy: they had different environments, colonial history, levels of institutional development, and demographic histories. It would not be surprising if some element in that set of characteristics had a direct effect on subsequent population or income growth.

3.5.4 Simulation Models

The reduced form estimates discussed in the previous section are one attempt to encompass all the different channels by which health affects economic growth in a single analysis. This reduced form approach is attractive precisely because there are so many different channels through which health could matter, each with its own long and variable lags. However, the difficulty of achieving identification in this context is severe.

The alternative to such reduced form regressions is to create a simulation model in which the different channels can be individually specified, based on credible microeconomic evidence. Crucially, it is easier to achieve identification of individual channels than it is to identify the reduced form effect of health. A further benefit of the simulation approach is that it allows the researcher to exploit a good deal of quantitative macroeconomic theory developed in the context of growth.²⁹

The progenitor of this type of analysis is [Young \(2005\)](#), who used a simulation model to examine the effects of HIV/AIDS on the development of the South African economy. His model combines a relatively standard aggregate production framework with a model of household optimization over fertility, labor supply, and children's education. AIDS is incorporated into the model via estimates of the fraction of the population that is HIV positive as well as transition times into illness (at which time labor input ceases) and

²⁹ [Ashraf et al. \(2013\)](#) discuss the history of the use of simulation models to address the somewhat related question of how fertility decline affects economic growth. Such models have been around for more than half a century, but they fell out of favor in the 1980s, being viewed as ad hoc and opaque.

death. The underlying parameters describing the fertility response to HIV and to female wages, returns to education, determinants of parental investment in children, and the elasticity of labor supply are estimated by Young using South African household data. His results are dominated by effects of reduced fertility, both as a collateral result of measures to prevent HIV transmission, and because of rising wages for women. Lower fertility, combined with adult mortality from AIDS, slows labor force growth, and through the Solow and Malthus channels (rising capital/labor, and land/labor ratios, respectively) raises income per capita. These effects outweigh the reduction in education that results from the high number of AIDS orphans. Young finds that income per capita in the HIV scenario is roughly 10% higher than in the non-HIV scenario in 2010 (15 years after the start of the simulation), and remains higher than the non-HIV scenario for the first 50 years of the simulation.

Ashraf et al. (2009) take a somewhat similar approach, simulating the effect of a generalized health improvement. The specific health intervention (a rise in life expectancy from 40 to 60 years) and its demographic consequences are discussed in [Section 3.3.1.3](#) above. Unlike Young, Ashraf et al. look to existing literature for estimates of the different channels relating health and income, rather than producing their own. Their model allows for several channels by which health affects output. First, there is the demographic effect of increased child survival that is discussed above. Second, there is the direct effect of improved health on worker productivity. This is calibrated in two different ways: first, using the methodology of [Weil \(2007\)](#), and second under the assumption that the Years Lost to Disability, as discussed in [Section 3.2.1.1](#), can also be used to measure the decrement to labor productivity associated with poor health. (The latter methodology yields productivity effects that are about half as large as the former). Third, the authors allow for effects of improved health on education. As in Young's paper, the model has an aggregate production function in which quality-adjusted labor is combined with physical capital (accumulated with a fixed saving rate) and land.

Ashraf et al. find a short-run effect that is consistent with Young's (as well as the findings of Acemoglu and Johnson discussed above): improving health lowers income per capita, primarily through the demographic channel of raising the ratio of dependent children to working age adults. Fifteen years into the simulation, income per capita is 5% lower than it would have been absent the health improvement. In the long run, the demographic effect is undone by endogenously falling fertility, while better health and higher education raise worker productivity, and so the effect on income reverses. Income per capita returns to its baseline level after 30 years, and in the long run is 15% higher thanks to the health improvement. While this long-run finding is in the same direction as empirical papers such as [Bloom et al. \(2004\)](#), the magnitude is far smaller. In addition to using their model to consider a general improvement in health, Ashraf et al. examine reductions in two particular diseases: malaria and tuberculosis. Again, the effects that they find are small. In the case of malaria, calibrating their model to the prevalence of the disease in Zambia,

they find that complete elimination would raise income per capita by only 2% in the long run—far below the estimates of, for example, [Gallup and Sachs \(2001\)](#).³⁰



3.6. HEALTH AS A COMPONENT OF ECONOMIC GROWTH

The above section discusses how improvements in health lead to increases in conventionally measured GDP. However, as mentioned in [Section 3.1](#), health plays an additional role of being, in itself, a measure of a country's development. This quality is not unique to health, of course. One can make a good argument that education, political freedom, gender equality, and many other social attributes are both themselves aspects of development and contributors to increases in conventionally measured income. But while health is not unique in this sense, it stands out as likely being the most important non-income component that one would want to include in a measure of economic development, for two reasons: first, the fact that individuals clearly assign very high value to a long and healthy life, and second, the large extent to which achievement of this aim varies among countries as well as historically.

The best known metric for combining measures of health and income (and education as well) into a single metric is the Human Development Index (HDI), created by Mahbub ul Haq and Amartya Sen in 1990 with the explicit goal of shifting analysis of economic development away from a focus on income per capita. The HDI is the geometric mean of three “dimension indices,” which in turn cover income, life expectancy, and education.³¹

$$HDI_i = I_{Income,i}^{1/3} \times I_{Life,i}^{1/3} \times I_{Education,i}^{1/3}.$$

Each dimension index is in turn defined as:

$$Dimension\ Index_i = \frac{actual\ value_i - minimum\ value}{maximum\ value - minimum\ value}.$$

Income is measured as the log of gross national income (GNI) and life expectancy in years. The minimum values used in both the numerator and denominator (ln(100) and 20 years) are conceived of as subsistence levels, while the maximum in each case is the highest value observed in the sample (\$87,478 and 83.6 years in 2012).

³⁰ [Gollin and Zimmermann \(2007\)](#) also construct a simulation model to study the effects of malaria. They pay particular attention to the behavioral responses of people living in malaria-endemic regions, such as sleeping under bed nets, that may limit the impact of the disease. The endogeneity of malaria prevalence leads to the possibility of multiple history-dependent steady states. Uncontrolled malaria, in the most extreme case, can reduce income per capita by up to half. A large part of the effect in their model is via asset holdings, which in turn determine the capital stock: malaria shortens lifespans and so reduces both the incentive to save and the time over which assets can build up. By contrast, the direct effect of malaria on labor productivity is small, with infected individuals losing only 10% of their labor input.

³¹ [Malik \(2013\)](#).

The HDI establishes an equivalence scale relating increases in life expectancy given by the formula to changes in income. Specifically, a rise in income by 1% (one log point) has the same effect on a country's HDI as a rise in life expectancy given by the formula:

$$\frac{(83.6 - 20)}{(\ln(87,478) - \ln(100))} \times \frac{I_{Life,i}}{I_{Income,i}} \times 0.01.$$

For example, in the case of Ghana where life expectancy in 2012 was 64.6 years and GNI was \$1684, a 1% rise in GNI would have an impact on HDI equivalent to raising life expectancy by 0.16 years. The country with the lowest implied gain in life expectancy equivalent to a 1% rise in income (0.097 years) is the United States (life expectancy of 78.8, GNI of \$43,480). At the other end of the spectrum, the country with the highest value (0.24 years) is Eritrea (life expectancy of 62.0, GNI of \$581).

3.6.1 A Utility-Based Approach

The HDI is of course somewhat arbitrary in its weighing of different components of development. Recently a number of economists have examined a more theoretically grounded approach toward synthesizing the value of gains in quality and quantity of life. Key papers in this literature include [Becker et al. \(2005\)](#), [Murphy and Topel \(2006\)](#), and [Jones and Klenow \(2010\)](#). All these papers use a similar theoretical structure, which I discuss below.

Murphy and Topel construct a framework for valuing improvements in overall longevity as well as progress against specific diseases. They demonstrate that the value of health gains is larger, the higher is lifetime income (because more utility is derived per year alive) and are also larger, the greater is the existing level of health (because it is more valuable not to die of a particular disease if you are less likely to die of something else). With a calibrated version of the model, they calculate the value of additional life years produced by health improvements in the United States for every decade in the 20th century. They call the value of these improvements “health capital.” They find that for the first half of the century, annual investment in health capital was only slightly less than conventionally measured GDP. In other words, almost half of properly measured GDP consisted of investments in health capital. By the last decades of the century, the fraction of properly measured GDP made up of such investments had fallen to roughly 20%. Even in this later period, the total value of health capital gains greatly exceeded medical expenditures.

Becker et al. employ a very similar approach, with a focus on inequality and income convergence among countries. They construct a measure of full income growth that incorporates the value of life expectancy gains in money-metric terms. Looking over the period 1960–2000, they find that in the poorer half of their sample, the annual growth rate of the part of full income that was due to health was 1.7% per year, vs. 0.4% per year in the richest half. Since the two parts of the sample had relatively similar growth rates of GDP,

convergence in full income was mostly driven by health. Further, in the poorer half of the sample, about 40% of full income growth was due to longevity improvement—a result that is similar to Murphy and Topel's finding for the US in the first half of the 20th century.

Jones and Klenow also look at cross-country data, examining both levels and growth rates of welfare. In addition to incorporating longevity into their calculations, they adjust their welfare measure for two other factors: within-country inequality of consumption and the average level of leisure. However, their finding is that by far the greatest contributor to welfare differences between rich and poor countries, other than consumption itself, is longevity (looking among rich countries, this is not the case, as longevity does not vary much while inequality and leisure do). The welfare differences that Jones and Klenow find are enormous, even by the standards of cross-country differences in income. For example, the average of income per capita in sub-Saharan Africa in their data is 5.3% of the US level, but the average level of welfare is 1.1% of the US level. Looking at welfare growth over time, their results are only partially consistent with the two papers discussed above. Over the period 1980–2000, longevity growth in the US contributed 1% point to annual welfare growth of 2.7% per year. However, looking across countries, they do not find evidence that convergence of welfare over the period examined greatly exceeded convergence of GDP per capita.

3.6.1.1 Underlying Theory

Health directly affects individual utility both by enhancing the quality of life (holding consumption constant) and by raising the quantity of life. Here I focus solely on the latter channel. The starting point for a theory that combines utility from consumption and length of life is to examine individual choices in which the two are traded off against each other. Consider a person who is faced with the opportunity to avoid taking a small risk to his life in return for a small payment. Let ϵ be the probability of death and x be the payment that makes the individual indifferent. The value of a statistical life (*VSL*) is defined as:

$$VSL = \frac{x}{\epsilon}.$$

VSL is most commonly estimated by looking at the wage premium associated with jobs that carry extra risk of mortality. The dollar value of marginal improvements in mortality can be directly assessed simply by using estimates of *VSL*. However, to assess the value of infra-marginal changes in mortality, and to compare changes in mortality to changes in consumption, one needs to impose more structure. A starting point is to assume that *VSL* is determined by setting equal the expected loss in utility from premature death and the addition utility from consuming x . Labeling V as the expected future utility, we have:

$$\epsilon V = (1 - \epsilon)u'(c)x.$$

In practice, the $(1 - \epsilon)$ term on the right-hand side of this equation can be ignored, since VSL is only measured in cases where ϵ is close to zero. The term V incorporates utility from both quality and quantity of life.

To incorporate quantity of life into a parametric utility framework requires that one insert another parameter into the utility function. A simple approach is to simply include a parameter $\bar{u}$, which can be interpreted as the “utility of being alive” that is addition to any utility from consumption. Allowing the consumption component of utility to be of the CRRA form, for example, we have:

$$U = \frac{c^{1-\sigma}}{1-\sigma} + \bar{u},$$

where utility from not being alive is normalized to zero.

To show how VSL can illuminate the relationship between life extension and growth, I examine a greatly simplified version of the model presented in the Murphy–Topel and Becker et al. papers. Following the “perpetual youth” approach of Blanchard (1985), I consider an individual who has constant mortality probability ρ and thus life expectancy of $1/\rho$. He has constant labor income and discounts future utility at rate θ , which is equal to the interest rate. Further, I assume that there is an actuarially fair annuity market. In such a setting, the optimum will be to maintain constant consumption, equal to the wage. Putting all this into the equation above and re-arranging:

$$VSL = \frac{x}{\epsilon} = c^\sigma \frac{\left(\frac{c^{1-\sigma}}{1-\sigma}\right) + \bar{u}}{\rho + \theta}.$$

In turn, we can solve for the parameter $\bar{u}$ in terms of the value of a statistical life as well as the other, more standard components of the utility function:

$$\bar{u} = VSL \times c^{-\sigma} (\rho + \theta) - \left(\frac{c^{1-\sigma}}{1-\sigma}\right).$$

With these parametric estimates in hand, one can undertake a number of quantitative exercises. The first is to calculate the value of consumption at which individuals are indifferent between being alive or dead. For the value of VSL I use \$4 million, which is broadly consistent with the literature for the United States according to Jones and Klenow.³² Personal consumption expenditures per capita in the United States in 2012 were approximately \$35,500. I use this figure for c , ignoring issues such as economies of scale in household production and the life cycle pattern of consumption expenditures. I use a value of $\rho = 0.0133$, to give life expectancy of 75 years, and a pure time discount rate of $\theta = 0.02$.

³² Murphy and Topel use \$6.3 million as the average VSL for adults aged 25–55. As they point out, estimates of VSL generally do not adjust by age – an approach that makes little sense in this utility framework, since an older person who dies is losing out on less utility than a young person. In their calibrated model, VSL falls from \$7 million at age 30 to \$5 million at age 50 and \$2 million at age 70. The perpetual youth model I use here avoids this issue.

Table 3.2 Implications of variations in curvature of the utility function

σ	$\bar{u}$	Break even consumption (\$)
0.8	-10.11	34
log	-6.72	830
1.5	0.03055	4286
2	0.000134	7465

The results of this exercise are very sensitive to the curvature of the utility function. Table 3.2 shows the implied value of $\bar{u}$ for different values σ , inverse of the intertemporal elasticity of substitution (Both Becker et al. and Murphy and Topel use 0.8 as their preferred value. Jones and Klenow use 1.0.) Since it is measured in utility terms, $\bar{u}$ itself is not very meaningful. The third column of the table shows the level of consumption at which individuals are indifferent between being alive or dead (labeled “break even” consumption), which can be seen to vary enormously with the value of σ . For some values of σ that would be considered empirically reasonable, the level of consumption at which life is not worth living is quite high.

I use this framework to carry out two exercises. The first is to re-visit the equivalence between increases in consumption and increases in life expectancy. Again, I consider the increase in life expectancy that provides increased utility equal to a 1% increase in c . This is derived by differentiating lifetime utility with respect to $\ln(c)$ and with respect to $(1/\rho)$ and taking their ratio. The formula is:

$$\text{gain in life expectancy} = \frac{c^{1-\sigma} \left(1 + \frac{\theta}{\rho}\right)^2}{(\rho + \theta) \left(\frac{c^{1-\sigma}}{1-\sigma} + \bar{u}\right)} \times .01.$$

I show the tradeoff at values equal to the US level, and then one-half, one-quarter, one-eighth, and one-sixteenth, and one thirty-second of that level. In each case, I calculate the rise in life expectancy (in years) that is equivalent to a 1% increase in the consumption measure c . (The entire exercise is conducted holding initial life expectancy constant at its US level of 75 years. Obviously, poor countries also have lower life expectancies than rich. However, in this setting, the effects of these differences are relatively muted.) I conduct the exercise for the same four values of σ considered above. Table 3.3 shows the results. The first column shows that for the parameters used in the calibration, people in the United States are made equally well-off by an increase in consumption of 1% and a gain in life expectancy of half a year. Compared to the HDI calculations discussed above, then, the model here weighs life relatively less. However, this is largely a matter of parameterization. For example, raising the value of a statistical life in the US to around

Table 3.3 Gain in life expectancy equivalent to 1% rise in consumption

σ	Consumption					
	35,000	17,750	8,875	4,437.5	2,218.75	1,109.38
0.8	0.499	0.525	0.558	0.602	0.662	0.747
1 (log)	0.499	0.612	0.791	1.12	1.91	6.46
1.5	0.499	0.906	2.14	53.6	—	—
2	0.499	1.36	9.92	—	—	—

\$20 million would set the implied gain in life expectancy equivalent to a 1% rise in consumption in the US equal to the value in the HDI. The more important results in Table 3.3 have to do with the variation in outcomes as income changes. One implication is that as a country grows richer, people are willing to give up more income in return for a given increment in health (this is the reciprocal of the number shown in the table). This effect is stressed by Hall and Jones (2007) as an explanation for increases in health spending as countries get richer: the marginal utility of consumption within a year declines as consumption rises, but the marginal utility of extra life years does not decline as life gets longer.³³ As the table also shows, this effect is magnified, the more curved is the within-period utility function, that is, the larger is the value of σ . In their baseline quantitative analysis, which forecasts that health spending in the United States could reach 30% of GDP by the middle of the 21st century, Hall and Jones use a value of $\sigma = 2$.³⁴ I return to other implications of the table in a moment.

The other, related exercise is to calculate the ratio VSL/c , which can be thought of as the value of a statistical life relative to the individual's ability to pay. It is not surprising that VSL rises with income, because richer people have more money to spend on everything. By contrast, the behavior of VSL/c gives more insight into the underlying economics. The values of this ratio are presented in Table 3.4 for the same values of σ and c that were considered in the previous table.

These two tables convey an interesting point. In this framework, the value that individuals place on extra years of life relative to income is strongly dependent on the level of income. As income falls, people raise the gain in life expectancy that is equivalent to an income increase, and similarly, they lower the ratio of the value of a statistical life to consumption. The reason, in both cases, is that according to the model, people who are significantly poorer than Americans get relatively little utility per period alive. Thus they value additions to consumption (raising the utility per year) far more than they value adding extra years of life. As the tables show, these effects are exacerbated for higher

³³ This result holds for standard, additively separable preferences. Bommier (2006) presents an alternative, intuitively appealing approach that allows for decreasing returns to lifetime as well.

³⁴ In addition to the curvature of the utility function, the optimal share of spending on health in their model depends critically on the elasticity of health status with respect to health status, which they estimate declines in the level of health spending.

Table 3.4 Ratio of value of statistical life to annual consumption

σ	Consumption					
	35,000	17,750	8,875	4,437.5	2,218.75	1,109.38
0.8	112.7	107.1	100.8	93.4	85.0	75.4
1 (log)	112.7	91.9	71.1	50.3	29.5	8.7
1.5	112.7	62.1	26.3	1.0	—	—
2	112.7	41.3	5.7	—	—	—

values of the σ , the inverse of the intertemporal elasticity of substitution. Indeed, many of the results in the tables seem downright crazy. For example, assuming log utility, the model implies that a person in a country with income per capita equal to 1/32 of the US level would be indifferent between increasing consumption by 1% and raising life expectancy by six years. Taking this model seriously would give the policy implication that aid to the poorest countries should be aimed at raising consumption far more than toward saving lives. However, the model clearly has something wrong with it—a point to which I return below.

3.6.1.2 Compensating and Equivalent Variations

The above analysis considers marginal changes in consumption and life expectancy. To evaluate non-marginal changes in life expectancy and consumption, authors in this literature have used the mechanisms of compensating and equivalent variation.³⁵ Denote life expectancy as e and expected lifetime utility as $V(e, c)$. Consider a comparison of two countries (or a single country at two points in time), denoted a and b , where a will serve as the benchmark. The equivalent variation measure asks how much consumption would have to be adjusted downward in country a such that expected utility in the two countries was equal:

$$V(e_a, \lambda_{ev} c_a) = V(e_b, c_b).$$

The compensating variation measure, in contrast, asks how much consumption has to rise in country b in order to set expected utility equal in the two cases:

$$V(e_a, c_a) = V\left(e_b, \frac{c_b}{\lambda_{cv}}\right).$$

Using the model of perpetual youth presented above, we can solve explicitly for both of these:

$$\lambda_{ev} = \frac{1}{c_a} \left(\left[\left(\frac{\rho_a + \theta}{\rho_b + \theta} \right) \left(\frac{c_b^{1-\sigma}}{1-\sigma} + \bar{u} \right) - \bar{u} \right] (1-\sigma) \right)^{\frac{1}{1-\sigma}},$$

³⁵ This treatment closely follows Jones and Klenow.

$$\lambda_{cv} = c_b \left(\left[\left(\frac{\rho_b + \theta}{\rho_a + \theta} \right) \left(\frac{c_a^{1-\sigma}}{1-\sigma} + \bar{u} \right) - \bar{u} \right] (1-\sigma) \right)^{\frac{1}{\sigma-1}}.$$

Important differences between the two measures arise when the level of flow utility (that is, utility from consumption plus $\bar{u}$, the utility from being alive) is near zero in a poor country. For example, consider the comparison of the United States and Zambia. Using data from the Human Development Report, GNI in the two countries is \$43,480 and \$1,358, respectively, while life expectancy is 78.8 and 49.4. The ratio of GNI (which I use as a proxy for consumption) in Zambia to that in the US is 3.1%. Assuming log utility, the value of λ_{ev} is 2.8%, reflecting only a small adjustment for the mortality gap: since life is barely worth living in Zambia, according to this calculation, the additional loss that would be incurred by someone from the US in switching to Zambian consumption and life expectancy (rather than just Zambian consumption) is relatively small. By contrast, the value of λ_{cv} is 1.3%, reflecting the fact that in order to give a Zambian lifetime utility equal to someone from the US, his annual flow utility would have to be raised enough to compensate for his lower life expectancy.³⁶

3.6.1.3 Variation in the Value of a Statistical Life Across Countries

Many of the problems in the above framework can be related to a single cause: the use of the valuation of a statistical life in the United States to impute a value of $\bar{u}$, the utility of being alive, that is then imported to other countries or time periods. People in the United States behave in a manner that suggests that they would rather be dead than consume at a level that characterizes many people in the developing world, but there is little reason to think that many people in developing countries feel the same way.

Direct evidence on *VSL* bears out this prediction. [Cordoba and Ripoll \(2013\)](#) examine data from [Viscusi and Aldy \(2003\)](#) on measures of *VSL* in a scattering of countries at different income levels, as well as *VSLs* of different income groups within the United States. Their analysis of the data leads them to conclude that the ratio of *VSL*/*c* is actually falling in the level of consumption, although looking at their data it seems equally reasonable to conclude that the ratio of *VSL* to consumption simply does not vary with consumption. In either case, however, the implication of the standard

³⁶ Jones and Klenow get much larger differences between CV and EV. In the most extreme case of Malawi, the two differ by a factor of 17. One reason that they get such large differences is that in their formulation there is no pure time discount factor, which leads to a larger effect of life expectancy on expected lifetime utility. To give an example, consider countries with life expectancies of 100 and 50 years. In the Jones-Klenow setup, the CV measure will increase in the low life expectancy country's consumption such that flow utility is twice as high as in the country with high life expectancy. By contrast, in the model I present, with a time discount rate of 2%, the CV measure will increase flow utility in the low life expectancy country to be one and one third times as large as in the high life expectancy country. In practice, Jones and Klenow present the geometric average of λ_{ev} and λ_{cv} as their main result, but they note that most of their conclusions hold using either measure alone.

model presented above, which is that VSL/c should be rising with the level of consumption, seems to be soundly rejected. Further, as Cordoba and Ripoll note, there is no evidence that poor people have negative values of VSL , as the standard theory predicts.

How can we reconcile these observations with the implications of the tried-and-true utility model? Cordoba and Ripoll propose to solve the problem by looking at a non-expected utility model, in which the coefficient of relative risk aversion is decoupled from the intertemporal elasticity of substitution. In their model, individuals have a high level of risk aversion toward the state of the world in which they are not alive, but a relatively high intertemporal elasticity of substitution. The specific mechanism that gets the result that VSL/c decreases as countries get richer runs through life expectancy, which in the data is correlated with income. In their view, the marginal willingness to pay for an extra chance of survival decreases with the probability of survival; in other words, a person will pay more to raise their chances of surviving from 5% to 6% than from 95% to 96%. Cordoba and Ripoll also note that their model matches reality better than the standard model in another dimension, specifically the preference of individuals for late rather than early resolution of uncertainty regarding risk of mortality.

An alternative approach to explaining the behavior of VSL across countries is proposed by [Prinz and Weil \(2013\)](#), who ground their approach in a simple model of habit formation, along the lines of [Carroll et al. \(2000\)](#). Consider an individual with instantaneous utility function:

$$u(c) = \frac{\left(\frac{c}{z^\gamma}\right)^{1-\sigma}}{1-\sigma} + \bar{u}, \quad (3.10)$$

where z denotes habitual consumption and $0 \leq \gamma \leq 1$ denotes the degree of importance of habit formation. In the case of “external habits,” z is determined by the average level of consumption in a country. An individual contemplating a small risk to his life in return for a small monetary benefit will take the value of z as fixed. Thus, for example, the values of “break even” consumption in [Table 3.2](#) at which a person would be equally happy dead or alive, calculated based on the observed VSL in the United States, are correct for someone with the US stock of habits. However, a person in a poor country, with a lower stock of habits, would have higher utility, and thus be happier alive than dead, at these same consumption levels.

For a given value of γ , and under the assumption that within a country z is equal to c , one can calculate $\bar{u}$ as well as the other quantities derived above, such as the trade-off between increased life expectancy and consumption. To give an example of how the Prinz-Weil approach leads to more sensible values for VSL in poor countries, I repeat the exercise of [Table 3.4](#), looking at the ratio of VSL to annual consumption, assuming habit formation of $\gamma = 0.5$. [Table 3.5](#) shows the results. Unlike the original version of the table, the ratio of VSL to consumption rises far more modestly with

Table 3.5 Ratio of value of statistical life to annual consumption with $\gamma = 0.5$

σ	Consumption					
	35,000	17,750	8,875	4,437.5	2,218.75	1,109.38
0.8	112.7	110.1	107.1	104.0	100.8	97.2
1 (log)	112.7	102.3	91.9	81.5	71.1	60.7
1.5	112.7	85.2	62.1	42.7	26.3	12.6
2	112.7	70.9	41.3	20.4	5.7	—

income, and there are fewer cases where VSL is negative.³⁷ Allowing for habit formation, the ratio of VSL to consumption for a country with $\frac{1}{32}$ of US consumption, in the case of log utility, is 61. The corresponding ratio without habit formation is 8.7.

This being said, however, the Prinz-Weil approach cannot, at least by itself, explain the observation that Cordoba and Ripoll make, that $VSL/\text{consumption}$ does not vary at all with income, unless one is willing to make the extreme claim that the degree of habit formation is one. This extreme case would imply that people in poor countries are just as happy with their level of consumption, adjusted for habits, as people in rich countries. An alternative explanation is that the values of $VSL/\text{consumption}$ observed in the data are partially a result of the habit formation effect and partly a result of something else, for example, higher expected consumption growth in poor than rich countries.



3.7. CONCLUSION

Income and health are strongly correlated. Looking across countries, higher income per capita is correlated not only with life expectancy, but with numerous other measures of health status. Within countries, there is also a strong correlation between an individual's place in the income distribution and his or her health outcomes. This within-country correlation is particularly strong in developing countries.

Comparing growth of income with improvements in health outcomes, things are a bit more complicated. In the short run, there is at best a weak correlation between changes in income and changes in life expectancy. Indeed, there are many examples of dramatic improvements in health taking place in the absence of notable income growth, and similarly of episodes of rapid income growth that are not accompanied by health improvements. On the other hand, we know that prior to the Industrial Revolution levels of income and life expectancy were roughly the same throughout the world while today the two are strongly correlated, and further that the pattern of initial divergence

³⁷ The formula for $\bar{u}$ is:

$$\bar{u} = c^{-\sigma-\gamma+\sigma\gamma}(\rho+\theta)VSL - \frac{c^{(1-\sigma)(1-\gamma)}}{1-\sigma}.$$

and later catch-up on the two series look similar. All these facts suggest that in the very long run income growth and health improvement are indeed correlated.

As is often the case in economics, the observation that income and health are correlated, is only the beginning of the discussion. Such a correlation can be induced by causation running in either direction, as well as by the effects of some third factor. A priori, there are good reasons to think that all of these are possibilities. People who are healthier can work harder and learn more in school; and where people live longer they will be incentivized to invest more in education. Thus, we would expect better health to cause economic growth. On the other hand, higher income allows individuals or governments to make investments that yield better health. Finally, differences in the quality of institutions (looking across countries), in human capital (looking across individuals), or in the level of technology (looking over time) can induce correlated movements in health and income. Further complicating the inference problem are the dynamic effects built into many of the potential causal channels. For example, improvements in health may only result in increased worker productivity with a lag of several decades. Similarly, when life expectancy rises there can be increases in population growth that may temporarily reduce income per capita.

The causal relation that has been most widely studied by researchers in this area is the effect of health improvements on economic growth at the country level. This is an issue with direct policy relevance. If improving health leads to growth, this would be a reason, beyond the welfare gain from better health itself, that governments might want to make such investments. However, the evidence for such an effect of health on growth is relatively weak. Cross-country empirical analyses that find large effects for this causal channel tend to have serious identification problems. The few studies that use better identification find small or even negative effects. Theoretical and empirical analyses of the individual causal channels by which health should raise growth find positive effects, but again these tend to be fairly small. Putting the different channels together into a simulation model shows that potential growth effects of better health are only modest, and arrive with a significant delay.

Regarding causality running from income to health, at least at the level of countries, there is also little evidence of much effect in the short run. For developing countries, there exists a large stock of health technologies that can be applied to great effect at low cost. Political will and institutional efficiency are more important than GDP in determining health. Looking across individuals, it is harder to sort out the extent to which it is knowledge of health improving behaviors or economic wherewithal (which is correlated with human capital) that is more important in contributing to the correlation between health and income. Possibly, this even differs as a function of level of economic development (as does the effect of health on income at the individual level).

In the short run, then, at least as regards differences among countries, one is forced to the conclusion that the strong relationship between income and health is a product of

some other factors. The same political will and institutional efficiency that lead to better health also lead to higher income, most of the time, but with some important exceptions.

Looking at historical changes, however, the picture is different. It is hard to escape the conclusion that in the long run, improvements in health have indeed been the result of economic growth. It is not hard to identify the scientific discoveries, medical advances, and public health initiatives that have produced enormous health gains in the most advanced countries. These achievements seem unlikely to have occurred outside the context of industrialization. As a counterfactual, it is possible to imagine a history in which economic growth (technological advance; accumulation of physical and human capital; institutional change; and so on) took place roughly as we have observed it, but in which life expectancy and other measures of health remained stuck at their 18th-century levels. But it is not similarly possible (at least for me) to imagine a history in which knowledge regarding health advanced and was implemented as it has been in the absence of economic growth.

In contrast to the uncertainty about causality, analysis of the welfare effects of health improvements is much more straightforward: they are very large. Depending on the period being examined, the welfare gain from better health may be as large or larger than the welfare gain from rising consumption.

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