Education and Longevity

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Abstract

Around 1700, a remarkable increase in life expectancy began in Europe and North America that spread to other parts of the world, eventually adding 48 years to expected length of life. The sudden and persistent increase in longevity was a departure from centuries of stagnation at a steady state equilibrium characterized by short lifespans lived at or below subsistence. This chapter reviews the antecedents to that increase in life expectancy including an agricultural revolution that increased average caloric consumption and health, an industrial revolution that increased income, and a human capital revolution that increased capacity to produce and grow. We show that increases in life expectancy led to rising investments in human capital, and that increased human capital had feedback effects on improved health. This virtuous cycle between health and education represents part of the endogenous growth mechanism that has increased life expectancy, education, income, and quality of life around the world, and offers a path out of poverty for the countries yet to develop.

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This chapter reviews the large literature that has examined the associations between education and life expectancy that have been observed across countries and across time periods. We review the historical evidence of the sequence of agricultural, industrial and human capital revolutions that led to the growth of the advanced economies around the world. We then present the theoretical arguments and econometric results that identify the causal pathways between education and health. The literature indicates that the relationship goes in both directions, so that rising life expectancy leads to increased schooling, and increased education leads to increased investments in health. Disentangling these complex associations is important for understanding the mechanisms of endogenous economic growth. The historical revolutions and the theoretical and empirical analyses of longevity and education contain lessons for improving the lives and livelihoods of the residents of developing countries.

One such lesson is that a large enough workforce with skills to utilize modern technologies is a necessary element for a successful industrialization and sustained economic growth. But while instituting the right set of education policies can make this skilling-up happen, the race between the supply of skills and the demand for skills cannot be won without also strengthening the incentives for parents to invest in their children's schooling, a change that requires an expected lifespan sufficiently long to provide an expected return on that investment.

In Section I, we review the historical record on how increases in life expectancy led to increases in education through successive economic revolutions that changed how people worked, how much they earned, and how they invested time and resources in schooling and health, and how the growing skill endowments embodied in the workforce helped to sustain the economic expansion beyond the initial industrial revolution. In Section II, we extend the model

initially developed by Heckman (1976) to show how an increase in life expectancy at birth can affect lifetime human capital investment. Under fairly general conditions, rising lifespans increase completed years of schooling. In Section III, we discuss the empirical issues related to identifying the effect of life expectancy on education. We conclude that the evidence is substantial that rising life expectancy increases human capital investments. Section IV reviews the studies that have addressed the reverse causal relationship of education affecting health status, health behaviors, and thus life expectancy. We present our conclusions in Section V.

1. How life expectancy leads to human capital investment: The historical record

Life expectancy did not rise appreciably in the world until the 1700s. Although data is spotty, Maddison's (2007) review suggests that life expectancy during the Roman Empire was 23 to 25 years for citizens, but undoubtedly lower for slaves. By 1000, life expectancy at birth was 24 years in Europe. Over the 1,700 years after the birth of Christ, life expectancy rose at a compound rate of 0.02% per year. Thereafter, as we show in Figure 1, life expectancy improved at a rate of 0.28% per year, rising by 49 years by 2018. World population and per-capita Gross Domestic Product also demonstrated dramatic growth after 17 centuries of stagnation. World population grew at a compound rate of 0.06 percent per year before 1700, and per-capita income rose even more slowly at 0.02 percent per year. The average person in 1700 had an income only 40 percent greater than the average person 1700 years earlier. When Malthus (1798) concluded that the population was doomed to live at subsistence levels, he offered a reasonably accurate portrayal of life before 1700. Ironically, by the time he published his essay on population, the subsistence steady state he described was breaking down. After 1700, world population grew at 0.8 percent per year or 13 times faster than the pre-1700 growth rate and per-capita GDP grew

even faster at 0.85 percent per year, over 50 times the pre-1700 rate. The average person in 2018 had a real income 15 times greater than the average person in 1700.

[Figure 1 about here]

In this chapter, we review the implications of the increase in life expectancy on human capital accumulation. To begin that review, it is useful to examine why life expectancy improved and how advances in agriculture, nutrition and public health infrastructure contributed. Most of the initial rise in expected length of life at birth are not due to the choices made at the household level, but are instead a result of exogenous changes in technologies and government policies and investments that raised life expectancy for the general population. These changes motivated and enabled shifts in individual behaviors that led to sustained improvements in human capital.

Agricultural revolutions

The remarkable improvement in the length and quality of life since 1700 began with what Robert Fogel (2004) called the second agricultural revolution. He dates the start of the British agricultural revolution in the early 1700s, while Aposolides et al. (2008) argue that the rise in agricultural productivity began in the 15th century but accelerated after 1700. In either case, it is apparent that, in the 1700s, agricultural production grew more rapidly than population, raising per capita caloric intake. Before the 18th century, the average European before the 18th century was malnourished and incapable of doing a full day's work (Fogel, 2012). Malnutrition also meant that individuals did not have the energy to fight off disease and infection, resulting in early deaths.

Huffman and Evenson (1998) documented the timing of many of the agricultural innovations that led to improvements in land productivity. One such innovation was selective breeding of livestock in 1760, credited to Robert Bakewell, nearly a century before Gregor Mendel's work established the existence of recessive and dominant genes in peas. Justus von Liebig, one of the founders of organic chemistry, proposed the use of chemical nutrients to enhance soil quality in Germany in the 1830s and extended this research to animal physiology in the 1840s. The enclosure movement in England which began in the 12th century but not completed until the 19th century, helped to control animal breeding which required segregation of preferred stock. The enclosure movement eliminated common grazing lands, accelerating the pace of livestock improvements, and solved the tragedy of the commons where overgrazing led to chronic animal malnutrition.

Another major source of the increases in land productivity was the development of new strategies for crop rotations. These consisted of methods to extend practical information on fertilizers to farmers, but they also tested new machinery, seed varieties, and crop rotation patterns. They were enhanced by the establishment of agricultural experiment stations in Germany and in the United Kingdom in the 1840s and 1850s.

While quantifying the link between agricultural productivity and overall economic growth in the 1700s is limited by poor data, the experience of the Green Revolution (also known as the third agricultural revolution) in the mid-1960s provides revealing evidence of how it could have happened. Gollin et al. (2021) show that the high-yield varieties developed in Asia boosted agricultural productivity by 44 percent during the period 1965-2010 and roughly doubled per-capita GDP in the developing world. A remarkably similar finding was reported by Fogel (2004) who estimated that one-half of the British economic growth between 1790 and 1980 could be

attributed to the increase in people's caloric intake and the more efficient use of calories for productive work rather than for fighting disease or surviving the elements.

Gollin, Parente and Rogerson (1993) present a growth model that shows that a slow-growing agricultural sector delays industrialization and, conversely, that agricultural output must rise above some threshold in order to release labor to the nonagricultural sector. Their stylized model is consistent with our assessment that populations had to attain sufficient calories to fuel industrialization. The manufacturing sector gave more opportunities for technological advancement including technologies that were complementary with human capital, and that creates a virtuous cycle of further growth and further demand for human capital.[1]

In sum, the industrial revolution was invariably preceded by an agricultural revolution that broke the Malthusian subsistence steady state (Huffman and Orazem, 2007). This was true in the industrial revolution in Japan as well, and true of the effects of the Green Revolution as it jumpstarted economic expansion in other parts of Asia. The only major exceptions to the pattern were in the deep-water ports of Hong Kong and Singapore which were able to trade into the agricultural revolutions occurring among their trading partners. To this day, the last remaining countries that have not experienced an industrial revolution are in Africa where per-capita caloric intake is at or below the levels in Europe in the 1700s. It is unlikely that Sub-Saharan Africa will experience a surge in per capita incomes and life expectancy without first experiencing a major surge in agricultural productivity.

Food and nutrition revolution

[1] Galor (2011) presents a unified growth framework that includes a Malthusian trap that is escaped by an acceleration in technological change induced by rising population. After that, increases in incomes and the pace of technological change raise the demand for and supply of human capital and moderate population growth. As his model requires some mechanism to escape the Malthusian steady state, it could also be consistent with an escape fueled by an agricultural revolution.

A significant development, emphasized by Fogel (2012), which followed the agricultural revolutions, is the increase in food availability among the poorest people in society. In response to bread riots, the British government greatly expanded its poor relief; this transferred resources to the poorest households, effectively doubling their income. As a result, at the start of the British industrial revolution, as agricultural productivity rose, the daily caloric intake of the poor in England was 18 percent greater than in France. This meant that the poorest 20 percent could average six hours of work, and that greater effective labor supply helped launch the British industrial revolution 50 years before that of France (Fogel, 2012). In comparison, the least nourished 10 percent of the French population was too malnourished to do any work, and the next 10 percent could manage just three hours of light work. Nevertheless, even in the better-fed British population, most were so stunted and wasted from persistent low caloric intake that they were prone to premature mortality. It was the continuing improvement of agricultural productivity growth that sustained the continued increase in per capita incomes and life expectancy through the course of the industrial revolution. Rising yields, however, were not sufficient to break the Malthusian subsistence steady state.

A coincident development which helped to enable the wider distribution of agricultural products and the greater availability of food supply was the development of the national road system, first in England and then elsewhere in Europe and North America. Guldi (2012) documents that, in 1726, the year the British government began developing its national roadway, each parish was responsible for its own roads and each had an incentive to free ride. As a result, travelers were mired in mud and muck, making agricultural trade quite difficult. By 1848, there were 40-foot-wide gravel highways connecting every British village, allowing rapid shipments of

agricultural products from countryside to city. The resulting development of national commodity markets broke local monopoly food pricing.

With the industrial revolution came higher real wages. Because demand for food is inelastic, the increase in available food supply through improved yields and better transportation lowered agricultural prices. As food took up to 75 percent of household budgets, this meant an increase in real incomes in urban relative to rural areas, incentivizing the shift of populations from rural to urban markets (Jorgenson, 1970; Huffman and Orazem, 2007).

Human capital revolution

The agricultural and industrial revolutions were followed by a human capital revolution, with literacy and schooling expanding rapidly in the 20th century. America epitomized the "human capital century," according to Goldin (2001), but all countries that experienced an industrial revolution in the 1800s experienced a surge in human capital investments in the late 19th and 20th centuries. Scholars have disagreed about the direction of causality, however. Some argue that human capital spurred and enabled economic growth, while others argue that growth generates and rewards investments in human capital. There are reasons to support both views.

The jobs in the first industrial revolution did not require literacy. As of 1842, only 4.2 percent of men and 2.2 percent of women in England held jobs that required literacy (Mitch, 1993). To the extent that skills were required, they were acquired on the job, not in schools. Literacy rates (as measured by the ability to sign marriage certificates) declined in some regions experienced expanding manufacturing, while literacy rates rose in other regions that did. As a result, some argue that the industrial revolution led to increasing literacy (West, 1978), and others remain unconvinced (Mitch, 1993).

The more important effect of the industrial revolution on human capital development was through its effect on household income. As shown by Becker and Tomes (1976), as income rises for poor households, investments in child quality (such as education) would respond elastically and positively while quantity of children would respond inelastically and perhaps negatively. This moderates population expansion as economic growth takes off, a pattern that has occurred in virtually all developed countries and in most developing countries.[2] Moreover, Becker and Tomes (1986) argue that, because the initial returns to human capital exceed the returns on financial assets, parents first transfer human capital assets to their children, and only revert to financial assets when the return to the marginal investments in human capital fall below the rate of return to physical assets.

During the industrial revolutions, a significant proportion of families' incomes was contributed by children because the growth of industries demanded an expansion of labor supply at low wages (Cunningham and Viazzo, 1996). Children below 15 years – and even as young as seven – worked. According to the censuses of England and Wales, in 1850 as many as 39 percent of boys and 21 percent of girls aged 5-14 were employed (Cunningham, 1996). U.S. Census data in 1870 show that one in eight children was employed, with this rate increasing to more than one in five children by 1900 (U.S. Bureau of Labor Statistics, 2017). That year, 32.4 percent of boys and 12.2 percent of girls aged 10-14 were employed (Carter and Sutch, 1996).

Child employment rates varied across areas, economic sectors and occupations, depending on differences in employment opportunities. For example, over one-half of boys aged 10-14 years were working in the textile mills of Yorkshire in 1850 (Cunningham and Viazzo,

[2] See Rosenzweig and Wolpin (1988) and Soares (2005) for examples of papers that examine how uncertainty of child survival or child mortality affect fertility behavior. As life expectancy rises and/or mortality falls, parents reduce fertility as they no longer have to have extra children beyond their planned number in anticipation that some will not survive childhood.

1996), and in France, more than one-third of girls and boys aged 10-14 worked in the textile town of Roubaix and over half of the boys aged 10-14 worked in the coal mining town of Anzin (Tilly and Scott, 1978). In 1817, many children in Ghent, Belgium, were working in spinning mills, paper mills, tobacco factories, brickworks, and coal mines (de Herdt, 1996).

Putting the historical data on child labor into current context, one can compare the percentages of children working during the early industrialization in Europe and the U.S. with the use of child labor in today's developing countries of similar GDP per capita level as Europe and the U.S. when they were experiencing the initial industrialization (Figure 2A). In 1870, when one in eight children was working in the U.S., the U.S. GDP per capita (in 2011 PPP dollars) was 3,876 (Bolt and Luiten van Zanden, 2014). In 2012, Ghana's GDP per capita was only 8 percent lower (in 2011 PPP dollars) than the U.S. GDP per capita in 1870; in Ghana in 2011, one in three children was working compared to one in eight. In Italy, also one in three children was working in 1936, but Italy's GDP per capita in 1937 (5,651 in 2011 PPP dollars) was 1.6 times that of Ghana's in 2012. Parents can better afford to sacrifice child labor as household incomes rise. However, the decline in child labor also reflects the coincident increase in life expectancy that raises the value of devoting child time to schooling rather than work (Figure 2B).

[Figure 2 about here]

In country after country, after the onset of industrialization, faster economic growth and rising incomes were followed by a decline in child labor. These changes were driven partly by a growing concern for the health and safety of young children working in factories and mines,[3] and

[3] For example, using 1843 anthropometric data cited by de Herdt (1996), the average height of boys aged 14 working in the Ghent textile factories was 92 percent of the normal height of 14-year-old boys in Ghent and their

partly by a recognition that an illiterate population was not good for industries that required greater and more distributed skills.[4] In 1880 in England and Wales, the government adopted compulsory education primarily to compel those children who were not working to attend school. The proportion of children aged 5-14 attending the so-called "inspected day schools" increased from 6.2 percent in 1851 to 74.8 percent in 1911 (Cunningham and Viazzo, 1996). In Belgium in 1845, 34 percent of children 7-14 were not educated at all, but by 1875 this percentage had declined to 27 percent (de Herdt, 1996). And in 1896, seven years after the passage of the first law to control child labor, there was a significant decrease in the number of children under 16 who were working in Ghent's textile mills (Scholliers, 1995).

It would be tempting to regard education compulsory laws as the principal factor for the decline in the use of child labor since the timing of the first truancy laws generally follows the onset of the country's industrial revolution. However, only the Prussian law predated the onset of its industrial revolution. More plausibly, countries established truancy laws after child labor was already in decline so that the laws would not interfere with industrialization. (Cunningham and Viazzo, 1996; de Herdt, 1996; Saito, 1996). For example, in the case of Belgium, the education law was targeted primarily at non-working children so that it would not disrupt the supply of cheap child labor (de Herdt, 1996). We prefer to interpret the timing of the truancy laws as the onset of the human capital revolution when the country places an emphasis on the acquisition of literacy and numeracy. As shown in Figure 3, the adoption of truancy laws follows closely after the surge in life expectancy at birth.

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average weight was 82 percent of the normal weight. These differences are, of course, due not only to working conditions but also to poor living conditions and diets.

[4] In England and Wales, by the turn of the 20th century, the percentage of children aged 10-14 who were working had fallen to 22 percent (Cunningham and Viazzo, 1996).

[Figure 3 about here]

Schultz (1964) observed that the importance of human capital development in economic growth was not at the start of the initial industrial revolution, but in the continuation of the industrial revolution. The implication for current developing countries is that to foster a successful shift from an agrarian to an industrial economy, they must have a large enough workforce with sufficient skill to utilize modern manufacturing technologies. As an example, plants located in developing countries financed by foreign direct investment use workers with education levels higher than the country average. Because the rise in life expectancy that coincided with the industrial revolution raises the return to human capital, there was an additional incentive for parents to invest in their children's schooling (Figure 4).

[Figure 4 about here]

It is worth noting that a critical part of the human capital revolution was the major public health improvements undertaken by governments after the start of industrialization. In Britain and Boston, for example, "sanitarians" were employed to control bad smells coming from polluted waters and open sewage, who, even though the science behind infection and diseases was not yet established.[5] Believing that illness and death were associated with unsanitary

[5] Investments in public health infrastructure date much earlier than the 1700s and not only in Europe (Aiello, Larson and Sedlak, 2008; Vuorinen, Juuti and Katko, 2008; Lofrano and Brown, 2010). The Mesopotamian empire, Indus and Greek civilizations all had their own system of managing the supply of clean water and the disposal of waste. The Roman Empire invested heavily in collecting and distributing water supply through dams and aqueducts, and also in early sanitation facilities such as public toilets. They also made the connection between swamps and disease, such as malaria, and the selection of habitation (Novick and Morrow, 2008). Lofrano and Brown (2010) identify the period after the fall of the Roman Empire and the start of the industrialization as a dark time for these water and sanitation facilities. Medieval cities in Europe had councils whose role included the supervision of disease

conditions,[6] swamps were drained, eliminating the breeding ground for mosquitos. In Germany and France, sewer systems were built in the mid-1800s, and in Italy, a few decades later (Lofrano and Brown, 2010). Sewage disposal systems reduced cholera epidemics. Garbage disposal reduced disease bearing rodent and insect populations (Aiello, Larson and Svedlak, 2008; Lofrano and Brown, 2010). In addition, a system of collecting vital statistics was introduced (Novick and Morrow, 2008), and not long after, medical laws were enacted as part of early public health action to emphasize disease prevention.

But improvements in longevity were also due to changes in people's own health-seeking behaviors. These behavioral changes were enabled not only by the infrastructural investments in water supply and sewerage discussed above but also by a growing understanding of the importance of clean drinking water and hygienic practices, long before the coming of major medical and pharmacological interventions such as vaccines and antibiotics. During the 19th century, an increased awareness of the benefits of personal and environmental cleanliness among people led to the decline of endemic diseases such as infant diarrhea and dysentery and thus to significant reductions in infant mortality (Aiello, Larson and Sedlak, 2008). Preston (1975) argues that it is the countries that succeeded in raising their literacy levels and in spreading public health and nutritional programs widely among the population that achieved remarkable advances in lengthening life.[7] Several other studies have concluded that the significant declines

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prevention, control of transmission of diseases, sanitation (Novick and Morrow, 2008; Tulchinsky and Varavikova, 2014). Some medieval cities built their own wells, fountains or cisterns to manage their water supply, and latrines, but these facilities were inadequate to stop the spread of epidemics.

[6] Industrialization was accompanied by the development of large settlements. London more than tripled in size in 1700 and then doubled in the first half of the 1800s to more than two million residents, resulting in overcrowding, filth, and disease outbreaks (Johnson, Johnson and Morrow, 2013). At about the same time, the growth of tenement housing in New York City transformed "typhus into an endemic slum disorder" (Johnson, Johnson and Morrow, 2013).

[7] While morbidity and mortality due to poor water and sanitation conditions have declined and largely disappeared in most parts of the world, in several countries in sub-Saharan Africa (e.g., Angola, Niger, Sierra Leone, Chad),

in child mortality over the past century were the result of increases in parental education, especially in contexts where health infrastructure were poor (Caldwell and McDonald, 1982; Caldwell, 1994). We return to this effect of education on health and life expectancy later in the chapter.

2. Theoretical Framework

Our review of the historical record suggests that rising life expectancy led to an increase in human capital investments including schooling. In this section, we will show how life expectancy at birth will affect lifetime human capital investment in the context of Heckman's (1976) life cycle model of earnings, learning, and consumption. The model relaxes several assumptions of the Ben-Porath (1967) model that are important to our analysis, including that life cycle labor supply decisions are endogenous and selected jointly with human capital investment decisions, that human capital enhances the utility from leisure consumption, and that initial endowments of assets and human capital can alter the life cycle entire trajectory of consumption, human capital acquisition, and labor supply. We focus narrowly on the role of expected length of life on these decisions, and so we refer readers interested in how these decisions respond to initial endowments, interest rates, and taxes to the initial Heckman (1976) paper.

An individual's instantaneous utility is defined by $U[X(t), L(t)H(t)]$, where utility is concave in its arguments $X(t)$ and $L(t)H(t)$. Goods and services consumption is represented by $X(t)$, and leisure by $L(t)$. The value of leisure consumption is augmented by the individual's current accumulated level of human capital $[L(t)H(t)]$. An implication of this specification is

infrastructure investments in water and sanitation remain a priority and require a long-term, sustained effort from national governments (Fuente et al., 2020)

that individuals have an incentive to invest in human capital for consumption as well as for pecuniary purposes, and so there is an incentive to continue investing in human capital in retirement. However, individuals with smaller accumulated human capital at older ages get less utility from leisure, a finding that may help to guide analysis of the rising incidence of so-called "deaths of despair".[8]

Individuals maximize lifetime discounted utility over their expected lifespan, *T,* subject to constraints on time, human capital accumulation, and budget. For simplicity, individuals have one unit of time in each of the *T* periods. Time can be allocated to leisure $L(t)$, to investment in human capital $I(t)$, and to work $(1 - L(t) - I(t))$. Human capital *H(t)* is generated by combining time with purchased inputs into human capital, $D(t)$. Human capital is accumulated at the rate

$$\dot{H(t)} = F[I(t)H(t), D(t)] - \delta H(t) \tag{1}$$

$$H(0) = H_0 \tag{2}$$

where *F* is a concave production function in its arguments. Note that the productivity of time in human capita production, $I(t)$ is augmented by past accumulation of human capital, $H(t)$. The specification implies that human capital accumulation grows only if time is allocated to it, *i.e.,* $I(t) > 0$. Human capital depreciates at the rate of δ per period, and so some investment is required to keep skills from declining.

Consumers can spend out of current income, they can borrow against future income, or they can spend out of accumulated assets. Labor income depends on current accumulated human capital that is valued at *R* per unit so that the wage per time unit is $RH(t)$. Income can be

[8] Case and Deaton (2020) find that the rising incidence of "deaths of despair" is concentrated among the least educated.

allocated to education goods ($D(t)$) or consumption goods ($X(t)$) at price P. Given an endowment of initial assets A_0 and the human capital endowment H_0, an individual will accumulate wealth $A(t)$ according to

$$\dot{A(t)} = rA(t) + RH(t)[1 - I(t) - L(t)] - PD(t) - PX(t), \tag{3}$$

$$A(0) = A_0. \tag{4}$$

In equation (3), r is the risk-free rate of return on accumulated assets. Individuals can borrow at the same rate.

The individual's lifetime utility maximization is to maximize lifetime utility, $\int_0^T e^{-\rho t} U[X(t), L(t)H(t)]dt$, subject to the constraints (1)-(4). The Hamiltonian is

$$J(t): e^{-\rho t} U[X(t), L(t)H(t)] + \lambda(t)\{ rA(t) + RH(t)[1 - I(t) - L(t)] - PD(t) - PX(t)\} + \mu(t)\{F[I(t)H(t), D(t)] - \delta H(t)\}, \tag{5}$$

where ρ is the discount rate on future consumption, $\lambda(t)$ is the shadow value of an additional unit of wealth and $\mu(t)$ is the shadow value of an additional unit of human capital in period t. The first-order conditions are

$$J(t)_{X(t)}: U_1(t) = \lambda(t) e^{\rho t} P. \tag{6}$$

$$J(t)_{L(t)}: U_2(t)H(t) = \lambda(t) e^{\rho t} RH(t). \tag{7}$$

$$J(t)_{I(t)}: \mu(t) F_1(t) H(t) = \lambda(t) RH(t). \tag{8}$$

$$J(t)_{D(t)}: \mu(t) F_2(t) = \lambda(t) P. \tag{9}$$

$$J(t)_{A(t)}: \dot{\lambda(t)} = -r\lambda(t) \ \rightarrow \lambda(t) = \lambda(0) e^{-rt} \tag{10}$$

$$J(t)_{H(t)}: \dot{\mu(t)} = \delta\mu(t) - R\lambda(t). \tag{11}$$

Equation (6) implies that the individual will consume $X(t)$ up to the point where the current utility equals the value of saving that expenditure. Equation (9) equates the value of the human

capital generated from the marginal expenditure on education goods against its value as a financial asset. Equation (7) equates the utility value of leisure against the value of assets accumulated from forgone earnings. Equation (8) equates the value of time in human capital investment against the value at work.

The transversality condition requires that $\lambda(T)A(T) + \mu(T)H(T) = 0$. Nonsatiation with finite resources means that the shadow value of wealth is always positive ($\lambda(t) > 0\ \forall t \in [0, T]$. Without a bequest motive, the individual will exhaust all resources and so $A(T) = 0$. The individual will still have utility from leisure in the last period and so $H(T) > 0$, so we need $\mu(T) = 0$, an assumption consistent with the shadow value of human capital going to zero at the end of life.

To simplify the analysis further, we define the shadow value of human capital in terms of wealth, that is, the ratio of the shadow value of human capital to that of assets: $g(t) = \frac{\mu(t)}{\lambda(t)}$. Applying equations (10) and (11), the time derivative is

$$\dot{g(t)} = \frac{\lambda(t)\dot{\mu(t)} - \mu(t)\dot{\lambda(t)}}{\lambda(t)^2} = \frac{\dot{\mu(t)}}{\lambda(t)} - \frac{\mu(t)}{\lambda(t)}\frac{\dot{\lambda(t)}}{\lambda(t)} = \delta g(t) - R - rg(t) = (\delta + r)g(t) - R.$$

With $\mu(T) = 0$, we have also that $g(T) = 0$, and so we can solve backwards to derive

$$g(t) = \frac{R}{(\delta + r)}\left[1 - e^{(\delta+r)(t-T)}\right]. \tag{12}$$

Comparative dynamics from increase in life expectancy at birth T

As shown by Heckman (1976), the two basic assumptions of strict concavity and differentiability of the utility and production functions allow us to derive the reduced form demand equations for consumption goods $X(t)$, effective leisure, $L(t)H(t)$, educational goods, $D(t)$, and effective time investments in human capital, $I(t)H(t)$. We can insert the solutions for $\lambda(t)$ and invert equations (6), (7), to obtain the $\lambda(0)$ constant (Frisch) equations:

$$X(t) = X\left[\lambda(0)e^{(\rho-r)t}P,\ R\lambda(0)e^{(\rho-r)t}\right], \tag{13A}$$

$$H(t)L(t) = HL\left[\lambda(0)e^{(\rho-r)t}P,\ R\lambda(0)e^{(\rho-r)t}\right]. \tag{13B}$$

The demand functions for the two human capital investment inputs can be obtained by inverting equations (8) and (9).

$$D(t) = D\left[\frac{P}{g(t)}, \frac{R}{g(t)}\right]. \tag{13C}$$

$$I(t)H(t) = IH\left[\frac{P}{g(t)}, \frac{R}{g(t)}\right] \tag{13D}$$

Equations (13C) and (13D) provide the key to evaluating how life expectancy affects huma capital investment and accumulation. First, using (12) and setting *t=0,* we have that $\lim_{T\to\infty}[g(0)] = \frac{R}{(\delta+r)}$. Then $g(t) = g(0)\left[1 - e^{(\delta+r)(t-T)}\right]$. The value of *g(t)* is greatest at *g(0),* and declines as *t* increases over the life cycle. As a result, the relative price of educational goods, $\frac{P}{g(t)}$, and the opportunity cost of time, $\frac{R}{g(t)}$, increase with age.

Since both of these inputs are assumed normal, the own price increase would decrease the demand for both inputs as *t* increases. We explicitly show the case for $D(t)$.

$$D(t) = D\left[\frac{P}{g(t)}, \frac{R}{g(t)}\right] \forall\ t \in [0, T]$$

$$\frac{dD(t)}{dt} = -\frac{P}{[g(t)]^2} * D_1 * \frac{dg(t)}{dt} - \frac{R}{[g(t)]^2} * D_2 * \frac{dg(t)}{dt} = -\left[\frac{P}{g(t)} * D_1 + \frac{R}{g(t)} * D_2\right] * \frac{\frac{dg(t)}{dt}}{g(t)}.$$

Strong concavity and twice differentiability of the human capital production function implies that the term in brackets must be negative. With $\frac{dg(t)}{dt} < 0$ and $g(t) > 0$, $\frac{dD(t)}{dt} < 0$. A similar argument results in $\frac{dI(t)H(t)}{dt} < 0$. Because both investments decline with age, there is a steady decrease in the rate of increase of human capital over the life cycle.

However, from (12), $\frac{\partial g(t)}{\partial T} = Re^{(\sigma+r)(t-T)} > 0\ \forall\ t \in [0, T]$, and so the shadow value of human capital relative to wealth increases with life expectancy.

$\frac{dD(t)}{dT} = -\left[\frac{P}{g(t)} * D_1 + \frac{R}{g(t)} * D_2\right] * \frac{\frac{dg(t)}{dT}}{g(t)}$ >0. A similar derivation results in $\frac{dI(t)H(t)}{dT} > 0$, and so for a fixed values of *P* and *R,* an increase in life expectancy will cause individuals to increase the expenditures on $D(t)$ and the effective time invested in human capital, $I(t)H(t)$, in every year of life. Increased use of both *D(t)* and *I(t)H(t)* in every period *t* means that $\dot{H(t)} = F[I(t)H(t), D(t)] - \delta H(t)$ must be larger in every period, and so rising life expectancy raises the accumulated human capital in every period of life.

This assumes that rising levels of human capital do not cause wages per unit of human capital to fall or that rising investments in educational goods do not bid up the price of those goods. Of course, it is equally plausible that agglomerations of human capital will bid up the value of human capital per unit. Ultimately, this is an empirical question that we will explore below.[9]

Reduced Form and Econometric Specifications

The model predicts that, in every period of life, increased life expectancy at the start of life will increase accumulated human capital. To test this proposition or the other reduced form relationships suggested by equations (13A-D), it is natural to conceptualize decisions at the start of life. The expected paths of goods consumption, educational goods investments, time allocations to human capital, leisure consumption, and planned accumulations of assets and human capital in every period will be based on available information on the exogenous variables at time 0. The exogenous variables include the rates of interest (*r*) and human capital depreciation (δ), the price of human capital inputs (*P*), the rental rate of human capital (*R*), the endowments of human capital and assets (H_0, A_0), and life expectancy at birth (*T*).

[9] This is Goldin and Katz's (2010) race between technology and education. If the supply of human capital outpaces the development of skill-using technologies, returns per unit of human capital will fall.

We can also estimate the expected shadow values of assets and human capital.

Because $g(t) = g(0)\left[1 - e^{(\delta+r)(t-T)}\right]$, the value of $g(t)$ is defined at the start of life by known values R, δ, r, T for every $t \epsilon [0, T]$. A change in T increases the projected lifetime wealth at the beginning of life, causing the marginal utility of wealth at birth, $\lambda(0)$, to fall. That increases $g(0)$, the value of human capital relative to wealth at the start of life, and this increase in $g(0)$ increases all the subsequent values of $g(t)$ $\forall t$.

At the time of birth, the individual can set the optimal trajectory of the human capital stock at every point in the life cycle based on information available at that time Ω_0 :

$$E[H(t)|\Omega_0] = f^H\left(r, \delta, P, R, H_0, A_0, T, g(0)\right) \forall t \in [0, T]. \quad (14)$$

The specification suggests that endogenous decisions on completed schooling in adulthood can be regressed on information known at the time of birth.[10] This can be viewed as the lifetime education equivalent of a Frisch labor supply equation of the sort advanced by MaCurdy(1981). The difference is that we are interested in a lifetime choice of time investment in, and the realized accumulation of human capital. The MaCurdy context was to analyze the time allocated to labor supply in one year, but in the context of a lifecycle choice framework.[11]

The reason equation (14) can generate valid inference is that, absent any unexpected shocks to any of the variables on the right-hand-side, the sequence of *g(t)* is perfectly predicted by information available at time 0. Unanticipated shocks to the exogenous variables in equation (14) will cause the individual to reconsider investments and the sequence of $g(t)$ evolves. Critically, however, the new information will be orthogonal to the information set Ω_0. As a

[10] The same reduced form variables enter the decisions for lifetime consumption, labor supply, leisure demand and earnings.

[11] See Blundell and MaCurdy (1999) for a review of alternative estimation strategies to estimate life-cycle time allocation decisions.

result, changes to the expected sequence of $g(t)$, and the resulting changes in the realized values of $H(t)$ compared the expected value at birth, will be uncorrelated with Ω_0. Regressing completed lifetime schooling on information known at the time of birth, including life expectancy, will generate the link between length of life and years of education.

A second strategy is to examine how schooling changes in response to innovations in life expectancy. For example, suppose at time *t',* the individual finds out that life expectancy has changed from *T* to *T'.* The individual will re-optimize, including new values of the $g(t)$ sequence from *t'* through the end of life at *T'.* However, $E[\mathcal{g}(t) - g(t)|\Omega_0] = 0 \; \forall \; t > t'$ where $\mathcal{g}(t)$ represents the re-optimized sequence of relative shadow values of human capital to assets. That means that changes made to planned sequences of human capital investments, labor supply, and lifetime consumption paths from the plans made at time 0 will be uncorrelated with the values of the exogenous variables at time 0, including the life expectancy at birth. The effect of life expectancy on schooling can be determined by regressing the change in planned schooling, $H(T') - H(T)$ on the change in expected life expectancy, $T' - T$.

One question that has appeared in the literature is whether to use life expectancy at birth or life expectancy at an older age (. Life expectancy at birth reflects known potential time before parents make decisions on the goods and time investments of their children in human capital. These decisions will alter the realized life expectancy of their children. As a result, life expectancy at time *t,* conditional on living until time *t,* will endogenously reflect parental decisions. Any regression of years of schooling on life expectancy at ages older than birth will be subject to the endogenous choices that affect life expectancy.

3. Estimation

Direct estimates using observations on T

The most direct estimations strategy used to test the hypothesized relationship between life expectancy and schooling is to employ direct estimates of T in (14). However, estimation is sensitive to the inclusion or exclusion of other covariate controls. For example, Acemoglu and Johnson (2007) examined the effects of increased life expectancy due to improved control of infectious disease on schooling. They argued that population growth induced by the decrease in mortality outpaced any increase in human capital or related productivity, leading to a decrease in per capita incomes. A reanalysis by Bloom, Canning and Fink (2014) found that Acemoglu and Johnson's findings reverse when controls for initial health, H_0 are added. Cohen and Leker (2014) also found that the link between life expectancy and schooling is decidedly positive when increases in life expectancy are tied to the specific cohorts affected. As the theory suggests, changes in life expectancy at birth are tied to the affected birth cohort as opposed to the overall population aged 15 and above.

Hazan (2009) took a different approach, using the Ben Porath (1967) model to argue that lifetime labor supply would have to increase if an increase in life expectancy would increase human capital investment. He found that American men born between 1840 and 1970 reduced lifetime labor supply as life expectancy rose. However, the Ben Porath model does not allow leisure to respond to life expectancy, a restriction relaxed by Heckman's (1976) formulation. As shown by Hoque et al (2020), lifetime leisure (and hence, lifetime labor supply) could increase or decrease as life expectancy rises, and so the Hazan test is not definitive.

A second paper by Hazan (2012) argues that life expectancy should be measured at age 5 rather than at birth because most of the increase in life expectancy in the poorest countries is due to declining infant mortality. Differences in life expectancy between rich and poor countries at older ages are much smaller. The argument is curious on the surface, as it suggests that tests of

changing life expectancy should be based on measures that have less variation, but that potentially lowers the correlation between life expectancy and schooling. Indeed, there is no correlation between changes in life expectancy and changes in average schooling for the population aged 15 and older, in contrast to the positive and significant correlation with changes in life expectancy at birth. The more substantial concern, however, is that the theory shows that changes in life expectancy at birth are plausibly exogenous while changes after birth will reflect the endogenous investments of parents in their children, conditional on information at birth. Hence, life expectancy at older ages will increasingly reflect endogenous health choices rather than exogenous changes in longevity.

The remaining papers have all found strong positive effects of rising life expectancy on schooling. Cervellati and Sunde (2013) show that the Hazan (2009) condition that rising labor supply is a requirement for a positive link between life expectancy and schooling does not hold when there are age-specific increases in survival rates. Their empirical evidence shows that rising life expectancy, especially in the working ages, increases years of schooling.

Several papers address the issue of endogeneity in life expectancy. Jayachandran and Lleras-Muney (2009) used improvements in maternal health to instrument for presumed endogenous life expectancy for girls whose life expectancy rose relative to boys. They showed that affected girls experienced 0.11 years of added schooling for every additional year of expected life. Hansen (2013) used the incidence of various diseases to instrument for life expectancy. He found that increased life expectancy was particularly affected by falling pneumonia mortality, and that affected children experienced 0.17 years of added schooling for every added year of life expectancy.

Hoque et al (2019) examined how life expectancy for different birth cohorts within a country affected completed schooling across 919 different household surveys. Their estimates showed that when the measure of life expectancy is tied specifically to the cohort affected, estimates are quite robust to the other issues raised by various researchers. For example, the effect of life expectancy on years of schooling differed only slightly when life expectancy was measured at birth, at 5 years, at 10 years, or at 15 years. In Figure 5, we show the cumulative distribution of their least-squares estimates representing 111 different countries. Nearly all (95%) of the estimates are positive with the mean estimate of 0.15 located between the estimates reported by Jayachandran and Lleras-Muney (2009) and Hansen (2013). Of the 5% that generated negative estimates of life expectancy on schooling, only three were statistically significant. Estimates using infant mortality to instrument for life expectancy were somewhat larger at 0.21 years of schooling per added year of life expectancy.

One additional finding is that increases in life expectancy of the parents translate to increased schooling of their offspring, holding constant the life expectancy of the children. This is consistent with the more common finding of intergenerational persistence of schooling (Becker and Tomes, 1986; Lindahl et al, 2015). The added benefit of the parents' longevity on their children creates a persistence in growth of human capital from the initial surge in life expectancy.

Fritsch equations

An alternative specification, following the work by MaCurdy (1981), would be to utilize longitudinal data on educational choices. The specification would control for the conditions at the start of life by using individual fixed effects. One could then use innovations on life expectancy to measure the changes in schooling. In the MaCurdy context, innovations in wages

could plausibly be viewed as exogenously influencing labor supply. The problem with this context is that the innovations in life expectancy are almost entirely going to be due to endogenous choices that influence health, and so the Fritsch strategy will require specialized data.

One such setting is when individuals receive exogenous information on length of life. Although not technically a Fritsch equation, the paper by Oster et al. (2013) provides an example of how exogenous innovations in life expectancy affect changes in schooling. It examines the schooling decisions of individuals who were tested for Huntington's disease, a fatal inherited disease that lowers life expectancy and reduces effective work life. The study shows that individuals who received the test information that they do not have the disease increased their schooling by 0.17 years for every additional year of expected life associated with the timing of the test.

Bhalotra and Venkataramani (2012) showed that the sudden availability of sulfa drugs to combat pneumonia resulted in a sharp decrease in mortality rates after 1937. Individuals experienced a reduction in death rates due to pneumonia equivalent to a drop from the 75th to the 25th percentile due to the availability of sulfa drugs at the time of their birth. These individuals increased subsequent schooling and income, consistent with a positive human capital response to unexpected increases in life expectancy. Lifetime health also improved, and so the increase in human capital is due, at least in part, to a greater capacity to learn and work that is associated with improved health.

Endogenous life expectancy

We have argued that studies should use measures of life expectancy at the time of birth because measures conditional on attaining higher ages will reflect endogenous choices by

parents. One of the first papers to explore infant mortality from the perspective of an economic model was Rosenzweig and Schultz (1982) who showed that Indian households favor boys or girls in household resource allocation and survival in areas with fewer market opportunities for women. Since then, numerous studies have explored the phenomenon of "missing girls" in India and other countries.

For example, Bhalotra et al. (2020) showed that, when gold prices rose, thus raising the expected cost of a dowry, neonatal and infant mortality rates for infant girls rose relative to boys in states where dowries are traditional. Bharadwaj and Lakdawala (2013) found that Indian mothers were more likely to have prenatal medical checkups and tetanus vaccines when the fetus was a boy, especially when their existing children were all girls. Differences in prenatal tetanus shots explains 2.6 -7.2 % of the difference in female mortality. Chen, Li and Meng (2013) showed that the ratio of boys to girls in China rose fastest under the one-child policy in provinces with the earliest access to ultrasound technologies, making it easier to use selective abortions to favor boys.

In section IV, we review the broader literature that examines how endogenous health behaviors, and resulting health outcomes such as mortality risk and life expectancy are influenced by education.

The effect of improved health early in life

As the Bhalotra and Venkataramani (2012) case indicates, it is not simple to distinguish between increases in life expectancy and improvements in health at the time of birth. While this Chapter focuses on changes in *T,* we cannot ignore the much larger literature that explores the effects of health endowments on lifetime schooling. We will focus on examples of papers that examine the effects of exogenous changes to health early in life on human capital production.

One line of analysis hearkens back to the original work by Robert Fogel that emphasized the role of nutrition and the agricultural revolution in the initial surge of life expectancy. Many different sources of shocks to child nutrition have been explored, including randomized placement into a nutritional supplement program (Maluccio et al., 2009); exposure to iodine supplements in utero Improvements to fetal or infant health such as maternal malnutrition (Field et al., 2009); and prenatal exposure to mothers fasting during Ramadan (Almond and Majumder, 2011) all had negative repercussions for schooling. Exposure to excessive or insufficient rainfall during the first year of childhood leads to malnutrition (Maccini and Yang, 2009; Shah and Steinberg, 2014) that also results in persistent adverse outcomes later in life. Children with low birthweight complete less schooling (Behrman and Rosenzweig, 2004). A recent analysis using developed country data (Maruyama and Heinesen, 2020) showed that birthweight provides protection against child mortality and other adverse health consequences associated with the diagnosis of placenta previa. While the added health consequences from high birthweight were not significantly correlated with better test scores, it is associated with earlier completion of the 9th grade and with higher labor force or schooling participation at age 24. The survey of the evidence by Almond et al. (2018) concluded that low birth weight is consistently associated with inferior adult outcomes in schooling, health, and earnings.

Another line of research focuses on exposure to illness early in life. Bleakley (2007, 2010a) and Lucas (2010) exploited variation in the timing and intensity of hookworm and malaria eradication in the American South and across developing countries in Latin America and Asia to demonstrate that the populations with the earliest access to treatments experienced improved education and earnings. *In utero* exposure to the Spanish flu also had permanent negative effects on completed schooling and adult earnings (Almond, 2006). Similarly, Miguel

and Kremer (2004) found that Kenyan children who received treatment for intestinal worms were absent 25% less frequently than students in the schools that were randomly assigned to receive the treatment 2-3 years later. Follow-up surveys of these same students revealed that compared to those in the control schools, those students in the treatment schools worked 13% more hours and earned 20-29% more (Karlan and Appel, 2011).

A third line of research examines how exposure to environmental pollution or hazards affects child health and schooling outcomes. Chay and Greenstone (2003) showed that a reduction in air pollution lowered infant mortality during the U.S. 1981-82 recession, with the greatest reductions from fetal exposure. Similar findings were reported by Foster et al. (2009) for Mexico, and Jayachandran (2009) for Indonesia. Almond et al. (2009) showed that Swedish children exposed to greater fallout from Chernobyl suffered loss of cognitive function. The Almond et al. (2018) survey concluded that the adverse effects of pollution are particularly large on a developing fetus.

All of these studies suggest that the link reported earlier between life expectancy and increased schooling is likely due to two margins. Longer life means increased time to recoup the investment in schooling, and improved health leads to greater productivity in the production of human capital per year.

4. Reverse causality: How education affects health and life expectancy

The relationship between life expectancy and education also goes in the other direction, reflecting a virtuous cycle in which health status and longevity cause education and education affects health status and longevity. Although this is not precisely what the endogenous growth theorists had in mind about the endogenous growth mechanism (Romer, 1986; Pack, 1994; Lucas, 2002), this two-way relationship exemplifies how initial advances in human capital

sustain future improvements in education and health through their effect on productivity, innovation and economic growth, which in turn increase the returns to human capital and spurs even more investments in human capital development.

This section reviews the studies that have estimated the influence of education on health and life expectancy. Most of the studies have focused on the U.S. and Europe (e.g., Elo and Preston, 1996; Currie and Moretti, 2003; Cutler and Lleras-Muney, 2006, 2010), but many have also examined education and health in low- and middle-income countries (e.g., Vogl, 2012; Burchi, 2012; Casale, Espi and Norris, 2018; Sudharsanan et al., 2020). Together, these studies help to understand the different pathways through which education affects health status and life expectancy, and in particular, the nonlinearity of this relationship and the critical role of maternal education on the health of the next generation.

Preston (1975) first showed that there was a strong positive relationship between per capita GDP and life expectancy. Over time, life expectancy rose at all income levels, a shift in life expectancy that Preston related to improvements in health technologies that raised life expectancy at all incomes. However, Lutz and Kebede (2018) recast the Preston analysis as a relationship between education and life expectancy, arguing that education raises both incomes and health. They show that the relationship between education and life expectancy is much stronger than between life expectancy and GDP per capita. In 1996, Elo and Preston estimated the percentage decrease in mortality per year of schooling for men and women aged 35-54 years in the U.S. and several European countries. An additional year of schooling lowered mortality rates by an average of 8.2% for men and 5.4% for women. College graduates have the lowest mortality rates and high school dropouts have the highest. The pattern suggests that mortality rates reflect the hazards associated with different occupations. More educated men tend to have

much less risky occupations than less educated men. Women have less risky work than men at all education levels, so women have a smaller reduction in mortality as education increases.

We illustrate the Preston Education Curves for 1960 and 2010 in Figure 6 which shows a positive correlation between adult education and health. The education measures reflect the average schooling completed for adults aged 25-65, and life expectancy at birth is information on the current birth cohort. In 1960, life expectancy ranged from a low of 28 in Mali to a high of 73 in Norway. By 2010, the range of life expectancies had risen from 45 in Lesotho to 83 in Japan, and the education-life expectancy plots have shifted up and to the right, indicating that education levels had also risen. Gains have occurred at all education levels, but the largest gains in life expectancy are at the lower tail of the education distribution, suggesting that education gains have been particularly important for improving the health in the countries with low initial life expectancy and education.

[Figure 6 about here]

While country plots are informative, they can mask true causal effects. We now turn to studies have examined the relationship between schooling and health at the individual level. Cutler and Lleras-Muney (2006) estimated the relationship between years of schooling and life expectancy, and posited that this relationship is nonlinear. They used a non-parametric estimator to measure the *education-health gradient,* the increasing effect of each year of schooling on measures of health status.[12] They find that the relationship between education and mortality is

[12] Cutler and Lleras-Muney (2010) use several self-reported measures of health such as mortality and incidence of various diseases in the U.S., and a set of control variables that include age, gender and race. They find that more highly educated persons are less likely to die within five years after the survey and have lower morbidity from the most common acute and chronic diseases, such as cardiovascular diseases and diabetes, and have better physical and mental functioning.

negative and linear after 10 years of schooling, but that there are no additional health benefits associated with the completion of a degree. Other studies that have estimated the education-health gradient also find that the gradient has become steeper in recent decades, consistent with the convex shape of the 2010 trend line in Figure 6. In 1980, at age 25, those with some tertiary education could expect to live another 54.4 years compared to 51.6 years for those with only a high school degree or less. By 2000, this gap of 2.8 years had increased to seven years (Cutler et al., 2008, 2012). Estimates for men and women born in 1905-15, some 40-50 years earlier, suggest that an additional year of schooling added only 0.4 years of life for individuals at age 35 (Lleras-Muney, Price and Yue, 2020).

Everett, Rehkopf, and Rogers (2013) explored another dimension of the education-health gradient – how the relationship differs across demographic groups of adults aged 35-65 years, and how this pattern has changed over time (that is, for birth cohorts 1921–1936, 1937–1953, and 1954–1969). Using the U.S. National Health Interview Survey-Linked Mortality Files, which link data from 1986 through 2006, they estimate the gradient by cohort, race/ethnicity, and gender. Overall, they find substantially nonlinear relationships between education and mortality risk that differ by race and gender. Among white men, for example, there has been a slow decline in the education–mortality relationship between 0 and 9 years of education, after which the mortality risk declines more steeply and crosses the mean risk at 13 years of education. This education–mortality slope becomes even steeper for younger birth cohorts, but less so for black men than for white men. Among white women, the education–mortality curve in the oldest cohort is relatively flat at less than eight years of schooling but it decreases more rapidly from 8 to 14 years of education before leveling off. As for white males, the education–mortality gradient for white and black women is steeper for younger birth cohorts.

At least two factors may have contributed to these black-white differences. Everett, Rehkopf, and Rogers (2013) show that the mortality gap between the lowest and highest levels of education is higher among white men than among black men. Among black men, higher levels of stress and discrimination are said to be correlated with cardiovascular health and thus with mortality risk, undermining the benefits of education for them. In addition, the quality of education has improved more in largely white communities than in largely black communities, and this difference has resulted in unequal returns to similar education levels in these communities.

Several studies have measured the education-health gradient in other countries, both developed and less developed. Cutler and Lleras-Muney (2006) show that the positive and nonlinear relationship between years of education and life expectancy is evident using the average years of education and life expectancy at birth in 138 countries. In Spain, Permanyer et al. (2018) find that life expectancy has been increasing for all education groups but more so for highly educated people. As a result, differences in life expectancy between the low- and highly-educated have increased, particularly among men. Sudharsanan et al. (2020) estimate the education-health gradient using harmonized longitudinal data from China, Costa Rica, Indonesia, Mexico, South Africa and South Korea by age group and gender. In all six countries, they find that only adults with tertiary schooling have a mortality advantage compared to those with no schooling, but this conclusion differs by gender in some of the countries. For example, among Indonesian women and Mexican men, even those with tertiary schooling do not have a mortality advantage relative to those with no schooling. The gradient is steeper among South African men than women, and among men than women at all education levels in South Korea.

What accounts for the positive and nonlinear relationship between education and health and life expectancy? What are the pathways through which education affects health? One obvious pathway is that more education is positively associated with the capacity to earn a higher income, which in turn buys health care and a less risky lifestyle.In other words, education shapes the likelihood of being unemployed and one's occupation, including the probability of working in a safe or less safe environment (von dem Knesebeck, Verde and Dragano, 2006). Another pathway is that persons with higher levels of education are better able to acquire and process health information, formulate more appropriate responses and decisions, and change their health behaviors accordingly. In addition to the indirect effect of education through higher incomes, more highly educated persons are less likely to smoke, and are more likely to exercise and to have better nutrition, all of which are associated with better health. Cutler and Lleras-Muney (2010) find that two-thirds of the education gradient is due to differences across education levels in access to information, material resources, cognition, and social interactions, and that the monetary value of the resulting difference in health lifestyle is about half of the return to education on earnings.

Many studies have also focused on the intergenerational effect of education on health outcomes, specifically the effect of maternal education on children's health. Regressions of child health on household wealth and maternal education in 42 developing countries show that higher education levels are significantly associated with better child health, although there is substantial variation across countries in both the strength and form of this association (Boyle et al., 2006). Mother's education is especially important in the case of at-risk children in households with low income and limited access to public services. This health return to education comes through different channels: more highly educated mothers tend to have better nutrition knowledge which

result in less stunting, as in the case of Brazil (Thomas, Strauss and Henriques, 1991) and Mozambique (Burchi, 2010); to be more open to modern health care such as prenatal care and vaccination programs, as in the case of the U.S. (Currie and Moretti, 2003), Indonesia (Streatfield, Singarimbun and Diamond, 1990), South Africa (Casale, Espi and Norris, 2018) and Nigeria (Burroway and Hargrove, 2018); to know how to protect their young children from the adverse effects of poverty and poor public services (Behrman and Wolfe, 1987; Dargent-Molina et al., 1994; Pena, Wall and Persson, 2000; Urke, Bull and Mittelmark, 2011); and to adopt healthier behaviors themselves, such as to not smoke (Currie and Moretti, 2003)

That both education and health could be determined by unobservable factors that are correlated with both education and health, and that, as discussed in the previous sections, education decisions are indeed affected by life expectancy put into question the interpretation of the coefficient of education in these studies as causal. Several studies have used different strategies to address this issue. Recognizing the endogeneity of education, Currie and Moretti (2003) use data on the availability of colleges in a woman's county at age 17 as an instrument for education in their analysis of the effect of maternal education on infant health in the U.S.. In justifying this choice of instrument, they show that maternal education and birth outcomes do not have a clear relationship ten years before the college availability, and that maternal education increases if the college availability goes up by age 17 and not at age 25 and above. Arendt (2005) uses Danish school reforms to instrument for schooling in a study of how education affects three health outcomes (self-reported health, body mass index, and an indicator for never having smoked). Illustrating the difficulty of choosing instruments, the study finds a significant relationship between education and only smoking behavior. Using a different approach to establishing the causal effect of education on health, Galama, Lleras-Muney, and Van

Kippersluis (2018) review the studies that have used randomized controlled trials, twin studies, and quasi-experiments to examine the causal effect of education on mortality, obesity, and smoking behavior. In particular, they examine the effect of institutional reforms, considered exogenous to household and individual behavior and are exploited as quasi-experiments in many empirical studies. Those institutions can regulate prices, wages, whether youth are supposed to be in school at a certain age, and the penalties associated with not conforming to such laws. The authors also argue that schooling and skills are two different concepts, a proposition that has gained more supporters (e.g., World Bank, 2018), and that it is not merely the number years of schooling that is important in changing health and life expectancy but also the quality of education.

5. Conclusion

Substantial public and private investments in health and education have been critical for the industrialization and economic growth in today's advanced economies countries, and they are key to achieving human capital development and growth in developing countries. Improving health and education also have significant , We have reviewed historical and econometric evidence of the linkage between longevity and education, and argue that the linkage goes both ways: that the initial increase in life expectancy eventually led to the rise in demand for schooling for children, and that increased education led to improved investments in health and longevity. This virtuous cycle that leads to the growth of both human capital and health can be viewed as one particular mechanism underlying the analysis of endogenous growth and the rise of cities.

In the Lucas (2002) endogenous growth formulation, the stock of human capital per person generates an external productivity effect for all members of the society, above and beyond their own human capital stock. This externality means that production could exhibit increasing returns to scale, even if we have diminishing returns to individual investments in capital and skill. The mechanism that generates endogenously growing stocks of human capital is an aggregation of the individual production of human capital we defined in equations (1) and (2). If the production of human capital, $H(\dot{t})$, depends on its current level, $H(t)$, and if the initial stock of human capital, H_0, is proportional to the parents' human capital, we can have a permanently growth in per capita income fueled by rising per capita human capital stocks. Increasing life expectancy with its associated greater accumulation of human capital per person and greater accumulation of knowledge from experience would be one way that both individual productivity and external productivity would grow.

An implication of the Lucas model of growth is that educated workers will have an incentive to concentrate in proximity to one another. An individual's skills are more valuable when combined with other skills, leading to the rise of cities (Glaeser, 2008). Worldwide, growth has led to urbanization with increased concentration of populations and production in urban areas. As demonstrated by Moretti (2004) using a matched data set of firms and workers, plants in cities experiencing rising shares of college graduates experiencing faster productivity growth than do similar plants in cities with smaller growth in educated labor. This pattern or urbanization reinforces the gains in per capita income attributable to the steadily rising education per person and its external productivity effect when the human capital is agglomerated in cities.

In the Lucas model, endogenous economic growth leading to a steady increase in per capita incomes only happens if individuals apply effort to the acquisition of additional human

capital. The Malthus steady state at subsistence is consistent with the lack of time allocation to human capital investment. The launch to the new endogenous growth state required a perceived gain from human capital investment, and that occurred when the increased caloric intake made possible by the agricultural revolution, improved transportation, and the development of national commodity markets allowed a lifespan sufficiently long to promise a payoff to human capital investments. Only then, in the Lucas model, would the conditions for permanent growth have become possible.

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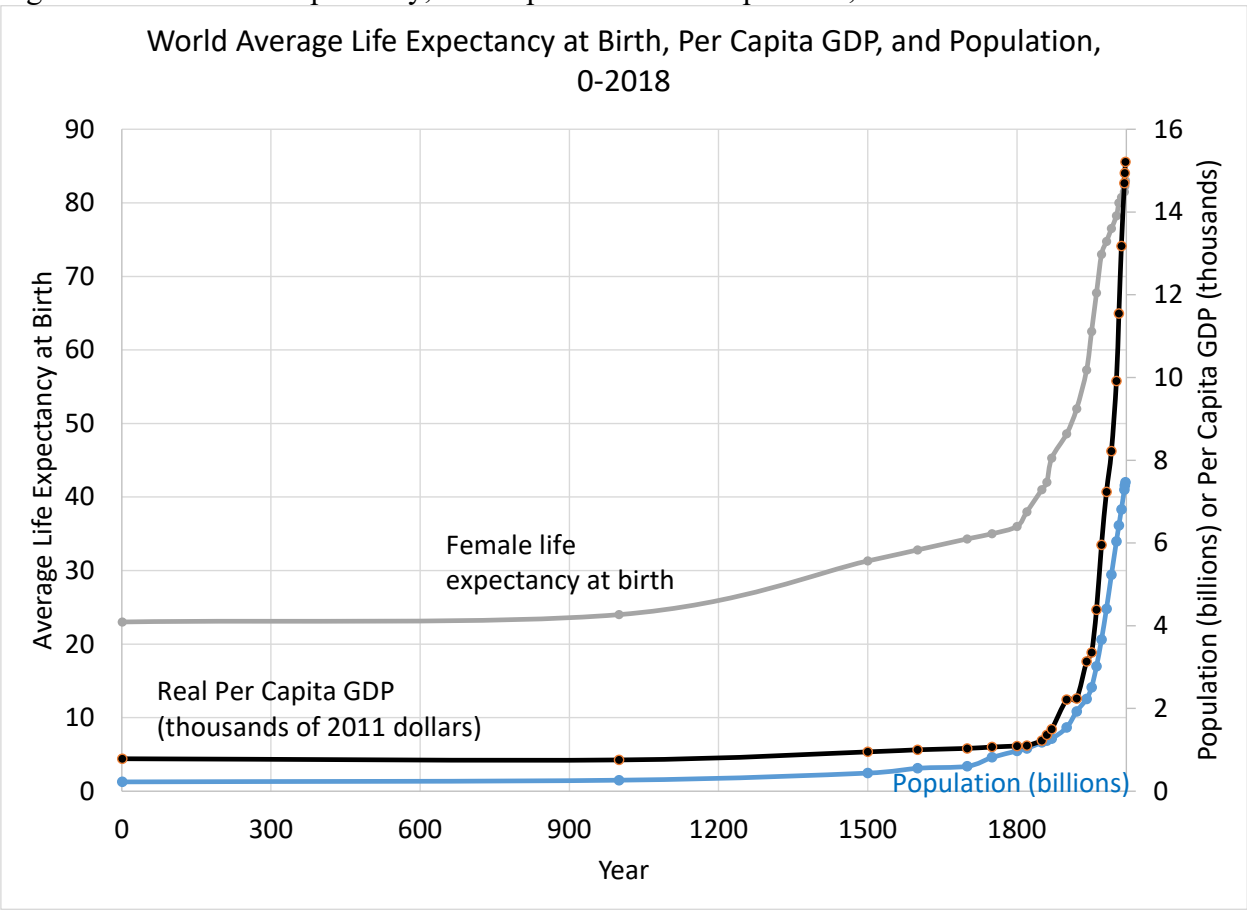
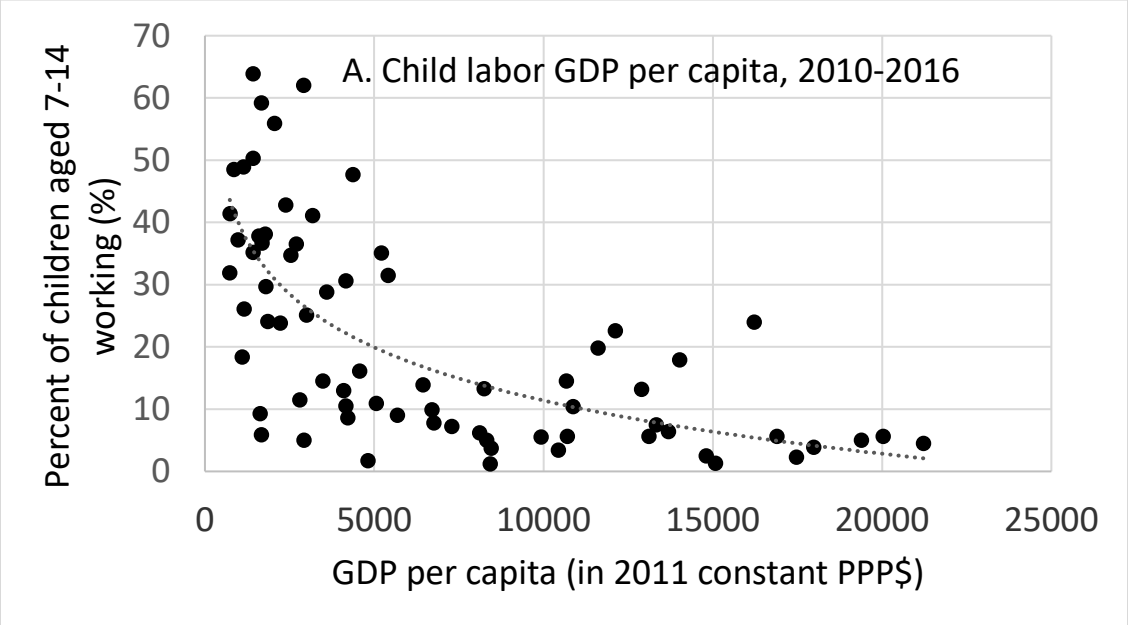
Figure 1: World Life Expectancy, Per Capita GDP and Population, 0 - 2018



Source: Authors compilation of data from Bolt and van Zanden (2020), Maddison (2007), and Vallin and Meslé (2009). Female life expectancy avoids reduction in life expectancy related to wars that generate misleading patterns of expected life span for men.

Figure 2: Per capita GDP, life expectancy and the incidence of child labor by country, various countries in 2016.



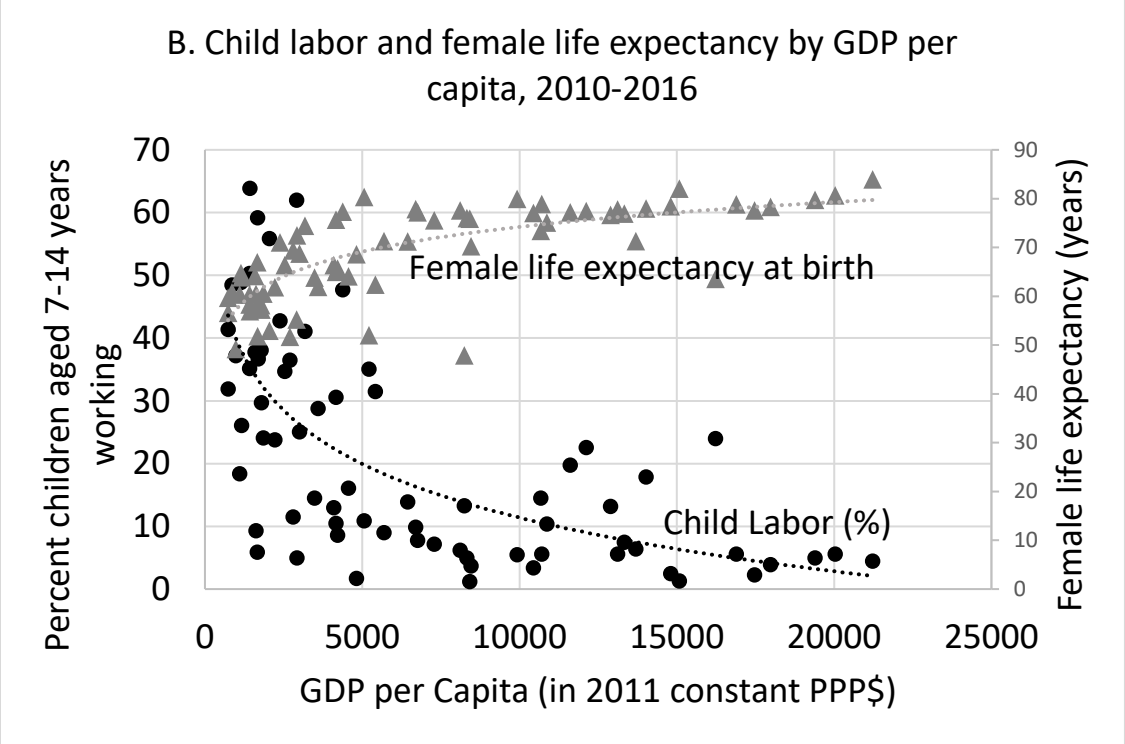
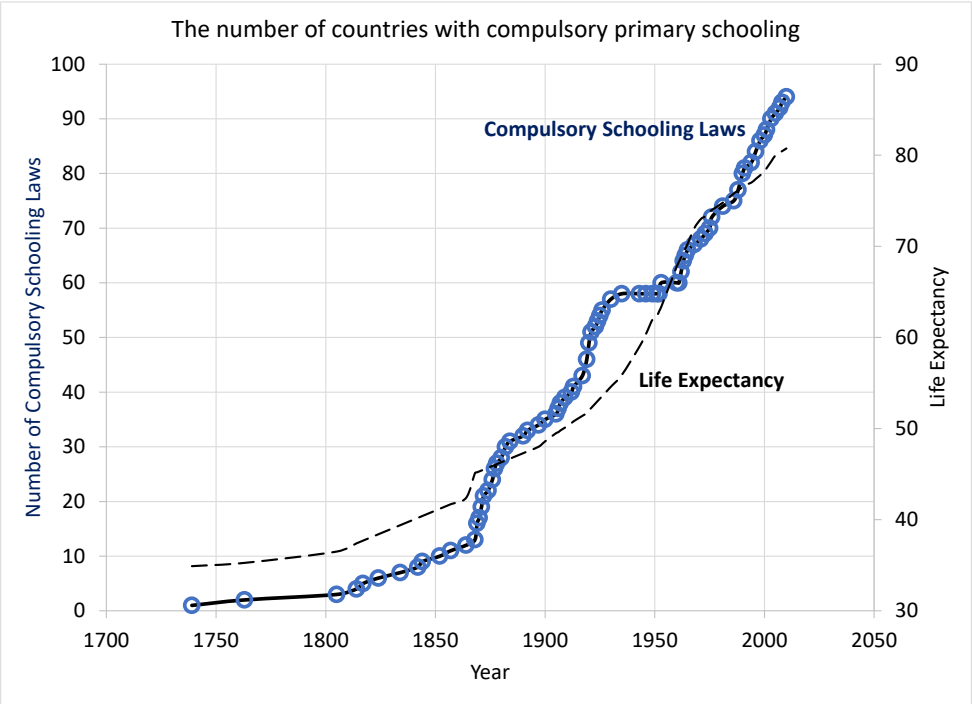


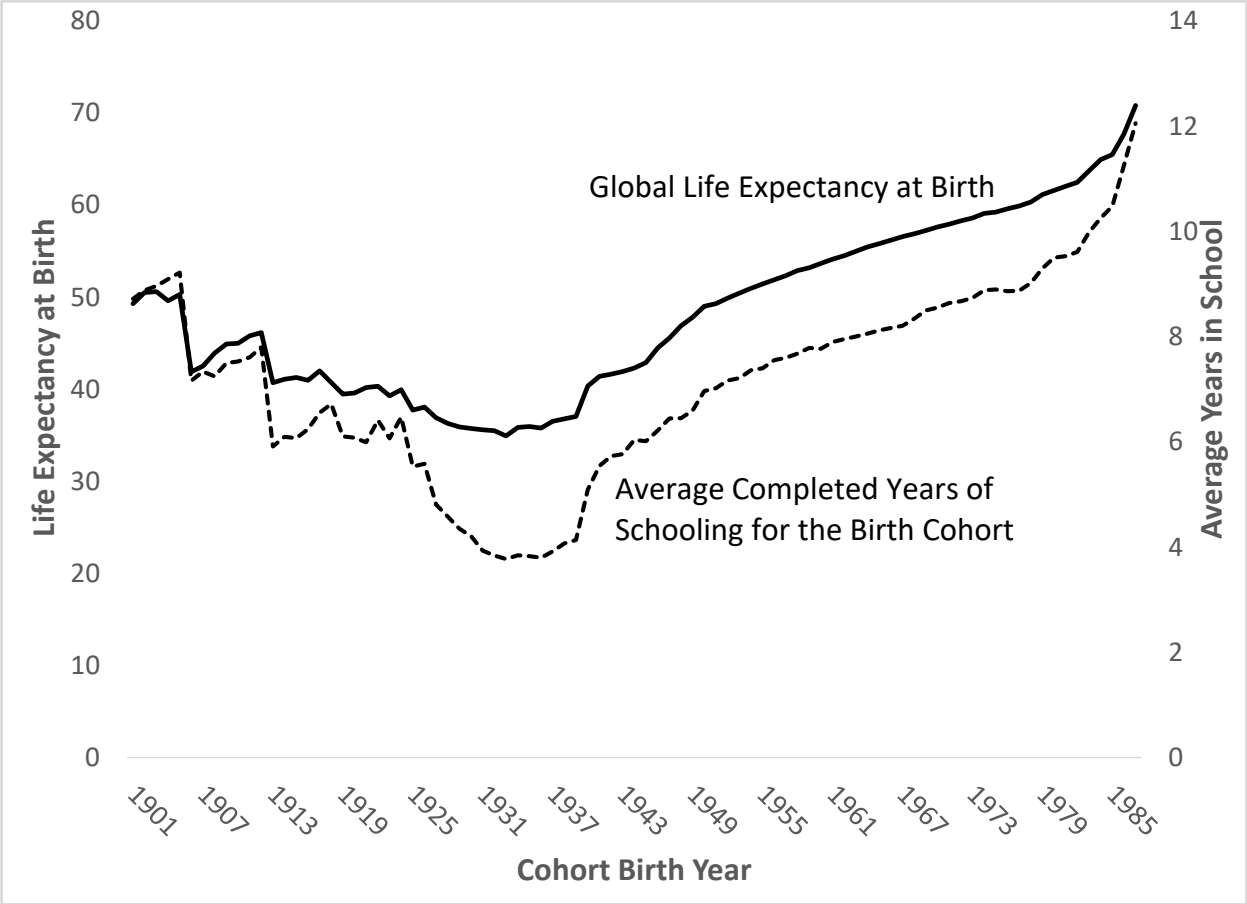
Figure. Relationship between child employment rates by economic activity and life expectancy
Data source: Data sources: World Development Indicators (World Bank, 2021); ILO Statistics
Notes: Child labor participation rates are the latest data during the period 2010-2016; female life expectancy rates are data in 2016. These data are matched with GDP per capita levels (in 2011 constant PPP dollars) for the same years.

Figure 3: Number of Countries with Compulsory Primary Schooling by World Life Expectancy at Birth



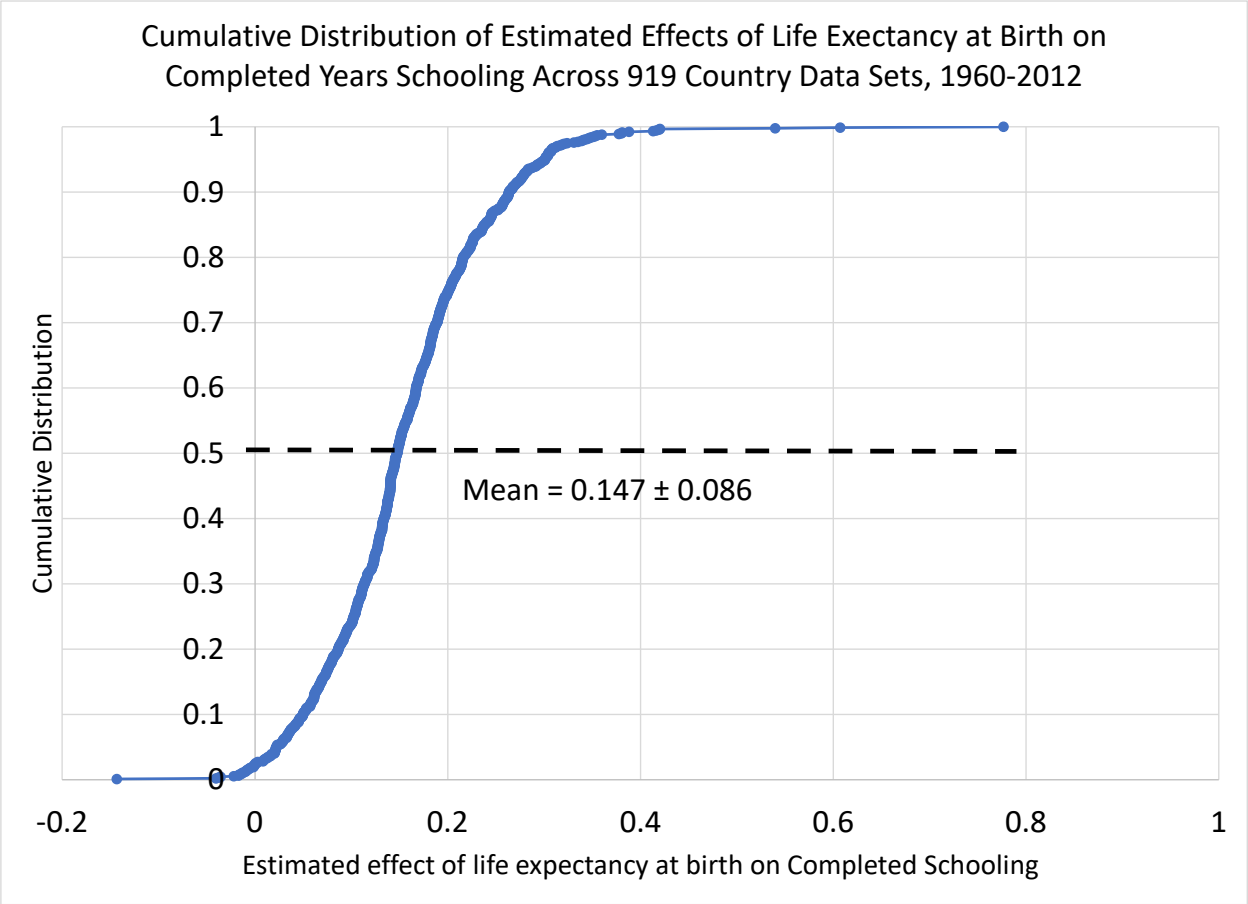
Source: Life expectancy from Bolt and van Zanden (2020), Maddison (2007), and Vallin and Meslé (2009). Date of compulsory schooling law from sources too numerous to list.

Figure 4: Life expectancy and completed years of schooling by birth cohort, World Average



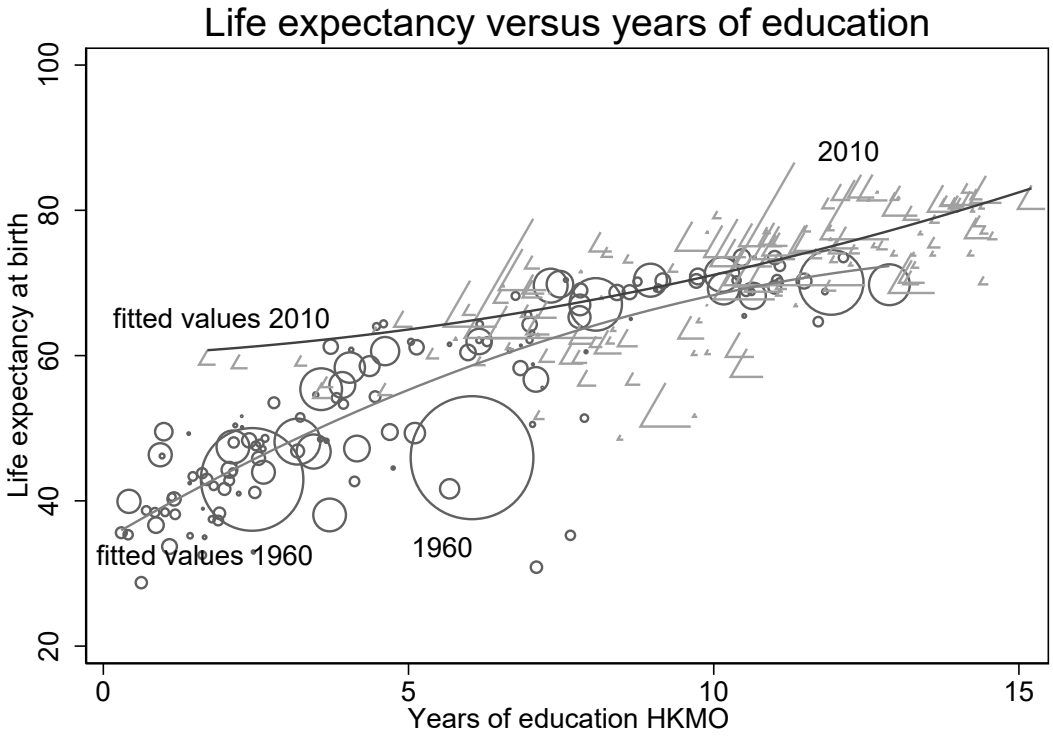
Source: Author's computations based on household data from 111 countries included in the World Bank's International Income Distribution Database

Figure 5: Cumulative distribution of estimated effects of life expectancy on completed schooling



Source: Estimated coefficients from 919 household data sets reported in Hoque et al (2019).

Figure 6: Preston Education Curves, 1960 - 2010



Source: Authors' estimates of life expectancy at birth were taken from the World Bank's World Development Indicators. Average schooling for individuals aged 25-65 was computed from household surveys used in Hoque, King, Montenegro and Orazem (2019, HKMO) for each country. The 1960 estimates are taken from available household surveys collected from 1960-1965. The 2010 estimates are taken from household surveys collected from 2011 – 2015. Symbol sizes reflect the country population.