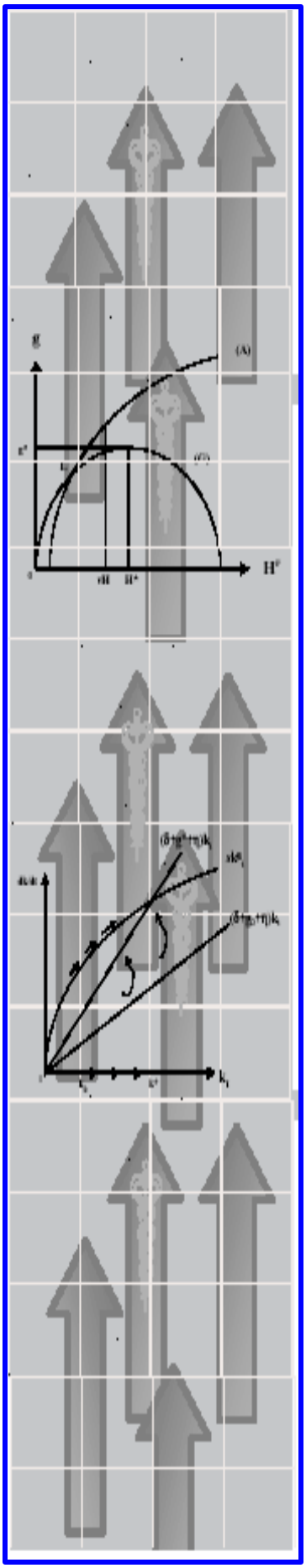

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Health, Human Capital and Economic Growth

On Epidemiologic and Economic Transitions: A Historical View *

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On Epidemiologic and Economic Transitions: A Historical View

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Based on data since the mid-nineteenth century, this article reexamines existing propositions of the Epidemiologic Transition. The data reveal perceptible health-related changes germane to human capital formation. At the same time, human capital frameworks provide the basic tools for studying long-term epidemiologic change. The synergy between epidemiologic change, human capital formation, and an economic transition from the Malthusian regime produces some evidence that health improvements have permanent long-term social consequences.

The rise of human capital since the nineteenth century is unique to history. Several theories posit that by permanently increasing the pace of economic growth it stimulated an economic transition from the Malthusian regime (Lucas 2002, Galor and Weil 1999). However, human capital in the form of skills has been their main emphasis. Few studies have tried to highlight the relevance of health to human capital formation and to growth.

This article tries to narrow the gap by uncovering a synergy between two fields of research: Economic Growth and the Epidemiologic Transition. The Transition refers to long-term change in the rate and composition of disease in the population.

Both fields assign a pivotal to human capital. However, they examine different but inseparable aspects of it. Growth theory lays emphasis on skills; whereas the Transition emphasizes health.

Moreover, escape from the Malthusian era is a central theme to both areas of research: skill invigorates an economic transition from the Malthusian regime to modern economic growth; whereas health invigorates an epidemiologic transition from the Malthusian regime to the modern era. Furthermore, the onset of both transitions in industrialized countries can be traced at least to the nineteenth century. Since they share a common theme in which human capital formation plays a central role, a simultaneous inquiry into epidemiologic and economic transitions may benefit both fields of research.

Studying both transitions simultaneously, however, constrains the choice of countries because reasonable data for only a few industrialized countries extend sufficiently into the past. Pertaining to hundreds of diseases, the data reveal perceptible epidemiologic changes germane to human capital

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formation since mid-nineteenth century. In turn, existing human capital models offer a framework relevant to the study of epidemiologic change and its long-term economic influence. This synergism allows an empirical evaluation of human capital-based growth theory.

The next section argues that the nineteenth century's unique stimuli enabled households and society to curtail deaths from hundreds of diseases, allowing human capital to surface as a productive force perhaps for the first time in centuries. To uncover the nature of those stimuli, however, we need to examine infectious and non-infectious diseases that comprise the epidemiologic transition.

The Epdemiologic Transition posits that the decrease of death rate from infectious diseases, which dominated the Malthusian regime, coincided with an increase in death rate from non-infectious maladies, which now dominate the modern epidemiologic experience. Several aspects of this view, however, appear to be inconsistent with the data. Existing propositions of the Transition are then refined to match the data and some new ones germane to human capital formation and growth are proposed.

The article then finds that neither current level of per capita GDP nor its growth rate suffices as explanatory variables for epidemiologic change. The results suggest that per capita income is too blunt an instrument for studying epidemiologic change, within and across countries.

On the other hand, intergenerational human capital frameworks provide the basic tools for organizing thought about epidemiologic change and its influence on economic growth. After discussing their relevance, the article tries to evaluate whether diminishing disease-caused fatality, a potent sign of improving human durability, influenced the slope of the growth path permanently—human capital-based growth theory's central prediction. The results indicate that health-related changes have a long reach.

2. Importance of Health to Human "Capital"

Health-related facets of human capital have been largely peripheral to discussions on economic growth. However, there are reasonable grounds for bringing them to the center.

Conceptually, economic theory assigns the term "capital" to factors that have three properties:

First, its durability should be able to function as a "storage technology," which provides benefits over time. It is self-evident that well-functioning organs allow people to work effectively. Few skills would be useful if organs and organ systems were to break down. Durability of organs, which is essential to health, is also essential to human capital.

Second, the factor should be alienable: like private property, the returns accrue to the owner (or to the owner's immediate family). Besides being self-evident, returns to health have been documented in several empirical studies (Grossman (1972), Bartel and Taubman (1979), Schultz (1997), Schultz and Tansel (1996), among others).

The third property that makes a factor "capital"—and the one most relevant to emergence of conventional forms of human capital, historically—is that its accumulation must entail some form of

current sacrifice for future benefit. Accumulating human capital—schooling, work experience, on-the-job training—requires a sacrifice of time, often years.

If it takes sacrifice of years to accumulate skill and experience, then it is informative to find out how years available to an average person have evolved historically.

Figure 1a shows that period life expectancy at birth fluctuated between 27 and 41 years for three centuries. It persisted in that range despite economic progress since the industrial revolution in the eighteenth century. Only toward the end of the nineteenth century did it breach that range permanently, the new direction evident in life expectancy at higher ages as well (Figure 1b).

Why would life expectancy increase? Calculation of life expectancy is based largely on disease-caused deaths. Instead of gauging the extent of sickness and deaths from hundreds of diseases annually, life expectancy merely summarizes the consequence in units of years. It follows that lasting declines in disease-caused deaths added years to people's lives. And these declines, disease-by-disease, resonate household and social choices that curtail sickness and deaths.

Curtailing of sickness and deaths is perhaps a potent signal of households' intent to foster human capital. To accumulate skill and experience, people would have tried to ensure that the life-years required for doing so were produced simultaneously. And that would happen only if people had protected their health or corrected health-related disorders. Therefore, accumulating human capital subsumes attending to human durability; otherwise human capital would not be "capital."

In this sense, ascending life expectancy itself signifies improving human capital of the masses. Social changes that triggered its ascent may have generated what Robert Lucas Jr. has called, "opportunities for human capital investment [schooling] that face the mass of households in a society ..." Furthermore, it steers attention toward the possibility that more people were able to school since the nineteenth century because life-years had started growing, increasing with it the present value of such an investment. Therefore, health improvements and rising life expectancy may have been essential to human capital.

Though rising life expectancy summarizes increasing usable time, to uncover the sources of its increase we need to examine disease-related shifts that have occurred beneath the surface—the Epidemiologic Transition.

3. The Epidemiologic Transition

The Transition is a set of propositions about the rate and composition of disease-caused deaths during the Malthusian regime, a transitional period and a post-transitional regime (Omran (1971)).

The Malthusian epidemiologic stage is widely characterized by three central features:

1. A high rate of infectious- and contagious disease persists (e.g. tuberculosis, cholera, influenza, plague, typhoid fever, smallpox, malaria, polio, among hundreds of others). Infectious diseases are a larger proportion of all disease-caused deaths and their outbreaks occur frequently.

2. In contrast, the rate of non-infectious maladies—non-contagious, chronic and degenerative conditions (diabetes, cancer, heart diseases and the like)—is low. Consequently, they account for a smaller proportion of all disease-caused deaths.
3. A high rate of aggregate disease-caused deaths perpetuates low life expectancy.

Then, for various reasons, there begins a transitional phase in which:

1. Infectious diseases start reducing; their share of all disease-caused deaths starts shrinking.
2. Life expectancy starts increasing permanently.
3. However, along with rising life expectancy, the rate of non-infectious-diseases starts increasing as well. Non-infectious diseases surface as leading causes, eventually accounting for the bulk of all disease-caused deaths in the post-transitional regime.

Recently, researchers have proposed a new phase in which death from non-infectious maladies too are being deferred to much older ages, increasing life expectancy among the elderly.[2] Others have noted Malthusian-stage-like features in epidemics like HIV/AIDS and other new drug-resistant microbes and infections. Nevertheless, it helps first to begin with the core outlined above.

To view the transition, annual age-specific deaths from each disease were assimilated into a variable, "All diseases." All disease-caused deaths were then partitioned into two main components dubbed as "infectious diseases" and "non-infectious diseases." Non-infectious diseases were then segmented further into its main components such as cancers, maladies of the circulatory system, digestive system, nervous system, musckuloskeletal system, genitourinary system and so on. External causes (accidents, injuries, homicides etc.) were excluded. For an extensive discussion on proper disease-compositions of each variable, the interested reader is referred to Preston et al. (1972), Preston (1976), D´Espaignet et al (1988) and Vallin (1987), Arora (1999, 2003) for details.

The next section examines main features of the transition in the United Kingdom. Since the data reveal only a single cause of death per person, they only allow statement of *plausible* propositions. Each death may have involved more than one malady but only one of them appeared in the vital statistics. Though the data pertain to the United Kingdom, the trends might be broadly relevant to western industrialized countries.

Infectious diseases

Marked by several significant outbreaks, the contour of infectious diseases shown in Figure 2 indicates roughly three regimes: (1) Pre-1870 dubbed as "Malthusian" regime, (2) A transitional phase, 1870–1940/50, and (3) Post-transitional regime after World War II.

Although reasonable data before 1850 are unavailable, Figure 3 gives the impression that infectious diseases might have been quite problematic before then. Advances in transportation technology during the 17th, 18th and 19th centuries had enabled mass migration of peoples,

[2] Olshansky and Ault (1986), Wilmoth and Lundström (1996), Kannisto et al. (1994), Thatcher (1992), Vaupel and Lundström (1994).

domestically and internationally. This may have led to the spread of contagious diseases and to the introduction of new diseases that were catastrophic for some populations (Crosby (1986), McNeil (1998)). This escalation of epidemics lasted through the first three-quarters of the nineteenth century. Outbreaks of smallpox, influenza, typhus, typhoid fever, cholera, diarrhea, among others, erupted frequently, spanning as many as 30 years during the first half of the 19th century.

Unsurprisingly, they featured in Malthus's (1766–1834) theory of population in which per capita output tended to return to a roughly constant level. Recurring outbreaks may have returned per capita output to roughly similar steady states over time. Their recurrence indicates persistence of unsanitary conditions and frailty of population health.

However, by most accounts, during the last quarter of the nineteenth century, ascendancy of the germ theory—the unique, knowledge-based stimulus—renewed human understanding of disease etiology, stimulated public sanitation and spurred further advances in bacteriology. Progressively, people learned how to check disease through effective arterial sewage and water supply systems; through pasteurization, sterilization and antiseptic procedures; through behavioral change brought about by education; through legislation; and through formation of institutions to contain outbreaks and infectious diseases in general. (Chadwick (1842), Bairoch 1988, Frazer (1950), Szreter (1988, 1997), Easterlin (1999), Mokyr (2000), Tomes (1990)).

The germ theory's ascendancy perhaps delivered a major blow to the Malthusian view. During the transitional stage, even as mass migration and urban crowding continued, infectious diseases and frequency of outbreaks began diminishing. Consequently, life expectancy started rising permanently for the first time in centuries, perhaps altering people's worldview of the opportunities they could avail themselves and their children. Ultimately, in the post-World War II period, antibiotics and vaccines may have helped contain infectious diseases to a low rate, cementing the new worldview as the standard.

That standard, however, did not always exist. Economic theory tells us that short life spans of the Malthusian era would have discouraged investments in human capital. High rate of infectious disease and the uncertainty caused by frequent outbreaks may have perpetuated the view that there is little point in investing because you may not be around to reap the benefit.

Theoretically, human capital develops because households exercise a quantity-quality tradeoff in nurture of offspring. In this Malthusian regime, fulfilling the desired tradeoff may have entailed a high fertility rate. Not knowing how to prevent childhood sickness and death, households may have had more children for two likely reasons. First, they may have sought replacement for a child who died, which happened all too frequently. Second, fear of juvenile death might have led to a heightened precautionary demand for children—having more of them increased the chances that some would survive. Fearing such loss, they may also have been reluctant to school each child until it was reasonably certain the child would live long enough for such an investment to be worthwhile (Sah (1991), Kalemli-Ozcan (2000), Kalemli-Ozcan et al (2001)).

In this sense, the germ theory likely shifted the production function of human capital. Joel Mokyr has called it a "quantum leap" in humanity's grasp of disease-causation (Mokyr (2000)). Equipped with new knowledge, households may have been able to check disease. This may have activated salutary dynamics in which they had fewer children with more human capital per child. Thus, life expectancy and human capital (including schooling) may have increased simultaneously. Elevated average ability then may have invigorated economic activity permanently, bringing about a transition—epidemiologic and economic—from the Malthusian era. Without the knowledge, the dynamic might have reversed, vindicating the Malthusian worldview again.

The relevance of infectious diseases to conventional forms of human capital is best illustrated in Figure 4. Circa 1850, children (0–14 years) and the age group 15–64 years accounted for about three-quarters of all infectious disease-caused deaths. They accounted for 95 percent of the total reduction in infectious diseases, about two-thirds of it occurring in 15- to 64-year olds, the stage when benefits from prior investments materialize. The sharp fall in their share immediately after World War II suggests that the use of antibiotics and vaccines may have resulted in considerable social returns.

In this post-transitional regime, children and the labor force largely eluded infectious disease-caused fatality, adding years that could be used for schooling and acquiring work experience. The rate of infectious diseases was contained largely to ages 65 and above (mostly influenza and pneumonia).

About 97 percent of the total decline in infectious diseases had been attained through 1960.[3]

Non-infectious diseases and the Transition

As infectious diseases started diminishing, an epidemiologic shift began. Figure 5 shows the fraction of all disease-caused deaths attributable to infectious and non-infectious diseases. In line with propositions of the Transition, the share of all disease-caused deaths attributable to non-infectious started increasing.

However, non-infectious diseases were a significant proportion even during the Malthusian stage. This is slightly different from the standard tenets of the Transition, which say that infectious diseases may have been larger then.[4]

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[3] It is impossible to discuss each infectious disease for each age-group in this limited space. Suffice it to mention that age group 0–14 benefited from the reduction of all infectious maladies whether they were water- and food- or air-borne (cholera, typhoid, diarrhea, dysentery, infective enteritis, smallpox, scarlet fever, diphtheria, whooping cough, measles, among hundreds of others). The age group 15–64 benefited from reduction in water-borne infections, and respiratory infections (respiratory tuberculosis, influenza and pneumonia); the same is true for age group 65 and above.

[4] A reason for this difference could be that non-infectious diseases include a category called "Ill-defined Causes," which accounted for 20–25 percent of all disease-caused deaths until about 1870/80. However, bulk of the ill-defined appeared as "old age (and senility)," a conditions pertinent to ages 65 and above. I have included them in non-infectious diseases because they are likely to have been some unidentifiable chronic or degenerative condition in the elderly. Ill-defined causes diminished rapidly early in the twentieth century; perhaps what was previously considered ill defined became identifiable and was assigned presumably to various non-infectious disease-categories.

Consequently, until about 1870/80, we have a regime in which life expectancy remained low, the rate of infectious diseases was high, and major outbreaks recurred frequently—distinct Malthusian features all. Yet, non-infectious maladies were also a considerable fraction of all disease-caused deaths.

> *Proposition 1: During the Malthusian regime, both infectious diseases and non-infectious diseases likely coexisted as significant proportions of all disease-caused deaths.*

The proportions do not necessarily reflect each disease-category's substantive importance because a particular disease may have been instrumental in causing other types as well.

Figure 6 shows the contour of non-infectious maladies along with infectious diseases (Figure 2 reproduced in a broken line). Since at least the mid-nineteenth century, the rate of non-infectious maladies has been larger than infectious diseases. Existing propositions of the Transition posit that infectious diseases were larger during the Malthusian epidemiologic regime. On the contrary,

> *Proposition 2: During the Malthusian regime, the rate of non-infectious disease-caused fatality was likely larger than infectious disease-caused fatality. This has continued through the transitional and the post-transitional regime.*

Given that life expectancy during the Malthusian stage persisted between 27 and 41 years, it is reasonable to infer that average life spans were short (life expectancy is *not* life span though the two may correlate positively). And shorter average life spans coincided with a much higher rate of non-infectious maladies than today. It follows

> *Proposition 3: During the Malthusian stage, on average, chronic and degenerative maladies likely surfaced or turned fatal, or both, at younger ages than during the post-transitional stage.*

Therefore, besides infectious diseases, non-infectious diseases likely constrained human capital formation as well. Furthermore, like infectious diseases, conditions that generate most non-infectious maladies were also much worse during the Malthusian era than during the post-transitional period. Their etiologies may have overlapped.

It is also clear from Figure 6 that as infectious diseases started declining, non-infectious diseases started to diminish shortly afterward. However, existing propositions say that the rate of non-infectious disease-caused deaths rises during the transitional and post-transitional stages. On the contrary,

> *Proposition 4: During the transitional phase, the rate of non-infectious diseases decreases. Their decrease lagged briefly the onset of reduction in infectious diseases. Though infectious diseases fell more rapidly, the fall of both categories at about the same time is suggestive of a synergy between them.*

This synergy appears to be a central feature of the epidemiologic transition. High rates of both disease categories coexisted during the Malthusian regime. Then during the transitional stage, both began diminishing, at about the same time. The following sections try to explore potential reasons.

Meanwhile, it is noteworthy that the downward trend in non-infectious diseases is not a recent phenomenon; it occurred throughout the twentieth century. Further, the declining trend has coincided with increasing average life spans—the converse of proposition 3. Therefore, fatality from non-infectious diseases was being deferred to older ages throughout the twentieth century. This, too, has been integral to the transition, though it may have become more visible in the oldest of the old only recently (the proposed "new" phase).

However, the decrease of aggregate non-infectious diseases needs to be qualified. Unlike infectious diseases, not all types of non-infectious diseases decreased.

A way of studying different trends in disease-categories is to calculate how much each category contributed to the reduction of All causes. This would highlight two features simultaneously. First, it clarifies the direction of change—a disease that had increased would have *added* to the aggregate death rate even as the aggregate diminished under the weight of diseases that fell. Second, it gives a perspective on the relative contributions of various disease-categories.

Table 1 shows the results of the accounting, for the aggregate population and for various age groups. There are two sets of numbers. In columns 2 through 5 the accounting is done through 1955/60, which is chosen as a reference point because through 1960 the transitional stage had ended and about 85 percent of the total increase in life expectancy had been achieved (Figure 1). It is informative to know which disease-categories contributed to the bulk of improvement during the transitional stage.[5] Columns 6 through 9 provide an accounting over the entire period. Expressed as percentages, a negative sign denotes that the disease-category helped *reduce* all-disease-caused death rate.

First consider the main italicized categories. Through 1960, reductions in infectious diseases contributed about two-thirds of the total decline for the aggregate population (column 5), for ages 0–14 (column 2), and for ages 15–64 (column 3). For ages 65 and above (column 4), the contribution is substantial as well.

Over the entire period (Columns 6 through 9), the contribution of infectious diseases is reduced because after 1960 much of the decrease in all disease-caused fatality has come from non-infectious maladies, particularly for age groups 15–64 and 65-and-above. Therefore,

> *Proposition 5: During the transitional stage, reduction in infectious diseases likely accounted for the bulk of decrease in all-disease-caused fatality for the aggregate population and for the broader age groups.*

The decrease of infectious diseases, therefore, was broad-based. It affected all age groups perhaps because of a scale effect of containment methods. Containing cholera means containing it for all.

Since information on all-disease-caused deaths is the basis for life expectancy calculations, it follows

[5] The calculations are based on 5-year averages.

Proposition 6: During the transitional phase, reduction in infectious diseases likely accounted for the bulk of increase in life expectancy at various ages; non-infectious diseases contributed a substantial amount too. However, infectious diseases have likely been restrained to a low rate in the post-transitional regime; whereas, non-infectious diseases have continued to diminish, adding further to life expectancy.

Ongoing decline of non-infectious diseases during the post-transitional phase suggests that life expectancy might increase further. Moreover, excepting cancers, the decline of all non-infectious disease categories in ages 15 to 64 seems to be in line with *Proposition 3*—the rate of fatalities from non-infectious diseases in this age group was much higher in the past. Furthermore, the decline in non-infectious maladies may not have ended yet.

However, through 1960, for the aggregate population (column 5), cancers and maladies of the circulatory system increased. For maladies of the circulatory system, all of that increase came from age group 65-and-above.[6] Only cancers have increased in all age groups (though trends of different types of cancers have varied). It follows these two disease-categories impeded the progress of life expectancy.

The negative contribution of both circulatory maladies and cancers shrinks when it is calculated over the entire period (columns 6 through 9) because their rates have reduced substantially since 1960 (even for ages 65 and above). Better medical technology may have contributed to that improvement.

About 65 percent of the total decline in non-infectious diseases too was achieved through 1960. This does not mean that they have been trivial after 1960. It just says that the conditions were much, much worse before.

4. Human Capital Frameworks and the Transition

Section 2 argued that declining disease-caused deaths signals households' intent to foster durability of human capital. If so, are human capital frameworks applicable to the study of epidemiologic change? In trying to answer this question, my goal is not to propose a particular theory. Given that health is essential to human capital, it is to explore whether human capital frameworks provide the tools for organizing thought about the Transition.

Human capital frameworks may be able to address the likely synergy between infectious and non-infectious diseases: During the Malthusian regime, high rates of infectious and non-infectious diseases coexisted; once infectious maladies began declining, most non-infectious maladies started diminishing as well.

[6] And some of the increase in cardiovascular maladies may be attributable to a mere transfer from what was previously thought to be ill defined. Though there is no a priori reason to believe that cardiovascular maladies were the only beneficiary of that transfer, it is likely that this may have occurred because "old-age and senility," which occurred in ages 65 and above, dominated the ill defined causes. And it is in this age group and not in ages 15–64 that circulatory maladies have increased.

In theory, the central mechanism of human capital formation is nurture of offspring. Moreover, this nurture has intertemporal consequences.

A growing body of knowledge indicates that early-age infection and other forms of deficiency in nurture result in poorer cellular development of organs. Unsalutary diets and frequent infections from infancy through adolescence impede cellular development because infections breakdown metabolic tissue, exacerbate nutrient deficiency, and deprive the human body of elements necessary for proper development of vital organs. Instead of cellular growth, the biological system diverts its resources toward repair of damaged tissue and toward synthesis of antibodies that fight infections. In doing so, it draws upon existing stores of protein (tissue), retarding cellular growth of vital organs, which increases their susceptibility to malfunction.

Furthermore, not all effects of early-age cellular impairment shows up immediately. Barker (1991, 1992, 1998) have reported that conditions such as coronary heart disease, hypertension, non-insulin dependent diabetes, autoimmune thyroiditis and stroke begin early in life but do not become apparent until mid-adult or later stages. If deficiencies persist then conditions may become irreversible, causing vital organs to malfunction. These include malfunctioning of the lungs, the circulatory system (Khosla 1981, Buck and Simpson 1982, Paneth and Susser 1995), the gastrointestinal tract (digestive system), impaired functioning of the endocrine system, the central nervous system (Idaquez 1988), and degenerative joint diseases. (Also see Scrimshaw and Gordon 1968, Scrimshaw 1970). Though exact biochemical mechanisms that turn early-age deficiencies to organ dysfunction later are not well established, it is reasonable to infer that poorly developed organs are susceptible to break down sooner than well-developed ones. (Tanner 1990, 1993, Martorell et al 1975).

Suppose the connection between infections, unsalutary diets and cellular development are true. Suppose too that households are unable to check infections and contagious diseases because effective knowledge either does not exist or is unavailable for various reasons. The current effect would show up in sickness and deaths from infectious diseases across age groups. But there is also an intertemporal effect. Vital organs of children that survived the onslaught of infections may have incurred irreversible cellular damage. This may manifest in frailty of their organs as adults. In turn, this frailty may lead to faster rate of organ degeneration that shows up as high rates of fatality from various non-infectious diseases. As long as infections persist and diets remain unsalutary, high rates of both infectious and non-infectious diseases may coexist—the Malthusian epidemiologic regime (*Proposition 1)*.

Now suppose effective knowledge to contain infections arrives and households use it to prevent childhood infections. The dynamic would reverse: as infectious diseases start diminishing, cellular development of successive cohorts may improve. As they reach adulthood, the intertemporal effect comes into play: better cellular development might reduce incidence or fatality (or both) from chronic and degenerative conditions. Non-infectious diseases may start diminishing.

The transitional stage would then involve improving human durability, which shows up in declining fatality from both infectious and non-infectious maladies. Consequently, life spans and life expectancy increase.

However, overall reduction in non-infectious diseases may become apparent only after a lag because several cohorts coexist in the population; some cohorts brought up under the older regime and others who grew up under regime of fewer infections. All else held constant, the reduction of non-infectious maladies would become apparent first in the younger age groups. The overall decline in non-infectious maladies, however, would become apparent only after cohorts with better cellular development begin dominating an "evolving" distribution. As cohorts with better cellular development age, they may suffer fewer chronic ailments; may recuperate at a faster rate than elder cohorts; or they may respond more positively to available medical treatments than elder cohorts. Non-infectious maladies may then turn fatal at progressively older ages than before (c*onverse of Proposition 3*). Although susceptibility to degenerative maladies increases with age, fatalities from such maladies may diminish even as average life spans increase.

Accordingly, the Transition would involve a decline in fatality from both infectious and non-infectious maladies (*Proposition 4*). However, infectious diseases may decline more rapidly because their containment methods involve a scale effect that does not necessarily, and immediately, apply to non-infectious diseases. Consequently, the *share* of infectious diseases would diminish rapidly, leaving behind non-infectious maladies to dominate the epidemiologic landscape even as non-infectious diseases decrease.

This health-related behavioral change in nurture and in environment does not rule out genetic influence. Genes as endowments of potential human capital may be transmitted stochastically to subsequent generations as well. Yet, their influence could be overlaid with behavior because humans use knowledge to benefit. If genes had rigidly programmed human life spans and life expectancy, and if behavior had no influence, average life spans of the Malthusian regime may have persisted. If genetic susceptibilities that drive non-infectious diseases had rigidly preset the age of fatality, then deaths from non-infectious diseases too may have persisted at younger ages. However, that has not happened; most non-infectious diseases diminished during the transition. Behavior and technology, therefore, may have been important.

Yet, this dynamic is likely bounded. Though its current and intertemporal effects might be occurring today as well, the post-transitional phase, however prolonged, is unlikely to last forever because life spans are finite.

However, that limit may not have been reached yet.[7] If falling death rates from infectious diseases has coincided with lowered incidence rates as well, cohorts born during the post-transitional regime (after World War II) may have grown up under significantly more salutary conditions than

[7] See Fries (1980, 1989) for limits to life expectancy that were proposed.

cohorts brought up during the transitional stage or earlier. All else held constant, its intertemporal effects might yet show up as additional reductions in non-infectious diseases, enabling people to reach ages older than the norm today. Consequently, life spans and life expectancy may increase further (*Proposition 6*).

Therefore, knowledge-induced behavioral choice emphasized in human capital frameworks appears to be directly relevant to the synergy between the two disease-categories and to the epidemiologic shift since the nineteenth century. And this mechanism has intergenerational effects. During the transitional stage, younger cohorts on average end up living longer than elder cohorts, presumably because irreversible juvenile cellular development combined with better medical technology enable them to do so.

Anthropometric indicators of cellular development since the nineteenth century also indicate intergenerational change. Figure 7 shows that adult stature of subsequent cohorts increased sharply toward the end of the nineteenth century, suggesting that Britons *succeeded* in nurturing better "quality" children since then.

Sadly, however, despite economic progress since the Industrial Revolution in the late eighteenth century, adult stature declined for almost fifty years before increasing later in the nineteenth century. Life expectancy too stagnated during that time. Impaired nutritional process—digestion, metabolism, absorption and storage—may have severely constrained cellular development, stunting physiological growth. This stunting might have coincided with a high rate of non-infectious maladies, and at younger ages. Consequently, human capital may have been much less durable than it is today (Fogel (1994)).

Researchers have found that countries that urbanized rapidly before the ascendancy of the germ theory—and the U. K. was one of them—experienced prolonged stagnation or worsening of their population's health (Steckel and Floud (1997)). This suggests that curtailing infections through various methods, particularly in urban areas, may have been important.

Though human capital frameworks seem broadly applicable, constructing particular models to capture every nuance of this complex epidemiologic shift is beyond the scope of this article. And this is because there are significant areas of ignorance about the biological pathways of disease causation, especially of non-infectious diseases. It is nevertheless clear, whether it is life expectancy or stature, infectious diseases or non-infectious diseases, there has been an underlying synergy in their developments since the nineteenth century.

Furthermore, health-related changes may work through intergenerational or intertemporal biological pathways. The hidden pathways may often confound studies that examine the influence of current economic conditions on current health-related change, particularly at the aggregate level. To understand this further we need to assess whether an increase in the level of GDP per capita or its growth rate, by itself, may have diminished disease-caused fatality.

5. The Transition and Per Capita GDP

Health improvements at the aggregate level are widely considered as a consequence of economic growth. Further, disease-caused fatality rates in richer countries tend to be lower than in poorer countries, suggesting that attainment of higher per capita incomes might cause disease-caused fatality to reduce. It is therefore prudent to ascertain whether the level of current per capita GDP or its growth rate might have directly reduced disease-caused deaths since the nineteenth century.

First consider the growth rate of per capita GDP. Results in Table 2 indicate growth rate of per capita output relates tenuously to reduction of All disease-caused deaths, infectious and non-infectious. Furthermore, some positive coefficients, albeit with large standard errors, suggest the opposite: disease-caused fatality increases as per capita output grows more rapidly, and faster growth would eventually lead us back into the Malthusian epidemiologic regime. The results indicate that the pace of growth is perhaps an inappropriate explanatory variable for epidemiologic change. Another set of results, not shown here, indicates weak relationship for short or long sample periods.

Now consider the level of per capita GDP. Table 3 shows that irrespective of disease-category, the level of per capita GDP offers scant explanatory power as well, although the sign on some coefficients is more in line with intuition.

Why might there be scant confidence in the role of *current* per capita income?

Consider the case for the United Kingdom. Historically, per capita GDP levels had been rising gradually since late eighteenth century (Crafts (1985), Clark (2001). However, that had not coincided with much life expectancy improvement for almost a century (Figure 1). It seems reasonable to suspect that direct effect of rising per capita income levels during that time may have been weak as well. (We saw that despite rising per capita incomes, adult stature even deteriorated for almost half a century). Lack of knowledge to check disease, rather than income, may have been the reason for stagnation in health.

Per capita GDP might also be too blunt an instrument for the matter at hand. Current changes in disease-caused fatality involve people from several cohorts simultaneously. These cohorts may have experienced different health-related pasts, which might have been more relevant to current disease-caused fatality than current per capita GDP. Arguably, one would try to relate each cohort to their past health-related choices and perhaps to past GDP levels. At the aggregate level, this is an impractical proposition because current fatalities include infants to 80 year-olds or more. Moreover, doing so itself is an acknowledgment that explanatory power of current level of per capita output is inadequate and that we need to look elsewhere for coherent explanations.

Furthermore, the Transition in industrialized countries began at per capita income-levels that were substantially lower than today. Triggered at least partly by the ascendancy of the germ theory into social consciousness, it involved behavioral change and newer forms of civil engineering. The lagged effects public health sanitation projects may lead to the weak relationship. The fixed costs of sanitation infrastructure may have featured prominently in GDP accounts for a short period but not

afterward whereas, subsequent health benefits of these investments may have continued over an extended period. Consequently, relationship between current per capita GDP and disease-caused fatality during the transitional and post-transitional stages would be weak.

Moreover, at the aggregate level, the notion that rising income by itself reduces disease-caused deaths is structurally unsound. As incomes trend upward, it implies immortality within finite time. Perhaps it is not surprising that the data indicate a weak relationship. Nevertheless, it is still possible there may have been a structural break in their relationship.

A type of structural break is an income-threshold. It is plausible that the level of per capita income matters up to a particular point but not afterward when factors other than income become dominant.

However, identifying such thresholds at the aggregate level is difficult. Consider the case for the United Kingdom shown in Table 4. If per capita income of, say 1870, is taken as a threshold, then according to this argument health should have improved significantly before 1870; but that is inconsistent with the data. If we consider 1960 as another arbitrary threshold, then per capita income level should relate strongly before 1960 but not afterward; this result does not obtain either. If we admit the possibility of a future threshold, then the two variables show associate closely over the entire period; the results, however, indicate otherwise.

Though thresholds may be relevant to extreme micro-level situations, identifying them at the aggregate level is difficult because the numbers of diseases that afflict people are large. Mechanisms that trigger a disease may be too idiosyncratic to associate meaningfully with particular income levels. Furthermore, the notion of income-thresholds also raises uncomfortable questions: What minimal income level, by itself, might contain maladies such as smallpox, influenza and cancer, among hundreds of others? Is there a minimal per capita GDP level that would reduce cancer, high blood pressure and obesity? Such questions likely lack clear and meaningful answers because new biomedical knowledge or technologies may arrive stochastically at any income level. For example, would we say that 1955's per capita GDP is a pivotal income-threshold for polio or would we say the fortuitous discovery of the Salk's vaccine was the catalytic event for polio containment in the United States and elsewhere?

Therefore epidemiologic change may occur for reasons other than GDP per capita and both types of diseases may occur at low incomes or high incomes (Preston 1975), Caldwell 1986, Easterlin 1999, Kunitz and Engerman 1992). In thinking about the Transition at the aggregate level, specific income-thresholds may not be a useful device. The numbers of diseases are too large and identifying income-thresholds for each may become an unproductive exercise. Thus,

> *Proposition 7: Explanatory power of growth rate and the level of per capita GDP appears to be weak. The hypothesis that rising income levels per se caused disease-caused fatality to diminish (in a linear way) is unsound because it implies immortality*

within finite time. And if we then turn to the notion of particular income-thresholds, it does not seem to provide a meaningful guide either.

This does not mean, however, that economic progress has no bearing on epidemiologic change. It only says there is little confidence in level of current GDP per capita or its growth rate as primary explanatory variables.

Examining factors more closely associated with disease-related phenomena might provide us with better understanding of the Transition. Epidemiologic change may have involved intergenerational or intertemporal pathways (as were discussed in the previous section) that current GDP per capita or its growth rate may not explain or capture adequately. Moreover, even if "living standards" correlate with coincident per capita GDP levels, the correlation could appear because of numerous aspects of the "standard" not included in GDP accounts. And if they are not, then it is informative to identify them and explore their significance instead.

Doing so, however, may require studying each country in the context of its epidemiologic regime. The Malthusian epidemiologic regime likely required a different set of institutional and technological arrangements than the post-transitional regime. Consequently, inference from studies that use GDP to explain disparity of health-related outcomes across countries may be fragile, and often unreliable, particularly if the sample periods are short.

Though underlying causes for the Transition may have been several, it has nevertheless coincided with substantial improvement in human durability: Life expectancy has increased by almost 100 percent; human body size has increased by almost 50 percent; few people die of infectious diseases anymore; non-infectious disease-caused death rate has reduced considerably too. Did this improvement in durability invigorate an economic transition from the Malthusian regime?

6. "Engine" of Growth and Economic Transition

In human capital-based growth theory, an economic transition from the Malthusian regime entails a permanent increase in the pace of growth. Acting as an engine of growth, human capital helps sustain a permanently faster pace.

On the other hand, in models of growth a la Solow (1956), physical capital did not provide such an engine because diminishing returns to its accumulation prevented an economy from maintaining a permanently faster pace, a prediction corroborated in numerous empirical studies.

How does this difference relate to the epidemiologic transition? And what are its implications for long-term growth?

Temporary versus Permanent Influence

Figure 8 stylizes temporary versus permanent influence on growth. Consider an already growing economy. Now suppose there occurs a stimulus to human capital, say eradication of hundred diseases that afflicted people before time T_0 (Panels A.1 and B.1). In an economy that behaves in

accordance with the Solow model, the stimulus increases the growth rate of per capita output to g_1 (panel A.1). However, after the transitional period, the growth rate reverts to its prior pace g_0—a temporary influence. Consequently, the slope of the long-term growth path remains unchanged (panel A.3). After the transitional period only the level of per capita output rises permanently: the level effect.

On the other hand, in an economy that behaves according to human-capital based theory, disease-eradication stimulates a permanently faster growth rate (panel B. 2). Its influence on the pace of growth lasts—an engine of growth. Consequently, the level of per capita output (in panel B.3) increases (the level effect). However, it does so more rapidly than before and the slope of the growth path becomes permanently steeper: the growth-rate effect.

Under both scenarios, the level of per capita output increases. However, only under the human-capital-based framework the pace of growth increases permanently. Therefore, it is necessary and sufficient to find out whether the stimulus influenced the growth rate. If it influenced the growth rate permanently, it follows that it influenced the final level as well. The converse is not necessarily true. The left-hand-side variable, therefore, must be the growth rate of per capita output. And a factor (like physical capital) known to generate only level effects, as such, does not belong to the right-hand-side.

The method of "cointegration" devised by Engle and Granger (1987), and Stock and Watson (1988) may assess temporary versus permanent effects. It entails estimating

$$\Delta \ln y_t = \alpha + \beta \ln d_t + \sum_{i=-m}^{m} \phi_i \ln d_{t+i} + \varepsilon_t \qquad t = 1,2,3,\ldots,T \qquad (1),$$

where, the left-hand-side variable is the first difference of the natural log of per capita GDP, $\ln d_t$ is the natural log of the disease variable, ε_t is an error term and m is a parameter. The hypothesis test

$$\begin{aligned} H_0 : \beta &= 0 \\ H_1 : \beta &< 0 \end{aligned} \qquad (2),$$

evaluates the influence. Under the null, the level of disease-caused fatality does not cointegrate with the long-term growth rate of per capita output (panels A.1 and A.2). Whereas under the alternative, the two cointegrate (panels B.1 and B.2). (For life expectancy variables and stature at adulthood, the alternative hypothesis would be $\beta > 0$).

However, this method requires a long sample period. Short-spanned data are unsuitable to because the transitional phases—economic and epidemiologic—tend to be prolonged.

Transitional periods of growth have been known to last two-to-three decades. A short sample period is unlikely to discern temporary influence from a permanent one because after the transitional stage growth may return to its prior pace (like panel A.2).

Similarly, as Figures 2 and 6 show, 97 percent of the total decrease in infectious diseases and 65 percent of the total decrease in non-infectious diseases had already occurred through 1960. A post-1960 sample period would overlook much of that improvement. It would be unable to assess the

central tenet of human capital-based theory: distinct levels of human capital would be associated with distinct steady state growth rates. Only a longer sample period—one that encompasses the time when disease-caused fatality was high (the Malthusian stage) and the time when it was restrained (post-transitional regime)—may reasonably assess that matter.

> *Proposition 8: To ascertain temporary versus permanent influence on growth, the sample period must encompass as much of the Transition as is possible. Shorter sample periods would likely conflate temporary and permanent effects because both economic and epidemiologic transitional stages tend to be prolonged.*

Therefore, this method may yield unreliable results for countries that have only recently begun their epidemiologic transitions.

However, the fact that the bulk of disease-caused fatality had diminished before 1960 does not imply their influence dissipated after 1960. Since 1960, the economy may have been able to sustain a faster growth *because* diseases that constrained people in the past ceased to do so. The influence would endure after 1960 (like panel B.2 of Figure 7). A post-1960 study of growth that does not account for this development might erroneously credit something else. Thus,

> *Proposition 9: Once a set of diseases is restrained or eradicated, their social influence likely endures. Since they ceased to be a hindrance, the population may become more able than the past and may sustain a permanently faster pace of economic activity than was possible before.*

This proposition is particularly relevant to infectious diseases because they were restrained substantially *before* 1960. Further, it points to a significant shortcoming of growth accounting conducted on data after 1960. It would pertain only to the *incremental* influence of health. Once a set of diseases is subdued, their incremental influence—not the total influence—is likely to be minimal. Only maladies that persist at a high rate may offer tangible incremental gains.

Consequently, short-spanned cross-country studies, typically 1960 onward, would provide unreliable guides for decision-making because such data would be unable to assess properly the importance of health-related change to long-term growth *within* industrialized countries.

Empirical results

Table 5 shows the estimate effects for "all diseases" along with results for other health-related variables from Arora (2001). The robustness of results across countries and variables suggest that emergence of human capital during the Transition influenced the pace of growth permanently, a finding that supports human capital-based growth theory.

Consider the case for the United Kingdom. If human capital had remained as fragile as it was during the Malthusian regime, the estimated effects imply that per capita output in 1994 would have been about 35–40 percent less than actuality. Alternatively, had the Malthusian epidemiologic regime persisted— high rate of disease-caused fatality, frequent recurrence of epidemics, low and

volatile life expectancy—it would have taken about 172 years to reach 1994's level of per capita output, about 50 years longer than actuality. Economic consequences of escaping the Malthusian epidemiologic regime have been substantial and permanent.

What would be the pace of growth before the transitional phase began? Assuming no health-related improvements, estimates imply that long-term growth rate would have been about 0.7 to 0.8 percent per annum before 1870. It would be slower if contribution of any prior health improvement to growth is subtracted; and slower still, if poorer health had impeded people's learning ability and other forms of human capital formation. Therefore, epidemiologic shifts that began toward the end of the nineteenth century may have hastened the onset of a steeper growth path.

Ascertaining the separate contribution of each disease-category, infectious and non-infectious, is beyond the scope of this article because their etiologies may have overlapped. It is nevertheless clear that a substantial proportion of the total effect would be attributable to infectious disease for several likely reasons: their percentage decline is much larger and the bulk of it was achieved before the post-transitional phase; their decline contributed to the bulk of increase in life expectancy; their decline may have contributed to the decline in non-infectious diseases; their decline has pertained most to ages critical for human capital formation.

However, looking ahead, the total influence of infectious diseases is unlikely to *recur* because their remarkable reduction would have to repeat in the future. Whereas there might be scope for further improvement in non-infectious diseases, particularly as new medical technologies become available (Murphy and Topel (2003)). But that improvement will likely pertain older people and economic gains may take the form of a level-effect rather than a growth-rate effect.

Discussion and conclusions

Though health is essential to human capital, sustained increases in skilled labor force essential to economic growth may not be guaranteed forever once health improves. By enabling more people into the labor force, health improvements are likely to reduced labor-market skill premiums as well. In turn, shrinking skill premiums may act as a disincentive to school and skill. Improving health may result in sustained increases in skilled labor force *only* in the presence of skill-biased technological change. Newer occupations brought about by technological change may prevent skill premiums from shrinking asymptotically to zero. For health-related policy to raise the level of long-term well being, economic policies that engender technological change are likely to be crucial.

Over the very long term, however, progress in food production is likely the bedrock of health-related outcomes in a society. Advances in food production are perhaps among the principal ultimate causes for human capital development. Besides enabling basic human function, food's synergistic role in curtailing disease is well known. Declining direct contribution of agriculture to annual GDP accounts over time significantly understates its social significance, particularly in industrialized countries. Furthermore, progress in food production in the past may have tipped the balance in favor

humans (instead of pathogens), only to be tipped further by new biomedical knowledge and public health sanitation.

Installing public health infrastructure to contain preventable diseases, particularly contagious and parasitic diseases may yield permanent social returns. This is particularly relevant to diseases whose etiology is known but technology to combat them is not yet discovered. Markets mechanisms are unlikely to directly provide such technologies. For example, there is considerable private disincentive to develop a vaccine if its subsequent use would wipe out the demand. Once a contagious disease is restrained or eradicated, the demand for the vaccine is likely to reduce or disappear. In industrialized countries, several contagious diseases diminished substantially before vaccines or effective medical technologies became available. Therefore, fixed investments in public health infrastructure and institutional mechanisms for containing contagious diseases may have been pivotal to the epidemiologic transition. This, however, does not mean that medical technologies have been irrelevant. It only implies that absent appropriate technologies, social losses may persist much longer, especially if institutional modes of disease containment remain undeveloped.

Furthermore, the returns from containing childhood diseases may have a long reach. Besides fostering human capital formation, it might lead to a decline in chronic and degenerative diseases down the road as well. Long-term trends of infectious and non-infectious diseases appear to have been synergistic, though the biological pathways of that synergism are not yet fully understood. Nevertheless, it does suggest that isolating infectious diseases from non-infectious diseases might significantly misrepresent their underlying causes.

Human capital-based frameworks provide a useful format for organizing thought about the synergy between infectious and non-infectious diseases during the epidemiologic transition. They also offer a way for evaluating the influence of health-related change on the economic transitions from the Malthusian regime. In a preliminary inquiry, this article has tried to offer some evidence that emergence of human capital during the epidemiologic shifts of the nineteenth century has influenced the slope of the growth path permanently. Social consequences of health-related change may have transcended generations.

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Table 1: Percentage contribution of principal disease-groups to reduction in All Disease-caused death rate

Disease category	*1871/75–1956/60*				*1871/75–1990/94*			
	Age groups				*Age groups*			
	0–14	*15–64*	*65 & +*	*Aggregate*	*0–14*	*15–64*	*65 & +*	*Aggregate*
	(2)	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*
Infectious diseases	*– 69.2*	*– 68.4*	*– 57.2*	*– 65.5*	*– 67.0*	*– 64.3*	*– 38.8*	*– 55.2*
Non-infectious diseases	*– 30.8*	*– 29.9*	*–42.8*	*– 34.5*	*– 33.1*	*– 35.8*	*– 61.2*	*– 44.8*
Circulatory system	– 1.6	– 6.6	+ 116.2	+ 34.1	– 1.5	– 14.7	+ 29.3	+ 5.4
Neoplasms (Cancers)	+ 0.1	+ 5.9	+ 23.6	+ 9.8	– 0.1	+ 3.4	+ 20.5	+ 8.9
Nervous system	– 0.4	– 6.7	– 22.7	– 10.1	– 0.5	– 6.1	– 15.0	– 8.1
Digestive system	– 0.7	– 7.0	– 5.4	– 4.8	– 0.8	– 6.4	– 3.9	– 4.1
Nutritional & metabolic disorders	– 0.4	– 2.3	– 1.1	– 1.3	– 0.4	– 2.1	– 1.1	– 1.1
Genitourinary system	– 0.3	– 3.4	– 2.6	– 3.4	– 0.4	– 3.7	– 3.2	– 2.7
Musculoskeletal system	– 0.3	– 0.1	– 0.33	– 0.6	– 0.3	– 0.9	0.0	– 0.4
Ill-defined causes	– 2.4	– 4.0	–116.6	– 39.2	– 2.1	– 4.0	–81.6	–33.2
Perinatal causes	–25.5	__	__	– 7.3	–28.2	__	__	– 6.7
Others	– 0.7	– 5.7	– 8.9	–15.4	0.0	– 1.3	– 6.2	– 2.8

Note: Time series for each cause, was considered separately for each age group and for the aggregate population. "Others" includes congenital anomalies, causes pertaining to pregnancy and childbirth, maladies of the skin and subcutaneous tissue, and of blood and blood-forming organs. Over 1870–1960, All-disease-caused deaths reduced by 93% for ages 0–14, by 73.5% for ages 15–64, by 32.5% for age 65 and above and by 54.4% for the aggregate population; over 1870–1994, the percentage reductions are, 98.1%, 82.8%, 47.4% and 66.4%, respectively.

Table 2: Explanatory Power of Growth rate of GDP per capita

	$\hat{\theta}$	*Standard error*	*t-statistic*
	All Diseases		
Australia	0.129	0.225	0.576
France	–0.026	0.246	–0.105
Netherlands	–0.070	0.157	–0.045
United Kingdom	–0.050	0.334	0.151
	Infectious diseases		
Australia	0.184	0.530	0.347
France	–0.113	0.374	–0.302
Netherlands	0.070	0.372	0.189
United Kingdom	0.539	0.832	0.648
	Non-infectious diseases		
Australia	0.116	0.164	0.707
France	0.032	0.324	0.099
Netherlands	–0.030	0.114	–0.265
United Kingdom	0.075	0.158	0.476

Notes: This table presents estimates of θ obtained from implementing the following equation:

$$\Delta \ln d_t = \alpha + \theta g_t + \sum_{i=-m}^{m} \varphi_i \Delta g_{t-i} + \varepsilon_t$$

where, the left-hand-side variable is the first difference of the natural log of the disease-variable, and g is the first difference of the natural log of per capita GDP.

Table 3: Explanatory power of current level of GDP per capita

	$\hat{\theta}$	*Standard error*	*t-statistic*
	All Diseases		
Australia	–0.0072	0.0087	–0.8331
France	–0.0003	0.0101	–0.0339
Netherlands	–0.0002	0.0084	–0.0277
United Kingdom	–0.0019	0.0099	–0.1930
	Infectious diseases		
Australia	0.0120	0.0278	0.4327
France	0.0146	0.0203	0.7315
Netherlands	0.0700	0.0262	2.6679
United Kingdom	–0.0128	0.0187	–0.6830
	Non-infectious diseases		
Australia	–0.0170	0.0062	–2.8335
France	–0.0034	0.0097	–0.3525
Netherlands	–0.0051	0.0070	–0.7273
United Kingdom	–0.0048	0.0078	–0.6230

Notes: This table presents estimates of θ obtained from implementing the following equation:

$$\Delta \ln d_t = \alpha + \theta \ln y_t + \sum_{i=-m}^{m} \varphi_i \Delta \ln y_{t-i} + \varepsilon_t$$

where, $\ln y_t$ is the natural log of the level per capita GDP.

Table 4: In search of income-thresholds, United Kingdom

Disease category	$\hat{\theta}$	*Standard error*	*t-statistic*
	1870–1994		
All diseases	– 0.0019	0.0099	– 0.193
Infectious diseases	– 0.0128	0.0187	– 0.683
Non-infectious diseases	– 0.0048	0.0078	– 0.623
	1870–1912		
All diseases	– 0.0010	0.0626	– 0.156
Infectious diseases	– 0.0176	0.1591	– 0.111
Non-infectious diseases	– 0.0101	0.0608	– 0.166
	1870–1960		
All diseases	– 0.0172	0.0252	– 0.682
Infectious diseases	– 0.0292	0.0539	– 0.587
Non-infectious diseases	– 0.0009	0.0199	– 0.045
	1960–1994		
All diseases	– 0.0001	0.0258	– 0.004
Infectious diseases	– 0.0167	0.1222	– 0.137
Non-infectious diseases	– 0.0036	0.0238	– 0.155

Notes: This table presents estimates of θ *obtained from implementing the following equation:*

$$\Delta \ln d_t = \alpha + \theta \ln y_t + \sum_{i=-m}^{m} \varphi_i \, \Delta \ln y_{t-i} + \varepsilon_t$$

where, $\ln y_t$ *is the natural log of the level per capita GDP.*

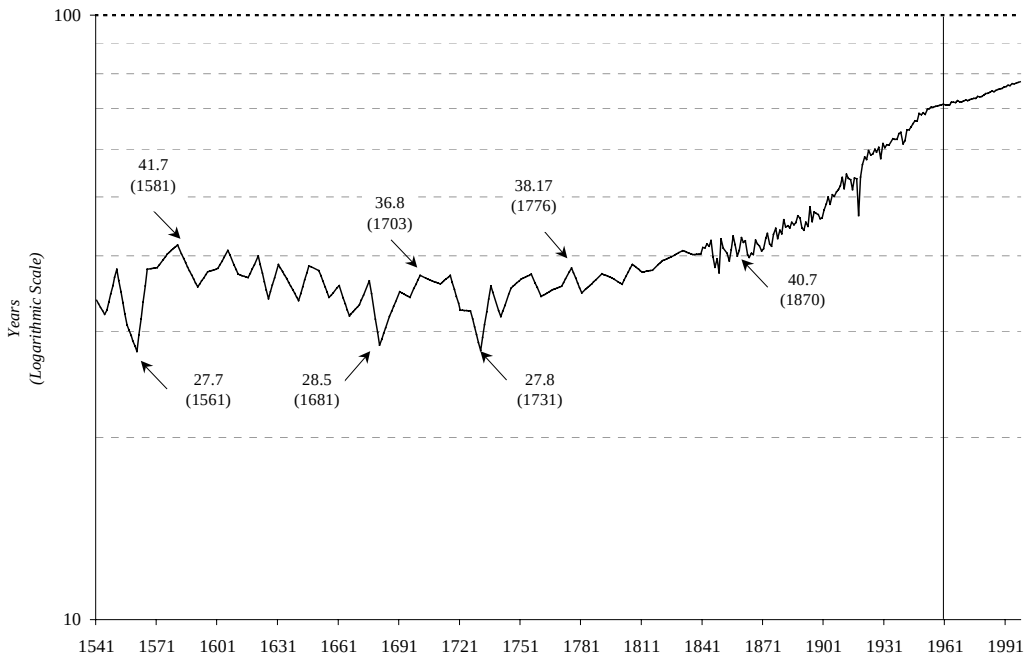
Table 5: Estimated long-term effects

(Fraction of total growth)

	e_0	e_{15} or e_{20}	*Stature*	*All diseases*
Australia	0.43	0.49	—	0.35
France	0.42	0.62	0.56	0.20
Netherlands	0.62	0.47	0.40	0.28
United Kingdom	0.35	0.35	0.31	0.27

Notes: e_0 is life expectancy at birth and e_{15} is life expectancy at age 15. The results for life expectancy and Stature come from Arora (2001). Excepting the United Kingdom, sample periods for life expectancy variables and" all diseases" vary. The detailed econometric results are available on request.

Figure 1: Period life expectancy at birth, England and Wales (1541–1998)



1b: Period life expectancy at birth, age 15 and age 20

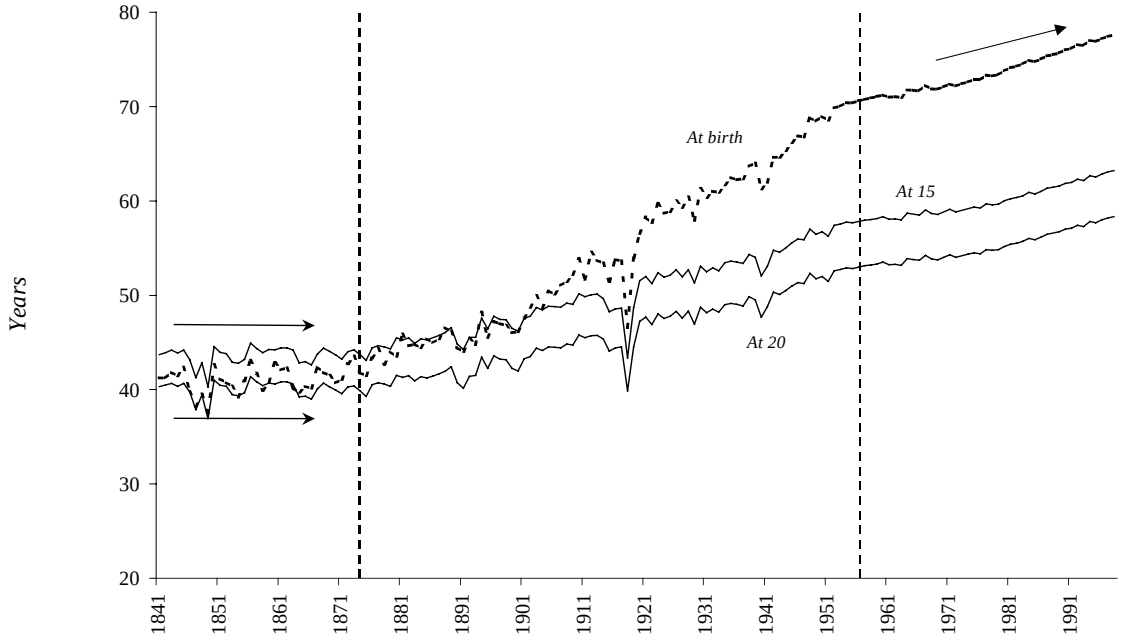


Figure 2: Infectious diseases in England & Wales, 1848–1994

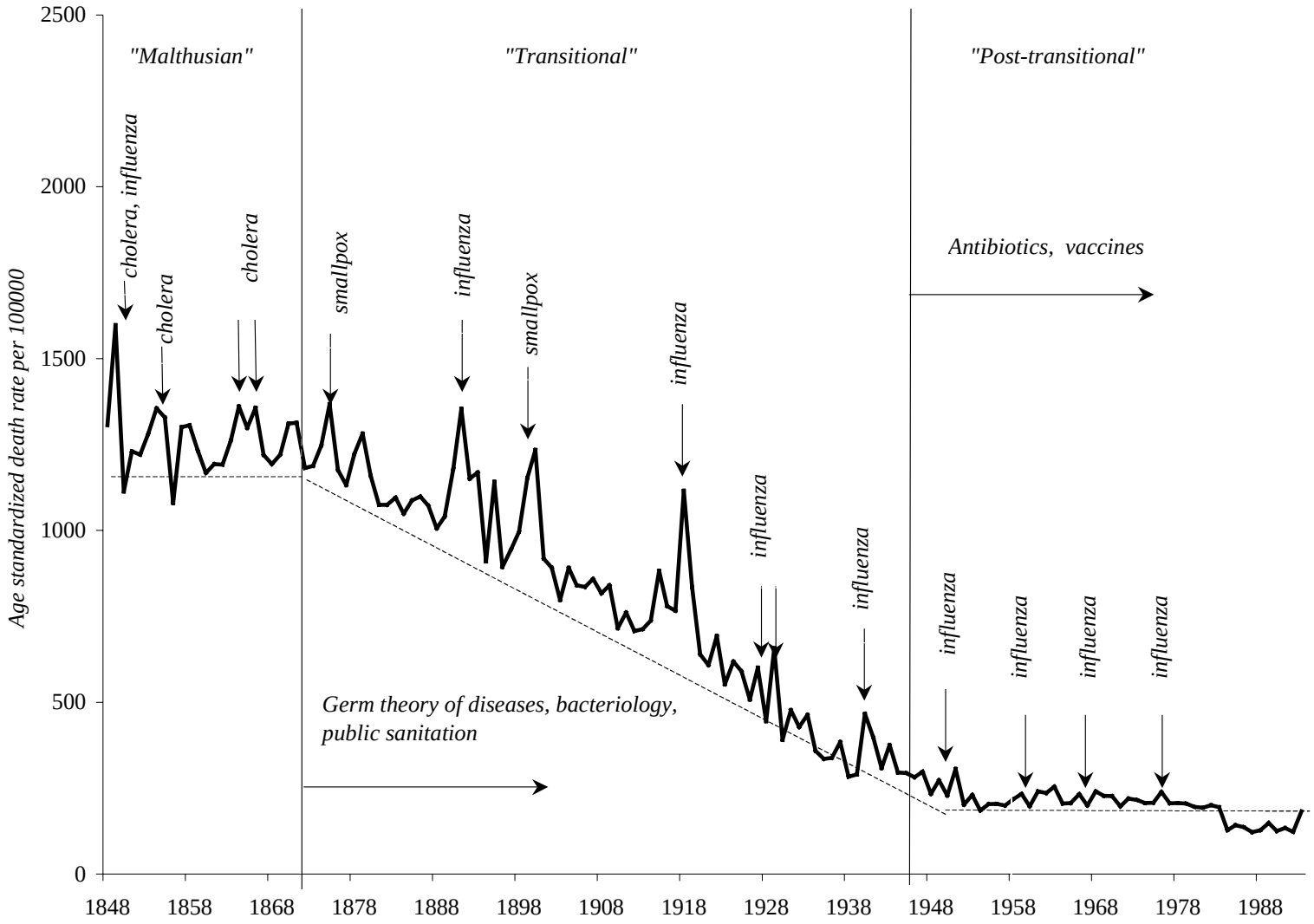


Figure 3: Frequency of epidemics since the 17th century

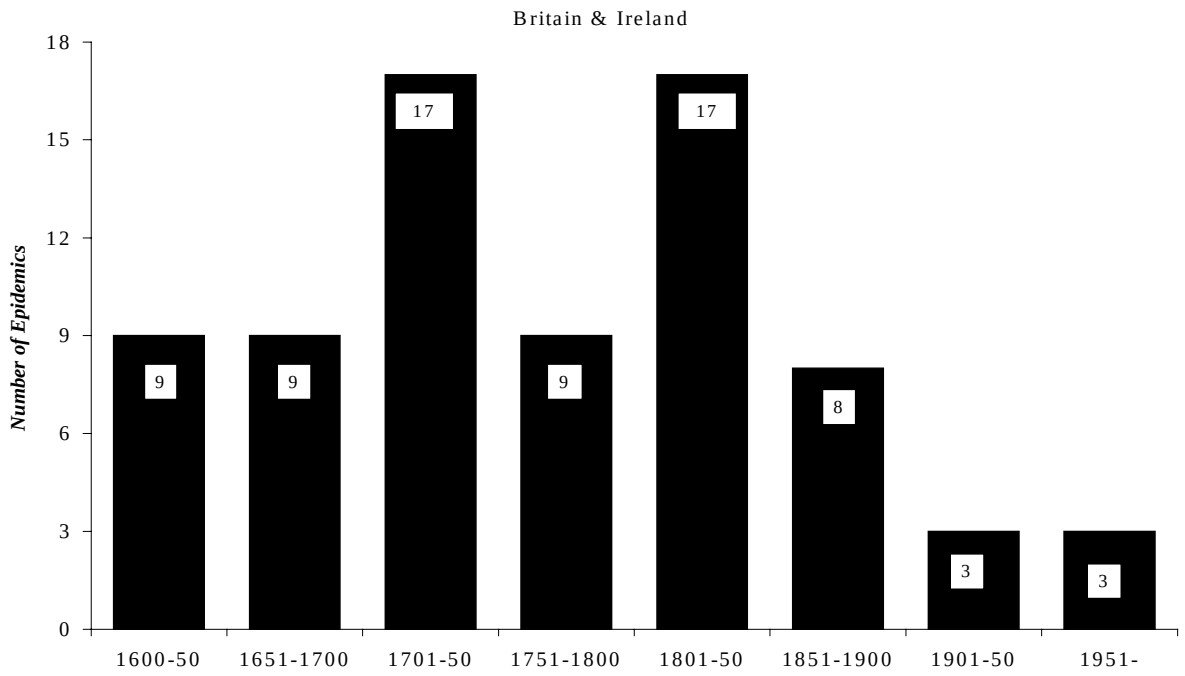


Figure 4: Age distribution of deaths from infectious diseases, 1848-1994

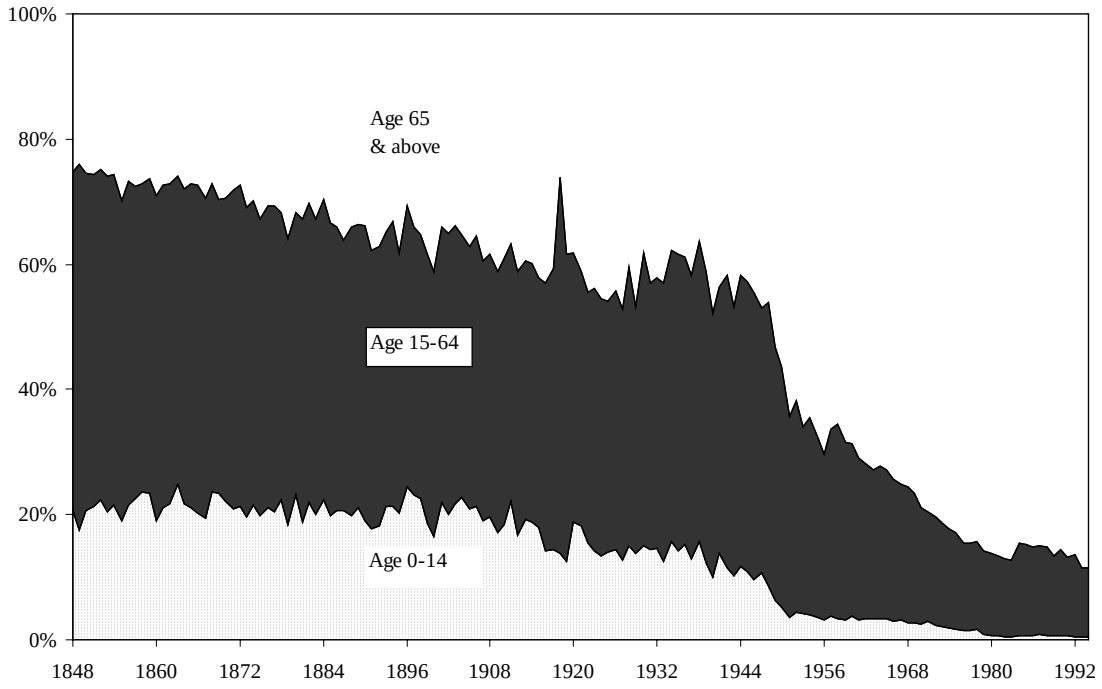


Figure 5: Infectious and non-infectious diseases as proportions of All disease-caused deaths

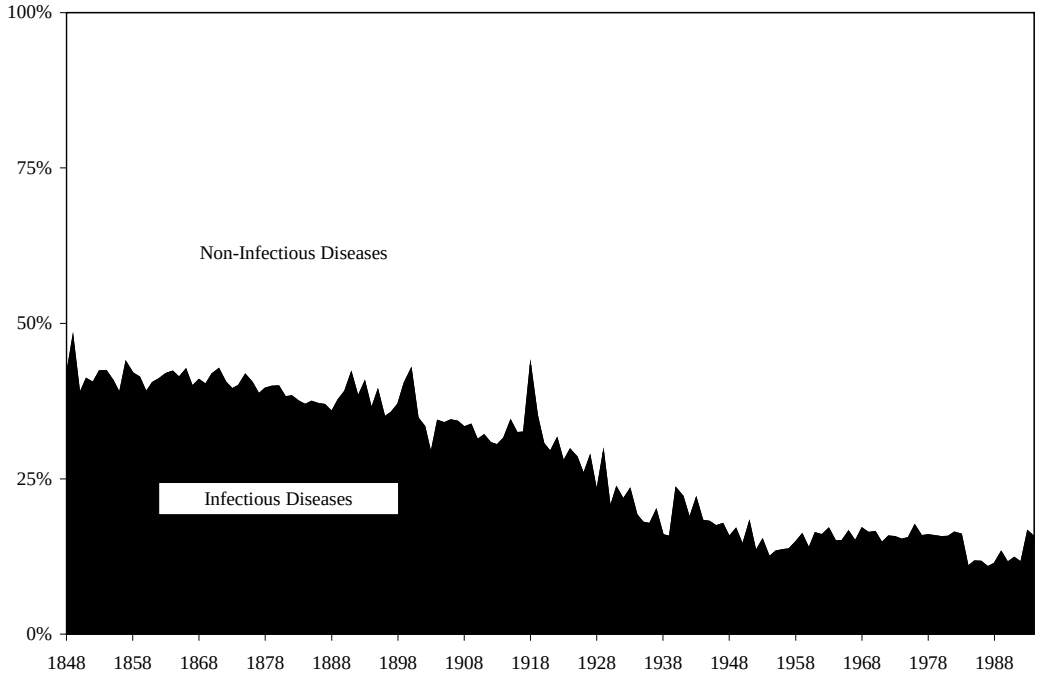


Figure 6: Non-infectious diseases and infectious diseases, England & Wales 1848–1994

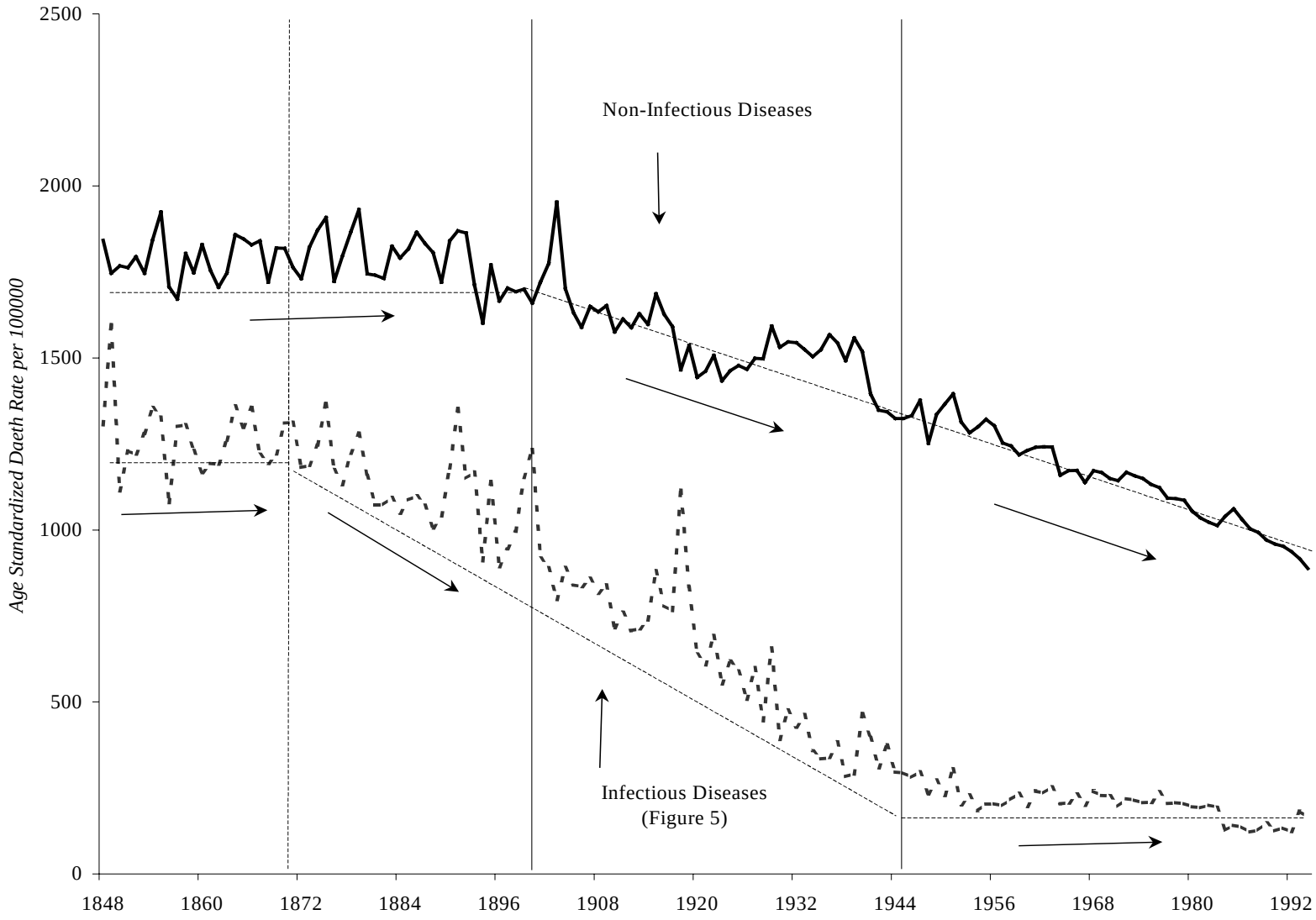


Figure 7: Stature of 18-year-old males in the U. K. 1800-1950

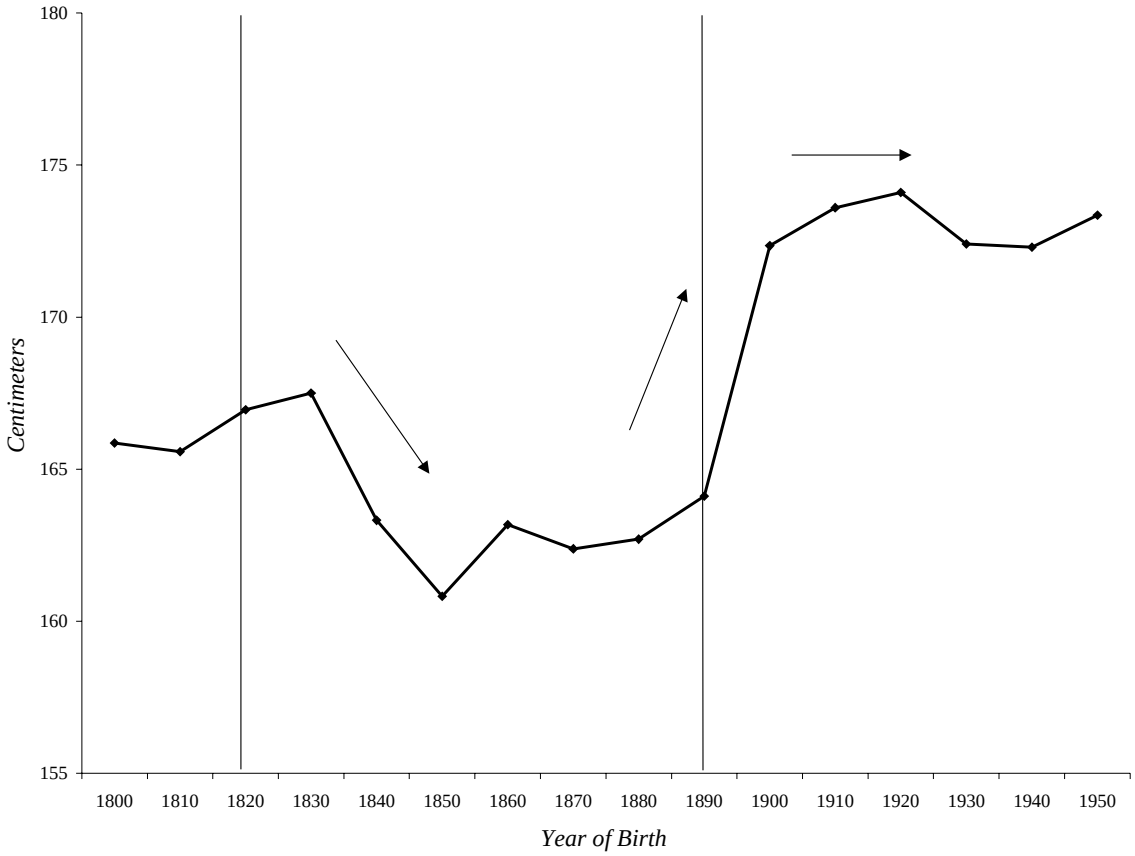


Figure 8: Stylized temporary versus permanent growth-rate effects