The reversal of the relation between economic growth and health progress: Sweden in the 19th and 20th centuries[1]

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Abstract

Health progress, as measured by the decline in mortality rates and the increase in life expectancy, is usually conceived as related to economic growth, especially in the long run. In this investigation it is shown that economic growth is positively associated with health progress in Sweden throughout the 19th century. However, the relation becomes weaker as time passes and is completely reversed in the second half of the 20th century, when economic growth negatively affects health progress. The effect of the economy on health occurs mostly at lag zero in the 19th century and is lagged up to two years in the 20th. No evidence is found for economic effects on mortality at greater lags. These findings are shown to be robustly consistent across a variety of statistical procedures, including linear regression, spectral analysis, cross-correlation, and lag regression models. Models using inflation and unemployment as economic indicators reveal similar results. Evidence for reverse effects of health progress on economic growth is weak, and unobservable in the second half of the 20th century.

1. Introduction

One of the unarguable proofs of social progress in recent centuries is the reduction of mortality rates and the associated increase in life expectancy. Thirty years ago, only 36 out of 178 countries had a life expectancy at birth of over 70 years, while there are now 87 nations exceeding this figure (UNDP, 2005). In a large number of countries today most humans are able to reach an advanced age, a privilege that was enjoyed worldwide by small minorities only one century ago (Riley, 2001).

The historical decline of mortality has been attributed to various factors associated with economic and social advancement, including the rising availability of material goods, urbanization and improvement of physical infrastructures and housing, increasing levels of education, improvement in personal and social hygienic behaviors, medical advances, the disappearance of slavery, and other significant reductions in discrimination for gender, religious, or ethnic reasons (Cutler et al., 2006; Fogel, 1994; Kunitz, 1986; Riley, 2001; Sen 1990). It is generally agreed that the dramatic reduction of mortality due to infectious disease during the past two centuries has been the major determinant of the transition from high to low levels of death rates in every country. Consequently, among the potential causes explaining the drop in mortality, a prominent one is the improvement in nutrition, leading to a strengthening of the immune resistance to infection (Fogel, 1991; Fogel, 1994; Harris 2004; Kunitz, 1986; McKeown, 1985; McKeown, 1988). Public policies improving the hygienic quality of drinking water, milk, and various other foods; sanitizing the urban environment; and cleaning up housing have also been claimed as major factors for the dramatic reductions in mortality rates (Szreter 1988; Szreter 1999). Though there are extant controversies and lacunae in knowledge (Fogel, 1997), because the microbiological knowledge and the pharmacological or biological tools to fight infections (antibiotics, chemotherapeutic drugs, and vaccines) became available only many years after the accelerated drop in infectious disease mortality had started, it is usually accepted that progress in medical technology may have had a quite limited role in the historical decline of mortality (Fogel, 1997; Grundy 2005; Kunitz 1987; Preston 1996). Views on the impact of medical technologies and medical care on mortality rates in recent decades cover a wide spectrum (Korda and Butler, 2006; McKinlay et al., 1989). At any rate, if the improvement in nutrition were the basic link in the chain leading to the secular decline in death rates; or if it were the public policies such as supply of clean drinking water,

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building of sewage networks, removal of garbage, and widespread vaccinations; or if several of these factors played important roles, what is undeniable is that the historical decline in mortality must be somewhat related to the process of economic development.

Assuming that particular aspects of the process of economic development were associated with the secular decline in mortality rates, it would be expected that the faster the process of economic development, providing health-improving goods, services, and infrastructures, the faster the progress in health as measured by declines in mortality rates. Indeed, research on preindustrial societies has shown links between harvest yields, grain prices, real wages, and changes in mortality. However, mortality responses to agricultural failures, grain price inflation, or changes in real wages become more muted as the level of development increases (Thomas, 1941; Lee, 1981; Bengtsson and Ohlsson, 1985; Galloway 1988). Moreover, in the United States and Britain, some historical periods of rapid economic growth during the early years of industrialization have been shown to coincide with increasing mortality (Easterlin 1999; Haines et al., 2003; Higgs 1979; Szreter 1998). In modern India and China during recent decades of strong economic growth, the declines in mortality rates have been small compared with strong drops in mortality during the slow-growth decades before economic liberalization (Cutler et al., 2006).

A whole body of research, mostly published in medical journals, claiming short-term and long-term effects of periods of economic slowdown in rising mortality rates in 20th century industrial economies (Brenner 1971; Brenner 1977; Brenner 1979; Brenner 1981; Brenner 1983; Bunn 1979) has been discredited by later and more solid studies showing short-term oscillations of mortality fluctuating up in expansions and down in recessions, with the death rate sometimes even reversing its declining long-term trend during periods of accelerated economic growth (Chay and Greenstone, 2003; Dehejia and Lleras-Muney, 2004; Gerdtham and Ruhm, 2006; Graham et al., 1992; Laporte 2004; Neumayer 2004; Ruhm 2000; Ruhm 2003; Ruhm, 2005; Tapia Granados, 2005a; Tapia Granados, 2005b). Recent research seems therefore to suggest an inverse relation between the rate of improvement in health conditions and the rate of economic growth, at least in the short run and in advanced economies in recent decades. Moreover, in modern industrialized nations, it is not hunger but harmful caloric overconsumption and its pathologic effects—overweight, diabetes, hypertension, cardiovascular disease, and cancer—that are the scourge of the poor (Isaacs and Schroeder, 2005). A little-known historical natural experiment was the one occurring in the Nordic countries during World War II, when major drops in mortality due to cardiovascular disease and diabetes took place, apparently as a consequence of food shortages and cuts in the consumption of dairy products (Malmros 1950). Something similar may have happened in the 1990s in Cuba (Franco et al., 2007).

Though it is increasingly accepted that in the short run economic growth may have harmful effects on health, in the long term a beneficial impact of economic growth on health improvement is usually accepted. For instance, in a recent commentary (Ruhm 2005), Christopher Ruhm—probably the author who has most forcefully shown in recent years the association between economic expansions and mortality increases—has stated,

> Higher mortality during temporary expansions need not imply negative effects of permanent growth. The key distinction is that transitory increases in output usually require more intensive use of labour and health inputs with existing technologies, whereas lasting changes result from technological innovations or expansions in the capital stock that have the potential to ameliorate any costs to health. Individuals are also more likely to defer health investments during temporary than permanent increases in work hours and sustained growth permits the purchase of consumption goods (like safer cars) that benefit health.

Since it has been proved that the poorest countries also have the worst health indicators, and in these countries income growth has a direct translation into improved health conditions (Pritchett and Summers, 1996), it is tempting to apply the same reasoning to high- or medium-income countries, assuming for instance that the capacity to generate higher earnings "facilitates an increase in the consumption of health-related goods such as adequate food or medicine" and healthy changes in lifestyle (López-Casasnovas et al., 2005).

In the field of historical demography, however, expectations about the impact of economic growth in the long-run decline of mortality are generally modest. For instance, according to al-

ready classical estimates by Samuel Preston, only between 10% and 25% of the massive international declines in mortality between the 1930s and the 1960s could be attributed to improved standards of living measured in terms of income per capita (Preston, 1976; Preston 1996), an estimate that has not been seriously challenged to date.

In each country, large increases in the output and availability of goods and services took place during the transition from an agrarian economy, mainly producing for self-consumption, to an industrial monetary economy in which markets and commerce play a much larger role. That is precisely the process through which Sweden passed during the past two centuries. The population involved in agriculture and subsidiary occupations was about 80% of all Swedes in 1800, still over 50% in 1900, but below 5% at the end of the 20th century. The share of "agriculture and ancillaries" in the Swedish gross domestic product (GDP) ranged between 35% and 40% from 1800 up to the 1870s, decreased to about 25% in the 1910s, and dropped to only 2.5% in 1990 (Krantz 2002; Swedish Board of Agriculture, 2005; Thomas, 1941).

Because of early development of a statistical registration system, Sweden has historical statistics that are probably the best in the world. Using these statistics, it is possible to analyze the long-term relation between economic growth and health progress. The results of the analysis that will be presented herein provide substantial evidence that the relation between economic growth and health progress reversed in Sweden during the past two centuries, from strongly positive in the first half of the 19th century to moderately negative in the late 20th century.

The data and methods used in the study are explained in the next two sections. Section 4 presents the results of the statistical analysis, and section 5 discusses the findings and presents the conclusions of the study. In the appendix we present and discuss (I) potential pathways connecting harvests, inflation, economic growth and mortality; (II) some issues related to the GDP estimates; and (III) Granger-causality tests.

2. Data

Economic growth for the years 1800–1998 was indexed by the annual rate of growth of GDP per capita.[4] To gauge inflation, the annual rate of change of the GDP deflator was used as an index of the year-to-year change in the general level of prices. The general crop index for the years 1800–1957 has been taken from Swedish official historical statistics (Sweden - Statistiska Centralbyrån, 1955). GDP, GDP per capita, and GDP deflator series are from Olle Krantz (2002), who kindly shared them with us. National account data by Olle Krantz have been considered of high quality (Backus and Kehoe, 1992; Maddison, 2003).[5] Also from Krantz is an unpublished series of annual unemployment rates for the years 1911–2000.

Demographic statistics from Sweden are taken from the Human Mortality Database, a common project of the University of California and the Max Planck Institute, available online (http://www.mortality.org/). The infant mortality rate and age-specific annual mortality rates for large age strata of youngsters (15-24), mid-age adults (35-54), and elderly individuals (70-89), for the whole population and for both males and females separately, were computed from the crude annual deaths, live births (for infant mortality), and population counts in each specific stratum of age and sex.

Life expectancy at birth (e_0), herein considered synonymous with longevity, is a direct indicator of population health, while age-specific mortality rates are inverse indicators. There is no

[4] Replacing GDP per capita growth with the rate of growth of GDP leads to similar results. These and other results not shown in the paper are included in a set of Supplementary Materials available on request from the authors and archived at http://deepblue.lib.umich.edu/.....

[5] However, Krantz himself states that in general, "for the 19th century, a lot of constructions and 'guesstimates' had to be made" (Krantz, 2002), According to the Swedish demographer Tommy Bengtsson (personal communication, New York, 2007), for the early decades of the 19th century both Krantz's estimates of GDP and the crop index series reported in Swedish historical statistics may have been computed using population statistics as an input. The reversal identified in this paper occurs well after the mid 19th century, by which time official statistics of trade and production had substantially improved.

univocal answer to the question of how to use health indicators to gauge the progress in population health; both absolute change and relative change have advantages and disadvantages (Sen 1981). In this investigation the annual gain in longevity and the annual relative decline in age-specific mortality rates were the indicators used to gauge health progress. The annual gain in longevity was computed as the yearly increase in life expectancy at birth ($\Delta e_{0,t} = e_{0,t} - e_{0,t\text{-}1}$), while the relative progress in population health using age-specific mortality rates as health indicators was computed as $-\Delta \ln (m_{a,t}) = -[\ln (m_{a,t}) - \ln (m_{a,t-1})]$, where $m_{a,t}$ is mortality at age a in the year t. Since differences in natural logs are very good approximations for rates of change, the negative difference of natural logs approximates a rate of decline, computed as $-(m_{a,t} - m_{a,t-1})/m_{a,t}$ for an age-specific mortality rate. Both the level and change in life expectancy are direct indicators of health or health improvement, but mortality is an inverse indicator of population health, so that referring to decline in mortality rates and changing signs after computing the first differences in logs is appropriate to avoid referring continuously to negative numbers. Therefore, when the paper refers to *improvement* or *progress* in health, what is implied is the absolute gain in longevity or the relative decline in age-specific or age-and-sex-specific mortality.

As measures of the dynamic status of the economy, both the rate of growth of GDP and the rate of growth of GDP per capita were used in this investigation. Since population usually changes slowly with respect to economic fluctuations, both rates correlate strongly (0.96 in the sample 1801-1998 and always above 0.92 in the four subsamples for half centuries). It can be argued that since increases in longevity increase GDP partly by increasing the size of the labor force, using GDP per capita growth instead of GDP growth might be a sensible if modest step towards reducing the problem of potential reverse causation in the relation between economic growth and health progress (Christopher Jencks, 2007, personal communication). For that reason, in this article we will present results using GDP per capita growth (computed as the first difference in log GDP per capita) as the indicator of the rate of growth of the economy.

During the two centuries under consideration, the Swedish mean annual GDP growth—as estimated by the difference in log GDP—was 2.3%, while the mean annual growth of GDP per capita was only 1.6%. The first half of the 20th century had the strongest economic growth, with an annual 2.4% mean growth of GDP per capita. In the long run, economic growth accelerated from 1800 to the middle decades of the 20th century, when GDP per capita growth reached average annual levels of 3.7%, to drop then quite dramatically to levels around 1% in the last quarter of the 20th century. The years after 1950 were also the most inflationary half century, with the GDP deflator growing, on average, 5.4% per year, versus 2.4% for the whole two centuries considered.

Life expectancy at birth, e_0, rose from values well below 40 years in the early 1800s to almost 80 years in the 1990s. In 1999 e_0 reached 76.9 years for males and 81.7 years for females. On average, during the period of study, longevity increased annually by one-fifth of a year, with annual mean increases of 0.10, 0.13, 0.38 and 0.16 years, respectively, for the four half-century subsamples of the period of study. The infant mortality rate—deaths before age one per 1,000 births—dropped from 240 in 1800 to 3.4 in 1999, about 2.1% per year throughout the period, with the fastest decline, 3.7% per year, in 1951–1999, and the slowest, 0.5%, in 1851–1900. Considering the other health indicators, the best period in terms of health progress was the first half of the 20th century for the decline in longevity shortfall and mortality at ages 15–24 and 35–54. However, the fastest decline in mortality at ages 70–89 occurred after 1950. The slowest health progress occurred for all considered health indicators in the second half of the 19th century.

GDP, the GDP deflator, life expectancy, and mortality rates have obvious trends, and the augmented Dickey-Fuller (ADF) test fails to reject the hypothesis of unit roots for all of them. When these series are converted into logs and then differenced, the ADF test rejects, at high levels of statistical significance, the hypothesis of unit roots. Therefore, the series of relative change of these variables which are used in the analysis are all trend-stationary.

The crop index that was used in some analyses evaluates annual crop yields on a scale from 0.5 for a disaster harvest to 4.5 for an excellent one.

Unemployment had a strong increase in Sweden in the 1990s, and indeed the ADF test does not rule out the hypothesis of unit roots, that is, the existence of trends in the unemployment se-

ries. As in other market economies, in the sample 1911–1999 of the Swedish economy for which unemployment data are available, economic growth correlates poorly with unemployment (– 0.13, $P = 0.24$), though it correlates strongly with the change in unemployment and with the rate of change in unemployment measured by the first difference in log unemployment (– 0.49 and – 0.56, respectively; for both correlations $P < 0.001$). For these reasons, for the years available the change in unemployment or the rate of change in unemployment were used in some models as indicators of the dynamic state of the economy.

3. Methods

We studied the coincidental or lagged covariations of "the economy" and health by using a variety of statistical models. Though there are many perspectives on causality in economics (Hicks, 1979; Darnell, 1993), not to mention social science (MacIver, 1973), epidemiology (Rothman et al., 1988; Susser, 1973), statistics (Eells, 1991; Pearl 2000), or philosophy (Mackie, 1974), a common view among empirical researchers is that statistical results alone cannot demonstrate causality, which is only "suggested" by statistical evidence placed in the context of a theoretical framework. In social science in general and economics in particular, causal statements do not readily generate consensus agreement. In natural science and often in public health research, causal judgments are usually the outcome of replicated results that also fit a theoretical model. The classical case is the attribution of causation to the relation between cancer and smoking. Time precedence (effects must follow causes), and dose-effect relation or concomitant variation (if the cause is greater, so must be the effect) are usually accepted criteria for suggesting causation in empirical research. Such statistical procedures as correlation, regression, or spectral analysis are tools to ascertain if these criteria exist. In time series analysis, leaving aside spurious correlations due to common trends, the coincidental or lagged covariation of two time series is strongly suggestive of one series causing the other, or a third factor causing both (Diggle, 1989; Glass et al., 1975). The scientific literature on the demographic transition and the so-called mortality revolution (Cutler et al., 2006; Easterlin, 1999; Easterlin, 2004; Harris 2004; McKeown, 1985; McKeown, 1988; Riley, 2001; Szreter 1988; Szreter 1998; Szreter 1999) provides a theoretical framework suggesting linkages between the process of economic development and the historical decline of mortality rates. Statistical evidence showing coincidental or lagged comovements of mortality rates with respect to "the economy" will therefore suggest a relation of causality between the latter and the former. Since there are plausible reasons for assuming that economic growth might affect health but there are also reasons to expect that health might affect the economy, we explored the relation in both directions by using various statistical procedures.

4. Results

4.1. Regression modeling of coincidental effects

The historical relation between economic growth and health progress can be modeled with an equation such as

$$\Delta e_t = b_0 + b_1 \cdot t + b_2 \cdot g_t + b_3 \cdot t \cdot g_t + \varepsilon_t \qquad [1]$$

where the progress in health indexed as a longevity gain (or alternatively, as a relative decline in a mortality-based variable) is regressed on a constant, time t, economic growth g_t, and the interaction of time and economic growth, $t \cdot g_t$. If economic growth has an impact on health progress, we will expect significant estimates of the parameter b_2. Moreover, if the impact of economic growth on health progress changes throughout time, we will expect a significant estimate of the parameter b_3.

The results for this type of model, in which time has been rescaled so that t = year – 1800 (table 1), provide strong evidence that the year-to-year short-term effect of economic growth on health reverses during the considered period. The general pattern of the regression results is that of a positive and significant effect of economic growth on health progress for $t = 0$ — that is, at the start of the 19th century. For instance, considering the models including data for the two centuries, during the early 19th century each percentage-point increase in GDP per capita growth increases the annual gain in longevity by 0.44 years (44.5 is the effect corresponding to an increase of 100 percentage points in GDP per capita) and the annual decline in male mortality at ages 35–54 by 1.9 percentage points. There is also a pattern of negative effects for the estimated

interaction term $t \cdot g_t$, indicating that the passing of time attenuates the beneficial effect of economic growth. Almost without exception, the interaction term is significant when data for the two centuries, or for the 20th century only, are included in the regression, but not in models including only data for the 19th century.

Indexing health progress by the annual gain in longevity, the model for the two centuries renders statistically significant estimates of 55.5 for the effect of GDP per capita growth and – 0.510 for the effect of the interaction between economic growth and time. Therefore, $55.5\, g - 0.510\, g \cdot t$ is the whole economic growth-related effect on health progress at year t. This would be zero if a time is reached when there is an effect reversal and economic growth starts having a negative effect on health progress. Solving $55.5\, g - 0.51\, g \cdot t = 0$, we get $t = 55.5/0.51 \approx 109$, which is the year 1909. Computing this tipping point for other models in which the interaction effect is significant, the tipping point is some year in the 20th century or late decades of the 19th century. Standard errors computed for this tipping point, calculated via the Delta method (Casella and Berger, 1990), are usually of the order of decades.

The Durbin-Watson d in these models was always above 2.2. Since $d = 2 \cdot (1 - \hat{a})$, where $\hat{a}$ is the estimated autocorrelation of the residuals, d values above 2.0 indicate that the autocorrelation is negative and, therefore, the estimate for the standard error is expected to be enlarged, which will bias the P-values up. No spurious statistical significance is therefore to be expected.

Following a rationale similar to that in equation [1], progress in health can be also modeled as a function of the natural log of GDP per capita x_t, economic growth g_t, and the interaction between them, $x_t \cdot g_t$, so that

$$\Delta \ln h_t = b_0 + b_1 \cdot \ln x_t + b_2 \cdot g_t + b_3 \cdot \ln x_t \cdot g_t + \varepsilon_t \qquad [2]$$

From this equation, $\mathrm{d}(\Delta \ln h_t)/\mathrm{d}g_t = b_2 + b_3 \cdot \ln x_t$, and this must be zero for a tipping point in which GDP per capita growth g_t starts having negative effects on health progress. The tipping point thus occurs when GDP per capita is $x_t = e^{-b_2/b_3}$ and from this value we can identify the corresponding year. In practice, this time point was identified by fitting a fourth degree polynomial to x_t; this also provided the derivative required to find the standard error via the Delta method.

Tipping points obtained from specifications based in equation [1] modeling the interaction between time and economic growth (table 1) are largely consistent with those computed from equation [2] (table 2) based on the interaction between the level of GDP per capita and its annual growth. Using values of R^2 and the Akaike information criterion (AIC) to quantify the goodness of fit of the models, the models based on the interaction of time and economic growth (equation [1]) are just slightly better than the models based on the interaction between level and rate of economic growth (equation [2]) (results not shown). Time and log of GDP per capita are highly colinear variables, and specifications combining equations [1] and [2] do not improve the model fit. We conclude that the influence of economic growth on health progress has changed over time, and that increasing levels of economic development give one plausible explanation.

4.2. Spectral analysis

Spectral analysis identifies the frequency components of time series and provides evidence on the relation between two time series at given frequencies. It may also reveal lagged relationships that might be overlooked when observing contemporaneous cross-correlations, particularly if the relation is spread over several lags.

The squared spectral coherency K^2 of two time series may be interpreted as the correlation between the series at a particular frequency band. The phase f of the cross-spectrum quantifies the degree in which the two oscillations are coincident (in phase) or lead-lag (out of phase). It makes sense to observe it at frequencies in which there is a high degree of spectral coherence among the series.

What follows will focus on the cross-spectral analysis for the decline in mortality at ages 35–54 and the growth of GDP per capita (figure 1).[6] In the analysis for the other health indicators we generally found similar behavior, though mortality at ages 70–89 behaves quite differently.

[6] For smoothing the periodograms, we used two iterations of a modified Daniell kernel of width 3, following the recommendations of Venables and Ripley (2002).

In the four half centuries, spectral coherency graphs reveal peaks of high and statistically significant K^2 at frequencies corresponding to period oscillations of 3 or 4 years, though in 1850–1899 the peak is at a slightly larger period of 6 years (figure 1, first and second rows).[7] The phase φ corresponding to the peaks in K^2 is between –1 and +1 rad for the three half centuries before 1950 (figure 1, third and fourth rows). Since $f = \varphi/2\pi$, where φ is the phase in radians, f in fractions of a cycle is between – 0.16 and + 0.16 cycles—that is, for cycles of four to six years, oscillations of economic growth and the health indicator are "in phase," following each other with a lag of less than a year. In the second half of the 20th century the peak of K^2 at an oscillation period of 3 years corresponds to a phase of –3 radians. Therefore, the decline in mortality 35–54 and GDP per capita growth oscillate separated $-3/2\pi$ cycles, that is, about half a cycle, or about a year and a half for a cycle of three years.

In summary, for health change measured by mortality 35–54, spectral analysis reveals a clear difference between the second half of the 20th century and the other half centuries considered. Results are similar for the other health indicators. While before 1950 health oscillates mostly in phase with the economy, in the second half of the 20th century the oscillations are out of phase.

4.3. Cross-correlation models

As mentioned previously, a whole body of literature by Harvey Brenner has posed lagged effects of economic growth on health with lags up to 11 years (Brenner, 1995; Brenner 2005). However, Brenner's models have been seriously criticized (Eyer 1976a, 1976b, 1977; Kasl 1979; Sogaard 1992; Tapia Granados, 2005c; Wagstaff 1985; Winter, 1983) and researchers who have intently investigated the possibility of lagged macroeconomic effects on health (Neumayer 2004; Ruhm 2000; Tapia Granados, 2005b) have been unable to find observable lagged effects beyond a very few years. With the Swedish data we explored the possibility of lagged effects of economic growth on health progress, not only by spectral analysis but also by cross-correlations and distributed lag regressions.

The cross-correlations (not shown) between health progress and lagged economic growth, with lags up to 15 or 20 years, did not reveal any significant correlations beyond those to be expected by chance. The correlations between economic growth and health progress, as measured by the annual gain in longevity or the year-to-year decline in mortality at ages 35–54, show a clear pattern. While in half-century periods during the 19th century there are positive correlations between economic growth and health progress at lag zero, with the passing of time this pattern fades and in the 20th century a pattern of negative signs appears when health progress is correlated with economic growth lagged one or two years. When economic growth is correlated with lagged health progress, some significant positive correlations appear in the first half of the 20th century at lags three and four. This may suggest a stimulating effect of health progress on economic growth a few years latter, though it could also be a statistical artifact consequence of outliers (see the section on Granger causality in the appendix) .

4.4. Lag regression models

Results of regression models with lags in which health progress is regressed on a constant and coincidental and lagged values of economic growth confirm the pattern observed in the exploratory cross-correlation models, showing significant positive effects of economic growth at lag zero and negative and marginally significant negative effects at lags two or three when data for the two centuries are included in the regression. When the data included in the regression are split by century (table 3), there is a clear pattern of positive effects of GDP per capita growth on health progress at zero lag in the 19th century, while in the 20th century the significant effects are weaker and the dominant effect appears at lag two and is negative. These results also show a reversal of the effect of economic growth on health progress from the 19th to the 20th century, and are consistent with the results already presented.

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[7] Using the hypothesis testing terminology, in the squared coherency diagrams the null hypothesis is $K^2 = 0$, so that the K^2 estimates are statistically significant at the 95% level when the 95% confidence interval for K^2 excludes zero.

In the lag regressions, the Durbin Watson *d* was consistently above 2.00 (most of the time, between 2.4 and 2.8), indicating again that the residuals have negative autocorrelation and, therefore, the regression results are biased *against* statistical significance.

Results of models including up to 15 lags (table 3 shows only the results up to lag 6, the remainder are not shown), were computed, but they do not reveal long-lagged significant effects. A few statistically significant effects at very long lags appear without any pattern, as is to be expected due to random variability.

Modeling the impact of economic growth on health progress for half-century samples and sex-specific health indicators in coincidental models without lags, and in models including one or two lags (results not shown), the results again show a general pattern of reversal of signs, though with particularities for different health indicators. For several age-and-sex-specific death rates, the negative effects of economic growth on health progress at lag two in the second half of the 20th century are marginally significant. For infant mortality, the reversal seems to happen at the turn of the 20th century (table 3) and no significant effects of GDP growth on infant mortality are observable after 1950. On mortality at specific ages 15–24, 35–54, and 70–89, the results show an impressive positive impact of economic growth in 1801–1850, particularly in the decline of mortality of young men and women aged 15–24. However, for the elderly aged 70–89, effects are no longer significant in the three half-centuries after 1850. In 1851–1900 and 1901–1950, positive effects at lag zero are still predominant for ages 15–24 and 35–54, though many are not significant, and for males aged 35–54 a marginally significant negative effect appears at lag two. After 1950, negative effects of economic growth on the decline in mortality predominate at ages 15–24 and 35–54, some of them being statistically significant at the 90% level of confidence.

Models using the annual change in unemployment as the indicator of the dynamic status of the economy (table 4) confirm that in the 20th century "the economy" has a lagged effect on health progress, with the lag being two years in the first half of the century and just one year after 1950. Models using the rate of change of unemployment as the economic indicator produce very similar results (not shown), but when unemployment in levels is used as the explanatory variable, no effects appear.

For the Swedish economy the correlation between annual GDP growth and the annual rate of change in the GDP deflator used as the inflation index is – 0.76 ($P < 0.001$) in 1801–1850, – 0.21 (P = 0.15) in 1851–1900, – 0.32 ($P = 0.02$) in 1901–1950, and – 0.26 ($P = 0.07$) in 1951–1998. That means that, generally, recessionary years are inflationary and expansionary years are deflationary (the correlation for the whole two-century sample is – 0.35, $P < 0.001$), but the relation is very intense in the first half of the 19th century, intense in the first half of the 20th century, and quite weak in the second half of both centuries.

Models in which gains in longevity are regressed on inflation (table 5) reveal a strong negative impact of increasing prices on the rate of improvement in health at lags zero and one in the years 1851–1900 and 1901–1950. In the first half of the 19th century, inflation does not seem to have significant effects either on gains in longevity (table 5) or on infant mortality declines, though it has statistically significant harmful effects on death rates at ages 15–24, 35–54, and 70–89 (not shown). In general, while inflation seems to have harmful effects on mortality at short lags of one or two years in the 19th century, particularly in the second half of the century, in the 20th century the effects are much weaker, almost disappearing after 1950. All this is also consistent with a regime change during the 20th century.

Models to explore reverse causality in which economic growth is regressed on coincidental and lagged values of health progress (not shown) reveal strong significant positive effects at lag zero in the three half-centuries before 1950, and marginally significant negative effects after 1950. These zero-lag effects may of course go in any direction, and they are indeed probably revealing effects in the direction from the economy to health. Some positive lagged effects of health on economic growth appear at lags three and four in the first half of the two centuries. This may suggest a stimulating effect of health progress on economic growth a few years later, though the effect in the first half of the 20th century may well be a consequence of a statistical artifact (see the section on Granger causality in the appendix).

4.5. Models with moving averages

It might be that the average level of economic growth during *k* years had an impact on the average progress of health during those years, or that the average economic growth during *k* years had an impact on the rate of change of health at the end of that period. It seems theoretically conceivable that this kind of averaged effect would not appear in the regression models already presented. With this rationale, economic growth and health progress were averaged with centered moving means in periods of 5, 11, and 15 years, and the correlations between these moving averages were computed (table 6, panel A). Confirming the evidence already presented, the pattern of such correlations provides strong evidence of a reversal in the relation between economic growth and health progress: for all averaging lengths and for all health indicators considered, the correlations are strongly positive in 1801–1850, negative in 1951–1999, and show intermediate values in the other two half centuries. For mortality at ages 70–89, the pattern is, again, much weaker. When GDP per capita growth is averaged (with moving means) in five-year windows and is plotted together with the rate of change of life expectancy at birth similarly smoothed (figure 2) the relation between the two variables appears clearly reversing from a strongly positive correlation (0.62) in the first half of the 19th century to a negative correlation (−0.33) in the second half of the 20th century.

If we consider that the effect of economic growth in *k* consecutive years will have an impact on health conditions at the end of that period, then what we need to look at is the correlation between average economic growth in periods of *k* years and health progress at the end of that period. However, the correlations of 5-, 11-, and 15-year moving averages of GDP per capita growth with the progress in health at the end of the considered period (table 6, panel B) are substantially smaller in magnitude than those for table 6, panel A. If there is any pattern in these correlations, it is precisely in the second half of the 20th century, where the correlations are slightly stronger, but showing, if anything, a negative association between economic growth and improvement in health.

The windows chosen for computing the moving averages are, of course, arbitrary, but if lagged effects of economic growth on health exist, it would be hard to conceive that those effects occur beyond 15 years. At any rate, such an effect appeared neither in cross-correlations nor in lag regressions, and, therefore, there is no evidence to support its existence.

5. Discussion and conclusions

The consistency of results using different statistical models and different economic and health indicators makes it very unlikely that the described reversal of the relation between economic growth and health progress is a spurious finding. All the models are consistent with a weakening of the strong positive association between economic growth and health progress found in the first half of the 19th century, and with a reversal of the relation between economic growth and health progress, which becomes negative in the last half of the 20th century. There seem to be some reverse effects of health progress on economic growth, with declines in mortality stimulating economic growth three or four years latter, but these effects are no longer observable in the most recent half century.

Therefore, this investigation shows that, year to year, economic growth was strongly associated with health progress in Sweden in the first half of the 19th century, with the association becoming weaker and weaker in the next hundred years, to be substituted by a negative lagged association in the second half of the 20th century, in which economic growth has a negative effect on health progress with a short lag of about one or two years. There is no evidence of effects of economic growth on health at longer lags in any of the periods of the two centuries included in the study.

What might be the reasons that the decline in mortality, strongly stimulated by economic growth in 19th-century Sweden, becomes negatively affected by economic growth in the last half of the 20th century? A brief answer may be that economic growth and affluence strongly reduce mortality at the population level when most deaths are due to infectious disease (as in largely agricultural 19th-century Sweden), but that increased business and industrial activity induce higher death rates when most fatalities are due to such causes as cardiovascular disease, cancer, traffic

injuries, diabetes, liver cirrhosis, and other pathologic processes related to work, consumption, or the environment. A more detailed examination of potential pathways is presented in the appendix.

The negative association found between economic growth and health progress in the most recent half century, though quite at odds with traditional views of the relation between economic growth and improvement in the various dimensions of human well-being, is consistent with modern studies revealing a short-term tendency of death rates to increase during economic expansions in industrialized countries in recent decades (Graham et al., 1992; Abdala et al., 2000; Ruhm 2000 and 2003; Chay and Greenstone, 2003; Dehejia and Lleras-Muney, 2004; Gerdtham and Ruhm, 2006; Laporte 2004; Gerdtham and Ruhm, 2002; Neumayer, 2004; Tapia Granados, 2005a and 2005b). Taking into account all these empirical results, it is indeed quite possible that a reversal of the relation between economic growth and health progress like the one described here in Sweden had occurred during the 20th century in other countries. Analyzing the British experience of health progress between 1900 and 1970, Amartya Sen concluded that the rate of decline of mortality in England and Wales during these eight decades was inversely related to economic growth, with decades of stronger growth associated with lower increases in life expectancy (Sen, 2001). This finding would also be consistent with a reversal of the relation between GDP growth and health advancement in Britain.

Life expectancy and age-specific mortality rates are among the most objective and solid components of that unobservable Holy Grail variously referred to by social scientists as "social welfare," "societal utility," "total ophelimity," or, more plainly, "common good." In the 1980s a famous economist and once governor of the Bank of Greece, Xenophon Zolotas, concluded that, in terms of social welfare, economic growth has diminishing and, eventually, negative returns (Zolotas, 1981). What this investigation shows is that economic growth can, and in Sweden's recent past did, produce detrimental effects on the evolution of such a major component of social welfare as life expectancy at birth and the probability of survival at various ages.

To consider the implications of the negative relation between economic growth and health advancement in a modern economy like Sweden raises difficult questions that go well beyond the scope of the present paper. Since the stakes are high and the policy implications of these findings are substantial, a key issue is to ascertain if this switch of the relation between growth and health evidenced in Sweden is also observable in other countries. For nations at low or medium levels of income and population health, it becomes a major issue to ascertain if they have reached the threshold where economic growth no longer promotes improvements in health.

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APPENDIX

I. Pathways connecting harvests, inflation, economic growth, and health progress. To disentangle the complex relations between health progress, harvest fluctuations, economic growth, inflation, and many other potential factors that may be involved in population health in Sweden during the two centuries included in the study is beyond the scope of this paper, but an outline of these relations will be presented here.

The correlations between economic growth, inflation, and harvest quality (table A1) show clearly the high degree of dependence of the Swedish economy on agriculture during the 19th century, particularly during its first half.

In the 19th century, the quality of harvests affected the economy contemporaneously and with a one-year lag, and had an impact on mortality the same year. Both in year-to-year terms (table A1, figure A1) and in 5- or 11-year moving averages (not shown), the harvest quality and inflation are negatively related, with the correlation being much stronger in the first half of the century. For instance, the correlations between the general crop index and the rate of inflation one year later are respectively – 0.57 ($P < 0.001$) and – 0.14 in the two halves of the century. Lack of ability to store grain from harvests of good years made good harvest years times of waste and falling prices, while scarcity after bad harvests had a strong impact on increasing prices (Thomas, 1941). This link is weakened, however, in the second half of the century, when the development of facilities for grain storage, as well as increasing ability to transport and import grain, considerably diminishes the impact of harvests on the level of prices.

The effect of harvests on health progress is revealed by the positive correlations between the crop index and the progress in health as measured by different indicators (table A1). The crop index is associated at statistically significant levels with the decline in infant mortality in 1801–1850 ($r = 0.36$) and 1825–1874 ($r = 0.47$), and with mortality of the very old in 1801–1850 ($r = 0.29$). As explained almost a century ago by the Swedish demographer Gustav Sundabärg and cited by Dorothy Thomas (1941), in the early 19th century, "if the harvest was good, marriage and birth rates were high and death rates comparatively low... when the harvest failed ... death devastated the land, bearing witness to need and privation and at times even to starvation."

The impact of inflation on mortality during the 19th century (table A1, figure A1) may be therefore largely explained by the link between the quality of harvests and the rate of inflation. That link between harvests and inflation weakens throughout the century, and then seems to reassert itself in the first half of the 20th century, when, on the other hand, the connection between harvests and mortality no longer operates, at least at the strong levels observable in infant mortality and mortality of the elderly up to the 1870s.

In summary, during the first half of the 19th century, the Swedish economy was highly dependent on agriculture, as proved (table A1) by the degree to which good harvests increased GDP ($r = 0.31$, $P < 0.05$) or GDP per capita (0.40, $P < 0.01$), diminished the level of prices ($r = – 0.38$, $P < 0.01$), and reduced mortality, for instance of infants ($r = 0.36$, $P < 0.05$) and the elderly aged 70–89 ($r = 0.29$, $P < 0.05$). On the other hand, increases in prices raised mortality less than in 1851–1900, probably because the food supply depended little on markets and much more on production for self-consumption. In the second half of the 19th century, the general level of economic activity was less dependent on harvests, probably because of a higher capacity to save food from years of good harvest and the ability to import (Thomas, 1941).

II. GDP estimates. GDP is always an imprecise measure of the size of the economy, much more so when historical data implying many rough estimates are considered. It could therefore be argued that the pattern of correlations found may be just a fluke produced by a series of volume GDP not measuring appropriately the growth of the Swedish economy. However, errors in measurement tend to blur relations between variables, not to create patterns like those found in this investigation. On the other hand, another series of historical GDP values for Sweden is available, though this one, by Maddison (2003), covers the 19th century from 1820 only. The correlation of annual economic growth computed with data from the series by Maddison (who used estimates by Krantz as one of his sources) and GDP growth computed with Krantz's values is 0.59 for all the overlapping years, 1821–1998; for subperiods the correlations are stronger as we get closer to

the present (1820–1900, 0.40; 1901–1999, 0.77; 1851–1900, 0.34; 1901–1950, 0.76, 1951–1999, 0.84, $P < 0.05$ for the period 1851–1900 and $P < 0.001$ for all other correlations). Since the correlations between economic growth rates computed from the two sources are quite low in some subperiods, it might be expected that when GDP growth from the Maddison data is used for the analysis, the estimates of the economic impact on health will change considerably. That was indeed tested and found. However, the correlations (not shown), though different, reveal a similar pattern of change through the subperiods of the study. With data either from Krantz or from Maddison, economic growth associates positively with health progress in the 19th century, with the association weakening as time passes and switching to negative after 1950**.**

III. Granger causality. The Granger causality test is often used in econometrics to ascertain causality, though deep controversies remain on how to interpret the test and the concept of causality itself. Some econometricians prefer to refer to the Granger test just as a method to establish "precedence," while others consider it a way to ascertain "predictive causality" or just "causality." In brief, it is said that *x* Granger-causes *y* if a regression of *y* on lagged values of *y* and *x* (expanded model) predicts the values of *y* better than a regression of *y* on its past values (restricted model). An *F* test is used to compare the two models (Gujarati, 2003).

We applied Granger causality tests to each of the two centuries separately and to the second half of the century, exploring effects lagged up to 10 years. These tests (results not shown) show strong evidence of economic growth Granger-causing health progress in each of the three periods considered. The specifications in which the null hypothesis of no effect is rejected by the *F* test are those with only one lag included in the 19th century, and with three or four lags in the 20th century, considering it in whole or in its second half. Granger-causality tests do not provide any evidence on the positive or negative sign of these effects, but the global pattern of Granger-causation from economic growth to health seems quite consistent with the regime change during the 20th century indicated by other statistical procedures.

In the opposite direction there is no evidence of Granger-causality in the 19th century or in the second half of the 20th century, but health progress Granger-causes economic growth in specifications with three or four lags in the first half of the 20th century. This evidence of Granger-causality from health to economic growth, also appearing as an effect in lag regressions, is very sensitive to excluding the 1910s. This suggests that this effect can be just a statistical artifact due to the fact that two bad years in terms of mortality, 1915 and 1918, in which life expectancy dropped respectively 1.1 and 9.1 years (1918 was the year of the world flu pandemics) were followed three years later by strong recessions in which GDP per capita shrunk 7.3% (in 1918) and 6.2% (in 1921). These recessions have been explained by reduced demand for Swedish exports related to the 1914–1918 war and its aftermath (Berend 2006).

In summary, Granger-causality tests are mostly consistent with regressions and correlations suggesting that the causal effects are mainly from the economy to health. The conclusion that economic growth no longer has consistent positive effects on longevity in the most recent decades is even stronger if part of the observed association is due to reverse causation, as long as the effect of health on the economy is positive.

Table 1
Parameter estimates (with standard errors, SE) of models in which the annual health progress (either *absolute gain* in years of life expectancy or *relative decline* in mortality) is regressed on a constant, time *t,* economic growth per capita *g,* and the interaction of time and economic growth, $g \cdot t$. For the explanation of the tipping point, see text.

Dependent variable	Sample (centuries)	Growth of GDP per capita (g)		Interaction $g \cdot t$		Tipping point	
		Parameter estimate	(SE)	Parameter estimate	(SE)	Parameter estimate	(SE)
Life expectancy	19th	55.5**	(19.0)	-0.510	(0.325)	1909	(40.8)
	20th	53.7*	(21.9)	-0.330*	(0.157)	1963	(16.6)
	19th & 20th	44.5***	(10.3)	-0.275**	(0.094)	1962	(27.8)
Life exp. females	19th	52.4**	(19.1)	-0.447	(0.326)	1917	(52.3)
	20th	48.6*	(21.6)	-0.297	(0.154)	1964	(18.5)
	19th & 20th	43.4**	(10.3)	-0.266**	(0.094)	1963	(29.1)
Life exp. males	19th	58.1**	(19.0)	-0.565	(0.324)	1903	(33.7)
	20th	58.5*	(22.5)	-0.361*	(0.160)	1962	(15.3)
	19th & 20th	45.4***	(10.4)	-0.282**	(0.094)	1961	(27.0)
Infant mortality	19th	1.5*	(0.7)	-0.012	(0.012)	1926	(79.6)
	20th	0.2	(1.1)	-0.001	(0.008)	...[a]	...
	19th & 20th	1.2**	(0.4)	-0.007*	(0.004)	1956	(37.7)
Mort. 15-24 females	19th	2.7**	(0.8)	-0.025	(0.015)	1907	(36.1)
	20th	3.8	(2.6)	-0.022	(0.019)	1976	(39.4)
	19th & 20th	2.0**	(0.7)	-0.010	(0.006)	...[a]	...
Mort. 15-24 males	19th	4.5***	(0.9)	-0.056***	(0.015)	1880	(11.0)
	20th	5.5*	(2.3)	-0.032	(0.017)	1974	(22.6)
	19th & 20th	2.8***	(0.6)	-0.015*	(0.006)	1987	(40.5)
Mort. 35-54 females	19th	3.2***	(0.8)	-0.038**	(0.014)	1883	(15.5)
	20th	2.3*	(1.1)	-0.014	(0.008)	1957	(17.3)
	19th & 20th	2.1***	(0.5)	-0.014***	(0.004)	1945	(20.6)
Mort. 35-54 males	19th	2.9**	(0.8)	-0.037**	(0.014)	1878	(14.5)
	20th	3.2**	(1.2)	-0.020*	(0.008)	1956	(12.6)
	19th & 20th	1.9***	(0.5)	-0.012**	(0.004)	1949	(24.8y
Mort. 70-89 females	19th	1.9***	(0.6)	-0.023*	(0.011)	1882	(18.9)
	20th	-0.1	(0.9)	0.000	(0.006)	...[a]	...
	19th & 20th	1.2**	(0.4)	-0.009**	(0.003)	1926	(20.0)
Mort. 70-89 males	19th	2.0***	(0.6)	-0.023*	(0.010)	1884	(17.5)
	20th	0.2	(0.8)	-0.002	(0.005)	1880	(151.9)
	19th & 20th	1.3***	(0.3)	-0.010***	(0.003)	1923	(15.5)

*$P < 0.05$ **$P < 0.01$ ***$P < 0.001$. Durbin-Watson $d > 2.0$ in all regressions.

[a] The tipping point is not well defined when the interaction is close to zero.

Table 2
Parameter estimates (with standard errors, SE) of models in which the annual health progress (either *absolute gain* in years of life expectancy or *relative decline* in mortality) is regressed on a constant, the log of GDP per capita, the growth of GDP per capita (g), and the interaction of these two ($g \cdot$ log GDPpc). For the explanation of the tipping point, see text.

Dependent variable	Sample (centuries)	Growth of GDP per capita (g)		Interaction $g \cdot$ log(GDPpc)		Tipping point	
		Parameter estimate	(SE)	Parameter estimate	(SE)	Estimate	(SE)
Life expectancy	19th	230.8	(116.5)	-62.3	(35.8)	1890	(16.4)
	20th	70.8*	(30.3)	-13.1*	(6.3)	1961	(16.8)
	19th & 20th	76.5***	(21.9)	-14.5**	(5.3)	1956	(21.1)
Life exp. females	19th	215.3	(117.1)	-57.5	(36.0)	1892	(18.5)
	20th	63.7*	(29.8)	-11.7	(6.2)	1962	(19.0)
	19th & 20th	75.1***	(21.9)	-14.3*	(5.3)	1956	(21.6)
Life exp. males	19th	244.3*	(116.3)	-66.5	(35.8)	1889	(14.8)
	20th	77.5*	(31.0)	-14.4*	(6.5)	1960	(15.2)
	19th & 20th	77.6***	(22.0)	-14.8*	(5.3)	1956	(20.8)
Infant mortality	19th	6.2	(4.2)	-1.6	(1.3)	1894	(24.2)
	20th	0.20	(1.5)	-0.0	(0.3)	...[a]	...
	19th & 20th	2.06*	(0.8)	-0.4	(0.2)	1953	(28.3)

*$P < 0.05$ **$P < 0.01$ ***$P < 0.001$. Durbin-Watson $d > 2.0$ in all regressions.
[a] The tipping point is not well defined when the interaction is close to zero.

Table 3

Parameter estimates in lag regression models $\Delta h_t = \alpha + \sum_{i=0}^{k} \beta_i \cdot g_{t-i}$ to estimate the lagged effect of economic growth (annual percentage change in GDP per capita, $g = \Delta \ln \text{GDPpc}_{t-i}$) on health progress (as measured by a health indicator h_t).

	Dependent variable: annual gain in life expectancy at birth							Dependent variable: annual rate of decline of mortality 35-54						
	Number of lags included in the regression							Number of lags included in the regression						
	0	1	2	3	4	5	15[a]	0	1	2	3	4	5	15
	Sample 19th century													
β_0	27.5**	30.3**	29.1**	28.3**	29.5**	29.3**	18.2	1.03**	1.13**	1.10**	1.06**	1.06*	1.06*	0.42
β_1		17.3	18.3*	18.1*	14.6	14.7	13.6		0.65†	0.67†	0.67†	0.60	0.63	0.51
β_2			–4.2	–4.4	–5.6	–5.4	–9.4			– 0.16	– 0.18	– 0.19	– 0.21	– 0.03
β_3				–3.9	–4.7	–4.8	-10.4				– 0.15	– 0.19	– 0.21	–0.67
β_4					–16.3†	–16.0†	–11.3					– 0.25	– 0.23	– 0.13
β_5						–0.4	–4.5						– 0.18	– 0.10
β_6							–1.5							–0.07
	Sample 20th century													
β_0	8.3*	8.5*	7.2†	6.7	6.8	5.7	1.1	0.32	0.34†	0.27	0.28	0.29	0.21	0.00
β_1		–1.3	0.3	0.8	0.9	1.0	6.3		– 0.12	– 0.05	– 0.06	– 0.08	– 0.07	0.16
β_2			–9.4*	-9.9*	-10.1**	–9.1*	-10.9*			– 0.37†	–0.36†	–0.34	–0.27	–0.38
β_3				2.4	2.4	1.1	-1.0				– 0.05	0.08	– 0.16	– 0.23
β_4					–0.1	1.1	5.5					0.13	0.21	0.39
β_5						5.6	– 8.8†						– 0.39†	–0.51*
β_6							3.2							0.13
	Sample 1951-1998													
β_0	–1.1	– 0.7	– 0.3	0.1	0.0	0.1		– 0.26†	– 0.23	– 0.21	– 0.13	– 0.15	– 0.11	
β_1		– 1.3	– 1.6	– 2.2	– 2.3	–2.5			– 0.11	– 0.16	– 0.23	– 0.20	– 0.24	
β_2			1.5	1.4	1.7	2.0				0.10	0.27†	0.25	0.30†	
β_3				– 2.2	– 2.6	–2.8					– 0.42**	– 0.38*	– 0.43*	
β_4					0.5	0.9						– 0.12	–0.02	
β_5						–1.0							– 0.21	

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$ † $P < 0.1$

[a] Only the estimates for lags zero to six are shown for the specification including 15 lags in models for a whole century.

Table 3 (cont.).

		Number of lags in the regression						Number of lags in the regression				
		0	1	2	3	4		0	1	2	3	4
		Sample 19th century						*Sample 20th century*				
Dependent variable: infant	β_0	0.84**	0.89**	0.85*	0.83*	0.86*		0.09	0.10	0.07	0.04	0.03
	β_1		–0.17	–0.16	–0.17	–0.24			–0.11	–0.06	–0.03	–0.01
	β_2			–0.41	–0.42	-0.45				–0.35†	-0.40*	-0.42*
	β_3				–0.10	–0.11					–0.26	0.29
	β_4					–0.35						–0.13
		Sample 19th century						*Sample 20th century*				
Dependent variable: mortality 15-24	β_0	1.43***	1.51***	1.45***	1.42***	1.44**		1.00**	1.01*	0.87*	0.81†	0.82†
	β_1		0.68	0.67	0.69	0.54			–0.07	–0.08	–0.15	–0.15
	β_2			–0.68	–0.70	–0.73†				-0.83*	-0.90*	-0.91*
	β_3				–0.07	–0.13					0.33	0.32
	β_4					–0.60						0.07
		Sample 19th century						*Sample 20th century*				
Dependent variable: mortality 70-89	β_0	0.67*	0.76*	0.74**	0.71*	0.71*		– 0.11	–0.09	– 0.15	–0.18	–0.17
	β_1		0.22	0.22	– 0.20	– 0.15			–0.11	–0.05	–0.02	–0.04
	β_2			– 0.22	– 0.21	– 0.21				-0.27†	-0.31†	-0.29†
	β_3				– 0.15	– 0.19					0.18	0.15
	β_4					– 0.17						0.15
		Sample 1950-1998						*Sample 1950-1998*				
Dependent variable: infant mortality	β_0	– 0.12	– 0.12	– 0.12	– 0.03	0.00	Dependent variable: mortality 15-24	– 0.28	– 0.21	– 0.24	– 0.26	– 0.44
	β_1		0.04	0.10	0.02	0.01			– 0.19	0.09	0.07	0.23
	β_2			- 0.19	0.02	0.01				– 0.85†	– 0.92	– 0.95†
	β_3				– 0.55	– 0.57					0.27	0.54
	β_4					0.07						– 0.81

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$ † $P < 0.1$

Table 4

Regression results in lag models $\Delta e_{0,t} = \alpha + \sum_{i=0}^{k} \beta_i \cdot \Delta u_{t-i}$ in which health progress, as measured by the annual gain in life expectancy at birth ($\Delta e_{0,t}$), is regressed on a constant and the change in the unemployment rate (Δu_t), in specifications with k lags, $k \leq 3$.

	1911-1999				1911-1950				1951-1999			
k =	0	1	2	3	0	1	2	3	0	1	2	3
β_0	0.043	0.041	0.104	0.102	0.047	0.046	0.122	0.118	0.024	– 0.015	– 0.029	– 0.031
β_1		0.014	– 0.013	– 0.052		0.013	-0.007	– 0.049		0.083	0.106†	0.126†
β_2			0.217*	0.233*			0.242	0.252†			– 0.026	– 0.067
β_3				–0.133				– 0.133				0.067
R^2	0.003	0.003	0.072	0.098	0.003	0.004	0.082	0.108	0.006	0.057	0.074	0.100

* $P < 0.05$ † $P < 0.1$

Table 5

Regression results in lag models $\Delta e_{0,t} = \alpha + \sum_{i=0}^{k} \beta_i \cdot \Delta \log d_{t-i}$ in which health progress (as measured by annual gain in life expectancy at birth) is regressed on a constant and inflation (as measured by the log difference in the GDP deflator), in specifications with k lags, $k \leq 2$.

	1801-1850			1851-1900			1901-1950			1950-1999		
k =	0	1	2	0	1	2	0	1	2	0	1	2
β_0	–12.2	–12.2	– 11.1	– 22.1**	–12.7†	– 11.2	– 5.9*	– 14.3***	– 17.3***	0.13	– 0.40	0.05
β_1		–4.0	– 5.5		– 19.8**	– 21.7*		12.2***	19.3***		0.04	– 0.09
β_2			1.00			3.5			–0.19†			– 0.45
R^2	0.05	0.07	0.07	0.16	0.29	0.29	0.09	0.28	0.35	0.00	0.01	0.01

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$ † $P < 0.1$

Table 6
Correlations between health progress and growth of GDP per capita, both smoothed with centered moving means in periods of 5, 11, and 15 years.

		A — Health progress and economic growth both averaged from $t-(k-1)/2$ to $t+(k-1)/2$				*B* — Economic growth averaged from $t-k+1$ to t, correlated with health progress at t			
Annual health progress as measured by	k	1801-1850	1851-1900	1901-1950	1950-1999	1801-1850	1851-1900	1901-1950	1950-1999
Increase in life expectancy	5	0.62	0.25	0.15	− 0.33	−0.08	0.00	0.03	−0.16
	11	0.55	0.03	0.38	− 0.10	0.02	0.09	0.09	− 0.04
	15	0.49	0.23	0.26	−0.02	−0.01	0.11	0.00	−0.16
Relative decline in mortality 15-24	5	0.69	0.12	0.42	− 0.53	0.19	−0.07	0.16	− 0.19
	11	0.65	−0.18	0.55	−0.16	0.10	0.01	0.08	− 0.05
	15	0.63	0.04	0.50	−0.04	0.09	0.04	0.07	− 0.13
Relative decline in mortality 35-54	5	0.59	0.15	0.11	− 0.39	0.18	−0.03	0.05	− 0.32
	11	0.50	− 0.16	0.33	− 0.38	−0.05	0.02	0.08	− 0.23
	15	0.54	− 0.25	0.30	− 0.32	0.04	0.06	0.00	− 0.24
Relative decline in mortality 70-89	5	0.48	0.23	−0.33	−0.25	0.06	−0.06	−0.04	− 0.05
	11	0.25	−0.07	0.06	−0.11	−0.06	−0.01	0.00	0 07
	15	0.21	−0.10	0.07	−0.12	−0.08	0.05	− 0.04	− 0.02

APPENDIX TABLE

Table A1
Correlations between annual indicators of harvest quality (crop index) economic growth (as measured by the rate of change of either GDP or GDP per capita), inflation (rate of change of the GDP deflator), and progress in health as measured by several health indicators.

		Correlation coefficients	
	Period	GDP growth	GDP per capita growth
Inflation and economic growth	1801-1850	– 0.76***	– 0.74***
	1825-1874	– 0.49***	– 0.45***
	1851-1900	– 0.21	– 0.17
	1875-1924	– 0.28†	– 0.23
	1901-1950	– 0.32*	– 0.29*
	1925-1974	– 0.25	– 0.21
	1951-*ca.* 1998	– 0.26†	– 0.15
Crop index and economic growth	1801-1850	0.31*	0.40**
	1825-1874	0.16	0.25
	1851-1900	0.08	0.07
	1875-1924	0.14	0.11
	1901-1950	0.36**	0.36*
Crop index lagged one year and economic growth	1801-1850	0.64***	0.58***
	1825-1874	0.52***	0.48***
	1851-1900	0.56***	0.56***
	1875-1924	0.50***	0.28†
	1901-1950	0.13	0.14
		Crop index at lag zero	Crop index lagged one year
Inflation and crop index	1801-1850	– 0.38**	– 0.57***
	1825-1874	– 0.10	– 0.35*
	1851-1900	– 0.23	– 0.14
	1875-1924	– 0.16	– 0.31*
	1901-1950	– 0.29†	– 0.40**
Relative decline in longevity shortfall and crop index	1801-1850	0.15	0.09
	1825-1874	0.22	0.09
	1851-1900	0.17	0.28†
	1875-1924	0.22	0.12
	1901-1950	0.24	0.02
Gain in life expectancy at birth and crop index	1801-1850	0.15	0.09
	1825-1874	0.22	0.09
	1851-1900	0.17	0.28†
	1875-1924	0.21	0.11
	1901-1950	0.26†	0.06

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$ † $P < 0.1$

Table A1 (Cont.).

		Correlation coefficients	
	Period	Crop index at lag zero	Crop index lagged one year
Relative decline in infant mortality and crop index	1801-1850	0.36*	– 0.06
	1825-1874	0.47**	– 0.16
	1851-1900	0.22	0.09
	1875-1924	– 0.04	0.18
	1901-1950	– 0.09	– 0.08
Relative decline in mortality ages 15-24 and crop index	1801-1850	0.18	0.21
	1825-1874	0.13	0.17
	1851-1900	0.00	0.24
	1875-1924	0.21	0.08
	1901-1950	0.25†	0.06
Relative decline in mortality ages 35-54 and crop index	1801-1850	0.20	0.15
	1825-1874	0.20	0.06
	1851-1900	0.03	0.11
	1875-1924	0.16	0.04
	1901-1950	0.24†	0.02
Relative decline in mortality ages 70-89 and crop index	1801-1850	0.29*	0.12
	1825-1874	0.23	0.19
	1851-1900	– 0.06	0.11
	1875-1924	0.03	0.12
	1901-1950	0.15	– 0.17

* † $P < 0.1$ $P < 0.05$ $^{**}P < 0.01$ $^{***}P < 0.001$

Figure 1
Spectral analysis for the annual rate of decline in mortality 35–54 and economic growth as measured by annual growth of GDP per capita. In the horizontal axis the period is in years. Dotted lines are 95% confidence limits.

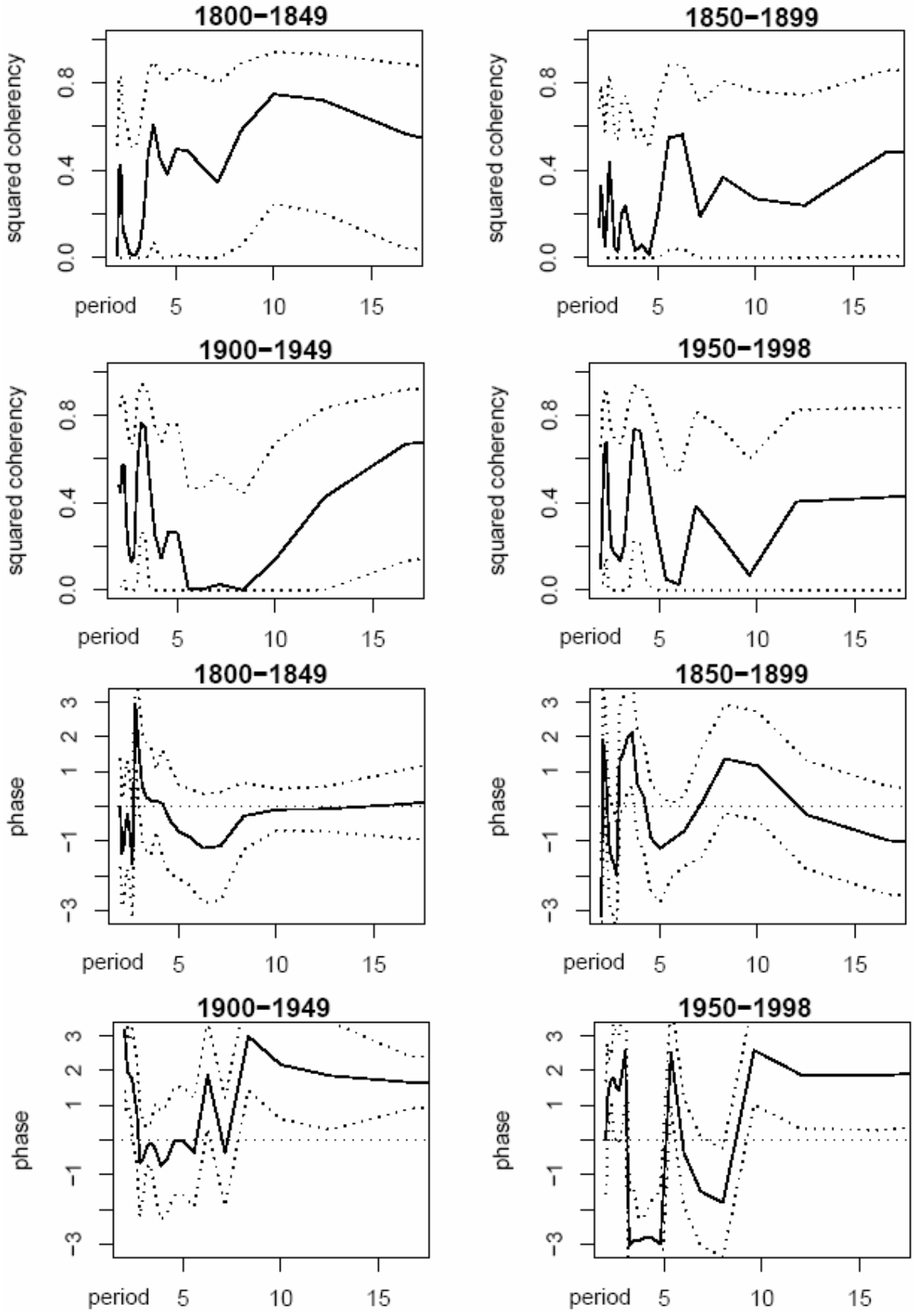
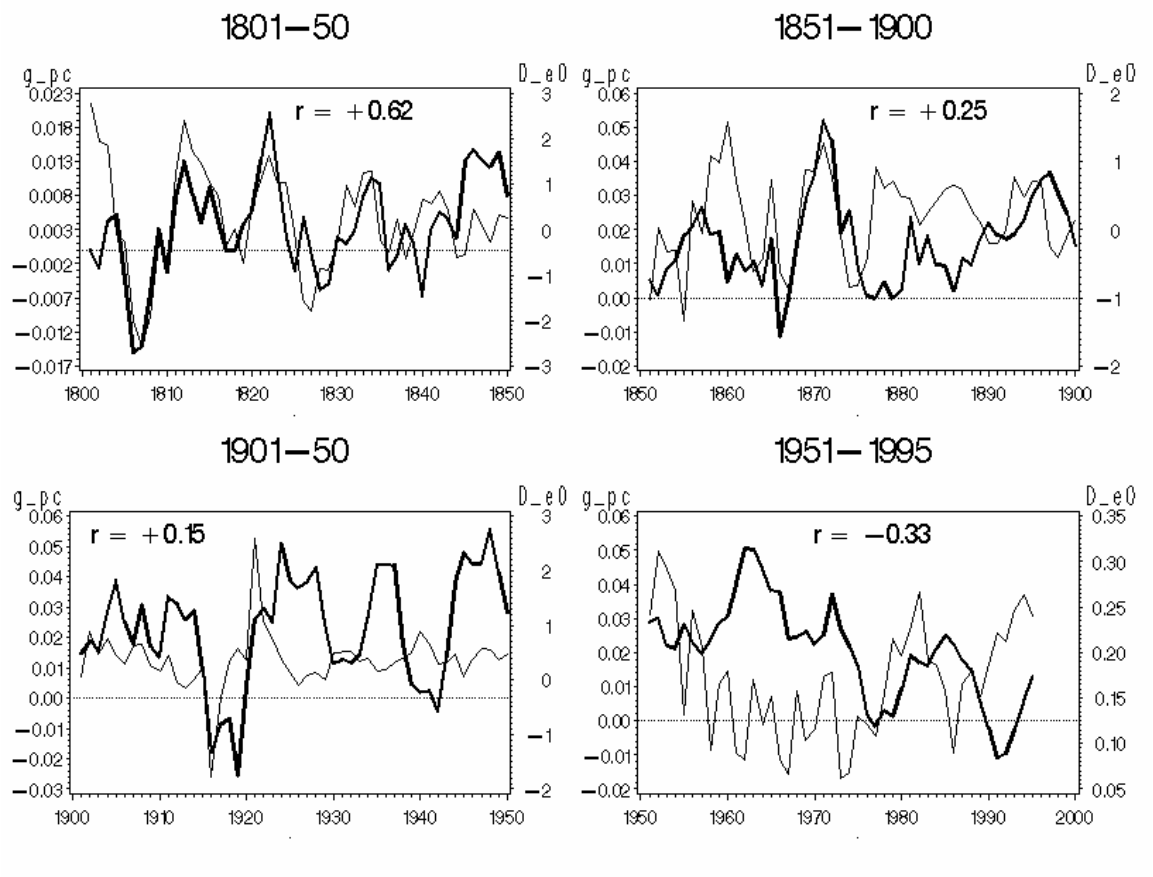
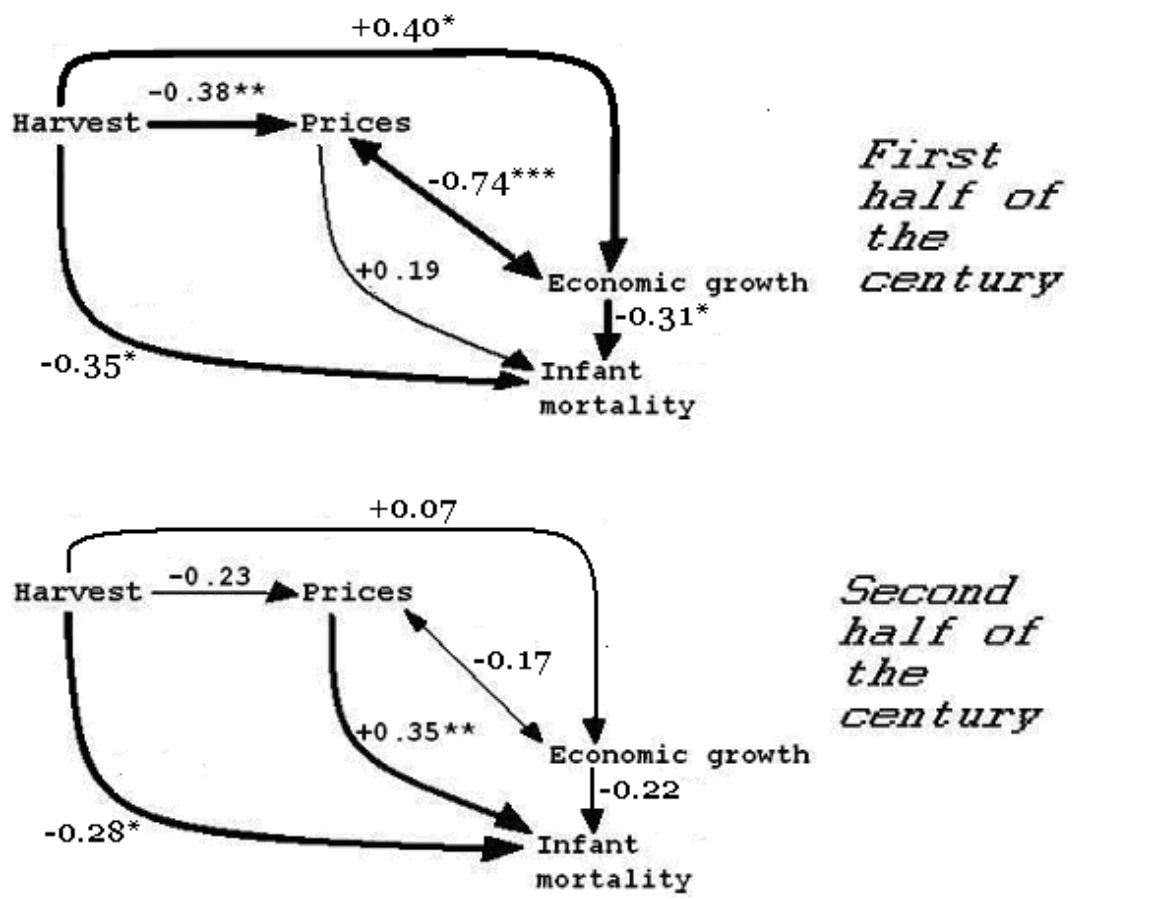


Figure 2
Annual rate of change of GDP per capita (*g_pc,* thick line) and annual gain in life expectancy at birth (*D_e0*) in 19th- and 20th-century Sweden. Both variables are transformed into 5-year centered moving means. Note that scales are different for each variable and in each panel.



APPENDIX FIGURE

Figure A1
Pathways between harvest quality, price level, economic growth, and infant mortality in Sweden during the 19th century. Correlations are computed between annual series of a general crop index in levels (harvest) and the annual rate of change of the GDP deflator (prices), GDP per capita (economic growth), and infant mortality. Thick lines indicate statistically significant correlations.



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