## Why were the 1940s so good and the 1960s so bad? An exploration of 20th century mortality trends in the United States

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#### Abstract

Although the decline of rates of mortality in the United States is considered to be major evidence of progress, not much attention has been paid to the rate of decline of mortality in different decades. In this investigation, the long-term trend in mortality rate was extracted, and rates of long-term mortality decline were computed. The rates of long-term decline of mortality for different demographic groups reveal substantial similarities in the patterns of mortality reduction through time. The 1940s and the 1970s were periods of accelerating decline of mortality for all ages; contrarily, in the 1960s, mortality declined at diminishing rates in all age strata—and even increased substantially for working-age and nonwhite males. At the end of the century, ageadjusted mortality is declining at accelerating rates for males, while it is stagnating at an almost zero rate of decline for women. Possible causes of these phenomena are discussed.

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### Introduction

In most countries, the 20th century was a period of steady decline of death rates for all age strata, with the consequent increase of life expectancy. In the United States, crude mortality fluctuated throughout the 20th century (figure 1), with occasional peaks such as the one produced by the world epidemics of flu in 1918, but its declining long-term trend is obvious until the recent decades, when the aging of population slowed down the long-term declining trend. Age-specific mortality rates continued their long-term decline (figure 2) until the present, though there are considerable differences in the evolution of age-specific mortality rates in different periods.

The decline in death rates has been repeatedly cited as evidence of health progress. However, not much attention has been paid to the differential rates of mortality decline in the United States throughout the decades. In his influential and controversial book on the effects of social inequalities on health, Richard Wilkinson (1996) argued that in the 20th century mortality in the United Kingdom dropped faster during the periods of war, in 1914–1918 and 1939–1945. Also in the British experience during the past century, Amartya Sen (2001) has found that the increase in life expectancy was slower in the decades when economic growth was stronger. The present study investigates the long-term trends in the reduction of mortality rates in the United States during the 20th century, examining specifically the rates of long-term mortality decline. The observed patterns suggest that the long-term evolution of death rates is subjected to influences that are substantially similar for both males and females, and for different age-strata, though the particular patterns of mortality decline shed light on important aspects of health progress through time.

#### Data and methods

Mortality rates are taken from the *Historical Statistics of the United States* (U.S. Bureau of the Census, 1997) and various editions of *Vital Statistics of the United States* (U.S.

Bureau of the Census). Mortality rates included in historical statistics represent civilian deaths only. Deaths caused by war are excluded for the computation of annual rates, which are also correspondingly adjusted in the denominator (U.S. Bureau of the Census, 1945).

Long-term mortality trends were computed with a Hodrick-Prescott (HP) filter, setting the smoothing parameter to 100. The HP filter (figure 1) smoothes the oscillations of a time-series, extracting a long-term trend that tends to a straight line when the smoothing parameter increases, and follows very closely the oscillations of the original series when the parameter is close to zero. For annual data, a smoothing parameter equal to 100 is recommended and commonly used (Backus and Kehoe 1992).

After mortality data were smoothed with the HP filter, the rate of change was computed as the percentage change from the former year, that is,  $100 \cdot (x_t - x_{t-1})/x_{t-1}$ , where  $x_t$  is the transformed value of mortality at the year *t* after applying the HP filter. Since mortality is almost always declining, the long-term *rate of change* so computed was transformed into a *rate of decline* just by multiplying it by -1. To check to what extent the long-term trends of decline computed from the HP-filtered values are sensitive to the smoothing parameter, this was set to several values between 50 and 200 which were then checked with the mortality data. They produced basically the same patterns in the graphs of the rate of decline of mortality.

### Results

The long-term rates of decline of crude mortality and age-adjusted mortality for different groups (figure 3) reveal a neat historical picture. For both men and women, for whites and nonwhites, the largest rates of reduction in mortality occurred during the first half of the 1920s, the mid-1940s, and the mid-1970s, when mortality dropped at rates between 2% and 3% per year, consistently larger for females. The opposite occurred in the 1960s, when age-adjusted mortality stagnated or increased in males, especially nonwhite males, while in females it declined at its smallest rate during the second half of the century. In the 1990s, the increasing rate of decline of male mortality dramatically contrasts with the evaporating improvement for females. Indeed, the rate of decline of age-adjusted male mortality (figure 3, second row) dramatically improves from the late 1980s, when

mortality was dropping around 1% per year, to the late 1990s, when the rate of reduction is above 2% per year. Female mortality, declining at 2.3% per year during the mid 1970s, stagnates at zero decline at the end of the century.

The long-term rates of decline for specific age and sex strata (figure 4) reveal different stories for different age groups, though some patterns are constant across age and sex boundaries. Suggesting that factors determining these patterns may be to a large extent environmental and socially conditioned, the long-term patterns of mortality decline for males and females throughout the century show apparent differences in each age strata, but they are almost indistinguishable for male and female infants below age 1, and very similar for boys and girls aged 5 to 14 years.

Confirming what was suggested by the rates of decline in age-adjusted mortality, compared with previous and later decades the 1940s were an excellent decade in terms of mortality reduction for all age- and sex-specific groups. Similarly, the late 1950s and 1960s were a very bad period, with dwindling declines of mortality for all demographic groups and increasing mortality for young and middle-aged adults, especially males (figure 4, third to sixth rows).

The flu pandemics of 1918–1919 is a determining influence on the evolution of mortality during early decades of the century. In the United States about 700,000 people died in the outbreak (Barry 2004), which strongly affected children, adolescents, and adults below 50, mildly affecting pre-retirement adults, and largely skipping infants and the elderly (figures 2 and 3). Reflecting the wane of the pandemics, in the early 1920s the rate of mortality decline reached peaks for almost all age groups, in both males and females. From there it went substantially down for infants below one and males above 25. During the "Roaring Twenties," the rate of decline of death rates not only slowed down substantially for many age groups, but mortality ceased declining and even rose for males above 55 and, in the early 1920s, for women above 75.

From the early 1930s to the mid-late 1940s, the decline of mortality accelerated for all age groups except males aged 15–24. In the mid-late 1940s, the rate of mortality decline shows peaks in all age groups, for both males and females. Following that golden age of health progress, from the mid-1940s to the mid-late 1960s, there was a substantial decrease in the rate of mortality decline. During the 1960s, mortality *increased* for all

male groups except children below 14 and the elderly. Death rates were also increasing for females aged 15–24 and 35–44.

During the 1970s, the decline of mortality substantially accelerated for all groups (reaching rates up to 3% or 4% per year in infants and children, and between 1% and 3% for adults), but the improvement tended to disappear after the mid-1970s, evaporating during the 1980s. Both males and females of ages 25–44 suffered mortality increases around 1% per year (larger for males) in the late 1980s and early 1990s. A dramatic gender differential appears beginning in the mid-1980s in the evolution of mortality of adults in pre-retirement age or older. Mortality of males aged 55–64 and 75–84 dropped about 1.5% per year, while the corresponding rate was only around 0.5% per year for females in these age groups.

#### Discussion

The long-term patterns of mortality decline reveal large improvements in the 1940s and the 1970s, and diminishing rates of improvement or even net deterioration in the 1960s for all demographic groups. This suggests that these patterns cannot be the effects of causes affecting the risk of death with a lag of many years. Causes having effects with lags of, say, two or three years, might be credible for adults. However, the peaks (1940s, 1970s), and troughs (1950s–1960s) of mortality decline for infants (figure 4) coinciding with those of males and females aged 55-64 or even 75-84 suggest that even these short lags can be too long to be operative. The conclusion must be that the causes ruling these patterns of long-term reduction in mortality have to be largely operating with a very short lag. What can these causes be? The short answer is that we don't know.

It is remarkable that the large acceleration of the decline of mortality during the 1930s and 1940s coincides and follows by only a few years what is arguably the most unstable period in U.S. society in the 20th century, the Great Depression that started in 1929 (figure 5). Industrial production and aggregate output (GDP) contracted in the early 1930s to about half of the levels reached in 1929; unemployment reached a historical record of 24.9% in 1933 and until 1939 was well above 15%. During the 1930s, the rate of joblessness averaged 18.8%, and only in 1942 reached 9.9%, the first unemployment rate below 10% since 1931. Except for infant mortality, which slowed down its decline

from 1920 until the mid-1930s and then substantially accelerated its reduction until the 1940s (figure 4, first row), the decline of mortality during the 1930s accelerated for all age groups except males aged 15–24, in which slightly slowed. In the 1940s, the improvement in mortality reductions was at its highest rates in all age groups.

In a number of contributions in the 1970s and 1980s, in a press release in 1992 and in a forthcoming comment, Harvey Brenner (Brenner 1995; Reuters Health 2002; Brenner 2005) has postulated that increased unemployment translates to lagged increases in mortality with lags of up to 10–15 years—the so-called "Brenner hypothesis" (Tapia Granados 2003). The substantial acceleration of the mortality decline during most of the 1930s and 1940s is strongly at odds with this hypothesis. Unless compensated by other influences, any lagged or unlagged effect of the large increases in unemployment during the 1930s would have had the effect of increasing mortality or at least reducing its rate of decline during the late 1930s and the 1940s. Mortality was, nevertheless, dropping at an increasing rate. The conclusion must be that the human misery caused by the Great Depression of the 1930s appears not to have translated into general mortality increases mortality due to suicide may be a different case (figure 6). Empirical results are sometimes counterintuitive, and this is probably one of the most counterintuitive results given that disease and death are often the consequence of misery, squalor, and poverty.

During the 20th century, the only important decline in income inequality in the United States was the one that took place during "a very specific and brief time interval," the 1930s and early 1940s (Picketty and Saez 2003). The most frequently used index of inequality, the Gini coefficient, reveals a large reduction in inequality during the 1930s and 1940s (figure 5). Since these drops in inequality immediately preceded and coincided with the large acceleration of mortality decline in the 1940s, this might be evidence in favor of Wilkinson's hypothesis—that inequality has harmful effects on population health. Income inequality remained substantially at constant levels or slightly declining from the end of World War II to the late 1960s (figure 5), while age-adjusted mortality decline substantially slowed down in the 1950s (figure 2) reaching its slowest pace in the mid-1960s. Then mortality strongly accelerated its decline from the mid-1960s to the mid-1970s, a period in which inequality was constant or slightly increasing. From the mid-1980s to the late 1990s, the decline of age-adjusted mortality slightly accelerated

(differing strongly in males and females), while inequality measured by the Gini coefficient or other indicators strongly increased. Therefore, while the 1930s-1940s experience seems to support Wilkinson's hypothesis, that of the second half of the century seems to be quite inconsistent with it.

The evolution of mortality due to particular causes (figure 6) may throw some light on the factors contributing to the patterns of long-term decline in mortality. Given the large contribution of deaths due to cardiovascular disease (CVD) to total mortality, the long-term maximum that CVD mortality reached in the 1960s is obviously a major factor explaining the poor performance of mortality decline in this decade. However, given the very small share of this group of causes of deaths in mortality of children and young people, it is unlikely that the epidemics of cardiovascular deaths peaking in the 1960s is an important factor to explain the slowing down and even reversal of the decline of mortality in these age groups from the mid-1940s to the 1960s. Indeed, homicides, suicides, and traffic injuries were identified as major causes of the rise in mortality of young people during the 1960s (Waldron and Eyer 1975).

The prohibition of sale and consumption of alcoholic beverages started in 1920 and lasted until 1933. Mortality owing to cirrhosis of the liver which had been dropping from the early years of the century was already at its lowest historical levels in 1920 (figure 6) and remained mostly unchanged or lightly increasing in waves from the baseline levels reached in 1920 all throughout the 1920s and early 1930s. It seems therefore doubtful that Prohibition had a significant impact on this cause of death. An associated impact of Prohibition in diagnostic fashions reflected in death certificates (which is a quite likely possibility) would even make it possible that real mortality due to cirrhosis had been increasing to some extent during the Prohibition years, though that was not reflected in registered mortality figures. Moreover, during the years 1920-1933 the decline of mortality substantially weakened for all male age groups above 25 and mortality even started increasing for males over 55 (figure 4). Contrarily, the decline of mortality moderately accelerated for most age groups of females. All this seems to be evidence against a noticeable impact of Prohibition on the evolution of general mortality rates, since such an impact would probably be reflected in a stronger acceleration of mortality decline in males, on whom presumably Prohibition would have a much stronger

impact in reducing alcohol intake.

Liver cirrhosis is not a major cause of death, accounting for only between 0.5% and 1.7% of all annual deaths throughout the century. Therefore, though the substantial increase in cirrhosis mortality from the early 1950s to the mid-1970s (figure 6) might be one of the factors accounting for the poor evolution of adult mortality in the 1960s, especially in males and young women, who suffered increasing mortality during this decade, it cannot be a major one. However, the historical rise in liver cirrhosis mortality from the early 1950s to the mid-1970s and its subsequent fall does seem to be related to the per capita consumption of distilled spirits, which increased during the post-war years until the 1970s and then dropped (Roizen et al. 2005). Since, besides liver cirrhosis, alcohol consumption is causally related (with lags varying from zero to a few years) with increased risk of CVD, injuries, cancer, and even infections and other diseases in which the immune-depressing effect of alcohol may have a pathophysiological role, alcohol might be a major factor contributing to the poor evolution of general mortality during the 1960s. Ruhm and Black (1991) have proved that overall drinking increases in periods of strong economic growth, with this effect being mostly dependent on greater consumption among existing drinkers rather than movements from the pool of previous non-drinkers to the pool of drinkers. Given that most of the 1950s and 1960s were years of strong economic growth, the increasing levels of drinking during that period might reflect mostly patterns of heavy drinking that through a variety of pathways contributed to the slowly declining, stagnating, or even increasing mortality rates during the 1960s.

Through cancer, CVD, and traffic injuries, cigarettes and cars are two of the major killers in 20th-century societies. Even accepting that the effects of secondary smoke may be significant on nonsmokers, tobacco smoking can hardly explain simultaneous accelerations or reductions in the pace of mortality decline of infants, children, adults, and the elderly. Traffic injuries, however, are a different issue.

The death rate due to traffic injuries steadily grew from the early decades of the century to its highest levels in the late 1930s (figure 6). Epidemiologists have long known that traffic mortality increases during economic booms and drops during recessions (Baker et al. 1984; Robertson 1992). Indeed, the historical rise of traffic mortality connected to the generalization of cars and road transportation was dramatically halted by

the Great Depression. Traffic deaths increased again in the boom of the early 1940s and dramatically dropped in the years of U.S. participation in World War II, when traffic mortality was almost halved, to 17.7 per 100,000 in 1943 from 30.0 in 1941, as result of gas rationing and reduction of traffic volume (figure 5). Traffic fatalities steadily increased again during the 1960s, reaching a plateau in the early 1970s, and then dramatically dropped in 1975 under the combined pressure of gas shortages (related to the Yon-Kippur War) and lower speed limits. In the 1980s, traffic mortality started a slightly declining path with strong oscillations linked to the fluctuations of the economy, probably led by the beneficial effects of the introduction of safety belts and other car safety measures.

Since traffic fatalities are often young persons, and traffic injuries often result in long-term disability, traffic injuries are a major cause of healthy life lost. However, mortality due to traffic injuries never accounted for more than 3% of all annual deaths, and therefore can hardly explain the patterns of long-term changes in mortality during the 20th century. Nevertheless, since traffic fatalities happen at all ages, they are a potential explanatory factor of coinciding peaks or troughs of the rate of decline of age-specific mortality rates. Indeed, the accelerating rates of mortality decline during the late 1930s coincided with the halt in rising traffic mortality related to the Great Depression, the peak in mortality decline in the mid-1940s coincided with the drop in traffic mortality associated with World War II, and the troughs in mortality decline of the 1960s coincided with substantially rising traffic mortality (figure 6).

The change in the social position of different groups may perhaps be an explanatory factor of the different patterns of mortality decline. For instance, the substantial differences in the evolution of mortality of males and females in the most recent decades might be related to the major changes in the social status of women and their participation in labor markets. On the other hand, in the period from the mid-1960s to the mid-1970s, there were substantial improvements in the social status of nonwhites associated with the civil rights movement. In that period there are also significant differences in the decline of mortality rates of whites and nonwhites. Mortality of white males (figure 3) accelerated its decline approximately 2 percentage points (from zero to about 2% per year) while that of non whites accelerated about 3 percentage points (from -

-0.5% to +2.5%). For white women the rate of decline of mortality was accelerated 1.5 percentage points (from about 0.7% to 2.2%), while that of non white women improved 2.5 percentage points (from 0.8% to 3.3%). For both men and women, the improvement between the early to mid-1960s and the mid-1970s is larger for non whites. Data are at least compatible with a positive effect of the civil rights movement on the health of non white U.S. citizens.

Using panel regressions with data from the states of the United States in recent decades, Ruhm (2000 and 2003) has provided strong statistical evidence that in the short run, excluding long-term trends, morbidity and mortality in the general population tend to fluctuate procyclically, increasing in expansions and decreasing in recessions. These results have been replicated with time series for the whole 20th century (Tapia Granados 2005a) and with panel data from the OECD countries (Gerdtham and Ruhm 2002), from Germany (Neumayer 2003), and from Spain (Tapia Granados 2005b). However, using individual data from recent decades in Sweden, Gerdtham and Johannesson (2005) found a countercyclical oscillation of mortality in males and no effect of economic fluctuations on female mortality.

Modern results showing a procyclical oscillation of mortality reproduce old findings, some of them almost a century old, in which an oscillation of mortality rising during economic upturns and falling during recessions was seen in the United States, the United Kingdom, and Canada (Ogburn and Thomas 1922; Thomas 1925; Eyer 1977a; Higgs 1979; Graham et al 1992, Adams, cited by Jin et al., 1995; LaPorte 2005). All authors who looked at the fluctuations of infectious disease mortality as related to the economic fluctuations in advanced countries (Eyer 1977a; Higgs 1979; Ruhm, 2000) found that infectious disease mortality waxes and wanes procyclically, in parallel to the expansions and recessions of the economy. Higgs (1979), who studied this phenomenon in thirty large U.S. cities during the late 19th century, attributed this increase of infectious disease mortality during economic upturns to the inflow of more virulent strains of bacteria and viruses brought by immigrants in periods of economic expansion, a point of view disputed by Chernomas (1984), who blamed the excess infectious mortality on overcrowding and overwork. In a recent discussion of the virological aspects of the 1918–1919 world flu pandemics, Kilbourne (2003) has mentioned the possibility that this pandemics were related to host and environmental factors, including a state of general susceptibility due to stress, crowding, and mixing of populations because of the World War. This has similarities with the point of view of Eyer, who attributed the increase in infectious disease during periods of rapid economic expansion to a decrease in the population's level of immunity due to overwork, overconsumption of toxic substances, and other stressful influences (Eyer 1977b, 1984; Eyer and Sterling 1977; Sterling and Eyer 1981). Since the concrete timing of deaths from chronic conditions such as CVD or cancer is subject to short-term influences (Anson and Anson 2000; Philips 1999), it may be determined by mild infections, and through this pathway short-term variations in the population level of immunity might have an impact on the fluctuations of general mortality, mostly due to chronic diseases. The possibility that long-term changes in the population's level of immunity can be related to the acceleration of the decline of mortality across all age strata of the U.S. population in the 1940s, and its deceleration and even reversal during the 1960s, is thus a hypothesis to be explored.

Leaving speculation and hypothesis aside, what is certain is that in the quartercentury following the end of World War II, real income consistently improved, unemployment rates were low, and income inequality was at steady or slightly declining levels (figure 5). In this period, frequently called the Golden Age of the American economy, the use of most antibiotics was generalized and many medical advances in diagnosis and treatment were implemented and generalized in clinical practice. Paradoxically, this period of unprecedented economic prosperity and medical progress coincided with shrinking rates of mortality decline for all demographic groups, culminating in a decade in which mortality was substantially increasing for most groups of working-age males, and even for some groups of working-age females. But data often contradict our expectations, and any improvement of our knowledge must begin with the facts.

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Figure 1. Crude mortality rate in the United States during the 20th century, deaths per 1000 population. The thin dotted line is a Hodrick-Prescott long-term trend line computed with a smoothing parameter equal to 100.





Figure 2. Age-specific mortality (natural log of deaths per 1,000) at different ages.



Figure 2 (cont.). Age-specific mortality (natural log of deaths per 1,000) at different ages.



Figure 3. Long-term rates of decline of crude mortality and age-adjusted mortality in the United States throughout the 20th century.



Figure 4. Long-term rates of decline of mortality for different age groups.



Figure 4 (cont.). Long-term rates of decline of mortality for different age groups.

Figure 5. Three changing aspects of U.S. society in the 20th century: unemployment, household income inequality, and use of motor vehicles



Figure 6. Annual mortality (deaths per 100,000) due to six causes of death. Note the different scales in the vertical axis. The first panel represents mortality due to CVD and renal disease before 1970 (thin line), and mortality due to major CVD after this year (thick line).

