

Losses of Expected Lifetime in the United States and Other Developed Countries: Methods and Empirical Analyses

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Abstract Patterns of diversity in age at death are examined using e^\dagger , a dispersion measure that equals the average expected lifetime lost at death. We apply two methods for decomposing differences in e^\dagger . The first method estimates the contributions of average levels of mortality and mortality age structures. The second (and newly developed) method returns components produced by differences between age- and cause-specific mortality rates. The United States is close to England and Wales in mean life expectancy but has higher life expectancy losses and lacks mortality compression. The difference is determined by mortality age structures, whereas the role of mortality levels is minor. This is related to excess mortality at ages under 65 from various causes in the United States. Regression on 17 country-series suggests that e^\dagger correlates with income inequality across countries but not across time. This result can be attributed to dissimilarity between the age- and cause-of-death structures of temporal mortality reduction and intercountry mortality variation. It also suggests that factors affecting overall mortality decrease differ from those responsible for excess lifetime losses in the United States compared with other countries. The latter can be related to weaknesses of health system and other factors resulting in premature death from heart diseases, amenable causes, accidents and violence.

Keywords Life expectancy · Premature death · Causes of death · Inequality

Introduction

There has been a long-standing tendency for mortality decreases to be steeper at younger than at older ages. This tendency, also called “rectangularization” of the

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survival curve (Wilmoth and Horiuchi 1999), facilitates an increase in the average length of life. It also leads to a strong negative correlation between life expectancy and the amount of diversity in the life-table ages at death both across time and across countries. In most cases, temporal increases in life expectancy correspond to decreases in this diversity; and life expectancy is, in most cases, higher in countries where the diversity is lower. Studies showed that since the 1970s, however, in some countries, one can observe increases in average life expectancy coinciding with stable or even increasing disparity in age at death (Shkolnikov et al. 2003; Wilmoth and Horiuchi 1999; Zhang and Vaupel 2008). These authors suggested that the new trend can be explained by an “expansion” of death to advanced ages and also to difficulties in further reduction of mortality at young and middle adult ages.

Shkolnikov et al. (2003) also pointed out considerable intercountry differences with respect to the relationship between life expectancy and the amount of diversity in age at death. In particular, the U.S. population is characterized by unexpectedly high diversity in age at death compared with the average life span because of relatively high proportions of deaths at ages that are much younger and much older than the average life span. It was also found that during a period of emergence of certain public health problems between the mid-1980s and the mid-1990s (Elo and Drevenstadt 2004; Kochanek et al. 1994), the relative interindividual difference in age at death for ages 15 and older increased (Shkolnikov et al. 2003).

Edwards and Tuljapurkar (2005) carried out an extensive study of potential reasons for temporal changes in the standard deviation of age at death for ages 10 and older (S_{10}), with special focus on its high value in the United States. After analyzing relations between S_{10} and external-cause mortality, race, and educational and income inequalities, the authors concluded that “... sources of differential background inequality in life spans between countries remain unclear and await further research” and that their measure of aggregate mortality inequality had not “simply followed trends in either educational or income inequality...” (Edwards and Tuljapurkar 2005:17, 23).

The present article extends prior work by contributing to methodology for the analysis of diversity in ages at death and also contributes substantive results to the discussion of reasons for slow progress in life expectancy in the United States that was initiated at the 2007 annual meeting of the Population Association of America (Blau et al. 2007).

Apart from S_{10} , interindividual diversity in age at death has been measured by the interquartile range (IQR; Wilmoth and Horiuchi 1999), the Gini coefficient and likely measures of relative inequality (Anand et al. 2001; Gakidou et al. 2003; Shkolnikov et al. 2003; Smits and Monden 2009), the Theil index of inequality (Smits and Monden 2009), and average interindividual difference and the related measures of absolute inequality (Moser et al. 2005; Shkolnikov et al. 2003). These measures differ from each other in some formal properties and also in the degree of their aversion to inequality (Anand 1983; Anand et al. 2001; Shkolnikov et al. 2003). In this article, we use e^\dagger , a measure highlighted by Vaupel and Canudas Romo (2003). Unlike S_{10} , it covers the entire range of ages and has an important public health interpretation. The value of e^\dagger quantifies the average life expectancy losses attributable to death. It generally follows Keyfitz’s idea that “everybody dies

prematurely” because every death “deprives the person involved of the reminder of his expectation of life” (Keyfitz 1977:61–68).

We will demonstrate that e^\dagger is also a measure of diversity in age at death equal to a weighted average of interindividual differences in age at death. We will introduce procedures for the decomposition of differences between two e^\dagger values according to direct and compositional components and according to age- and cause-specific components. The latter allows one to quantify the impact of mortality at different ages and from different causes upon life expectancy losses. Reduction of mortality at the ages, and from the causes, that produce greater impacts on life expectancy losses is the most direct way to accelerate the increase in a population’s longevity. Analysis of age- and cause-of-death components of the decrease in life expectancy losses in the United States and England and Wales and of the equivalent components of the difference in life expectancy losses between the two countries reveals a difference between the two structures. This meaningful difference helps to explain our finding that regression analysis of 17 country-series shows that life expectancy losses correlate with income inequality across countries but not across time. Decomposition analysis also provides information for a discussion of the reasons for particularly high lifetime losses in the United States.

Methods

e^\dagger as a Measure of Lifetime Losses and of Diversity in Age at Death

The quantity e^\dagger can be traced back to Mitra (1978). It was further developed by Vaupel (1986) and recently by Zhang and Vaupel (2008).

$$e_x^\dagger = \frac{1}{l_x} \int_x^\infty l(y)\mu(y)e(y)dy, \tag{1}$$

where $l(y)$, $\mu(y)$, and $e(y)$ are survivorship, the force of mortality, and life expectancy, respectively, expressed as functions of age. The definition makes it clear that e_x^\dagger is the average life expectancy losses caused by death at age $[x, x+1)$ and older ages. For empirical calculations, the following discrete formulae can be used:

$$e_x^\dagger = \frac{1}{l_x} \sum_{y=x}^{\omega-1} d_y \bar{e}_y = \frac{1}{2l_x} \sum_{y=x}^{\omega-1} d_y (e_y + e_{y+1}), \tag{2a}$$

$$e_x^\dagger = \frac{1}{l_x} \sum_{y=x}^{\omega-1} d_y \bar{e}_y = \frac{1}{l_x} \sum_{y=x}^{\omega-1} d_y [(1 - a_y) + e_{y+1}]. \tag{2b}$$

Equation 2b is slightly more precise because it includes $1-a_y$, the share of the

elementary age interval $[y, y+1)$ lost by those dying in this interval. If $x = 0$ and $l_0 = 1$, Eq. 2b yields

$$e^\dagger = e_0^\dagger = \sum_{y=0}^{\omega-1} d_y e_{y+1} + \left(1 - \sum_{y=x}^{\omega-1} d_y a_y \right), \tag{3}$$

where a_y is the share of the elementary age interval $[y, y+1)$ lived by those dying in this interval.

In the latter expression, the first term is the average amount of expected lifetime lost after ages y ($y = 0, 1, \dots, \omega-1$) attributable to deaths in elementary age intervals $[y, y+1)$; the second term is the average amount of lifetime lost within elementary age intervals $[y, y+1)$. The second term usually takes values close to 0.5 years.

Life expectancy at age $x+1$ can be also expressed as

$$e_{x+1} = \frac{1}{l_{x+1}} \sum_{y=x+1}^{\omega-1} d_y (\bar{y} - x - 1) = \frac{1}{l_{x+1}} \sum_{y=x+1}^{\omega-1} d_y (\bar{y} - \bar{x}) - (1 - a_x),$$

where \bar{y} and \bar{x} are mean ages at death within elementary intervals $[y, y+1)$ and $[x, x+1)$, respectively.¹ Substituting life expectancy by the latter expression in the first term of Eq. 3 yields

$$e^\dagger = \sum_{x=0}^{\omega-1} \frac{1}{l_{x+1}} \left[\sum_{y=x+1}^{\omega-1} d_x d_y (\bar{y} - \bar{x}) \right]. \tag{4}$$

Equation 4 suggests that the core part of e^\dagger is equal to a weighted interindividual difference in age at death. Thus, there is a clear similarity between e^\dagger and the numerator of the Gini coefficient, also called the average interindividual difference (AID) in length of life (Moser et al. 2005; Shkolnikov et al. 2003).

$$AID = \sum_{x=0}^{\omega-1} \left[\sum_{y=x+1}^{\omega-1} d_x d_y (\bar{y} - \bar{x}) \right]. \tag{5}$$

The presence of weights $(1 / l_{x+1})$ in Eq. 4 suggests that e^\dagger is somewhat more sensitive than AID to mortality at advanced ages.

The Pearson correlation coefficient between e^\dagger and AID across all country-year life tables of the Human Mortality Database ($n=1,972$) is close to .99 both for males and females. The corresponding correlation coefficients between e^\dagger and S_{10} are lower: .71 and .81 for males and females, respectively. Different inequality measures sometimes suggest different judgments about relative levels of inequality (Anand et al. 2001; Shkolnikov et al. 2003; van Raalte 2008). Comparisons within all possible pairs of countries of the Human Mortality Database for the last available year (666 comparisons) reveal about a 4% difference between e^\dagger -based and AID-based country rankings both for males and females. The corresponding percentages of disagreement between the e^\dagger -based and S_{10} -based rankings are 8% and 10% for males and females, respectively.

¹ $\bar{x} = x + a_x, \bar{y} = y + a_y.$

Direct and Compositional Components of Differences and Changes

Keyfitz (1977) and Vaupel (1986) performed the first analyses of the relationship between increases in life expectancy on one side and magnitudes of mortality reduction and shapes of mortality distributions by age on the other. Vaupel and Canudas Romo (2003) considered two factors driving longevity progress: the average rate of mortality reduction and the age pattern of this reduction. Longevity progress depends on each factor and on their interaction.

This idea can be applied to life expectancy losses as well. Any difference between two values of life expectancy losses can be presented as the result of a general mortality reduction undifferentiated by age and of a change in the age pattern of mortality. Consider a population with mortality determined by vectors of age-specific death rates equal to M_0 and M_1 . Then the total difference between the life expectancy losses $\Delta e_{tot}^\dagger = e^\dagger(M_1) - e^\dagger(M_0)$ is a sum of two components produced by the amount of mortality change (direct component) and the age structure of this change (compositional component):

$$\Delta e_{dir}^\dagger = e^\dagger(\lambda \cdot M_0) - e^\dagger(M_0), \tag{6a}$$

$$\Delta e_{cmp}^\dagger = e^\dagger(M_1) - e^\dagger(\lambda \cdot M_0), \tag{6b}$$

where the mean rate of mortality change is $\lambda = \frac{1}{\omega} \cdot \sum_x (m_{x,1}/m_{x,0})$ with $m_{x,1}$ and $m_{x,0}$ denoting elements of the vectors M_1 and M_2 , respectively, for age $[x, x+1)$. Equations 6a and 6b represent a simplified calculation procedure corresponding to that expressed in the continuous form for mean life expectancy by Vaupel and Canudas Romo (2003). The decomposition is based on calculation of the life expectancy losses resulting from application of the same rate of change to each of the initial age-specific death rates. Note that the second (compositional) component (6b) is a residual that combines a “pure” effect of the change in the age distribution of mortality with the effect of interaction between this “pure” effect and the change in the average level of mortality.

Age- and Cause-Specific Components of Differences and Changes

Each $d_x \cdot \bar{e}_x$ term in Eqs. 2a and 2b is a complicated quantity. Indeed, $d_x = l_x \cdot q_x$ and therefore depends on mortality at age $[x, x+1)$ and also ages younger than x . \bar{e}_x depends on mortality at age $[x, x+1)$ and older ages. The purpose of age decomposition is to estimate the *net* contribution of mortality change at a specific age to the total change or difference in an aggregate demographic measure (Andreev et al. 2002). The age-specific contribution must be free from side influences of other ages. Such decompositions of time changes or intercountry differences provide valuable information about the relative importance of mortality dynamics at different ages. Further decomposition by causes of death indicates the relative importance of various diseases and health conditions within the age groups.

In earlier work, we proposed a general algorithm for decomposition of differences between aggregate demographic measures (Andreev et al. 2002). If an aggregate

measure (say, life expectancy at birth) is calculated from a vector of age-specific death rates M , the age-specific component of the total difference between two values $e_0(M') - e_0(M)$ related to age $[x, x+1)$ is

$$\delta_x = e_0\left(M^{[x+1]}\right) - e_0\left(M^{[x]}\right). \tag{7a}$$

In this formula, $M^{[x]}$ stands for a vector of age-specific death rates containing elements m'_y at ages y from 0 to $[x, x+1)$ and elements m_y at ages y from $[x+1, x+2)$ to ω . This implies that Eq. 7a defines a decomposition method based on a stepwise replacement of the elements of the vector M by elements of the vector M' . Results of the procedure Eq. 7a are *not* exactly the same, depending on whether one replaces M by M' or, vice versa, M' by M . Hence, it is useful to calculate the second set of components by making the opposite-direction stepwise replacement of the elements m'_y by the elements m_y :

$$\delta'_x = e_0\left(M'^{[x+1]}\right) - e_0\left(M'^{[x]}\right). \tag{7b}$$

Equations 7a and 7b can be used directly for a numerical decomposition but can also be transformed into formulae for the components. It has been shown that these procedures result in well-known formulae for the decomposition of differences between two life expectancy values (Andreev 1982; Andreev et al. 2002; Arriaga 1984):

$$\delta_x = l'_x\left(e'_x - e_x\right) - l'_{x+1}\left(e'_{x+1} - e_{x+1}\right), \tag{8a}$$

$$\delta'_x = l_x\left(e_x - e'_x\right) - l_{x+1}\left(e_{x+1} - e'_{x+1}\right). \tag{8b}$$

The final age-specific components are calculated by averaging $\bar{\delta}_x = \frac{1}{2}\left(\delta_x - \delta'_x\right)$.

The same procedure can be applied to e^\dagger :

$$\eta_x = e^\dagger\left(M^{[x+1]}\right) - e^\dagger\left(M^{[x]}\right). \tag{9a}$$

As in the case of life expectancy, the replacement Eq. 9a can be used not only for a numerical decomposition but also for developing a formula for the age-specific component η_x (Appendix A):

$$\eta_x = \frac{\delta_x}{2} \cdot \sum_{y=0}^{x-1} \left[\frac{d'_y}{l'_y} + \frac{d_y}{l_{y+1}} \right] + \frac{d'_x}{2} \left(e_x + e_{x+1} + \frac{\delta_x}{l'_x} \right) - \frac{d_x \cdot l'_x}{2l_x} (e_x + e_{x+1}) + \left(\frac{l'_{x+1}}{l_{x+1}} - \frac{l'_x}{l_x} \right) \cdot l_{x+1} \cdot e^\dagger_{x+1}. \tag{10a}$$

The opposite-direction replacement corresponds to the components

$$\eta'_x = e^\dagger\left(M'^{[x+1]}\right) - e^\dagger\left(M'^{[x]}\right). \tag{9b}$$

Then the formula similar to Eq. 10a is

$$\eta'_x = \frac{\delta'_x}{2} \cdot \sum_{y=0}^{x-1} \left[\frac{d_y}{l_y} + \frac{d_y}{l_{y+1}} \right] + \frac{d_x}{2} \left(e'_x + e'_{x+1} + \frac{\delta'_x}{l_x} \right) - \frac{d'_x \cdot l_x}{2l'_x} \left(e'_x + e'_{x+1} \right) + \left(\frac{l_{x+1}}{l'_{x+1}} - \frac{l_x}{l'_x} \right) \cdot l'_{x+1} \cdot e'_{x+1}. \tag{10b}$$

The final components are: $\bar{\eta}_x = \frac{1}{2}(\eta_x - \eta'_x)$. As we know, the life expectancy components $\bar{\delta}_x$ can be further split by causes of death (Andreev 1982):

$$\bar{\delta}_{x,i} = \bar{\delta}_x \cdot \frac{m'_{x,i} - m_{x,i}}{m'_x - m_x}, \tag{11}$$

where $m_{x,i}$ denotes death rate at age $[x, x+1)$ from cause i .

Age- and cause-of-death components $\bar{\eta}_{x,i}$ can be calculated in the same manner (Appendix B):

$$\bar{\eta}_{x,i} = \bar{\eta}_x \cdot \frac{m'_{x,i} - m_{x,i}}{m'_x - m_x}. \tag{12}$$

Equations 11 and 12 suggest that for a given elementary age interval $[x, x+1)$ relative cause-specific shares of the corresponding age-specific component are the same for life expectancy losses and for life expectancy. This is certainly not the case for broader ranges of ages that include several elementary age intervals.

Regression of Life Expectancy Losses on Economic Inequality Across Countries and Time

To identify relationship between life expectancy losses and economic inequality, we use a matrix of observations of values of e^\dagger and the Gini index of income inequality by country and year. The cross-sectional time series regression is performed on a set of 17 countries for which it was possible to acquire consistent series of Gini indexes based on household incomes since 1975. These countries and regions are Australia, Austria, Belgium, Canada, Denmark, Finland, France, Italy, Japan, New Zealand, Norway, Spain, Sweden, Taiwan, the Netherlands, the United Kingdom, and the United States. Data for the calculations were extracted from the World Income Inequality Database and two additional sources (WIID2 2008; OECD 2008; the World Bank 2008). We did not include country series with numerous gaps or implausible ruptures nor the series for Eastern European countries. For the latter, income data are distorted by periods of political instability and also are likely to contain incomparable segments related to periods before and after the fall of communism. Before running the statistical analysis, we filled in the missing values of the Gini coefficient by interpolation and also by using some additional data sources wherever possible.

Exploratory analysis showed that time lags of 1, 2, and 3 years did not improve the results and that the logarithmic transformation did not change them. In the regression analysis, only quinquennial data were used, including years 1975, 1980, 1985, 1990, 1995, 2000, and 2004–2005. Such data are less likely to be serially correlated than the annual data.

Fixed- and random-effects regressions are performed with the help of the panel regression commands in Stata10 (Stata Corporation 2007). In the regressions, e^\dagger is

taken as the dependent variable and the Gini index of income inequality, and time dummy variables serve as independent variables.

Results

Trends in Life Expectancy and in Life Expectancy Losses

It is generally known that measures of diversity in age at death are strongly and negatively correlated with average length of life (Wilmoth and Horiuchi 1999), and e^\dagger can be expected to have the same property. In spite of this correlation, the balance between life expectancy and life expectancy losses can differ from one country to another. Figure 1 shows country trajectories for the United States, England and Wales, Japan, and Sweden in the e^\dagger vs. e space. The figure covers the years after World War II from the moment when life expectancy in each of these countries reached 60 years for males and 65 years for females. Accordingly, the Japanese series covers the period after 1951, while the other three country series start from 1946.

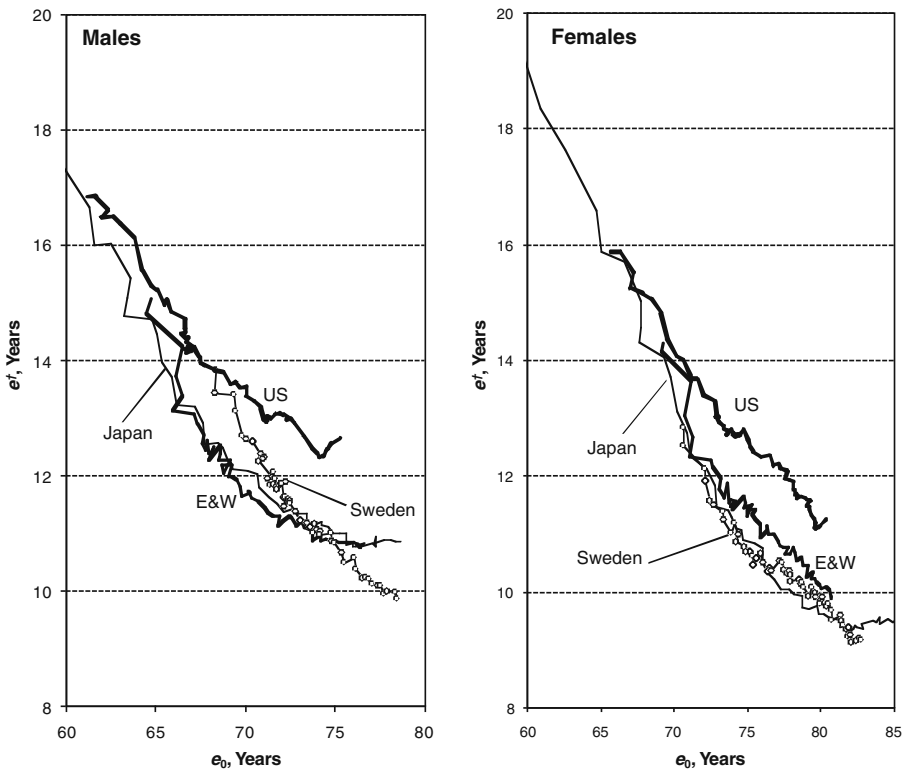


Fig. 1 The e^\dagger versus e_0 trajectories in England and Wales (E&W), Japan, Sweden, and the United States (US) after the year when life expectancies in these countries reached 60 years for males and 65 years for females. Time periods covered are 1946–2003 for England and Wales, 1951–2004 for Japan, 1946–2005 for Sweden, and 1946–2004 for the United States. Data are from author’s calculations on data from the Human Mortality Database (2007)

Figure 1 demonstrates a close negative correlation between life expectancy and life expectancy losses and also a relatively high level of life expectancy losses in the United States for most of the period. Although at certain moments in the past, the U.S. e^\dagger values had been quite close to e^\dagger values in Japan, England and Wales, and Sweden, the gap between the United States and other countries later widened because of a flatter e^\dagger trajectory in the United States compared with the other countries. The trajectories for Japan, England and Wales, and Sweden converge remarkably (especially for females) and constitute a clear difference from the United States. Over a long period of time, the U.S. and the English life expectancies have been close to each other. Since the mid-1990s, life expectancy values in England and Wales were higher than those in the United States by about one year for males and by about one-half year for females. In spite of the closeness in average longevity, life expectancy losses are substantially greater in the United States than in England and Wales.

Figure 2 reflects a cross-sectional correspondence in 2002 between life expectancy losses and life expectancy for the 29 developed countries represented in the Human Mortality Database. Once again, one can see a tight negative association between e_0 and e^\dagger ($r = -.95$ and $r = -.82$ for males and females, respectively). There are also some differences in e^\dagger between countries with the same level of life expectancy. The U.S. e^\dagger values lie considerably higher than the expected values on the trend line. For example, in 2002, an average male death in the United States caused a loss of 12.5 years of lifetime, whereas the corresponding expected value on the trend line is 11.4 years (left panel of Fig. 2). This means that in 2002, an average male death in the United States caused an excess loss of 1.1 years of lifetime compared with what can be expected from experience of other countries. For U.S. females, the observed and the expected values of e^\dagger in 2002 were 11.1 and 10.0 years, respectively, suggesting the same excess loss of 1.1 years of lifetime.

According to Eq. 2a, the high life expectancy losses in the United States must be produced by peculiar shapes of the $d_x \cdot \bar{e}_x$ distributions. Figure 3 provides a comparison of these distributions between the United States and England and Wales in 1950 and 2002. During this period, in both countries, a gross shift of the whole

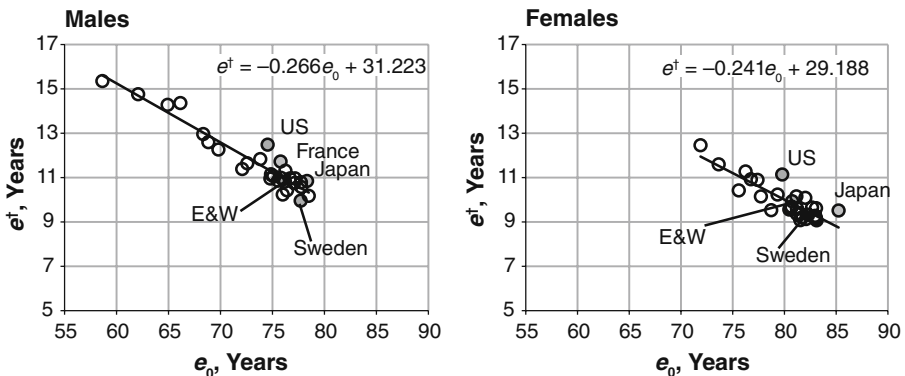


Fig. 2 The e^\dagger versus e_0 correspondence for 29 industrialized Human Mortality Database countries in 2002. Data are from authors' calculations on data from the Human Mortality Database (2007)

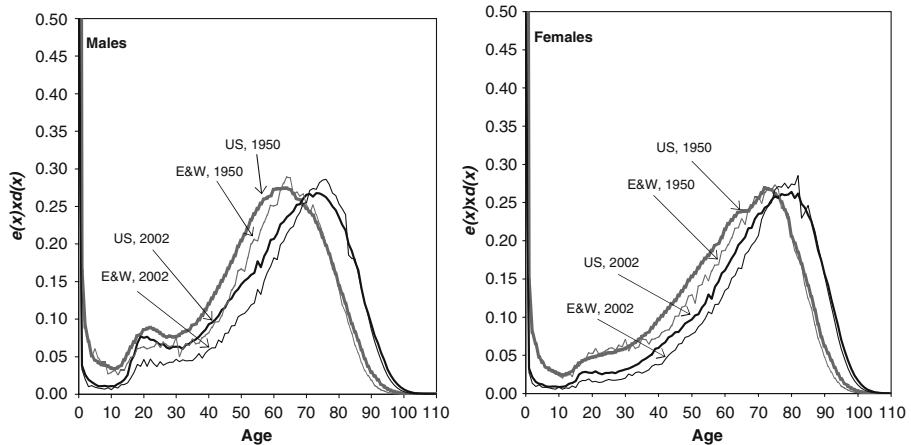


Fig. 3 Comparison of the $d_x \cdot \bar{e}_x$ curves between the United States (US) and England and Wales (E&W) in 1950 and 2002. Data are from authors' calculations on data from the Human Mortality Database (2007)

death distribution toward older ages occurred. At both time points, there is almost no difference between the modes of the two distributions; however, the U.S. distributions in both 1950 and 2002 are more dispersed than those of England and Wales. The U.S. distributions have heavier left tails corresponding to young-middle ages. For males, the intercountry difference reaches maxima at ages around 20 and at ages between 40 and 60. For females, the maximum differences are observed at ages between 40 and 65.

It is remarkable that for males at ages between 25 and 45, the $d_x \cdot \bar{e}_x$ values in the United States in 2002 are almost the same as the ones observed in England and Wales in 1950.

In 1950, age-specific death rates in the United States were, on average, higher than those in England and Wales by about 7% for males and 9% for females. By 2002, these differences increased to 29% and 25%, respectively. Table 1 shows the results of decomposition of differences in life expectancy losses between England and Wales and the United States for the two years. The e^\dagger differences in favor of England and Wales increased from 1.6 to 1.7 years for males and from 1.0 to 1.2 years for females. Calculations according to Eqs. 6a and 6b make it clear that both in 1950 and 2002, more than 90% of the intercountry difference in e^\dagger was produced by the compositional component determined by differences between the mortality age structures. The part of the direct component determined by differences between average levels of mortality is minor.

Oeppen (2008) introduced the concept of efficiency of the age pattern of mortality change, which is based on an intuitively clear relationship between progress in average longevity and the amount of life expectancy losses. An “efficient” age pattern of mortality reduction (in terms of the overall longevity gain) is the one that produces greater mortality reductions at ages where the $d_x \cdot \bar{e}_x$ fractions are greater. In this regard, age patterns of mortality change in the United States are far from being optimal because the excess in lifetime losses in the United States relative to other countries has not been decreasing with time.

Table 1 Direct and compositional components of differences between life expectancy losses in England and Wales (E&W) and in the United States (US) in 1950 and 2002 (in years)

	Values			Components of the Differences		
	EW (a)	US (b)	After Even Reduction (c)	Total Difference (a)–(b)	Direct (a)–(c)	Compositional (c)–(b)
Males						
1950	13.68	15.24	13.81	–1.56	–0.12	–1.44
2002	11.09	12.76	11.23	–1.66	–0.14	–1.52
Females						
1950	12.94	13.98	12.93	–1.04	–0.01	–1.05
2002	10.19	11.41	10.32	–1.22	–0.13	–1.09

Data are from authors' calculations on data from the Human Mortality Database (2007)

Age- and Cause-of-Death Components of Change in Life Expectancy Losses Between 1980 and 2002

Equations 7a, 7b, 9a, and 9b allow one to decompose by age the increase in e_0 and decrease in e^\dagger , respectively. Figure 4, which presents age-specific components of changes between 1980 and 2002² in the United States and England and Wales, shows a difference between reactions of the two measures to the same change in age-specific mortality. The life expectancy increases in both countries are largely determined by decreases in death rates at ages from 50–85 (upper panels of Fig. 4). Smaller contributions are produced by reductions of infant deaths and of younger adult mortality at ages 15–50. The younger adult mortality contribution is more important for males than females. In England and Wales, components produced by infant and older adult ages are greater than the equivalent components in the United States. At the same time, the younger adult-age components are greater in the United States than those in England and Wales.

For the life expectancy losses (lower panels of Fig. 4), age patterns of change are quite different. Depending on age, the mortality reduction produces negative or positive contributions to the total e^\dagger change. At ages younger than a threshold age (denoted by a^\dagger by Zhang and Vaupel [2008]), mortality reduction contributes to e^\dagger negatively, whereas mortality reduction at ages older than a^\dagger contribute to e^\dagger positively. These two balancing forces producing negative and positive effects on life expectancy losses were defined by Zhang and Vaupel (2008) as mortality *compression* and mortality *expansion*, respectively. Lack of mortality compression (e.g., elevated sum of components $d_x \cdot \bar{e}_x$ at ages under age a^\dagger) is seen as unfavorable from the public health point of view. For the mortality change between 1980 and 2002 in both countries, sums of the negative components of change (mortality compression) were two to three times greater than sums of the positive components

² The last year for which the cause-of-death data are currently available for the two countries in the WHO Mortality Database.

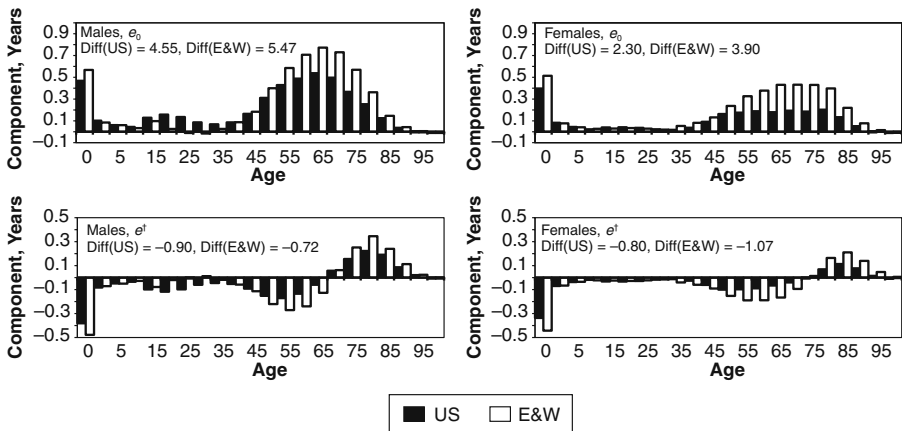


Fig. 4 Age-specific components of increases in life expectancy and decreases in life expectancy losses between 1980 and 2002 in the United States (US) and England and Wales (E&W) Data are from authors' calculations on data from the Human Mortality Database (2007)

(mortality expansion). Finally, the lower panels of Fig. 4 demonstrate that England and Wales experienced greater compression and expansion components of the total e^{\dagger} change than the United States.

The threshold age has tended to increase over the last decades, always being somewhat lower than the average life expectancy (Zhang and Vaupel 2008). Between 1980 and 2002, the male a^{\dagger} increased from 67.5 to 72.5 years in the United States and from 68.5 years to 74.5 years in England and Wales. During the same period, the female a^{\dagger} increased from 76.5 to 78.5 years in the United States and from 75.5 to 79.5 years in England and Wales.

From a public health perspective, the degree of mortality compression is especially important. It shows the extent to which a society is able to protect people from premature death. The fact that the threshold age increases with time means that the ages at death that are considered to be premature are rising. Using cause of death data to examine mortality expansion is more problematic because of the difficulties of determining a single cause of death for the very elderly and because of the use of open age intervals.

Figure 5 shows cause-of-death components of mortality compression in the two countries. As the threshold ages are located within age groups 65–75 and 75–80 for males and females, respectively, the male and female decompositions are being made for ages under 70 and ages under 75, respectively. The cause-specific components of the e^{\dagger} decrease between 1980 and 2002 are computed from Eqs. 11 and 12. Both in the United States and in England and Wales, the greatest contributions are produced by coronary and other circulatory diseases. In both countries, major causes of infant death, such as perinatal conditions and congenital abnormalities, are the second greatest contributor to decreasing life expectancy losses under the threshold age. In both countries, considerable contributions are also produced by lowering mortality from lung cancer (males), breast and other cancers (females), traffic accidents (males), and violent causes of death (only for males in the United States).

Comparison between the two countries shows that during 1980–2002, England and Wales experienced greater reduction in e^{\dagger} because of chronic conditions, such as circulatory diseases, male lung cancer, and female breast cancer. The United States

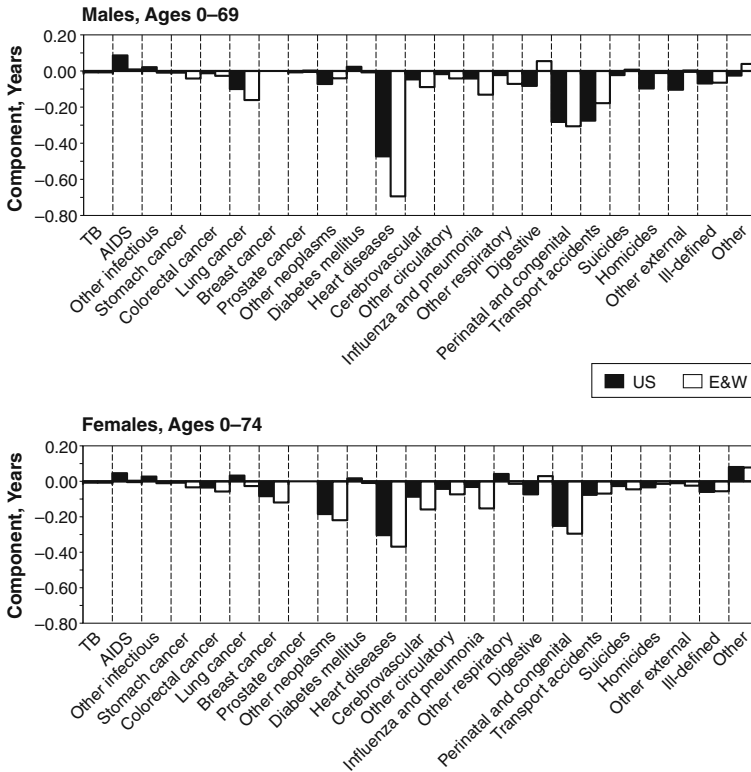


Fig. 5 Cause-specific components of decreases in life expectancy losses between 1980 and 2002 in the United States (US) and England and Wales (E&W) for the range of ages under the threshold. Data are from the authors' calculation on data from the Human Mortality Database (2007) and the WHO Mortality Database (2007)

experienced greater effects related to the reduction of male mortality from accidents and violence.

The Intercountry Differences in Life Expectancy Losses in 2002

Figure 6 exhibits age-specific components of differences between England and Wales and the United States in life expectancy and in life expectancy losses in the year 2002 and reveals marked differences from the age patterns of temporal change in Fig. 4. First, the lower U.S. death rates at ages over 75 produce negative contributions to the e_0 difference between the two countries and partly counter-balance positive components produced by the higher U.S. death rates at ages under 75. At the same time, the components of intercountry difference in e^\dagger are now negative at all ages. Second, the role of older adult age and infant age components is relatively less important in the intercountry differences than the role of the equivalent components in the temporal change. At the same time, the contributions of younger adult ages in the intercountry differences are more important than the equivalent contributions to the temporal change.

The cause-of-death pattern of the intercountry difference in mortality compression (Fig. 7) also differs substantially from the equivalent pattern of temporal change

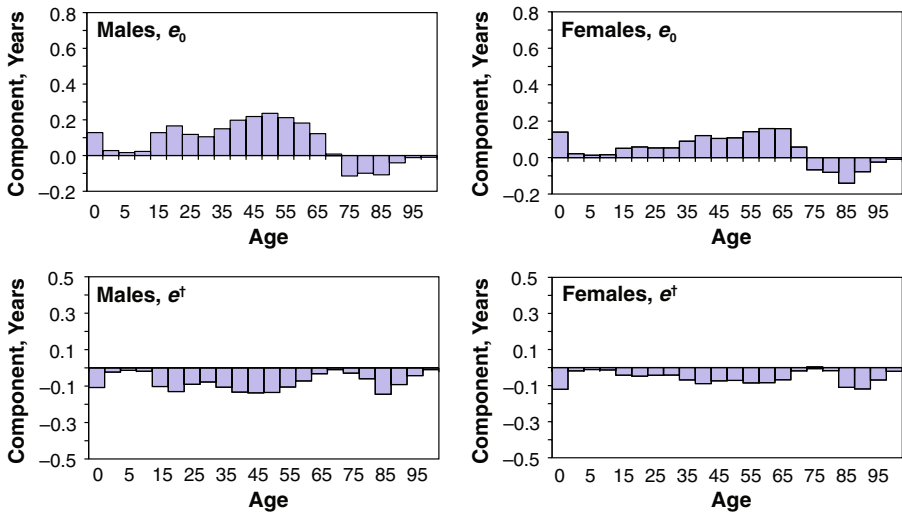


Fig. 6 Age-specific components of differences between England and Wales and the United States in life expectancy and in life expectancy losses in 2002. Data are from the authors' calculations on data from the Human Mortality Database (2007)

(Fig. 5). Indeed, the role of heart diseases is relatively less prominent in the intercountry difference than in the temporal change, and causes of death characteristic of younger adult ages (such as transport and other accidents, violence, and HIV/AIDS) are more important in the intercountry difference than in the temporal change.

Table 2 shows that if the mortality difference between the United States and England and Wales at ages under the threshold age was instantly eliminated, U.S. average longevity would exceed the values for England and Wales. U.S. life expectancy losses would become much lower, but they would still exceed corresponding values in England and Wales.

Associations Between Life Expectancy Losses and Economic Inequality

Is it only a coincidence that the United States is characterized by one of the developed world's highest levels of disparity in ages at death and by one of the developed world's highest levels of economic inequality? To answer this question, we examine whether variation in economic inequality across countries and time is statistically associated with life expectancy losses. Edwards and Tuljapurkar (2005) evaluated the link between time changes in the standard deviation of ages at death above age 10 (S_{10}) and the Gini index of income inequality by visual inspection of the trajectories of the United States and four other countries in the S_{10} -Gini space. We approach it somewhat differently by means of regression analysis of cross-sectional time series connecting life expectancy losses with the Gini index for a greater number of countries and years.

As a preliminary step, the Pearson correlation coefficients are computed across the whole set of country-year points. Correlations between life expectancy and the Gini index appear to be negative (as expected) but small and statistically insignificant: -0.20 and -0.25 for males and females, respectively. Coefficients of

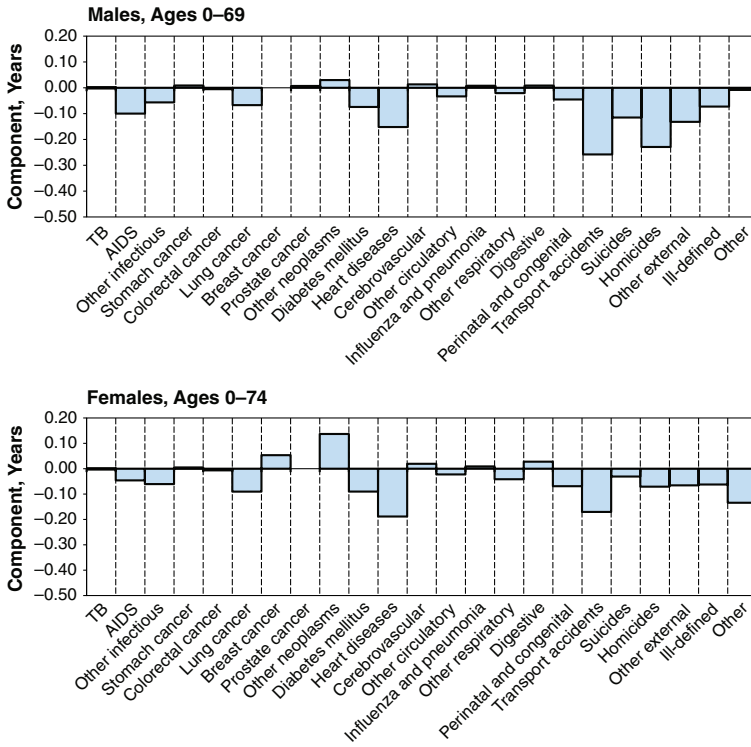


Fig. 7 Cause- and age-specific components of differences between England and Wales and the United States in life expectancy losses in 2002 for the range of ages under the threshold. Data are from authors’ calculations on data from the WHO Mortality Database (2007)

correlation between e^\dagger and the Gini index are much higher and statistically significant ($p < .05$): .70 for males and .73 for females.

Table 3 shows the outcomes of three regression models. The first “between” regression is based on the cross-sectional setup. It connects the time-averaged values of e^\dagger to the corresponding values of the Gini index. For both males and females, e^\dagger is positively and statistically significantly associated with the Gini index. The second “within” model is a pure longitudinal model with fixed country-effects. The model examines whether, on average, the e^\dagger trends are associated with the Gini index

Table 2 Life expectancy and life expectancy losses in 2002: Effects of elimination of the excess mortality in the United States (US) compared with England and Wales (E&W) at ages under 70 (males) and ages under 75 (females)

	e_0			e^\dagger		
	US	E&W	US After Elimination	US	E&W	US After Elimination
Males	74.54	76.21	76.57	12.51	10.84	11.22
Females	79.79	80.74	81.14	11.16	9.93	10.27

Data are from authors’ calculations on data from the Human Mortality Database (2007)

Table 3 Relationship between e^{\dagger} and Gini index of income inequality across countries and time

	Pooled LS ^a With Fixed Country Effects		Generalized LS ^a With Random Country Effects		Pooled LS ^a With Fixed Country Effects		Generalized LS ^a With Random Country Effects	
	Between	Effects	Between	Effects	Between	Effects	Between	Effects
	Males				Females			
Regression Coefficients								
Gini	0.072 (0.012)	0.013 (0.090)	0.019 (0.014)	0.081 (0.003)	0.008 (0.174)	0.011 (0.044)		
Intercept	9.514 (0.000)	12.123 (0.000)	11.927 (0.000)	7.828 (0.000)	10.828 (0.000)	10.706 (0.000)		
Fixed time effects	No	Yes	Yes	No	Yes	Yes		Yes
Statistical Tests								
Chow. H_0 : Absence of fixed effects	–	36.78 (0.000)	–	–	55.33 (0.000)	–	–	–
Hausman. H_0 : No systematic difference between random and fixed effects	–	–	17.67 (0.014)	–	–	104.78 (0.000)	–	–
Breusch-Pagan. H_0 : Absence of random effects	–	–	–	–	–	–	–	178.71 (0.000)

Data are for Australia, Austria, Belgium, Canada, Denmark, Finland, France, Italy, Japan, New Zealand, Norway, Spain, Sweden, Taiwan, the Netherlands, United States, United Kingdom. Quinquennial data of the years 1975, 1980, 1985, 1990, 1995, 2000, 2004–2005 are used. Data are from authors' calculations of e^{\dagger} on data from the Human Mortality Database (2007); OECD Statistics (2008), World Bank (2008), and WIID (2008) for the Gini index

p values are given in parentheses

^a LS refers to least squares

trends. The Chow test suggests that significant fixed country-effects do exist. There is only a very weak positive association for males ($p < .10$) and no significant association for females. Even if the association between e^\dagger and the Gini index exists for males, the regression coefficient of 0.013 indicates that the Gini index would have to be increased by a factor of 10 to produce a moderate increase in e^\dagger equal to 0.13 year. Although the random effects model (that combines the cross-sectional and the longitudinal approaches) indicates significant and positive relationships ($p < .05$), the Hausman test suggests priority for the fixed-effects model.

All in all, there is a cross-sectional association between life expectancy losses and income inequality, but temporal changes in life expectancy losses are independent or almost independent of changes in income inequality.

To understand whether a high diversity in age at death in the United States can be attributed to socioeconomic inequalities in health, Edwards and Tuljapurkar (2005) compared distributions of ages at death between broad educational groups and between broad income groups on the basis of data from the National Longitudinal Mortality Survey (NLMS 2007; Rogot et al. 1992). They found that groups of higher income and higher education experienced lower values of the standard deviation of ages at death for ages 10 and older, S_{10} ; but even in these groups, S_{10} values were high when compared with the international standard. Using the same NLMS data, we calculated life expectancy at age 30 and life expectancy losses at ages 30 and older for a larger number of more finely defined educational, income, and racial groups, and also for their two-dimensional combinations (Table 4). During 1979–1985, the differences in life expectancy losses between the most advantaged and the most disadvantaged groups were nearly 5 years. Values of e^\dagger are about 15 years for African American males and females in the lowest income group compared with e^\dagger values of 10.5–11 years for white males and females in the highest income group. However, our conclusion remains the same as the one by Edwards and Tuljapurkar (2005): even the most advantaged groups experienced values of e^\dagger that were still slightly higher than the contemporary values for the entire population of England and Wales, which were below 10 years during 1979–1985.

Summary of Results

This study further develops a toolkit for the analysis of interindividual inequality in the face of death. We focus on e^\dagger , a quantity measuring diversity in ages at death that is also equal to the amount of expected lifetime lost. We introduce procedures for calculation of this measure from empirical data and two ways to decompose a difference between two e^\dagger values. The first type of decomposition reflects two fundamental aspects of the mortality pattern and permits estimation of a component produced by the difference between average levels of mortality and a component produced by differences between mortality age structures. The second (and more traditional) type of decomposition is public health-oriented. It allows one to compute components produced by differences between age- and cause-specific mortality rates. Its usage allows one to evaluate the relative importance of contributions of different ages and causes of death to the overall difference between life expectancy losses.

Table 4 Values of e_{30} and e^{\dagger}_{30} in 1979–1985 in the national populations of the United States and England and Wales, and in racial, educational, income, and combined groups in the National Longitudinal Mortality Survey

			Males			Females		
			Person-Years (thousands)	e_{30}	e^{\dagger}_{30}	Person-Years (thousands)	e_{30}	e^{\dagger}_{30}
National populations (from HMD)								
US, HMD population			630,240	43.34	11.24	720,001	49.70	10.52
UK			147,790	43.38	9.97	147,790	48.72	9.76
Groups (from the NLMS)								
Race	Education	Income (\$)						
All	All	All	1,217	44.77	11.67	1,438	51.44	11.61
White	All	All	1,027	45.03	11.41	1,106	51.70	11.30
Black	All	All	90	40.64	13.60	127	48.09	14.01
All	Elementary	All	211	42.23	12.49	238	49.52	12.27
All	High school	All	528	44.24	11.66	754	51.48	11.92
All	College	All	410	47.30	11.04	360	53.05	10.88
All	All	<9,999	227	39.43	13.21	406	48.90	12.91
All	All	10,000–24,999	536	45.25	11.83	577	51.96	11.33
All	All	25,000+	386	47.83	10.62	370	52.94	10.75
White	All	<9,999	186	40.17	12.81	330	49.75	12.30
White	All	10,000–24,999	481	45.47	11.68	516	52.11	11.10
White	All	25,000+	360	47.80	10.55	344	52.92	10.75
Black	All	<9,999	34	36.05	14.77	66	45.97	15.03
Black	All	10,000–24,999	41	42.77	12.83	46	49.79	13.42
Black	All	25,000+	15	46.75	11.30	145	50.38	11.03
White	Elementary	All	187	42.91	12.13	208	50.24	11.75
White	High school	All	502	44.64	11.46	714	51.91	11.63
White	College	All	401	47.40	10.91	346	53.22	10.84
Black	Elementary	All	30	38.15	14.48	36	46.22	14.95
Black	High school	All	45	41.07	13.99	71	47.80	13.79
Black	College	All	19	44.58	12.05	25	54.78	15.71
All	Elementary	<9,999	101	39.46	13.09	147	48.31	13.09
All	Elementary	10,000–24,999	88	44.05	12.30	74	50.27	11.32
All	Elementary	25,000+	21	45.00	11.64	18	51.65	10.64
All	High school	<9,999	95	38.82	13.37	209	49.22	12.97
All	High school	10,000–24,999	285	45.15	11.52	355	52.35	12.00
All	High school	25,000+	147	46.84	10.74	190	52.61	11.24
All	College	<9,999	30	41.97	12.46	49	49.69	12.21
All	College	10,000–24,999	162	46.22	11.73	149	53.52	11.38
All	College	25,000+	217	49.17	10.55	162	54.66	11.38

Data are from authors' calculations based on the data from the National Longitudinal Mortality Study (NLMS 2007). The value of family income in unadjusted dollars is inflated (deflated) to 1980 dollars, and then each member of the family is assigned the appropriate category for the variable as indicated in the table

Greater focus on the public health aspect of age-at-death disparity constitutes the main difference between this study and the prior work by Edwards and Tuljapurkar (2005). We use an alternative measure of disparity in age at death that covers the entire range of ages and is more public health-oriented than S_{10} . In addition to temporal change, we pay serious attention to cross-sectional differences among countries that (unlike temporal change) appear to be associated with economic inequality. Using decomposition, we show that excess life expectancy losses in the United States are attributable to certain public health problems related to particular age groups and death causes.

Two empirical analyses of life expectancy losses are completed. First, we consider trends and made intercountry comparisons. The analysis reveals persistently high values of e^\dagger in the United States that are caused by relatively low mortality compression and relatively high mortality expansion in this country. In 2002, lifetime losses among U.S. males and females were greater by 1.1 year than the values expected on the basis of the experience of other developed countries. Compared with England and Wales, the United States had slightly lower average longevity and much greater life expectancy losses. The first type of decomposition demonstrates that both in the past and now, the e^\dagger gap between the two countries is mostly determined by the difference between the mortality age structures.

Second, we apply the decomposition by ages and causes of death to decreases in life expectancy losses between 1980 and 2002 in the two countries and to the differences in life expectancy losses between the two countries in 2002. We find the following:

- Falling infant mortality is a considerable component of the decrease in life expectancy losses in both countries and is much less important as a component of the difference in life expectancy losses between the two countries.
- Decreasing mortality from cardiovascular diseases and some other chronic conditions (male lung cancer and female breast cancer) at middle and older adult ages constitute a major component of the decrease in life expectancy losses. From 1980 to 2002, this component of mortality compression was greater in England and Wales than in the United States.
- Decreasing mortality at younger adult ages 15–50 from accidents (especially traffic accidents), violence, heart attacks, HIV/AIDS, and diabetes is less important than the decreasing older-age mortality from major chronic diseases as a component of the decrease in life expectancy losses, but it is more important as a component of the gap in life expectancy losses between the United States and England and Wales.

Seeking interpretations of the observed patterns of life expectancy losses, we carry out additional analyses. We perform regressions connecting e^\dagger with the Gini index of income inequality on time series for 17 industrialized countries since 1975. We find statistically significant associations across countries but not across time.

Using the NLMS data, we calculate e^\dagger values for categories of education, income, and race and all their pairwise combinations for the United States. Although life expectancy losses vary significantly across groups, even the lowest e^\dagger values in the most advantaged groups are still slightly higher than values observed in the total population of England and Wales. In this regard, our substantive conclusion confirms that of Edwards and Tuljapurkar (2005) even though we use much more detailed socioeconomic groupings.

Discussion

The substantive results of this study allow us to discuss two issues. First, they provide insights into determinants of variation in life expectancy losses across time and countries. Second, they prompt more specific explanations for the particularly high level of life expectancy losses in the United States.

When thinking about reasons for variation in life expectancy losses across time and countries, it is useful to combine the regression results with results of decompositions by age and cause for the United States and England and Wales. In this way, one can see that the complete or nearly complete absence of the longitudinal association between life expectancy losses and income inequality corresponds to the important role of decreasing circulatory disease and major cancers at old ages in the temporal decline in life expectancy losses. At the same time, the significance of the cross-sectional association between life expectancy losses and income inequality corresponds to a greater role of mortality at younger adult ages and to more acute and avoidable causes of death in cross-sectional differences in life expectancy losses.

The dissimilarity between the longitudinal and cross-sectional health-income inequality associations is consistent with the conclusions of John Lynch and colleagues (2004), whose extensive review of 98 epidemiological studies pointed out the specificity of the strength of association between income inequality and health in regard to type of health outcome. Empirical studies (especially those involving longitudinal evidence and control for compositional effects) provide little evidence for a general relationship between income inequality and total mortality (Deaton and Lubotsky 2003; Lynch et al. 2001; Lynch et al. 2004b; Mellor and Milyo 2001; Osler et al. 2002; Shibuya et al. 2002). In particular, there is little research support for the relationship between income inequality and mortality or morbidity from major cardiovascular and other chronic diseases of old age (Lynch et al. 2004a:74–76, 81–82). At the same time, certain health outcomes are significantly associated with income inequality. Such associations were detected for the mortality of children and adults of working age and for mortality from certain causes such as homicide, stroke and heart attacks (Daly et al. 2001; Franzini and Spears 2003; Kennedy et al. 1998; Kennedy et al. 1996; Lobmayer and Wilkinson 2000; Lynch et al. 2004a; Osler et al. 2003; Shi et al. 2003; Sohler et al. 2003; Szwarcwald et al. 1999; Wilkinson et al. 1998). The work by Backlund et al. (2007) is especially instructive. The authors applied advanced multilevel techniques to NLMS data and showed that state-level income inequality in 1990 in the United States was associated with differences in state level mortality at ages 25–64 after controlling for the compositional effects of individual characteristics, such as income, education, unemployment, and race. The association was much stronger for men than for women. No such relationship was found for mortality at ages above 65.

This result makes it clear why income inequality can not be a major determinant of general mortality decrease. Indeed, in the United States and other advanced countries, general mortality decrease is determined primarily by cardiovascular and other chronic diseases at older ages that are unrelated to income inequality (Salomon and Murray 2002; Vallin and Meslé 2004).

Thus, our results based on age-cause decompositions and regression analysis of 17 country series agree with the detailed epidemiological evidence. Both suggest that

factors for a temporal decrease in life expectancy losses differ from factors for intercountry differences. The decrease in these losses is mostly driven by reduction of major chronic diseases at old ages that can be related to advancement in medical technologies for treatment and diagnostics as well as favorable behavioral changes, such as reduction in smoking (Pampel 2003). The intercountry differences are, to a greater extent, related to health and mortality at younger adult ages that are associated with socioeconomic inequality and *relative* deprivation, leading in turn to elevation of psychosocial stress (Marmot and Wilkinson 2001; Siegrist 2000; Wilkinson et al. 1998).

In rare cases, large changes in working-age mortality cause substantial changes in total mortality, mean length of life, and life expectancy losses. Such changes were observed in Russia and other ex-USSR countries in the 1990s. Both some of the historical health crises, such as the one in nineteenth century Sweden (Sundin and Willner 2004; Willner 2001), and the recent crisis in the former USSR were largely based on excess mortality of men from causes associated with alcohol and were attributed to psychosocial stress (Marmot and Bobak 2000; Leon and Shkolnikov 1998; Shapiro 1995).

In the modern developed world, such outbreaks of working-age mortality are exceptions from mainstream health progress. In the United States (as in other advanced countries), changes in life expectancy losses are mainly driven by decreasing chronic disease among older people and are unrelated to income inequality. However, the excess in life expectancy losses in the United States *relative* to other countries is determined by the higher U.S. mortality at younger ages and from causes of death that can be linked to income inequality.

A high level of premature death is a long-standing health problem in the United States. Progress in this area is not rapid enough, and separation between the United States and other countries tends to be sustained. High life expectancy losses in the United States can be seen as a result of persistent adverse conditions, such as cigarette smoking among some groups, and also weaknesses of a health system that is unable to assure accelerated reduction of premature death. Our regression analysis signals that it is likely that at least part of these conditions and weaknesses is related to high income inequality in the United States. It is noteworthy that the United States is the country where income inequality is most consistently linked to population health by research evidence, which is not the case in most other developed countries (Lynch et al. 2004a).

Socioeconomic disparities in health between population groups comprise a part of the total amount of interindividual disparity in respect to age at death (and in life expectancy losses). In the second half of the twentieth century, mortality reversals have been observed twice in the African American population (Elo and Drevenstadt 2004; Geronimus et al. 2001; Kochanek et al. 1994; Preston and Elo 1995). The last episode, lasting from 1984 to 1991, coincided with an increase in age at death disparity among U.S. males aged 15 and older (Shkolnikov et al. 2003). Our analysis of NLMS data demonstrated large differences between higher losses in disadvantaged groups and lower losses in advantaged groups. It is possible that some other important types of inequalities play a role. In the United States, huge geographical differences in mortality are related not only to the variable socioeconomic status of individuals in

various places but also to highly variable regional contexts (Ezzati et al. 2008; Murray et al. 2006).

However, the socioeconomic health contrasts are unlikely to be responsible for the total amount of excess life expectancy losses in the United States. Indeed, on the basis of NLMS data, we find that even the lowest losses in advantaged groups are still slightly higher than losses in the total population of England and Wales. Therefore, one can guess that the whole range of variation of losses within the United States is shifted toward higher values compared with the equivalent range in England and Wales. This suggests that there are some adverse factors that affect all or many of the U.S. population groups.

The American health system is one of the candidate factors. An important disadvantage of the United States is the incomplete population coverage and variable performance characteristic of the health system. A detailed investigation by Schoen and How (2006) revealed a range of concrete problems in medical care that are especially significant for working people paying their own medical bills. It was reported that about one-third of Americans at ages under 65 do not have any health insurance, and the same share of people have difficulties paying their medical bills. For people of working age, availability and quality of medical care not only depends on their wealth but also significantly varies across health care plans, states, and hospitals. Only one-half of adults receive the recommended preventive health care, including screening for cancer. Health insurance premiums rose far faster than wages, rising as a share of median incomes. Readmissions to hospitals within 30 days remained high and were variable across the country.

It is likely that the disadvantage of the U.S. health system relative to other advanced countries is tending to increase. Two studies by Nolte and McKee (2003, 2008) estimated mortality from medically amenable causes such as bacterial infections, treatable cancers, cerebrovascular disease, part of ischemic heart disease, and complications of common surgical procedures at ages under 75 in 19 OECD countries. During 1997–1998, the United States occupied the 15th place, with amenable mortality exceeding that in 14 other OECD countries. By 2002–2003, the decline in amenable mortality comprised 17% for all OECD countries and only 4% for the United States. As a result, the United States fell to 19th place.

As we demonstrated, excess mortality from lung cancer and heart diseases at ages under 70 for males and under 75 for females contributes to the lack of mortality compression in the United States. This can be largely related to smoking. In the mid-1960s, the United States was among the nations of the world with the highest rates of smoking, with a smoking prevalence of about 50% and 30% for males and females aged 18 and older, respectively (Garfinkel and Silverberg 1991). By 2005, smoking was dramatically reduced to 26% among males and 22% among females. The steeper decrease in smoking of men from the mid-1960s to 2005 is considered to be the central reason for the recent narrowing of the female-male longevity gap (Pampel 2002; Preston and Wang 2006). Smoking-related mortality in the United States has been estimated directly from survey data (Rogers et al. 2005) and also by indirect methods (Peto et al. 2006; Preston et al. 2010). All these estimates are consistent with each other. According to Peto et al. (2006), 29% of male deaths and 27% of female deaths at ages 35–69 in the United States in 2000 were

attributable to smoking. Preston et al. (2010) reported similar figures and demonstrated that among 20 developed countries in 2003, the share of smoking-attributable death in the United States was the highest for females and the sixth highest for males.

Death rates among young and middle-aged adults, especially men, are also related to risks caused by alcohol and substance use. France is a classic example of high alcohol-related mortality that contributes to the high level of disparity in age at death in this country (Edwards and Tuljapurkar 2005; Nizard and Muños-Perez 1993). The United States is also not free of such problems. Between 1992 and 2002, U.S. mortality from unintentional injuries increased by 11% (Paulozzi et al. 2006). In the age group 40–64 years, death rates increased for falls, poisonings, and motor vehicle accidents. The increase was particularly pronounced for poisonings at ages 15–24 and 40–59. The increase in unintentional injuries is likely to be related to rises of drug abuse, alcohol consumption, and binge drinking, and also to the use of prescribed psychoactive substances (CDC 2004a, b; Compton and Volkow 2006; Fingerhut and Cox 1998; Serdula et al. 2004).

Some risk factors are especially characteristic of the United States and also contribute to life expectancy losses at working ages. These are traditionally easy access to firearms, resulting in higher risk of homicide (Kaplan and Geling 1998), and especially active use of automobiles, which increases the risk of fatal traffic accidents (Heuveline 2002).

Finally, it is worth mentioning the rapid spread of obesity that is considered to be a serious public health concern in the United States (Breslow 1952; Kim and Popkin 2006; Olshansky et al. 2005; Sturm 2003; WHO 1998). This process has accelerated: between 1988–1994 and 1999–2000, age-adjusted prevalence of overweight ($BMI \geq 25$) increased from 56% to 65%, prevalence of obesity ($BMI \geq 30$) increased from 23% to 31%, and prevalence of clinically severe obesity ($BMI \geq 40$) increased from 3% to 5% (Flegal et al. 2002; Sturm 2003). In the early 2000s, the U.S. prevalences of overweight (25%) and obesity (7%) among children aged 10–16 years were the second highest among 34 countries (Janssen et al. 2005). Obesity increases the risk of a number of circulatory diseases, Type 2 diabetes, certain cancers, gallbladder disease, and osteoarthritis (Kim and Popkin 2006). The minimal number of annual deaths in the United States attributable to obesity is estimated at 112,000 (Flegal et al. 2005; Mark 2005). Although mortality among the obese tends to decrease with time, the spread of obesity contributes to premature death in the United States, contributes to the gap between the United States and countries with lower levels of obesity, and has the potential to slow down the general mortality decline.

All in all, during the last decades, health progress in the United States was slower than in other advanced nations. It was attenuated by high life expectancy losses. Further monitoring and analysis of these losses, their components, and determinants is a research priority.

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Appendix A. Derivation of Formulae for the Age-Decomposition of a Difference Between Two e^\dagger Values

The general decomposition Eq. 9a yields

$$\begin{aligned} \eta_x &= e^\dagger(M^{[x+1]}) - e^\dagger(M^{[x]}) \\ &= \frac{1}{2} \sum_{y=0}^{\omega} \left\{ d_y(M^{[x+1]}) [e_y(M^{[x+1]}) + e_{y+1}(M^{[x+1]})] - d_y(M^{[x]}) [e_y(M^{[x]}) + e_{y+1}(M^{[x]})] \right\}. \end{aligned}$$

By definition of $M^{[x]}$, the quantity $d_y(M^{[x]})$ is equal to d'_y for ages $y \leq x$ and is equal to $\frac{l'_{x+1}}{l'_{x+1}} \cdot d_y$ for ages $y > x$.

$$\begin{aligned} \eta_x &= e^\dagger(M^{[x+1]}) - e^\dagger(M^{[x]}) \\ &= \frac{1}{2} \cdot \left\{ \sum_{y=0}^{x-1} [d'_y (e_y(M^{[x+1]}) - e_y(M^{[x]}) + e_{y+1}(M^{[x+1]}) - e_{y+1}(M^{[x]}))] \right. \\ &\quad + d'_x [e_x(M^{[x+1]}) + e_{x+1}(M^{[x+1]})] - d_x [e_x(M^{[x]}) + e_{x+1}(M^{[x]})] \\ &\quad \left. + \frac{l'_{x+1}}{l'_{x+1}} \sum_{y=x+1}^{\omega} [d_y \cdot (e_y + e_{y+1})] - \frac{l'_x}{l'_x} \sum_{y=x+1}^{\omega} [d_y \cdot (e_y + e_{y+1})] \right\}. \end{aligned}$$

Three lines of the latter expression are parts of the age-specific component related to ages younger than age x , to the age group $[x, x+1)$, and to ages $x+1$ and older, respectively.

Formulae for age-specific components of differences between life expectancies are given in Eq. 8a:

$$e_0(M^{[x+1]}) - e_0(M^{[x]}) = \delta_x = l'_x(e'_x - e_x) - l'_{x+1}(e'_{x+1} - e_{x+1})$$

and

$$e_y(M^{[x+1]}) - e_y(M^{[x]}) = \delta_x(y) = \frac{1}{l'_y} [l'_x(e'_x - e_x) - l'_{x+1}(e'_{x+1} - e_{x+1})] \text{ for any age } y, y < x.$$

Using these formulae, one can obtain the final expression for the component η_x contributed by age interval $[x, x+1)$ to the total difference $e^{\dagger'} - e^\dagger$:

$$\eta_x = \frac{\delta_x}{2} \cdot \sum_{y=0}^{x-1} \left[\frac{d'_y}{l'_y} + \frac{d'_y}{l'_{y+1}} \right] + \frac{d'_x}{2} \left(e_x + e_{x+1} + \frac{\delta_x}{l'_x} \right) - \frac{d_x \cdot l'_x}{2l'_x} (e_x + e_{x+1}) + \left(\frac{l'_{x+1}}{l'_{x+1}} - \frac{l'_x}{l'_x} \right) \cdot l_{x+1} \cdot e^\dagger_{x+1}.$$

Appendix B. Derivation of Formulae for the Age- and Cause-of-Death Decomposition of a Difference Between Two e^\dagger Values

The continuous definition of e^\dagger in Eq. 1 together with the general decomposition Eq. 9a allow one to express the component of the total difference $e^\dagger_0(M') - e^\dagger_0(M)$

produced by a small age interval $[x, x + \Delta x)$:

$$\begin{aligned} \Delta x \eta_x &= e_0^\dagger(M^{[x+\Delta x]}) - e_0^\dagger(M^{[x]}) = \int_0^x l'_y \cdot \mu'_y \cdot [e_y(M^{[x+\Delta x]}) - e_y(M^{[x]})] dy \\ &+ \int_x^{x+\Delta x} [l'_y \cdot \mu'_y \cdot e_y(M^{[x+\Delta x]}) - l_y \cdot \mu_y \cdot e_y(M^{[x]})] dy + \int_{x+\Delta x}^\infty [l_y(M^{[x+\Delta x]}) - l_y(M^{[x]})] \mu_y \cdot e_y dy. \end{aligned} \tag{B1}$$

Three integrals in Eq. B1 are parts of the age-specific component related to ages younger than x , to the age group $[x, x + \Delta x)$, and to ages $x + \Delta x$ and older, respectively.

From definitions of the survivorship and the life expectancy functions, it is easy to derive the following relations that hold true for a small Δx (see also Shkolnikov et al. 2003:328):

$$l_{x+\Delta x} \cong l_x \cdot (1 - \mu_x \Delta x), \tag{B2}$$

$$e_{x+\Delta x} \cong e_x - (1 - \mu_x e_x) \Delta x. \tag{B3}$$

Using Eqs. B2 and B3, the second integral in Eq. B1 can be simplified:

$$\int_x^{x+\Delta x} [l'_y \cdot \mu'_y \cdot e_y(M^{[x+\Delta x]}) - l_y \cdot \mu_y \cdot e_y(M^{[x]})] dy = -l'_x \cdot (\mu_x - \mu'_x) \cdot e_{x+\Delta x} \cdot \Delta x. \tag{B4}$$

Taking into account that $l_y(M^{[x]}) = \frac{l'_x}{l'_x} \cdot l_y$, $l_y(M^{[x+\Delta x]}) = \frac{l'_{x+\Delta x}}{l'_{x+\Delta x}} \cdot l_y$ ($x > y$) and also Eq. B2, the third integral can be transformed:

$$\begin{aligned} &\int_{x+\Delta x}^\infty [l_y(M^{[x+\Delta x]}) - l_y(M^{[x]})] \mu_y e_y dy \\ &= \left(\frac{l'_{x+\Delta x}}{l_{x+\Delta x}} - \frac{l'_x}{l_x} \right) \cdot \int_{x+\Delta x}^\infty l_y \mu_y e_y dy \cong \frac{l'_x}{l_x} (\mu_x - \mu'_x) \Delta x \cdot \int_{x+\Delta x}^\infty l_y \mu_y e_y dy. \end{aligned} \tag{B5}$$

Using (B3), it is possible to show that

$$e_y(M^{[x+\Delta x]}) - e_y(M^{[x]}) \cong \frac{l'_x (\mu_x - \mu'_x) e_x}{l'_y} \Delta x, x > y.$$

This relation helps to transform the first integral in Eq. B1:

$$\int_0^x l'_y \cdot \mu'_y \cdot [e_y(M^{[x+\Delta x]}) - e_y(M^{[x]})] dy = l'_x \cdot e_x \cdot (\mu_x - \mu'_x) \Delta x \cdot \int_0^x \mu'_y dy. \tag{B6}$$

Replacement of the three original integrals in Eq. B1 by their equivalents from Eqs. B4, B5, and B6 yields

$$\begin{aligned}\Delta x \eta_x &= \left[l'_x \cdot e_x \cdot \left(\int_0^x \mu'_y dy - 1 \right) + \frac{l'_x}{x} \int_{x+\Delta x}^{\infty} l_y \cdot \mu_y \cdot e_y dy \right] \cdot (\mu_x - \mu'_x) \cdot \Delta x \quad (\text{B7}) \\ &= \phi_x (\mu_x - \mu'_x) \cdot \Delta x\end{aligned}$$

This formula is the final expression for the component of the difference between two e^\dagger values produced by a mortality change within a small age interval $[x, x + \Delta x)$. Most important, it includes the $\mu_x - \mu'_x$ multiplier. Following our earlier study, one can integrate the left- and right-hand sides of Eq. B7 over the age interval $[x, x+1)$ (Shkolnikov et al. 2003:328–29) and obtain

$$\eta_x = (m_x - m'_x) \cdot \int_x^{x+1} \phi_y dy.$$

Consequently,

$$\eta_{x,i} = \frac{m_{x,i} - m'_{x,i}}{m_x - m'_x} \eta_x,$$

where $m_{x,i}$ is the death rate by cause i at age x .

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