Losses of expected lifetime in the US and other developed countries:
methods and empirical analyses

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ABSTRACT

Patterns of diversity in age at death are examined using \( e^+ \), a dispersion measure that also equals the average expected lifetime lost at death. We apply two methods for decomposing differences in \( e^+ \). 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 US 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. The difference is related to excess mortality at ages under 65 from various causes in the US. Regression on 17 country-series suggests that \( e^+ \) 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 inter-country mortality variation. It also suggests that factors affecting overall mortality decrease differ from those responsible for excess lifetime losses in the US in particular. The latter can be related to weaknesses of health system and other factors resulting in premature death including heart diseases, amenable causes, accidents and violence.
INTRODUCTION

There has been a long standing tendency for mortality decrease to be steeper at younger than at older ages. This tendency, also called “rectangularisation” of the 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. 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 (Wilmoth and Horiuchi 1999; Shkolnikov, Andreev, Begun 2003; Zhang and Vaupel 2008). It was suggested that this 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 inter-country differences with respect to the relationship between life expectancy and the amount of diversity in age at death. In particular, the US population is characterized by unexpectedly high diversity in age at death compared to the average life span, due to 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 (Kochanek, Mauer, Rosenberg 1994; Elo and Drevenstadt 2004), the relative inter-individual difference in age at death over 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 US. After analyzing relations between S_{10} and external-cause
mortality, race, educational, and income inequalities, the authors conclude 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…”.

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 US that was initiated in 2007 at the Annual Meeting of the Population Association of America (Panel Discussion 2007).

Apart from $S_{10}$, inter-individual diversity in age at death has been measured by the inter-quartile range IQR (Wilmoth and Horiuchi 1999), Gini coefficient and likely measures of relative inequality (Anand et al. 2001; Gakidou et al. 2003; Shkolnikov et al. 2003; Smits and Monden 2009), Theil index of inequality (Smits and Monden 2009) and average inter-individual difference and the related measures of absolute inequality (Shkolnikov et al. 2003; Moser, Shkolnikov and Leon 2005). 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 $e^\dagger$ (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 due to death. It generally follows Keyfitz’s idea that “everybody dies prematurely” since 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 inter-individual 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 US 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 US.

METHODS

$e^\dagger$ as a measure of lifetime losses and of diversity in age at death

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

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

where $l(y), \mu(y), e(y)$ are survivorship, the force of mortality, and life expectancy 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:
\[ e_x^+ = \frac{1}{l_x} \sum_{y=x}^{\omega-1} d_y \bar{e}_y = \frac{1}{2l_x} \sum_{y=x}^{\omega-1} d_y (e_y + e_{y+1}), \]  \hspace{1cm} (2a) \\
\[ e_x^+ = \frac{1}{l_x} \sum_{y=x}^{\omega-1} d_y \bar{e}_y = \frac{1}{l_x} \sum_{y=x}^{\omega-1} d_y (1-a_y) + e_{y+1}. \]  \hspace{1cm} (2b) 

Formula (2b) is slightly more precise since 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 \( l_0=1 \), formula (2b) yields

\[ e^+ = e^+_0 = \sum_{y=0}^{\omega-1} d_y e_{y+1} + (1 - \sum_{y=0}^{\omega-1} d_y a_y), \]  \hspace{1cm} (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 \( y \) due 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

\[ e_{x+1} = \frac{1}{l_{x+1}} \sum_{y=x+1}^{\omega-1} d_y (\bar{y} - \bar{x}), \]  where \( \bar{y} \) and \( \bar{x} \) are mean ages at death within elementary intervals \( [y, y+1) \) and \( [x, x+1) \), respectively. Substituting life expectancy by the latter expression in the first term of formula (3) yields

\[ e^+ = \sum_{x=0}^{\omega-1} \frac{1}{l_x} \left[ \sum_{y=x+1}^{\omega-1} d_y (\bar{y} - \bar{x}) \right] + (1 - \sum_{y=0}^{\omega-1} d_y a_y). \]  \hspace{1cm} (4) 

\(^1\bar{x} = x + a_y, \hspace{1mm} \bar{y} = y + a_y\)
Formula (4) suggests that the core part of $e^\dagger$ is equal to a weighted inter-individual difference in age at death. Thus, there is a clear similarity between $e^\dagger$ and the numerator of the Gini coefficient, that can be also called the average inter-individual difference (AID) in length of life (Shkolnikov et al. 2003; Moser et al. 2005).

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

(5)

The presence of weights ($1/l_x$) in formula (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 ($n=1972$) is close to 0.99 both for males and females. The corresponding correlation coefficients between $e^\dagger$ and $S_{10}$ are lower: 0.71 and 0.81 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.

**Direct and compositional components of differences and changes**

average rate of mortality reduction and the age-pattern of this reduction. Longevity progress depends on each of the two factors 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 \( \Delta e_{tot} = e^\dagger (M_1) - e^\dagger (M_0) \) is a sum of two components produced by the amount of mortality change (direct component) and the age-structure of this change (compositional component):

\[
\Delta e^\dagger_{dir} = e^\dagger (\lambda \cdot M_0) - e^\dagger (M_0), \tag{6a}
\]

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

where the mean rate of mortality change is \( \lambda = \frac{1}{\omega} \sum_x (m_{x,1} / m_{x,0}) \) with \( m_{x,1} \) and \( m_{x,0} \) denoting elements of the vectors \( M_1 \) and \( M_0 \), respectively. Formulae (6a) and (6b) represents 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 average 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 definitions (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 younger ages. \( \bar{e}_x \) 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, Shkolnikov, Begun 2002). The age-specific contributions must be free from side influences of other ages. Such decompositions of time changes or inter-country 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

\[
\delta_x = e_0 (M^{[x+1]}) - e_0 (M^{[x]}).
\]

(7a)

In this formula, \( M^{[x]} \) stands for a vector of age-specific death rates containing elements \( m'_y \) at ages from 0 to \([x, x+1]\) and elements \( m_y \) at ages \( y \) from \([x+1, x+2]\) to \( \omega \). This implies that formula (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 (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 $m_y$:

$$
\delta_x = e_0(M^{[x+1]}) - e_0(M^{[x]}).
$$

(7b)

Procedures (7a)-(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; Arriaga 1984; Andreev et al. 2002):

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

(8a)

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

(8b)

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

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

$$
\eta_x = e^\dagger(M^{[x+1]}) - e^\dagger(M^{[x]}).
$$

(9a)

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

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

(10a)

The opposite-direction replacement corresponds to the components

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

(9b)
Then the formula similar to (10a) is

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

The final components are: 

\[
\bar{\eta}_i = \frac{1}{2} (\eta_x - \eta'_x).
\]

As we know, the life expectancy components \( \bar{\delta}_i \) can be further split by causes of death (Andreev 1982)

\[
\bar{\delta}_{x,i} = \bar{\delta}_x \cdot \frac{m'_{x,i} - m_{x,i}}{m'_{x} - m_{x}},
\] (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)

\[
\bar{\eta}_{x,i} = \bar{\eta}_i \cdot \frac{m'_{x,i} - m_{x,i}}{m'_{x} - m_{x}}.
\] (12)

Expressions (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 \( e^{+} \) 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 UK, and the US. Data for the calculations were extracted from the World Income Inequality Database and a small number of additional sources (WIID2 2008; the World Bank 2008; OECD Statistics 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 either. In the regression analysis, only quinquennial data were used including years 1975, 1980, 1985, 1990, 1995, 2000, and 2004-5. 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). The $e^\dagger$ statistic is taken as the dependent variable and the Gini index of income inequality and time-dummies 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 US, England and Wales, Japan, and Sweden. The figure covers the years after the Second World War 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.

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 US for most of the period. Although at certain moments in the past the US $e^\dagger$ values had been quite close to $e^\dagger$ values in Japan, England and Wales, and Sweden, later on the gap between the US and other countries has widened due to a flatter $e^\dagger$ trajectory in the US compared to the other countries. The trajectories for Japan, England and Wales and Sweden converge remarkably (especially for females) and constitute a clear difference from the US. Over a long period of time the US and the English life expectancies have been close to each other. During the last decade life expectancy values in England and Wales were higher than those in the US by about one year for males and by about half a year for females. In spite of the closeness in average longevities, life expectancy losses are substantially greater in the US 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 present in the Human Mortality Database. Once again, one can see a tight negative association between $e_0$ and $e^\dagger$ ($r=-0.95$ and $r=-0.82$ for males and females, respectively). There are also some differences in $e^\dagger$ between countries with the same level of life expectancy. The US $e^\dagger$ values lie considerably higher than the expected values on the trendline. For example, in 2002 an average male death in the US caused a loss of 12.5 years of lifetime, whereas the corresponding expected value on the trendline is 11.4 years (left panel of Figure 2). This means that in 2002 an average male death in the US caused an excess loss of 1.1
years of lifetime compared to what can be expected from experience of other countries. For US females, the observed and the expected values of $e^\dagger$ in 2002 are 11.1 and 10.0 years respectively, suggesting the same excess loss of 1.1 years of lifetime.

Figure 1. The $e^\dagger$ vs. $e_0$ trajectories in England and Wales, Japan, Sweden, and the US after the year when life expectancies in these countries reached 60 years for males and 65 years for females.


According to definition (2a), the high life expectancy losses in the US 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 US and England and Wales in 1950 and 2002. During this period, in both countries a
A gross shift of the whole death distribution toward older ages has occurred. At both time points, there is almost no difference between the modes of the two distributions, however the US distributions in both 1950 and 2002 are more dispersed than those of England and Wales. The US distributions have heavier left tails corresponding to young-middle ages. For males, the inter-country 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.

![Figure 2. The $e^\dagger$ vs. $e_0$ correspondence for 29 industrialized Human Mortality Database countries in 2002.](image)


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

In 1950, age-specific death rates in the US were on average higher than those in England and Wales by about 7% for males and 9% for females. By the year 2002 these differences have 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 US for the two years. The $e^\dagger$ differences in favor of England and Wales have increased from 1.6 to 1.7 years for males and from
1.0 to 1.2 years for females. Calculations according to formulae (6a) and (6b) make it clear that both in 1950 and 2002 more than 90% of the inter-country difference in $e^t$ 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.

![Graph showing comparison of $d_x \cdot \bar{e}_x$ curves between the US and England and Wales in 1950 and 2002.](image)

**Figure 3. Comparison of the $d_x \cdot \bar{e}_x$ curves between the US and England and Wales in 1950 and 2002.**


Oeppen (2008) introduced the concept of efficiency of the age pattern of mortality change. It 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 US are far from being optimal since the US excess in lifetime losses 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 and in the US in 1950 and 2002. (in years).

<table>
<thead>
<tr>
<th></th>
<th>Values</th>
<th>Components of the differences</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EW</td>
<td>US</td>
</tr>
<tr>
<td></td>
<td>(a)</td>
<td>(b)</td>
</tr>
<tr>
<td>1950</td>
<td>13.68</td>
<td>15.24</td>
</tr>
<tr>
<td>2002</td>
<td>11.09</td>
<td>12.76</td>
</tr>
<tr>
<td>1950</td>
<td>12.94</td>
<td>13.98</td>
</tr>
<tr>
<td>2002</td>
<td>10.19</td>
<td>11.41</td>
</tr>
</tbody>
</table>

Source: Authors’ calculations on data from the Human Mortality Database.

Age- and cause-of-death components of change in life expectancy losses between 1980 and 2002

Formulae (7a, b) and (9a, b) allow one to decompose by age the increase in $e_0$ and decrease in $e^\uparrow$, respectively. Figure 4 presents age-specific components of changes between 1980 and 2002 in the US and England and Wales\(^2\). The Figure 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 to 85 (upper panels of Figure 4). Smaller contributions are produced by reductions of infant deaths and of younger adult mortality at ages 15 to 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 US. At the same time, the younger adult-age components are greater in the US than those in England and Wales.

For the life expectancy losses (lower panels of Figure 4), age patterns of change are quite different. Depending on age, the mortality reduction produces negative or positive contributions to

\(^2\) The last year for which the cause-of-death data are currently available for the two countries in the WHO Mortality Database.
the total $e^\dagger$ change (Zhang and Vaupel 2008). At ages younger than a threshold age (named $a^\dagger$ 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 $d_e \cdot \bar{\varepsilon}_e$ 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-fold greater than sums of the positive components (mortality expansion). Finally, the lower panels of Figure 4 demonstrate that England and Wales experienced greater compression and expansion components of the total $e^\dagger$ change than the US.

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 $a^\dagger$ has increased from 67.5 to 72.5 years and from 68.5 years to 74.5 years in the US and England and Wales, respectively. During the same period, the female $a^\dagger$ has increased from 76.5 to 78.5 years and from 75.5 to 79.5 years in the US and England and Wales, respectively.

From a public health perspective, the degree of mortality compression is especially important. It shows to what extent 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 as 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 4. Age-specific components of increases in life expectancy and decreases in life expectancy losses between 1980 and 2002 in the US and England and Wales.
Source: Human Mortality Database

Figure 5 shows cause-of-death components of mortality compression in the two countries. As the threshold ages are located within age groups 65-69 and 70-74 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 $e^\uparrow$ decrease between 1980 and 2002 are computed from formulae (11)-(12). The greatest contributions are produced by coronary and other circulatory diseases. 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. Considerable contributions are also produced by lowering mortality from lung cancer (males) and from breast and other cancers (females), by lowering mortality from the traffic accidents (males) and violent causes of death (males in the US).
Comparison between the two countries shows that in 1980-2002 England and Wales experienced greater reduction in $e^{\dagger}$ due to chronic conditions such as circulatory diseases, male lung cancer and female breast cancer. The US experienced greater effects related to the reduction of male mortality from accidents and violence.

**The inter-country differences in life expectancy losses in 2002**

Figure 6 exhibits age-specific components of differences between England and Wales and the US in life expectancy and in life expectancy losses in the year 2002 and reveals marked
differences from the age patterns of temporal change in Figure 4. First, the lower US 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 US death rates at ages under 75. At the same time, the components of inter-country difference in \( e^† \) life expectancy losses are now negative at all ages. Second, the role of older adult age and infant-age components is relatively less important in the inter-country differences than the role of the equivalent components of the temporal change. At the same time, the contributions of younger adult ages are more important than the equivalent contributions to the temporal change.

![Graphs showing life expectancy and life expectancy losses for males and females in England and Wales and the US, 2002.](image)

**Figure 6.** Age-specific components of differences between England and Wales and the US in life expectancy and in life expectancy losses in 2002.


The cause-of-death pattern of the inter-country difference in mortality compression (Figure 7) also differs substantially from the equivalent pattern of temporal change (Figure 5). Indeed, the
role of heart diseases is relatively less prominent in the inter-country difference, while causes of
death characteristic of younger adult ages such as transport and other accidents, violence and
HIV/AIDS are more important for the inter-country difference.

Figure 7. Cause- and age-specific components of differences between England and Wales and
the US in life expectancy losses in 2002 for the range of ages under the threshold.

Table 2 shows that if the mortality difference between the US and England and Wales at
ages under the threshold age was instantly eliminated, US average longevity would exceed the
values for England and Wales. US life expectancy losses would become much lower, but would
still exceed corresponding values in England and Wales.
Table 2. Life expectancy and life expectancy losses in 2002: effects of elimination of the excess mortality in the US compared to England and Wales at ages under 70 (males) and ages under 75 (females).

<table>
<thead>
<tr>
<th></th>
<th>( e_0 ) US</th>
<th>( e_0 ) EW</th>
<th>( e_0 ) US after elimination</th>
<th>( e'_{0} ) US</th>
<th>( e'_{0} ) EW</th>
<th>( e'_{0} ) US after elimination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>74.54</td>
<td>76.21</td>
<td>76.57</td>
<td>12.51</td>
<td>10.84</td>
<td>11.22</td>
</tr>
<tr>
<td>Females</td>
<td>79.79</td>
<td>80.74</td>
<td>81.14</td>
<td>11.16</td>
<td>9.93</td>
<td>10.27</td>
</tr>
</tbody>
</table>

Source: Authors’ calculations on data from the Human Mortality Database, 2007.

Associations between life expectancy losses and economic inequality

Is it only a coincidence that the US 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 US 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 \( e_0 \) and the Gini index across countries appear to be negative (as expected) but small and statistically insignificant: -0.20 and -0.25 for males and females, respectively. Coefficients of correlation between \( e'_{0} \) and the Gini index are much higher and statistically significant (\( p<0.05 \)): 0.70 for males and 0.73 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 \( e'_{0} \) 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 trends. The Chow test suggests that significant fixed country-effects do exist. There is only a very weak positive longitudinal association for males (p<0.10) and no significant relationship 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<0.05), the Hausman test suggests priority for the fixed effects model.

All in all, the regression results suggest that there is a cross-sectional association between lifetime losses and income inequality, but temporal changes in lifetime losses are independent or almost independent of changes in income inequality.

To understand whether a high diversity in age at death in the US 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, Sorlie, Johnson et al. 1992). It was found that the better-off groups experienced lower values for the standard deviation of ages at death for ages 10+, $S_{10}$, but even in these groups $S_{10}$ values were high when compared to the international standard. Using the same NLMS data, we calculated life expectancy at age 30 and life expectancy losses at ages 30+ for larger numbers of more finely defined educational, income, and racial groups and also for their two-dimensional combinations (Appendix C). It appears that in 1979-1985, the differences in life expectancy losses between the most advantaged and the most
disadvantaged groups was nearly 5 years. Values of $e^\dagger$ are about 15 years for African American males and females in the lowest income group vs. $e^\dagger$ 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 experience 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 in 1979-1985.

**SUMMARY OF RESULTS**

This study further develops a toolkit for the analysis of inter-individual 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 due to death. 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 3. Relationship between $e^{t_0}$ and Gini index of income inequality across countries and time. *

<table>
<thead>
<tr>
<th>Model:</th>
<th>Between</th>
<th>Pooled LS with fixed country effects</th>
<th>Generalized LS with random country effects</th>
<th>Between</th>
<th>Pooled LS with fixed country effects</th>
<th>Generalized LS with random country effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Males</td>
<td></td>
<td></td>
<td>Females</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gini</td>
<td>0.072</td>
<td>0.013</td>
<td>0.019</td>
<td>0.081</td>
<td>0.008</td>
<td>0.011</td>
</tr>
<tr>
<td></td>
<td>(0.012)§</td>
<td>(0.090)</td>
<td>(0.014)</td>
<td>(0.003)</td>
<td>(0.174)</td>
<td>(0.044)</td>
</tr>
<tr>
<td></td>
<td>(0.000)</td>
<td>(0.000)</td>
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<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Statistical tests:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chow. H0: Absence of fixed effects</td>
<td>-</td>
<td>36.78</td>
<td>-</td>
<td>55.33</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Hausman. H0: No systematic difference between random and fixed effects</td>
<td>-</td>
<td>-</td>
<td>17.67</td>
<td>-</td>
<td>-</td>
<td>104.78</td>
</tr>
<tr>
<td>Breusch-Pagan. H0: Absence of random effects</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>199.19</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Notes: * Populations: Australia, Austria, Belgium, Canada, Denmark, Finland, France, Italy, Japan, New Zealand, Norway, Spain, Sweden, Taiwan, the Netherlands, USA, UK.

§ p-values are given in parentheses.


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 attention to cross-sectional differences among countries that (unlike temporal change) appear to be associated with economic inequality. Using a decomposition, we show that excess life expectancy losses in the US 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 inter-country comparisons. The analysis reveals persistently high values of $e^{\dagger}$ in the US that are caused by relatively low mortality compression and relatively high mortality expansion in this country. In 2002, lifetime losses among US males and females were greater by 1.1 year than the values expected on the basis of the experience of other developed countries. Compared to England and Wales, the US 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 life expectancy losses’ differences between the two countries in 2002. We find that:

- 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 US.

- mortality at younger adult ages 15 to 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 is more important as a component of the gap in life expectancy losses between the US and England and Wales.

Seeking interpretations of the observed patterns of life expectancy losses, we carry out additional analyses. We perform regressions connecting $e^+$ 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 $e^+$ values for categories of education, income, and race and all of their pair wise combinations for the US. Although life expectancy losses vary significantly across groups, even the lowest $e^+$ 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 US.
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 age- and cause-decompositions for the US 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 from 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 points out the specificity of the strength of association between income inequality and health in regard to type of health outcome. It was demonstrated that 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 (Lynch et al. 2004; Deaton and Lubotsky 2003; Lynch et al. 2001; 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. 2004, pp. 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 (Lynch et al. 2004; Lohmayer and Wilkinson 2000, Kennedy, Kawachi and Prothrow-Stith 1996; Sohler et al. 2002; Daly, Wilson and Vesdev 2001; Kennedy et al. 1998; Szwarcwald et al. 1999; Wilkinson, Kawachi and Kennedy 1998; Franzini and Spears 2003; Osler et al. 2003; Shi et al.
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 US was associated with differentials in state level mortality at ages 25 to 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 US and other advanced countries general mortality decrease over the last three decades 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 temporal decrease in life expectancy losses differ from factors for inter-country differences. Decrease in the life expectancy 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 and also to favorable behavioral changes such as reduction in smoking (Pampel 2003). The inter-country differences are to a greater extent related to health and mortality at younger adult ages that is probably 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 temporal 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 the historical health crises such as the one in 19th century Sweden (Willner 2001, Sundin and Willner 2004) and the recent crisis in
the former USSR were largely caused by excess mortality of men from causes associated with alcohol that was attributed to psychosocial stress (Shapiro 1995; Bobak and Marmot 2000; Leon and Shkolnikov 1998).

In the modern developed world such outbreaks of working-age mortality are exceptions from mainstream health progress. In the US (as in other advanced countries) changes in life expectancy losses are mainly driven by decreasing chronic disease among older people and are mostly unrelated to income inequality. However, the US excess in life expectancy losses relative to other countries is related to the higher US 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 US. So far, progress in this area has not been rapid enough and separation between the US and other countries tends to be sustained. High life expectancy losses in the US 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 US. It is noteworthy that the US 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. 2004).

Socioeconomic disparities in health between population groups comprise a part of the total amount of inter-individual disparity in respect to age at death (and in life expectancy losses). In the second half of the 20th century mortality reversals have been observed twice in the African American population (Kochanek et al. 1994; Preston and Elo 1995; Geronimus et al. 2001; Elo and Drevenstadt 2004). It was shown that the last episode, lasting from 1984 to 1991, coincided with an increase in age at death disparity among US 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 US, there are huge geographical differences in mortality that are related not only to the variable socioeconomic status of individuals in various places but also to highly variable geographic contexts (Murray et al. 2006; Ezzati, Friedman, Kulkarni 2008).

However, the socioeconomic health contrasts are unlikely to be responsible for the total amount of excess life expectancy losses in the US. Indeed, on the basis of NLMS data we found 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 US is shifted toward higher values compared to the equivalent range in England and Wales. This suggests that there are some adverse factors that affect all or many of the US population groups.

The American health system is one of the candidate factors. An important disadvantage of the US is the incomplete population coverage and variable performance characteristic of the health system. The detailed investigation by Schoen and How (2006) reveals a range of concrete problems in medical care, which 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 while the same share of people have difficulties in paying their medical bills. For people of working age, availability and quality of medical care depends not only on their wealth but also significantly varies across health care plans, states, and hospitals. Only 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 very variable across the country.
It is likely that the disadvantage of the US 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. In 1997-1998, the US occupied the 15th place according to amenable mortality. By 2002-2003, the decline in amenable mortality comprised 17% for all OECD countries and only 4% for the US. As a result, the US has fallen 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 US. This can be largely related to smoking. In the mid-1960s the country was among the most smoking nations of the world with a smoking prevalence of about 50% and 30% for males and females aged 18+, respectively (Garfinkel and Silverberg 1991). Since then smoking has been dramatically reduced to 26% and 22% in 2005 among males and females, respectively. The steeper decrease in smoking of men over the last forty years 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 US has been estimated directly from survey data (Rogers et al. 2005) and also by indirect methods (Peto et al. 2006; Preston, Glei, Wilmoth 2009). 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 to 69 in the US in 2000 were attributable to smoking. Preston, Glei and Wilmoth report similar figures and demonstrate that among 20 developed countries in the year 2003, the share of smoking-attributable death in the US 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 (Nizard and Muños-Perez 1993; Edwards and Tuljapurkar 2005). The US is also not free of such problems. Between 1992 and 2002, US mortality from unintentional injuries has increased by 11% (Paulozzi, Ballesteros, Stevens, 2006). In the age group 40 to 64 years, death rates increased for falls, poisonings, and motor vehicle accidents. The increase was particularly pronounced for poisonings at ages 15 to 24 and 40 to 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 (Fingehrut and Cox 1998; CDC 2004a; CDC 2004b; Serdula et al. 2004, Compton and Volkow 2006).

Some risk factors are especially characteristic of the US 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 that 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 US (Breslow 1952; WHO 1998; Sturm 2003; Olshansky et al. 2005; Kim and Popkin 2006). During the last decades, this process has accelerated. Between 1988-1994 and 1999-2000 age-adjusted prevalence of overweight BMI\(\geq25\) has increased from 56% to 65%, prevalence of obesity (BMI\(\geq30\)) has increased from 23% to 31% and prevalence of clinically severe obesity (BMI\(\geq40\)) has increased from 3% to 5% (Flegal et al. 2002; Sturm 2003). In the early 2000s, the US 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-II diabetes, certain cancers, gallbladder disease, and osteoarthritis (Kim and Popkin 2006). The minimal number of annual deaths in the US attributable to obesity is estimated at 112 thousand (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 US, contributes to the gap between the US 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 US 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.

ACKNOWLEDGEMENTS

This study is a part of project “Methods of measurement and decomposition in mortality studies” at the Max Planck Institute for Demographic Research. We would like to express our sincere gratitude to Demography’s invited editor Ken Hill for his constructive and insightful comments to the paper. We are also grateful to two anonymous reviewers for their helpful comments and rigorous check of the text, formulae, and tables. Finally, we extend our gratitude to Edelgard Katke from MPIDR for final formatting of the manuscript.
APPENDIX A.
Derivation of formulae for the age-decomposition of a difference between two \( e^x \) values.

The general decomposition formula (8a) yields

\[
\eta_x = e^x (M^{x+1}) - e^y (M^x) = \\
= \frac{1}{2} \sum_{y=0}^{\omega} \left\{ d'_y (M^{x+1}) [e'_y (M^{x+1}) + e_{y+1} (M^{x+1})] - d_y (M^{x+1}) [e_y (M^{x+1}) + e_{y+1} (M^{x+1})] \right\}
\]

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 \( \frac{l'_{x+1}}{l_{x+1}} d_y \) for ages \( y > x \).

\[
\eta_x = e^x (M^{x+1}) - e^y (M^x) = \\
= \frac{1}{2} \sum_{y=0}^{\omega} \left\{ d'_y [e'_y (M^{x+1}) - e_y (M^{x+1}) + e_{y+1} (M^{x+1}) - e_{y+1} (M^x)] \right\} + \\
+ d'_y [e'_y (M^{x+1}) + e_{y+1} (M^{x+1})] - d_y [e_y (M^{x+1}) + e_{y+1} (M^x)] + \\
+ \frac{l'_{x+1}}{l_{x+1}} \sum_{y=x+1}^{\omega} \left[ d_y \cdot (e_y + e_{y+1}) \right] - \frac{l'_{x}}{l_{x}} \sum_{y=x+1}^{\omega} \left[ d_y \cdot (e_y + e_{y+1}) \right]
\]

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 known:

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

and

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

Using these formulae, one can obtain the final expression for the component \( \eta_x \) contributed by age interval \([x, x+1)\) to the total difference \( e' - e^y \)
\[\eta_x = \frac{\delta_x}{2} \sum_{y=x}^{x+\Delta x} \left[ \frac{d^+}{d^+} + \frac{d^+}{l_y^+} \right] + \frac{d^+}{2} \left( e_x + e_{x+1} + \frac{\delta_{x+1}^+}{l_x} \right) - \frac{d^+}{2l_x} (e_x + e_{x+1}) + \left( \frac{l_{x+1}^+}{l_{x+1}^+} - \frac{l_x^+}{l_x} \right) \cdot l_{x+1}, e_{x+1}^+ \]

**APPENDIX B.**

**Derivation of formulae for the age- and cause-of-death decomposition of a difference between two \(e^+\) values.**

The continuous definition of \(e^+\) (1) together with the general decomposition formula (8a) allow one to express the component of the total difference \(e_0^+(M') - e_0^+(M)\) produced by a small age interval \([x, x+\Delta x]\)

\[\Delta \eta_x = e_0^+(M^{[x+\Delta x]}) - e_0^+(M^{[x]}) = \int_0^{x+\Delta x} l_y^+ \cdot \mu_y^+ \left[ e_y(M^{[x+\Delta x]}) - e_y(M^{[x]}) \right] dy + \int_x^{x+\Delta x} l_y^+ \cdot \mu_y^+ \cdot e_y(M^{[x+\Delta x]}) - l_y^+ \cdot \mu_y^+ \cdot e_y(M^{[x]}) \right] dy + \int_x^{x+\Delta x} l_y^+ \left( M^{[x+\Delta x]} \right) - l_y^+ \left( M^{[x]} \right) \bigg] dy \]

(B-1)

Three integrals in (B-1) are parts of the age-specific component related to ages younger than \(x\), of the age group \([x, x+\Delta x]\) and of 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 \(\Delta x\) (see also Shkolnikov et al. 2003: 328):

\[l_{x+\Delta x} \equiv l_x \cdot (1 - \mu_x \Delta x), \quad (B-2)\]

\[e_{x+\Delta x} \equiv e_x - (1 - \mu_x e_x) \Delta x \quad (B-3)\]

Using (B-2) and (B-3) the second integral in (B-1) can be simplified

\[\int_x^{x+\Delta x} l_y^+ \cdot \mu_y^+ \cdot e_y(M^{[x+\Delta x]}) - l_y^+ \cdot \mu_y^+ \cdot e_y(M^{[x]}) \bigg] dy = \int_x^{x+\Delta x} l_y^+ \cdot (\mu_x^+ - \mu_y^+) \cdot e_{x+\Delta x} \cdot \Delta x \]

(B-4)

Taking into account that \(l_y(M^{[x]}) = \frac{l_y'}{l_x'}, l_y(M^{[x+\Delta x]}) = \frac{l_y'}{l_x'} \cdot l_y' (x > y)\) and also (B-2), the third integral can be transformed

\[\int_x^{x+\Delta x} l_y' \left( M^{[x+\Delta x]} \right) - l_y' \left( M^{[x]} \right) \bigg] dy = \left( \frac{l_y'}{l_x'} - \frac{l_y'}{l_x} \right) \cdot \int_x^{x+\Delta x} l_y' \cdot e_y \cdot dy = \left( \frac{l_y'}{l_x'} - \frac{l_y'}{l_x} \right) \cdot \int_{x+\Delta x}^{x} l_y' \cdot e_y \cdot dy \quad (B-5)\]
Using (B-3) it is possible to show that

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

This relation helps to transform the first integral in (B-1)

\[ \int_0^x l'_y \cdot \mu'_y \cdot [e_y(M^{(x+\Delta x)}) - e_y(M^{(x)})] dy = l'_x \cdot e_x \cdot (\mu_x - \mu'_x) \Delta x \cdot \int_0^x \mu'_y dy. \quad (B-6) \]

Replacement of the three original integrals in (A2-1) by their equivalents from formulae (B-4, B-5, B-6) yields

\[ \Delta \eta_x = l'_x \cdot e_x \left( \int_0^x \mu'_y dy - 1 \right) + \frac{l'_x}{l'_x + \Delta x} \int_{x+\Delta x}^\infty \mu'_y \cdot e_x \cdot dy \cdot (\mu_x - \mu'_x) \cdot \Delta x = \]

\[ = \varphi_x \cdot (\mu_x - \mu'_x) \cdot \Delta x \quad (B-7) \]

This formula is the final expression for the component of the difference between two \( e^i \) values produced by a mortality change within a small age interval \([x, x+\Delta x]\). Most importantly, it includes the \( \mu_x - \mu'_x \) multiplier. Following our earlier study, one can integrate the left- and right-hand sides of equation (B-7) over the age interval \([x, x+1]\) (Shkolnikov et al. 2003:328-329) and obtain

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

Consequently,

\[ \eta_{x,i} = \frac{m_{x,i} - m'_{x,i}}{m_x - m_x} \eta_x, \text{ where } m_{x,i} \text{ is the death rate by cause } i \text{ at age } x. \]
APPENDIX C.
Values of $e_{30}$ and $e^\dagger_{30}$ in 1979-1985 in the national populations of the US and England and Wales and in racial, educational, income, and combined groups in the National Longitudinal Mortality Survey.

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<td>$e_{30}$</td>
<td>$e^\dagger_{30}$</td>
<td>Person-years (thousands)</td>
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Source: 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.
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