The Threshold between Compression and Expansion of Mortality

Zhang Zhen
zhang@demogr.mpg.de

James W. Vaupel
jwv@demogr.mpg.de

Max Planck Institute for Demographic Research
Konrad-Zuse-Str. 1, 18057 Rostock, Germany

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Abstract

As a result of the steady increments in life expectancy and the relatively slow increase of the maximum lifespan, mortality compression is generally hard to avoid. However, empirical findings are mixed, both for and against compression. In this study we develop a method that allows us to closely investigate the dynamic process of mortality compression. It turns out that there exists a "threshold age", say A^*, such that reductions in mortality before it lead to mortality compression, whereas those after age it to mortality expansion. The overall effect of mortality reduction on compression can thus be decomposed into two components, the compression of deaths at younger ages and the expansion of deaths at older ages. Whether the compression of mortality occurs in the whole population depends on which component is greater.

Introduction

Record life expectancy has steadily risen in the past 160 years, and shows no sign of slowing down (Oeppen and Vaupel, 2002). Meanwhile, the maximum lifespan of humans has increased too, though at a relatively slower rate (Wilmoth et al., 2000). As more and more people live longer but the upper limit of lifespan increases slowly, the distribution of deaths will be compressed at old age, which is called "mortality compression"(Fries, 1980). However, empirical findings are mixed, both for and against compression (Myers and Manton, 1984; Nusselder and Mackenbach, 1996; Wilmoth and Horiuchi, 1999; Lynch and Brown, 2001).

Mortality compression is subject to the age pattern of survival improvement. Typically, because of reductions in deaths at younger ages, some deaths that would have happened at those ages will be postponed to older ages, leading to mortality compression. Whereas, mortality improvement at older ages can make old people to live longer and thus leads to the expansion of deaths among the elderly, which will partly offset the compression of mortality resulting from reductions in mortality at younger ages. Clearly, the compression of mortality in the whole population is the result of comparison between the compression of deaths at younger ages (henceforth, *CDYA*) and the expansion of deaths at older ages (*EDOA*). Whether mortality compression occurs in the whole population depends on which impact is greater: If the *CDYA* is greater than the *EDOA*, then mortality compression will occur, and vice versa; if both of them are roughly equal, neither compression nor expansion will appear.

The coexisting of *CDYA* and *EDOA* implies that different combinations of them may lead to the same phenomenon, either compression or expansion of mortality in the whole population. For instance, the expansion of mortality may result from worsening survivorship at younger ages, which, though much less prevalent nowadays, can be found in some countries where many young adults are suffering from HIV/AIDS. Alternatively, mortality expansion may occur when reductions in mortality are greater at older ages than at younger ages (i.e., *EDOA* is greater than *CDYA*). Despite the same expansion, the implications on health and mortality are rather different or even inverse. In the former case the worsening survivorship among younger people is bad news in any sense, whereas in the latter case more and more people living longer corresponds to achievement in pursuing longevity.

To distinguish different combinations of *CDYA* and *EDOA*, it is required to precisely identify them. In doing so, we need explicitly define the threshold age, instead of using it in a vague way through the usage of "younger" or "older" ages. The threshold age is such that reductions in mortality before it lead to *CDYA*, while those after it to *EDOA*. Note

that the definition of threshold age also provides the clue to estimate it. In this study, we develop a method that allows us to estimate the threshold age and closely investigate the dynamic process of mortality compression.

The threshold age

The compression of mortality, characterized by decreasing variation of ages at death, is usually related to the equalizing of life chances. Hence, measures of lifespan disparity or variation of ages at death are also used for measuring mortality compression. In the present study we prefer lost life expectancy (*LLE*), despite other alternatives such as standard deviation (*SD*) and Inter-Quartile Range (*IQR*) of the distribution of ages at death, Keyfitz' $\mathscr{H}$, the Gini coefficient, and so on (Keyfitz, 1977; Wilmoth and Horiuchi, 1999; Shkolnikov et al., 2003; Cheung et al., 2005; Edwards and Tuljapurkar, 2005). First, *LLE* is highly correlated with all other measures.[1] Second, *LLE* has the characteristics of a life table function so that lifespan disparity can readily be investigated together with other life table functions.

LLE may date back to the concept of life deprivation due to death, which was proposed first by Keyfitz (1977). Following the line of of life table entropy (Keyfitz, 1977; Demetrius, 1978), Mitra (1978), Goldman and Lord (1986), and Vaupel (1986) independently obtained the mathematical expression for *LLE*. Recently, Vaupel and Canudas-Romo (2003) further derived the relationship between *LLE* and the rate of change in life expectancy.

In conventional notation, *LLE* is given as below

$$e^{\dagger}(t) = \int_0^{\omega} e(a,t) f(a,t) \mathrm{d}a \tag{1}$$

where $e(a,t) = \int_a^{\omega} \ell(x,t)\mathrm{d}x/\ell(a,t)$ is the remaining life expectancy at age a, $f(a,t) = \ell(a,t)\mu(a,t)$ the life table distribution of deaths, $\ell(a,t)$ the survival function at age a and time t, $\mu(a,t)$ mortality force at age a, and ω the maximum age to reach. $e^{\dagger}$ thus measures the homogeneity of a population's life chances. If $e^{\dagger}$ is small, then people die at roughly the same age, indicating mortality compression; if $e^{\dagger}$ is large, then people die at very different ages, indicating mortality expansion.

The age-specific impact of survival improvement on lifespan disparity can be specified by the derivative of $e^{\dagger}$ with respect to mortality change over time as below

$$g(a,t) = \frac{\mathrm{d}e^{\dagger}(t)}{-\mathrm{d}\ln\mu(a,t)} = f(a,t)\left[e^{\dagger}(a,t) - e(a,t)(1+\ln\ell(a,t))\right], \tag{2}$$

[1] The results of correlation coefficients are not shown here, but available for request.

where $e^\dagger(a,t) = \int_a^\omega e(x,t)f(x,t)\mathrm{d}x/\ell(a,t)$ is lost remaining life expectancy of the survival up to age a at time t. Hence, the function $g(a,t)$ measures how much change in $e^\dagger$ is due to an equal reduction in mortality at age a and time t. As shown by Figure 1(A), $g(0,t) < 0$ suggests that the decline in infant mortality may bring $e^\dagger$ up; and the bigger $|g(0,t)|$, the more contribution to the decline in $e^\dagger$ that is made by the equal reduction in infant mortality. On the contrary, $g(\omega,t) > 0$ means that $e^\dagger$ may go up because of reductions in old age mortality.

Equation (2) is helpful for obtaining threshold age, though without a very straightforward demographic interpretation. Let

$$k(a,t) = e^\dagger(a,t) - e(a,t)(1 + \ln \ell(a,t)), \tag{3}$$

with

$$g(a,t) = f(a,t)k(a,t). \tag{4}$$

As shown in Figure 1(B), $k(a,t)$ is a continuous function of a, and monotonic mostly on $[0,\omega]$. It proves that there exists only one $A^* \in [0,\omega]$ at time t such that $k(A^*(t),t) = 0$, $k(a,t) < 0$ when $a < A^*(t)$, and $k(a,t) > 0$ when $a > A^*(t)$. (See Appendix for details about proof of the uniqueness of $A^*(t)$.)

Because $f(a,t) > 0$ for all $a \in [0,\omega]$, it follows from (4) that

$$\begin{aligned} g(a,t) &< 0 \quad \text{if } a < A^*(t), \\ g(a,t) &= 0 \quad \text{if } a = A^*(t), \text{ and} \\ g(a,t) &> 0 \quad \text{if } a > A^*(t). \end{aligned}$$

$A^*(t)$ thus works as a "threshold age" such that mortality improvement at ages younger than $A^*(t)$ can contribute to compression of mortality while that at ages older than $A^*(t)$ to expansion.

As mentioned above, the function $g(a,t)$ measures how much $e^\dagger$ will be changed by an equal reduction in mortality, and thus indicates the efficiency of mortality improvement on lifespan disparity. The magnitude of change in $e^\dagger$ due to mortality decline is obtained after taking into account the progress against mortality, that is,

$$G(a,t) = g(a,t)\rho(a,t), \tag{5}$$

where $\rho(a,t) = -\mathrm{d}\ln\mu(a,t)/\mathrm{d}t$ is the rate of progress of mortality improvement (Figure 1(C)). Hence, $G(a,t)$ measures the change in $e^\dagger$ resulting from the reduction in mortality at

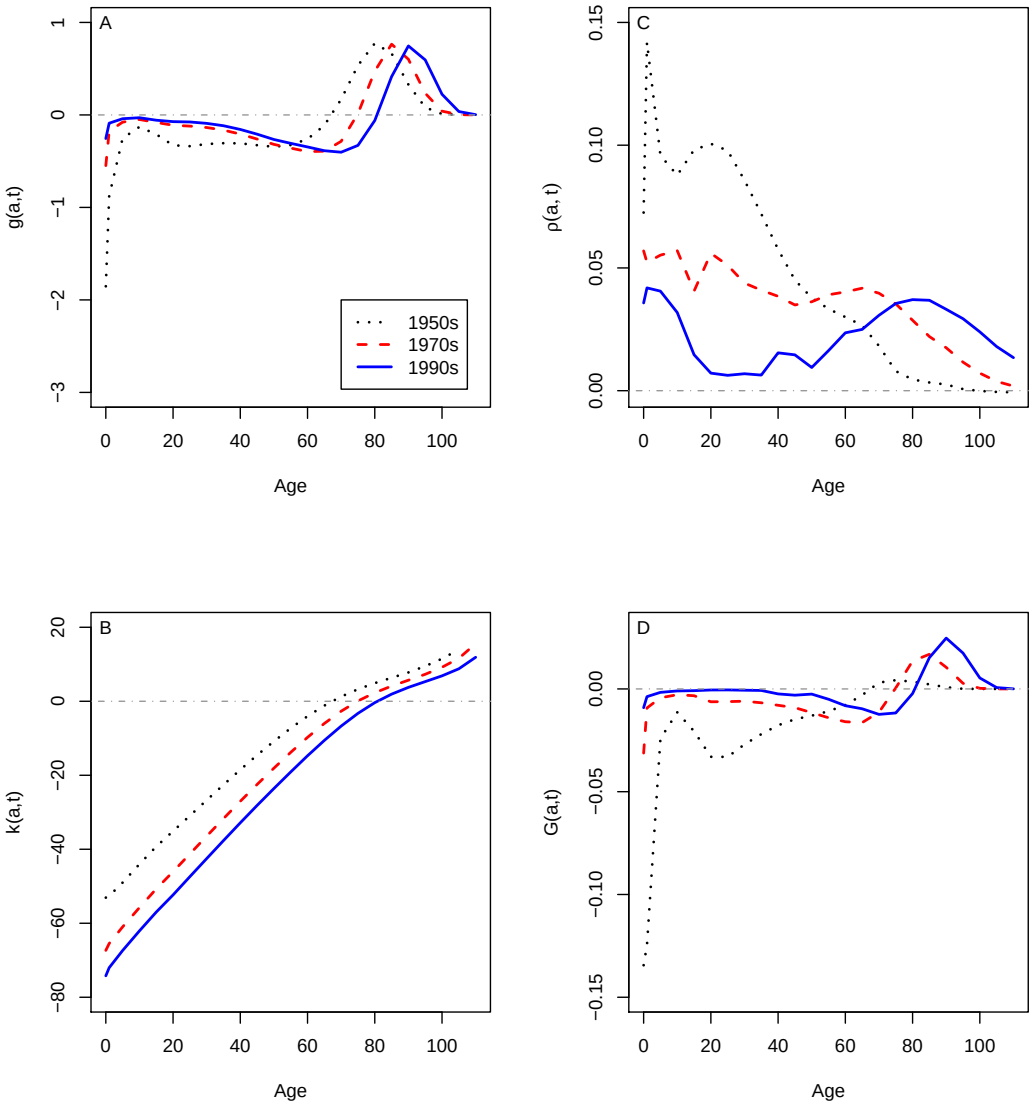

Figure 1: The functions $g(a,t)$ (A), $k(a,t)$ (B), $\rho(a,t)$ (C), and $G(a,t)$ (D) for Japanese females in 1950s, 1970s, and 1990s. *Source: Human Mortality Database 2007*

age a and time t, and its sum over age a amounts to the change in $e^\dagger$

$$\dot{e}^\dagger(t) = \mathrm{d}e^\dagger(t)/\mathrm{d}t = \int_0^\omega g(a,t)\rho(a,t)\mathrm{d}a = \int_0^\omega G(a,t)\mathrm{d}a, \tag{6}$$

Based on the threshold age A^*, $\dot{e}^\dagger$ can be decomposed into two components

$$\dot{e}^\dagger(t) = G_c(t) + G_e(t), \tag{7}$$

where,

$$G_c(t) = \int_0^{A^*(t)} G(a,t)\mathrm{d}a \text{ and } G_e(t) = \int_{A^*(t)}^\omega G(a,t)\mathrm{d}a \tag{8}$$

are *CDYA* and *EDOA* at time t, respectively.[2]

As discussed above, whether $\dot{e}^\dagger$ is below zero depends on which of G_c and G_e is greater. If $G_c < 0$ and $|G_c| > G_e$, then $\dot{e}^\dagger < 0$. This means the decrease in $e^\dagger$, indicating the occurrence of mortality compression. Instead, if $G_c \geq 0$ or $G_c < G_e$, then $\dot{e}^\dagger > 0$, indicating mortality expansion. In addition, if $G_c < 0$ and $|G_c| = G_e$, neither compression nor expansion will take place.

As mentioned above, the same change in $e^\dagger$ may result from different combinations of G_c and G_e. Consider the case of $\dot{e}^\dagger > 0$, meaning mortality expansion. A first combination is $|G_c| < G_e$ when $G_c < 0$. That is, the progress against mortality is more favorable to the older people. A second possibility is that both G_c and G_e are positive. In this case, the survival worsening happens to people younger than A^*, while the survival improvement happens to the elderly aged above A^*. The two combinations of G_c and G_e underlying the same $\dot{e}^\dagger > 0$ can give the inverse evaluation on mortality conditions.

Since A^* is determined by life table functions like $\ell(a,t)$, $f(a,t)$ and $\mu(a,t)$, the changes in such functions over time will change A^* too. As shown in Figure 1(B), as $k(a,t)$ shifts to the right, the point A^* at which $k(a,t)$ crosses the horizontal line moves to the right accordingly. The feature of A^* increasing over time can be illustrated in analysis relevant to age-specific contributions to mortality expansion, as in a previous study by Wilmoth and Horiuchi (1999).[3] More important, time-varying threshold age reminds us of the comparability of mortality compression in population subgroups. For example, assume that

[2]Note that $G_c(t) > 0$ does not mean that death rates at all ages before A^* increases. Instead, the relatively realistic situation is that the survivorship deteriorates at some ages, but improves at others. The sum of $G_c(a,t)$'s at the ages with survival worsening is greater than that at ages with survival improvement, so $G_c(t) > 0$. The same logic can be applied to negative $G_e(t) < 0$ although $G_e(t)$ is usually positive.

[3]Although the threshold age was not explicitly marked in that study, it still can be seen between the ages with negative contribution to change in *IQR* and those with positive contribution; Moreover, the threshold age increased over time (Tables 3 and 5 on pp. 484, *ibid.*). Unfortunately, the age ranges of 5 or even 20 years used in decomposition analysis are too big to delineate the trajectory of threshold age.

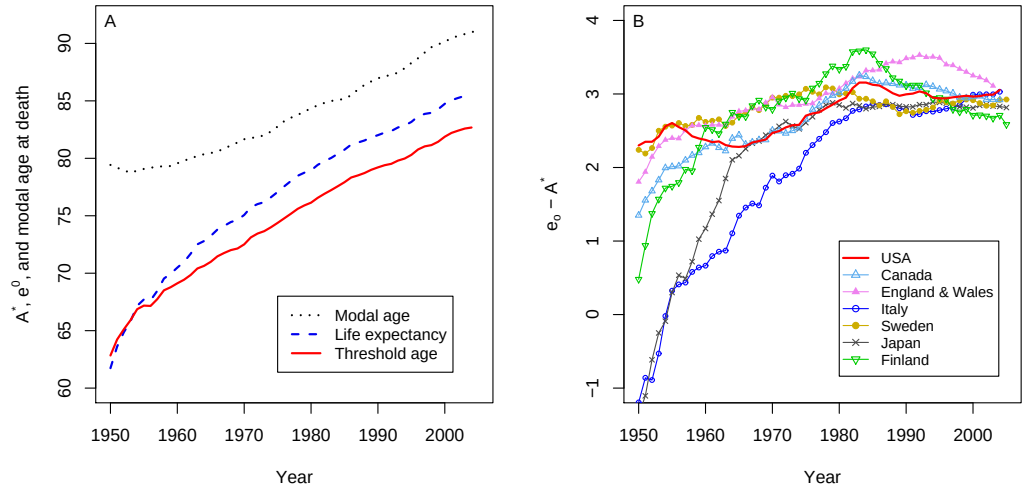

Figure 2: (A) The threshold age, life expectancy, and old-age mode for Japanese females, 1950-2005; (B) The gaps between life expectancy and threshold age in selected countries. *Source: Human Mortality Database 2007*

threshold age in a population has increased from 60 to 70 over the past fifty years. The rise of $e^{\dagger}$ in the subgroup aged 65+ that occurred fifty years ago can be totally attributed to *EDOA*, because all reductions in mortality after age 60 contributed to mortality expansion at that time. However, when threshold age goes up to 70, the change in $e^{\dagger}$ in the subgroup aged 65+ is mixed with *CDYA* at age 65-70 and *EDOA* at age 70+. In this sense, the subgroup aged 65+ fifty years ago is different from the current one. Usually, it is problematic to compare two such different groups in examine change in mortality expansion over time. For instance, the increase in $e^{\dagger}$ currently observed in the subgroup aged 65+ underestimates the magnitude of mortality expansion at older ages, because *CDYA* can offset *EDOA* in part.

Mean age, modal age, and threshold age

The widening gap between A^* and e^o in Figure 2(B) may seem puzzling. Suppose that deaths of a population follow a normal distribution that is symmetrical about the mean and mode. A decrease in the left half of such a distribution resulting from mortality decline will compress the deaths to the right. As the deaths are increasingly concentrated on the right half of the distribution, the mean will increase. However, the determinants of change in the mean are different from those in threshold. Any progress in reducing mortality can increase the mean length of life, whereas a change in threshold age is largely determined

by the distribution of such a progress over age. For instance, if mortality reductions largely benefit to the elderly (or the right half of the death distribution), threshold age may fall behind the mean age. In an extreme case in which the progress in reducing mortality is uniformly distributed over age, the differences among A^*, e^o and the mode will remain identical.

The death distribution of humans is bimodal so that these three ages are mostly different. Moreover, since the progress against mortality becomes greater among the elderly than that among the young people, threhold age becomes gradually lower than mean. Of these three age, threshold age is the smallest (Figure 2(A)). That means that reductions in death rates before mean or mode do not necessarily lead to mortality compression, because mortality decline occurring between the threshold age and mean or mode will contribute to mortality expansion.

The threshold age has generally been lower than life expectancy over the past fifty years, with a few exceptions in the early 1950s (Figure 2(B)). And the gaps between life expectancy and threshold age tend to converge at a roughly constant level of three years. Since threshold age is lower than mean age, mortality expansion is more likely to occur than mortality compression. When $A^* < e^o$, even if people fail to live as long as an average individual of the population, they can make a contribution to mortality expansion. On the contrary, if $A^* > e^o$, only people living longer than both e^o and A^* can contribute to *EDOA*. That is, *EDOA* is relatively easy in the case with a lower A^* than $e^\dagger$. On the other side, *CDYA* becomes increasingly difficult because the mortality at younger ages is already very low, and compared to that at older ages, is unlikely to further decline. Taken together, the two sides suggests that *EDOA* tends to catch up with and even exceeds *CDYA* in magnitude. As a result, the compression of mortality in the whole population may fade away, and further be substituted by the expansion.

Decomposition of mortality compression

Figures 3(A) and (B) graph the historical trends of $e^\dagger$ and $\dot{e}^\dagger$ for females in selected countries since 1950. First, $e^\dagger$ mostly converges across countries. In the 1950s $e^\dagger$ in Japan was the highest while that in Sweden was very low; but $e^\dagger$'s in the two countries have closed forty years later. Many other countries between the two countries in terms of $e^\dagger$ have converged by the 1990s. Second, $e^\dagger$ is inversely related to $\dot{e}^\dagger$, namely, the lower $e^\dagger$, the slower decline in $e^\dagger$, and vice versa. The country with a high $e^\dagger$ like Japan experienced a quick drop in $e^\dagger$, while some other countries with a low $e^\dagger$ like Sweden had a very modest decline in $e^\dagger$. Third, the inverse and the convergence of $e^\dagger$ implies that $\dot{e}^\dagger$ converges across countries too.

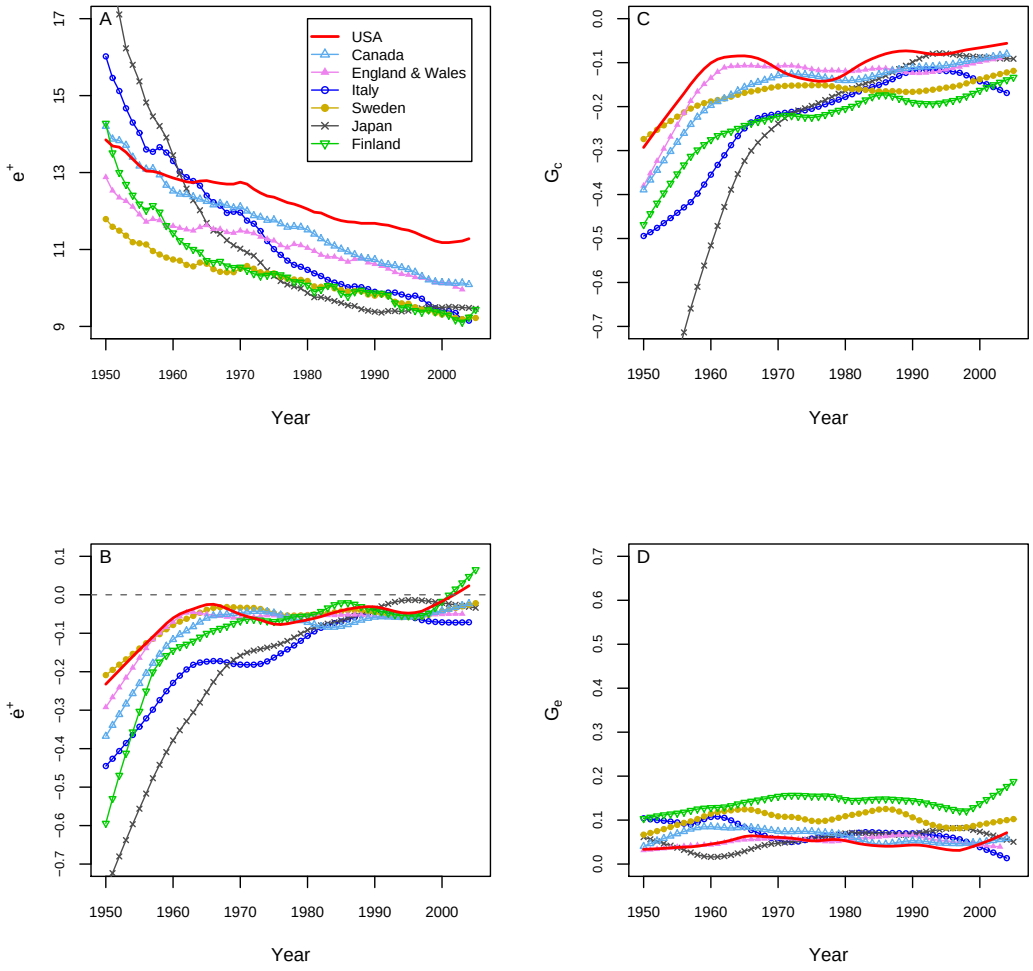

Figure 3: Lost life expectancy (A) and the rate of its change (B) with two components G_c (C) and G_e (D) in selected countries, females, 1950-2005. Except for $e^\dagger$, the rest three graphs are based on the smoothened line. *Source: Human Mortality Database 2007*

The trajectory of $\dot{e}^{\dagger}$ plotted in Figure 3(B) looks roughly like a mirror image of $e^{\dagger}$ in Figure 3(A), particularly during the period 1950-1990.

Of selected countries or broadly developed countries, the US is one of a few exceptions from the convergence of $e^{\dagger}$. In the 1950s the US stayed in the middle of selected countries in terms of $e^{\dagger}$, but became an outlier fifty years later because its $e^{\dagger}$ was as high as some other poor-practice countries like Ukraine. This is largely due to the slow rate of decline in $e^{\dagger}$ in the US. For example, the US had the similar $e^{\dagger}$ to Finland in 1950, but then fell behind because Finland had experienced a quick decline in $e^{\dagger}$. Some other countries also had slow decline in $e^{\dagger}$, but had a relatively low $e^{\dagger}$ at the beginning of the period 1950-2005, such as England & Wales. Then the decline in $e^{\dagger}$ in England & Wales remains nearly parallel with that in the US until now. Consequently, the US gradually stepped away from the convergence trend of $e^{\dagger}$ among developed countries.

The divergence in $\dot{e}^{\dagger}$ since the early the mid-1990s is remarkable, mainly characterized by the increasing of $\dot{e}^{\dagger}$ in the US and Finland. As we know, when $\dot{e}^{\dagger}$ is positive, its increase simply indicates an accelerating trend of increase in $e^{\dagger}$, meaning a quick expansion of mortality[4]. Since the historical experience of humans so far suggests the association between mortality expansion and survival worsening, which is often indicated by falling of life expectancy. However, life expectancy has increased in the two countries by now, and survival worsening is hard to account for mortality expansion.

With (7), the changes in $e^{\dagger}$ for selected countries are decomposed into two such components, G_c and G_e. To be comparable, the same scale is used for the vertical axis in Figures 3(B), (C) and (D). The trajectory of G_c almost resembles that of $\dot{e}^{\dagger}$, meaning that the decline in $e^{\dagger}$ is largely attributable to mortality compression resulting from mortality decline before A^*. Then the quick convergence in $\dot{e}^{\dagger}$ is also reflected by the trajectory of G_c. As shown in Figure 3(C), G_c in most countries had quickly converged by the mid-1980s.

G_c represents the contribution of survival improvements before the threshold age to the decline in $e^{\dagger}$: the lower $|G_c|$, the less contribution. Strikingly, except for the 1970s, the US has been the country with the least contribution of G_c to its decline in $e^{\dagger}$ almost throughout the whole period 1950-2005. By contrast, G_c in Finland has contributed the most to its decline in $e^{\dagger}$ since 1970s when all countries finished the quick fall of G_c. As death rates at younger ages keep decreasing, G_c usually declines. Hence, the gap in G_c between the US and Finland measures the room for further decline in $e^{\dagger}$ resulted from G_c.

Relatively, G_e appears much less variable than G_c. Apart from a few small fluctuations in Sweden and Italy, the noticeable change in G_e is the rise of G_e in the US and Finland very

[4]When $\dot{e}^{\dagger}$ is below zero, its increase (i.e., the decline in its absolute value) amounts to the slowing down of decline in $e^{\dagger}$.

recently. It is important to realized that a high G_e is not sufficient for mortality expansion in the whole. For instance, Finland's G_e has been the highest among selected countries, but mortality expansion appeared only in the late 1990s when G_e outweighed G_c. On the contrary, a small G_e may lead to expansion if it is relatively higher than G_c, as in the US. The US' G_e is smaller compared to Finland's, but higher than G_c; so even a modest increase in G_e increased $e^{\dagger}$, leading to mortality expansion.

The decomposition analysis above makes it evident that mortality expansion occurring in the US and Finland very recently is due to *EDOA* that outweighs *CDYA*, rather than a deterioration in survivorship. In particular, G_c in the two countries remains negative until today, meaning the continuing progress of reducing mortality at younger ages regardless of the speed of such a progress. That is, the expansion of mortality in the two countries results from the survival improvement, especially among the elderly people.

Discussion and Conclusion

The steady increment in life expectancy over the past 160 years is closely associated with mortality compression. This is mainly attributable to the change in the age pattern of survival improvement, as summarized in the theory of epidemiological transition (Omran, 1971; Olshansky and Ault, 1986; Horiuchi, 1997). By the mid-20th century, reductions in mortality had been considerable among children and adults at reproductive ages, but modest among the elderly (Horiuchi, 1997). Accordingly, mortality compression occurred because compression of mortality at younger ages was greater than the expansion of deaths among the elderly. For example, in Sweden, the compression of mortality was largely attributed to decreasing mortality among the very young (Wilmoth and Horiuchi, 1999).

Since the 1950s, however, many wealthy countries have gradually stepped into a new stage, which is characterized by reduction of mortality due to degenerative diseases. Mortality at advanced ages has declined considerably since the 1950s (Kannisto, 1994; Kannisto, 1996; Rau et al., 2005). The expansion of deaths among the elderly tends to be greater than before. On the other hand, the death rates at younger ages are already very low so that further compression of deaths among the younger people become increasingly difficult. Consequently, the compression of mortality in the whole population might gradually disappear. For instance, compression in the U.S. had been occurring by the 1960s, but disappeared since (Wilmoth and Horiuchi, 1999). And further, the latest data show that compression in the U.S. emerged again in the last decade. Besides the US, Finland also experienced mortality expansion in very recent years. Put another way, the lifespan disparity in the two countries has *increased*. Following the logic derived from the historical trends

of lifespan disparity of humans, the two countries would be regarded as having greater inequality of life chances. Is this correct? Maybe not. Considering that both Finland and the US enjoy relatively high life expectancy, which continues to increase, we should be cautious about interpreting the increase in lifespan disparity or mortality expansion.

Since the compression of mortality in the whole population is determined by the result of comparison between *CDYA* and *EDOA*, we develop a method to distinguish the two effects of mortality decline at different ages. It turns out that there exists a threshold age such that reductions in mortality before it will lead to mortality compression, whereas those after it to mortality expansion. Furthermore, based on the threshold, the change in mortality compression or lifespan disparity can be decomposed into two components. With the aid of such a decomposition, we find out that increases in lifespan disparity in the US and Finland are attributable to survival chances that have improved more for older people than for younger people.

The present study is mainly concerned with the change in lifespan disparity so far. However, considering the continuous improvement of survivorship of the elderly, it is interesting to further ask what will be happening in the future. For instance, will more and more countries join the list of countries with mortality expansion? – Because of the spreading of reductions in mortality of the elderly over the world. Will another compression follow after the mortality expansion? – Because of the spreading of knowledge about survival improvement from the oldest old to younger old. Indeed, these questions are as challenging as forecasting human lifespan.

Appendix

It is evident from (3) that the function $k(a,t)$ is continuous on $[0,\omega]$, as other functions in the right side of (3). Moreover, $k(a,t) < 0$ when $a = 0$ because $\ln \ell(0,t) = 0$ and then $k(0,t) = e^{\dagger}(0,t) - e(0,t)$ while $e^{\dagger}(0,t) < e(0,t)$. Moreover, $k(a,t) > 0$ when $a = \omega$ because both $e^{\dagger}(a,t)$ and $e(a,t)$ approach to 0^{+} when $a \to \omega$, but $\lim_{a\to\omega} \ln \ell(a,t) = -\infty$. According to the intermediate value theorem, there exists *at least* one $A^{*}(t) \in [0,\omega]$ at time t such that $k(A^{*}(t),t) = 0$.

If $e(a,t)$ is monotonic on $[0,\omega]$, so is $k(a,t)$. Further, if $k(a,t)$ is strictly monotonic on $[0,\omega]$, there will be *only* one $A^{*}(t)$. However, $e(a,t)$ in the population with very high infant mortality is not monotonic because it increase more or less at the very beginning, and goes down after reaching the peak at certain age (e.g., age three for Swedish females in 1900). Correspondingly, $k(a,t)$ falls slightly first and then turns up. Let $\underline{a}(t)$ be the age at which $k(a,t)$ reaches the minimum at time t, and $e(a,t)$ the maximum, then $k(0,t) > k(\underline{a}(t),t)$

and $e(0,t) < e(\underline{a}(t),t)$. It is evident that $e(a,t)$ is strictly increasing on $[\underline{a}(t),\omega]$, and so is $k(a,t)$. In practice, $k(0,t)$ is very close to $k(\underline{a}(t),t)$ that is usually far lower than zero. This suggests little possibility that there is another value $c \in [0,\underline{a}(t)]$ such that $k(c,t) = 0$. As infant mortality has dramatically dropped in the past decades, both $e(a,t)$ and $k(a,t)$ in many cases are monotonic. So A^* is generally unique with $k(A^*(t),t) = 0$, $k(a,t) < 0$ when $a < A^*(t)$, and $k(a,t) > 0$ when $a > A^*(t)$.

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