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The new trends in survival improvement require a revision of traditional gerontological concepts

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Abstract

In 1960, Strehler and Mildvan (SM) theoretically predicted that the parameters of the Gompertz approximation to a mortality curve are negatively correlated. This means that the changes in the human mortality rate resulting from improvement in living standards, progress in health care or the influence of other factors must follow certain regularities prescribed by dependence between the Gompertz parameters. Such dependence, called SM correlation, was then confirmed in a number of empirical studies using period data on human mortality. Since the SM theory was based on the cohort model of mortality, it was tacitly assumed that period and cohort SM correlation patterns are similar. The remarkable stability of the SM correlation pattern revealed in these studies was often regarded as manifestation of a universal demographic law regulating changes in the age pattern of mortality rates. In this paper, we investigated trends in mortality decline in France, Japan, Sweden and the United States. In contrast with traditional expectations, we found that the SM correlation pattern was relatively stable only in certain periods of a population's survival history. Recently, several new correlation patterns emerged and, despite some differences in the timing of the changes, the new patterns are remarkably similar in all four countries. Contrary to traditional expectations, the patterns are not the same for cohort and period mortality data when SM correlations are calculated for France, Sweden and the United States. We show that some changes in the patterns of SM correlation admit interpretation in terms of a biological mechanism of individual adaptation (survival trade off). Some other patterns, however, contradict basic postulates of the SM theory. This indicates the need for revision of traditional concepts establishing the relationship between physiological and demographic patterns of aging.

Keywords: Mortality rate; Strehler and Mildvan model; Biodemography of aging; Rectangularization

1. Introduction

In 1960, Science magazine published a paper by Strehler and Mildvan (1960) where a new theory of mortality and aging was suggested. The authors related the exponential increase in human mortality between the ages of 35 and 85 (the Gompertz law $\mu(x) = a\,e^{bx}$) with the linear decline of a vitality index $V(x) = V_0(1 - Bx)$ and parameters of environmental stresses (the frequency K and the average magnitude ϵD). Both kinds of dependence were observed in aging studies. Gompertz curve is a

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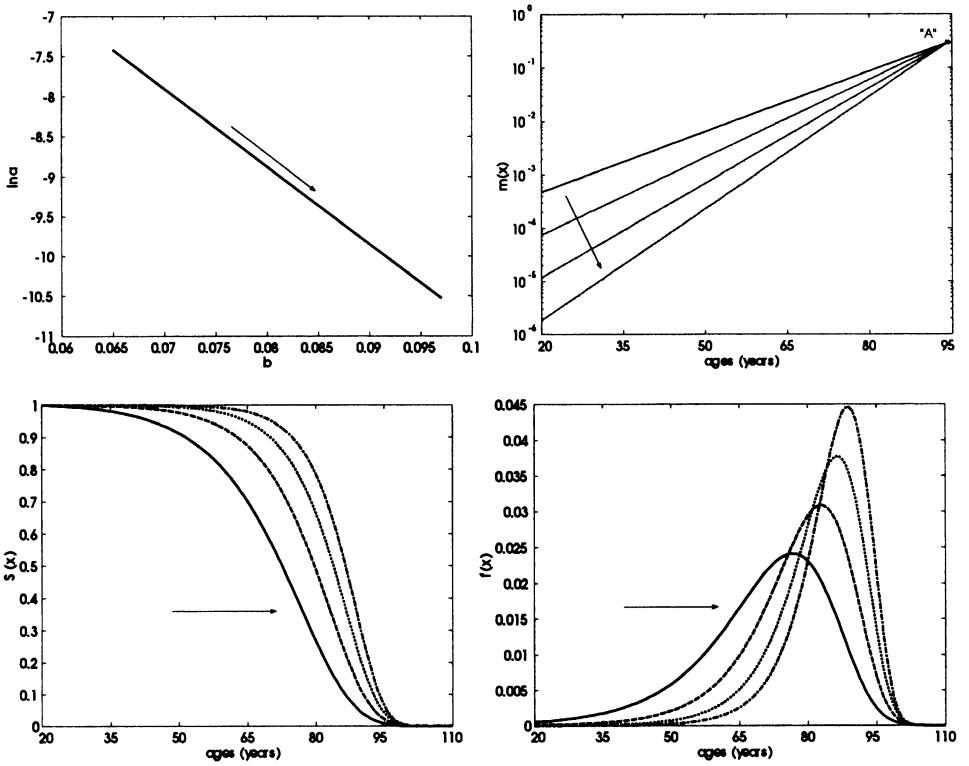

Fig. 1. The regular pattern of SM correlation (top left panel) and respective patterns of mortality decline (top right panel), survival improvement (bottom left panel), and of change in p.d.f. of the life span distribution (bottom right panel) corresponding to regular SM correlation pattern.

standard description of mortality between the ages 35–85 represented by demographic life tables (Pollard, 1991; Pollard and Valcovics, 1992). The mortality rate at old ages deviates from Gompertz curve. For humans, this rate is approximated by logistic (gamma-Gompertz) curve (Vaupel et al., 1998; Yashin et al., 1994). The decline of many important physiological functions was first shown to be nearly linear by Nathan Shock (1950) and Shock and Yiengst (1955, 1957). The curves describing age related changes in variables such as nerve conduction velocity, basal metabolic rate, cardiac output, kidney blood flow and maximum breathing capacity were shown to have a constant slope. These observations were confirmed in later studies of aging (Bafitis and Sargent, 1977; Sehl and Yates, 2001)), and stimulated several other approaches to modeling mortality and aging (Yashin et al., 2000). Note that the rate of decline in vitality index with age is characterized by two parameters V_0 and B, where V_0 also determines the initial value of vitality index. Such decomposition opens an avenue for genetic studies of aging using ideas of Strehler and Mildvan, since changes in V_0 may be associated with genetic or epigenetic effects.

1.1. Mortality rate in the Procrustean Bed of SM correlation

An important theoretical finding of Strehler and Mildvan (1960) was that Gompertz parameters are negatively correlated: $\ln a$ is a linear function of b.

$$\ln a = \ln K - \frac{1}{B} b \tag{1}$$

This result means that the evolution of the Gompertz part of the mortality curve must follow a certain regularity pattern prescribed by Eq. (1). Note that, although the rate of decline in vitality index is the product of V_0 and B, the Eq. (1) depends only on the parameter B. The typical pattern of SM correlation,

described by Eq. (1), corresponds to fixed parameters K and B. This pattern is shown in Fig. 1 (top left-hand panel). Such a correlation pattern was observed in a number of empirical studies.

One can see from Fig. 1 that, when the value of b increases, the value of $\ln a$ must decrease. In this case, mortality rate declines. This process is not uniform. The most significant log mortality decline occurs at young and adult ages (Fig. 1, the right top panel). When K and B are constants (as in the original Strehler and Mildvan model), the logarithms of all Gompertz-like mortality curves (with different parameters a and b) satisfying Eq. (1) must intersect at one point $(1/B, \ln K)$. When b increases, the line representing the logarithm of the mortality rate rotates counterclockwise around the point with coordinates $(1/B, \ln K)$. This is because, in the case of Gompertz mortality

$$\ln \mu(x) = \ln K + b\left(x - \frac{1}{B}\right) \tag{2}$$

In such a pattern of mortality changes, survival functions look more rectangular over time (Fig. 1, the bottom left-hand panel). Such a process is called the rectangularization of the survival curve. It yields the decline in the variance of the life span distribution (Fig. 1, the bottom right-hand panel). Note that vitality index $V(x^*) = 0$ when $x^* = 1/B$. So for ages larger than x^*, the SM model is not specified. The value $1/B$ is also the abscissa of the point where mortality rates intersect. If it is larger than the current life expectancy at birth, then the mean value and the median of the life span distribution tends to increase (Fig. 1, the bottom right-hand panel).

For several decades, following the Strehler and Mildvan (1960) publication, the pattern of SM correlation established for the total mortality rate was believed to be a kind of universal demographic law valid both for period and cohort mortality data. Since, for the wide range of ages, required cohort data on human mortality is limited, most SM analyses were performed using period (cross-seinctional) data. For the periodic data corresponding to the first several decades of the last century, the SM correlation pattern looked stable (Riggs, 1992; Riggs and Millecchia, 1992; Riggs et al., 1998; Prieto et al., 1996). This means that the intensity of stresses K reaching organisms and parameter K characterizing the rate of vitality decline were constants during this period. In this case, the rectangularization of the survival curve is the only pattern of survival improvement compatible with the SM theory. Fries (1980, 2000) emphasized the importance of such a pattern for prediction of the limits of mortality decline. The extended model of Strehler and Mildvan suggested by Khalavkin (1998) admits possibility of non-aging organisms. Michalski et al. (2000) used the Strehler and Mildvan model to explain the survival patterns observed in stress experiments with nematode worms *C. elegans*.

1.2. First signs of deviation of survival trend from rectangularization pattern

The signals about deviations of the trend in survival improvement from the traditional rectangularization pattern appeared in demographic literature about two decades ago and continued to be discussed in more recent publications. Myers and Manton (1984) found evidence that, in the second half of the 20th century, the tail of the survival curve in the United States tended to increase with years. Gavrilov and Gavrilova (1991) observed a tendency to derectangularization of the survival curve. Manton and Tolley (1991) discussed deviations from rectangularization trend in US data. Horiuchi and Wilmoth (1997, 1998) confirmed an increase in the tail of the life span distribution. Wilmoth and Horiuchi (1999) found that the decline in variability of the life span associated with the rectangularization pattern of changes in the survival curve ended in Sweden and in the United States around 1950. Kannisto (1994) and Jeune and Vaupel (1995) found that a steady decline in mortality at ages over 80 years started in many developed countries around 1950. Jeune and Kannisto (1997) observed that until this time, centenarians were quite rare and their number had been growing slowly. However, around 1950, their numbers began to increase considerably in the countries of Western Europe and North America. Wilmoth et al. (2000) found that an increase in the maximum life span in Sweden accelerated in the second half of the century. Robine (2001) discussed recent deviations from rectangularization trend in survival improvement in France. Lynch and Brown (2001) quantified the decompression of mortality in the United States between 1968 and 1992. Despite

these observations and numerous other data showing deviations of recent trends from the rectangularization of the survival curve, the new pattern of survival improvement was neither identified nor specified.

1.3. Signs of instability in SM correlation pattern

It is clear that if a deviation from rectangularization trend of survival improvement really took place in the last century, it would change the SM correlation pattern as well. According to the SM model, it could only happen if parameters K and B in Eq. (1) were not constant in certain periods of the survival history of populations. Hence, the basic postulates used in the derivation and implementation of the Strehler and Mildvan result (constant values of K and B) must be revised to take into account possible changes in K and B. Riggs (1994), Imaizumi (1996) and Lestienne (1988) discussed instability in SM correlation in the analysis of cause-specific mortality data for the United States. To avoid difficulties in the interpretation of the results of their studies, they introduced dependence between parameters K and B. However, the nature of this dependence was not discussed. Moreover, the value of the critical age $x^* = 1/B$ in the SM model describing cause specific mortality data was often smaller then the maximum age of the interval where the Gompertz approximation of the mortality rate is valid. The signs of instability can also be seen in the patterns of SM correlation for total mortality provided by Riggs et al. (1998) and by Prieto et al. (1996). However, to our knowledge, nobody has ever expressed concern about possible non-universality of SM correlation described by Eq. (1) for the total mortality rate in human populations. Gavrilov and Gavrilova (1991) modified the SM theory suggesting that, one first eliminates the background component of human mortality, and then calculates the SM correlation pattern for the remaining Gompertz part of the mortality curve. The authors claimed that, after such an elimination, the remaining Gompertz part of mortality curve is stable for the population of a given country, with different values of $\ln a$ and b for different countries. Our analysis reveals instability in SM correlation calculated with and without elimination of the background component of mortality. Here, we discuss the results of analysis performed in accordance with the traditional Strehler and Mildvan (1960) formulation. The regular pattern of SM correlation is supposed to characterize mortality rates in all countries between ages 35 and 80. In this paper, we analyzed mortality data in France, Japan, Sweden and the United States to check whether traditional patterns of SM correlation really take place for the large range of the mortality data.

2. Materials and methods

2.1. Mortality data

We use mortality data for males and females in France, Japan, Sweden and the United States. The data can be obtained from Wilmoth's web site at University of Berkeley, California, USA: http://demog.berkeley.edu/wilmoth/mortality/Sweden/notes.html. The data allow the analysis of period mortality data in France from 1899 till 1995, in Japan from 1950 till 1996, in Sweden from 1861 till 1999, and in the United States from 1890 till 1995. The data also contain information about cohort mortality for ages between 40 and 80 for 1859–1915 birth cohorts in France, 1821–1919 birth cohorts in Sweden, and 1900–1915 birth cohorts in the United States. The Japanese data series is too short to be used for cohort analysis.

2.2. Methods

We used demographic life table data to estimate survival functions for each year of available data in each country. Then, we visualized trends in survival improvement by putting all these survival functions in one graph and using different colors for different trends. Then, we estimated parameters a and b of the Gompertz period mortality rate for each year of available data in all four countries by fitting the curve $\ln \mu(x) = \ln a + bx$ to the logarithms of empirical period mortality in the age interval between 40 and 80, using the weighted least squares method. We put the respective estimates of $\ln a$ and b on the plane with coordinates $(b, \ln a)$ to evaluate the SM correlation pattern. To test the stability of the cohort SM correlation pattern and to compare it with the period one, we approximated cohort mortality rate in the age interval between 40 and 80 by the Gompertz curve $\mu(x) = a\,e^{bx}$. Using weighted least squares, we estimated

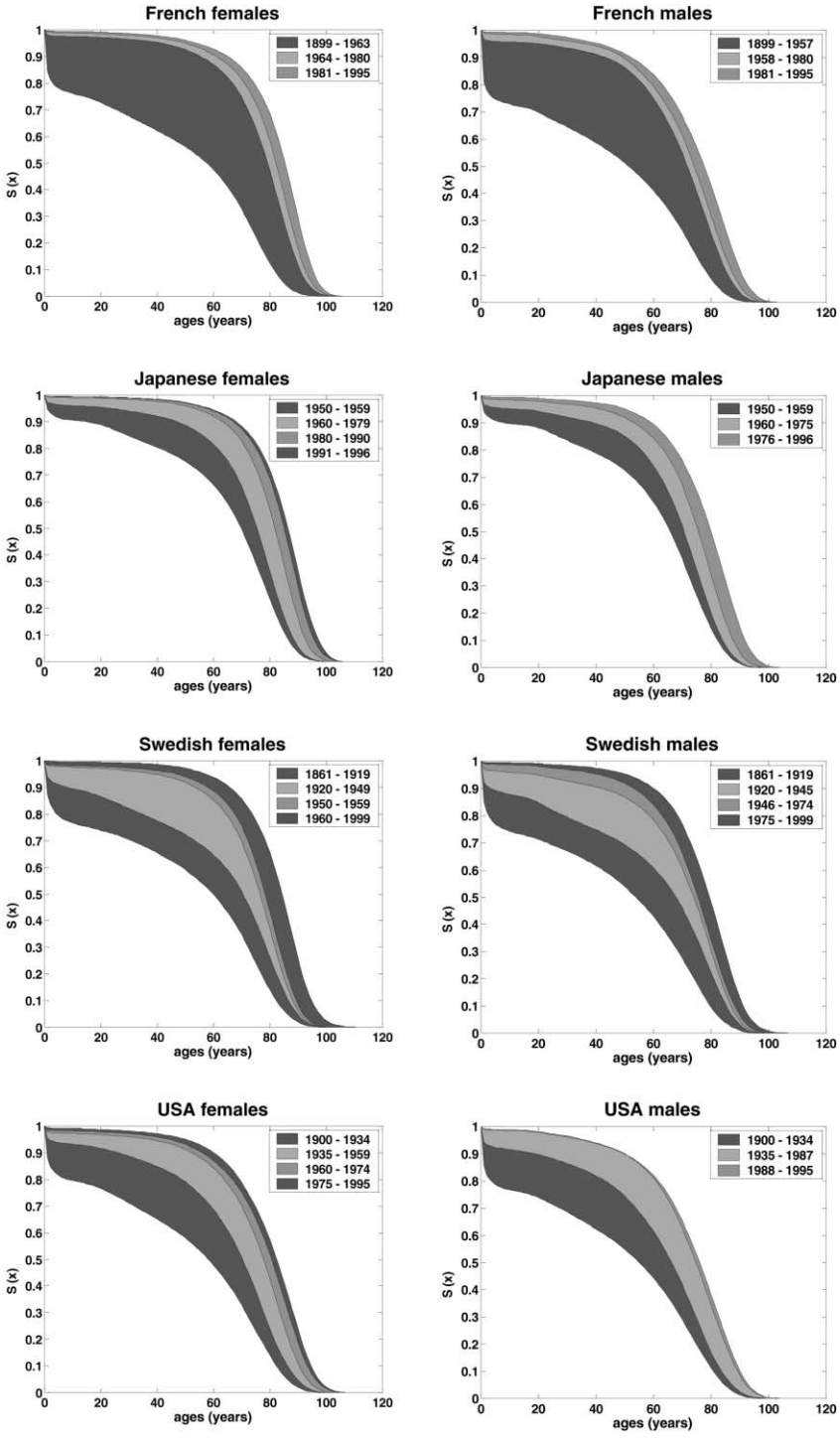

Fig. 2. Period patterns of survival improvement in France, Sweden, Japan and the United States.

values of $\ln a$ and b for the mortality data on each available cohort in France, Sweden and in the United States and put them on the plane with coordinates $(b, \ln a)$, to evaluate the cohort SM-correlation pattern.

3. Results

3.1. Changes in the age pattern of survival improvement

Fig. 2 shows the periodic patterns of survival improvement in France, Japan, Sweden and the United States for males and females.

One can see that, in France (both for males and females), changes in mortality in the period from 1899 to 1938 reveal an evident tendency to rectangularization of the survival curves (calculated from period mortality data), with almost no change in the tail of survival distribution. After 1938, except for the years associated with the World War II in Europe, this tendency was accompanied by an increase in the tail of the survival distribution. This trend was replaced by a near parallel shift of the survival curve to the right around 1981 and continued until 1995 (the end of available data). This shift increases the mean value of the life span (the life expectancy at birth). The variance of the life span decreases slightly, mostly because of the continuing mortality decline in young and adult ages. An increase in the tail of the life span distribution makes an additional contribution to an increase in empirical maximum life span, and to the growth of the proportion of centenarians discussed by Jeune and Vaupel (1995) and by Jeune and Kannisto (1997).

The data for Japan show typical rectangularization in the pattern of changes in the survival curve that occurred between 1950 and 1960 for both sexes. This is because the major demographic transition in Japan started about 1950 and continued with a higher rate than that in developed countries of Europe and North America (Kagava, 1978). After 1960, this tendency was accompanied by an increase in the tail of the survival distribution. This trend continued until 1979 for females and until 1975 for males. Around 1980, this pattern for females was replaced by a near parallel shift of the survival curve to the right, and this trend continued for about a decade until 1990. From 1991 until 1996, a slight tendency to derectangularization of the survival curve emerged for females. For Japanese males, this tendency continued for about two decades from 1976 until 1996 (the end of available data).

The pattern of changes in survival in Sweden (both for males and females), in the period from 1861 to 1919 combines rectangularization and an increase in the tail of life span distribution. From 1920 until 1949 for females and until 1945 for males, survival improvement followed the typical rectangularization pattern. Then for about a decade, between 1950 and 1960, the rectangularization trend for females was accompanied by an increase in the tail of the survival curve. For males, this process took place between 1946 and 1974. Then the parallel shift of the survival curve to the right with a slight tendency to derectangularization emerged for Swedish females. This trend continued from 1960 to 1999. For males, the parallel shift to the right with a slight tendency to rectangularization took place between 1975 and 1999 (the end of available data).

In the United States, for both males and females, the major change in the trend happened around 1935 when a tendency to rectangularization accompanied by an increase in the tail of the survival curve replaced the rectangularization pattern of survival improvement dominant since 1900. This trend continued until 1959 for females and until 1987 for males. Then this trend was replaced by a parallel shift of the survival curve to the right with a slight tendency to derectangularization. This trend extended from 1960 to 1974 for females and from 1988 to 1995 for males. Since 1974, the parallel shift of the female survival curve to the right was accompanied by a slight tendency to rectangularization. This trend continued until 1995 (the end of available data).

The similarity in the patterns of changes in developed countries indicates that their populations were probably exposed to similar conditions. These conditions may be associated with improvements in living standards and medication, an increase in the access to health care facilities and other beneficial aspects of industrial development, which can easily cross national borders. Cultural, climatic and genetic differences among the countries and their populations are likely to be responsible for the differences in survival trends.

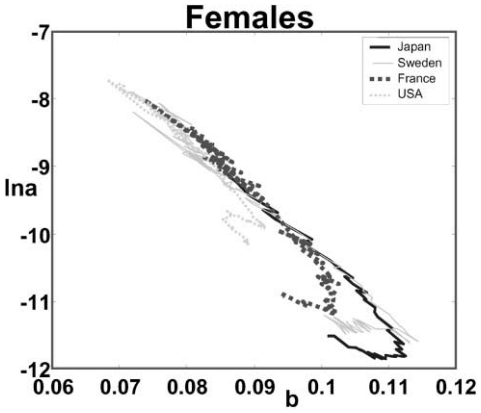

Fig. 3. Period patterns of SM correlation for females in France, Sweden, Japan and the United States.

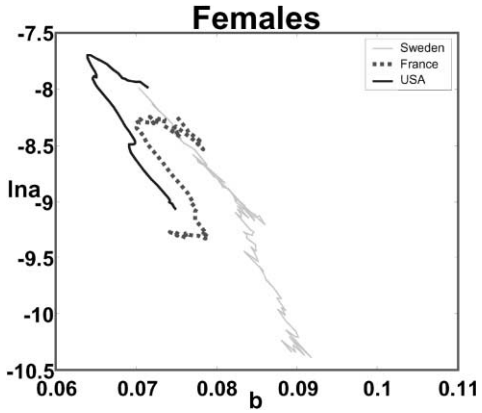

Fig. 5. Cohort patterns of SM correlation for females in France, Sweden, Japan and the United States.

3.2. Recent changes of the period SM correlation pattern

The period SM correlation patterns for female mortality data in France, Japan, Sweden and the United States are shown in Fig. 3.

One can see from this figure that, in all four countries, the SM correlation does not look like the regular pattern predicted by the Strehler and Mildvan theory (1960). The common part of the dependence between ln a and b for these countries can be approximated by the linear function. The majority of points on this line correspond to the mortality rates observed in these countries in the first half of the 20th century. Although in the second part of the century, each country followed its own pattern of dependence between ln a and b, these patterns certainly have one common feature. All these patterns have typical 'hooks' corresponding to recent changes in survival. For different countries, these hooks emerged in different places on the $(\ln a, b)$ plane. Only recent changes in female mortality in the United States followed the pattern of SM correlation, which is not represented in other countries. Fig. 4 shows the SM correlation pattern for male mortality in France, Japan, Sweden and the United States.

One can see the hook pattern of SM correlation similar to that of females in France, Japan and the

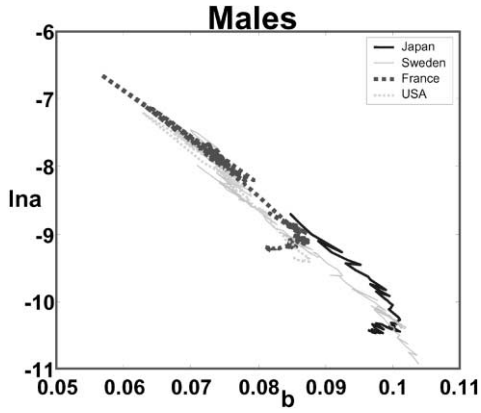

Fig. 4. Period patterns of SM correlation for males in France, Sweden, Japan and the United States.

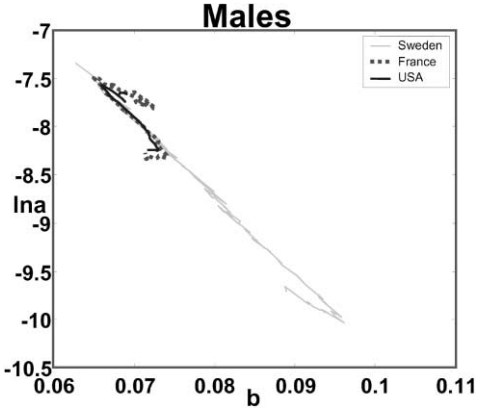

Fig. 6. Cohort patterns of SM correlation for males in France, Sweden, Japan and the United States.

United States. However, such a pattern did not emerge for Swedish males.

Since the SM model was derived to explain exponential increase in cohort mortality, it might be that the instability of the empirical SM correlation pattern is the result of the cross-sectional nature of the data. For this reason we decided to perform the SM analysis on cohort data.

3.3. Instability of the cohort SM correlation pattern

The patterns of cohort SM correlation for females in France, Sweden and the United States are shown in Fig. 5 and for males in Fig. 6.

One can see several distinct patterns of dependence between Gompertz parameters of mortality both for males and females. Swedish females show two distinct patterns of SM correlation. The first one characterizes mortality changes in the 1821–1874 birth cohorts. The second one describes changes in 1875–1919 birth cohorts. One can see that the slope of SM correlation corresponding to the second period of mortality improvement is higher than that in the first one. French females show three distinct patterns. The first and the third patterns are characterized by a relatively low slope of SM correlation. They characterize changes in mortality among the 1859–1884, 1885–1905, and 1906–1915 birth cohorts, respectively. The mortality rates in 1885–1905 follow a regular pattern of SM correlation.

Similar 'low slope' patterns of SM correlation took place for male mortality in the last available cohorts for the United States and France. The SM correlation for female mortality in the United States is characterized by two major patterns. The low slope pattern describes mortality changes in the 1860–1875 birth cohorts. The 1876–1915 birth cohorts follow a regular pattern of SM correlation. The male 1821–1907 birth cohort in Sweden follows a regular pattern of SM correlation. However, the slope of the SM correlation decreased for the 1908–1919 birth cohorts. This confirms that an improvement in cohort survival in these countries occurred in several phases.

3.4. The period and cohort data have different SM correlation patterns

The comparison of Figs. 3 and 4 with Figs. 5 and 6 shows that the period and cohort SM correlation patterns are not the same for both sexes. Since the original Strehler and Mildvan model establishes a relationship between physiological changes in individual organisms and cohort mortality the correct analysis must use data on cohort mortality. In the case of a stable pattern of cohort SM correlation, the respective parameters K and B can be interpreted as the intensity of stresses reaching organisms, and the rate of decline of their physiological capacity, respectively. We performed a simulation study and showed that the period and cohort patterns of SM correlation are similar if the changes in mortality rate are slow. However, fast changes in cohort mortality following a regular cohort SM correlation pattern produce irregular patterns of period of SM correlation and vice versa. This is because fast changes in Gompertz parameters of the cohort mortality rate may substantially disturb the shape of the period mortality rate. In this case, the approximation of such a period mortality rate by the Gompertz curve is not the best choice. Similar consideration with respect to cohort mortality and cohort SM correlation pattern is correct if fast changes happen in Gompertz parameters of period mortality. Therefore, stability of the SM correlation pattern for cohort data does not necessarily mean stability of such a pattern for period data and vice versa.

4. Discussion

4.1. The instability of SM correlation requires further extension of the SM theory

The deviation of the new SM correlation pattern from that described by Strehler and Mildvan means that parameters K and B in Eq. (1), which were relatively stable in the first part of the last century experienced changes in the second part. The analysis of these changes may shed some light on the nature of the individual aging and adaptation processes when the environmental situation changes. Some mortality changes (e.g. in Swedish females) show a steeper pattern of SM correlation for the group of latest birth cohorts. Such a situation, i.e. a switch from one correlation pattern to another may be explained by a change in the defense strategy of the population of individual organisms as a response to changes in environmental stress loading. The new (steeper) line

describing SM correlation may again, be well interpreted in terms of traditional SM theory. Unfortunately, not all new patterns have such simple interpretation. The patterns with a lower slope in the SM correlation line contradict the original postulates of the Strehler and Mildvan theory. For their explanation, continuous changes in all parameters of the SM model have to be assumed. For example, the slope of the pattern of SM correlation is lower for the later than for the earlier birth cohorts of Swedish males (Fig. 6). This change corresponds to an increase in the value of parameter B. With such a value, the vitality index $V(x)$ declines unrealistically fast and reaches zero value at young ages, i.e. before the Gompertz approximation to the mortality rate becomes valid.

4.2. Which forces may increase the slope of cohort SM correlation?

Since the description of cohort SM correlation patterns involves parameters associated with physiological and biological changes in the human organism during aging, as well as parameters characterizing environmental processes, it is natural to seek interpretation of observed changes in terms of interaction between processes in the human organism and the environment. Let us consider the change in the cohort SM correlation pattern for Swedish females as an example. This change is characterized by an increase in the slope of the SM correlation (i.e. by the decline in parameter B representing vitality). It is also accompanied by an increase in the frequency of stresses K.

The sharp increase in the frequency of stresses K, observed during transition from one phase of survival changes to another is difficult to interpret if the rate of vitality decline B does not always change in the opposite direction. These simultaneous changes of two, at first glance completely independent, parameters suggest an idea about the existence of some mechanism regulating the relationship between their values. The dependence between parameters K and B has also been observed in the SM analysis of cause specific mortality (Riggs, 1998). The biological interpretation of this phenomenon may be better understood if we recall that K is interpreted as an intensity of the events associated with energy demands for an organism to eliminate stress consequences. This intensity does not necessarily coincide with the intensity of environmental stresses K_0. One may assume a biological protective mechanism, capable of reducing the effective intensity of stresses that reach an organism.

A reduction of the energy used for protection allows an organism to save a certain amount of biological resources, which may be spent for its other functions. In particular, these resources may be spent on maintenance and repair of damages from internal stresses, such as oxidative stress, or on better functioning of the immune system. If the value of parameter B characterizing rate of decline in physiological capacities is determined by the amount of such resources then it will decline each time that the level of protection declines (i.e. when the value of parameter K increases), and vice versa. This is exactly what we observe in the case of the cohort pattern of SM correlation for females in Sweden. Our simulation showed that fast changes in mortality explain the patterns of SM correlation with a low slope.

4.3. The trade off for survival

The disposable soma theory of aging (Kirkwood, 1996) postulates the presence of a trade off mechanism allocating resources between reproduction and somatic maintenance and repair. The adaptation strategy used in such trade off works in the evolutionary scale. It may require many generations to approach maximum fitness, when environmental conditions become stabilized. The results of this paper admit an interpretation that the evolutionarily justified resource allocation between reproduction and somatic maintenance leaves room for adaptation in a smaller time scale: resources assigned for maintenance and repair of somatic cells must be used efficiently during an individual's life. The analysis of the 20th century changes in the cohort patterns of SM correlation in France, Sweden and the United States allows us to hypothesize a mechanism of adaptation, which modulates the organism's sensitivity to internal and external stresses. This is realized by the selection of the level of 'thinning' of the sequence of stress events characterizing different energy demands for an organism which yields respective changes in the rate of decline of physiological functions. During the thinning procedure some stress events become

neutralized, so the effective intensity of stresses K reaching an organism becomes smaller than the intensity of environmental stresses K_0. It means that parameter K, in the SM model may be represented as $K = K_0(1 - p)$, where K_0 is the intensity of environmental stresses, and p characterizes the efficiency of the protective mechanism. With probability p (which may be age and time dependent), the consequences of each stress-event arriving with intensity K_0 can be prevented or neutralized. A similar operation of thinning of the process of initial defects in the cells by biological protective mechanisms is described in the literature on cancer modeling (Yakovlev and Tsodikov, 1996). Thus, parameter K measures the frequency of stresses that reach an organism after passing through the system of biological and physiological defense.

At first glance, the procedure of elimination or neutralizing some stress events and leaving others unchanged (this is the idea of thinning of the flow of events) look strange and contradictory to the biological nature of the processes in the organism. The situation becomes more understandable if one takes into account the periodic (oscillatory) nature of many biological processes. Due of this, the protective properties of defense mechanisms may also oscillate. In this case, the thinning procedure is the natural outcome of such a defense. The frequency and magnitude of respective oscillations can vary to provide additional adaptation to stresses.

The adaptation to external stresses involves changes in the proportion of resources spent on protection from and elimination of the harmful effects of the internal stresses associated with oxidative damage to DNA or other bio-molecules, spontaneous mutations, etc. This involves DNA repair, elimination and removal of undesirable products of metabolism, etc. The amount of these resources determines the rate of decline in physiological capacities described by parameter B in the SM model. Although the models of such a trade off for biological organisms are not yet developed, the indications of its existence can be found in the biological literature. Recently, Jazwinski (2000) discussed two adaptation strategies in yeast aging. One, called the 'retrograde response', stimulates maintenance and repair of the cells in a response to improper mitochondria functioning. Another deals with the response of yeast cells to caloric restriction. It stimulates an increase of robustness in yeast cells.

5. Conclusions

Thus, in the second half of the last century, the rectangularization trend observed in France, Japan, Sweden and the United States was gradually replaced by a near parallel shift of the survival curve to the right. Neither cohort, nor period patterns of SM correlation were stable in these countries during the last century. The shape of the new correlation patterns differs from that established in Strehler and Mildvan's (1960) publication. The cohort pattern of SM correlation differs from that of the period pattern. This is an important observation since in most applications of the Strehler and Mildvan model to human data the SM correlation was calculated using period mortality curves. Some changes in the cohort pattern of SM correlation may be explained in terms of a biological mechanism of adaptation to stress working in individual organisms. Some others contradict basic assumptions of the SM theory. These results indicate the need to revise existing theoretical concepts of aging and mortality. New models are needed to explain non-standard patterns of SM correlation observed in the last century in developed countries. New concepts relating patterns of biological and physiological aging to the pattern of age specific mortality have to be developed. These concepts must relate manifestation of aging at the population level with the adaptive strategies associated with aging at the level of the individual organism in adult as well as in old ages.

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