

Decrease in Human Aging Rate Since the Middle of the 20th Century

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Abstract—The plot of the mortality rate minus the constant A of the Gompertz–Makeham equation and the plot of the mortality intensity increment $d(m)$ reflect the actual rate of biological aging. It was shown that since the middle of the 20th century there has been a slowdown in aging for all the countries of the world that were studied (for available periods in the history), in all parameters: R_0 and k coefficients of the Gompertz equation, mortality intensity increment $d(m)$ and maximum life span. The slowdown in the aging rate of continues to the present. The probable cause is a significant improvement in medical and social care and quality of life since the mid-20th century and the possible influence of the therapy of chronic diseases on the aging processes.

Keywords: aging, mortality, Gompertz equation, aging rate, retardation of aging

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A number of questions about the nature of aging and the interpretation of methods for its quantitative calculation still remain in the spotlight: the possibility of the existence of biological limits of lifespan [1, 2]; change in the aging rate in the history and its differences in different countries; change in the aging rate during life, including at the ages of long-livers; increase in the maximum life expectancy [3, 4] and others. Analysis of the age-related mortality has been the main method for studying aging [5] since the time of Gompertz research [6].

The aim of the study was to analyze changes in the aging rate using the age mortality for a number of world's countries in the history, mainly in the 20th century.

Although the Gompertz equation was originally derived purely empirically from the mortality statistics [6], now it can be developed theoretically. From the general definition as “aging is a decrease in the total viability with age” and the notion that this is a spontaneous, probabilistic process, we can consider the decrease in viability X with age as a process similar to the process of radioactive decay, when the rate of a decrease in the number of elements is proportional

their current number at the moment: $dX/dt = -kX$, where k is the proportionality coefficient. Accordingly, the number of remaining viable elements for the time t will be as follows:

$$X(t) = X_0 \exp(-kt) - A.$$

Hence, assuming that the total mortality m for a population is inversely proportional to the viability, i.e., $m = 1/X$, the well-known Gompertz–Makeham equation with generally accepted coefficients can be obtained:

$$m(t) = R_0 \exp(kt) + A.$$

To assess the rate of aging, one can use the following indicators: $m - A$ (the mortality intensity without background external component A , which is independent of aging) and the age-specific mortality coefficient k , which determines the rate of an increase in the aging-dependent mortality, as well as R_0 , which is thought to determine the initial level of the aging rate.

One can also use the mortality rate increment $d(m)$, which levels the constant A ; in this case, the indicator $d(m)$ better reflects the actual aging rate than $m - A$ because, in the latter case, the average value of A , which in reality can significantly vary in different age periods, is used. A decrease in the aging rate allows one to live to later ages, which allows using the maximum life span (MLS), the age of complete extinction for a standard cohort (100 000 people), for its estimation.

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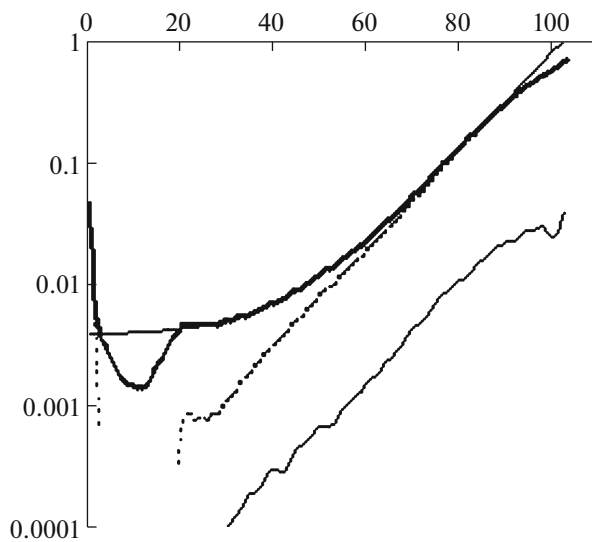


Fig. 1. Typical curve of the change in mortality intensity with age, France, 1930.

We conducted the study of the age-related mortality based on the data from the *Human Mortality Database* [7], covering the survival data of a standard cohort from 1741 to 2010 for 40 countries (with different available periods in the history). Changes in the total mortality rate m and its increment $d(m)$ for neighboring ages were plotted in a logarithmic scale at the ages of 1–110 years with 10-year intervals in the history, and indicators of the Gompertz–Makeham equation were calculated using known methods [8].

A typical Gompertz–Makeham plot is a straight line encompassing periods from the age of the end of growth and development (20–25 years) to the ages of long-livers (85–90 years) in a semilogarithmic scale. However, real plots usually show different shapes of the mortality intensity curve in the history, for different countries and at different age periods. This may be the result of the influence of either external conditions (which are reflected by the constant A) or a change in the aging rate, which is believed to reflect parameters of the exponent R_0 and, mainly, the coefficient k , as well as the mortality intensity increment $d(m)$, which tracks changes in the age-related intensity of the mortality, excluding the constant A in the calculations.

An example of a typical plot of the mortality intensity with a complex shape is shown below (Fig. 1). The curve calculated according to the Gompertz–Makeham equation is also shown there, which differs significantly from the real one at the young age and during the longevity period, but even in this case it is so far from the linearity (due to the influence of the A component).

At the same time, the use of the plots of the mortality intensity minus the external component of the mortality $m - A$, and the plots of the mortality inten-

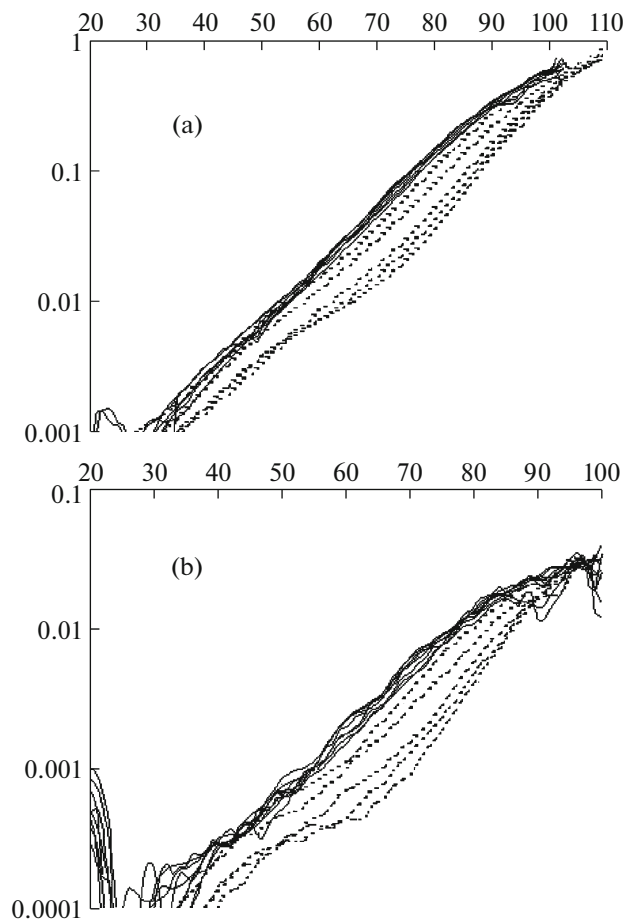


Fig. 2. Aging rate for a country in the history, France.

sity increment $d(m)$, show that the linear shape of the plot (in the semilogarithmic scale) is preserved from the period of the end of growth and development until the age of long-livers: the patterns of change in the rate of biological aging remain the same throughout the entire life cycle despite the pronounced changes in the plots of the total mortality, which are significantly affected by external factors.

The mortality intensity is along the vertical axis (logarithmic scale), the age of survivors is along the horizontal axis. From the top to the bottom: Gompertz–Makeham calculated curve (thin line), actual mortality rate (bold curve), biological component of the mortality rate, which reflects aging ($m - A$, dashed line), and the mortality intensity increment ($d(m)$, bottom line).

Using France as an example, for which there are well reliable data from the 1850s, it can be seen (Fig. 2) that the aging rate is the same in the country's history until the middle of the 20th century (Figs. 2a and 2b, solid lines of the plot). However, from the middle of the 20th century there has been a progressive sharp decrease in curves of the aging rate (Figs. 2a and 2b, dotted lines of the plot) for all ages.

The mortality intensity is along the vertical axis (logarithmic scale). The age of survivors is along the horizontal axis. a is the mortality without the background component $m - A$; b is the mortality intensity increment $d(m)$. For France, years: 1850–1870–1890–1900–1910–1920–1930–1940–1950–1970–1990–2000–2010. The years after 1950 are marked by dashed line.

Using 20 countries, for which the data since 1950 are available (Russia since 1960), as the example, it can be seen that the aging rate (the mortality intensity increment) sharply and constantly decreases as exemplified by the 75-year-old persons (Fig. 3).

The decrease in aging rate for the countries of the post-Soviet space (dashed lines), especially Russia, is slightly behind; a significant improvement in demographic indicators for Russia was noted only by 2014 [9].

The mortality intensity increment $d(m)$ is along the vertical axis (logarithmic scale). The age of survivors is along the horizontal axis. From the top to the bottom countries: Portugal, Finland, Japan, Hungary, New Zealand, Czech Republic, Switzerland, Estonia, Poland, Sweden, Italy, Australia, England, Spain, France, Latvia, Norway, Russia (Fig. 3, bold line), United States, Canada. Post-Soviet countries are indicated by dashed lines.

For 65-year-old persons for 12 countries, for which the data since 1900 are available (Belgium, Denmark, England, Finland, France, Iceland, Italy, the Netherlands, Norway, Scotland, Sweden, and Switzerland), the parameter $m - A$ decreased for 100 years by 2000 on average 2.79-fold (from 0.0313 ± 0.0070 to 0.0112 ± 0.0019 ; $P < 0.001$); similarly, the parameter $d(m)$ decreased on average 2.76-fold (from 0.00273 ± 0.00058 to 0.00099 ± 0.00026 ; $P < 0.001$). The decrease was the greater, the higher the initial level of aging in 1900 was: $r = -0.51$ for the parameter $d(m)$.

From the middle of the 20th century, the k term of the Gompertz–Makeham equation also decreases; it reflects an exponentially increasing intensity of the mortality with age, which is taken as the main characteristic of the aging rate: whereas, for 12 countries, it was 0.106 ± 0.008 in 1920, then after 50 years, by 1970 it was 0.095 ± 0.005 ($P < 0.001$): the decrease more than by 10%. The correlation of the k component with the current year, using the history of France as an example, for 1900–1940 is absent (determination coefficient $R^2 = 0.21$), whereas since 1950 the correlation becomes highly significant ($R^2 = 0.88$). At the beginning of the 21st century, the A component in many countries becomes negative, while the k component, which reflects the aging rate, decreases to the greatest extent.

Similarly, the coefficient R_0 , which reflects the basic level of aging, decreases, for 12 countries, from 1900 to 2000, 2.6-fold ($P < 0.02$).

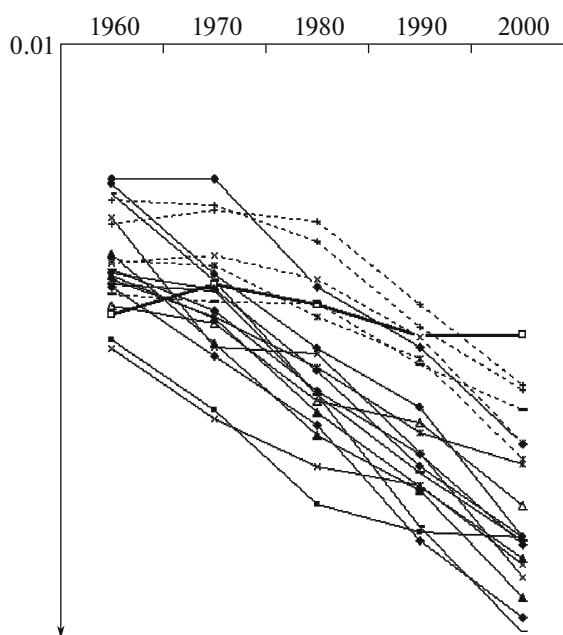


Fig. 3. Change in the aging rate of 75-year-old persons from the middle of the 20th century (20 countries).

During the same time, the indicator of external influences on the mortality intensity, the Makeham coefficient A , is reduced 24-fold.

The maximum life span, as the age of the complete extinction of the standard cohort, increases: for France, for example: from 105–106 years for 1840–1940 to 114 in 2010; and for 12 countries: from 104–109 years in 1900 to 111–113 years in 2000 (on average by 5 years: from 106.3 ± 1.5 years to 111.5 ± 0.7 years, $P < 0.001$).

Heredity can apparently contribute up to 25% to the life span and forms the phenomenon of long-livers [10, 11], affecting, however, only the final stages of life. The influence of external conditions on the aging rate as a whole is also quite probable [12, 13]. We proposed a look at aging, bringing together pathological changes during natural aging and under age-related diseases [14]: changes in the total viability in both processes are equivalent to the effect on biological aging. In this case, the prevention of age-related diseases and high level of medical and social assistance under high quality of life will affect the apparent rate of aging.

Thus, the use of several indicators of the aging rate for a number of countries of the world clearly indicates a decrease in the rate of biological aging from the middle of the 20th century. This is a very encouraging result, since so far the search for geroprotectors for humans has not yielded significant results [15], although in the experiment it is possible to restrain and even reverse aging of animals, while the effects on the regulatory vegetative centers give the most interesting results [16].

COMPLIANCE WITH ETHICAL STANDARDS

The authors declare that they have no conflict of interest. This article does not contain any studies involving animals or human participants performed by any of the authors.

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