Debate

Human Life Span Stopped Increasing: Why?

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Abstract. To account for the cessation of human life span increase in developed countries, we have studied the Swedish vital statistics over the period of 1901–1978. Approximating age-related mortality dynamics as the sum of the constant (age-independent mortality) and exponential (age-dependent mortality), we have discovered a striking phenomenon consisting in historical stability of age-dependent mortality. It appeared that decrease in total mortality was exclusively due to age-independent mortality which is close now to the limiting (zero) level. The results obtained prove the existence of the biological limit for the average life span and show that the conventional reserves for decrease in mortality have been exhausted. Thus, the problem of life prolongation requires a new way of thinking.

In recent years a paradoxical situation has been observed in all developed countries. Despite the obvious progress in medicine and public health service, the average life span stopped growing [13, 19, 23]. For example, the annual increase in Swedish males' life expectancy was 190 days in 1936–1945, but it became close to zero last decade, with the average life span being equal to 72.2 years (fig. 1) [21]. Moreover, an increase in mortality rate and decrease in life expectancy of adults are observed now in many developed countries (fig. 1) [4, 13, 21, 23]. Since this disturbing phenomenon of able-bodied people mortality increase has serious economic and social con-

sequences [15, 24], an explanation, quantitative description and forecasting of the historical dynamics of human mortality become the key problem of human ecology.

There are three extreme points of view in the explanation of the life span paradox. The first, most optimistic one may be called the hypothesis of temporary stagnation in medicine. According to this hypothesis, success achieved by public health service in its combat with infective diseases has resulted in more prominent mortality from other causes (heart diseases and neoplasms) which cannot so far be cured effectively [3]. Some authors believe, however, that further progress in

medicine will make it possible to attain an additional 50-year increase in life span by the beginning of the next century [16].

The hypothesis of ecological crisis is less optimistic. According to it, the cessation of increase in human life span has resulted from the disturbance of ecological equilibrium, environment pollution and the negative consequences of modern living (stress, hypodynamia, smoking, etc.) [3, 15].

The third hypothesis is that of a biological limit. In conformity with this hypothesis, the mean expectation of human life is already close to the limit determined by the biological constitution of *Homo sapiens* [12, 19].

Although each of these hypotheses is quite reasonable, none can be preferred and used for making a scientifically grounded quantitative forecast.

We found that the historical dynamics of human mortality rate can be easily accounted for, described quantitatively and reliably predicted on the basis of the most simple and general ideas on the nature of mortality. These ideas were originally formulated in 1825 by *Benjamin Gompertz* [9] and developed in 1860 by *William Makeham* [11].

According to the Gompertz-Makeham paradigm, the adult mortality rate from all causes may be approximated as the sum of two items: (1) age-independent mortality rate from causes unrelated to age (e.g., exogenic deaths from accidents, highly infective and lethal diseases); and (2) age-dependent mortality rate that grows with age following the geometrical progression law. This idea can be written as:

$$\mu(t) = -\frac{dN}{Ndt} = A + R_o \exp(\alpha\,t),$$

where $\mu(t)$ is mortality rate at age t, N is the number of surviving to age t, A is the Make-

ham parameter representing age-independent mortality rate (first item), $R_o \exp(\alpha t)$ is the Gompertz function representing age-dependent mortality rate which increases due to aging (second item); R_o and α are the Gompertz parameters.

The Gompertz-Makeham equation may be considered as the biological law of mortality since it holds not only for humans [7–9, 11] but also for many other species [7, 17, 18, 22] including horses, rats, mice and drosophila. Recently this equation has been theoretically grounded in terms of the reliability theory [1, 5, 7, 20]. Thus, the Gompertz-Makeham paradigm has now sufficient factual and theoretical support to be used as a tool for mortality study. The efficiency of such an approach has already been demonstrated [8].

Method

The Gompertz-Makeham parameters were estimated for 21 Swedish life tables in the age interval of 20–80 years by the least square method using our original nonlinear regression program in a EC-1022 computer. The theoretical equation was drawn by integrating the Gompertz-Makeham equation:

$$N_t/N_o = (N_{t_o}/N_o)\exp[-A(t-t_o) - \frac{R_o}{\alpha}(\exp\{\alpha\,t\} - \exp\{\alpha\,t_o\})],$$

where N_t/N_o is the probability of survival to age t, i.e. the hypothetical cohort number at age t (N_t) divided by its initial number (N_o).

Results and Discussion

To elucidate why the adult mortality rate has ceased to decrease in developed countries, we studied the historical dynamics of each of the Gompertz-Makeham parameters.

To that end, we investigated vital statistics of Swedish males over the period of 1901–1978. The Swedish statistics published in the form of 21 complete life tables was chosen for its extreme reliability and completeness. During the period under study the average life span of Swedish males increased from 54.5 to 72.2 years, with the cessation of growth being observed in recent years (fig. 1).

The Gompertz-Makeham parameters were estimated for each of these 21 Swedish life tables (table I). It appeared that the only parameter which has reliably changed during the period under study was that of Makeham (A). Figure 2 illustrates the biological interpretation of this phenomenon. It can be noted that the decrease in age-independent mortality rate (A) is the only cause for mortality decline. Age-dependent mortality rate, $R_o \exp(\alpha t)$, has hardly changed from 1901 to 1978. In 1956–1960, the age-independent mortality rate has dropped to the limiting (zero) level, hence this conventional reserve of the life span increase proved exhausted. It is in this period that the stabilization of adult mortality and life expectancy has taken place (fig. 1, 2).

Similar results were obtained using the vital statistics of other developed countries (data not shown). Thus, the average life span in developed countries is already close to the biological limit and cannot be increased significantly by conventional measures of medicine and public health service.

This conclusion is in accord with the results obtained from the analysis of the historical dynamics of mortality from selected causes [2, 14] and is shared by many investigators [2, 14, 19]. Our approach, however, has made it possible for the first time to determine the reserves and limits for life span increase solely on the basis of life tables,

Table I. Parameter values of the Gompertz-Makeham equation for Swedish male populations

Years	Parameters and uncertainties[*]		
	$A \cdot 10^3$ (year^{-1})	$R_o \cdot 10^6$ (year^{-1})	$\alpha \cdot 10^3$ (year^{-1})
1901–1910	5.57 ± 0.16	35.6 ± 6.5	100.5 ± 2.6
1911–1915	5.57 ± 0.14	26.0 ± 4.5	104.9 ± 2.5
1911–1920	6.96 ± 0.23	11.5 ± 4.3	115.7 ± 5.3
1921–1925	4.11 ± 0.14	22.6 ± 4.2	106.1 ± 2.7
1926–1930	3.76 ± 0.08	27.4 ± 2.5	103.8 ± 1.3
1931–1935	3.03 ± 0.08	28.5 ± 2.7	103.4 ± 1.4
1936–1940	2.46 ± 0.11	34.0 ± 4.1	101.5 ± 1.8
1941–1945	2.17 ± 0.12	24.8 ± 3.8	104.4 ± 2.2
1946–1950	1.21 ± 0.10	29.3 ± 3.4	102.7 ± 1.7
1951–1955	0.76 ± 0.08	28.7 ± 2.7	102.5 ± 1.3
1956–1960	0.63 ± 0.08	25.4 ± 2.7	104.2 ± 1.5
1961–1965	0.58 ± 0.09	24.0 ± 2.9	105.1 ± 1.7
1963–1967	0.62 ± 0.08	24.3 ± 2.5	104.8 ± 1.5
1966–1970	0.68 ± 0.07	24.4 ± 2.0	104.8 ± 1.2
1967–1971	0.62 ± 0.26	26.2 ± 8.5	103.8 ± 4.7
1968–1972	0.70 ± 0.05	24.9 ± 1.7	104.4 ± 1.0
1969–1973	0.68 ± 0.05	25.9 ± 1.7	103.9 ± 1.0
1970–1974	0.67 ± 0.04	26.7 ± 1.4	103.3 ± 0.8
1971–1975	0.70 ± 0.04	26.7 ± 1.3	103.4 ± 0.7
1972–1976	0.69 ± 0.04	27.0 ± 1.4	103.3 ± 0.8
1974–1978	0.68 ± 0.04	28.7 ± 1.5	102.3 ± 0.7

[*] The uncertainties correspond to 95% confidence intervals.

with the particular causes of death being unknown. That is why the method based on the Gompertz-Makeham paradigm can be used even when data on the causes of death are lacking (e.g., for developing countries and for earlier historical periods). Although attempts to analyze the total mortality have also been made previously [10, 17, 19], they yielded inaccurate results since the authors have ignored the Makeham parameter [19] and preferred a visual analysis of the graphs [10, 19] to tedious but precise calculations. Thereby,
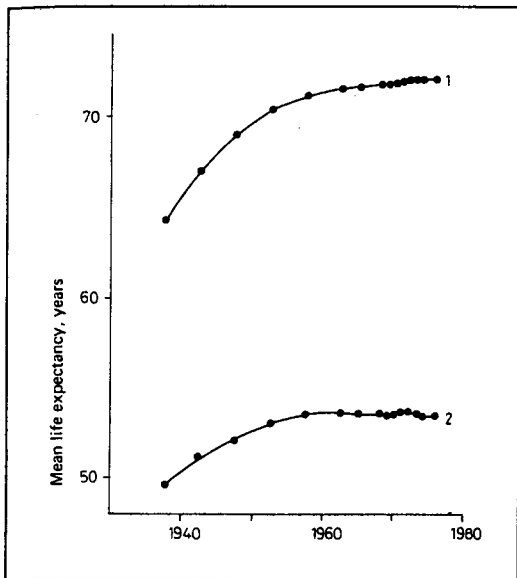
Fig. 1. Life expectancy of Swedish males at birth (1) and at age 20 (2) over the period of 1936–1978 (data from Swedish statistical abstracts [21]).
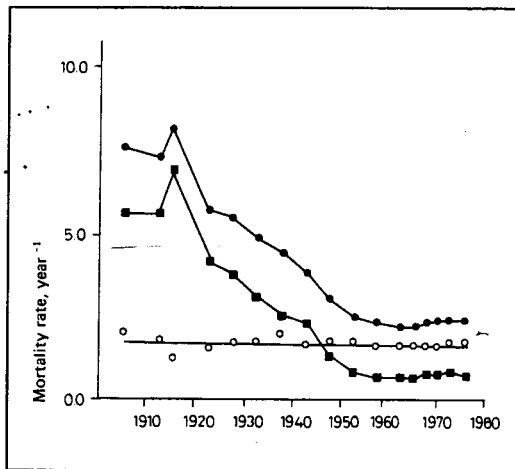

Fig. 2. Change in total mortality rate (●), age-independent mortality rate (■) and age-dependent mortality rate (○) for Swedish males over the period of 1901–1978. The values of mortality rates are multiplied by 10^3 and reflect the annual death number per 1,000 males at age 40. Mortality rates were taken or calculated from table I.

the erroneous conclusion as to the decrease in parameter R_o in the historical aspect has been drawn [17].

The data obtained in this work show that the mortality rate of adult Swedish males can be decreased only by an insignificant value of 0.007 $year^{-1}$ (table I), i.e. they actually prove that there is a biological limit for the average life span. Our preliminary calculations, however, reveal that the value of this limit, although historically stable, depends significantly on sex [6] and country [6, 7]. This gives us hope that one can discover factors determining the biological limit of human life span and find means to overcome it. The first step of this work is to draw the geographical map of biological limits of mortality. This work is in progress now.

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