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Biological Implications of the Weibull and Gompertz Models of Aging

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ABSTRACT: Gompertz and Weibull functions imply contrasting biological causes of demographic aging. The terms describing increasing mortality with age are multiplicative and additive, respectively, which could result from an increase in the vulnerability of individuals to extrinsic causes in the Gompertz model and the predominance of intrinsic causes at older ages in the Weibull model. Experiments that manipulate extrinsic mortality can distinguish these biological models. To facilitate analyses of experimental data, we defined a single index for the rate of aging (ω) for the Weibull and Gompertz functions. Each function described the increase in aging-related mortality in simulated ages at death reasonably well. However, in contrast to the Weibull ω_W, the Gompertz ω_G was sensitive to variation in the initial mortality rate independently of aging-related mortality. Comparisons between wild and captive populations appear to support the intrinsic-causes model for birds, but give mixed support for both models in mammals.

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Senescence (or aging) is a decline of physiological function with age. This decline is manifested in populations as an increase in mortality rate at older ages, which is often referred to as actuarial senescence (AS). In the absence of detailed studies on organism function, the increase in mortality rate with age has been used to compare the rate of aging in different populations and species of animals (1,2). AS also directly influences population growth potential and measures the strength of natural selection to postpone aging and its demographic consequences. Thus, increase in mortality rate with age has figured prominently in evolutionary studies of aging (3–6). When many populations are compared, it is most useful to describe the rate of aging by a single index for each population (7). This is usually accomplished by fitting a mathematical function to the relationship between rate of mortality and age or, alternatively, to the relationship between the proportion of individuals surviving and age. The coefficients of an aging model fitted to the data are used to describe the course of AS. Ideally, the rate of aging should be represented by a single index having units of 1/time (i.e., time^{1}). Many mathematical functions have been used to describe actuarial senescence (8,9). The most prominent of these are the Gompertz and Weibull equations.

The Gompertz and Weibull models differ in the way that early adult mortality and age-dependent mortality are related. Gerontologists ought to prefer the function that represents the underlying causes of increasing mortality with age most accurately (9,10). However, because both models are commonly used, it is also important to understand the relationship between the coefficients of the two functions (10,11). In this contribution, we distinguish essential properties of the two models, show how their coefficients are related, use simulated data sets to show the basic interchangeability of the two models and the circumstances under which they differ, and discuss some biological arguments for preferring one or the other function. Most of these points have been discussed in the literature; however, distinctions are often based on fine points of model fitting rather than the biological processes represented by the models. We argue that biological considerations should be paramount in distinguishing between models of aging, as they are likely to identify issues for future research.

Characteristics of Gompertz and Weibull Models of Aging

The Gompertz and Weibull models of AS differ primarily in the way in which age-independent and age-dependent components of mortality are related to each other. Both models ignore the typical decline in mortality that accompanies growth and development prior to maturity, although this component of mortality can be added to either model, as shown, for example, by Witten (12). Both models also incorporate a minimum mortality rate suffered by young adults prior to the onset of their physiological decline. This is usually referred to as the initial mortality rate (m_o). The models differ in the way in which mortality increases with age. In the Gompertz model, aging-related mortality increases exponentially as a multiple of the initial mortality m_o. In the Weibull model, the aging-related component of mortality is a power function of age that is added to the initial mortality rate. Thus, the initial mortality rate may be zero in the Weibull model, but it must be a positive number in the Gompertz model. Biologically, the Gompertz model implies that the increase in mortality rate with age represents increasing vulnerability to causes of mortality suffered by young adults. The exponential term of the Gompertz equation describes how rapidly this vulnerability increases with age. The vulnerability model assumes that the probability of death of each individual rises as its physiological function declines with age. In contrast, the Weibull model implies that causes of death of young adults and old individuals are different, independent, and additive. In addition, the Weibull model incorporates death that is due to catastrophic intrinsic causes whose probability increases with age. Thus, the Weibull function has been used in conjunction with failure-time models in which failure depends on the occurrence of one or more rare events, such as genetic mutations or cell deaths (10,13,14).

Mathematical characterization.—Instantaneous, or exponential, mortality rate (m) can range between 0 and infinity. In both the Gompertz and Weibull models, m increases continuously without limit. Some models of aging-related mortality, such as the logistic function (8,9), have upper mortality plateaus and thus better describe the leveling of mortality rate at old age observed in large cohorts of flies and humans (15–19). However, the deceleration of the mortality rate among the oldest old likely reflects, at least in part, heterogeneity in aging processes among

individuals (19–22) rather than a deceleration in the probability of death of a single individual. Regardless, we shall restrict this discussion to the nonasymptotic Gompertz and Weibull functions because of the practical consideration that small cohorts of individuals normally do not survive long enough to show marked deceleration of mortality rate. In addition, as we indicate below, in the Weibull model the rate of increase in the mortality rate slows with increasing age and thus can describe most survival data adequately.

The Gompertz function represents the increase in mortality rate (m) as a function of age (x) by the expression

$$m_x = m_o e^{\gamma x} \quad (1)$$

where m_o is the initial mortality rate experienced by young adults and γ (the Greek lowercase gamma) is the exponential rate of increase in mortality rate with age. Here m_o and m_x are instantaneous rates and are expressed in units of time^{-1}. Often γ is referred to as the Gompertz aging parameter and has units of time^{-1}. The product x is dimensionless, as it is an exponential. In addition, the relative, or exponential, rate of increase in mortality rate ($d \log m_x/dx$) under the Gompertz model is the constant γ. Thus, the acceleration of mortality rate is constant with increasing age. Finally, the mortality rate at a given age depends on the initial mortality rate, m_o, and on the exponential rate of increase in mortality with age, γ. The rate of aging under the Gompertz model is often expressed as the mortality rate doubling time (MRDT), where

$$\text{MRDT (time)} = \frac{\log_e 2}{\gamma} \quad (2)$$

(23,24). Note that MRDT in the Gompertz aging model also is independent of age.

According to the Weibull function,

$$m_x = m_o + \alpha x^{\beta} \quad (3)$$

the age-dependent component of mortality (αx^{β}) is added to the initial mortality rate, and its value at any particular age is independent of m_o. Here β is a dimensionless parameter, characterizing the shape of the curve relating mortality rate and age; determines the magnitude of the mortality rate at any given age for a particular value of β. Because the term αx^{β} has units of time^{-1}, the coefficient α has units of time$^{-(\beta+1)}$. Note that, in contrast to the Gompertz model, the relative rate of increase in mortality rate is age dependent, according to

$$\frac{d \log m_x}{dx} = \frac{\alpha\beta x^{\beta-1}}{m_o + \alpha x^{\beta}} \quad (4)$$

As x becomes large, particularly when m_o is small, $d \log m_x/dx$ approaches β/x, and the relative rate of increase in mortality becomes inversely related to age. This results in a deceleration of the increase in mortality rate with age.

One modification of the Gompertz model, the Gompertz– Makeham function, separates the initial mortality rate into components that remain constant and that increase with age (8). Thus,

$$m_x = m_o + ae^{\gamma x} \quad (5)$$

However, the added parameter does not avoid coupling aging-related mortality to a component of the mortality suffered by young adults. In addition, we have found that nonlinear curve fitting does not estimate the partitioned initial mortality components m_o and a efficiently for small samples, and the calculated aging parameter for the Gompertz– Makeham model is extremely variable. The additional parameter in principle allows for a closer fit of the model to data. However, even with one fewer parameter the Gompertz function often fits small samples better than the Weibull function (see Results) and so it is not disfavored in this regard. Additionally, our concern in this paper is not the precision of the fit but the meaning of the parameters. Thus, the Gompertz– Makeham model will not be considered further here.

The rate of aging in Gompertz and Weibull models.— The increase in mortality rate over the initial adult mortality rate measures the aging-related decrease in physiological function in demographic terms. Thus, an index to the rate of aging should bear some relationship to the rate of mortality at a particular age and have the same units as mortality, that is, time^{-1}. The Gompertz has units of time^{-1} but does not by itself indicate the magnitude of the mortality rate at a particular age. By analogy to the Weibull function, in which initial and aging-related components of mortality add, the aging-related component in the Gompertz function is $m_x - m_o = m_o(e^{\gamma x} - 1)$. This cannot be expressed simply in terms of a single

parameter, and so it is necessary to calculate an index incorporating both m_o and γ. We refer to such an index to rate of aging as ω (Greek lowercase omega). For the Gompertz model, a suitable expression for the rate of aging is

$$\omega_G = \sqrt{m_o \gamma}, \quad (6)$$

which has units of time^{-1}. This is the square root of the slope of the relationship between mortality rate and age at age 0.

For the Weibull function, α and β may be combined to give a single index to rate of aging, with units time^{-1}, by the expression

$$\omega_w = \alpha^{1/(\beta + 1)} \quad (7)$$

(2). Although different combinations of and can give the same value of ω_w, is often close to 3 in natural and captive populations and the value of ω_w also is relatively insensitive to variation in the value of used to fit a particular data set (25). Regardless of the value of α or β, all curves of $m_x = \alpha x^\beta$ with the same value of ω_w cross at a value of $m_x = \omega_w$ when $x = 1/\omega_w$ (25). Put another way, any Weibull function with parameters α and β has a value ω_w such that the curve of x versus x reaches a value of ω_w at age $x = 1/\omega_w$. Thus, the single index ω_w provides an estimate of the rate of aging that is independent of the extrinsic mortality and that may be compared among populations and species.

Fitting Data With Gompertz and Weibull Aging Models

The most direct approach to estimating the parameters of Gompertz and Weibull models is to fit Equations (1) and (3) to the relationships between mortality rate and age. The primary data for this purpose are the ages at death of individuals in a cohort, which can be grouped by age to calculate the proportion of individuals in a cohort dying during a particular age interval. Individual ages at death are independently sampled from the population distribution and therefore satisfy the assumption of independence for statistical inference (unless, for example, contagious diseases were prevalent in the population under study).

Ages at death also can be converted to a survivorship curve for a population, which portrays the individuals alive at age x as a proportion of those in the population alive at age 0. Survivorship (l_x) is related to the age-specific mortality rate by

$$l_x = \exp\left(-\int_{i=0}^{x} m_i di\right). \quad (8)$$

Thus, for the Gompertz function,

$$l_x = \exp\left[-\frac{m_o}{\gamma}(e^{\gamma x} - 1)\right] \quad (9)$$

and for the Weibull function

$$l_x = \exp\left(-m_o x - \frac{\alpha x^{\beta+1}}{\beta + 1}\right). \quad (10)$$

The parameters for both functions may be obtained by nonlinear curve fitting. We use the logarithmic form of the relationship between l_x and age; that is,

$$\log l_x = -\frac{m_o}{\gamma}(e^{\gamma x} - 1) \text{ for Gompertz} \quad (11)$$

and

$$\log l_x = -m_o x - \frac{\alpha x^{\beta + 1}}{\beta + 1} \text{ for Weibull.} \quad (12)$$

A disadvantage to estimating parameters from survivorship is that values of l_x are not independent and goodness-of-fit statistics must be obtained by bootstrapping or by Monte Carlo simulations. An advantage to using the survivorship function rather than the mortality rate is that parameters may be estimated for very small samples. Samples of 100 individuals were chosen for simulation to resemble typical samples in studies of natural and captive populations. For such small samples, maximum likelihood estimates of parameters rarely converge (2). Details of curve fitting and confidence limits for estimated parameters will be provided elsewhere.

Here we use simulated data to explore the relationship between parameters of Gompertz and Weibull equations fitted to data generated by both models. In general, both equations performed well in recovering the parameters used to generate the data. Curve fitting leads to no clear preference of one model over the other. Parameters ω_W and ω_G of the two models are correlated when fitted to the same sets of data as long as the aging parameters γ or α and the initial mortality rate m_o are also correlated with each other. However, when aging parameters and the initial mortality rate vary independently, the Weibull function provides a more stable estimate of aging-related mortality. When agingrelated mortality and initial mortality have different causes, the Weibull model also provides a distinct advantage for comparative analyses in having a single index (ω_W) that describes only the aging-related component of mortality.

METHODS

Ricklefs (26) fitted Weibull models to mortality rates or survival curves of 40 species of birds. He found that m_o ranged from 0.007 (for the Blue-and-Yellow Macaw *Ara macao* in a captive population) to 0.989 (for Great Tit *Parus major* males in a natural population). The aging index ω_W varied from 0.038 (for a natural population of the Wandering Albatross *Diomedea exulans*) to 0.279 (for male Arabian Babblers *Turdoides squamiceps* in a natural population). The initial mortality rate and the rate of aging were strongly positively correlated (2). For data simulations, we chose four different parameter combinations from the regression line relating ω_W to m_o. These parameters characterize four hypothetical bird species that lie at roughly equal intervals along the short-lived/long-lived continuum (Table 1). Values of m_o and γ were obtained for the Gompertz function by fitting Gompertz equations to data for four representative species of birds. Thus, the parameter values used to generate survival data for the Gompertz and Weibull models are similar but not exactly comparable. However, we only compare different model fits to the same data set and do not compare the data generated by the different models, and so comparability is not a problem in these analyses.

For both the Gompertz and Weibull models, we generated 100 data sets comprising 100 individual ages at death for each combination of parameter values. One hundred individuals were chosen as representative of the size of many studies. Time units were years. For each simulation, the time scale was divided into 0.1-year intervals, and the probability of death during the interval was calculated from the model as the difference in the expected survival to the beginning and end of each time interval. Then, for each individual in the simulation a random number from a uniform distribution between 0 and 1 was generated for each age interval beginning at age 0. The age at death for each individual was the midpoint of the first age interval in which its random number was less than the probability of mortality during that interval. Ages at death for all 100 individuals in each simulation were arranged to produce a survival curve, which was then fitted by both Weibull and Gompertz equations. This was repeated for each set of parameter values. All calculations were performed by using the Statistical Analysis System (SAS, Release 6.12, SAS Institute, Cary, NC) and curve fitting was accomplished by the nonlinear regression procedure. Aging indices (ω) were calculated from the parameter estimates.

To investigate further the ability of the Gompertz and Weibull models to estimate the aging-related component of mortality when initial mortality m_o varies, we generated three data sets using Weibull models, each having the same value for the rate of aging ($\omega_W = 0.0813$) but with a different value of m_o. The difference between these models may be compared with the difference between populations in nature and in captivity (or in any contrasting environments with respect to extrinsic mortality factors) when the rate of aging is an intrinsic characteristic of the species uninfluenced by external conditions.

RESULTS

Comparison of Recoveries of Initial Mortality (m_o) by Gompertz and Weibull Models

The results of this exercise clearly show that both models recover values for initial mortality reasonably well regardless of whether the data were generated by Gompertz or Weibull equations (Figures 1A and 1B). However, the Weibull equation has a tendency to greatly underestimate m_o in a small proportion of cases, whereas Gompertz estimates are tightly clustered. This is because mortality rates at all ages contribute to the estimation of m_o by the Gompertz equation, whereas only mortality rates at the youngest ages weigh heavily in this estimate for the Weibull equation. Moreover, the Weibull equation tends to estimate m_o rather poorly in data sets generated by the Gompertz model, and the Gompertz equation tends to underestimate m_o for data generated by the Weibull model, especially when values of m_o are low (see Table 2). Values of m_o estimated by fitting the two equations appear to be uncorrelated within each data set, except for the parameter set with the highest value of m_o (correlations not shown).

Table 1. Four Combinations of Aging Parameters Serving as Models for Our Simulations of Aging Data

Parameter Set	Weibull		Gompertz			
	m_o	ω	m_o	γ	ω	Typical Bird Spec.
A	0.3	0.1893	0.2204	0.1890	0.2041	Red-billed Leiothrix
B	0.1	0.1265	0.0605	0.1804	0.1045	Cockatiel
C	0.03	0.0813	0.0186	0.1500	0.0528	Scarlet Ibis
D	0.01	0.0543	0.0086	0.1100	0.0308	Greater Flamingo

Comparison of Estimates of the Rate of Aging Index

Estimates of rate of aging index showed less variation than estimates for m_o and were significantly correlated between fits produced by the two models, especially for scenarios with lower rates of aging (see Figures 2A and 2B; Table 3). Both equations also estimated the rate of aging without bias when data were generated by the same model (see Table 4). Because the Gompertz and Weibull rates of aging are not comparable, we cannot decide whether either is biased when one equation is used to fit data produced by the other model. However, rates of aging estimated by the Gompertz equation tended to be less variable than those estimated by the Weibull equation when the rate of aging was high.

Retrieving Parameters From Data Sets Generated by a Weibull Model Using a Constant Rate of Aging but Different Values of m_o

The Weibull equation retrieved essentially the same value for ω_w for all three data sets, whereas the Gompertz model yielded different estimates for the rate of aging depending on m_o (see Figure 3). Because ω_G is calculated from the product of m_o and γ, reducing the value of m_o tends to reduce the value of ω_G. Evidently, the increase in γ necessary to fit the unchanged aging-related component of mortality is not sufficient to offset this. Thus, ω_G is sensitive to the value of m_o and is therefore not a robust measure of the rate of aging. This does not necessarily mean that the Gompertz equation cannot fit the data adequately, but rather that the index ω_G cannot be used in comparisons among data sets when aging-related mortality has intrinsic causes that are nonexistent at age 0. Alternative indices for the Gompertz model to characterize aging-dependent mortality resulting from intrinsic causes have not come to mind.

DISCUSSION

How Well Does Each Model Retrieve the Input Parameters?

One objective of our simulations was to determine how well the Gompertz and Weibull models fit the same data sets. We found that both equations could be fitted reasonably well to simulated data, regardless of which model was used to generate the data. Thus, the two models of actuarial aging are roughly equivalent in their ability to characterize aging-related mortality. We also found that variation in the Gompertz estimates for a given simulation was generally lower than that for Weibull estimates, regardless of whether the curves were generated from Gompertz or Weibull models. Gompertz models yielded better retrieval of input m_o, especially when the input values were low. The Gompertz equation also yielded less variable estimates of ω when initial mortality m_o was high, but not otherwise. Thus, it would appear that the Gompertz equation provides somewhat more consistent parameter estimates for a particular sample of ages at death, although both equations appear to produce unbiased estimates of parameter values under a variety of parameter values. Additional simulations (not shown) indicate that parameter estimates vary less as cohort (sample) size increases, to the point that differences in the quality of the fits for each

equation largely disappear for samples of 1000 or more.

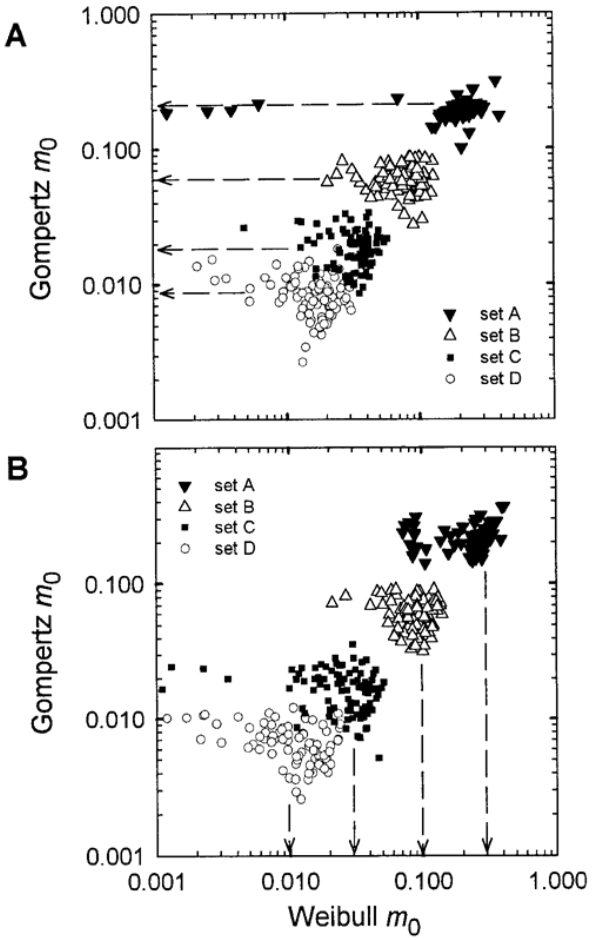

Figure 1. The relationship between Gompertz and Weibull estimates of initial mortality (m_o) when the respective models were used to fit data sets generated by **A** a Gompertz model or **B** a Weibull model. Combinations of parameters characterizing four different parameter sets (A–D; see Table 1) were used. For each species 100 simulations were performed for a cohort size of 100 individuals. Arrows indicate parameter values used in simulations.

The Weibull function tends to give more consistent estimates of the rate of aging for data in which the rate of aging is low. Because the rate of aging decreases more slowly than initial mortality in natural populations of birds, a larger proportion of individuals die of intrinsic causes in species with low rates of aging (2). Thus, the better performance of the Weibull model under these conditions argues in favor of its use for studying actuarial senescence in long-lived organisms.

Differences Between Gompertz and Weibull Models of Aging

The most important difference between the Gompertz and Weibull models is that the increase in mortality that results from senescence is a multiple of the initial mortality

Table 2. Normalized Bias and *SD*s of Initial Mortality Estimated by Gompertz and Weibull Functions

	Gompertz Function						Weibull Function					
	Gompertz Fit			Weibull Fit			Gompertz Fit			Weibull Fit		
Parameter Set	Bias	*SD*	NC	Bias	*SD*	NC	Bias	*SD*	NC	Bias	*SD*	NC
A	-0.12	0.12		-0.42	0.17	12	-0.27	0.15	6	-0.14	0.25	12
B	-0.02	0.22		-0.21	0.24		-0.40	0.15		-0.06	0.25	
C	+0.03	0.33		+0.12	0.33	5	-0.42	0.20		-0.03	0.35	7
D	+0.05	0.35	1	+0.66	0.67	8	-0.25	0.27		+0.20	0.59	7

Notes: Gompertz and Weibull functions are fitted to 100 data sets generated by Gompertz and Weibull models, using parameter sets A–D. *SD* standard deviation; NC number of cases out of 100 for which the program did not converge on a solution.

rate in the first case and is independent of the initial mortality rate in the second case. This difference has a parallel in the biological basis for actuarial senescence. The initial mortality (m_o) rate applies to individuals prior to the onset of physiological senescence for which causes of death are largely extrinsic to the organism: accidents of life that strike individuals independently of their age. Such causes of mortality include predation, physical trauma from accidents, starvation resulting from failed food supplies, extreme weather conditions, and infectious diseases. Aging may cause an increase in mortality rate above the initial level in two ways. First, general physiological decline at advancing age may increase the individual's vulnerability to the same extrinsic causes of mortality that affect young adults. Second, physiological aging may result in disease states that kill the individual independently of extrinsic mortality factors. Deaths resulting from cancers, stroke, heart disease, severe autoimmune disease, and other intrinsic causes fall into this category. Although such intrinsic aging processes may increase the vulnerability of the individual to extrinsic mortality factors, death is inevitable regardless of extrinsic agents, whose intensity has only minor direct influence on the individual's age at death.

Whether actuarial senescence in animals expresses an increase in death from extrinsic or intrinsic causes can be determined, in principle, by manipulating the strength of extrinsic causes of death. If actuarial senescence resulted from increasing vulnerability to extrinsic mortality factors, the mortality rate at a particular age would vary in direct proportion to the mortality of presenescent individuals (m_o) in the population. If actuarial senescence resulted from disease processes that cause death irrespective of external conditions, then the increase in mortality with age would be independent of m_o.

These two possibilities have mathematical parallels in the Gompertz and Weibull functions. Suppose that the aging parameters γ (Gompertz) and α and β (Weibull) represent intrinsic physiological changes in the organism that presumably are independent of most extrinsic causes of mortality in the environment. This is not to say that many environmental factors, such as radiation, diet, toxins, and stress, do not influence the rate of physiological aging. However, to the extent that aging-related mortality increases independently of the intensity of external mortality, measured values of aging

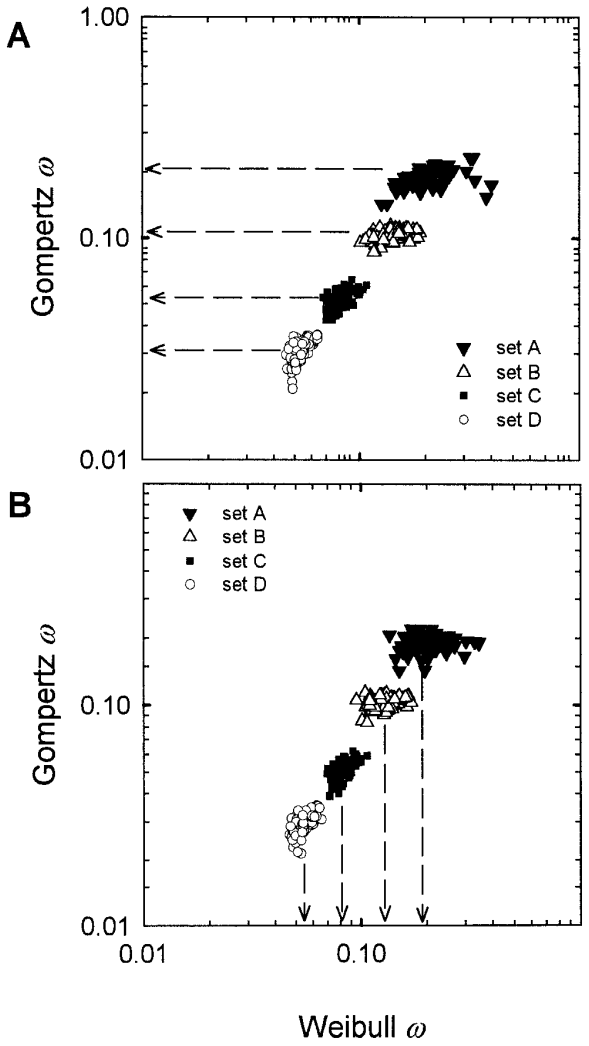

Figure 2. The relationship between Gompertz and Weibull estimates of the rate of aging ω_G and ω_w when the respective models were used to fit data sets generated by **A** a Gompertz model or **B** a Weibull model. Combinations of parameters characterizing four different parameter sets (A–D; see Table 1) were used. For each species 100 simulations were performed for a cohort size of 100 individuals. Arrows indicate parameter values used in simulations.

parameters should be independent of variation in the value of m_o in a particular population.

If the exponential parameter (γ) of the Gompertz equation represented the rate of increase in vulnerability of individuals to primarily extrinsic mortality factors that affect young adults, then the rate of mortality would be the product of this exponential term and the intensity of initial causes of mortality (m_o). Accordingly, the mortality rate at a particular age (m_x) would vary in direct proportion to the extrinsic mortality rate (m_o). If aging-related causes of death were primarily intrinsic, then deaths over and above the

initial mortality ($m_x - m_o$) would be largely independent of the

Table 3. Pearson's Correlation Coefficients Between Gompertz and Weibull Estimates of the Rate of Aging for Data Sets Generated by a Gompertz or a Weibull Model

Function	Gompertz Function		Weibull	
Parameter Set	Pearson's r^2	Probability	Pearson's r^2	Probability
A	0.1174	0.0011	0.1575	0.0002
B	0.1844	0.0001	0.1088	0.0008
C	0.3249	0.0001	0.4349	0.0001
D	0.1289	0.0005	0.4471	0.0001

environment. As a consequence, variation in environmental conditions causing a change in m_o would require a compensating change in the fitted Gompertz aging parameter γ.

In contrast, in the Weibull model, aging-related mortality is independent of the intensity of extrinsic mortality. If aging-related mortality were intrinsically caused and if extrinsic mortality were reduced experimentally even to nil ($m_o = 0$), mortality rate would still increase with age as a result of disease processes that eventually resulted in death, and the estimates of α, β, and ω_w would not vary. However, if aging-related mortality reflected increased vulnerability to extrinsic causes, then ω_w would vary in relation to m_o.

Evaluating Gompertz and Weibull Models by Using Biological Rationales

In the human population, the causes of deaths of young adults and old individuals differ. Excluding infant mortality, these causes are mostly extrinsic in the case of the young and intrinsic in the case of the old (27). Captive populations of rhesus macaques show a similar pattern (28). This suggests that the Weibull model may have a stronger biological rationale than the Gompertz model, but in the absence of a suitable experiment we cannot determine how the mortality rate at a particular age would change in response to a change in m_o. A relevant experiment is performed when animals are brought into captivity in laboratories or zoos, where extrinsic causes of mortality are minimized. Accordingly, the Gompertz model predicts that the increase in mortality rate as a function of age should diminish in proportion to the decrease in m_o. The intrinsic-mortality model predicts that the age-dependent increase in mortality should remain

Table 4. Normalized Bias and *SD*s of Rate of Aging Estimated by Gompertz and Weibull Functions

	Gompertz Function						Weibull Function					
	Gompertz Fit			Weibull Fit			Gompertz Fit					
Weibull Fit Parameter Set	Bias	*SD*	NC	Bias	*SD*	NC	Bias	*SD*	NC	Bias	*SD*	NC
A	0.02	0.09		0.26	0.25	24	0.02	0.08	6	0.01	0.21	16
B	0.01	0.05		0.11	0.15		0.02	0.06		0.02	0.13	
C	0.01	0.09		0.00	0.10	5	0.04	0.10		0.03	0.10	7
D	0.00	0.10	1	0.02	0.08	8	0.06	0.10		0.00	0.08	7

Notes: Gompertz and Weibull Functions are fitted to 100 data sets generated by Gompertz and Weibull models; using parameter sets A–D. *SD* standard deviation; NC number of cases out of 100 for which the program did not converge on a solution.

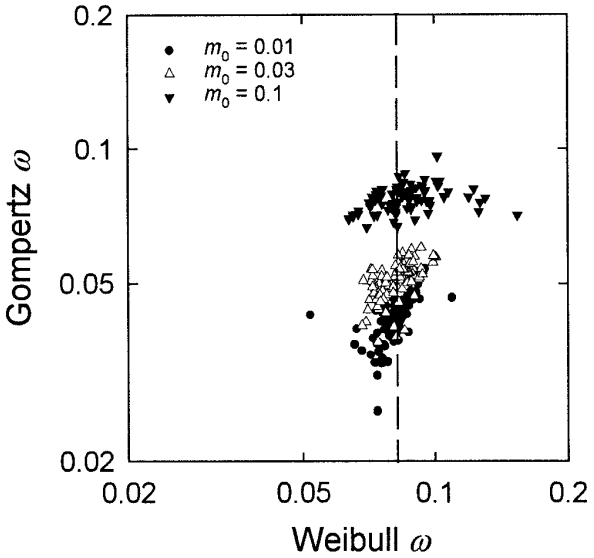

Figure 3. The relationship between Gompertz and Weibull estimates of the rate of aging ω_G and ω_w when the respective models were used to fit data sets generated by a Weibull model; was kept constant at 0.0813, whereas m_o was variable in the source data set. A total number of 100 simulations were performed for a cohort size of 100 individuals per combination.

the same in captivity as in nature. Thus, for the Weibull function, the aging parameters α and β should be independent of variation in m_o; that is, they should be the same in captive and natural populations. For the Gompertz function, γ should increase to compensate for the decrease in m_o and maintain a constant aging-dependent component of mortality.

One comparison of Weibull parameters between wild and captive populations of birds showed that although m_o decreased markedly in captivity, ω_w remained unchanged (26). Unfortunately, the sample size in this comparison

was small and few of the species in the wild and captivity were closely matched. Ricklefs and Scheuerlein (25) compared Weibull aging parameters of 12 conspecific or congeneric pairs of mammals in the wild and in captivity. In this case, 9 of the pairs exhibited lower values of ω_w in captivity than in the wild. This was true for all the species in the sample that inhabit open savannalike environments in nature where a decrease in physiological function with age is likely to reduce an individual's ability to hunt prey or escape predators. Thus, for many species of mammals the increase in mortality rate with age may reflect increasing vulnerability to extrinsic mortality factors. Nonetheless, the rate of aging remained relatively high in captivity in the absence of extrinsic mortality factors experienced in the wild, and so some component of aging-related mortality may also be intrinsic. One of the difficulties with studies of captive populations is that initial mortality rates (m_o) are only partly reduced in captivity. Thus, captivity may impose novel mortality factors, perhaps related to stress and contagious disease, which confound analyses of aging processes and may affect the course of aging.

Conclusions

The Gompertz and Weibull functions make clear distinctions between the manner in which mortality rate increases with age within a population. From an empirical standpoint, each function appears to fit age-at-death data equally well, particularly when sample sizes are large. However, the Weibull function appears to lend itself better to a single parameter () describing the rate of aging in comparative studies when aging-related mortality has intrinsic causes rather than simply reflecting vulnerability to extrinsic causes. Comparisons of the rate of aging between wild and captive populations should allow one to distinguish between the Gompertz and Weibull functions on biological grounds, but results are equivocal because of (a) difficulties in finding suitable phylogenetically matched comparisons, (b) novel sources of mortality in captivity, and (c) mixed results from available comparisons. Our understanding of the causes of aging-related mortality can be guided by considering the biological implications of the mathematical functions we use to describe aging data. The distinction between intrinsic and extrinsic causes of death is difficult but also has meaning for the way mortality relates to the processes of normal aging in organisms. If aging-related mortality primarily reflected intrinsic causes that kill regardless of extrinsic factors, then each individual would maintain a high level of personal fitness until his or her relatively sudden death. If aging-related mortality reflected increasing vulnerability to extrinsic causes of death, then normal aging would be accompanied by continual deterioration of function. These Weibull-like and Gompertz-like scenarios have very different implications for how we view normal aging and the prospects for human life span and the health of the elderly population.

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