Evolutionary Theories of Aging: Confirmation of a Fundamental Prediction, with Implications for the Genetic Basis and Evolution of Life Span

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ABSTRACT: Evolutionary considerations predict that rate of aging should vary in direct relation to the mortality rate of presenescent young adults (extrinsic mortality rate) independently of differences in physiology, such as rate of metabolism. This prediction emerges from theory irrespective of the particular genetic mechanisms responsible for variation in aging. Yet this critical relationship has not been confirmed in comparative studies of natural populations. In the present analysis, rate of aging is estimated by the rate of increase in mortality rate (m_x) with age (x). Comparisons between natural and captive populations of birds suggest that the Weibull model ($m_x = m_0 + \alpha x^{\beta}$) provides a better description of aging than the Gompertz model ($m_x = m_0 e^{\gamma x}$). Rate of aging is quantified by the parameter ω (dimension: 1/time), which is calculated from the Weibull parameters α and β ($\omega = \alpha^{1/(\beta+1)}$). In this analysis, rate of aging in birds and mammals is directly related to extrinsic mortality (estimated by the initial mortality rate, m_0) independently of taxonomic group and of variation in body size and, by implication, metabolic rate. When time is expressed in years, rate of senescence is related to initial mortality rate by $\omega = 0.294 m_0^{0.367}$. This result implies that natural selection in response to variation among taxa in m_0 has resulted in the evolutionary modification of factors that influence the rate of aging in natural populations. The potential strength of selection on factors that could further reduce rate of aging is indicated by the proportion of deaths due to aging-related causes. Although species with low initial mortality rates also exhibit reduced rates of increase in mortality rate with age (i.e., delayed senescence), the relatively high proportion of aging-related deaths in such species suggests that further evolutionary responses leading to long life are severely constrained. This argues against mutation accumulation and antagonistic pleiotropy as genetic mechanisms underlying senescence and suggests, instead, that rate of aging represents a balance between wear and tear, on the one hand, and genetically controlled mechanisms of prevention and repair, on the other. Evidently, remedies for extreme physiological deterioration in old age either are not within the range of genetic variation or are too costly to be favored by selection.

Keywords: aging, antagonistic pleiotropy, birds, mammals, mortality rate, senescence.

Senescence (or aging) is a decline in physiological functioning with age that results in a decrease in reproductive rate, increase in mortality rate, or both (Finch 1990; Abrams 1991; Rose 1991; Holmes and Austad 1995*b*). Defined as such, senescence may result in part from accumulated effects of cellular wear and tear on the individual associated with life itself (Sohal 1986; Finch 1990). However, differences in maximum potential life span among organisms with similar metabolic rates suggest that rate of aging may be modified by biological processes that are under genetic control and subject to evolutionary adjustment (Rose 1991; Charlesworth 1994). For example, sparrows can live 15 yr while rodents of similar size rarely attain a third of that age under ideal conditions. This contrast suggests that wear and tear related to rate of living cannot fully explain variation in rate of aging in natural populations (Holmes and Austad 1995*b*).

Evolutionary theories of aging portray the pattern of senescence as a balance between factors that promote aging and factors that retard aging, at least some of which exhibit genetic variation (Williams 1957; Hamilton 1966; Partridge and Barton 1993*a,* 1993*b*). Evolutionary modification of aging depends on the variance in fitness resulting from differences among individuals in a population with respect to genetic factors expressed in old age. The variance in fitness within a population is a measure of the relative potential strength of selection. For a given effect on the phenotype of the individual, this value is age related and is a function of the proportion of indi-

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viduals reaching a particular age weighted by their expected future reproductive output (Charlesworth 1994). Even in a population without senescence (i.e., with constant adult survival and fecundity), the relative strength of selection declines with advancing age: as potentially immortal individuals die as a result of causes that affect all ages alike, relatively few survive to express genetic factors manifested only late in life.

Evolutionary theories of aging predict that the rate of senescence should be directly related to age-independent mortality rates experienced by young adult individuals (Williams 1957; Rose 1991; Charlesworth 1993; Keller and Genoud 1997). This extrinsic mortality rate influences the proportion of adult individuals in a population that reaches old age, hence the relative strength of selection on genetic factors that influence rate of aging. Thus, in populations with lower extrinsic mortality, more individuals survive to older ages, and selection more strongly favors attributes that delay senescence.

Although the predicted relationship between rate of aging and extrinsic mortality is key to interpreting senescence as an evolved life-history trait, the existence of such a relationship has not been confirmed directly in comparisons among natural populations. To be sure, animals such as elephants (and humans), which are assumed to have low extrinsic mortality, age slowly (Finch 1990). However, one cannot readily disentangle the statistically related effects of body size (associated with variation in metabolic intensity) and mortality rate in such comparisons (Austad and Fischer 1991). Because of their ability to fly, birds are thought to avoid much of the predation and adverse weather that causes death among mammals of similar body size, and they are also known to have longer maximum life spans (Calder 1983; Holmes and Austad 1995*b*), implying low extrinsic mortality. Bats tend to be relatively long-lived, as well, apparently for the same reasons as birds (Austad and Fischer 1991). Yet methods of sampling life span generally differ between birds and mammals, and maximum reported life span itself does not directly estimate either extrinsic mortality or rate of aging. Keller and Genoud (1997) have recently pointed to the association of the long life spans of ant queens and the protected environment of social insect colonies as confirming a relationship between extrinsic mortality and rate of aging. However, neither mortality rates nor aging parameters were quantified in their comparison. Summarizing earlier empirical evidence in support of evolutionary theories of senescence, Rose (1991, p. 98) admitted that "all these patterns [relating rate of aging to mortality rate] have exceptions, and the comparative predictions of the theory concerning rates of aging do not provide material for critical tests." More strikingly, in his analysis of life-history patterns of mammals, Promislow (1991, p. 1876) stated that "while short-lived taxa have high rates of senescence, there is no correlation between mortality rate at age of maturity and rate of senescence."

Many genetic factors are known to influence the pattern of physiological aging or to affect the age-specific schedules of mortality and fecundity that express changes in underlying physiological processes (Finch 1990; Rose 1991; Rose and Finch 1993; Holmes and Austad 1995*a;* Shmookler Reis and Ebert 1996; Finch and Tanzi 1997). It is unclear, however, whether single gene effects identified within human or laboratory populations resemble genetic factors that differentiate patterns of aging between species. Artificial selection, particularly applied to populations of *Drosophila melanogaster,* has also revealed considerable evolutionary responsiveness in age-specific patterns of mortality and fecundity (Rose 1984, 1991; Partridge and Barton 1993*a*), even though heritabilities for longevity are low (Fukui et al. 1996; Promislow et al. 1996). Still, one could argue that responses to powerful artificial selection do not closely mimic the results of weaker selection in natural populations. Although considerable circumstantial evidence suggests that patterns of aging in natural populations are subject to evolutionary modification, it is crucial to show a direct connection between a population's demography and its pattern of senescence. Without this empirical link, it is difficult to conclude that senescence has important fitness consequences for individuals in natural populations and that patterns of senescence have been molded by natural selection.

In this article, I use data from the literature on age-specific mortality rates of birds and mammals to evaluate the relationship between the rate of increase in mortality rate with age and extrinsic adult mortality. First, however, I discuss several issues that impinge on the analysis and interpretation of mortality data. These include the relationship between selection and population demography and the way in which age-dependent mortality is characterized mathematically. I argue that the Weibull function, rather than the more familiar Gompertz function, should be used to characterize the pattern of increase in mortality with age. Taking this approach, analyses of the coefficients of Weibull models fitted to data show not only that rate of senescence is directly related to extrinsic adult mortality but also that this relationship is the same for birds and mammals. This result suggests that senescence produces unique causes of mortality unrelated to extrinsic factors and that the model of the evolutionary modification of senescence envisioned by Medawar (1952) and Williams (1957) is essentially correct for birds and mammals. Furthermore, the analyses reveal that populations with lower extrinsic mortality suffer a

higher proportion of senescent deaths. This result is inconsistent with genetic models of senescence based on mutation accumulation and certain types of antagonistic pleiotropy, and it suggests instead that the pattern of senescence balances somatic wear and tear against maintenance and repair mechanisms whose efficacy is under genetic control.

Analysis and Interpretation of Mortality Data

The exact form of the relationship between relative strength of selection and age depends on how genetic factors influence age-specific values of mortality and fecundity (Abrams 1991; Abrams and Ludwig 1995). In a hypothetical nonsenescing population (age-independent adult mortality and fecundity), genes that turn on at adult age x and have a continued influence on mortality rate m for the rest of the individual's life influence fitness (the intrinsic exponential growth rate of the population, r) according to

$$\frac{\partial r}{\partial m} = -\frac{1}{(b+1)^x}, \tag{1}$$

where b is the rate of fecundity (Abrams 1991). Thus, the strength of selection on genetic factors that cause a given change in adult mortality rate declines rapidly with increasing age, especially in populations with high fecundity.

As it is defined in this article, senescence refers to an increase in adult mortality rate with age associated with progressive physiological decline. The relationship between mortality and age is sometimes called *actuarial senescence* (Holmes and Austad 1995*a*) to distinguish demographic and physiological manifestations of aging. In order to interpret analyses of life-table data, one must assume that actuarial senescence is related to decline in physiological function. Theoretical studies have shown that factors other than senescence may cause mortality to increase with age (Abrams 1993; Blarer et al. 1995; McNamara and Houston 1996). When the expectation of future reproduction declines with age, an optimized life history may include increased reproductive effort late in life, which in turn may cause mortality to increase (Taylor et al. 1974; Pianka and Parker 1975; Charlesworth and Leon 1976; Blarer et al. 1995). For organisms that live more than 1 yr, this model presupposes some senescence-like process that causes reduced expectation of future reproduction with age, and thus it cannot be used to argue against physiological decline being responsible for increasing mortality rate with age. Reproductive effort may also increase when size or condition increases with age, and this may lead to higher mortality (McNamara and Houston 1996). However, reproductive output typically does not increase in old age in organisms having a fixed adult size and is often observed to decline instead (e.g., birds and mammals: Newton 1989*a;* Forslund and Pärt 1995; Holmes and Austad 1995*a*). Twenty-five reported patterns of avian senescence in natural populations (19 species; Holmes and Austad 1995*a,* table 1, excluding three zoo populations) included 13 cases of actuarial senescence, seven cases of reproductive senescence, three cases of apparent physiological decline, and only two cases of apparent increase in reproductive success at old age (for the last, see Wooller et al. 1989; Pugesek and Diem 1990). It is therefore unlikely that actuarial senescence results directly or solely from higher investment in reproduction.

There is little direct evidence from natural populations for physiological decline in function of adult birds with increasing age, although laboratory mammals and, to a lesser extent, domesticated birds have been thoroughly characterized in this respect (see references in Finch 1990; Holmes and Austad 1995*a*). Domestic fowl and Japanese quail exhibit decreased function of the reproductive tract with age (Ottinger 1991), but these species are strongly selected for early egg laying and may no longer provide useful models for natural populations. A. P. Møller and F. de Lope (unpublished manuscript) have demonstrated decreases with age in the size of sexually selected characters, developmental stability, migration characteristics, reproductive performance, and ability to control infestations of ectoparasites in the barn swallow *Hirundo rustica.* These results support a direct relationship between increasing mortality rate and declining physiological condition in this species. Other species kept in zoos, and several natural populations of birds, exhibit declining reproductive performance with age (Clum 1995; Holmes and Austad 1995*b;* Ottinger et al. 1995).

Abrams (1993) showed how the relationship between extrinsic adult mortality rate and senescence can be affected by the pattern of density-dependent effects on age-specific values of survival and fecundity in a population. An important result is that when density feedbacks resulting from change in adult survival influence fecundity independently of age or when they influence juvenile survival or both (generally, recruitment), the rate of senescent mortality at any given age—hence, the rate of senescence—will be directly related to extrinsic adult mortality. A decrease in the rate of senescent mortality with increasing age will be favored when density mainly affects survival or fecundity late in life. That is, when reduced extrinsic mortality leads to an increase in population size, older age classes increase disproportionately, which increases the relative strength of selection on ge-

netic factors expressed in old age. This model presupposes senescence in the population and also that senescent individuals are most adversely affected by crowding. Abrams (1993) also pointed out that higher mortality can select for decreased senescent mortality when a senescent trait increases the vulnerability of an individual to a particular mortality factor. For example, declining physiological function may impair an individual's ability to escape predators or survive severe weather. In this case, more powerful mortality factors tend to increase the strength of selection against senescence, thereby tending to obscure a relationship between extrinsic adult mortality and rate of senescence.

In birds and mammals, therefore, a relationship between rate of senescence and extrinsic adult mortality would support a population model of the evolutionary modification of senescence in which density dependence falls most heavily on prereproductive individuals and in which vulnerability to extrinsic mortality factors is mostly independent of the aging-related physiological state of the organism. The exact form of the relationship between mortality and age, and of the relationship between rate of increase in age-specific mortality and extrinsic mortality, cannot be predicted without knowing how genetic factors influence fitness. However, failure to find a direct relationship between rate of senescent mortality and extrinsic mortality rate (Promislow 1991) would require a reevaluation of the demographic contexts of both the evolutionary modification of aging and the evolutionary optimization of reproductive investment.

Methods

Models of Aging-Related Parameters

Several mathematical models are commonly used to characterize the consequences of senescence for mortality rates in populations (Gavrilov and Gavrilova 1991; Wilson 1994). These relate mortality rate at age x (m_x [dimension: time^{-1}]), to initial mortality rate at age 0 (m_0 [time^{-1}]) by one or more "aging" parameters. The Gompertz equation (Mueller et al. 1995) describes an exponential increase in mortality rate with age at rate γ (time^{-1}), that is, $m_x = m_0 e^{\gamma x}$. Rate of aging has been compared among populations by the parameter γ and by the derived mortality rate doubling time (MRDT [time] $= \log_e 2/\gamma$) (Finch 1990). To interpret the Gompertz parameter as an index to the rate of aging, one implicitly assumes that physiological senescence increases the vulnerability of individuals to the same mortality factors that kill young adults; mortality is a multiple ($e^{\gamma x}$) of the initial rate (m_0), which grows exponentially with age. According to Abrams's (1993) theoretical results, this assumption concerning the interaction between extrinsic mortality and senescence reduces the evolutionary influence of increased extrinsic mortality on rate of senescence because rate of senescent mortality responds phenotypically to variation in extrinsic mortality and therefore the proportion of senescence-related deaths in the population does not change. Indeed, Promislow (1991), in his comparison of age-dependent mortality rates in mammals, found that the value of the exponential "aging" parameter γ was statistically uncorrelated with the estimated extrinsic mortality. As a result, Promislow concluded that rate of aging was independent of initial mortality rate. Of course, in comparisons having constant γ, senescent mortality (m_x at any given age) actually increases in direct proportion to m_0. Hence, the Gompertz parameter γ is not, by itself, an informative measure of absolute mortality resulting from actuarial senescence.

Alternatively, aging-related mortality may be caused by factors different from those that affect young adults (e.g., degenerative disease, reduced resistance to infections, tumors), in which case initial (extrinsic) mortality and aging-related (intrinsic) mortality should add together to give the total at any particular age, and they should be computationally independent. The Makeham modification of the Gompertz equation partitions mortality into separate terms for initial and aging mortality, that is, $m_x = m_0 + ae^{\gamma x}$ (Gavrilov and Gavrilova 1991; Wilson 1994). However, because the aging term is exponential, the initial mortality rate ($x = 0$) is now $m_0 + a$, and actuarial aging is still expressed as an age-dependent multiple of a portion of the initial mortality rate.

The Weibull equation $m_x = m_0 + \alpha x^{\beta}$ separates the initial mortality and aging terms computationally and characterizes aging-related mortality by two parameters, one (β [dimensionless]) controlling shape and the other (α [time$^{-(\beta+1)}$]), magnitude for a given shape. The variable ω, which equals $\alpha^{1/(\beta+1)}$ [time^{-1}], provides a shape-adjusted index to the rate of aging that is comparable to parameter γ of the Gompertz equation but portrays the rate of aging independently of the initial mortality rate.

In both Gompertz and Weibull models, rate of mortality continues to increase, at an accelerating rate, with increasing age. Empirical studies of large populations of flies and humans have shown, however, that a plateau in mortality rate may be reached in extremely old age (Carey et al. 1992; Fukui et al. 1993, 1996). Mueller and Rose (1996) have suggested that this occurs because the force of selection at extreme old age becomes significantly less than forces of mutation and drift, which are independent of age. However, the point at which selectively responsive senescence leaves off is attained by only a small fraction of a population and is mostly beyond the reach of the samples compared here or in any study of

natural populations of vertebrates. Thus, conventional models of senescence are appropriate for this analysis.

Choice of Aging Model

Which aging model is appropriate depends on both statistical and biological considerations. Statistically, it is important that estimates of extrinsic mortality and aging parameters are independent. For the Gompertz equation, m_0 and γ are not independently estimated. Variation in data that result in a higher estimate of one parameter result in a lower estimate of the other. This is why Promislow (1991) estimated extrinsic mortality (μ) independently from survival curves. For the Weibull equation, estimates of α and β are highly intercorrelated, but these are relatively independent of m_0. For example, in maximum likelihood estimates of aging parameters for female pied flycatchers and male Dall sheep (see below), the asymptotic correlations for estimates of m_0 and γ were -0.91 and -0.97, respectively. For the Weibull equation, asymptotic correlations between α and β were -0.99 in both cases, but correlations between these parameters and m_0 averaged -0.69 and -0.55, respectively.

The more important consideration concerning a choice of aging model is whether the effects of senescence on mortality multiply (Gompertz), or add to (Weibull), the initial mortality rate. One way to distinguish between the assumption of factorial increase and that of incremental increase would be to manipulate the initial mortality rate experienced by a population and estimate the aging-related mortality (Promislow 1991). The Gompertz model would be implied by a proportional response of m_x to m_0, and the Weibull model would be implied by independence between the two. This "experiment" is conducted inadvertently with captive populations that are removed from natural sources of extrinsic mortality. To take advantage of this circumstance, Weibull equations of the form

$$l_x = \exp\left(-m_0 x - \frac{\alpha x^{\beta+1}}{\beta + 1}\right), \qquad (2)$$

where l_x is the proportion of the population surviving to age x, were fitted to survival data for five species of bird in the London Zoo (Comfort 1962; fig. 1). Initial mortality rates (m_0) for these captive populations (table 1) were lower by one to two orders of magnitude than values in natural populations of comparable species. In two cases, it was possible to estimate values for β, which were 2.4 (*Pavo cristatus*) and 2.0 (*Nycticorax nycticorax*); these fits yielded $m_0 = 0.11$ (0.01 SE) yr^{-1} and 0.024 (0.010 SE) yr^{-1}, and $\omega = 0.19$ yr^{-1} and 0.10 yr^{-1}, respectively. When β was fixed at 3.0, which is close to the average for birds

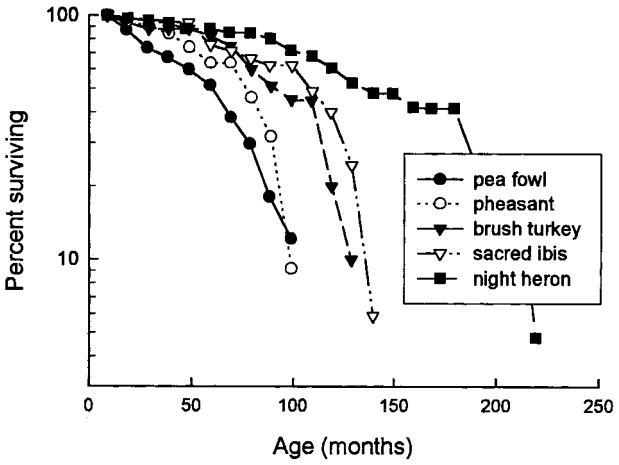

Figure 1: Survival curves for five species of bird in the London Zoo. Data from Comfort (1962). See table 1 for fitted parameters of Weibull equations.

as a whole (see below), estimates of m_0 for all five species varied from 0 to 0.11 yr^{-1} and estimates of ω varied between 0.09 and 0.21 yr^{-1} (table 1). None of the species included in this sample is well known in nature, but initial mortality rates are likely to exceed 0.10, for which ω estimated from the observed relationship between ω and m_0 in figure 6 below is 0.12 yr^{-1}. North American black-crowned night herons (*N. nycticorax*) have adult mortality rates of $m = 0.27$ (Henny 1972), for which estimated ω is 0.17 yr^{-1}. A natural population of ring-necked pheasant (*Phasianus colchicus*) in Denmark had a value of $m = 0.88$ (Cramp 1980; the zoo population of Reeve's pheasant *Syrmaticus reevesi,* had $m_0 = 0.011$), for which estimated ω is 0.27 yr^{-1}. Thus, aging-related mortality in zoo populations ($\omega = 0.156 \pm 0.019$ SE, $n = 5$) is similar to that observed in the wild ($\omega = 0.160 \pm 0.018$ SE, $n = 18$; app. A, table A1) and therefore appears to be insensitive to experimental manipulation of m_0. In contrast, Gompertz equations of the form

$$l_x = \exp\left(-\frac{m_0}{\gamma}[e^{\gamma x} - 1]\right) \qquad (3)$$

fitted to the London zoo data yielded initial mortality rates of 0.003–0.07 yr^{-1} and mortality rate doubling times of 0.09–0.27 yr (1–3 mo). These MRDTs are more than an order of magnitude lower than values estimated for natural populations of birds (Finch 1990) because of the low initial mortality rates of captive populations. In contrast, Weibull aging parameters (ω) calculated for zoo populations are consistent with initial mortality rates of natural populations. Thus, the Weibull function is a more appropriate model than the Gompertz function for describing aging-related mortality. Patterns of aging

Table 1: Parameters of the Weibull equation fitted to survival curves of five species in the London Zoo

Species	N	m_0*(SE)*	α*(SE)*	ω
Pea fowl *Pavo cristatis*	80	.111 (.007)	1.1E−3 (.8E−3)	.181
Reeves pheasant *Syrmaticus reevesi*	107	.011 (.009)	1.8E−3 (.3E−3)	.205
Brush turkey *Alectura lathami*	34	.019 (.007)	6.0E−4 (.4E−4)	.155
Sacred ibis *Threskiornis aethiopicus*	30	.000 (.000)	5.2E−3 (.4E−3)	.151
Night heron *Nycticorax nycticorax*	44	.024 (.009)	6.6E−5 (1.0E−5)	.090

Note: Shape parameter of Weibull function (β) fixed at 3; N = number of individuals; m_0 = initial mortality rate (yr^{-1}); α = scale parameter of Weibull function; ω = Weibull aging parameter (yr^{-1}). Data from Comfort (1962).

in zoo populations of birds would appear to reject one of the conditions cited by Abrams (1993; senescence-dependent expression of extrinsic mortality factors) as reducing the evolutionary relationship of rate of senescence on extrinsic adult mortality. Therefore, we can assume that senescence is associated with unique mortality factors related to physiological deterioration, which cause an increase in mortality with age independently of extrinsic mortality.

Estimation of Extrinsic Mortality

The extrinsic mortality of a population may be defined operationally as the minimum mortality exhibited by an adult age class. In birds and mammals, mortality rates typically decrease dramatically between independence and sexual maturity and often continue to decrease for some period after the onset of reproduction (e.g., Newton and Rothery 1997). Thus, the age-specific pattern of mortality includes both increasing mortality with age due to senescence and decreasing mortality with age associated with size and experience (Sæther 1990; Forslund and Larsson 1992; Forslund and Pärt 1995). Because the latter is not incorporated into mathematical models of aging, the value of m_0 is not strictly an estimate of extrinsic mortality. When the effects of senescence on mortality do not begin to increase dramatically until well after the benefits of experience have leveled off, adult mortality should exhibit a broad valley over which mortality rate is low and does not change rapidly with respect to age. This minimum mortality rate should be close to the extrinsic mortality of the population. In addition, under these circumstances values of m_0 obtained by fitting aging equations to data should also estimate extrinsic mortality reasonably well. This is because a period of constant mortality rate through young adulthood establishes a lower asymptote for mortality rate, which extrapolates with little change to age 0 (see, e.g., fig. 4).

In this analysis, I have taken a two-tiered approach to the estimation of extrinsic mortality rate. First, fitted parameter values for m_0 are compared to the pattern of age-specific mortality rate exhibited by the data. When m_0 was similar to mortality rates of young adult age classes, it was accepted as an estimate of extrinsic mortality rate. When m_0 differed substantially (usually being too low, even negative), I calculated the average mortality rate of adults over age classes showing a low and relatively constant mortality rate (m_e). Handling of data in particular cases is explained in appendix A.

In his analysis of mammalian life tables, Promislow (1991) estimated extrinsic mortality rate as the instantaneous rate of mortality at the age of maturity (μ). This was calculated by fitting a third-order polynomial curve to the log-transformed survival curve and evaluating the slope of the curve at the age of maturity. For 12 mammalian data sets for which I accepted m_0 as an estimate of extrinsic mortality rate, m_0 was highly correlated with Promislow's μ (log-transformed values: $r = 0.79$, $P = .0024$; $r_s = 0.79$, $P = .0023$). In a regression of m_0 on μ, the intercept did not differ significantly from 0 (0.14 [0.26 SE]) and the slope did not differ significantly from 1 (1.15 [0.28 SE]). Thus, estimates of m_0 and μ were similar in these cases. For 11 mammalian data sets for which I estimated extrinsic mortality by the mortality rates of young adults, my estimate and Promislow's μ were not significantly correlated ($r = 0.48$, $P = .13$; $r_s = 0.60$, $P = .051$). This sample included primarily species with low extrinsic mortality, and μ for these species ($\log_{10}$ units, -1.39 ± 0.77 SD) was much more variable than m_e ($-1.08 \pm .36$ SD). Unlike μ, the estimate of m_e is not influenced by mortality outside of young adult age classes and may be more representative for that reason.

Parameter Estimation

Much has been written about estimating the parameters of survival models (e.g., Eakin et al. 1995; Mueller et al. 1995; Nichols et al. 1997). Approaches generally involve

Table 2: Estimates of coefficients of Weibull functions fitted to the relationship between mortality rate and age in natural populations of birds and mammals

Species	*Sex*	*Age groups*	m_0		α		β		ω	*Reference notes*
			Coefficient	*SE*	*Coefficient*	*SE*	*Coefficient*	*SE*		
Birds:										
Larus canus	MF	13	.097	.021	3.32E−5	.00E−5	2.96	1.08	.074	2
Puffinus tenuirostris	MF	13	.051	.035	2.28E−3	6.13E−3	1.34	.81	.074	6
Diomedia exulans	MF	4	.013	.000	6.64E−5	1.34E−5	2.01	.00	.041	7
Ficedula hypoleuca	F	7	.664	.032	2.35E−3	1.64E−3	3.14	.36	.232	8
F. hypoleuca	M	7	.778	.045	1.35E−3	1.42E−3	3.47	.54	.228	8
Parus major	F	6	.043	.162	.60E−3	2.67E−3	3.80	2.25	.213	9
Turdoides squamiceps	F	5	.183	.211	.62E−1	1.55E−1	1.18	1.31	.279	11
T. squamiceps	M	6	.115	.031	.84E−3	1.68E−3	3.39	1.10	.199	11
Passerina cyanea Reserve	M	5	.437	.041	3.69E−3	5.24E−3	2.59	.76	.210	12
Mammals:										
Hippopotamus amphibius	MF	12	.000	.00	1.21E−7	.12E−7	4.00	.00	.041	9
Ovis dalli	M	11	.043	.00	3.11E−6	.59E−6	5.35	.08	.136	16

Note: Table includes only those studies for which nonlinear curve fitting converged to a solution. For references and notes, see appendix A, table A2 for birds and table A1 for mammals.

a trade-off between the number of parameters and other effects in a model and ability to estimate parameters accurately. In general, maximum likelihood approaches are preferred to linear and nonlinear regression (Petersen 1986; Mueller et al. 1995), although sample sizes in studies of natural populations are often so small that maximum likelihood approaches may not converge on stable solutions. Following the application of several estimation procedures to a few data sets and the recovery of parameter values from simulated data (app. B), I ended up by fitting Weibull functions to the relationship between mortality rate (m_x) and age (x) by unweighted nonlinear regression (Procedure NLIN of SAS; SAS Institute 1985). This approach performs best with small sample sizes and does not require information on the numbers of individuals per age class, which are not given in many published studies. Thus, unweighted nonlinear least squares permitted the estimation of parameters from the largest number of data sets and introduced little bias compared to differences between species.

I calculated m_x as the instantaneous (exponential) mortality rate—that is, the negative of the natural logarithm of the proportion of individuals surviving over a unit interval of time (1 yr). The quality of the life tables for natural populations of mammals were poor compared with those of birds (see below), and the Weibull parameters α and β could not be estimated simultaneously by nonlinear curve fitting in most cases. These data were then fitted (Procedure NLIN of SAS) by Weibull equations with $\beta = 3$, which is approximately the average value among birds (see table 2). The effect of fixing the value of β on estimates of the rate of actuarial senescence (ω) is illustrated in figure 2. In fact, ω is rather insensitive to the value of β chosen for nonlinear curve fitting, and values of ω for parameter estimates with β free and β fixed are strongly correlated (fig. 3).

Data

Field Studies

Most studies of demography in mammal populations are based on collections of the remains of individuals whose ages are estimated from size, tooth wear, and other morphological indices (Caughley 1977). The resulting tabulation of ages at death is then converted to a survivorship table, from which age-specific survival rates are calculated. In this study, survival rates were converted to instantaneous mortality rates ($m = -\log_e S$), which were then fitted by Weibull functions to the relationship between m_x and age. Most life tables of bird populations are obtained from local, longitudinal studies of individuals marked in the year of birth, from time-specific survival rates in a population of known-age birds, or from a combination of these two approaches (Newton 1989*a;* LeBreton et al. 1992). Age-specific annual survival is estimated directly from the persistence of known-age individuals in the population. Survival data from such studies were converted to instantaneous mortality rates for fitting with Weibull functions.

Data on age-specific survival rates were obtained primarily from sources listed in (Promislow 1991) for mammals (app. A; table A2), and from (Newton 1989*a*) and

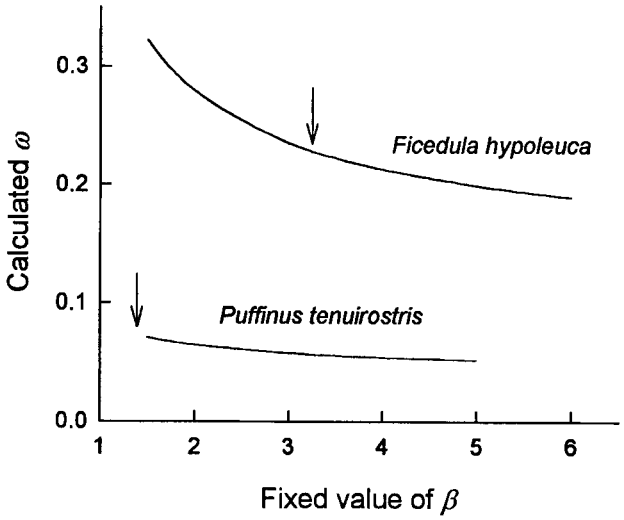

Figure 2: Sensitivity of the Weibull aging parameter (ω) to variation in the value of β used in fitting the Weibull model to the increase in mortality rate with age, using data for the pied flycatcher (*Ficedula hypoleuca*) and the short-tailed shearwater (*Puffinus tenuirostris*). Arrows indicate the fitted values of β when it is retained as a free parameter.

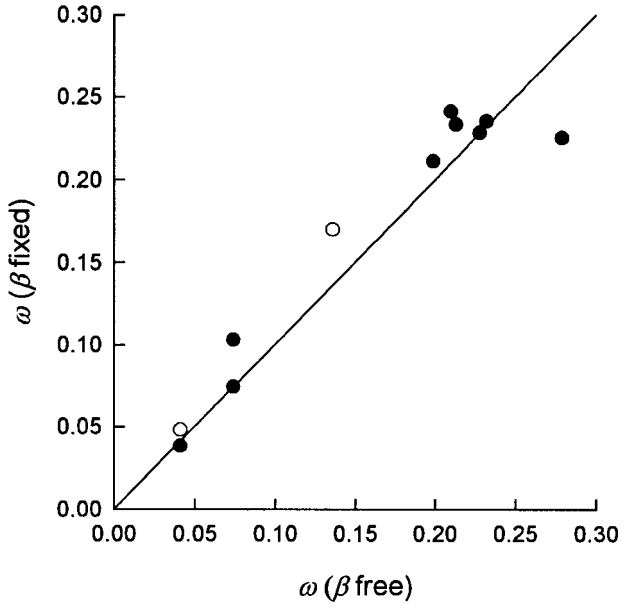

Figure 3: Relationship between the Weibull aging parameter (ω) when it is calculated from fitted Weibull equations with β as a free parameter compared with β as a fixed parameter with a value of 3. Solid symbols represent species of bird; open symbols, species of mammal.

sources listed in (Holmes and Austad 1995*b*) for birds (app. A, table A1). Data for males and females, when presented separately, were considered as independent estimates of aging parameters. Studies on mammals were included only if they revealed a significant increase in mortality rate with age in Promislow's analysis. This selection criterion presumes that studies failed to show a senescent pattern of age-specific mortality rate because of inadequate sampling either of ages or of number of individuals. In Promislow's (1991) sample of 56 mammal data sets, half showed significant ($P < .05$) actuarial senescence. Of these, 22 out of 28 were represented by eight or more age classes. Of the 28 nonsignificant data sets, 18 included fewer than eight age classes. Furthermore, mammalian life tables constructed by sampling the age structure or the distribution of ages at death in a population are less likely to reveal actuarial senescence than are age-specific survival data because the variance in the estimate of mortality at a particular age is greater. These considerations, and the simulations presented in appendix B, emphasize that parameter estimates from small data sets must be interpreted cautiously. For this reason, several analyses in this study were run both with the entire data set and with species represented by small samples deleted.

Most of the reported data were accepted at face value, although age-specific mortality rates were recalculated in some cases. Higher mortality rates reported for younger age classes (e.g., Newton and Rothery 1997), which express lack of maturity or experience (Forslund and Larsson 1992; Forslund and Pärt 1995; Sæther 1990), were deleted from the analyses.

Phylogenetic Comparisons

In comparative studies, phylogenetic relationship among taxa may influence the statistical interpretation of correlations and regressions among traits and has been claimed to produce biased or spurious results in some cases (Harvey and Pagel 1991). I have not applied phylogenetically corrected methods to this analysis because comparisons of statistics based on phylogenetically independent contrasts (PIC) and species data generally do not differ; calculation of PICs introduces its own sources of error to the data and embodies restrictive assumptions; and phylogenetic relationships, especially for mammals, are not well resolved (Ricklefs and Starck 1996). Among mammals, the 27 samples of life tables reported in appendix A, table A2, are drawn from 13 families belonging to seven orders (authority for taxonomy: Wilson and Reeder 1993).

Among birds, the 18 life tables analyzed (app. A, table A1) belong to 12 species in nine families (authority for

taxonomy: Monroe and Sibley 1993). However, taxa with rapid aging ($\omega > 0.15$) were all Passeriformes except for the European sparrowhawk (*Accipiter nisus:* Ciconiiformes). Taxa with delayed aging were either Anseriformes or Ciconiiformes. Thus, there appears to be a strong taxonomic association to rate of aging, but this is associated with similar taxonomic patterns in body mass and initial mortality rate. The most compelling argument against a strong phylogenetic component to rate of aging is the parallel patterns and similar ranges of variation in birds and mammals. Potential problems for statistical inference of phylogenetic relationship are addressed in part by estimating the minimum degrees of freedom that would support statistical significance of a particular relationship.

Results

The fit of a Weibull equation to age-specific mortality rates of female pied flycatchers (*Ficedula hypoleuca:* Muscicapidae) is shown in figure 4. In this example, the shape parameter β is 3.14 $\pm$ 0.36 SE, and the rate of aging-related increase in mortality, ω, is $0.00235^{0.24} = 0.23\ \text{yr}^{-1}$. Weibull shape constants, which could be estimated for seven species of bird, tended to increase with increasing initial mortality rate but not markedly so (fig. 5). For these species, the logarithmic relationship between ω and m_0 was significant ($F = 18$, df = 1, 7, $P = .0037$, $R^2 = 0.72$; $r_s = 0.76$, $P = .017$); the regression is described by $\log_{10}(\omega) = -0.55$ (0.09 SE) + 0.40 (0.09 SE) $\log_{10}(m_0)$. When the number of degrees of freedom was reduced by two to account for two species in which males and females were analyzed separately, the F test had a significance value of $P < .005$, and Spearman's rank correlation (r_s), $P < .05$. When the two species of mammals for which β could be estimated were added to the regression analysis, the regression equation is $\log_{10}(\omega) = -0.54$ (0.10 SE) + 0.41 (0.09 SE) $\log_{10}(m_0)$ ($F = 20$, df = 1, 9, $P = .0015$, $R^2 = 0.69$; $r_s = 0.84$, $P = .0012$); reducing the degrees of freedom by two changed the P values to $<.005$ and $<.01$, respectively. These results are consistent with predictions of evolutionary models of aging. However, nonlinear regression algorithms failed to converge on stable solutions when applied to many data sets, and so it was not possible to estimate β for some birds and most mammals. Consequently, rate of senescence was further compared by estimating α and ω under the assumption that $\beta = 3$, which is approximately the average value for birds in table 2. Because the fitted value of α is strongly inversely correlated with the estimated value of β for a given set of data, fixing β results in a shape-adjusted estimate of the rate of increase in mortality with age (ω) that is relatively insensitive to variation in β over the range observed in this study (see fig. 2).

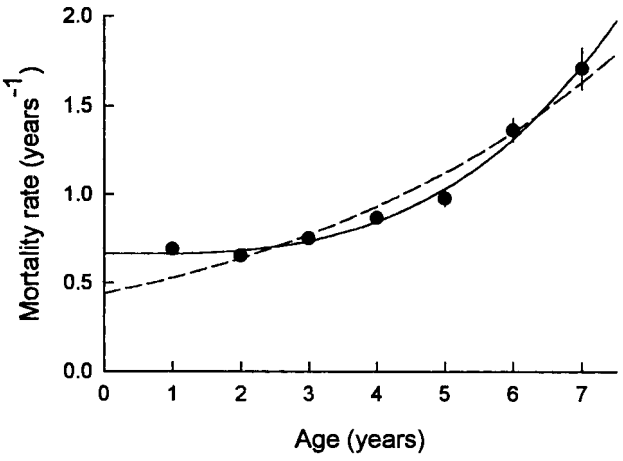

Figure 4: Relationship of mortality rate (m_x) to age in female pied flycatchers (*Ficedula hypoleuca*), with fitted Gompertz (*dashed line*) and Weibull (*solid line*) aging models. The Weibull equation is $m_x = 0.664 + 0.00235x^{3.467}$. The Gompertz equation is $m_x = 0.440e^{0.187x}$. Vertical bars are ± 1 SE of the mean. Data from Sternberg (1989).

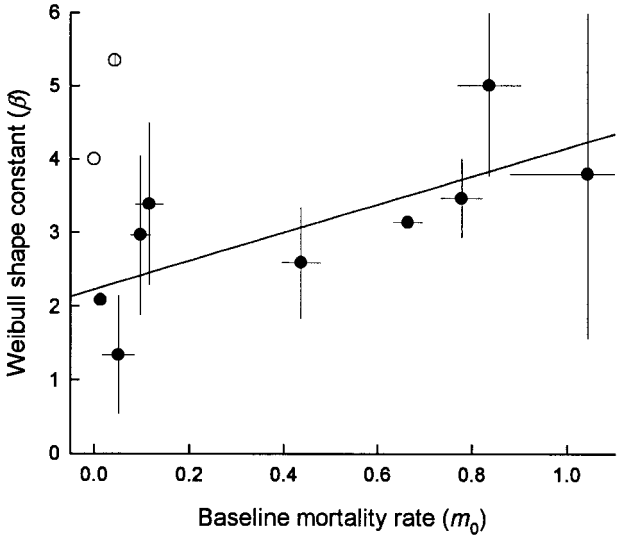

Figure 5: Relationship of the shape constant (β) of the Weibull equation to initial mortality rate (m_0) in seven species of bird (table 2). The vertical and horizontal lines are ± 1 SE of the estimate. The regression has the equation $\beta = 2.2$ (0.4 SE) + 1.9 (0.7 SE) m_0 ($F = 7.7$, df = 1, 7, $P = .028$, $R^2 = 0.52$; nonparametric $r_s = 0.64$, $P = .035$). Sexes are combined for five of the species and separated for two of them; when the denominator degrees of freedom are reduced by two to account for two species with values for both males and females, $.025 < P < .05$; the average of β is 3.1. When values of β for the two species of mammals in table 2 are added to the statistical analysis, the relationship between β and m_0 becomes insignificant ($F = 0.8$, df = 1, 9, $P = .4$).

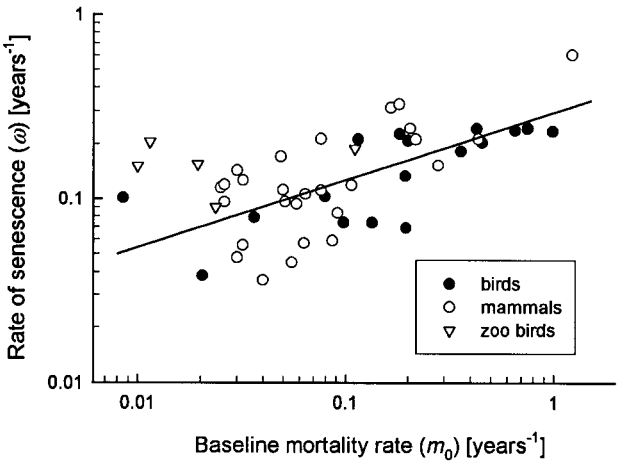

Figure 6: Relationship between rate of senescence (ω of the Weibull equation, when β = 3) and initial mortality rate in populations of birds (*solid circles*) and mammals (*open circles*). Data from captive populations in the London Zoo are indicated by open triangles; m_0 is the value estimated from the zoo data and does not refer to natural extrinsic mortality.

At a given body mass, rate of senescence (ω) varied over an order of magnitude. Furthermore, ω increased in both birds and mammals in direct relation to initial mortality rate (fig. 6), independently of body mass. An ANCOVA revealed that rate of aging (ω) decreased with respect to increasing mass, specifically with mass raised to the −0.17 (±0.04 SE) power, and was 0.39 $\log_{10}$ units (2.4-fold) lower in birds than in mammals of similar mass ($F = 10.6$, df = 2, 40, $P < .0002$, $R^2 = 0.35$; interaction term $F = 1.2$, df = 1, 39, $P = .28$, NS). However, m_0 also decreased with increasing mass, specifically with mass raised to the −0.39 (±0.05 SE) power, and m_0 was 0.69 (±0.15 SE) $\log_{10}$ units (4.9-fold) lower in birds than in mammals of comparable size ($F = 43.4$, df = 2, 40, $P < .0001$, $R^2 = 0.68$; interaction $F = 4.8$, df = 1, 39, $P = .034$, marginally significant and deleted). The relationship of ω to m_0 turned out to be independent of body mass. In a multiple logarithmic regression relating ω to m_0 and body mass (M), both the $m_0 \times M$ interaction ($F = 0.1$, df = 1, 39, $P = .77$) and M ($F = 0.43$, df = 1, 40, $P = .52$) were insignificant effects. When birds and mammals were distinguished as a main effect (TAXON) in this analysis, neither the $m_0 \times$ TAXON interaction ($F = 0.7$, df = 1, 39, $P = .40$) nor TAXON itself ($F = 1.4$, df = 1, 40, $P = .25$) was significant. Rate of senescence ω was directly related to initial mortality rate m_0 ($F = 31.2$, df = 1, 41, $P < .0001$, $R^2 = .43$) by the equation $\log_{10}\omega = -0.531$ (±0.074 SE) + 0.367 (±0.066 SE) $\log_{10} m_0$. Thus, rate of senescence in this sample increased as the approximately 3/8 or 1/3 power of m_0. With an F value of 31, the denominator degrees of freedom could drop to as low as 7 and still have a significance level of $P < .001$.

When values for males and females were averaged, the number of observations (species) decreased to 30, but the relationship between $\log_{10}\omega$ and $\log_{10} m_0$ remained strong ($F = 34$, df = 1, 28, $P < .001$, $R^2 = 0.55$); the slope of the regression increased to 0.441 (±0.076 SE). When the analysis included only data sets with >100 individuals (32 data sets), the results were qualitatively identical, and the relationship between $\log_{10}\omega$ and $\log_{10} m_0$ was basically unchanged ($F = 32.5$, df = 1, 30, $P < .0001$, $R^2 = 0.52$), with a regression slope of 0.41 (±0.07 SE). Reducing the analysis to data sets with >200 individuals (18 data sets) further confirmed this result: regression of $\log_{10}\omega$ on $\log_{10} m_0$ ($F = 46.0$, df = 1, 16, $P < .0001$, $R^2 = 0.74$); intercept −0.512 (±0.059 SE); slope 0.410 (±0.060 SE). These results confirm, for mammals and birds, the fundamental prediction of evolutionary theories of aging.

Proportion of Senescent Deaths

Senescence is an important source of mortality in natural populations, particularly those with low initial mortality rates. The impact of senescence may be estimated from the proportion of deaths in a population that can be attributed to aging-related causes (P_S). For the Weibull model, this proportion is

$$P_S = \int_{x=0}^{\infty} \alpha x^{\beta} l_x \, dx, \tag{4}$$

where probability of survival to age x (l_x) is

$$l_x = e^{-\{m_0 x + [\alpha/(\beta+1)] x^{\beta+1}\}}. \tag{5}$$

For predicted values of α (= $\omega^{\beta+1}$) at empirically determined values of m_0 (assuming β = 3) observed in birds and mammals (fig. 6), the maximum fraction of senescent deaths (P_S) varies from 79% at $m_0 = 0.01$ yr^{-1} to less than 3% at $m_0 = 1.0$ yr^{-1} (table 3). For analyses in which β was a free parameter (table 2), the relationship between P_S and m_0 is shown in figure 7. The best model relates the logarithm of P_S to m_0 by the equation $\log_{10} P_S = -0.20$ (±0.06 SE) − 1.85 (±0.13 SE) m_0 ($F = 212$, df = 1, 9, $P = < .0001$, $R^2 = 0.96$). Another way to assess the impact of senescence is to compare the average life span (T) of adult individuals in natural populations to that in hypothetical nonaging populations ($T = 1/m_0$). For the Weibull model of aging, the average life span is

$$T = \int_{x=0}^{\infty} e^{-\{m_0 x + [\alpha/(\beta+1)] x^{\beta+1}\}} dx. \tag{6}$$

Senescence reduces average life span by only 2% when $m_0 = 1.0$ yr^{-1} but by almost 80% when $m_0 = 0.01$ yr^{-1} (table 3 and fig. 8). These comparisons suggest that senescence should be easier to detect in populations of rel-

Table 3: Proportion of senescence-related deaths (P_S) and average life span (T) in populations with different initial mortality rates (m_0)

				Average life span (T)	
$m_0(yr^{-1})$	α	$\omega(yr^{-1})$	*% senescent death* (P_S)	*Years*	*% nonsenescent*
.01	7.8E−6	.053	78.6	21.4	21.4
.02	2.1E−5	.068	69.0	15.5	31.0
.05	8.2E−5	.095	51.9	9.6	48.2
.10	2.2E−4	.122	36.6	6.4	63.7
.20	6.2E−4	.158	19.5	3.9	78.9
.50	2.4E−3	.220	7.6	1.86	92.9
1.00	6.5E−3	.284	2.4	.98	98.1

Note: Values of α and ω are those predicted by the regression equation relating α to m_0 with β = 3 (see fig. 6).

atively long-lived individuals provided that studies on such populations are long enough to include large numbers of old individuals. Indeed, this result clears up an enigma evident in the data of Botkin and Miller (1974). These authors compared maximum observed longevities with predicted longevity in a population of a particular size and age-independent (nonsenescent) mortality rate. They found reasonable correspondence between observed and predicted values in species with high adult mortality rates, which is consistent with little senescent death. In species with low adult mortality, however, observed maximum life spans were far short of the nonsenescent prediction, suggesting that mortality rate increased in old age.

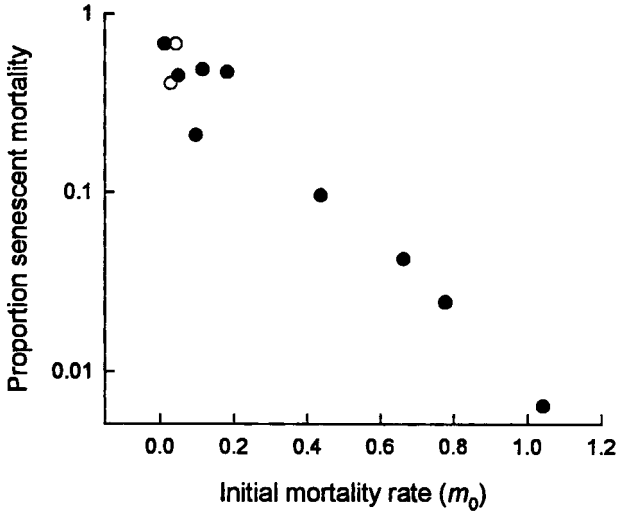

Figure 7: Relationship between the proportion of mortality caused by senescence (P_S) and the initial mortality rate (m_0) in a population for samples in which Weibull equations were fitted with β as a free parameter (table 2). Solid symbols represent species of bird; open symbols, species of mammal.

Discussion

For a particular intrinsically determined schedule of senescent mortality defined by α and β, the proportion of deaths resulting from aging would increase if initial extrinsic mortality were to decrease. In response to such increasing selection resulting from factors that cause aging-related deaths, populations should evolve modifications that reduce senescent mortality at a given age, resulting in lower α, β, or both. The results of this analysis suggest, however, that although such evolutionary responses do occur, they are not sufficient to offset fully the larger proportions of individuals that survive to old age and suffer deaths hastened by physiological decline. As a result, a larger proportion of individuals succumbs to factors related to physiological aging in populations with older age distributions. The inability of evolutionary re-

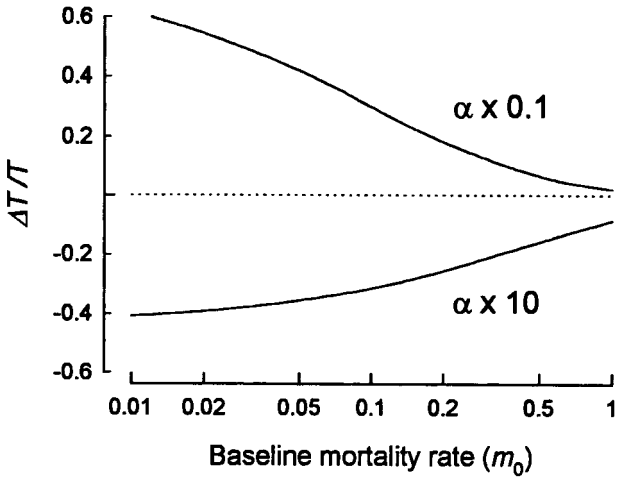

Figure 8: Fractional change in the average life span (T) of individuals in a population resulting from a 10-fold increase or decrease in the value of α (1.8-fold change in ω) when β = 3 and values of α are determined by the regression in figure 6.

sponses to fully remedy high proportions of senescent mortality could be due to (1) weaker relative strength of selection to retard senescence at lower initial mortality rates, which clearly is false, (2) increased costs of delaying senescence at progressively older ages, which would be considered as an expression of negative antagonistic pleiotropy, or (3) lack of suitable genetic variation to enhance mechanisms that reduce senescence at extreme old age, that is, a physiological constraint. As pointed out by Mueller and Rose (1996), at some point selection becomes weaker than the forces of mutation and genetic drift and adaptive evolutionary responses cease (mechanism 4). However, this point occurs well after 99% of individuals in a population have died, and the mechanism therefore is not relevant to mechanisms of senescent death occurring up to this point.

The strength of selection on changes in rate of senescence (α) can be estimated from the effect of such changes on fitness. In the absence of information on the age-specific schedule of fecundity, a relative measure of fitness, which assumes stable population size and constant fecundity with respect to age, is the average life span (T) of individuals in a population. For $m_0 = 0.01$ and $\alpha = 7.8 \times 10^{-6}$, $T = 21.3$ yr. Decreasing and increasing α by a factor of 10 (equivalently, ω by a factor of 1.8 when $\beta = 3$) changes T by +62% and −41%, respectively. For $m_0 = 0.1$ and $\alpha = 2.2 \times 10^{-4}$ ($T = 6.4$ yr), changing α by factors of 0.1 and 10 alters T by +29% and −31%; for $m_0 = 1.0$ and $\alpha = 6.5 \times 10^{-3}$ ($T = 1.0$ yr), these values are +2% and −9% (fig. 8). Thus, the change in life span that would be associated with comparable factorial changes in the rate of senescence (α) is larger for populations with lower initial mortality rates. Accordingly, populations with lower initial mortality rates experience potentially stronger selection to further postpone senescence than do populations with higher initial mortality rates, contrary to proposition 1 above. This implies that delaying senescence is more difficult to accomplish at older than at younger ages and that either the costs for greatly extending natural life span are prohibitive (proposition 2) or the possibilities for doing so regardless of cost (proposition 3) are severely limited.

This apparent constraint on the evolutionary response sheds some light on the genetic bases of variation among species in rate of senescence. Senescence is generally thought to be caused by one or more of three mechanisms (Rose 1991; Ricklefs and Finch 1995): (*a*) the accumulated effects of deleterious mutations having their expression at increasing age (Edney and Gill 1968; Mueller 1987; Partridge and Barton 1993*b*); (*b*) negative effects of the expression in old age of genes that enhance survival and/or reproduction at younger ages (antagonistic pleiotropy; Williams 1957; Rose 1991); and (*c*) environmental factors and use (wear and tear), with variation in rate of senescence among populations and species being modified by genetically controlled preventive and repair mechanisms (Kirkwood and Holliday 1979; Kirkwood 1990). The last type of mechanism, often referred to as the "disposable soma" theory of aging, creates an apparent antagonistic pleiotropy when such mechanisms impose costs that reduce the fecundity or survival of young individuals.

Mutations with deleterious effects at old age (mechanism *a*) are removed by selection in proportion to the variance in fitness that they produce within populations. In mutation-selection models, evolutionary equilibria are achieved that balance the rates of mutation and selective removal of mutants (Ewens 1979; Charlesworth 1994). Thus, unless the rate of appearance of new mutants increased (i.e., more loci expressed or higher mutation rates) as a function of age of expression or in populations with older age structures, the total fitness consequence of senescence should be similar for all populations regardless of age structure. This clearly is not the case because the proportion of senescence-related deaths is greater in populations having average age of individuals. Some form of antagonistic pleiotropy is thus implicated in the evolutionary modification of aging in birds and mammals.

According to hypothesis *b,* genes that accelerate aging must confer commensurate fitness benefits early in life. Because populations with older age structure suffer proportionately more deaths from aging-related causes than do populations with younger age structure, the total benefit conferred by antagonistically pleiotropic genes early in life would have to be greater in populations with older compared with younger age structures. Few plausible candidates for antagonistically pleiotropic genes have been recognized, and the physiological mechanisms connecting opposing early and late effects on fitness are not well characterized (Rose 1991; Ricklefs and Finch 1995; Tatar et al. 1996). Furthermore, there is no reason to suspect that the earlier beneficial effects of antagonistically pleiotropic genes should increase consistently with increasing age of expression of their later deleterious effects. Thus, the observation reported here of increasing senescence-related mortality in populations with progressively older age structure (lower initial mortality rate, m_0) weighs against two popular hypotheses (mutation accumulation and antagonistic genetic pleiotropy) for the genetic basis of aging in birds and mammals.

The repair hypothesis of senescence (*c*) could be consistent with observed patterns of aging-related mortality if genetic variation for repair capabilities decreased with increasing age of expression of physiological deterioration. That is, the lesser damage experienced by younger individuals may be more readily prevented or repaired

than the more serious damage suffered by older individuals. As physiological deterioration progresses further, it may simply be more difficult, or more costly, to prevent or reverse. The key difference between this hypothesis and both *a* and *b* above is that in case *c* the degenerative bases of senescence have environmental or somatic causes, rather than being genetically determined. This relaxes the tight correlation between genetic variation and senescent mortality implicit in *a* and *b*. As a result, evolutionary responses to selection generated by aging-related deaths may be increasingly limited at older age by lack of genetically determined physiological or biochemical avenues of repair and prevention, that is, genetic variation that could create genetic variance in fitness and hence selection. The effects of aging simply become increasingly irreversible.

Regardless of the underlying genetic mechanisms that control the rate of physiological senescence, the fact that senescent mortality increases as initial adult mortality decreases and thus creates an older age structure implies a strong constraint on the improvement of life span in long-lived organisms, including humans. If populations of long-lived organisms lacked genetic variation for mechanisms that would further retard aging and increase life span, the most sensible approaches to aging as a public health problem would include behavioral control of wear and tear and therapeutic remedy of damage, rather than manipulation of gene products and engineering of the genetic material itself.

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APPENDIX A

Table A1: Parameters of the Weibull equation fitted to the relationship between mortality rate and age in natural populations of birds

Species	*Sex*	N	*Oldest age*	m_0		α		ω	*Mass (kg)*	*Reference notes*
				Coefficient	*SE*	*Coefficient*	*SE*			
Anseriformes:										
Anatidae:										
Cygnus columbianus	M	32	18	.036	.030	3.89E−5	1.33E−5	.079	6.4	1
C. columbianus	F	27	18	.009	.055	1.05E−4	.29E−4	.101	5.7	1
Ciconiiformes:										
Laridae:										
Larus canus	MF	5,422	18	.098	.010	2.99E−5	.35E−5	.074	.404	2
Rissa tridactyla	M	>160	>12	.196	.014	2.31E−5	1.10E−5	.069	.421	3
R. tridactyla	F	>181	>12	.135	.022	3.02E−5	1.72E−5	.074	.393	3
Accipitridae:										
Accipiter nisus	F	· · ·	>8	.201	.089	1.84E−3	.32E−4	.207	.202	4
Spheniscidae:										
Eudyptula minor	MF	246	16	.195	.077	3.13E−4	.75E−4	.133	1.105	5
Procellariidae:										
Puffinus tenuirostris	MF	· · ·	25	.080	.010	1.12E−4	.02E−4	.103	.543	6
Diomedia exulans	MF	1,254	>28	.020	.004	2.12E−6	.30E−6	.038	7.65	7
Passeriformes:										
Muscicapidae:										
Ficedula hypoleuca	M	953	7	.748	.034	3.35E−3	.21E−3	.241	.012	8
F. hypoleuca	F	1,298	7	.656	.021	3.07E−3	.13E−3	.235	.012	8
Paridae:										
Parus major	M	· · ·	7	.756	.085	3.43E−3	.48E−3	.242	.019	9
P. major	F	· · ·	7	.989	.103	2.93E−3	.59E−3	.233	.019	9
Parus atricapillus	MF	~150	6	.361	.059	1.10E−3	.50E−3	.182	.011	10

Table A1 (*Continued*)

Species	Sex	N	Oldest age	m_0		α		ω	Mass (kg)	Reference notes
				Coefficient	SE	Coefficient	SE			
Sylviidae:										
Turdoides squamiceps	F	71	6	.297	.054	2.58E−3	.93E−3	.225	.074	11
T. squamiceps	M	69	7	.097	.006	1.99E−3	.15E−3	.211	.074	11
Fringillidae:										
Passerina cyanea (Reserve)	M	184	6	.429	.092	3.35E−3	.87E−3	.241	.015	12
P. cyanea (Niles)	M	173	6	.454	.016	1.71E−3	.14E−3	.203	.015	12

Note: Taxonomy based on Monroe and Sibley (1993). Body masses from Dunning (1993), unless otherwise noted. "Oldest age" refers to the oldest age class used in fitting the Weibull parameters. References and notes are as follows: (1) Scott (1988), table 14.2; survival from ages 7 to 18 yr. (2) Rattiste and Lilleleht (1987); from fourth year of breeding (approximately 6 yr) to sixteenth year of breeding; oldest individual in population was 24 yr. (3) Coulson and Wooller (1976); survival calculated for age classes based on number of years of breeding experience: 1, 2, 3, 4–5, 6–7, and 8–17 yr; age at first reproduction about 4 yr. (4) Newton (1989*b*), figure 17.1; 3–8+ yr (survival lower in years 1 and 2); 465 bird-years. (5) Dann and Cullen (1990); life table constructed from ages of known-age birds last recovered dead (table 3.1); ages 4–15 yr included; ages 9–10 and 11–16 combined. (6) Bradley et al. (1989); Wooller et al. (1989), table 24.2; data for years 3 to 20–27 after first breeding, which begins at an average age of 7 yr; sample size 2,127 bird-years, 126–268 individuals per age class. (7) Weimerskirch (1992); survival rates calculated from recaptures of 998 individuals aged 4–22 yr, and 256 individuals older than 28 yr; age of individuals in the latter group unknown because banding program was only 24 yr old; Weibull function fitted to survival for age classes 10–12, 13–15, 16–22, and >28 yr. (8) Gustafsson (1989), table 4.1; based on years 1–7; breeders only; 1,946 bird-years for males and 1,504 bird-years for females. (9) McCleery and Perrins (1989). (10) Loery et al. (1987). (11) Zahavi (1989), table 16.2; ages 1–6 for females and 1–7 for males, includes hatchlings banded in 1979–1983, whose survival was followed through 1989; birds older than 6 and 7 yr, respectively, exhibited increased survival rates, but samples were only 20 and 22 bird-years, respectively; adult mass 64–83 g (Cramp and Perrins 1993). (12) Payne (1989), table 10.1; ages 2–6 yr for Reserve population (first-year survival low) and 1–6 for Niles population; females showed no decrease in survival rate through 6 yr.

Table A2: Parameters of the Weibull equation fitted to the relationship between mortality rate and age in natural populations of mammals

Species	Sex	N	Oldest age	m_0		α		ω	Mass (kg)	Reference notes
				Coefficient	SE	Coefficient	SE			
Primates:										
Cercopithidae:										
Macaca mulatta	F	164	21	.087	.017	1.21E−5	.57E−5	.059	4.6	1
Hominidae:										
Pan troglodytes	F	22	37	.055	.011	4.22E−6	.83E−6	.045	39	2
Carnivora:										
Felidae:										
Panthera leo	F	412	16	.032	· · ·	2.52E−4	.47E−4	126	156.0	3
Mustelidae:										
Martes zibellina	?	2,598	12	.280	.076	5.42E−4	1.54E−4	.153	1.18	4
Otaridae:										
Callorhinus ursinus	F	8,797	23	.051	· · ·	8.60E−5	4.40E−5	.096	250.0	5
Phocidae:										
Phoca hispida	M	130	40	.063	.008	1.07E−5	.07E−5	.057	65.0	6
P. hispida	F	160	37	.032	.004	9.75E−6	.28E−6	.056	65.0	6
Proboscidea:										
Elephantidae:										
Loxodonta africana	MF	174	60	.040	· · ·	1.70E−6	.24E−6	.036	6,607.0	7
Perrisodactyla:										
Equidae:										
Equus burchelli	M	55	23	.057	.015	7.72E−5	.61E−5	.094	237.7	8
E. burchelli	F	49	20	.064	· · ·	1.24E−4	.21E−4	.106	· · ·	8

Table A2 (*Continued*)

Species	*Sex*	N	*Oldest age*	m_0		α		ω	*Mass (kg)*	*Reference notes*
				Coefficient	*SE*	*Coefficient*	*SE*			
Artiodactyla:										
Hippopotamidae:										
Hippopotamus amphibius	MF	207	43	.030	· · ·	5.28E−6	.62E−7	.048	1,200.0	9
Cervidae:										
Cervus elaphus	F	328	14	.050	.012	1.56E−4	.31E−4	.112	50.0	10
C. elaphus	F	· · ·	13	.025	· · ·	1.74E−4	.28E−4	.115	50.0	11
Rangifer tarandus	M	105	11	.076	· · ·	2.03E−3	.37E−3	.212	105.0	12
R. tarandus	F	26	16	.076	.029	1.52E−4	.18E−4	.111	105.0	12
Bovidae:										
Syncerus caffer	M	70	22	.026	· · ·	8.51E−5	.94E−5	.096	469.0	13
S. caffer	F	55	17	.026	· · ·	2.03E−4	.11E−4	.119	469.0	14
S. caffer	M	86	22	.092	.037	4.88E−5	.97E−5	.084	469.0	15
S. caffer	F	93	16	.030	· · ·	4.21E−4	.53E−4	.143	469.0	15
Ovis dalli dalli	M	130	13	.049	· · ·	8.35E−4	.72E−4	.170	73.0	16
O. dalli dalli	F	101	13	.107	.009	1.96E−4	.12E−4	.118	73.0	17
Rupicapra rupicapra	F	275	11	.219	.076	1.98E−3	.23E−4	.211	34.0	18
Kobus kob thomasi	M	170	8	.206	.057	3.41E−3	.54E−4	.242	117.5	19
Damaliscus korrigum	M	154	6	.166	.087	9.40E−3	1.40E−3	.311	98.0	20
D. korrigum	F	94	6	.182	.092	1.13E−2	.14E−2	.326	98.0	20
Rodentia:										
Sciuridae:										
Tamiasciurus hudsonicus	MF	412	8	.437	.175	2.05E−3	.64E−3	.213	.190	21
Lagomorpha:										
Leporidae:										
Sylvilagus floridanus	MF	1,300	3.3	1.225	.331	1.32E−1	.28E−1	.602	1.21	22

Note: Unless otherwise noted, life tables were generated from ages at death, usually from collections of skulls aged by tooth wear or horn development. Taxonomy based on Wilson and Reeder (1993). Body mass from Promislow (1991), unless noted otherwise. "Old age" refers to the oldest age class used in fitting the Weibull parameters. References and notes are as follows: (1) Sade et al. (1976); time-specific life table constructed from survival of females over 1-yr period; 1+ yr; age classes 7–9, 11–12, 13–15, 16–17, and 18–21 combined. (2) Teleki et al. (1976); compiled from ages at death; ages 3+; ages 3–5, 5–9, 9–13, 13–27, and 27–37 combined; adult mass from Eisenberg (1981), app. 2. (3) Packer et al. (1988); woodlands population only; sample size of 412 applies to males and females in three populations; ages 3+ included in analysis; NLIN estimate (SAS Institute 1985) of m_0 not significantly different from 0; m_0 estimated from average mortality rate between ages 3 and 8. (4) Monakhov (1983); based on estimated age structure of living population; ages 2–10. (5) Lander (1981); ages 8–23 yr; NLIN estimate of m_0 was significantly less than 0; m_0 was estimated as the mortality rate at 8–10 yr. (6) Helle (1980); life table constructed from age structure of a sample of seals caught in fishing nets; ages 9–30 yr. (7) Laws (1966); populations in Queen Elizabeth Park and Murchison Falls Park combined; regression estimate for m_0 negative and not significantly different from 0; m_0 estimated from the average annual mortality rate between 10 and 50 yr, which is approximately constant. (8) Spinage (1972); male data combined for age intervals 1–4, 4–9, 9–12, 12–15, and 15–20 yr; female data combined for age intervals 1–5, 5–9, 9–13, 13–16, and 16–19; NLIN estimate for female m_0 did not differ from 0; female m_0 estimated from survival between 1 and 5 yr as 0.105 yr^{-1}. (9) Laws (1968); data from Queen Elizabeth Park; shape parameter (β) for Weibull model is 4.00 $\pm$ 0.00063; for $\beta = 3$, regression estimate of m_0 was negative and did not differ significantly from 0; m_0 is estimated to be 0.03 yr^{-1} based on survival rates of young adults 8–17 yr. (10) Clutton-Brock (1984); milk hinds only, ages 3+; m_0 estimated from average mortality rate between 3 and 10 yr of age; yeld (nonbreeding) hinds show virtually no increase in mortality rate with age ($n = 290$). (11) Clutton-Brock et al. (1988); survivorship estimated from graph in figure 21.2; age 2+; NLIN estimate of m_0 not significantly different from 0; m_0 estimated as average mortality rate of hinds between ages 2 and 8 yr. Males exhibited no mortality between 2 and 8 yr; NLIN estimate of m_0 not different from 0, $\alpha = 4.50E{-}4 \pm 0.86E{-}4$, that is, almost three times the value for females. (12) Bie (1976); males, ages 3–11; regression estimate of m_0 negative and not significantly different from 0; m_0 estimated from data for females; females, ages 4–16; body mass from Eisenberg (1981), app. 2. (13) Spinage (1972); data combined for age intervals 1–7, 7–9, 9–11, 12–14, 14–17, and 17+ yr; NLIN estimate of m_0 (0.060 $\pm$ 0.060) did not differ significantly from 0; m_0 was estimated as the average mortality rate between ages 1 and 7 (0.026 yr^{-1}). (14) Sinclair (1977), data taken from figure 76; mortality patterns for males similar; NLIN estimate of m_0 did not differ significantly from 0; m_0 estimated as in (13). (15) Mertens (1985); ages 1+; average mortality rate of males between 1 and 7 yr is 0.037 yr^{-1}; m_0 for females estimated to be 0.03 yr^{-1}. (16) Hoefs and Bayer (1983); 130 skulls of males that died of natural mortality, ages 2+; Weibull shape parameter (β) estimated as 5.35 $\pm$ 0.08, for which $m_0 = 0.043 \pm 0.0$ and $\alpha = 3.11E{-}6 \pm 0.59E{-}6$; for $\beta = 3$, m_0 estimated from survival between 2 and 5 yr as NLIN estimate of m_0 was negative. (17) Simmons et al. (1984); life table constructed from age structure of sheep sampled by shooting. (18) Caughley (1970); based on estimated age structure of living population; ages 1+. (19) Mertens (1985); ages 1+. (20) Mertens (1985); ages 1+; species name changed to *lunatus* according to Wilson and Reeder (1993); adult mass from Dorst and Dandelot (1970); 200–300 lbs. (21) Davis and Sealander (1971); from 1 yr of age; Cree Lake population; sex ratio approximately even. (22) Lord (1961); mortality rates calculated from age structure of population by 4-mo period beginning at 1 mo; age estimated by weight of eye lens.

APPENDIX B

Comparison of Approaches to Estimating Aging Parameters

Unweighted nonlinear least squares (LS), weighted nonlinear least squares (WLS), and maximum likelihood (ML) approaches to estimating aging parameters are compared. For the LS approach, the data are the instantaneous mortality rates m_x estimated as the negative of the natural logarithm of the survival from time x to time $x + 1$. In the WLS approach, I weighted observations by the square root of the initial number of individuals in each age class, which is inversely related to the standard error of the estimate of the survival fraction. Calculations were done in the NLIN procedure of SAS (SAS Institute 1985).

For the ML approach, the data were the number of individuals living through each age class ($S(x)$) and the number dying in each age class ($D(x)$). The sum of $S(x)$ and $D(x)$ is the total number entering each age class ($N(x)$). We define $L(x)$ as the probability that an individual will live to the beginning of age class x, and $q(x)$ as the probability that a newborn individual will die during the age interval x to $x + 1$. In this case, $q(x) = L(x) - L(x + 1)$. Given a particular value of $q(x)$, the likelihood of a particular number of deaths in the interval x to $x + 1$ is

$$\text{Likelihood } (D(x)) = \frac{N(x)!}{S(x)!\,D(x)!} \times [q(x)^{D(x)} \cdot (1 - q(x))^{S(x)}]. \tag{B1}$$

The logarithm of this expression is

$$\begin{aligned} \ln(\text{likelihood } (D(x)) = {} & \ln(N(x)!) \\ & - \ln(S(x)!) - \ln(D(x)!) \\ & + D(x) \ln(q(x)) \\ & + S(x) \ln(1 - q(x)), \end{aligned} \tag{B2}$$

and the total log-likelihood is the sum of this expression over all age classes.

For the Weibull function, the fraction of individuals in the population dying during age interval x to $x + 1$ is

$$q(x) = 1 - \exp\left\{-m_0 - \frac{\alpha}{\beta}[(x + 1)^{\beta+1} - x^{\beta+1}]\right\}. \tag{B3}$$

Values of $N(x)!$, $D(x)!$, and $S(x)!$ were approximated by Stirling's formula

$$n! = e^{-n} n^n \sqrt{2\pi n}, \tag{B4}$$

and therefore

$$\ln(n!) = -n + n \ln(n) + \frac{1}{2}\ln(2\pi n). \tag{B5}$$

Log-likelihood functions were evaluated using the NLIN procedure of SAS with a weight equal to the variance in $D(x)$, that is, _WEIGHT_ $= 1/N(x) \cdot q(x) \cdot (1 - q(x))$, and a criterion (_LOSS_) function equal to the sum of the log-likelihood terms divided by their weights.

In general, parameter estimates obtained by the LS, WLS, and ML approaches did not differ markedly. This point is illustrated in table B1, where results for the three approaches are shown for female pied flycatchers and male Arabian babblers. In these comparisons, estimates of the initial mortality rates are nearly identical. Maximum likelihood tended to produce higher shape parameters (β), but lower values of α resulted in similar estimated values of the aging parameter ω. Other similar comparisons revealed little difference in parameter estimates of Weibull functions, particularly with respect to the value of m_0 and the secondary parameter ω.

Gompertz equations were also fitted by ML to the data for female pied flycatchers and to a sample of male Dall sheep *Ovis dalli.* The values for the convergence functions (_LOSS_) for each of the species were −20.0 (Weibull: $F = 2083$, df = 3, 4) and −23.0 (Gompertz: $F = 960$, df = 2, 5) for *Ficedula hypoleuca,* and −20.1 (Weibull: $F = 310$, df = 3, 7) and −21.4 (Gompertz: $F = 192$, df = 2, 8) for *O. dalli.* It is not surprising that the three-parameter Weibull equation provides a better fit to the data than the two-parameter Gompertz equation does in each case.

In order to examine the influence of sample size on estimates of the parameters of the Weibull equation, I performed three exercises patterned after the approach of Mueller et al. (1995). First, the data for female pied flycatchers were randomly rarified to one-half and one-quarter of the 1,504 bird-years in the original sample of data. This was accomplished by reducing the number of individuals in each class, randomly selecting individuals from the group that survived and the group that died. Weibull equations were fitted to the rarified data by ML. This was repeated 10 times for each level of rarefaction. Second, life tables were generated randomly using particular values of Weibull parameters, and the resulting numbers of surviving and dying individuals in each age class were fitted using an ML approach. Ten life tables were generated with each of 250, 500, 1,000, 2,000, 5,000, and 10,000 individuals. Third, randomly generated life tables having either 1,000 or 10,000 individuals were generated using parameters of the Weibull equation, and the resulting data were fitted by Weibull equations using LS, WLS, and ML approaches.

Table B1: Least squares (LS), weighted least squares (WLS), and maximum likelihood (ML) estimates of Weibull parameters for female pied flycatchers (*Ficedula hypoleuca*) and male Arabian babblers (*Turdoides squamiceps*)

	m_0		α		β		
Species and fit	*Estimate*	*SE*	*Estimate*	*SE*	*Estimate*	*SE*	ω
Ficedula hypoleuca:							
LS	.664	.032	2.53E−3	1.64E−3	3.14	.36	.232
WLS	.667	.023	2.03E−3	1.53E−3	3.22	.40	.230
ML	.663	.032	.75E−3	1.55E−3	3.62	1.16	.211
Turdoides squamiceps:							
LS	.115	.031	.84E−3	1.68E−3	3.39	1.10	.199
WLS	.114	.029	.96E−3	1.87E−3	3.31	1.09	.199
ML	.113	.038	.35E−3	1.46E−3	3.71	2.29	.185

Note: For details of the data sets, see appendix A, table A1.

With the rarified data sets for the pied flycatcher, ML fitting often failed to converge on a stable solution. In table B2, which summarizes these results, convergence is tabulated under three categories: "yes," meaning that the solution converged within 50 iterations; "no," meaning that the algorithm was approaching a stable solution after 50 iterations but had not reached it; and "failed," meaning that one or more of the estimated parameters had become negative during the course of the iterations. In the case of a "no" outcome, the last set of parameters (iteration 50) was retained. In the case of a "failed" outcome, the set of parameters corresponding to the lowest value of the _LOSS_ function during the iterations was retained.

Table B2 indicates considerable stability in m_0 with respect to rarifaction but a decrease in α, a corresponding increase in β, and a slight decrease in ω, particularly compared with the variation among species in this analysis.

In the second set of simulations, I used Weibull parameter values of $m_0 = 0.6$, $\alpha = 0.002$, and $\beta = 3$. These yield a calculated value of $\omega = 0.211$. Either 1,000 or 10,000 individuals were simulated up to a maximum age of 10 yr. Each of these cases was repeated 10 times. When either $D(x)$ or $S(x)$ for an age class was 0, that age class was dropped from the data set. The different categories of convergence success were handled as above. Estimated parameters were divided by the parameter values used to generate the data, giving a relative value normalized to 1 (fig. B1). Down to a sample of 500 individuals, parameters estimated by ML were very close to the generating parameter values, although the standard deviations of the data were large. With 250 individuals, the estimate of the shape parameter β (3.80 $\pm$ 0.32 SE) significantly exceeded the generating value (3). This was compensated by a reduced value of α (0.00131 $\pm$ 0.00054 SE), so that the estimated value of ω (0.193 $\pm$ 0.012 SE) was not very different from the generating value. Convergence success with the ML approach fell from $^9/_{10}$ at 10,000 individuals to $^5/_{10}$ at 1,000, and $^2/_{10}$ at 250 individuals.

The third set of simulations, with either 1,000 or 10,000 individuals per data set were fitted by LS, WLS, and ML approaches. The first two approaches, LS and WLS, consistently overestimated the value of β. However,

Table B2: Maximum likelihood estimates of Weibull parameters for female pied flycatcher data rarified to one-half and one-quarter

	Convergence			*Mean of estimates (SD)*			
	Yes	*No*	*Failed*	m_0	α	β	ω
Unreduced	. . .	. . .	. . .	.663	.75E−3	3.62	.210
Reduced to .5	5	5	0	.651	4.81E−3	3.38	.213
				(.030)	(11.1E−3)	(.97)	(.014)
Reduced to .25	3	4	3	.691	1.75E−3	3.68	.180
				(.065)	(3.17E−3)	(1.36)	(.041)

Note: Mean and standard deviation (in parentheses) based on 10 repetitions.

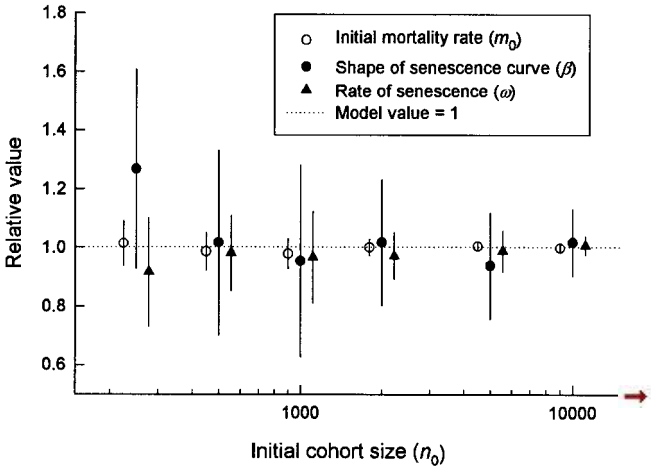

Figure B1: Maximum likelihood estimates of parameters m_0 and β, and the calculated rate of aging ω, as a function of sample size for simulated data sets using Weibull survival functions. Estimated values are shown relative to the parameter values used to generate the data sets ($m_0 = 0.6$, $\alpha = 0.002$, $\beta = 3$, $\omega = 0.211$). Each simulation was repeated 10 times. Estimated values are reported as means ± 1 SD.

with respect to the critical aging parameter ω, all three estimation approaches performed reasonably well, with the biases in ω averaging less than 20% in all cases, and even less for ML approaches at the smaller sample size (fig. B2). With samples of 100 individuals (results not shown), none of the estimating approaches led to stable solutions with this set of parameter values.

The basic conclusion from this exercise is that while ML produces less biased estimates of parameters, this performance is only marginally better than LS and WLS approaches. Biases in β and ω are generally small compared with variation among species. Values of m_0 are estimated well regardless of the sample size and approach taken. The general failure of estimation at small sample sizes, regardless of the approach, explains in part why so many data sets fail to show evidence of actuarial aging but also cautions against using parameter estimates from such small populations.

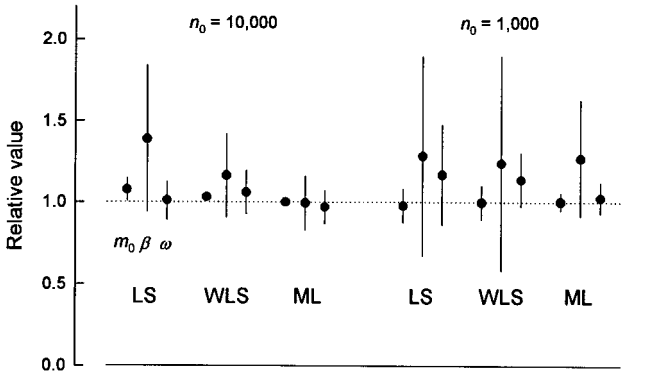

Figure B2: Least squares (*LS*), weighted least squares (*WLS*), and maximum likelihood (*ML*) estimates of parameters m_0 and β, and the calculated rate of aging ω, for simulated data sets of 10,000 and 1,000 individuals using Weibull survival functions. Estimated values are shown relative to the parameter values used to generate the data sets ($m_0 = 0.6$, $\alpha = 0.002$, $\beta = 3$, $\omega = 0.211$). Each simulation was repeated 10 times. Estimated values are reported as means ± 1 SD.

Literature Cited

Abrams, P. A. 1991. The fitness costs of senescence: the evolutionary importance of events in early adult life. Evolutionary Ecology 5:343–360.

———. 1993. Does increased mortality favor the evolution of more rapid senescence? Evolution 47:877–887.

Abrams, P. A., and D. Ludwig. 1995. Optimality theory, Gompertz' law, and the disposable soma theory of senescence. Evolution 49:1055–1066.

Austad, S. N., and K. E. Fischer. 1991. Mammalian aging, metabolism, and ecology: evidence from the bats and marsupials. Journal of Gerontology: Biological Sciences 46:B47–B53.

Bie, S. de 1976. Survivorship in the Svalbard reindeer (*Rangifer tarandus platyrhynchus* Vrolik) on Edgeøya, Svalbard. Norsk Polarinstitutt Årbok 1976:249–270.

Blarer, A., M. Doebeli, and S. C. Stearns. 1995. Diagnosing senescence: inferring evolutionary causes from phenotypic patterns can be misleading. Proceedings of the Royal Society of London B, Biological Sciences 262:305–312.

Botkin, D. B., and R. S. Miller. 1974. Mortality rates and survival of birds. American Naturalist 108:181–192.

Bradley, J. S., R. D. Wooller, I. J. Skira, and D. L. Serventy. 1989. Age-dependent survival of breeding short-tailed shearwaters *Puffinus tenuirostris*. Journal of Animal Ecology 58:175–188.

Calder, W. A., III. 1983. Body size, mortality, and longevity. Journal of Theoretical Biology 102:135–144.

Carey, J. R., P. Liedo, D. Orozco, and J. W. Vaupel. 1992. Slowing of mortality rates at older ages in large medfly cohorts. Science (Washington, D.C.) 258: 457–461.

Caughley, G. 1970. Population statistics of chamois. Mammalia 34:194–199.

———. 1977. Analysis of vertebrate populations. Wiley, New York.

Charlesworth, B. 1993. Evolutionary mechanisms of senescence. Genetica 91:11–19.

———. 1994. Evolution in age-structured populations. 2d ed. Cambridge University Press, Cambridge.

Charlesworth, B., and J. A. Leon. 1976. The relations of

reproductive effort to age. American Naturalist 110: 449–459.

Clum, N. J. 1995. Effects of aging and mate retention on reproductive success of captive female peregrine falcons. American Zoologist 35:329–339.

Clutton-Brock, T. H. 1984. Reproductive effort and terminal investment in iteroparous animals. American Naturalist 123:212–229.

Clutton-Brock, T. H., S. D. Albon, and F. E. Guinness. 1988. Reproductive success in male and female red deer. Pages 325–343 *in* T. H. Clutton-Brock, ed. Reproductive success: studies of individual variation in contrasting breeding systems. University of Chicago Press, Chicago.

Comfort, A. 1962. Survival curves of some birds in the London Zoo. Ibis 104:115–117.

Coulson, J. C., and R. D. Wooller. 1976. Differential survival rates among breeding kittiwake gulls *Rissa tridactyla* (L.). Journal of Animal Ecology 45:205–214.

Cramp, S. 1980. Handbook of the birds of Europe, the Middle East, and North Africa: the birds of the Western Palearctic. Vol. 2. Hawks to bustards. Oxford University Press, Oxford.

Cramp, S., and C. M. Perrins. 1993. Handbook of the birds of Europe, the Middle East and North Africa: the birds of the Western Palearctic. Vol. 7. Flycatchers to shrikes. Oxford University Press, Oxford.

Dann, P., and J. M. Cullen. 1990. Survival, patterns of reproduction, and lifetime reproductive output in little blue penguins (*Eudyptula minor*) on Phillip Island, Victoria, Australia. Pages 63–84 *in* L. S. Davis and J. T. Darby, eds. Penguin biology. Academic Press, San Diego, Calif.

Davis, D. W., and J. A. Sealander. 1971. Sex ratio and age structure in two red squirrel populations in northern Saskatchewan. Canadian Field-Naturalist 85:303–308.

Dorst, J., and P. Dandelot. 1970. A field guide to the larger mammals of Africa. Collins, London.

Dunning, J. B., Jr. 1993. CRC handbook of avian body masses. CRC, Boca Raton, Fla.

Eakin, T., R. Shouman, Y. Qi, G. Liu, and M. Witten. 1995. Estimating parametric survival model parameters in gerontological aging studies: methodological problems and insights. Journal of Gerontology: Biological Sciences 50A:B166–B176.

Edney, E. B., and R. W. Gill. 1968. Evolution of senescence and specific longevity. Nature (London) 220:281–282.

Eisenberg, J. F. 1981. The mammalian radiations: an analysis of trends in evolution, adaptation, and behavior. University of Chicago Press, Chicago.

Ewens, W. J. 1979. Mathematical population genetics. Springer, Berlin.

Finch, C. E. 1990. Longevity, senescence, and the genome. University of Chicago Press, Chicago.

Finch, C. E., and R. E. Tanzi. 1997. Genetics of aging. Science (Washington, D.C.) 278:407–411.

Forslund, P., and K. Larsson. 1992. Age-related reproductive success in the barnacle goose. Journal of Animal Ecology 61:195–204.

Forslund, P., and T. Pärt. 1995. Age and reproduction in birds—hypotheses and tests. Trends in Ecology & Evolution 10:374–378.

Fukui, H. H., L. Xiu, and J. W. Curtsinger. 1993. Slowing of age-specific mortality rates in *Drosophila melanogaster*. Experimental Gerontology 28:585–599.

Fukui, H. H., L. Ackert, and J. W. Curtsinger. 1996. Deceleration of age-specific mortality rates in chromosomal homozygotes and heterozygotes of *Drosophila melanogaster*. Experimental Gerontology 31:517–531.

Gavrilov, L. A., and N. S. Gavrilova. 1991. The biology of life span: a quantitative approach. Harwood Academic, New York.

Gustafsson, L. 1989. Pied flycatcher. Pages 55–74 *in* I. Newton, ed. Lifetime reproduction in birds. Academic Press, London.

Hamilton, W. D. 1966. The moulding of senescence by natural selection. Journal of Theoretical Biology 12: 12–45.

Harvey, P. H., and M. S. Pagel. 1991. The comparative method in evolutionary biology. Cambridge University Press, Cambridge.

Helle, E. 1980. Age structure and sex ratio of the ringed seal *Phoca (Pusa) hispida* Schreber population in the Bothnian Bay, northern Baltic Sea. Zeitschrift für Säugetierkunde 45:310–317.

Henny, C. J. 1972. An analysis of the population dynamics of selected avian species. U.S. Fish and Wildlife Service Wildlife Research 1:1–99.

Hoefs, M., and M. Bayer. 1983. Demographic characteristics of an unhunted Dall sheep (*Ovis dalli dalli*) population in southwest Yukon, Canada. Canadian Journal of Zoology 61:1346–1357.

Holmes, D. J., and S. N. Austad. 1995*a*. Birds as animal models for the comparative biology of aging: a prospectus. Journal of Gerontology: Biological Sciences 50A:B59–B66.

———. 1995*b*. The evolution of avian senescence patterns: implications for understanding primary aging processes. American Zoologist 35:307–317.

Keller, L., and M. Genoud. 1997. Extraordinary lifespans in ants: a test of evolutionary theories of ageing. Nature (London) 389:958–960.

Kirkwood, T. B. L. 1990. The disposable soma theory of aging. Pages 9–19 *in* D. E. Harrison, ed. Genetic effects on aging. Telford, Caldwell, N.J.

Kirkwood, T. B. L., and R. Holliday. 1979. The evolution of ageing and longevity. Proceedings of the Royal Society of London B, Biological Sciences 205:531–546.

Lander, R. H. 1981. A life table and biomass estimate for Alaskan fur seals. Fisheries Research 1:55–70.

Laws, R. M. 1966. Age criteria for the African elephant, *Loxodonta a. africana.* East African Wildlife Journal 4: 1–37.

———. 1968. Dentition and ageing of the hippopotamus. East African Wildlife Journal 6:19–52.

LeBreton, J.-D., K. P. Burnham, J. Clobert, and D. R. Anderson. 1992. Modeling survival and testing biological hypotheses using marked animals: a unified approach with case studies. Ecological Monographs 62: 67–118.

Loery, G., K. H. Pollock, J. D. Nichols, and J. E. Hines. 1987. Age-specificity of black-capped chickadee survival rates: analysis of capture-recapture data. Ecology 68:1038–1044.

Lord, R. D., Jr. 1961. Mortality rates of cottontail rabbits. Journal of Wildlife Management 25:33–40.

McCleery, R. H., and C. M. Perrins. 1989. Great tit. Pages 35–54 *in* I. Newton, ed. Lifetime reproduction in birds. Academic Press, London.

McNamara, J. M., and A. I. Houston. 1996. State-dependent life histories. Nature (London) 380:215–221.

Medawar, P. B. 1952. An unsolved problem in biology. H. K. Lewis, London.

Mertens, H. 1985. Structures de population et tables de survie des buffles, topis et cobs de Buffon au Parc National des Virunga, Zaire. Revue d'Écologie la Terre et la Vie 40:33–51.

Monakhov, V. G. 1983. Age structure of the sable (*Martes zibellina*) populations [in Russian]. Zoologicheskii Zhurnal 62:1398–1406.

Monroe, B. L., Jr., and C. G. Sibley. 1993. A world checklist of birds. Yale University Press, New Haven, Conn.

Mueller, L. D. 1987. Evolution of accelerated senescence in laboratory populations of *Drosophila.* Proceedings of the National Academy of Sciences of the USA 84: 1974–1977.

Mueller, L. D., and M. R. Rose. 1996. Evolutionary theory predicts late-life mortality plateaus. Proceedings of the National Academy of Sciences of the USA 93: 15249–15253.

Mueller, L. D., T. J. Nusbaum, and M. R. Rose. 1995. The Gompertz equation as a predictive tool in demography. Experimental Gerontology 30:553–569.

Newton, I. 1989*a.* Lifetime reproduction in birds. Academic Press, London.

———. 1989*b.* Sparrowhawk. Pages 279–296 *in* I. Newton, ed. Lifetime reproduction in birds. Academic Press, London.

Newton, I., and P. Rothery. 1997. Senescence and reproductive value in sparrowhawks. Ecology 78:1000–1008.

Nichols, J. D., J. E. Hines, and P. Blums. 1997. Tests for senescent decline in annual survival probabilities of common pochards, *Aythya ferina.* Ecology 78: 1009–1018.

Ottinger, M. A. 1991. Neuroendocrine and behavioral determinants of reproductive aging. Critical Reviews of Poultry Biology 3:131–142.

Ottinger, M. A., I. C. T. Nisbet, and C. E. Finch. 1995. Aging and reproduction: comparative endocrinology of the common tern and Japanese quail. American Zoologist 35:299–306.

Packer, C., L. Herbst, A. E. Pusey, J. D. Bygott, J. P. Hanby, S. J. Cairns, and M. B. Mulder. 1988. Reproductive success of lions. Pages 363–383 *in* T. H. Clutton-Brock, ed. Reproductive success: studies of individual variation in contrasting breeding systems. University of Chicago Press, Chicago.

Partridge, L., and N. H. Barton. 1993*a.* Evolution of aging: testing the theory using *Drosophila.* Genetica 91: 89–98.

———. 1993*b.* Optimality, mutation and the evolution of ageing. Nature (London) 362:305–311.

Payne, R. B. 1989. Indigo bunting. Pages 153–172 *in* I. Newton, ed. Lifetime reproduction in birds. Academic Press, London.

Petersen, T. 1986. Estimating fully parametric hazard rate models with time-dependent covariates: use of maximum likelihood. Sociological Methods and Research 14:219–246.

Pianka, E. R., and W. S. Parker. 1975. Age-specific reproductive tactics. American Naturalist 109:453–464.

Promislow, D. E. L. 1991. Senescence in natural populations of mammals: a comparative study. Evolution 45: 1869–1887.

Promislow, D. E. L., M. Tatar, A. A. Khazaeli, and J. W. Curtsinger. 1996. Age-specific patterns of genetic variance in *Drosophila melanogaster.* I. Mortality. Genetics 143:839–848.

Pugesek, B. H., and K. L. Diem. 1990. The relationship between reproduction and survival in known-aged California gulls. Ecology 71:811–817.

Rattiste, K., and V. Lilleleht. 1987. Population ecology of the common gull *Larus canus* in Estonia. Ornis Fennica 64:179–186.

Ricklefs, R. E., and C. E. Finch. 1995. Aging: a natural history. Scientific American Library, New York.

Ricklefs, R. E., and J. M. Starck. 1996. Applications of phylogenetically independent contrasts: a mixed progress report. Oikos 77:167–172.

Rose, M. R. 1984. Laboratory evolution of postponed senescence in *Drosophila melanogaster.* Evolution 38: 1004–1010.

———. 1991. Evolutionary biology of aging. Oxford University Press, New York.

Rose, M. R., and C. E. Finch. 1993. The Janiform genetics of aging. Genetica 91:3–10.

Sade, D. S., K. Cushing, P. Cushing, J. Dunaif, A. Figueroa, J. R. Kaplan, C. Lauer, D. Rhodes, and J. Schneider. 1976. Population dynamics in relation to social structure on Cayo Santiago. Yearbook of Physical Anthropology 20:253–262.

Sæther, B. E. 1990. Age specific variation in reproductive performance in birds. Current Ornithology 7:251–283.

SAS Institute. 1985. SAS user's guide: statistics, version 5 ed. SAS Institute, Cary, N.C.

Scott, D. K. 1988. Breeding success in Bewick's swans. Pages 220–236 *in* T. H. Clutton-Brock, ed. Reproductive success: studies of individual variation in contrasting breeding systems. University of Chicago Press, Chicago.

Shmookler Reis, R. J., and R. H. Ebert II. 1996. Genetics of aging: current animal models. Experimental Gerontology 31:69–81.

Simmons, N. M., M. B. Bayer, and L. O. Sinkey. 1984. Demography of Dall's sheep in the Mackenzie Mountains, Northwest Territories. Journal of Wildlife Management 48:156–162.

Sinclair, A. R. E. 1977. The African buffalo: a study of resource limitation of populations. University of Chicago Press, Chicago.

Sohal, R. S. 1986. The rate of living theory: a contemporary interpretation. Pages 23–44 *in* R. S. Sohal and K. G. Collatz, eds. Insect aging, strategies and mechanisms. Springer, Berlin.

Spinage, C. A. 1972. African ungulate life tables. Ecology 53:51–78.

Sternberg, H. 1989. Pied flycatcher. Pages 55–74 *in* I. Newton, ed. Lifelong reproduction in birds. Academic Press, London.

Tatar, M., D. E. L. Promislow, A. A. Khazaeli, and J. W. Curtsinger. 1996. Age-specific patterns of genetic variance in *Drosophila melanogaster*. II. Fecundity and its genetic covariance with age-specific mortality. Genetics 143:849–858.

Taylor, H. M., R. S. Gourley, C. E. Lawrence, and R. S. Kaplan. 1974. Natural selection of life history attributes: an analytical approach. Theoretical Population Biology 5:104–122.

Teleki, G., E. E. Hunt, Jr., and J. H. Pfifferling. 1976. Demographic observations (1963–1973) on the chimpanzees of Gombe National Park, Tanzania. Journal of Human Evolution 5:559–598.

Weimerskirch, H. 1992. Reproductive effort in long-lived birds: age-specific patterns of condition, reproduction and survival in the wandering albatross. Oikos 64: 464–473.

Williams, G. C. 1957. Pleiotropy, natural selection and the evolution of senescence. Evolution 11:398–411.

Wilson, D. E., and D. M. Reeder. 1993. Mammal species of the world: a taxonomic guide and geographical reference. Smithsonian Institution Press, Washington, D.C.

Wilson, D. L. 1994. The analysis of survival (mortality) data: fitting Gompertz, Weibull, and logistic functions. Mechanisms of Ageing and Development 74:15–33.

Wooller, R. D., J. S. Bradley, I. J. Skira, and D. L. Serventy. 1989. Short-tailed shearwater. Pages 405–417 *in* I. Newton, ed. Lifetime reproduction in birds. Academic Press, London.

Zahavi, A. 1989. Arabian babbler. Pages 253–275 *in* I. Newton, ed. Lifetime reproduction in birds. Academic Press, London.

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