Heritability of Longevity in Captive Populations of Nondomesticated Mammals and Birds

Robert E. Ricklefs and Carlos Daniel Cadena

1Department of Biology, University of Missouri, St. Louis.
2Departamento de Ciencias Biológicas, Universidad de los Andes, Bogotá, Colombia.

We used variance components analysis and offspring–parent regression to estimate the heritability of age at death in zoo populations of several species of mammals and birds. A meta-analysis over 14 species of mammals indicated a variance–component heritability of 0.53. More conservative regression estimates of heritability for the same species averaged 0.17. Offspring–parent regressions were not significant for any of eight species of birds. Heritabilities for data simulated with frailty and age-at-death models showed that sources of variation in age at death cannot be distinguished from observed heritabilities. The CV_A in age at death in six mammal species, based on parent–offspring regression, ranged from 0.20 to 0.54. The absence of substantial genetic variation for age at death in birds might be related to the stringency of flight, allowing for little variation in the optimization of life-history trade-offs.


Because age at death contributes to the lifetime reproductive success of individuals, genetic factors that influence individual life span are often under strong selection (1–4). These factors presumably determine attributes of the phenotype that influence the risk of death at all ages [e.g., (5,6)], as well as factors that influence the rate of physiological senescence and age-dependent mortality [see (7–13)]. Evolutionary diversification of the rate of aging among species (14–16), differences in life span in inbred lines of rats and mice [e.g., (17), pages 290–318], and response to artificial selection on life span or reproductive life span (3,18–23) demonstrate that risk of mortality and its increase with age are under genetic influence [e.g., (24)] and respond to selection. Indeed, most traits that influence performance exhibit genetic variation within populations [e.g., (25,26)]. This is true of the age at death, as well, which exhibits moderate heritability within the human population (27–30) and in populations of laboratory and domesticated animals (31).

Few studies have addressed genetic variation in the age at death in wild populations (26,32), in which aging-related mortality is overshadowed by time-dependent mortality caused by environmental factors, such as predation, inclement weather, and food shortages. Because age at death is directly correlated with lifetime reproductive success, hence evolutionary fitness, one expects selection to remove most genetic variation in natural populations. In addition, prevalent environmental causes of death and the general stochasticity of mortality suggest that environmental components of variation in age at death should be large and heritability correspondingly low. Gustafsson (33) famously demonstrated an inverse relationship between heritability and fitness contributions of traits in collared flycatchers (Ficedula albicollis) in a wild population in Sweden. Similar results have been reported for the great tit (Parus major) by McCleery and colleagues (34). Merila and Sheldon (35) pointed out, however, that although heritability of life span and lifetime reproductive success are often low, the genetic coefficient of variation (CV) can be relatively large because of the large phenotypic variance in these measures. Thus, although environmental variation obscures genetic variation in these traits, populations might nonetheless harbor considerable variation [for a review, see (36)].

Genetic influences over risk of death and its increase with age are poorly understood. Research on model systems has identified many gene mutations with marked effects (usually negative) on age at death or rate of biological aging (3,17[Chapter 7],24), but the extent to which such genes constitute genetic variation in age at death in natural populations is an open question. The nature of these genetic factors bears on discussions of the evolution of rate of aging in populations and, by inference, potential interventions to extend life span (2,29,37,38).

The strength of selection against deleterious genetic variation declines with age in most organisms as a smaller proportion of a population remains alive to express genetic effects on fitness (4,16,37,39,40). Based on this principle, evolutionary biologists have proposed several hypotheses concerning the genetic basis of aging and its accompanying increase in mortality rate: (a) accumulation of deleterious mutations expressed at older ages (mutation accumulation hypothesis) (3,40–42); (b) selection of genes conferring benefits early in life but having deleterious effects at older
ages (antagonistic pleiotropy hypothesis) (2, 43); and (c) selection of genes—or their expression—involves in the prevention and repair of damage to the organism as it ages (disposable soma hypothesis, a special type of antagonistic pleiotropy) (44–46).

Only the mutation accumulation hypothesis necessarily creates genetic variation within a population, and this variation is restricted to traits expressed late in life. Antagonistic pleiotropy and disposable soma theories would permit the accumulation of genetic variation through mutation when fitness landscapes were relatively flat or when different alleles favored fitness at different ages and heterozygotes produced the optimum phenotype. Such variation would primarily affect death at relatively old age, with effects on younger individuals influencing reproduction. In addition, genetic factors unrelated to aging undoubtedly influence mortality among young individuals in populations, before the onset of senescence, and such factors would have a disproportionate effect on lifetime reproductive success compared to influences of similar magnitude later in life. Because of these possibilities, inheritable genetic variation in the age at death does not readily differentiate between alternative genetic mechanisms for the evolution of aging.

Heritability ($h^2$), in its narrow sense, is the proportion of the variance in a trait attributable to additive genetic factors, i.e., differences in the expression of different alleles in the phenotype. Calculations of the heritability of life span in human, laboratory, and domesticated vertebrate populations are variable and often relatively low. At the higher end of the range of values, Klebanov and colleagues (47) estimated $h^2$ of age at death to be 0.44 and 0.62 in two populations of laboratory mice, but significant heritability in their study depended on crossing inbred laboratory strains with a wild-derived inbred strain of mice to introduce genetic variation. In addition, such mice, as in the case of domesticated animals and human populations, are maintained under controlled conditions that minimize environmentally caused mortality. As a result, although heritabilities might be significant and even substantial, the genetic standard deviation ($SD$) in life span as a percentage of the mean value is often small, and might be overwhelmed in natural populations exposed to extrinsic mortality factors.

Reported heritabilities for longevity in domesticated animals are consistently low: 0.06 (48), 0.14 (49), and 0.10 and 0.11 (50) for dairy cows; 0.11–0.27 (51) and 0.10–0.22 (52) for pigs; 0.076 for Boxer dogs (53); and 0.088 for domesticated rabbits (54). Presumably, strong selection and inbreeding might have eliminated some of the genetic variation present in the wild ancestors of these animals, although the domestic environment also reduces environmental variation in age at death. For example, a captive population of baboons recently derived from nature and maintained for medical studies exhibited a heritability in life span of 0.23 ± 0.08 (55). Estimates of heritability of life span in various studies on humans range from 0% to 32% in genealogical studies and from 10% to 50% in twin studies (8, 17 [Chapter 8], 56). In the latter case, heritabilities based on differences between twins raised apart and together in one study were 0% for men and 15% for women (57), indicating that maternal and common environment effects pose significant limitations to estimating heritability (56). The study by Mitchell and colleagues (58) of Old Order Amish longevity exceeding 30 years (i.e., excluding childhood and young-adult causes of death and emphasizing aging-dependent mortality) revealed a heritability of $0.25 ± 0.05$ standard error ($SE$).

As in the case of Mitchell and colleagues (58), several studies have attempted to isolate genetic factors affecting the rate of aging by analyzing longevities exceeding a certain cutoff point, whether an absolute age or a specified proportion of the deaths of all individuals in a population (47). In the most extreme cases, some studies of humans have analyzed only the oldest of the old and find substantial genetic determination of longevity (59, 60). In most cases, deaths at older ages continue to exhibit significant heritability, but it is difficult to separate the effects of genes with age-specific effects from those that influence risk throughout life.

Most information on heritability of life span in nonhuman species comes from highly selected domesticated or laboratory populations that experience little environmentally caused death. Few studies have addressed wild populations. Reale and Festa-Bianchet (61) estimated heritability of age at death in wild bighorn sheep (Ovis canadensis) from mother–daughter correlations to be $0.46 ± 0.24 SE$ ($p = .07$) and $0.32 ± 0.42 SE$ ($p = .22$) in two populations. Clearly, sample size is a major issue in heritability studies in wild populations. The study by Reale and Festa-Bianchet included only 85 mother–daughter pairs in one population and 43 in the second, and estimated heritabilities did not differ significantly from zero. Maternal effects also might have increased apparent heritability. Further analysis of this population based on added data and a new pedigree including paternal links showed no significant heritability for life span of either male or female individuals (62). Kruuk and colleagues (63) obtained a similar result (i.e., no significant heritability) for red deer (Cervus elaphus). Charmantier and colleagues (32) used a restricted maximum likelihood (REML) analysis based on genealogical data to estimate the heritability of the age at last reproduction in a large sample ($n = 648$) of female mute swans (Cygnus olor) to be marginally significant ($h^2 = 0.083 ± 0.049 SE$, $p < .05$). Thus, the few estimates of heritability of age at death in the wild are low or insignificant. However, environmental and stochastic components of variance in life span are large, and when significant heritabilities have been estimated the additive genetic coefficient of variation ($CV_A$) can be substantial.

In this study, we estimate heritabilities for longevity in captive (zoo) populations of nondomesticated mammals based on analysis of variance (ANOVA) components in the longevity of offspring resulting from matings of individual males (sires) with several females (dams) (64). We also estimate $h^2$ in mammals and birds from the regression of offspring age at death on the midparent age at death. With respect to nongenetic causes of death, zoos are intermediate between nature and the highly controlled environments of domesticated and laboratory animals. Although protected from predators and starvation, individuals suffer mortality from contagious disease, accidents, and social stress in the

\[ h^2 \text{ (mammals and birds) } = \frac{\text{ Variance in Age at Death (Midparent) - Variance in Age at Death (Individual)}}{\text{Variance in Age at Death (Midparent)}} \]

\[ h^2 \text{ (zoo populations) } = \frac{\text{ Variance in Age at Death (Midparent) - Variance in Age at Death (Individual)}}{\text{Variance in Age at Death (Midparent)}} \]
zoo environment. In addition to these empirical analyses, we simulated ages of death in family groups based on different genetic models of longevity to determine the range of parameters that produce heritabilities matching observed values.

Several caveats must be recognized with respect to analyses of data from captive populations maintained in zoos. First, populations are inevitably inbred to some extent, although zoos regularly exchange individuals expressly to avoid these effects, and most long-lived species are represented by relatively few generations of captive breeding. Second, all of the offspring in these analyses were born in zoos, as were most of their parents, and many influences from natural environments on growth, development, and health status are missing. Zoos are not natural environments, and the expression of genotype–environment interactions in age at death undoubtedly differ from those of wild populations and are, in any event, unknown to us. Third, although zoos provide excellent care and husbandry, they are not entirely safe environments owing to stresses of captivity and exposure to contagious diseases, and all zoos are not the same in this regard. Thus, causes of death in zoos, particularly among younger individuals, which constitute the bulk of the mortality in these studies, differ from those of wild populations. In addition, zoo effects can be important sources of variation in age at death; however, confounding effects are minimized in heritability studies because many individuals are traded among institutions and parents and their offspring often die in different locations. Nonetheless, we explicitly consider the zoo effect in some of our analyses. Finally, environmental components of variance influence heritability estimates, and, because zoo and natural environments differ substantially, estimates of heritability in zoo environments cannot be translated directly to natural settings. What can be determined, however, is the existence of genetic variation that is expressed in the captive environment and the magnitude of its effect on longevity.

Materials And Methods

Estimation of Heritability of Life Span in Zoo Populations

Although zoological institutions have not designed their mating schemes with the goal of estimating the heritability of life-history traits, they keep detailed genealogical records, and individual animals are often mated with several individuals of the opposite sex over their lifetimes. These data are maintained by the International Species Information System (ISIS) in Apple Valley, Minnesota (www.isis.org), and were generously provided to us by Dr. Nate Flesness with the permission of the ISIS Board of Directors. Access to this unique data resource allowed us to determine whether zoo populations of mammals and birds harbor additive genetic variation for life span.

We used variance component estimation in half-sib designs and parent–offspring regressions to assess the heritability of life span. We did not use the Animal Model (65) to estimate heritabilities because our data included only a single generation (parent and offspring), sample sizes were small, and the sampling design was relatively well balanced, which reduces the advantage to using the Animal Model (66). Estimates of the heritability of life-history traits (not including age at death) based on parent–offspring regression and the Animal Model presented in Table 1 of Kruuk (65) were significantly correlated \( r^2 = 0.47, n = 11, p = 0.02 \) although the parent–offspring regression estimates were 1.34 \( (\pm 0.48 SD) \) times greater than the animal model estimates.

To create hierarchical half-sib designs, we selected from the database only males that mated and produced multiple offspring with two or more females. This selection would produce some bias in age at death relative to the population as a whole if such males had multiple mates because they lived long enough to do so or were judged to be in good condition relative to other males. Although this might reduce genetic variance in age at death, it would not inflate estimates of heritability. Indeed, both sires and dams in this study on average outlived their offspring, many of which did not reproduce. Parent–offspring regressions were not restricted to males that bred with multiple females, hence the larger sample sizes in these analyses. In addition, although average age at death was greater in parents than in offspring, variances were similar enough that regression slopes were not biased in this way.

Based on the half-sib designs, we estimated variance components using REML. Models for REML analyses included sire, and dam nested within sire, as random effects. Negative variance components were not allowed by the analysis; however, sire effects were positive for all but one species; dam-within-sire effects were zero for 6 of 14 species. Based on variance components obtained for the effect of sire, dam, and error, we calculated \( h^2 \) values and their \( SE \) values using equations described by Falconer and Mackay (64). The \( h^2 \) and its \( SE \) were also estimated as the slope of the regression of the mean age at death of full sibs (or the age at death of single individuals for cases in which matings produced a single offspring) on the midparent value of age at death. When males were mated to more than one female, sires were represented in the regressions more than once, thus inflating the degrees of freedom. Accordingly, statistics based on regression analyses should be viewed conservatively. We additionally estimated the \( CV_A \) for studies with significant heritabilities as the square root of the

<table>
<thead>
<tr>
<th>Species</th>
<th>Minimum Age (y)</th>
<th>Sample</th>
<th>( \alpha )</th>
<th>( \beta )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Addax</td>
<td>2</td>
<td>101</td>
<td>0.0778 ± 0.0057</td>
<td>0.40 ± 0.37 ( \times 10^{-4} )</td>
</tr>
<tr>
<td>Lion</td>
<td>4</td>
<td>114</td>
<td>0.0527 ± 0.0089</td>
<td>1.13 ± 0.81 ( \times 10^{-4} )</td>
</tr>
</tbody>
</table>
additive genetic variance in age at death divided by the mean age at death. For all analyses, we excluded individuals dying before 1 year of age because many individuals die in zoos shortly after birth. These deaths are largely environmentally caused and cannot contribute to estimates of genetic variation in natural adult longevity. For some of the species with larger sample sizes, we explored the effect of considering the fates of individuals that survived to older ages (e.g., 2, 4, 6, 8, 10 years in lions, Panthera leo). All analyses were conducted by using SAS statistical software using Proc Mixed for REML and Proc GLM for regressions (SAS Institute, Cary, NC).

Obtaining precise estimates of heritability requires large sample sizes. Accordingly, many previous studies on the heritability of life span have used samples comprising thousands of individuals [e.g., (29)]. In contrast, the samples available in the ISIS database were relatively small (e.g., the largest number of sires on which REML analyses were based was 36). Consequently, the statistical power of our analyses was limited, and individual estimates of \( h^2 \) had large SE values. Therefore, to assess the general level of additive genetic variation for life span across species, we analyzed the combined estimates of \( h^2 \) obtained for individual species in meta-analyses. Using meta-analysis to obtain a single overall estimate of additive genetic variation across species assumes that heritabilities are uniform across populations, which probably is not valid [e.g., (67)]. However, our goal was not to obtain a generally applicable estimate of \( h^2 \). Rather, we use this statistical technique only as a tool to address the question of whether, despite large SE values and low statistical power owing to small sample sizes, life span is influenced by genetic factors. Thus, rather than focusing on the cross-species point estimate of heritability, we focus on the confidence intervals (CI) around it, specifically on whether they include zero.

For meta-analyses, we entered estimates of \( h^2 \) obtained for each species as effect sizes and their SE values as variances, and we summarized these parameters in the program MetaWin version 2 (68). Because the statistical distribution of \( h^2 \) is not well characterized, we did not know a priori whether the SE is an appropriate estimator of its sampling variance, so we addressed this question using simulations (see below). Although the SE appears to be a biased estimator of the sampling variance of \( h^2 \) (SE values calculated for simulated data were consistently greater than the SD around the mean estimates of \( h^2 \) over many simulations), the direction of the bias is such that it allows for conservative meta-analyses. In addition, because the distribution of heritability estimates is not normal, parametric statistics can be biased. Therefore, we also calculated nonparametric bootstrapped CI values on the estimate of heritability obtained by meta-analysis (68). Bootstrapping involved resampling multiple times with replacement and determining the 2.5% and 97.5% range for the estimated heritabilities for all the samples. If the cross-species CI values of \( h^2 \) obtained in the meta-analysis do not include zero, although the sampling variances are overestimated, we can be confident that life span exhibits additive genetic variation. In the case of the capybara (Hydrochaeris hydrochaeris), the variance component estimated for the sire effect (and thus \( h^2 \)) was zero, and so the variance around the estimated \( h^2 \) was undefined. To include this species in the meta-analysis, we assigned it an SE for \( h^2 \) based on a regression of observed SE on sample size (\( n \)) for cases in which estimated heritability was not zero, that is, \( SE = 1.15 - 0.027n \).

Variation in age at death among zoological institutions introduces a potentially confounding effect in estimating heritability when a part of the variance among sires results from variation in zoo conditions, and offspring experience these same conditions. We addressed this potential problem for species individually exhibiting significant heritability by evaluating the contribution of zoo to the variance in age at death in both regular and mixed model analyses of variance in which the main effects were zoo, sire, and dam within sire. Frequently, more zoos than sires were represented in the data set, and so it was not possible to nest sires within institutions. Nonetheless, these analyses provide an indication of the relative contribution of institution to variance in age at death.

**Simulations**

We simulated several genetic models for the heritability of age at death to determine the compatibility of our empirical results with different theories concerning the basis of the age-specific mortality curve in populations. Natural populations of species in the wild and, to a lesser extent, captive populations exhibit two types of mortality as mature individuals (69). Initially, adults are subjected to a minimum level of mortality, which is thought to reflect time-dependent, accidental death from extrinsic factors such as predators, disease, inclement weather, and food shortages. These mortality factors presumably are present regardless of the age of the individual, and risk from these mortality factors presumably is under genetic influence. A second component of mortality increases with the age of the individual and is thought to reflect declining physiological function or increased probability of system failure resulting from the aging process. The change in mortality rate with age has been described by several different functions, each having different biological implications (70,71). The most commonly applied function is the Gompertz equation and several variants, which describe an exponential increase in the rate of mortality (\( m \)) from a minimum (\( m_0 \)) at age \( x = 0 \). Thus,

\[
m_x = m_0 e^{\gamma x},
\]

where \( \gamma \) (lowercase gamma) is the exponential rate of increase in the initial mortality rate. The Gompertz function is frequently modified by the addition of an independent component of the initial mortality rate, which is not subjected to the exponential increase with time, creating the Gompertz–Makeham function:

\[
m_x = m_i + m_a e^{\gamma x},
\]

where \( m_i \) is the age-independent component of mortality, and the initial mortality rate in the population is \( m_0 = m_i + m_a \).

The Weibull aging function describes the increase in mortality rate with age as a power function (hence
dependent only on age and not on the initial mortality rate),
to which an age-independent mortality component can be
added to give:
\[ m_t = m_0 + ax^b. \]

Unlike the Gompertz or Gompertz–Makeham functions, the
Weibull function requires no initial component of mortality
to which aging processes contribute. If extrinsic sources of
mortality were removed from a population, \( m_0 \) would
disappear but the aging-related term \( ax^b \) would remain
unchanged. The Gompertz function assumes that the
mortality rate at age 0 cannot be reduced below \( m_0 \), which
reflects intrinsic properties of the individual. Tests of these
predictions, available when wild populations are brought
into captivity or domesticated, favor a Weibull interpretation
of aging-related mortality in that initial mortality rate can be
greatly reduced without a change in the aging-related
component (69) (Scheuerlein A, Ricklefs RE, unpublished
data, 2008).

Accordingly, we have modeled age-dependent mortality in
our simulations after a Weibull process with an initial level of
age-independent mortality. We simulated two different
genetically based sources of variation in age at death. In the
first case, we modeled genetic variation in the rate parameter
\( \alpha \) of the Weibull function, which influences the
probability of age-dependent mortality uniformly at all ages.
This is a proportional hazards model (72, pages 483–489),
which can also be thought of as a frailty model in which each
individual ages more or less rapidly, depending on its genetic
constitution (73,74). In the second case, we distributed life-
ending mutations at random to each individual according to
a Weibull probability function, and the age at death was
determined by the earliest expression of a lethal allele.

In the frailty model, values of \( \alpha \) had an exponential
normal distribution of the form \( \alpha = \alpha_0 \exp(\alpha_{RND}) \),
where RND is a random normal deviate with a mean of 0 and an
SD of 1. Thus, \( \alpha \) controlled the amount of genetic variation
among individuals in \( \alpha \). In the mutation model, at each age
increment for each parent individual, we drew a random
number from a uniform 0–1 distribution. If that number was
lower than the Weibull mortality rate at that age \( ax^b \),
the individual was assigned a lethal mutation expressed at that
age. We also included a genetic component \( m_G \) to some
proportion (0–1) of the initial mortality rate \( m_0 \) in some
simulations, in which case this component was added to the
aging-related mortality to determine the distribution of lethal
alleles.

Inheritance of age-at-death factors was Mendelian. In the
frailty model, each offspring inherited the average of the
value of \( \alpha \) of its parents. In the mutation model, offspring
inherited each age-specific mutant allele from each parent
with probability = 0.5. This function does not account for
the selective benefits accruing to offspring born late in the
lives of their parents (which clearly do not have early-onset
lethal factors), but most offspring are produced before the
onset of high rates of genetically determined mortality, and
therefore this bias is minimal. At each age increment, an
individual could have genotype 00, 01, or 11 for a lethal
mutation (1); genotypes 01 and 11 were equally lethal.

We simulated the distribution of genetic factors and the
survival of each individual over 50 “years” at increments of
0.1 year. Parameters were chosen so that no individuals
survived the end of the simulation (see below). For each
simulation, we established populations of 20 or 50 males,
representing the higher end of our sample size for captive
populations. Each male in the simulation was mated to two
females, each of which produced two offspring. The ages at
death were determined for all three adults and four offspring
related to each male in the population, and the heritabilities
were calculated by nested ANOVA as described above for
the empirical data. Each simulation with a particular set of
parameters was repeated 25 times to build a sample of
studies approximating the number of species included in our
empirical analyses. Additional details are presented in the
Results section.

Expectations for additive genetic correlations \( r_A \)
between relatives in an outcrossed population are 0 between
sire and dam, 0.5 between parent and offspring, 0.25 among
full sibs, and 0.25 among half sibs (64). To test these
expectations for the mutation model, we simulated the
families of 100 sires with the following Weibull parameters:
\( \alpha = 0.00001 \), \( m_0 = 0.05 \), and \( \beta = 3 \). In this case, the
mean age at death was 13.7 years and the sire component of
variance in age at death was 20.9% of the total, indicating a
heritability of \( h^2 = 4 \) times the sire component of variance =
84%. Phenotypic correlations, which included only additive
genetic variation, were: sire–dam (two dams per sire), 0.23
and –0.07; sire–offspring (two offspring per dam), 0.48,
0.38, 0.51, and 0.50; dam–offspring, 0.47, 0.46, 0.34, 0.32;
full sib, 0.30 and 0.37; and half sib, 0.23, 0.28, 0.20, and
0.14. Considering the small sample of family groups, these
values met expectations reasonably well.

Parameters of the Weibull Aging Model

To determine Weibull parameters on which to base
simulations, we fitted Weibull functions to survival in
captive populations of the lion (P. leo) and the addax (Addax
nasomaculatus). Survival data were constructed from ages
depth used in the analysis of heritability and were fit by the
cumulative Weibull survival function:
\[ l_x = \exp \left( -m_0x - \frac{ax^b+1}{\beta + 1} \right) \]
(14). The fitting incorporates a lower cutoff age to eliminate
elevated “juvenile” mortality, and estimates the survival to
that age as \( \log(S) = -m_0 \times \text{cutoff} \) (Scheuerlein A,
unpublished data, 2001). The fitted parameters are summa-
rized in Table 1, and the function is compared to the
empirical survival function in Figure 1.

Based on these results, we used \( m_0 = 0.05 \), \( \alpha = 10^{-4} \),
and \( \beta = 3 \) in our simulations of age at death. In a simulation
of the mutation model over 100 families with \( m_0 = 0.05 \), \( \alpha =
10^{-5} \), and \( \beta = 3 \), the Weibull function fit the ages at death of
one offspring from each of the 100 sires with the parameters
\( m_0 = 0.0478 \pm 0.0014, \alpha = 1.02 \pm 0.59 \times 10^{-5} \), and \( \beta =
3.80 \pm 0.18 \), which represent a reasonable recovery of the
input parameters. In the simulated curve, the rate of increase
in mortality ($\beta = 3.80$) accelerates more rapidly than in the underlying Weibull function used to distribute lethal alleles ($\beta = 3.00$). As a result, the value of $\alpha$ is correspondingly reduced.

**RESULTS**

**Life Span in Zoo Populations**

We calculated the average age at death for individuals living beyond 1 year in the 14 species of mammals and 8 species of birds included in this analysis. Averages across species for sires (mammals: 10.5 ± 3.3 SD; birds: 10.3 ± 2.9) and dams (mammals: 10.9 ± 3.2; birds: 9.9 ± 2.9) were similar between the sexes and between mammals and birds. Offspring tended to die at younger ages (mammals: 6.4 ± 1.9; birds: 5.1 ± 0.6) because many individuals die before reaching reproductive age. However, maximum life span among offspring (mammals: 17.9 ± 4.0; birds: 16.8 ± 5.2) also was somewhat lower than that of their parents (mammals: sires = 20.9 ± 7.7, dams = 23.6 ± 9.3; birds: sires = 22.2 ± 6.8, dams = 19.4 ± 5.1).

**Heritability of Life Span in Zoo Populations**

We estimated variance components using REML for 14 species of mammals. These analyses revealed significant additive genetic variation for life span (i.e., statistically significant sire effects) in three species, and close to significance ($p < .10$) in two others (Table 2). Four of these five species were among the six with the largest sample sizes, suggesting that the detection of significant sire effects depends on having adequate statistical power. The weighted mean estimate of $h^2$ obtained across species in the meta-analysis was 0.53, and although the CI around this estimate was wide, it did not include zero. The 95% CI calculated parametrically ranged from 0.11 to 0.95, whereas the nonparametric bootstrap CI ranged from 0.28 to 0.87.

When we analyzed ages at death of offspring born to parents beyond 2, 4, 6, 8, and 10 years of age in species for which sample sizes were adequate, we found that the estimate of heritability tended to decrease with age. Nonetheless, heritability remained significantly above 0, or marginally insignificant, for the lion (Panthera leo) and red kangaroo (Macropus rufus), two species with large samples, until more than 60% and 80% of individuals had died, respectively (Figure 2).

Regression analyses revealed significant additive genetic variation for life span (i.e., slopes significantly >0) in five species of mammals, and near significance in one additional species (Table 2). The three species for which REML variance component estimation indicated significant ($p < .05$) heritability also showed significant (or marginally insignificant: $p < .10$) heritability in regression analyses, suggesting that the two methods yielded similar results. However, the estimates of $h^2$ obtained from regression analyses were consistently lower than those obtained from the estimation of variance components (Table 2). The discrepancy in the magnitude of the heritability estimates is clear in the results of meta-analyses: The weighted mean estimate of $h^2$ obtained across species in regression analyses was only 0.17, and, in contrast to the variance components analyses, the CI values around this estimate did, in one case, include zero (parametric CI, −0.044 to 0.378; bootstrap CI, 0.08 to 0.26).

For the six species having significant values for either or both of the variance component and the mid-parent regression estimates of heritability (Table 2), the Pearson parameteric correlation between the two was $r_p = 0.87$ ($p = .024$) and the Spearman rank-order correlation was $r_s = 0.83$ ($p = .042$). The mean variance component estimate of heritability for these species was 0.91 ± 0.52 SD, and the regression estimate of heritability was 0.33 ± 0.14 SE. The regression of $h^2$(reg) on $h^2$(var) had an insignificant intercept ($t = 1.8$, $p = .14$). In a regression passed through the origin, the slope of the regression was 0.33 ± 0.04 SE. The source of this threefold discrepancy will be considered in the Discussion.

The CV$_A$ values, expressed as a proportion of the average age at death, for five species of mammal with significant or marginally insignificant sire component heritabilities varied between 0.35 and 1.06. For six species exhibiting significant or marginally insignificant regression-based heritabilities, CV$_A$ varied between 0.20 and 0.54.

Zoo effects estimated by regular ANOVA were substantial, but not significant, for P. leo and the blackbuck (Antilope cervicapra). In mixed models, these effects were significant ($p < .05$) and accounted for 38% and 78%, respectively, of the total variance; A. nasomaculatus was third on this list at 20% ($p > .05$). These species also exhibited the highest estimates of sire–component heritability, all three of which exceeded 1. Although the institution effect appears to confound estimation of heritability, this effect was small (<16%) and statistically insignificant for M. rufus, Acinonyx jubatus, Leontopithecus rosalia, and...
Capra hircus, for which sire–component heritabilities ranged from 0.24 to 0.77 and regression heritabilities ranged from 0.18 to 0.31.

Sample sizes for birds were smaller than for mammals, and we were unable to estimate variance components in half-sib designs because cases in which males produced multiple offspring with more than one female were scarce.

Parent–offspring regressions were not significant for any of the eight species we examined (Table 2), and CI values around the cross-species estimate of $h^2$ obtained in meta-analysis (0.07) did include zero (parametric: −0.26 to 0.41; bootstrap: −0.003 to 0.18). With respect to the parent–offspring regressions, the probability of obtaining positive and negative values is equal under a hypothesis of no

### Table 2. Heritability Estimates for Ages at Death in Zoo Populations of Mammals and Birds Determined from Analysis of Variance Components (REML) and Parent–Offspring Regressions

<table>
<thead>
<tr>
<th>Species</th>
<th>N (Sires)</th>
<th>$h^2$ (SE)</th>
<th>p</th>
<th>N (Families)</th>
<th>$h^2$ (SE)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mammals</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red kangaroo (Macropus rufus)</td>
<td>36</td>
<td>0.66 (0.45)</td>
<td>.071*</td>
<td>151</td>
<td>0.18 (0.08)</td>
<td>.049*</td>
</tr>
<tr>
<td>Amur tiger (Panthera tigris altaica)</td>
<td>29</td>
<td>0.07 (0.14)</td>
<td>.308</td>
<td>138</td>
<td>0.08 (0.14)</td>
<td>.250</td>
</tr>
<tr>
<td>Reindeer (Rangifer tarandus)</td>
<td>21</td>
<td>0.48 (0.61)</td>
<td>.212</td>
<td>88</td>
<td>0.13 (0.15)</td>
<td>.400</td>
</tr>
<tr>
<td>Addax (Addax nasomaculatus)</td>
<td>18</td>
<td>1.38 (0.71)</td>
<td>.025</td>
<td>76</td>
<td>0.43 (0.15)</td>
<td>.006</td>
</tr>
<tr>
<td>Cheetah (Acinonyx jubatus)</td>
<td>18</td>
<td>0.73 (0.42)</td>
<td>.040</td>
<td>96</td>
<td>0.23 (0.13)</td>
<td>.081*</td>
</tr>
<tr>
<td>Lion (Panthera leo)</td>
<td>17</td>
<td>1.68 (0.87)</td>
<td>.027</td>
<td>89</td>
<td>0.55 (0.14)</td>
<td>.003</td>
</tr>
<tr>
<td>Patagonian cavy (Dolichotis patagonum)</td>
<td>13</td>
<td>0.62 (0.57)</td>
<td>.139</td>
<td>54</td>
<td>−0.11 (0.16)</td>
<td>.506</td>
</tr>
<tr>
<td>Golden lion tamarin (Leontopithecus rosalia)</td>
<td>13</td>
<td>0.24 (0.49)</td>
<td>.313</td>
<td>84</td>
<td>0.27 (0.11)</td>
<td>.016</td>
</tr>
<tr>
<td>Cotton-top tamarin (Saguinus oedipus)</td>
<td>13</td>
<td>0.07 (0.75)</td>
<td>.460</td>
<td>98</td>
<td>0.001 (0.11)</td>
<td>.990</td>
</tr>
<tr>
<td>Capybara (Hydrochaeris hydrochaeris)</td>
<td>13</td>
<td>0.00 (0.80)</td>
<td>1.000</td>
<td>67</td>
<td>0.08 (0.11)</td>
<td>.503</td>
</tr>
<tr>
<td>Leopard (Panthera pardus)</td>
<td>10</td>
<td>0.39 (0.73)</td>
<td>.296</td>
<td>72</td>
<td>0.11 (0.14)</td>
<td>.457</td>
</tr>
<tr>
<td>Domestic goat (Capra hircus)</td>
<td>9</td>
<td>0.77 (0.73)</td>
<td>.145</td>
<td>45</td>
<td>0.31 (0.13)</td>
<td>.020</td>
</tr>
<tr>
<td>Blackbuck (Antilope cervicapra)</td>
<td>9</td>
<td>2.23 (1.51)</td>
<td>.070*</td>
<td>31</td>
<td>0.01 (0.25)</td>
<td>.958</td>
</tr>
<tr>
<td>Gray wolf (Canis lupus)</td>
<td>6</td>
<td>1.23 (1.21)</td>
<td>.154</td>
<td>52</td>
<td>−0.04 (0.18)</td>
<td>.819</td>
</tr>
</tbody>
</table>

**Notes:** *Marginally insignificant value (.05 < p < .10).

†Significant value (p < .05).

REML = restricted maximum likelihood; SE = standard error; $h^2$ = heritability; — = analysis not done.

**Parent–Offspring Regressions**

**REML**

**Parent–Offspring Regressions**

**Figure 2.** Heritability estimated from variance components and midparent regression in four species of mammal as a function of minimum age at death of sires (variance components estimates) or both parents (regression estimates). For each species, points moving from left to right represent analysis of deaths occurring after 1, 2, 4 (Acinonyx), 6 (Addax), 8 (Macropus), and 10 (Panthera) years. However, data are plotted on a scale of the proportion of offspring surviving to each age to normalize differences in life span among the species.
relationship. For birds, obtaining as few as 2 negative values of 8 does not differ significantly from the null hypothesis (binomial test, \( p = .145 \)). For mammals, however, as few as 2 of 14 is significant by binomial test (\( p = .006 \)).

We also used a Kolmogorov–Smirnov one-sample test to compare the values of \( p \) for parent–offspring regressions to the uniform distribution expected for the null hypothesis of no significant heritability. For mammals, \( D = 0.348 \) and the critical value for \( N = 14 \) and \( p = .05 \) is 0.349; for birds, \( D = 0.075 \) and the critical value for \( N = 8 \) and \( p = .05 \) is 0.457. For the significance levels of the REML variance component heritabilities for mammals, \( D = 0.544 \) and the critical value for \( N = 14 \) and \( p = .01 \) is 0.418. Thus, in contrast to mammals, birds exhibit no significant heritability in the age at death. However, neither parametric ANOVA nor nonparametric (Wilcoxon, Kruskal–Wallis, two-sample Kolmogorov–Smirnov) tests indicated that the regression-based heritability estimates for bird and mammal samples differed significantly from each other.

Simulated Heritabilities

The meta-analysis of the mammalian data estimated heritability in age at death to be 0.53 (variance component values), which occupies the high end of the range of values obtained from larger samples of humans and domesticated animals. The point of the simulations was to determine the type of model and parameter spaces that could reproduce similar levels of heritability. Simulations were run 25 times for each set of parameters for either 20 or 50 sire groups. The Weibull parameters \( \alpha = 0.0001 \) and \( \beta = 3 \) were applied in all simulations.

In the frailty models, all variation in the age at death resulted from stochastic variation, with probabilities of death (hazards) varying genetically among individuals through variation in the value of \( \alpha \). The Weibull function has two sources of stochastic variation, namely the exponentially distributed component produced by \( m_0 \) and the component produced by the power function \( \alpha \). The variance produced by the exponential function alone would be \( (1/m_0)^2 \), that is, 400 for \( m_0 = 0.05 \), and that produced by the power function alone would be \( ([\beta + 1]/2\alpha)^2/(\beta + 1) \).

The results of the simulations are presented in Table 3 along with the SD values of each of these variables over the 25 trials in each simulation. The SD of the heritability estimates across the 25 trials should approximate the SE of the heritability estimated within each trial. Where the average heritability is high and most of the estimates exceed 0, the correspondence between SD\((h^2)\) over trials and the average SE\((h^2)\) within trials is reasonably close.

Estimates of heritability in frailty models approached the empirical variance–component value of 0.53 only when the age-independent component of mortality (\( m_0 \)) was set equal to 0 (simulation D). In this case, the residual (nongenetic) variance was equal to 12.2 years\(^2\), close to the predicted value for the power function. The heritable component of variance was generated by variation among individual parents in the value of \( \alpha \). In this simulation, the expected life spans of individuals for which \( \alpha \) is 1 SD below the mean, at the mean, and 1 SD above the mean, including about two thirds of the individuals in the population, are 16.5, 12.8, and 10.0 years, respectively. Variance–component heritabilities are not achieved in frailty models with levels of nongenetic age-independent mortality typical of \( m_0 \) estimated from zoo age-at-death data, although regression estimates of heritabilities were reasonably well matched in all the simulations. Estimated values of \( m_0 \) in natural populations range from about 0.03 to 0.10 in large mammals (\( P. leo \), 0.032; \( Rangifer tarandus \), 0.076; \( Syncerus caffer \), 0.026–0.092; \( C. elaphus \), 0.050; \( Ovis dalli \), 0.049 for males and 0.107 for females) to about 0.20 in mid-sized species.

Table 3. Heritability Estimates Based on the Sire Component of Variance in Simulated Data

<table>
<thead>
<tr>
<th>Trial</th>
<th>( m_0 )</th>
<th>( m_0 )</th>
<th>( SD(\alpha) )</th>
<th>Males</th>
<th>Heritability ((h^2))</th>
<th>( SE(h^2) )</th>
<th>( SE(SE) )</th>
<th>( h^2 = 0 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.050</td>
<td>0.5</td>
<td>20</td>
<td>0.128</td>
<td>0.221</td>
<td>0.463</td>
<td>0.068</td>
<td>13</td>
</tr>
<tr>
<td>B</td>
<td>0.050</td>
<td>1.0</td>
<td>20</td>
<td>0.242</td>
<td>0.417</td>
<td>0.550</td>
<td>0.102</td>
<td>14</td>
</tr>
<tr>
<td>C</td>
<td>0.050</td>
<td>1.0</td>
<td>50</td>
<td>0.122</td>
<td>0.138</td>
<td>0.299</td>
<td>0.031</td>
<td>8</td>
</tr>
<tr>
<td>D</td>
<td>0.000</td>
<td>1.0</td>
<td>50</td>
<td>0.576</td>
<td>0.354</td>
<td>0.355</td>
<td>0.114</td>
<td>2</td>
</tr>
<tr>
<td>E</td>
<td>0.050</td>
<td>( SD(m_0) = 1.0 )</td>
<td>50</td>
<td>0.439</td>
<td>0.313</td>
<td>0.325</td>
<td>0.079</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mutation model</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
</tr>
<tr>
<td>B</td>
</tr>
<tr>
<td>C</td>
</tr>
<tr>
<td>D</td>
</tr>
<tr>
<td>E</td>
</tr>
</tbody>
</table>

Notes: In all simulations \( \alpha = 0.0001 \), \( \beta = 3 \), and the number of replicates was 25. The column labeled \( h^2 = 0 \) contains the number of simulations of 25 exhibiting no significant heritability of age at death.

\( SD \) = standard deviation; \( SE \) = standard error.
(Kobus kob, 0.206; Damalisus korrigum, 0.166 for males and 0.182 for females) and >0.40 in small species (Tamiasciurus hudsonicus, 0.44; Sylvilagus floridanus, 1.23) (14).

Estimated heritabilities in mutation models also were low when a nongenetic component of age-independent mortality was included in the simulations. As in the frailty models, heritabilities approached empirical values determined from variance components only when genetic variation was added to the age-independent component of mortality \( (m_0) \) or when age-independent mortality was excluded from the model, although regression estimates were matched in all the simulations, including those with no genetic variation in the initial mortality rate. Thus, the simulations suggest that the observed heritabilities of age at death in captive populations of mammals are compatible with genetic variation in the age-dependent component of mortality. Trial E of the mutation model, in which age-independent mortality \( m_0 = 0 \), resulted in a heritability of age at death of \( h^2 = 0.79 \). Thus, it is clear that a random distribution of lethal genes with Weibull-distributed ages at expression can produce high heritability in age at death, as can genetic variation in frailty.

The CV_A values in the simulations were remarkably similar, with values ranging between 0.20 and 0.26 for all trials except for trial E of the frailty model (CV_A = 0.40) and trial D of the mutation model (CV_A = 0.49). Thus, even with the large stochastic variation in the age at death, genetic effects can have a substantial presence.

**Discussion**

Three observations are noteworthy in this study. First, heritabilities in zoo populations of nondomesticated mammals are substantial, generally exceeding those observed for domesticated animals. Second, heritabilities calculated from sire variance components in mammals exceeded values calculated by parent–offspring regression, by a factor of three. Third, heritabilities of age at death for birds based on parent–offspring regression did not differ from 0, although the sample also did not differ significantly from that for mammals. It is likely, however, that birds have lower, perhaps negligible, heritabilities for age at death, in contrast to mammals.

Simulations indicated that, with substantial initial mortality \( (m_0) \), heritabilities exceeding 0.30 were obtained only with genetic variation in \( m_0 \). For example, in trial E of the frailty model, we introduced variation in the age-independent mortality rate \( (m_0) \), and removed variation in the hazard scaling parameter \( \alpha \). In this case, the variance component estimate of heritability was similar to simulation D, in which age-independent mortality was removed and a similar level of variation was applied to \( \alpha \). Thus, observed heritability in longevity need not implicate mortality factors with age-dependent expression, although such variation was compatible with heritabilities estimated by offspring–parent regression.

Age-independent mortality exceeds age-dependent mortality in a Weibull process until age \( (x) = (m_0 \sqrt{2})^{1/\beta} \), which in the simulations used in this study \( (m_0 = 0.05; \alpha = 0.0001, \beta = 3) \) is \( x = 7.94 \) years. In simulation trials A and B of the mutation model, the average and median longevities were about 10 years, and the maximum was between 18 and 19 years, which resemble values for the species included in this study. Additional simulations showed that genetic variation in age-independent mortality can create high heritability in age at death in the absence of genetic variation in the aging-related component of mortality. Thus, our results suggest that natural populations contain substantial genetic variation for qualities that influence survival, but it is difficult to allocate these genetic effects between age-dependent and age-independent components of variation.

For the mammal data, we observed that heritabilities of age at death were about three times higher for variance–components estimates than for parent–offspring regression estimates. This is not apparent in the simulated data. We ran an additional simulation of mutation model C using the same parameters and 25 trials, and also calculated the regression slope of one offspring against the average of its parents \( (n = 50) \). The sire variance–components estimate of heritability was close to the first set of simulations \( (0.35 \pm 0.27 SD) \); the midparent regression slope was somewhat (but not significantly) higher \( (0.51 \pm 0.13 SD) \). These and other simulations (not shown) provided no indication that variance components gave higher values of heritability than parent–offspring regressions. In simulations of mutation model C, the female variance component provided an estimate of heritability \( 4 \) times the proportion of the dam variance component \( 0.59 \pm 0.42 SD \), which did not differ significantly from the regression slope. Also, the average of the sire and dam heritabilities was \( 0.47 \pm 0.37 SD \), which was close to the regression slope estimate.

One explanation for the higher heritability resulting from variance components estimation is that age at death varies significantly among zoos and that this component of variance is absorbed in the sire component of variance, thereby inflating the apparent heritability. Because most of the data sets include only one or a few sires per zoo, variation among sires and among zoos is confounded. We conducted ANOVAs of the ages at death of offspring included in this study for six species of mammals \( (A. jubatus, A. nasomaculatus, A. cervicapra, L. rosalia, M. rufus, and P. leo) \), for which the zoo effect alone accounted for between 24% and 70% of the variance and was significant \( (p < .01) \) in three of the six species. When we entered the zoo in which death occurred as the first effect in the nested ANOVA, the sire effect was reduced substantially in \( A. cervicapra \) \( (p \) nonetheless < .05) and \( P. leo \) \( (p > .05) \), the two species with the highest heritability estimates. In contrast, the sire effect increased in \( L. rosalia \), where adding zoo as an effect apparently transferred a substantial fraction of the residual variation to the sires. In this species, however, most offspring were transferred to other zoos after birth, so the effect of zoo on the heritability estimate is complex. In general, in ANOVA components, common-environment effects might be allocated either to the dam effect, in which case they would reduce apparent heritability based on the sire component of variance, or to the sire effect, having the opposite consequence. The slope of the offspring–midparent regression is increased somewhat by
common environment, but the effect on heritability is not so great as in a nested analysis. The slope would be decreased by genotype–environment interactions, but we had insufficient samples to analyze this component of variation.

Nondomesticated populations of mammals maintained in zoos appear to contain more genetic variation in age at death than do domesticated populations, based on the more conservative offspring–parent regression estimates. One possibility is that most of the heritability in wild populations arises from genetic variation in age-independent causes of death. Thus, when these causes are eliminated in the environments of domesticated and laboratory animals, the heritability in age at death drops dramatically. Populations of mammals in zoos are exposed to intermediate levels of age-independent mortality from contagious diseases, accidents, and social interactions, so they might retain genetic variation for susceptibility to these factors. Deaths at old age in zoo populations are too few to determine whether these have a significant heritable component, as one would expect from genetic variation in the rate of aging, and is evident in large samples of humans (30,60). It is also possible that strong selection associated with domestication removes much genetic variation for age at death.

Based on parent–offspring regressions, birds show little heritability in age at death in zoo populations (Table 2). If most of the genetic variation in age at death detected in zoo populations were related to the age-independent component of mortality, then this component would appear to be greatly reduced in birds compared to mammals. Captivity in zoos reduces age-independent mortality by about the same amount in birds and mammals (76), so the relative safety of birds and mammals in captivity cannot explain the difference in heritability of longevity. Alternatively, birds might have relatively little genetic variation for age at death in natural populations compared to mammals. We stress, however, that low sample sizes for the zoo populations of birds might account for our inability to detect significant genetic variation in life span.

Charmantier and colleagues (32) found a relatively low heritability (0.083 ± 0.049) in the age at last reproduction, which might be related to age at death, in mute swans. Other data for wild populations of birds are lacking. In any event, most detectable genetic variation in age at death in small samples occurs early in the potential life span of individuals and probably represents primarily age-independent components of mortality. It is unclear why heritability should be reduced in birds, unless strong directional selection for performance and life span weeds out genetic variation. For their body size, birds have relatively low adult mortality rates and long life spans compared to mammals (14,77), despite high metabolic rates, high blood glucose levels, and other indicators unfavorable to longevity in mammals. It is interesting to speculate that the stringencies of flight push birds to physiological limits that permit little tolerance for variation in physical performance. Mechanisms that minimize decline in performance with age might also extend life span and reduce its genetic variation. Data on the genetic basis of longevity in bats would be informative in this regard, but little more can be said at this point in the absence of relevant data.

Does the heritability of life span in captive mammals have any implications for the genetic basis of longevity in natural populations? Even in the absence of genetic variation in genes expressed late in life, variation in genes controlling age-independent causes of death would result in measurable heritability in age at death, as observed in populations of mammals in this study. Thus, estimates of heritability in small samples of captive individuals in zoos have relatively little to say about the mechanisms of senescence and the underlying causes of evolutionary differentiation in the rate of senescence among species. Nonetheless, heritability of the age at death remained significant beyond the age at which 60% of individuals had died (in captivity) in the lion (P. leo) and 80% in the red kangaroo (M. rufus). Presumably, death rates of young adults in the wild are much higher than in captivity, and these ages therefore represent old individuals. Thus, it would appear that at least a part of the heritability in age at death in zoo populations represents genetic factors expressed in individuals of advanced age.

This study illustrates the potential of zoo populations for studies of genetic variation in life-history traits, including age at death. Variation in animal care and husbandry practices among zoos is a potentially confounding factor, but the detailed record keeping of genealogies in zoo populations permits the estimation of genetic components of variation. Parent–offspring regression appears to be more conservative and less prone to biases than is nested variance components analysis owing to the confounding of zoo and sire effects in the latter. In addition, we have found in simulations that random variation in the magnitude of dam effects can obscure heritability estimates based on the sire component of variation.

Overall, our study revealed substantial genetic variation in age at death in captive populations of nondomesticated mammals, and perhaps much less in populations of birds. The magnitude of heritability and its distribution across the potential life span of individuals reveals little about the kinds of genes responsible for these effects. Thus, studies of age at death in small captive populations are unlikely to shed much light on the causes of senescence in wild populations other than to emphasize that populations of mammals contain considerable genetic variation for factors that influence survival in a captive setting. Presumably, this variation is maintained by balancing influences on other components of fitness, although the nature of these trade-offs is not understood.

ACKNOWLEDGMENTS
This study was supported by National Institutes of Health/National Institute on Aging grant R01 AG20263-01.

We are grateful to the International Species Information System (ISIS) for providing data on zoo populations. Insightful comments from Brian Charlesworth, Anne Charmantier, Dave Colman, Marco Festa-Bianchet, Ben Sheldon, and several reviewers greatly improved the manuscript.

Dr. Cadena is now with Departamento de Ciencias Biológicas, Universidad de los Andes, Bogotá, Colombia.

CORRESPONDENCE
Address correspondence to Robert E. Ricklefs, PhD, Department of Biology, University of Missouri-St. Louis, 8001 Natural Bridge Rd., St. Louis, MO 63121-4499. E-mail: ricklefs@umsl.edu
References


Received May 14, 2007
Accepted January 7, 2008
Decision Editor: Huber R. Warner, PhD

---

**Now Available!**

**Improving Practice Through Research In and About Assisted Living: Implications for a Research Agenda**
- Rosalie A. Kane and Keren Brown Wilson

**Historical Evolution of Assisted Living in the United States, 1979 to the Present**
- Keren Brown Wilson

**The Place of Assisted Living in Long-Term Care and Related Service Systems**
- Robyn I. Stone and Susan C. Reinhard

**Definition and Classification of Assisted Living**
- Sheryl Zimmerman and Phillip D. Sloane

**Defining Quality in Assisted Living: Comparing Apples, Oranges, and Broccoli**
- Catherine Hawes and Charles D. Phillips

**Dementia and Assisted Living**
- Joan Hyde, Rosa Perez, and Brent Forester

**Physical Environments of Assisted Living: Research Needs and Challenges**
- Lois J. Culler

Families and Assisted Living • Joseph E. Gaugler and Robert L. Kane

Improving Health Care for Assisted Living Residents • Robert L. Kane and John R. Mach, Jr.

Assisted Living and Special Populations: What Do We Know About Differences in Use and Potential Access Barriers? • Mauro Hernandez and Robert Newcomer

Assisted Living and Residential Care in Oregon: Two Decades of State Policy, Supply, and Medicaid Participation Trends • Mauro Hernandez

Assisted Living Literature Through May 2004: Taking Stock • Rosalie A. Kane, Jane Chan, and Robert L. Kane

Developing a Research Agenda for Assisted Living • Rosalie A. Kane, Keren Brown Wilson, and William Spector

Available for purchase through GSA’s online store at www.geron.org.

All members will automatically receive a complimentary copy in the mail.