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Intrinsic aging-related mortality in birds

Robert E. Ricklefs

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Actuarial senescence in captive populations of 28 species of bird was quantified by estimating the parameters of Weibull models fitted to survival curves constructed from data obtained from zoos. Samples of natural and captive populations were compared using phylogenetically independent contrasts, which revealed that extrinsic mortality rates in captive populations are, on average, less than 30% of those of natural populations but that the component of mortality related to aging does not differ significantly between natural and captive birds. This result supports the hypothesis that aging-related mortality is associated with intrinsic causes of death that kill independently of the external environment. A logical implication of this result is that birds in natural populations maintain a high level of physical fitness into old age and do not become more vulnerable to extrinsic mortality factors with increasing age. Additional comparisons showed that the rate of aging in this sample of birds is correlated with body mass, but not with embryonic or postnatal growth rate. These analyses suggest that studies of aging in captive populations can provide powerful tools to help us understand senescence in natural populations.

R. E. Ricklefs, Department of Biology, University of Missouri-St. Louis, 8001 Natural Bridge Road, St. Louis, MO 63121-4499, USA. E-mail: ricklefs@jinx.umsl.edu

Decline in physiological function associated with aging results in an increase in mortality rate (actuarial senescence) within populations both in nature and in captivity (Finch 1990, Holmes and Austad 1995a, b). Species exhibit different rates of actuarial senescence (Promislow 1991, Ricklefs 1998), although the physiological causes and evolutionary significance of this variation are not well understood (Rose 1991). Two hypotheses can account for actuarial senescence. First, physiological deterioration that accompanies aging may increase the vulnerability of organisms to the same extrinsic factors that cause death among young adults (e.g., predation, contagious disease, starvation, and weather-related stress). Alternatively, actuarial senescence may reflect an increase with age in intrinsic causes of death (e.g., from vascular disease, cancer, autoimmune disease, and acquired genetic damage) that kill more or less independently of the external environment. Human aging is associated primarily with an increase in intrinsic causes of death (Coni et al. 1992, Hayflick 1994), but it is not known whether this is typical of natural populations. The two hypotheses can be distinguished by comparing populations in nature and in captivity. In captivity, extrinsic mortality is greatly reduced. If actuarial senescence were caused by increased susceptibility to extrinsic mortality factors, then the rate of death associated with aging would be reduced in captive compared to natural populations. If aging resulted in an increase in intrinsic causes of mortality, then rate of actuarial senescence in natural and captive populations would not differ. Here it is shown that rate of actuarial senescence, which is quantified in this study by parameters of the Weibull aging function, does not differ between natural and zoo populations. This suggests that aging affects death rate in both natural and captive populations through an increase in intrinsic causes of mortality. Because susceptibility to extrinsic mortality does not appear to increase appreciably with age, individuals in natural populations of birds evidently remain physically fit into old age.

Maximum life span of warm-blooded vertebrates, whether in nature or in captivity, increases with body mass (Sacher 1959, Calder 1983, Promislow 1991) and has been shown to differ between birds and bats, on one hand, and non-volant mammals, on the other (Austad and Fischer 1991, Holmes and Austad 1994).

This pattern of variation is consistent with the greater ability of larger animals and, especially, flying animals to avoid predators and escape other extrinsic causes of mortality. The degree to which life span also is related to aging processes in natural populations is, however, poorly understood. Furthermore, whether deaths of older individuals result from increasing vulnerability to the same causes suffered by young adults, or from degenerative diseases that are ultimately lethal in their own right, is not known.

The pattern of actuarial senescence may be addressed directly by fitting mathematical functions to data describing the increase in mortality rate with age (Gavrilov and Gavrilova 1991, Wilson 1994). These functions partition the force of mortality into an initial rate typical of young adults usually denoted m_0, and a component whose rate increases with age (aging-dependent mortality). In this study, the initial mortality rate is assumed to reflect primarily extrinsic mortality, and the age-dependent term is assumed to measure the intrinsic component of mortality associated with senescence. These components are additive in the Weibull function, $m_x = m_0 + \alpha x^{\beta}$, which is used in this analysis. Accordingly, actuarial senescence is quantified by the shape of the curve relating mortality rate to age (β, [dimensionless]) and the magnitude of the curve (α, [time$^{-(\beta+1)}$]). Because rates have units of inverse time [time^{-1}], the derived parameter $\omega = \alpha^{1/(\beta+1)}$ [time^{-1}] is used here to compare rates of actuarial senescence among populations (Ricklefs 1998). β has an average value of about 3 in both natural and captive populations (Ricklefs 1998), indicating that intrinsic mortality increases approximately with the cube of age.

Weibull equations fitted to survival curves of five species in the London Zoo (Comfort 1962) showed that, although m_0 was much lower in captive than natural populations, the parameter ω did not differ significantly between them (Ricklefs 1998). This observation was consistent with the hypothesis that aging-related mortality reflects intrinsic causes rather than increased susceptibility to extrinsic causes. A larger sample of data from captive populations, obtained from a consortium of zoological institutions throughout the world and analysed in this study, permits a more detailed comparison of aging parameters in captive and natural populations and a better test of hypotheses concerning aging-dependent mortality.

Materials and methods

Data for natural populations of birds were obtained from 12 studies reporting survival of marked individuals of known age in local populations (see Ricklefs 1998). Ages at death in 23 species of bird in captivity were used to construct survival curves, from which the parameters of the Weibull model were estimated. These data were obtained from the International Species Information System (ISIS). ISIS is an international private organization located in Apple Valley, MN, with over 500 member zoological institutions. Data were edited to remove cases of death occurring (a) during the first year of life (which are concentrated within 60 days after hatching), (b) within 30 days of transfer between institutions, (c) when the date of death was imperfectly known (as is often the case with birds in large enclosures, particularly waterfowl), and (d) when siblings died on the same day or within a short interval, suggesting trauma or contagious disease as causes of death. In addition, the analysis retained only individuals born k years before the last entries in the database, where k is the maximum age at death among all individuals in the sample.

Ages at death were rank-ordered and ranks were converted to proportion of individuals surviving by dividing by the highest rank in the population (i.e., the sample size). This transformation provides a survivorship (l_x) curve describing the proportions of individuals alive at age x. This curve was then fitted by nonlinear curve fitting (NLIN procedure of the Statistical Analysis System® [SAS]) to estimate the parameters α and β. The procedure used a form of the Weibull aging model in which the dependent variable is the natural logarithm of the survivorship

$$\ln(l_x) = -m_0 - \left(\frac{\alpha}{\beta+1}\right)x^{\beta+1} \quad (1)$$

Additional survivorship curves of five species in the London Zoo were obtained from Comfort (1962) and analysed in the same manner.

The Weibull aging model is used in this study instead of the Gompertz model, $m_x = m_0 e^{-\gamma x}$, and the related Gompertz-Makeham model, $m_x = A + m_0 e^{-\gamma x}$ (Gavrilov and Gavrilova 1991), because the Weibull model quantifies actuarial senescence independently of extrinsic mortality (m_0). As a result, actuarial senescence can be compared among populations having manipulated levels of extrinsic mortality (e.g., natural versus captive populations).

Three additional methodological issues are addressed here. The first of these is the use of the derived measure omega (ω) to express rate of aging. The intrinsic component of mortality rate in the Weibull equation has two parameters, α [time$^{-(\beta+1)}$] and β [dimensionless], neither of which has dimension [time^{-1}], which would be required of a measure of rate. α and β can be combined into a single measure ω [time^{-1}] according to $\omega = \alpha^{1/(\beta+1)}$. The inverse of ω has dimension [time] and is of the same order of magnitude as the average and maximum life span in a population. For a given value of β, intrinsic mortality rate at every age increases with increasing ω ($dm_x/d\omega = [\beta+1][\omega x]^{\beta}$), as does the total

intrinsic mortality up to a given age ($d[-\ln\{l_x\}]/d\omega = x^{\beta+1}\omega^{\beta}$). When β and α both vary, the relationship between m_x and ω becomes more complex, but the total mortality up to a given age still parallels values of ω.

The second methodological issue is the way in which aging models are fitted to mortality data. When samples are large, maximum likelihood (ML) applied to ages at death provides unbiased estimates of model parameters (Lawless 1982). However, when samples are small, ML may fail to converge on a solution. In one study using simulated ages at death, ML failed to estimate parameters of a Weibull model 5 out 10 times for samples of 250 individuals and 10 of 10 times for samples of 100 individuals (Ricklefs 1998). When ages at death are not available, one may fit model equations for m_x or l_x to age-specific mortality rates or survivorship curves by nonlinear curve fitting (Eakin et al. 1995, Shouman and Witten 1995). Age-specific mortality rates provide statistical independence of the sample data but are subject to greater variability than survivorship. Consequently, age-specific mortality rates frequently produce poor estimates with small sample sizes. Most of the data for natural populations included in this study (Ricklefs 1998) were analysed using nonlinear regression to fit the Weibull model for m_x to age-specific mortality rates obtained from long-term studies of marked populations. Even though samples were as small as 27 marked individuals, each individual contributed to the sample at risk for each age until it died. Even so, many older age classes had to be combined to obtain reasonable samples. The zoo data were obtained as ages at death. However, because the edited samples were small (22 to 86), it was not possible to calculate reasonable estimates of age-specific mortality rates, and Weibull model parameters were estimated by non-linear curve-fitting of survival curves reconstructed from the ages at death (equation 1). This brings up the third methodological issue, namely how it is possible to obtain reasonable estimates of model parameters with such small samples.

I investigated the sampling distributions of parameter estimates for the Weibull aging model by fitting Weibull functions to data simulated with specified values for m_0, α, and β. Typical results, in this case for 10 data sets for each of 1,000, 100, 50, and 25 ages at death, are shown in Table 1. Bias in estimated initial mortality (m_0) increased to -34% of its model value for samples of 25 and the relative standard deviation (SD/model parameter) increased from 7.5 to 47.5% between samples of 1000 and 25. Thus, this curve-fitting technique tends to underestimate m_0, by about 25% on average for samples of 50, which is about the middle of the range of sample sizes in this study. The scaling parameter α is estimated very poorly with small samples because it enters the Weibull model as a multiplier of age raised to the power of β and is highly negatively correlated with the estimated value of β. Consequently, estimated α is hypersensitive to variation in β, which tends to be underestimated with small samples. However, because of the way in which ω is derived from α and β, variations in both tend to cancel. As a result, ω can be estimated with relatively little bias and variation. With a sample size of 50, the bias in ω was $+7.5\%$ and the relative standard deviation was only 11% of the model parameter; with a sample size of 25, these values were 13 and 17%, respectively. Thus, it is practical to estimate parameters of the Weibull equation with samples as small as 25 ages at death for comparative analyses that span large ranges in model parameters.

For comparative analyses, a phylogenetic tree was constructed for the sample of birds represented in this analysis from the phylogeny of Sibley and Ahlquist (1990). Trait values for each node were calculated as the mean of the pair of descendant node or tip values. Contrasts were calculated as the difference between the trait values for the descendant lineages from each node. Contrasts were neither standardized by branch lengths nor rectified. Intercepts of regressions involving contrasts did not differ significantly from 0.

Results

With the exception of the Bewick's Swan *Cygnus columbianus*, natural populations included in this study belong to the avian orders Ciconiiformes and Passeriformes, whereas captive species are spread more widely among birds as a whole (Table 2 and Fig. 1). Because of this heterogeneous sampling, one cannot compare captive and natural populations independently of their evolutionary relationships (Felsenstein 1985, Harvey and Pagel 1991). To circumvent this problem, values of m_0 and ω were compared for seven pairs of sister

Table 1. Parameters of the Weibull growth model estimated from data sets of 1000, 100, 50, and 25 ages at death produced with input parameter values of $m_0 = 0.053$, $\alpha = 0.0000095$, and $\beta = 3.626$. The corresponding value of ω is 0.082. Ages at death were generated by exposing each of N individuals to probabilities of death determined by the Weibull equation each 0.1 time unit until all N individuals had died. Relative values are the estimated parameter values divided by the input parameter values.

Value relative to model parameters	Number of ages at death (N)			
	1000	100	50	25
Initial mortality (m_0)	0.981	0.902	0.769	0.659
SD (m_0)	0.075	0.243	0.367	0.475
Scaling parameter (α)	1.297	10.72	48.12	320.5
SD (β)	0.847	19.13	57.80	620.2
Shape parameter (β)	1.005	0.909	0.857	0.768
SD (β)	0.061	0.184	0.373	0.441
Rate (ω)	1.028	1.031	1.075	1.133
SD (ω)	0.057	0.073	0.110	0.169
Maximum ω of 10	0.979	0.917	0.871	0.866
Minimum ω of 10	1.170	1.130	1.221	1.327

Table 2. Aging parameters and other life-history traits of the species included in this study. Sources of data were: body mass (Dunning 1993); incubation period (Ricklefs 1993); Gompertz growth rate (Starck and Ricklefs 1998). Original survival data for *Alectura*, *Syrmaticus*, *Pavo*, *Nycticorax*, and *Threskiornis* are from Comfort (1962). Data for species in natural populations are from Ricklefs (1998). In the "Species" column, M = male and F = female; male and female values were averaged for species in which both were reported separately; in the case of *Passerina* the two samples were for males in different localities. In the "Status" column, C = captive and N = natural. Values of $\beta = 3.00$ without standard errors (SE) represent fixed parameters used when the full Weibull model failed to converge.

	Genus	Species	Status	N	Oldest (yr)	m_0 (yr^{-1})	SE (m_0)	β	SE (β)	α	SE (α)	ω (yr^{-1})	Mass (g)	Inc. per. (days)	Growth rate (K, day^{-1})
1.	*Alectura*	*lathami*	C	34	.	0.019	0.007	3.00	.	6.00×10^{-4}	4.00×10^{-5}	0.155	2330	49.5	0.020
2.	*Chrysolophus*	*pictus*	C	71	13.4	0.174	0.004	4.31	0.48	1.03×10^{-6}	1.18×10^{-6}	0.074	625	22.0	0.038
3.	*Chrysolophus*	*amherstiae*	C	39	19.6	0.089	0.007	1.78	0.29	1.27×10^{-3}	1.03×10^{-3}	0.091	738	24.0	0.038
4.	*Syrmaticus*	*reevesi*	C	107	.	0.011	0.009	3.00	.	1.80×10^{-3}	3.00×10^{-4}	0.205	949	24.0	0.018
5.	*Pavo*	*cristatis*	C	80	.	0.111	0.007	3.00	.	1.10×10^{-3}	8.00×10^{-4}	0.181	3375	29.0	0.020
6.	*Cygnus*	*columbianus* M	N	32	18.0	0.036	0.030	3.00	.	3.89×10^{-5}	1.33×10^{-5}	0.079	6400	32.0	0.054
6.	*Cygnus*	*columbianus* F	N	27	18.0	0.009	0.055	3.00	.	1.05×10^{-4}	2.90×10^{-5}	0.101	5700	32.0	0.054
7.	*Lophodytes*	*cucullatus*	C	53	15.7	0.041	0.005	3.54	0.25	5.30×10^{-5}	3.38×10^{-5}	0.115	610	31.8	0.055
8.	*Ramphastos*	*toco*	C	62	16.2	0.092	0.003	4.96	0.37	9.04×10^{-7}	8.81×10^{-7}	0.097	540	15.5	0.135
9.	*Coracius*	*caudata*	C	72	13.7	0.105	0.013	1.60	0.25	6.65×10^{-3}	4.15×10^{-3}	0.145	108	21.0	0.169
10.	*Momotus*	*momota*	C	22	20.3	0.049	0.009	3.86	0.79	4.75×10^{-6}	1.07×10^{-5}	0.080	133	21.0	0.170
11.	*Dacelo*	*novaguineae*	C	47	26.8	0.116	0.004	1.89	0.79	1.32×10^{-4}	3.20×10^{-4}	0.046	305	25.0	0.098
12.	*Colius*	*striatus*	C	69	11.5	0.163	0.012	3.05	0.46	5.31×10^{-4}	5.42×10^{-4}	0.155	51	13.5	0.305
13.	*Ara*	*chloropterus*	C	42	26.7	0.000	0.012	1.43	0.23	2.91×10^{-3}	2.17×10^{-3}	0.091	1250	25.0	0.076
14.	*Ara*	*ararauna*	C	23	33.0	0.008	0.008	2.35	0.05	7.28×10^{-5}	1.22×10^{-4}	0.058	1125	25.0	0.086
15.	*Ara*	*macao*	C	25	37.1	0.007	0.006	2.35	0.39	5.08×10^{-5}	6.92×10^{-5}	0.053	1015	26.0	0.082
16.	*Tyto*	*alba*	C	33	16.7	0.065	0.017	1.65	0.42	3.39×10^{-3}	3.84×10^{-3}	0.117	524	30.8	0.116
17.	*Caloenas*	*nicobarica*	C	25	20.5	0.070	0.007	2.91	0.54	5.37×10^{-5}	8.24×10^{-5}	0.081	519	17.0	0.090
18.	*Balearica*	*regulorum*	C	71	27.2	0.033	0.005	2.14	0.22	2.80×10^{-4}	1.89×10^{-4}	0.074	3372	29.5	.
19.	*Grus*	*antigone*	C	36	41.8	0.036	0.006	1.79	0.41	1.83×10^{-4}	2.67×10^{-4}	0.046	8663	31.8	0.035
20.	*Vanellus*	*armatus*	C	48	18.6	0.112	0.012	1.78	0.45	1.59×10^{-3}	1.95×10^{-3}	0.098	156	29.0	0.078
21.	*Vanellus*	*spinosus*	C	25	12.7	0.048	0.011	2.98	0.39	3.79×10^{-4}	3.50×10^{-4}	0.139	152	23.0	0.078
22.	*Larosterna*	*inca*	C	49	26.0	0.069	0.002	3.24	0.29	8.90×10^{-6}	7.88×10^{-6}	0.064	180	25.4	0.093
23.	*Larus*	*canus*	N	5422	18.0	0.097	0.021	2.96	1.08	3.32×10^{-5}	0.35×10^{-5}	0.074	404	24.0	0.130
24.	*Rissa*	*tridactyla* M	N	160	12.0	0.196	0.014	3.00	.	2.31×10^{-5}	1.10×10^{-5}	0.069	421	27.0	0.120
24.	*Rissa*	*tridactyla* F	N	181	12.0	0.135	0.022	3.00	.	3.02×10^{-5}	1.72×10^{-5}	0.074	393	27.0	0.120
25.	*Haliaeetus*	*leucocephalus*	C	30	37.0	0.016	0.010	1.82	0.61	2.43×10^{-4}	5.12×10^{-4}	0.052	4740	35.0	0.066
26.	*Accipiter*	*nisus*	N	.	8.0	0.201	0.089	3.00	.	1.84×10^{-3}	3.20×10^{-5}	0.207	202	33.5	0.177
27.	*Nycticorax*	*nycticorax*	C	44	.	0.024	0.009	3.00	.	6.60×10^{-5}	1.00×10^{-5}	0.090	883	25.0	0.120
28.	*Eudocimus*	*ruber*	C	45	31.0	0.033	0.003	3.00	0.28	1.10×10^{-5}	1.00×10^{-5}	0.057	645	22.0	0.051
29.	*Threskiornis*	*aethiopicus*	C	30	.	0.000	0.000	3.00	.	5.20×10^{-3}	4.00×10^{-4}	0.151	1378	28.5	.
30.	*Eudyptila*	*minor*	N	246	16.0	0.195	0.077	3.00	.	3.13×10^{-4}	7.50×10^{-5}	0.133	1105	39.0	0.102
31.	*Puffinus*	*tenuirostris*	N	.	25.0	0.051	0.035	1.34	0.81	2.28×10^{-3}	6.13×10^{-3}	0.074	543	53.0	0.040
32.	*Diomedia*	*exulans*	N	1254	28.0	0.020	0.004	3.00	.	2.12×10^{-6}	3.00×10^{-7}	0.038	7650	78.0	0.022
33.	*Ficedula*	*hypoleuca* F	N	953	7.0	0.664	0.032	3.14	0.36	2.35×10^{-3}	1.64×10^{-3}	0.232	12	14.0	0.340
33.	*Ficedula*	*hypoleuca* M	N	.	.	0.778	0.045	3.47	0.54	1.35×10^{-3}	1.42×10^{-3}	0.228	12	14.0	0.340
34.	*Leucopsar*	*rothschildi*	C	86	22.5	0.053	0.003	3.63	0.24	9.50×10^{-6}	6.55×10^{-6}	0.082	70	13.0	0.192
35.	*Parus*	*major* F	N	.	7.0	0.756	0.085	3.00	.	3.43×10^{-3}	4.80×10^{-4}	0.242	19	12.5	0.316
35.	*Parus*	*major* M	N	.	7.0	0.989	0.103	3.00	.	2.93×10^{-3}	5.90×10^{-4}	0.233	19	12.5	0.316
36.	*Parus*	*atricapillus*	N	150	6.0	0.361	0.059	3.00	.	1.10×10^{-3}	5.00×10^{-4}	0.182	11	12.5	0.326
37.	*Turdoides*	*squamiceps* F	N	71	6.0	0.183	0.211	1.18	1.31	6.20×10^{-2}	1.55×10^{-1}	0.279	74	13.5	0.272
37.	*Turdoides*	*squamiceps* M	N	69	7.0	0.115	0.031	3.39	1.10	8.40×10^{-4}	1.68×10^{-3}	0.199	74	13.5	0.272
38.	*Leiothrix*	*lutea*	C	58	9.5	0.053	0.032	1.28	0.23	4.06E-02	2.04×10^{-2}	0.246	22	14.0	0.250
39.	*Chloebia*	*gouldiae*	C	80	6.0	0.254	0.006	7.71	0.49	3.88×10^{-6}	3.15×10^{-6}	0.239	12	13.0	0.210
40.	*Passerina*	*cyanea* M (Res.)	N	184	6.0	0.437	0.041	2.59	0.76	3.69×10^{-3}	5.24×10^{-3}	0.210	15	12.5	0.374
40.	*Passerina*	*cyanea* M (Niles)	N	173	6.0	0.454	0.016	3.00	.	1.71×10^{-3}	1.40×10^{-4}	0.203	15	12.5	0.374

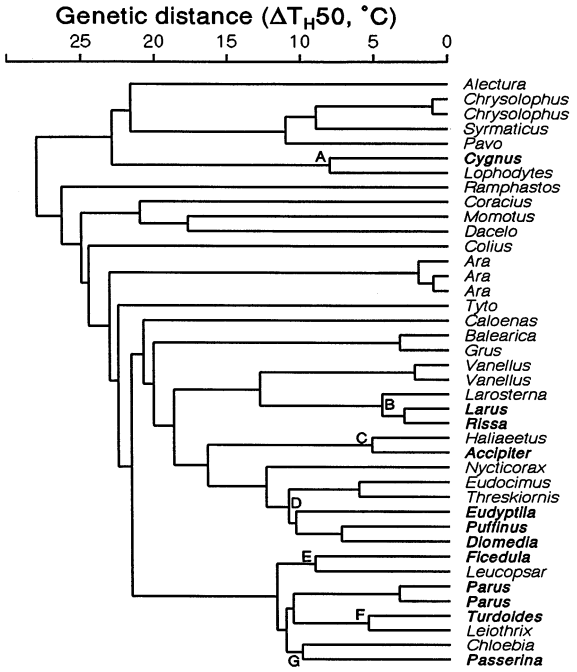

Fig. 1. Phylogenetic hypothesis for the taxa of birds included in this analysis. Taxa are identified by their genus names. The phylogeny is based on genetic distances obtained through DNA hybridization (Sibley and Ahlquist 1990). Genetic distance is the difference in melting point temperatures of homo- and heteroduplex DNA. Taxa represented by natural populations are shown in boldface. Letters indicate contrasts between pairs of lineages represented by natural and captive populations. Higher taxonomy is after Sibley and Ahlquist (1990).

lineages contrasting captive and natural populations. The nodes connecting these sister lineages are indicated by the letters A–G in Fig. 1. Profiles of these contrasts (Fig. 2) show that initial mortality rate is significantly lower in captive than in natural populations, as one would expect, but that the rate of actuarial senescence did not differ significantly. A two-way analysis of variance (paired comparisons test), in which the main effects were pairs of lineages (random) and captive versus natural (fixed), revealed a significant effect of captivity on m_0 ($F_{1,6} = 7.7$, $P = 0.03$). The geometric mean value of m_0 in captive populations (0.065 $yr^{-1} \pm 0.060$ SD) was 29% of the geometric mean of natural populations (0.26 $yr^{-1} \pm 0.24$ SD). The only comparison that showed the reverse trend was between the Bewick's Swan (natural) and the Hooded Merganser *Lophodytes cucullatus* (captive), for which there was also a pronounced difference in body mass (6050 versus 610 g). Rate of actuarial senescence (ω) did not differ significantly between natural (0.164 $yr^{-1} \pm 0.076$ SD) and captive (0.126 $yr^{-1} \pm 0.074$ SD) populations ($F_{1,6} = 1.4$, $P = 0.28$). In two cases (c and e) in which the captive member of the pair of lineages exhibited lower ω, it was an order of magnitude larger in body mass. The lineage pair itself was not a significant effect for either initial mortality rate or rate of actuarial senescence ($F_{6,6} < 2.1$, $P > 0.20$). The observation that ω does not differ between natural and captive populations, in spite of a nearly four-fold difference in m_0, is consistent with the prediction of the intrinsic-mortality hypothesis that captive and natural populations have similar rates of actuarial aging, particularly when body size is closely matched.

Because aging-related mortality (ω) apparently is not affected by captivity, natural and captive populations may be combined to examine how the rate of actuarial senescence is related to other aspects of the life history: body mass, length of the period of embryonic development, and postnatal growth rate of the chick. Larger birds have lower mass-specific metabolic rates (Calder 1984, Ricklefs et al. 1996) which may influence rate of senescence through the production of reactive forms of oxygen (Harman 1982, Beckman and Ames 1998). Embryonic and postnatal development, like rate of aging, measure the duration of different components of the life spans of individuals (Calder 1984), and although the mechanisms are not understood, they have been linked to variation in life span in birds (Ricklefs 1993). For these variables, phylogenetically independent con-

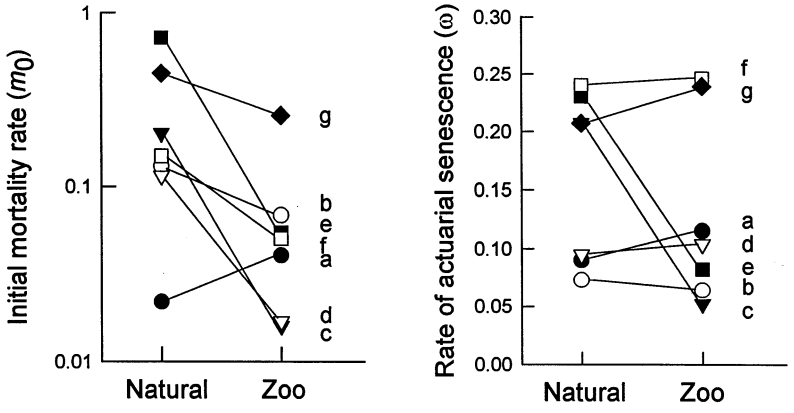

Fig. 2. Initial mortality rates (left) and rates of actuarial senescence (right) contrasted between seven pairs of sister lineages (joined by lines), of which one is represented by natural populations and the other by captive populations. Letters a–g correspond to the nodes identified in Fig. 1.

Table 3. Simple correlations of phylogenetically independent contrasts (PICs) for rate of aging (ω) with PICs of the natural logarithms of body mass, incubation period, and postnatal growth rate.

Contrast	N	SD	Correlation with ω	P
Rate of aging (w)	29	0.55	–	–
Body mass	29	1.16	−0.42	0.023
Incubation period	29	0.24	−0.07	0.70
Postnatal growth rate	27	0.49	0.25	0.21

trasts (PICs) (Garland et al. 1992) may be calculated more widely within the phylogenetic tree of the species included in this sample. Twenty-nine such contrasts were calculated here, including all but the deepest and most poorly resolved nodes in the phylogenetic tree in Fig. 1, and representing 36 of the 40 species for which ω has been calculated.

Contrasts in ω were significantly correlated only with contrasts in body mass (Table 3). The regression equation for rate of aging on body mass is PIC(ω) = 0.004 (± 0.093 SE) − 0.215 (± 0.079) PIC(mass) ($F_{1,27} = 7.3$, $P = 0.012$, $R^2 = 0.23$). Neither incubation period nor the growth rate of the chick had significant simple correlations with ω, nor were they significant effects in a multiple regression model that also included body mass. Analysis of the natural logarithms of the species data gave the same result (Fig. 3, right). In a multiple regression model, ω was significantly related only to mass ($P = 0.006$; incubation period and postnatal growth rate, $P > 0.25$). The least-squares regression for the combined data was ln(ω) = − 1.167 (± 0.232 SE) − 0.182 (± 0.038) ln(mass) ($F_{1,38} = 23.4$, $P < 0.0001$, $R^2 = 0.381$). The allometric constant (− 0.18) was similar to that obtained for contrasts (− 0.22). Analysis of covariance detected no significant difference in the relationship between natural and captive populations (status × mass interaction, $F_{1,36} = 1.0$, $P = 0.32$; status main effect with interaction deleted, $F_{1,37} = 0.75$, $P = 0.39$).

The species data were also used to confirm that initial mortality rate (m_0) among captive populations was lower than that among natural populations when corrected for body mass (Fig. 3, left). In an ANCOVA of log-transformed data, including captive versus natural as the main effect and body mass as the covariate, the status × mass interaction was not significant ($F_{1,40} = 3.2$, $P = 0.08$), indicating a common regression slope for both groups. With the interaction term removed from the model, the slope of the regression was − 0.43 (0.06 SE; $F_{1,41} = 51$, $P < 0.0001$) and the intercepts of the regression for captive (− 0.17) and natural (0.17) populations differed significantly from each other ($F_{1,41} = 11$, $P = 0.002$). The intercept for captive populations was 45% that of natural populations, which is somewhat higher than the value of 29% obtained in paired comparisons.

Discussion

The absence of a response in actuarial senescence when extrinsic mortality is reduced in captivity indicates that deaths associated with aging are caused by intrinsic factors that are lethal in their own right, as is largely the case with the human population (Coni et al. 1992, Hayflick 1994). In fact, rate of aging in human populations is independent of differences in the baseline mortality rate among nations (Strehler and Mildvan 1960). This contrasts with the idea that senescence weakens individuals and makes them more vulnerable to extrinsic causes of mortality in old age. This may happen, but the analyses reported here provide no such indication. To the contrary, they suggest that birds retain a high level of physical fitness to old age, eventually succumbing to intrinsic disease processes that kill rapidly. If physiological function did decrease with age, this could be balanced by increasing experience and acquired immunity, resulting in approximately constant extrinsic mortality through life. Although causes of death are determined infrequently in captive birds, and rarely in natural populations, captive populations appear to provide valid models for investigating aging processes in nature. Aging-related death rates appear to be unaffected by captivity, although different causes of death conceivably could arise from conditions of captivity. The lower extrinsic mortality rates of captive populations result in a larger proportion of individuals living long enough to die of old age. Although variation in rate of aging among species appears to reflect evolved differences, the capacity to postpone physiological decline evidently is constrained by intrinsic upper limits to potential life span in warm-blooded vertebrates. The nature of those limits and the evolutionary forces that influence them are not clear.

Fewer data are available for aging-related changes in reproduction in natural and captive populations and there appears to be less evidence for reproductive senescence than for mortality senescence (Holmes and Austad 1995a, b). Although records of reproduction are kept for birds in zoos, many birds are not bred or do not have the opportunity to breed because of caging arrangements or unavailability of suitable mates. If birds were to maintain a high level of fitness to old age, one would not expect a marked decrease in fecundity with age among birds that were capable of reproduc-

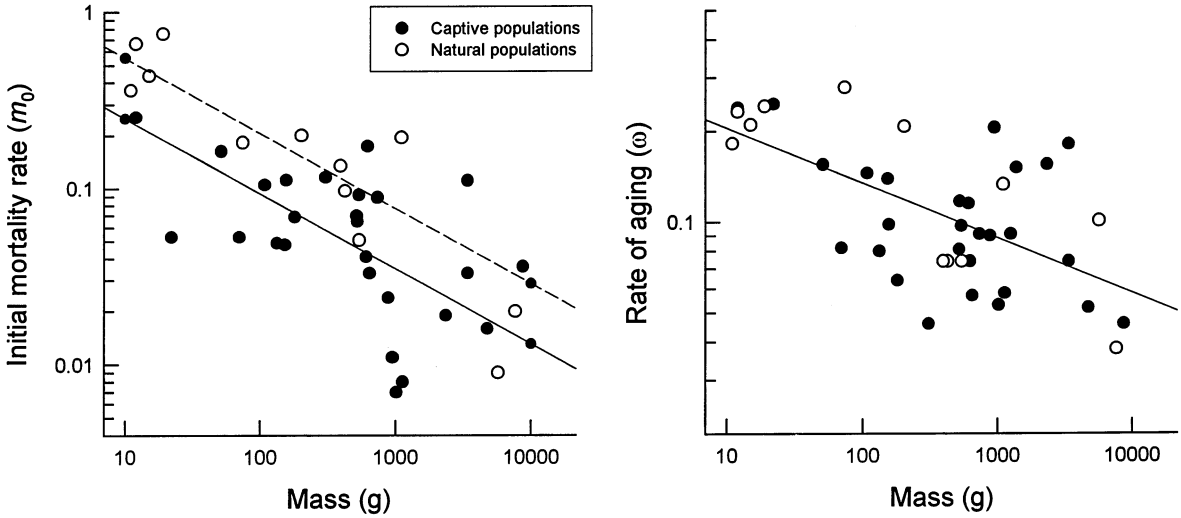

Fig. 3. Initial mortality rate (m_0) and rate of aging (ω) as a function of body mass in natural and captive populations.

tion. Intrinsic factors might preclude reproduction after a certain age, but an individual's ability to provide for offspring should not be diminished with age. Reproductive success has been found to decline with age in some studies (e.g., McCleery and Perrins 1989) but increase with age in others (Pugesek 1981). Unfortunately, it is not possible at present to assess general patterns of age-specificity in reproduction with the limited data available from natural or captive populations.

A direct relationship between rate of actuarial senescence (ω) and extrinsic mortality rate (m_0) that is independent of body mass has been established for natural populations of birds and mammals (Ricklefs 1998). In the sample of natural populations of birds included in this study, phylogenetically independent contrasts of the natural logarithms of ω and m_0 are strongly correlated (Fig. 4). However, extrinsic mortality rate (m_0) is also strongly related to mass in this sample. Body mass and m_0 explain variation in ω equally well statistically, and neither explains variation in ω independently of the other.

Larger body size is associated with lower extrinsic mortality and slower aging. Whether the connection between rate of aging and size is a direct consequence of physiological scaling parameters or reflects evolutionary responses to selection to reduce aging-related deaths in populations with older age structures, is not clear. If, on one hand, aging is allometrically related to body size through various physiological mechanisms, then, as body size increases, extrinsic mortality decreases (allometric constant, -0.48; Fig. 4, legend) relatively faster than the rate of aging decreases (-0.18), thereby exposing increasing numbers of individuals to aging-related death. If, on the other hand, rate of aging is under independent genetic control, then as body size increases and the age structure of the population increases, stronger selective pressure to postpone aging is constrained by intrinsic physiological or genetic limits to the evolutionary response.

The statistical effects of body mass and extrinsic mortality on the rate of senescence have been disentangled in analyses that combine mammals and birds

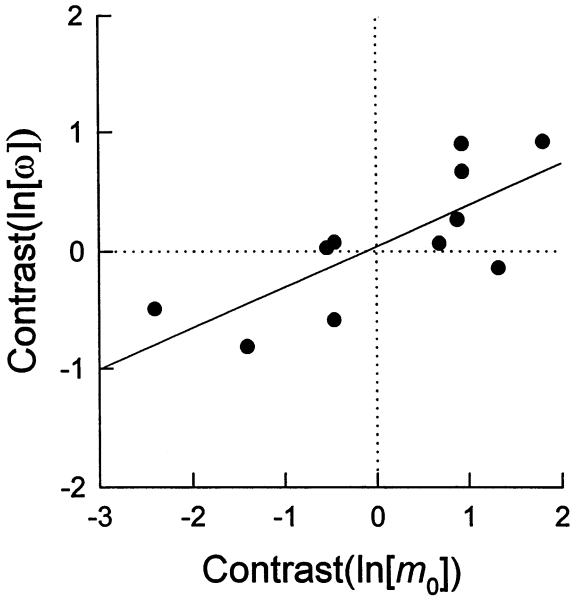

Fig. 4. Correlation between contrasts in rate of actuarial senescence (ω) and initial mortality rate (m_0) among 12 natural populations. The relationship is described by PIC(ln[ω]) = 0.04 ($\pm$0.12 SE) + 0.35 ($\pm$0.10 SE) PIC(ln[m_0]) ($F_{1,9} = 12.5$, $P = 0.006$, $R^2 = 0.58$). The relationship between the natural logarithms of the species values is similar: ln(ω) = -1.30 ($\pm$0.20 SE) + 0.41 ($\pm$0.09 SE) ln(m_0) ($F_{1,10} = 20.1$, $P = 0.001$, $R^2 = 0.67$). Extrinsic mortality rate (m_0) is related to mass in this sample (not shown) by ln(m_0) = 0.68 ($\pm$0.40 SE) $-$ 0.48 ($\pm$0.07 SE) ln(mass) ($F_{1,10} = 46.1$, $P = 0.0001$, $R^2 = 0.83$).

(Holmes and Austad 1995a, Ricklefs 1998). For a given body mass, birds have lower extrinsic mortality and correspondingly slower aging. Thus, assuming that differences between birds and mammals *per se* are not physiologically relevant to the aging process, extrinsic mortality controls rate of aging through selection and evolutionary response rather than body mass controlling aging through direct intrinsic physiological consequences of size.

It has been argued that few individuals survive long enough in natural populations of birds to experience aging-related loss of function, not to mention death from aging-related processes. This is reinforced by the observation that most birds continue to reproduce until they die (Holmes and Austad 1995b). Birds have no menopause and there is little evidence of declining condition in old individuals of many species (Newton 1989, Finch 1990, Holmes and Austad 1995a, Ottinger et al. 1995), which is consistent with the implication of this study that birds maintain high levels of fitness into old age. In fact, however, in populations with lower levels of extrinsic mortality, more deaths are related to actuarial senescence and fewer are related to the extrinsic (initial) component of Weibull mortality (Botkin and Miller 1974). The proportion of aging-related deaths according to the Weibull model is

$$P_S = \int_{x=0}^{\infty} \alpha x^{\beta} l_x \, dx \qquad (2)$$

(Ricklefs 1998). Evaluated with parameters from natural populations of birds, P_S is less than 10% when $m_0 = 0.50\ yr^{-1}$, as in many small songbirds and gamebirds, but increases to over 50% when m_0 is below 0.05 yr^{-1}, which is typical of many seabirds. The increasing proportion of senescent deaths with decreasing m_0 indicates that evolutionary responses to delay physiological aging cannot compensate for the increasing exposure of individuals to aging-related causes of mortality as the proportion of old-age individuals increases.

Finally, it is important to consider what is meant by extrinsic and intrinsic sources of mortality (Carnes et al. 1996). In this study, I have defined these in terms of the Weibull equation for age specific mortality rate, $m_x = m_0 + \alpha x^{\beta}$ (cf. Gage 1991). Of course, causes of mortality and how these change with age are poorly known for birds. When one defines extrinsic mortality as death caused by environmental factors whose force is independent of age (e.g., predation and adverse weather), the magnitude of this mortality component should be revealed by the death rate of young adults. Intrinsic mortality is defined in this study as death directly related to physiological factors arising as an individual ages, whether resulting from inherited genetic factors expressed at progressively older age, acquired genetic damage, or physiological deterioration of cells and tissues. A broader definition of intrinsic mortality would include death resulting from inherited lethal genes regardless of the age of expression (Pearl and Miner 1935, Bourgeois-Pichat 1978). Thus, congenital conditions that may cause death might be expressed in the initial mortality rate if these conditions make an individual more vulnerable to extrinsic mortality factors. Rate of death due to this component of "intrinsic" mortality presumably would decrease with age as the proportion of individuals carrying lethal genetic factors decreased in the population. Death due to extrinsic factors might also decrease with age owing to accumulated experience, but this would not be evident from the estimated parameters of an aging model. Clearly, the separation of mortality into intrinsic and extrinsic components is a theoretical construct at this point, although supported in its general form by evidence from studies on humans and laboratory animals. This distinction allows us to predict the responses of both intrinsic and extrinsic mortality to captivity from theories concerning the increase in mortality rate with age. In this case, the data support the idea that this increase is caused directly by the lethal consequences of physiological deterioration rather than indirectly through increased vulnerability of older individuals to extrinsic mortality factors.

This result focuses attention on the need for more research on the causes of death in natural and captive populations, as well as the physiological condition of individuals of different age. Regardless of the outcome of such studies, detailed comparisons of aging in captive populations of birds and mammals will likely provide a powerful tool to help us understand the biological basis of senescence in natural populations and the potential for modifying the rate of senescence in humans.

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