Exploring the Genetics of Aging in a Wild Passerine Bird

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Abstract: Senescence is the decline in survival and reproduction as an organism ages and is known to occur in collared flycatchers Ficedula albicollis. We consider annual fitness (the estimated genetic contribution that an individual makes to next year’s gene pool) as a measure of age-specific fitness. We apply a restricted maximum likelihood linear mixed-model approach on 25 years of data on 3,844 male and 4,992 female collared flycatchers. Annual fitness had a significant additive genetic component (h² of about 4%). Annual fitness declined at later ages in both sexes. Using a random regression animal model, we show that the observed age-related phenotypic changes in annual fitness were not present on the additive genetic level, contrary to predictions of genetic hypotheses of senescence. Our study suggests that patterns of aging in the wild need to be interpreted with caution in terms of underlying genetics because they may be largely determined by environmental processes.

Keywords: evolution, quantitative genetics, animal model, senescence, mutation.

Phenotypic senescent declines have been shown in a variety of traits in the wild (e.g., Gustafsson and Pärt 1990; Orell and Belda 2002), but a quantitative assessment of possible genetics behind these patterns is typically lacking (but see Charmantier et al. 2005). Senescence theories recognize that selection in later age classes is proportionally much lower than in young age classes because there are inevitably fewer older individuals alive to express the trait (e.g., Hamilton 1966; Baudisch 2005). Quantitative genetic theory predicts that a senescent decline will be coupled to an age-related change on the genetic level (Rose 1991; Charlesworth 2001), but little is known about the genetics of aging in the wild. Laboratory-based studies have documented that aging is affected by genes (e.g., Charlesworth and Hughes 1996), but it is unclear to what extent such findings can be extended to a natural population, because most laboratory studies are carried out under standardized conditions and on inbred lines (Rose 1991). Empirical tests of whether aging is heritable under natural conditions are therefore important.

Although senescence theories apply to any given trait, studies of senescence need to be based on a metric that translates unambiguously into individual performance and evolutionary dynamics. The majority of studies focus on survival, typically considered as life span (Rose 1991), but survival alone is not a complete estimate of fitness since survival and reproduction possibly undergo a trade-off (e.g., Lessels 1991). Here, we focus on an annual measure of individual fitness, the sum of an individual’s survival and its reproductive output. An attractive feature of annual fitness is that it allows the assignment of an age-dependent fitness value to an individual and thereby the study of senescence on the phenotypic and the genetic level. Evolution requires heritable variation. Iteroparous individuals must differ in their rate of aging, and aging must be heritable in order for senescence to evolve (Rose 1991; Partridge and Barton 1996). Whereas the standard genetic model views an individual’s breeding value as constant as age increases (fig. 1A), genetic theories of senescence predict changes in the breeding values as age increases (fig. 1B, 1C; Medawar 1952; Williams 1957).

We here analyze age-specific annual fitness based on an extensive data set of a wild population of individually marked collared flycatchers Ficedula albicollis. Although this species is fairly short lived, it experiences senescence in its reproductive output (Gustafsson and Pärt 1990). Experimental increase of reproductive output of a 1-year-
A, The standard model assumes that an individual’s breeding value is constant. B, Mutation accumulation in older age classes leads to a family-specific decline in breeding values, predicting an increase in the additive genetic variance (the variance in breeding values) with age. C, Antagonistic pleiotropy theory predicts a negative genetic covariance in the breeding values for early and late life. In addition, additive genetic variance may change over age classes.

old causes a steeper senescent decline in offspring production at later ages (Gustafsson and Pärt 1990). This experiment suggests pleiotropic effects between early and late life are possible. Furthermore, aspects of the soma senesce in collared flycatchers, since maternal immune function capacities decline with age (Cichon et al. 2003).

Annual fitness quantifies the sum of survival and reproduction per age class and therefore is the proper metric for the rate of aging (sensu Partridge and Barton 1996). We show that phenotypic values of annual fitness decline in older age classes, and we use a random regression animal model (RRAM) to test for evidence of any age-related genetic effects (fig. 1) in collared flycatcher annual fitness. An RRAM models the individual genetic effect as a continuous function of age (Meyer 1998; Schaeffer 2004). This approach is based on the infinite dimensional model (Kirkpatrick et al. 1990). The RRAMs have been extensively used in the context of animal breeding studies, but they also facilitate estimation of age-specific genetic variances (and covariances) in data sets characteristic of natural populations (Wilson et al. 2005). This approach has—to our knowledge—not been used to assess patterns of senescence in the wild.

**Study Species and Methods**

We have studied a collared flycatcher population breeding in nest boxes on the Swedish island of Gotland since 1980. Each year, practically all females and the majority of males were trapped at the nest box, and all offspring and unringed adults were ringed with aluminum rings in order to allow lifelong individual identification. The study area consisted of a set of 16 forest patches in the southern part of Gotland, where intense monitoring of all pairs has taken place (core patches), and eight additional forest patches, where monitoring has been less intensive (surrounding patches). We considered data from all areas but included a variable to account for whether an observation was from a core patch or a surrounding patch in all statistical models (discussed below). Focusing only on the core patches produces qualitatively the same results (cf. Qvarnström et al. 2006) but is less powerful for the study of age-related change.

Parental (apparent) survival (p) was based on whether the individual was caught again after the breeding season. Recruitment (r) was based on recapture of an offspring of either sex as a breeding adult at any time in the future. When a bird was not caught at time t but was caught in a later season (i.e., known to have survived), its annual fitness for time t was not defined. Annual fitness was thus always based on records of individuals whose life-history actions in a given year could be verified. For a description of annual fitness in this species, see appendix A. Errors in apparent survival and recruitment are probably minimal, because this population is characterized by high adult return rates and local recruitment of offspring (Pärt and Gustafsson 1991).

Age of a captured adult was based on either its known year of hatching when ringed as an offspring or plumage characteristics (either yearling or adult) for unringed individuals. When first caught, unringed individuals with

\[
W_{i,t} = p_i + \frac{1}{2} r_{i,o}
\]

where \( p_i \) is the survival of the individual (0 or 1) and \( r_{i,o} \) is the total number of recruits of both sexes that were produced at time t and enter the breeding population at some time step in the future. The factor one-half accounts for Mendelian inheritance, since offspring of either sex inherit half of an individual’s genes. Such an annual estimate of individual fitness on the annual timescale has been used in evolutionary studies (e.g., Gustafsson 1987; Coulson et al. 2006). A theoretical outline for annual fitness is provided in appendix A in the online edition of the *American Naturalist*.

### Material and Methods

#### Evolutionary Dynamics

An individual’s genetic contribution to the next time step can be considered the sum of the survival of its fully related self and the recruitment into the breeding population of those offspring sharing its genes. Hence, individual i at time t has an annual fitness

![Figure 1: Schematic presentation of three different quantitative genetic predictions of how the breeding values for annual fitness (a.) vary over age classes. Lines illustrate three individuals and here, for convenience, are assumed to be linear but need not be so for the theories to apply. A. The standard model assumes that an individual’s breeding value is constant. B, Mutation accumulation in older age classes leads to a family-specific decline in breeding values, predicting an increase in the additive genetic variance (the variance in breeding values) with age. C, Antagonistic pleiotropy theory predicts a negative genetic covariance in the breeding values for early and late life. In addition, additive genetic variance may change over age classes.](image)
adult plumage were assumed to be 2 years old because most individuals (males, 90.0% [1,600/1,777] of recruits; females, 92.1% [1,812/1,968] of recruits) start to breed before their third year in life.

This population has been subject to experimental studies in the past. We therefore excluded from the data set all breeding attempts in which an experiment was conducted that may have affected an individual’s survival or its offspring’s recruitment (e.g., clutch and brood size manipulation, moving a male between areas, swapping clutches to create a time advance or delay). We included all data on nests where the offspring were cross-fostered without any further manipulation (462 for males, 627 for females) because we wanted to maximize the power of our approach in terms of sample size. Hence, annual fitness considers the individual parent’s own propensity to survive and raise successful offspring. If an individual bred twice during one season (laying again or, for males, polygynous breeding), the total number of recruits from both breeding attempts was used (males: 206 cases of polygyny, with 25 successful; females: 72 double breeding attempts, with two successful). Finally, breeding attempts up to and including 2002 were included in order to allow for the detection of most recruits by 2004. These restrictions resulted in a data set of 7,405 breeding attempts of 4,992 females and 5,571 breeding attempts of 3,844 males. Most of the data (5,731 of the other ones (i.e., effects are additive) and that each random effect specifies the deviation from the overall fixed-effect mean with a mean of zero and variances to be estimated.

Our basic model had the following fixed effects: constant (μ), age (AGE fitted as an eight-level factor), and whether the individual bred in a central study plot (CSP; two-level factor). As random effects, we included the additive genetic (ai), individual-specific (permanent environment PEi; Lynch and Walsh 1998), year-specific (YR), and study plot-specific effects (plot). The individual-specific (or “permanent”) environment effect includes among-individual sources of variance that are conserved across records but are not due to additive effects (e.g., maternal environment effects, any nonadditive genetic effects). The additive genetic effect on individual i, ai, was assumed to be normally distributed with mean of zero and variance of σ2i (the additive genetic variance). This is estimated from the variance-covariance matrix of additive genetic effects that is equal to Ασ2i, where Α is the additive numerator relationship matrix containing the individual elements Aij = 2Θij, and Θij is the coefficient of coancestry between individuals i and j (obtained from the pedigree structure). All other random effects and residual errors (εi) were assumed to be normally distributed, with zero means and variances to be estimated. Residual errors were assumed to be uncorrelated within individuals across measurements. The phenotype of an individual i at time t is then specified as

\[ w_{i,t} = \mu + \text{AGE} + \text{CSP} + a_i + \text{PE}_i + \text{YR}_t + \epsilon_{i,t}. \]  

In order to describe age-specific variation on the genetic level, we fitted a random regression animal model (RRAM), an explicit implementation of the infinite-dimensional model, under which an individual’s age-specific breeding value is described by a continuous function of age. More specifically, we modeled the functions describing the individual-specific age-related change in the additive effect (f(ai, X)) as first- and second-order Legendre polynomial functions of age (Kirkpatrick et al. 1990). Age was standardized to a scale from −1 (1 year old) to 1 (8 years old), and separate analyses were again performed for males and females. Annual fitness w at time t for individual i of age X was then written as

\[ w_{i,t} = \mu + \text{AGE} + \text{CSP} + f(ai, X) + \text{PE}_i + \text{YR}_t + \epsilon_{i,x}, \]  

where \( f(ai, X) \) was the random regression function specifying how the age-specific breeding value of i deviates from the mean depending on age and \( \epsilon_{i,x} \) was the residual
error at age $X$. The RRAM was fitted with both an assumed constant residual variance across age classes and multivariate error structures (the latter allowing residual, or environmental, variance to change with age; see Wilson et al. 2006). Variance components associated with additional random effects were assumed constant with age.

Under model 3, the additive variance-covariance matrix of coefficients specifying the random regression functions was directly estimated. This was transformed to give the additive (co)variance matrix of age-specific annual fitness traits (e.g., Schaeffer 2004) and associated confidence limits (Fischer et al. 2004).

The RRAM’s ability to view additive genetic merit as a function of age makes it more powerful than the classic approach in which each age class is considered separately (character-state approach). The character-state approach needs to estimate the entire additive genetic variance-covariance matrix across age classes and can for each age class consider only the resemblance between relatives that expressed the trait at that particular age. In contrast, a RRAM uses a maximum-likelihood approach on information on age-specific trait expression of all relatives simultaneously to find the most parsimonious continuous function describing the change in additive genetic variance and covariance over age classes.

**Pedigree Information and Testing of Random Effects**

In all animal models, the additive genetic effect was estimated using pedigree information based on 3,715 individuals that recruited back into the breeding population and for which at least one genetic parent was known, with the remaining individuals considered as base population. Specifying a maternal effect (identity of the mother as a random effect) did not change the additive genetic effects and showed no evidence of maternal variance, and it was therefore not included in the modeling approach.

We tested the statistical significance of the model’s random effects by a conservative likelihood ratio test where the number of degrees of freedom equaled the number of (co)variance terms removed (Pinheiro and Bates 2000). Linear mixed models were implemented using a restricted maximum likelihood (REML) approach in the program S-Plus 6.1 (Insightful) and in ASReml (VSN International). These mixed models were based on normally distributed errors. We verified an absence of any pattern in the plot of residual versus fitted values and investigated the distribution of the models’ residuals for deviations from normality using Wilkinson-Shapiro statistics (statistic $W$, which is 1 in case of a normal distribution). All confidence limits reported are standard errors unless mentioned otherwise. ASReml calculates standard errors for variance components and for the ratios of variance components using the delta method (see, e.g., Lynch and Walsh 1998).

**Results**

**Age-Specific Variation in Phenotypic Annual Fitness**

There was significant variation across study plots, years, and estimated age classes in annual fitness for both sexes (table 1). The model’s residuals approximated a normal distribution (Wilkinson-Shapiro, males: $W = 0.95$; females: $W = 0.93$). In general, annual fitness peaked at age 2 for females but stayed equal for the first three age classes in males (fig. 2). Annual fitness declined after the first year in both sexes (weighted regression on BLUE for ages 2–8 [fig. 2], males: $b = -0.041 \pm 0.010$, $t = -4.1$, $P = .009$; females: $b = -0.046 \pm 0.012$, $t = -3.7$, $P = .014$). We therefore conclude that annual fitness showed senescence in both sexes.

**Variance Components of Annual Fitness**

We used an animal model to further partition the phenotypic variances. Most of the variance in annual fitness was residual (unexplained) variance (females, 90.2%; males, 91.9%; table 2). The residuals approximated a normal distribution (Wilkinson-Shapiro, males: $W = 0.94$;

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**Table 1: Exploration of age-specific effects on phenotypic annual fitness in males and female collared flycatchers**

<table>
<thead>
<tr>
<th>Term</th>
<th>Fixed effect:</th>
<th>Random effect:</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Constant</td>
<td>Individual</td>
<td>$\chi^2 = 3.39$, $P = .07$</td>
</tr>
<tr>
<td>Study plot</td>
<td>$F = 10.7$, df = 23, 1,697</td>
<td></td>
<td>$\chi^2 = 4.86$, $P = .03$</td>
</tr>
<tr>
<td>Year</td>
<td>$F = 8.81$, df = 22, 1,697</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>$F = 2.0$, df = 7, 1,697</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coefficient</td>
<td>$0.001$</td>
<td></td>
<td>$&lt;.001$</td>
</tr>
<tr>
<td>Test</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$P$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Spatial (study plot) and temporal (year) differences are accounted for, as well as the repeated measures (individual random effect). Age-specific coefficients are presented in figure 2. For the random effect $\chi^2$ test, there is one degree of freedom.
Figure 2: Best linear unbiased estimate (BLUE) of annual fitness for age classes 1–8. BLUES are derived from the linear mixed-model in table 1 and indicate the deviation from the overall fixed-effect mean annual fitness (constant in table 1), corresponding in this plot to the first age class and indicated by a dashed horizontal line. Values for males are denoted by circles filled with black connected by a solid line; values for females are denoted with gray squares connected by a dashed line. Error bars denote standard errors, but only the positive direction is plotted for the last age class. The number of observations are, for age class 1–8, 3,229, 2,376, 1,001, 478, 211, 82, 22, and 6 (made on 4,992 females); and for males, 1,896, 2,202, 846, 379, 157, 67, 18, and 6 (made on 3,844 males).

In females, fitting the additive genetic effect as a first-order Legendre polynomial function of age was not a significant improvement on fitting it as a constant (i.e., the conventional animal model described above; table 2). Although clearly statistically insignificant, a pattern of additive genetic variance in female annual fitness increasing with age was shown by the estimated first-order Legendre polynomial (app. B in the online edition of the American Naturalist). In males, there was clearly no effect, because the variance associated with the first-order term of the polynomial function being estimated was zero (with the model constrained to positive parameter space). Model likelihoods were not significantly improved by the use of second-order functions, and convergence problems were encountered when attempting to fit models in which residual variance varied with age. We therefore conclude that the constant additive genetic and residual model discussed above provided the most parsimonious description of the data.

Discussion

Variance Components of Annual Fitness

We studied variation in annual fitness, the annual genetic contributions that individuals make to the breeding population, calculated as the sum of an individual’s own apparent survival and half the number of its offspring that recruited later in life. There is a substantial variation in annual fitness in the collared flycatcher. Not surprisingly, most of this variation was due to residual (unexplained) effects, but we also found evidence of significant additive genetic, spatial, and temporal effects. Mixed modeling revealed that, phenotypically, annual fitness declines in later ages on the individual level. Using random regressions, a statistical technique that implements infinite-dimensional modeling (Kirkpatrick et al. 1990; Schaeffer 2004), we show that this phenotypic decline does not leave a signature on the genetic level. That is, individuals differ in their breeding values for annual fitness, but these breeding values do not vary over age classes (fig. 1A as opposed to fig. 1B, 1C).

We find significant additive genetic variance in annual fitness in both sexes. The standardized genetic annual fitness gives the expected relative change in population mean annual fitness (Burt 1995; app. A) and is around 4% in collared flycatchers. This value sets an upper rate to expected rates of evolutionary change in phenotypic (Falconer 1981) or molecular characters (Barton 1995), including changes due to indirect sexual selection (Kirkpatrick and Barton 1997). In general, these values are expected to be small and to fall between 1% and 10% (Burt 1995).

As a description of the environment, we have here fo-
Table 2: Standard and random regression animal models of annual fitness, for male and female collared flycatchers

<table>
<thead>
<tr>
<th>Term</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate (× 10^{-2})</td>
<td>Test (χ^2)</td>
</tr>
<tr>
<td>Standard model:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Additive</td>
<td>1.7 ± .7</td>
<td>5.3</td>
</tr>
<tr>
<td>Permanent</td>
<td>0.00</td>
<td>0</td>
</tr>
<tr>
<td>Year</td>
<td>1.4 ± .5</td>
<td>121.1</td>
</tr>
<tr>
<td>Area</td>
<td>.77 ± .4</td>
<td>37.9</td>
</tr>
<tr>
<td>Random regression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>animal model:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First-order Legendre</td>
<td>.13</td>
<td>.94</td>
</tr>
</tbody>
</table>

Note: Age (in eight classes) is used as a fixed effect. The standard model partitions variance in additive genetic, permanent environmental, and year-specific and area-specific variation. Estimated variances are provided only for statistically significant components. The random regression animal model (RRAM) is given by equation (2), assuming first-order Legendre polynomials of the additive genetic effect, keeping the other variance components the same as in the standard model. Higher-order polynomials did not lead to a better fit. The significance of terms in the standard model are tested for by testing twice the decrease in log likelihood after dropping a term (likelihood ratio test), which is distributed as a χ^2 value with one degree of freedom. The significance of the random regression term is tested by a likelihood ratio test compared to the standard model, which has two degrees of freedom.

...cused on the most important broadscale environmental factors that are shared by many individuals (i.e., year and study plot). Study plots are not homogeneous, and there may be, for example, systematic differences on the level of territories (nest boxes) occupied by individuals within study plots. Such spatially small-scale environmental effects are likely to be random over the pedigree and therefore do not affect estimates of the genetic variance component, although they are likely to explain parts of the large residual variance in annual fitness.

**Age-Related Change in Additive Genetic Variance in Annual Fitness**

One standing evolutionary question is how senescence, a decline in survival and reproductive output at later ages, can evolve. We have here shown age-related changes in phenotypic values of annual fitness in the collared flycatcher. This finding corroborates previous work in this population on senescence in offspring production (Gustafsson and Pärt 1990). Quantitative genetic theories of senescence based on mutation accumulation (MA; Medawar 1952) or antagonistic pleiotropy (AP; Williams 1957) specifically predict that aging is heritable. Additive genetic variances are expected to be age dependent because breeding values change over age (Rose 1991; fig. 1). MA theories predict increasing additive genetic variance and no negative genetic correlation across early and late age classes, whereas AP theory predicts a negative genetic covariance in breeding values at early versus late ages, possibly coupled with a change in additive genetic variance over age classes (Charlesworth 2001). Although these theories have been repeatedly tested under laboratory conditions (e.g., Hughes and Charlesworth 1994; Snoke and Promislow 2003), we know practically nothing about their occurrence under natural conditions. In the only study in the wild, Charmantier et al. (2005) showed that additive genetic variance in laying date (a correlate of reproduction) dramatically increases in old age classes in mute swans Cygnus olor, but they could not reliably assess the genetic covariance between early and late life. Furthermore, laying date in this species was not heritable (except in the oldest age classes), suggesting that most of the additive genetic variance was eroded, except in the oldest age classes.

We have used random regression analysis to analyze senescence. This approach is based on the infinite-dimensional model (Kirkpatrick et al. 1990) and thus models reaction norms of annual fitness as a function of age. This approach can be applied to any trait that varies with increasing age in iteroparous species, and—provided a pedigree is available—the genetic effects can be modeled directly (see also Nussey et al. 2007). One potential limitation for modeling annual fitness in long-lived species may be a low annual recruitment, causing annual fitness to be highly bimodally distributed (around 0 and 1), which is likely to violate the assumption of the animal model of normally distributed residual errors. In the collared flycatcher, recruitment is fairly high, and we indeed find that the residuals of the model fit a normal distribution reasonably well.

Despite a phenotypic decrease in annual fitness in later age classes and overall heritable annual fitness, we find...
that the most parsimonious genetic model states that breeding values for collared flycatcher annual fitness are constant (fig. 1A). In females, we detected an insignificant pattern that was consistent with increasing additive genetic variance as age increased, but in males no pattern emerged. A constant breeding value as age increases implies a complete correlation between performance in early and late life \( (r = 1) \). Hence, we find no clear evidence of an age-related change in additive genetic effects on annual fitness and also no evidence of negative genetic covariance between early and late life, despite considerable sample sizes and a powerful statistical technique.

There are three reasons why an age-related change on the genetic level may be difficult to detect. First, we used a pedigree based on social parentage, but collared flycatchers have about 15% extrapair paternities. In general, such a relatively low pedigree error rate has little influence on the estimation of additive genetic variance (Charmantier and Réale 2005). The probability of obtaining extrapair paternity may depend on a male’s age, which would reduce the power of our approach. However, this issue does not apply to females, because a mother’s relatedness to her offspring is not subject to error. Second, variation in the rate of aging across genotypes (fig. 1B, 1C) is likely to be much more difficult to detect than variation in the average value (fig. 1A) because it requires more information on the age-related performance of relatives. Our data set may have low power because it lacks information of, in particular, repeated records of old individuals. However, we believe that the structure of our data set is typical for passerine birds, with relatively few individuals surviving to breed many times (and at old ages), and we thus expect our findings to be general for an organism with a similar life history. It will be worthwhile to compare our results with a longer-lived organism that undergoes senescent reduction in annual fitness. Third, natural selection may act to reduce the possibility to detect additive genetic variance in older age classes in case only individuals with a high breeding value for annual fitness survive. Nevertheless, most (>90%) of the variation in annual fitness is residual, suggesting that survival to old age is more a chance event than determined by breeding value for annual fitness. Further, our random regression animal model will be robust to selection since records of short-lived individuals will be used for estimation of the infinite-dimensional function describing the change in breeding values over all age classes. The degree of such robustness, however, will crucially depend on the available information of relatives.

Although we cannot formally assess the power of our approach, our results show that if a genetic pattern underlying the phenotypic decline in annual fitness as age increases indeed exists, its effect size is probably small. Our results thus imply that much of the observed phenotypic decline in annual fitness with age is due to environmental effects. One possibility is that such environmental effects of aging are related to the aging of the soma per se. The somatic theory of aging assumes senescence occurs due to a decline in the functioning of the soma and the soma’s repair mechanisms (Kirkwood 1977). Further empirical tests of whether aging is heritable are therefore important, especially under natural conditions, and one major future challenge is to integrate such studies with measuring somatic aspects.

Provided a pedigree of the population is available, animal model methodology allows the study of a trait’s age-specific additive genetic variances and the covariances in the wild (e.g., Charmantier et al. 2005, 2006). An organism’s age-specific schedule of reproduction and survival has evolved under natural conditions, and studying predictions of quantitative genetic senescence theory in the wild, therefore, clearly would be elucidating. We have shown that annual fitness may be an attractive metric in the phenotypic and quantitative genetic exploration of senescence, and it has the potential to provide further insight in the evolution of senescence in the wild.

**Acknowledgments**

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