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ORIGINAL ARTICLE

The rate of ageing in a long-lived bird is not heritable

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A senescent decline in performance occurs in late age in many organisms, and is thought to be partly due to additive genetic effects. Here annual fitness, estimated as the age-specific sum of survival and reproduction, was used to test for genetic variance in ageing in a population of common gulls, *Larus canus*. Data on 3986 individuals collected over a 34-year period indicate a dramatic senescent decline in late life. We also find that annual fitness is heritable and that individuals vary in their rates of ageing. However, counter to theoretical expectations, we find no support for a heritable component to the variance in rates of senescence. Increases in the among-individual (permanent environment) and residual variance components initiate an increase in the total phenotypic variance for annual fitness with age. This finding suggests that older birds are more sensitive to environmental effects, and that old age causes an overall pattern of declining h^2 of annual fitness. Our findings suggest that individual-specific factors do have a role in determining the rate of senescence in this population, but that additive genetic variance for the rate of senescence is either absent or small.
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Introduction

Senescence, which can be defined as a decline in performance in old age, is typical in many mammals and birds (Jones *et al*., 2008). Evolutionary explanations for this phenomenon generally assume that the rate of ageing is heritable (Rose, 1991) due to genotype by age interactions. This means that an individual's additive genetic merit for a performance trait will be age dependent, whereas the population-level additive genetic variance for that trait will consequently change with age. Two mechanisms leading to this expectation have been proposed, both stemming from the assumption that net selection is weaker in old age classes (an expected consequence of proportionally fewer individuals living at later ages; Hamilton, 1966; Baudisch, 2005). First, genotypes with late-acting deleterious effects may be maintained in the population because they perform better at early life (antagonistic pleiotropy, Williams, 1957). Second, if selection is weak in late life, senescence may simply reflect the accumulation of mutations that have deleterious effect in old age (Medawar, 1952). At present, the challenge for quantitative genetic research in natural populations is not so much to distinguish between these two explanations (as has been performed using sophisticated breeding in the laboratory, for example Hughes and Charlesworth, 1994; Snoke and Promislow, 2003), but rather to explore whether there is evidence for a genetic basis of ageing in a variety of organisms in nature (Wilson *et al*., 2008).

In this paper, we apply the concept of the infinite-dimensional reaction norm (Kirkpatrick *et al*., 1990) to characterize an individual's genetic merit for fitness as a continuous function of age. This approach recognizes that complex (co)variance structures across ages can be effectively summarized by using comparatively simple functional forms to describe individual reaction norms. By comparison to estimating genetic parameters for a series of discretely defined age-specific traits, this approach reduces the dimensionality of the (co)variance structures to be estimated. This is a particularly useful technique in long-lived organisms with many age classes and has been implemented in the form of random regression animal model (RRAM) that is commonly used by animal breeders (Meyer, 1998; Schaeffer, 2004), and—in more recent years—by evolutionary biologists (reviewed in Nussey *et al*., 2007; Wilson *et al*., 2008).

Here, we use individual-based data on common gulls *Larus canus* breeding in Estonia to study senescence at both phenotypic and genotypic levels. We define senescence as a decline in fitness with age and test for it using a measure of annual performance that integrates both survival and fecundity. Although many studies of senescence focus only on a single component or correlate of fitness (for example survival), unexplored trade-offs among fitness components may complicate interpretation in these cases (Partridge and Barton, 1996). In contrast, we believe that a decline in annual fitness with increasing age represents an unambiguous indicator of senescence. We therefore test the following

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hypotheses: (1) individual common gulls exhibit senescent declines in fitness; (2) individuals differ in their rates of senescence; (3) heritable differences in the rate of senescence are present and can be detected as genotype by age interactions for annual fitness in this population.

Materials and methods

Common gull study

The data used in the present study were collected in 1968–1983 and 1986–2007 on three offshore islets, Kakrarahu (3.7 ha), Paljarahu (2.5 ha) and Hoorahu (0.5 ha), situated in Matsalu National Park, Estonia (58°46′ N 23°26′ E). Monitoring of common gulls on these islets started in 1962. Each islet forms a geographically distinct breeding colony of common gulls. All nestlings were ringed with a metal ring on their fist day of life. Adults were caught when breeding for the first time, with any unringed adults assumed to be immigrants ringed for lifelong identification with a metal ring at this time. In addition, all adult birds were sexed and individually marked at their first breeding event using plastic rings with clearly visible unique codes. In subsequent breeding years, adults were identified primarily from these plastic rings with observations made from a hide. On average, 94% of nest owners (both males and females) were identified each year.

Common gulls are migratory birds that usually start breeding at the age of 3 or 4 when they have their adult plumage (Rattiste and Lilleleht, 1986). Many birds starting to breed in the colonies were immigrants and could thus not be reliably aged. We therefore used 'breeding age' (previously termed 'experience' by Rattiste, 2004) for all individuals as a proxy for chronological age. Breeding age was defined as the number of years the individual has been part of the breeding population, starting with 1 in the year the individual bred for the first time. Note that a common gull will typically reproduce every year during its breeding career.

The study population has been characterized by a high influx of immigrants in some years, particularly from the mid 1970s onwards when birds breeding outside the focal colonies started to suffer from competitive displacement by other (larger) gull species. Surrounding islands in the western Estonian archipelago were surveyed, although with a lower intensity than the focal colonies and many individuals that were later recorded in our focal colonies were initially recorded breeding elsewhere. However, as breeding success outside the focal colonies was not consistently monitored, we include only those observations made within the focal colonies on individuals of known breeding age in our analyses. As a consequence of immigration, not all individuals have been recorded from breeding age 1 onwards.

Annual fitness and random regression animal model

Following Brommer *et al*. (2007), we define annual fitness w as the number of gene copies that individual i spreads from year t to year $t+1$.

$$w_{i,t} = p_{i,t} + \frac{1}{2} r_{i,t} \quad (1)$$

where $p_{i,t}$ is the survival of the individual (0 or 1) and $r_{i,t}$ 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. Only half the recruits are included in this measure, because of Mendelian inheritance.

Here we consider the survival and reproduction of individuals that were identified as breeding individuals in the focal colonies from 1968 up to and including 2003 for their offspring to be recruited by 2007 and for survival to be estimated as reliably as possible. Out of 4074 individuals recorded during this study period, the breeding age of 88 could not be reliably determined and these were omitted from the analyses. We thus considered 3986 individuals, with a total of 20 480 records of annual fitness in our analyses.

Survival (p) was based on whether the individual was observed again after the breeding season (survived, 1) or not (presumed to have died, 0). Recruitment (r) was based on recapture of offspring of either sex as breeding adults at any time point in the future either within the focal colonies or outside. Annual fitness was only defined for birds that were recorded (observed or caught) when breeding in one of the study colonies in a given year. Errors in apparent survival were probably minimal, because this population is characterized by high adult return rates, and the opportunities for breeding outside the focal colonies have—especially in recent decades—been restricted. Owing to sex-biased natal dispersal, about 80% of the recruits were males. Because annual fitness did not differ between the sexes (see Results), we pooled both sexes in the analyses. We restricted our analysis to breeding ages up to and including 25, omitting three individuals that reached a breeding age of 26 (producing no recruits at that age).

Modelling annual fitness

Annual fitness was modelled using a hierarchical set of linear mixed models that included a constant set of fixed effects but differed in their random effects structure. All models included a fixed effect mean (μ_F) that accounted for a number of factors (see below), and explicitly included breeding age AGE_F fitted as a factor with 25 levels to correct for differences in the mean annual fitness across breeding ages. We considered the hierarchical mixed models

$$w_{i,t} = \mu_F + \mathrm{AGE}_F + f(\mathrm{ind}_{xi}, \mathrm{age}) + \varepsilon_{\mathrm{age},i} \quad (2a)$$

$$= \mu_F + \mathrm{AGE}_F + f(a_{xi}, \mathrm{age}) + f(\mathrm{pe}_{xi}, \mathrm{age}) + \varepsilon_{\mathrm{age},i} \quad (2b)$$

Here, the first model (Equation (2a)) allows among-individual variation around the population average response of annual fitness to age (that is, individual variance in reaction norms), with the individual-specific deviation modelled as a function of age $f(\mathrm{ind}_{xi}, \mathrm{age})$. Having characterized among individual variation, we then partitioned it (Equation (2b)) into an additive genetic component $f(a_{xi}, \mathrm{age})$ and a non-genetic ('permanent environment', Lynch and Walsh, 1998) component $f(\mathrm{pe}_{xi}, \mathrm{age})$. The former was estimated using an animal model, with pedigree data allowing the relatedness matrix among individuals to be specified (see below; Lynch and Walsh, 1998; Kruuk, 2004). The permanent environment effect includes any among-individual sources of variance that are conserved across records

but are not due to additive effects (for example environmental conditions associated with an individual, maternal environment, any non-additive genetic effects).

In all models, we specified the individual-specific random effects (ind_{xi}, a_{xi}, pe_{xi}) as continuous functions of age, comparing model fits with different assumed functional forms (details below). Effects were modelled as polynomial functions of age with order x, where we compared constant ($x=0$), linear ($x=1$) and non-linear ($x>1$) forms of the reaction norm. For example, fitting a zero-order function for a_{xi} (Equation (2b)) results in breeding values, and hence genetic variance, being constrained to be constant with age (no genotype by age interaction). This is equivalent to the 'standard' repeated-measures animal model that ignores potential age effects on variance components. This can then be compared with a first-order polynomial function ($a_{0i}+a_{1i}$ age) in which the breeding value is modelled as a linear reaction norm model across age such that variances in elevation (a_{0i}) and slope (a_{1i}) are estimated, as well as the covariance between these. Age was standardized to a zero median (scale from -12 (breeding age 1) to 12 (breeding age 25)) such that variance in elevation can be interpreted as the among-individual variance at breeding age 13. The functions describing individual-specific (phenotypic) variance (ind_{xi}) and permanent environment variance (pe_{xi}) can be interpreted in an analogous manner.

Random effects, and residual errors, were assumed to be normally distributed with zero means and variances to be estimated. Residual errors ($\varepsilon_{\text{age},i}$) were assumed to be age specific (estimating 25 age-specific error variances) and uncorrelated across ages. Apart from breeding age (as a factor), additional fixed effects included in all the mixed models were sex, year, colony (three colonies) and an individual's breeding status (new partner, partner prior established or unknown). The latter factor was shown to lead to affect the seasonal timing of laying, and as laying early is under recruitment selection (Brommer and Rattiste, 2008), it is expected to have consequences for an individual's annual fitness.

Pedigree information

Equation (2b) can be solved for the genetic (co)variance function by using information on the coefficient of coancestry Θ_{ij} between individuals i and j, which is directly obtained from the pedigree. The additive genetic effects on individual i, a_{xi}, were assumed to be normally distributed with mean of zero and additive genetic variance of σ^2_{xA} (the variance in a_x). This variance (and the additive genetic covariance between all a_x if $x>0$) was estimated from the variance–covariance matrix of additive genetic effects which is equal to $\mathbf{A}\sigma^2$, where $\mathbf{A}$ has elements $A_{ij}=2\Theta_{ij}$.

The common gull pedigree included all recruited offspring recorded up to and including 2007. In total, there were 1234 recruited offspring. Of the 3986 individuals included in the analysis, 43.7% (1743 of 3986) were connected to at least one relative in the pedigree.

Model comparison and inference

Equation (2) allows for a hierarchical step-wise forward approach to test the statistical significance of all the random effects by a likelihood ratio test (LRT). The test statistic is twice the difference in log-likelihood between hierarchical models, and is distributed as χ^2 with the degrees of freedom equal to the difference in the number of (co)variance parameters estimated. First we tested for phenotypic variance in the parameters describing individual fitness functions of breeding age (Equation (2a)). We started out with a model that included only fixed effects and residuals (model 1), then tested for differences among individuals by sequentially fitting individual effects as zero-order (model 2, ind_{0i}), first-order (model 3, $\text{ind}_{0i}+\text{ind}_{1i}\times\text{age}$) and higher-order random regressions. With each increases in order of the polynomial function f(ind_x, age), we, using the LRT test outlined above, tested whether there was a significant improvement in model fit. We started by characterizing the age-specific variance in annual fitness on the individual-specific (phenotypic) level, because absence of significant individual-specific variance of order x directly implies the absence of genetic differences across individuals of order x. We then continued by testing whether partitioning variation in reaction norm elevation ind_0 into additive and permanent environmental effects (a_0 and pe_0) significantly improved the model (while retaining the random regression across individuals; $a_{0i},+\text{pe}_{0i}+f(\text{ind}_{ni},\ \text{age})\,|\,n>0$). This model tests the hypothesis that annual fitness is heritable, under the assumption that additive genetic variance is constant with age, while allowing individual-specific variance to vary with age. We then tested for genotype by age interactions (allowing additive genetic variance to vary with age) by testing for higher-order polynomials of age-specific additive genetic and permanent environment effects (see Brommer *et al*. (2008) for an equivalent modelling approach).

Results

Senescence in annual breeding success

Most (83%, 657 of 790) common gulls ringed as nestlings that were included in our analyses started to breed at age 3 or 4 (females: 3.81 ± 0.064 (s.e.), $n=153$; males: 3.41 ± 0.03 (s.e.), $n=637$). Given this restricted variation around the mean age of first breeding, we assume that our measure of breeding age effectively captures the variation in chronological age and is therefore an appropriate surrogate.

Annual fitness varied from 0 to 2.5 with a phenotypic mean of 0.889 and variance of 0.169. Each individual was recorded up to 21 times, with an average of 5.14 ± 1.75 (s.e.) repeated measures per bird. Mixed model analysis showed that annual fitness depends strongly on breeding age (fixed effects in Table 1), with predicted age-specific means showing an initial slight increase before a rapid decline from around 10 years of breeding age (Figure 1; age-specific sample sizes in caption). To a good approximation, the expected annual fitness of a common gull is relatively stable for the first 10 years of breeding after which it is halved over the next 10–15 years. Thus, as measured by annual fitness, common gull individuals show strong senescence.

It should be noted that recruitment of daughters is lower than that of sons because of sex-biased dispersal. Consequently, a trend in offspring sex ratio with female breeding age could introduce bias into our estimate of

Table 1 Hierarchical mixed models on the age-specific annual fitness in common gulls

Model	*Random effects*							*LogL*	*LRT*	*d.f.*	P-*value*
	ind_0	$ind_1 \times age$	$ind_2 \times age^2$	a_0	$a_1 \times age$	pe_0	$pe_1 \times age$				
1	—	—	—	—	—	—	—	8466.76			
2	0.882 (0.108)	—	—	—	—	—	—	8485.43	37.3	1	<0.001
3	2.02 (0.338)	3.56 (0.828)	—	—	—	—	—	8489.58	8.3	2	0.016
4	0.56 (0.322)	4.11 (1.34)	2.00 (0.996)	—	—	—	—	8498.33*	17.5	3	0.0006
5	**—**	**4.12 (1.36)**	**1.94 (1.00)**	**0.269 (0.11)**	**—**	**0.322 (0.339)**	**—**	**8502.84***	**6.06**	**1**	**0.014**
6	—	—	1.74 (0.988)	0.763 (0.542)	0.822 (1.13)	−0.126 (0.596)	3.35 (1.76)	8503.39*	0.90	2	0.64

Fixed effect	*Coefficient*	*Wald's F*	*d.f.*	P-*value*
Constant	0.827 ± 0.0187	67673.7	1	<0.001
Sex		0.93	1	0.37
Male	$-0.621 \times 10^{-2} \pm 0.644 \times 10^{-2}$			
Colony		18.76	2	<0.001
Status		10.50	2	<0.001
Prior established	$0.307 \times 10^{-1} \pm 0.671 \times 10^{-2}$			
Unknown	0.141 ± 0.391			
Year		21.84	33	0.001
Breeding age		9.43	24	<0.001

In the top half, the REML estimated variances are given for each effect fitted with their s.e. in brackets and effects that were not fitted are indicated by a dash '—'. First, the phenotypic variance across individuals is modelled as polynomial functions of order x ($f(\text{ind}_x, \text{age})$, Equation (2a); models 1–4). Second, the individual-specific variance is further partitioned into its additive genetic and permanent environmental components (Equation (2b)), starting with partitioning the individual-specific elevation ind_0 into its additive genetic and permanent environment components a_0 and pe_0 respectively (model 5). Lastly, model 6 tests whether the breeding values vary linearly with age ($a_1 \times \text{age}$). Including higher-order polynomials for the effects listed here did not produce a better fit (see Results). The most parsimonious model is printed in bold. The number of degrees of freedom between consecutive models differs, because fitting a higher-order polynomial model also includes the covariances between all model terms (see Results). All variances reported are in units of 10^{-2} and are conditional upon the same fixed effect structure as reported in the second half of the table. Asterisk (*) indicates model log likelihood where the estimated permanent environment covariance matrix was not general positive. Constraining models 4 and 5 to positive matrices led to qualitatively the same result (with LogL of 8497.45 and 8501.36 respectively); constraining model 6 did not allow model convergence.

mean senescence. However, the sex ratio (proportion of daughters) of 1083 common gull recruits produced by females included in our analysis showed no systematic pattern of change with maternal breeding age (linear regression coefficients and their s.e.: constant = 0.22 ± 0.038, $t = 5.9$, $P < 0.001$; age: 0.0030 ± 0.0097, $t = 0.31$, $P = 0.76$; age²: -0.00006 ± 0.0005, $t = -0.11$, $P = 0.91$; residuals were normally distributed). Hence, recruitment senescence is not biased by a change in sex ratio over breeding age.

Random regression analyses of senescence

Random regression models (Table 1) provided support for among-individual variance in annual fitness (model 2), and for significant first- (model 3) and second-order (model 4) individual by age interactions. Hence, there is evidence that individuals differed in both the elevations and the curvature of their annual fitness–age relationships. Third-order (cubic) functional forms of ind_{xi} did not allow model convergence when covariances were unconstrained. Constraining the covariance matrix to be general positive produced a lower likelihood (LogL = 8496.91) than the constrained model 4 (see Table 1). We therefore assume here that second-order (quadratic) functions present an adequate description of the data.

Genetics of the rate of senescence

Partitioning the among-individual variance in reaction norm elevation into its additive genetic and permanent environment component (model 5) was strongly statistically supported. Hence, our analyses show that annual fitness is heritable. However, we found no statistical support for the corresponding partition of the among-individual variance in the linear annual fitness–age relationship (model 6), nor did we find support for the combined partitioning of the first- and second-order terms (that is, model $a_0 + \text{pe}_0 + a_1 \times \text{age} + pe_1 \times \text{age} + a_2 \times \text{age}^2 + \text{pe}_2 \times \text{age}^2$; LRT compared to model 5: $\chi^2 = 1.78$, d.f. = 5, $P = 0.88$). We therefore conclude that we find no statistical evidence of a genotype by age interaction. The most parsimonious model (model 5) is one in which breeding value, and hence the additive genetic variance, is constant with age, whereas the permanent environment effect varies as a non-linear function of age. Visual inspection of the residual errors of mixed model 5 showed a reasonable approximation to normality (Shapiro–Wilk test statistic, $W = 0.81$).

Age-specific variance components of annual fitness

The second-order functional form of the among-individual variance indicated that individuals differed in the age of their peak performance. The correlations between individual-specific linear (ind_1) and elevation (pe_0) coefficients, and between linear (ind_1) and quadratic coefficients (ind_2) were positive (0.643 ± 0.234 (s.e.) and 0.207 ± 0.279 (s.e.) respectively). That is, individuals with a high performance at mean age 13 tend to have a higher performance also at later ages. Conversely, the correla-

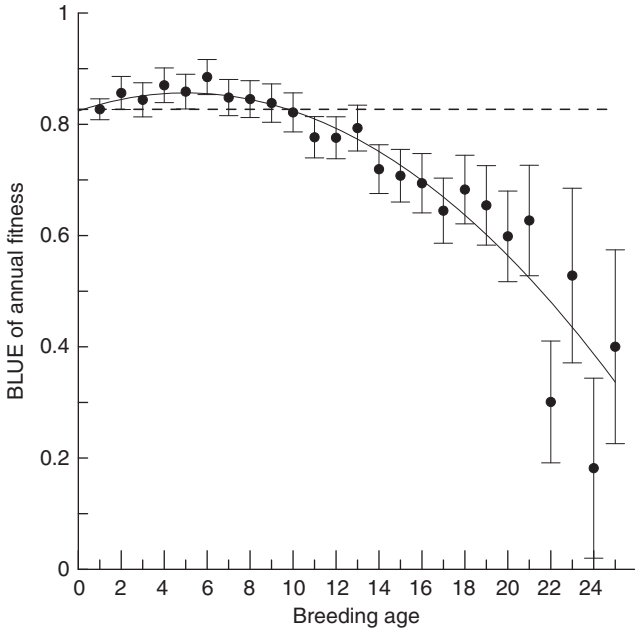

Figure 1 Best linear unbiased estimate (BLUE) of annual fitness with standard error as a function of breeding age. BLUE derived from the mixed model 5 given in Table 1. The first breeding age and the dashed line show the expected annual fitness (least square mean; see Table 1), and BLUEs are relative to this value. Both sexes are combined, as there was no significant difference between them (see Table 1). The solid line is a weighted polynomial regression fitted to the BLUEs: constant: 0.824 ± 0.013, $t = 64.1$, $P < 0.001$; breeding age: 0.013 ± 0.003, $t = 4.0$, $P < 0.001$; (breeding age)^2: $-0.0013 \pm 1.64 \times 10^{-4}$, $t = 8.0$, $P < 0.001$. Sample sizes per breeding age analysed in the model are: 1, 2885; 2, 2317; 3, 2059; 4, 1837; 5, 1715; 6, 1595; 7, 1488; 8, 1259; 9, 1070; 10, 889; 11, 754; 12, 652; 13, 486; 14, 385; 15, 306; 16, 239; 17, 176; 18, 120; 19, 84; 20, 66; 21, 41; 22, 26; 23, 14; 24, 13; 25, 4.

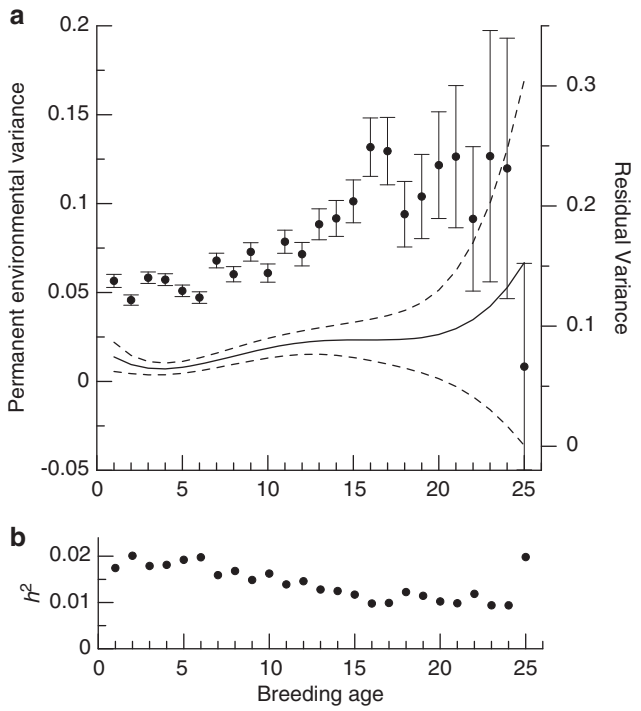

Figure 2 Restricted maximum likelihood (REML) estimated components of variance following mixed model 5 (Table 1). Shown are in (**a**) the increase in variance across individual with age due to non-genetic (permanent environmental) effects (solid line) with approximate confidence interval (dashed line). The approximate confidence interval is double the approximate standard error calculated following Fischer *et al*. (2004). In addition, the residual (error) variance for each age is shown with its standard error. (**b**) Age-specific heritability (h^2) is the ratio of additive genetic variance over the sum of estimated age-specific variances.

tion between elevation and quadratic coefficients was strongly negative (-1.49 ± 1.23 (s.e.)) indicating that high performance at mean age 13 also was associated with a convex shape of the annual fitness–breeding age function. Note that this correlation below -1 resulted from an unconstrained model (such that the covariance matrix of random regression coefficients was not positive definite). Model 5 remained the most parsimonious model if this covariance matrix was constrained to such that the correlation was $\geqslant -1$, but under these conditions the uncertainty around the (co)variance function cannot be estimated. Because the covariance estimates of the unconstrained model 5 produced sensible estimates of age-specific covariances (see below), we assumed that this model provides a satisfactory description of the data.

The random regression coefficients of model 5 were transformed to a variance–covariance matrix for age-specific annual fitness (for example Kirkpatrick *et al*., 1990). The variance across individuals is larger at both the youngest and the oldest age classes (Figure 2a). Because of the strong positive correlation between elevation and the linear coefficient, the permanent environment variance shows only a marginal initial decrease in variance with age, followed by a general increase in variance, which is very pronounced at late ages (Figure 2a). In addition, residual variances, interpretable as the within-individual variance attributable to short-term environmental effects on fitness, increased with age (Figure 2a), although residual variance in the last age class was estimated to be low. This general increase in residual variance was statistically significant, because constraining the residual variance to a single homogenous variance for all ages resulted in a significant decline of model fit (all other random effects as in model 5, $\chi^2 = 139.3$, d.f. $= 24$, $P < 0.001$). Estimated heritability declined with breeding age, from about 2 to about 1% over 25 years (Figure 2b). This decline is driven by increasing permanent environment and residual variances with ages, whereas the additive genetic variance is necessarily constant under the assumptions of model 5. Note that the increase in variance of annual fitness with age was not due to scaling effects as mean annual fitness declined with age (Figure 1).

Discussion

Evolutionary theories of senescence predict that genotypes should differ in their rate of ageing, thereby creating a change in additive genetic variance over age (Rose, 1991; cf. Brommer *et al*., 2007). We have tested this assumption using an RRAM on long-term data from a pedigreed wild population of common gulls. We find that common gulls show a slight initial improvement in annual fitness (a metric combining survival and reproduction) during the first 10 years, but then suffer from a pronounced senescence in annual fitness after being part

of the breeding population for 10 years. We further find strong evidence of additive genetic variance for annual fitness. However, although we do find a clear pattern of increasing variance among individuals with age, there is no statistical support for significant changes in additive genetic variance with age.

In accordance with our findings, a clear pattern of senescence coupled with a lack of evidence of changing additive genetic variance in annual fitness with age was also found in the collared flycatcher *Ficedula albicollis* (Brommer *et al*., 2007). In Soay sheep *Ovis aries*, there was some evidence for an increase in additive genetic variance over age in a population measure of annual fitness (Wilson *et al*., 2007), and in red deer *Cervus elaphus*, age-related changes in additive genetic variance of annual fitness did occur (Wilson *et al*., 2007). Furthermore, rates of ageing in guillemots *Uria aalge* did not differ across individuals (Reed *et al*., 2008), implying an absence of genetic differences in the rate of ageing. In the case of the common gull, we do find evidence of heterogeneity across individuals in their rate of senescence (estimated as the variance in the slopes of the reaction norms). Although not statistically better than model 5, effect sizes under a model that does include a linear genotype by age interaction (model 6; Table 1) suggest that 22.8% (Table 1, $0.855/(0.855+2.90)$) of the heterogeneity of linear slopes is due to genetic differences across individuals. Interestingly, this model predicts that additive genetic variance increases with age, causing an increase in heritability of annual fitness with age (Supplementary Figure 1). However, we stress that this models was not statistically supported and also note that the additive genetic variance (and its change with age) it predicts is clearly smaller than the permanent environment variance (except for the oldest age classes where uncertainty also is largest; Supplementary Figure 1). We therefore find no statistical support for genetic variance in rates of senescence. This lack of statistical significance was not due to a lack of additive genetic variance *per se*, because we did find that annual fitness is heritable. However, we note that our data do have more power to detect phenotypic than genotypic differences in individual-specific slopes of annual fitness across ages. This is because 44% of individuals considered here have a relative in the population, and only these individuals are informative for estimating the additive genetic covariance function. In contrast, all records contribute to estimation of the covariance structures associated with non-additive genetic components of the individual reaction norms. These limitations are inherent in the pedigree structure of any wild population. In conclusion, we find that additive genetic effects on the change in common gull annual fitness with age are relatively small compared to the permanent environmental effects and are not statistically significant.

Declining fitness with age

Senescence is a decline in performance with age, and is best quantified using a trait as closely related to fitness as possible (Partridge and Barton, 1996). This is because fitness components are likely to be facing trade-offs (Stearns, 1989). Senescence in one fitness component does not necessarily indicate senescence in other components, and—arguably—the importance of senescence in a trait would be measured by how much it correlates to fitness. Annual fitness is a metric that integrates the two major life-history components, survival and reproduction. In a long-lived species as the common gull, survival rates are relatively high, and recruitment rates are relatively low. As a consequence, the age-specific pattern of annual fitness mostly captures changes in survival with age. In accordance with the pattern of annual fitness (Figure 1), Rattiste and Lilleleht (1986) analysed common gull survival in a capture–recapture framework and documented a decline in common gull survival after breeding age 6. Recruitment production initially improves with age; recruitment increases threefold during the first 10 years of breeding in the common gulls, after which it starts to decline (Rattiste, 2004). The pattern of age-specific production of recruits may be biased if offspring produced later in life are more dispersive (and thus less likely to be recorded as a recruit) than offspring produced early in life (Ronce *et al*., 1998). We have insufficient data to explore fully the dispersal of offspring as a function of maternal breeding age, but we do note here that the proportion of the more dispersive offspring sex (daughters) remains constant over breeding ages.

One advantage of annual fitness as estimated here is that by summing survival and reproduction, we obtained a trait distribution sufficiently close to normal as to result in model residuals that were reasonably approximated by Gaussian distribution (see also Brommer *et al*. (2007) for similar finding in a passerine bird species). Hence, analysis of annual fitness allows us to use restricted maximum likelihood (REML) random regression analysis. Although it is clearly of great biological interest, we have not explored senescence in fecundity and survival as separate but potentially co-varying processes here. This would require modelling the (co)variance structures within and between two non-normal traits, which is (in principle) possible using generalized linear mixed models (see Wilson *et al*., 2008 for discussion). However, issues relating to both parameter estimation and valid procedures for statistical inferences remain to be resolved (Bolker *et al*., 2009) and the implementation of such models (particularly for the case of estimating genetic variance in an animal model) is not recommended using currently available software (Gilmour *et al*., 2006). For example, ASReml (used here) uses penalized quasi likelihood to approximate the likelihood function for non-Gaussian models but this method is expected to be unreliable in our data structure (Bolker *et al*., 2009). Conversely, available implementations of more robust likelihood approximation methods are not presently able to fit RRAMs as used here. However, we expect that appropriate tools, including Bayesian approaches currently in development (for example Ovaskainen *et al*., 2008), will facilitate quantitative genetic analyses of age-specific patterns of survival and reproduction and their genetic components of (co)variance in the near future.

We find that the individual-specific deviations from the mean annual fitness are a non-linear function of age. Note that these non-linear functions describe individual reaction norms of breeding age relative to the population mean (which in itself is also non-linear; Figure 1). To the best of our knowledge, this is the first demonstration of a statistically significant second-order functional form of

ageing on the individual level in a wild population as previous studies have documented linear effects only (reviewed in Wilson *et al*., 2008). This finding highlights the possible complexity behind describing variation in reaction norms of ageing in the wild. Because a considerable number of repeated records are needed to model higher-order functional forms effectively, such complexity may remain undetected in species with a short life history (or in long-lived species that are studied for insufficient time). In addition, partitioning higher-order age-specific among-individual variation into additive genetic and permanent environment components requires estimating several genetic (co)variances, possibly exacerbating power limitations.

Closer inspection of the age-specific variance indicates that the differences across individual performance are particularly enhanced at older ages. In statistical terms, increasing among-individual variance in annual fitness is likely caused by one or more factors that create positive covariances across ages. Accepting that the rate of ageing is not itself heritable, then individuals presumably differ in one or more environmental factors (for example quality of their breeding site) that influence senescence rates because the consequences of such qualitative differences become more pronounced in older age classes. Another contributing factor may be that consequences of life-history decisions made in early life carry-over to consecutive years, increasingly compounding variance across individuals as they age (for example Atchley and Zhu, 1997; Houle, 1998). In addition to the within-individual processes leading to an increase in variation in annual fitness with age, we also found an increase in residual variance with age. Residual variation can be considered as 'general' environmental variance, not associated with individual-specific factors, suggesting that older individuals become increasingly sensitive to environmental conditions. For example, winter conditions are the main determinant for survival probability of a common gull (Rattiste and Lilleleht, 1995), and older individuals may find it increasingly difficult to cope with such conditions.

In conclusion, we find a clear senescent decline in annual fitness in common gulls. Annual fitness is itself heritable in this population, but—contrary to theoretical predictions—we find no evidence for additive genetic variance in the rate of senescence. Although more studies on wild organisms are needed, the lack of clear evidence for a genotype by age interaction on annual fitness in three out of the four populations tested thus far (including this study) suggests that additive genetic sources of variation in ageing may be small. Certainly common gulls do exhibit pronounced differences in individual-specific rates of fitness decline, but the reasons for this may be mostly ecological, rather than genetic.

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