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Evolvability of an avian life history trait declines with father's age

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Abstract

Studies of laboratory organisms have suggested that parental age affects the genetic variance of offspring traits. This effect can engender age-specific variance in genetic contributions to evolutionary change in heritable traits under directional selection, particularly in age-structured populations. Using long-term population data of the blue-footed booby (*Sula nebouxii*), we tested whether genetic variance of recruiting age varies with parental age. Using robust quantitative genetic models fitted to pedigree, we found a significant genotype-by-paternal age interaction for recruiting age. Genetic potential for adaptive change in recruiting age was greater in progeny of young (age 1–6 years) fathers (males: $CV_A = 6.68$; females: $CV_A = 7.59$) than those of middle age (7–9 years) fathers (males: $CV_A = 4.64$; females: $CV_A = 5.08$) and old (10–14 years) fathers ($CV_A = 0$ for both sexes). Therefore, parental age dependence of heritable variance, in addition to age-related variation in survival and fecundity, should affect the strength of natural selection for evolutionary changes. Our results provide rare evidence for the influence of parental age on the evolutionary potential of a life history trait in a wild population.

Introduction

In recent decades, a great effort has been made to understand the processes involved in the adaptation of life history traits to stable and fluctuating environments (Roff, 2002). A key aspect of life history evolution is the variation in life history traits observed within a population because of genetic and environmental influences. Heritable (genetic) variation determines the response rate of a trait to the directional selection that generates evolutionary change (Falconer & Mackay, 1996; Lynch & Walsh, 1998). As most life history traits affect individual fitness (Stearns & Hoekstra, 2000), the maintenance of their genetic variation in the face of selection is a central paradox of evolutionary biology (Roff, 1997). In this context, studies of genetic and environmental influence on phenotypic variation in life history traits can provide potentially important insights into the evolutionary dynamics of wild populations and the maintenance of viable populations in changing environments (Lande & Shannon, 1996; Storfer, 1996; Coltman *et al.*, 2003).

An important and neglected aspect of phenotypic variation in life history traits is the inconstant nature of genetic and environmental variation, for example, because of differences in genetic quality and environmental conditions among individuals of different ages and cohorts. Recent quantitative genetic studies of wild animals have shown that additive genetic variance in life history traits such as timing of breeding and annual fitness can depend on the age when the trait is measured (Charmantier *et al.*, 2006a; Brommer *et al.*, 2007; Wilson *et al.*, 2007). These studies focused on changes in genetic variation over the lifetime of individuals using repeated measures of age-specific life history traits. However, similar evidence for effects of the age of parents when they give birth on the genetic variation of their progeny is scarce.

Effects of parental age on offspring life history traits, beyond any genetic mechanism, have been studied in diverse animal taxa. For instance, older parents often have shorter-lived offspring (i.e. 'Lansing effect'; Priest *et al.*, 2002 and references therein). As far as we know, only two studies examined the effects of parental age on

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offspring genetic variation in laboratory animals; heritability (h^2) of morphological traits was higher in progeny of older parents than in progeny of younger parents in both *Drosophila melanogaster* (Beardmore *et al.*, 1975) and the guppy *Poecilia reticulata* (Beardmore & Shami, 1976). Although it has been argued that these changes in heritability could arise from age-related decline in the number of progeny produced (Caligari & Baban, 1981), these early findings suggest that the effect of parental age on genetic variation may reflect an ageing process based on mutation accumulation or antagonistic pleiotropy (Beardmore & Shami, 1985). As the influence of parental age on genetic variation has only been examined in laboratory organisms, its generality and overall importance are uncertain (e.g. Moore & Harris, 2003; Partridge & Gems, 2007).

In wild populations, phenotypic variation among offspring has been assumed not to change with parental age (Williams, 1966; Charlesworth, 1994; Kirkwood & Austad, 2000), but the validity of this assumption remains to be explored. In age-structured populations, this issue is important because changes in genetic variation with parental age can modify the force of natural selection over the lifetime, with different age classes contributing differently to the gene pool (Hamilton, 1966; Baudisch, 2005). Negative effects of ageing on reproductive performance have been documented in wild populations only recently (see Jones *et al.*, 2008; Nussey *et al.*, 2008) and imply that older individuals make a minor contribution to the next generation. Nevertheless, if genetic variation among individuals varies with the age of their parents, the age structure of the population and age-specific fitness will have complex repercussions on the genetic response of the population to selection.

We test whether evolutionary potential is dependent on parental age by estimating genetic variance of a life history trait, recruiting age (age at first reproduction), in relation to paternal and maternal ages, using data from a long-term population study of the blue-footed booby (*Sula nebouxii* Milne-Edwards). Studies of some long-lived wild populations have shown that recruiting age is heritable (Kruuk *et al.*, 2000; Charmantier *et al.*, 2006b) and that selection acts on this trait through lifetime fitness (Brommer *et al.*, 2002; Oli *et al.*, 2002; Charmantier *et al.*, 2006b). We use a robust statistical procedure (the 'animal model') to simultaneously exploit similarities between relatives of varying degrees known from their observed pedigree (Lynch & Walsh, 1998; Kruuk, 2004; Kruuk & Hadfield, 2007; Wilson *et al.*, 2010). We estimate breeding value for recruiting age of blue-footed boobies as a function of parental age by applying random regression animal models (RRAMs). RRAM allows us to specifically test for additive genetic variance in recruiting age and to estimate parental age-specific additive genetic (co)variances without the serious loss of power associated with subdividing related individuals into parental age classes. We assess whether breeding value of recruiting age differs among progeny of parents of different age classes and predict how, under directional selection, such parental age dependence will influence evolutionary changes in males and females.

Materials and methods

Study system and field procedures

Reproduction of the blue-footed booby on Isla Isabel, Nayarít (21°52′N, 105°54′W), off the Pacific coast of Mexico, has been monitored since 1981. The majority of males and females recruit into their natal breeding population at age 2–6 years, females doing so on average nearly half a year earlier than males (Osorio-Beristain & Drummond, 1993; Drummond *et al.*, 2003). Roughly 37% of fledglings recruited (were resighted at least once as a breeder) in the period 1988–2004, including males and females in nearly equal proportions (51% and 49%, respectively; Oro *et al.*, 2010). Both sexes then show fidelity to their first breeding site over at least the first 8 years of life (Kim *et al.*, 2007). Annual adult mortality is roughly 9% (Oro *et al.*, 2010), and some males and females in the study population have been observed breeding at age 19 years (our unpublished data). After reaching their reproductive peak at age 8–10 years, both sexes in this population experience reproductive senescence (Velando *et al.*, 2006; Beamonte-Barrientos *et al.*, 2010).

Reproduction was recorded in two study areas measuring 20 800 and 6089 m^2 and lying roughly 400 m apart, from 1982 and 1989, respectively. Every year, all nests with a clutch or brood were marked with wooden stakes and monitored every 3–6 days between February and July. Breeders and fledglings were individually marked with PVC rings from 1982 to 1987 and with steel rings from 1988 onwards. Most breeders' ring numbers were confirmed by up to three independent readings.

Sampling and pedigree information

We sampled recruits from the 15 cohorts of fledglings (1988–2002) that bred for the first time at ages 1–6 years, using the long-term population data collected every year up to 2008. Therefore, there was no sampling bias in recruiting age among different cohorts. Among male and female recruits from the 15 cohorts, only 7.6% were observed to breed for the first time at ages 7–16 years (see also Drummond *et al.*, 2003 for distribution of male and female recruiting ages). We used first breeding records of all recruits for Model 1 (n = 3150) and first breeding records of recruits with one or two marked parents of known age for Models 2–4 (with known paternal age: n = 942; with known maternal age: n = 956). Parental age class (young: 1–6 years; middle: 7–8 years; old: 9–14 years) rather than annual parental

age was used in Models 2–4 to avoid bias in estimates of genetic variance because of autocorrelation between parental age and parental recruiting age in individuals whose parental age at birth is very young and to ensure an adequate sample in each age class. Indeed, convergence problems were encountered when attempting to fit annual parental age in the random regression models. Each parent produces a brood of only 0–3 recruits (often only 0 or 1 recruit) per year, and the majority of relatedness is based on full- and half-siblings in our data set. Therefore, it was inappropriate to estimate genotype-by-parental age interaction by using annual parental age for our data set. Effects of paternal and maternal age may not be independent when animals mate assortatively by age, but in the study population, the ages of mated breeders were not correlated ($r = 0.078$, $P = 0.072$, 537 pairs in 2007; our unpublished data).

Estimation of the additive genetic effect requires pedigree (family tree) information for analysed individuals. The pedigree for our quantitative genetic analyses is based on 4642 individuals, including 1221 paternal identities and 1187 maternal identities. In Model 1, 347 birds whose two parental identities are unknown are included to better estimate total phenotypic variance, but they do not function in the estimation of genetic variance. The pedigree used for the present study possibly includes some paternal errors because of extra-pair fertilization. Although extra-pair fertilization occurs rarely in the study population (B. C. Faircloth, A. Ramos, H. Drummond and P. Gowaty, unpublished data), its possible influence on paternal age-specific patterns in genetic variation should be considered to draw a careful conclusion.

Quantitative genetic analysis

The animal model estimates the genetic effect (heritability) of a trait by assessing the phenotypic covariance between all pairs of relatives in the pedigree (see Lynch & Walsh, 1998; Kruuk, 2004; Kruuk & Hadfield, 2007; Wilson *et al.*, 2010 for a detailed description); it is a form of mixed model, with fixed and random effects. In our study, animal models with a restricted maximum likelihood (REML) were fitted to the pedigree of recruits to estimate variance components and total phenotypic variance for recruiting age using ASReml v2 (VSN International; Gilmour *et al.*, 2006). Before analysis, recruiting age was log transformed to make the distribution of the data and model error terms more normal.

In Model 1, we partitioned variance in recruiting age into additive genetic and environmental components across all individuals whose parental identities and ages are either known or unknown; recruiting age of an individual i is specified as:

$$r_i = \mu + \text{sex} + a_i + c + e_i \qquad \text{(Model 1)}$$

In Model 1, the population mean (μ) and sex (as a factor) were included as fixed effects to account for the study population's sexual difference in recruiting age. Cohort (c) was included as an additional random effect to avoid upward bias in additive genetic variance by accounting for the annual differences in environmental conditions. As with most linear models, the distribution of all random effects, additive genetic and cohort effects (a_i and c) and the residual term (e_i) is assumed to have a mean of zero and a variance to be estimated (V_A, V_C and V_R). For Model 1, total phenotypic variance (V_P) was partitioned as: $V_P = V_A + V_C + V_R$, then heritability and common environmental effect (cohort random effect) were calculated as $h^2 = V_A/V_P$ and $c^2 = V_C/V_P$. The statistical significance of each random effect was assessed using likelihood ratio tests that compare models based on −2 times the difference in REML log-likelihood scores distributed as chi-square, where the degrees of freedom equalled the number of variance terms removed (Littell *et al.*, 2006). The significance of each variance component function (h^2 and c^2) was assessed using a one-tailed t-test.

In order to model an individual's breeding value as a function of parental (paternal or maternal) age at birth (i.e. genotype-by-parental age interactions, G × A), we fitted RRAMs (Model 3 and 4) to individuals whose paternal or maternal age is known (i.e. 30% of available recruits for both paternal age- and maternal age-specific analyses). For the initial model (Model 2), we considered breeding values of recruiting age as well as other random effects to be constant across paternal or maternal age classes. The model including paternal or maternal age as an additional fixed effect (a factor) is specified as:

$$r_i = \mu + \text{sex} + \text{P(M)age} + a_i + c + e_i \qquad \text{(Model 2)}$$

Although paternal age (as a factor) was not significant in Model 2 ($P = 0.150$), it was retained as an additional fixed effect in subsequent models because additive genetic variances were estimated as a function of standardized paternal age. Then, we modelled the functions describing the change in the additive effect as first- and second-order Legendre polynomial functions of standardized paternal or maternal age. Parental age class was standardized to a scale from −1 to 1. Individual breeding values were modelled as linear functions of parental age, such that $a_i = a_{i0} + b_i.\text{stP(M)age}$, where a_{i0} is the additive genetic variance of individual i at stP(M)age = 0, and b_i is the slope of the genetic reaction norm. Therefore, recruiting age of individual i was specified as:

$$r_i = \mu + \text{sex} + \text{P(M)age} + (a_{i0} + b_i.\text{stP(M)age}) + c + e_i \qquad \text{(Model 3)}$$

Although V_A can change with parental age under Model 3, the residual variance (e_i) is constrained to be constant. Assuming such homogeneity might not always be appropriate, so we relaxed the assumption of homogenous residual variance by fitting error structures as diagonal matrices with dimension equal to the number of parental age classes (Wilson *et al.*, 2007) as:

$$r_i = \mu + \text{sex} + \text{P(M)age} + (a_{i0} + b_i.\text{stP(M)age}) + c + e_i.\text{stP(M)age} \quad \text{(Model 4)}$$

Significance of G × A (first-order Legendre) under Models 3 and 4 was assessed by likelihood ratio tests to a reduced model in which the additive effect is a zero-order function of age (i.e. constant; Model 2). Similarly, significance of additive genetic variance as a second-order function of parental age was assessed by likelihood ratio comparison of first- and second-order models. Variance components for recruits from each parental age class were calculated using estimates of intercept and slope obtained from Model 4. We also provide the coefficient of parental age-specific additive genetic variance CV_A (i.e. evolvability; Houle, 1992), in which the additive genetic variance is scaled by the trait mean $(\overline{X})$: $CV_A = 100 \times \sqrt{V_A}/\overline{X}$, to enable comparison of evolvability among parental age classes.

Results

Variance components of recruiting age

In Model 1 fitted to $\log_{10}$-transformed recruiting age, which takes into account the significant sexual difference in recruiting age ($P < 0.001$; mean recruiting age ± SD: males: 4.212 ± 0.954, $n = 1609$; females: 3.652 ± 1.051, $n = 1541$), additive genetic and common environmental effects were statistically significant (Table 1). In Model 1, 6.4% and 11.0% of the total phenotypic variance in recruiting age was explained by additive genetic and common environmental effects, respectively. The same model was fitted to nontransformed recruiting age to estimate natural variances and standard errors for this trait in the study population: $V_A = 0.066 \pm 0.030$; $V_C = 0.111 \pm 0.048$; $V_P = 0.996 \pm 0.053$.

Breeding value as a function of paternal age

There was significant variation between males and females in $\log_{10}$-transformed recruiting age in Model 2, fitted to recruits whose paternal ages at birth are known ($P < 0.001$; Fig. 1a), and in Model 1; hence, sex was retained as a fixed effect in the subsequent models.

Table 1 Quantitative genetics (Model 1) on $\log_{10}$-transformed recruiting age in all recruits with known and unknown parental age at birth ($n = 3150$). A significant fixed effect (sex of recruits: $P < 0.001$) was included in the animal model.

	Estimate ($\times 10^{-2}$)	Test	*P*
$V_A \pm SE$	0.090 ± 0.042	$\chi^2_1 = 5.38$	0.020
$V_C \pm SE$	0.155 ± 0.067	$\chi^2_1 = 362.4$	< 0.001
$V_P \pm SE$	1.414 ± 0.074		
$h^2 \pm SE$	0.064 ± 0.030	$t = 2.133$	0.017
$c^2 \pm SE$	0.110 ± 0.042	$t = 2.619$	0.004

Additive genetic and common environmental effects of Model 2 were greater than those from Model 1, but the former was nonsignificant in Model 2 (Table 2).

In the RRAM in which residual variance was assumed to be constant (Model 3), fitting the additive genetic effect as a first-order Legendre polynomial function of paternal age significantly improved on fitting it as a constant (i.e. zero-order function). Therefore, the linear G × A interaction was significant (Table 2). Similarly, there was a highly significant linear G × A interaction in the RRAM in which residual variance varied with paternal age (Model 4; Table 2). Under Model 4, both additive genetic variance and residual variance decreased with paternal age (Fig. 1b,c). Neither Model 3 nor Model 4 was significantly improved by the use of second-order functions (Table 2). Therefore, the coefficient of paternal age-specific additive genetic variance (evolvability) for recruiting age decreased with paternal age class in both male and female recruits (Fig. 1d), suggesting greater evolutionary potential for recruiting sons and daughters of younger fathers.

Additionally, we estimated additive genetic variance and heritability in three paternal age-specific data subsets from Model 1 to show that the paternal age-specific pattern (Fig. 1b) was not simply because of the negative linear function of paternal age for genetic variance ($b_i < 0$) in Model 4. Although nonsignificant, Model 1 fitted to recruits from each paternal age class suggested that indeed additive genetic effect for recruiting age decreased with paternal age ($h^2 \pm SE$: young: 0.108 ± 0.110; middle: 0.050 ± 0.171; old: 0).

Breeding value as a function of maternal age

Similar to the models fitted to recruits with known paternal age, the significant sex effect and nonsignificant effect of maternal age class (sex: $P < 0.001$; maternal age: $P = 0.084$ in Model 2) were included as fixed effects in Model 2 fitted to recruits whose maternal ages at birth were known. However, in Model 2, the estimate of additive genetic variance was 0, whereas common environmental variance was comparable to that of Model 1 ($V_C = 0.146 \pm 0.084$ ($\times 10^{-2}$), $\chi^2_1 = 54.206$, $P < 0.001$). Therefore, it was difficult to make a meaningful interpretation regarding additive genetic effects as a function of maternal age despite the significant linear G × A interaction (first-order Legendre in Model 3: $\chi^2_2 = 6.904$, $P = 0.032$; in Model 4: $\chi^2_2 = 17.314$, $P < 0.001$) with a negative linear function of maternal age for genetic variance.

Discussion

We estimated heritable variation in $\log_{10}$-transformed recruiting age of the blue-footed booby by partitioning phenotypic variance into genetic and environmental components in a large data set, and then examined

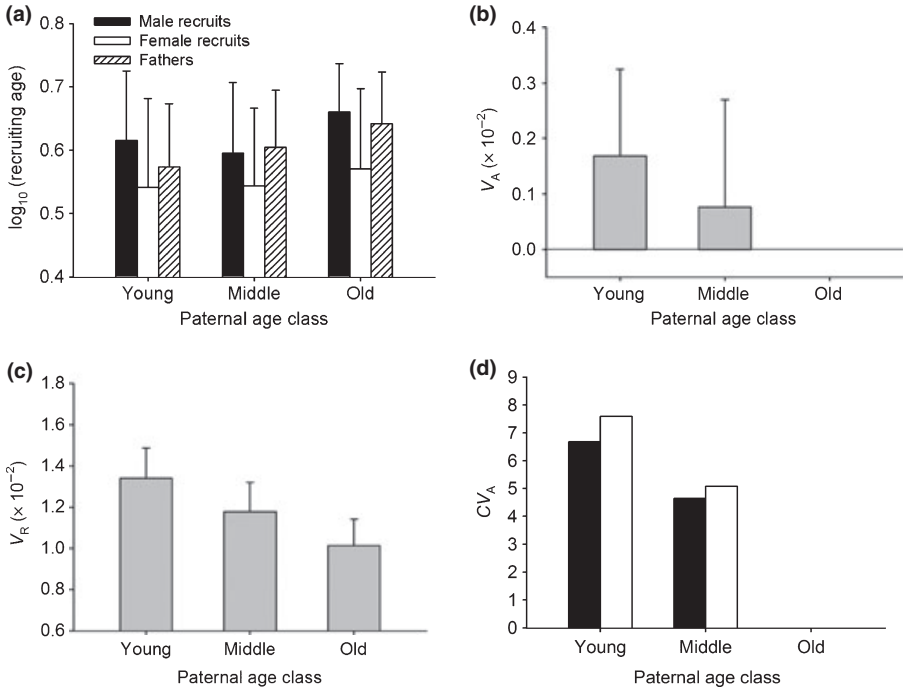

Fig. 1 Quantitative genetic parameters for $\log_{10}$-transformed recruiting age of blue-footed boobies across three paternal age classes at birth (young: 1–6 years; middle: 7–9 years; old: 10–14 years). (a) Trait means + SD for recruiting age of male and female recruits and their fathers. (b) and (c) Additive genetic variance + SE and residual variance + SE as a function of standardized paternal age at birth estimated from a random regression animal model (Model 4, first-order Legendre), respectively. (d) Coefficients of additive genetic variance for male and female recruits, calculated using the sex-specific trait mean (a) and additive genetic variance from Model 4 with a first-order Legendre polynomial function of paternal age (b).

additive genetic variation as a function of parental age at birth in a series of animal models. We found that additive genetic and cohort effects explained 6.4% and 11.0% of the variance in recruiting age, respectively. Although heritability of life history traits tends to be low compared to other traits (Price & Schluter, 1991), our results suggest that recruiting age is heritable in the study population and, more interestingly, its additive genetic variation depends on the father's age at the time of reproduction. The power of a quantitative genetic analysis depends crucially on sample size and pedigree structure (Wilson *et al.*, 2010). Indeed, additive genetic variance was not significant, although higher (almost double) than in the larger data set, in samples with known paternal age ($h^2 = 0.124$) and it was none in samples with known maternal age ($h^2 = 0$).

We found significant evidence for a genotype-by-paternal age interaction, suggesting stronger genetic influence of younger fathers on this heritable life history trait. Boobies are long-lived, so both age-specific fitness (Velando *et al.*, 2006) and age-specific genetic influence on progeny should affect the adaptive response of the population to directional selection. Our population data allowed us to calculate fitness of two early cohorts, the 1989 and 1991 cohorts (no fledglings were ringed in 1990, and in 1992, few chicks fledged because of a severe El Niño event), using number of fledglings produced during the lifetime up to age 17 years. Selection differentials for recruiting age, calculated using lifetime fitness, were negative in the two cohorts (male $S = -0.062$; female $S = -0.138$; S.-Y. Kim, A. Velando, R. Torres &

Table 2 Standard (Model 2) and random regression animal models (RRAMs) (Model 3 and 4) of $\log_{10}$-transformed recruiting age for recruits with known paternal age ($n = 942$).

Term	Estimate ($\times 10^{-2}$)	Test	*P*
Standard animal model (Model 2)			
$V_A \pm$ SE	0.197 ± 0.194	$\chi^2_1 = 1.220$	0.269
$V_C \pm$ SE	0.182 ± 0.098	$\chi^2_1 = 78.734$	< 0.001
$V_P \pm$ SE	1.584 ± 0.153		
$h^2 \pm$ SE	0.124 ± 0.115	$t = 1.084$	0.139
$c^2 \pm$ SE	0.115 ± 0.056	$t = 2.065$	0.020
RRAM with constant residual variance (Model 3)			
First-order Legendre		$\chi^2_2 = 7.010$	0.030
Second-order Legendre		$\chi^2_2 = 0.074$	0.964
RRAM with inconstant residual variance (Model 4)			
First-order Legendre		$\chi^2_2 = 11.898$	0.003
Second-order Legendre		$\chi^2_2 = 0.038$	0.981

H. Drummond, unpublished data), suggesting that directional selection favours early recruitment. Our results highlight a potentially important issue in evolutionary quantitative genetics and provide rare evidence for the influence of parental age on the evolutionary potential of a life history trait in a wild population. In the blue-footed booby, offspring of younger fathers had greater genetic potential for recruiting age (as shown by the coefficient of paternal age-specific additive genetic variance). Therefore, the potential for evolutionary changes in response to natural selection in this life history trait should decrease as reproductive males age.

The 'animal model' enables novel advances in evolutionary analysis, but there are some methodological issues that need careful consideration, particularly regarding specification of models, statistical power, interpretation of results and biased estimation of additive genetic variance (Charmantier *et al.*, 2006a; Kruuk *et al.*, 2008; Wilson, 2008). Recruits born to earlier paternal age classes comprised more cohorts (young: 11 cohorts; intermediate: 9 cohorts; old: 5 cohorts), and in early cohorts, estimates of additive genetic variance could be based mostly on full-sibling comparisons, because of lack of pedigree information from older generations. Because siblings from the same brood share more similar environments than other relatives, genetic variance could be partly overestimated in some individuals from earlier cohorts because genetic and environmental effects were not fully teased apart. However, our exploratory analysis showed that the common environment of broodmates explained little variance in recruiting age when nest identity was included as an additional random effect in Model 1 ($V_{nest} = 0.028 \pm 0.060$, $P = 0.624$) and no variance in Model 2 ($V_{nest} = 0$). Therefore, paternal age dependence of additive genetic variance is unlikely to be as a result of the interference of common environmental effects in early cohorts included in the young paternal age class.

Decreased genetic variance in progeny of old fathers could be as a result of increased extra-pair fertilization of partners of old males, if this occurs. Loss of paternity is often observed in young males of other species (e.g. Weatherhead & Boag, 1995; Wagner *et al.*, 1996; Richardson & Burke, 1999), but if females copulate with extra partners to obtain better quality genes for their offspring (Andersson, 1994), then females paired with old males should show increased extra-pair fertilization whenever old males carry unconditionally deleterious viability mutations and less adapted genes (Hansen & Price, 1995; Kokko & Lindström, 1996; Brooks & Kemp, 2001). Females in the study colony commonly copulate with extra partners (Osorio-Beristain & Drummond, 1998), but it is not known how female infidelity relates to age of the social partner. Extra-pair fertilizations and quasiparasitical egg dumping both occur in the colony at very low frequencies (Osorio-Beristain *et al.*, 2006; B. C. Faircloth, A. Ramos, H. Drummond & P. Gowaty, unpublished data), and evidence for extra-pair fertilization in congeners is lacking (Anderson & Boag, 2006; Baião & Parker, 2009). Thus, extra-pair paternity probably has a negligible influence on our estimates of genetic variance.

Parental age-specific environmental effects can also contribute to total phenotypic variance (Priest *et al.*, 2002). Offspring born to old fathers may show low genetic variance because they are raised under stressful conditions by senescent parents. The developmental environment can limit genetic potential (Gebhardt-Henrich & Van Noordwijk, 1991; Hoffmann & Merilä, 1999), and decreased genetic effect can be expected in individuals that experience more stressful conditions through depressed expression of additive genetic variance (Charmantier & Garant, 2005). However, this is unlikely to be important in our study because residual variance, which comprises mostly unexplained common environmental effects, also decreased with paternal age class when we relaxed the assumption of constant residual variance. Moreover, male recruits born to older fathers disperse less far from their natal nests, suggesting that they were more competitive than those born to young fathers at recruitment in the study population (Drummond *et al.*, 2010). This contrasts the assumption that offspring from old fathers suffer stressful environmental conditions during the development.

Decrease in genetic variance with the father's age may be as a result of age-related changes in fathers' genetic material and a general reduction in gene expression. Senescent parents, particularly old fathers, are likely to carry unconditionally deleterious viability mutations because of the accumulation of spontaneous germline mutations over the lifetime (Rose, 1991; Charlesworth, 2001; Velando *et al.*, 2008), affecting the resemblance in life history traits with their offspring. Our findings contrast the assumption of standard quantitative genetic models that an individual's breeding value is constant and possibly provide insight into the evolution of senescence. Whereas the mutation accumulation theory of ageing predicts a family-specific decline in breeding values and an increase in the additive genetic variance with age, the antagonistic pleiotropy theory predicts a negative genetic covariance in the breeding values for early and late life. Our results partly agree with the latter theory of ageing by showing that additive genetic variance changes over paternal age classes.

The evolutionary theory of senescence predicted a decline in the strength of natural selection with age while assuming that genetic variance is constant across ages (Hamilton, 1966). In the blue-footed booby, older males contribute less to the gene pool than other age classes because of reduced reproductive success (Velando *et al.*, 2006), and our results indicate that their contribution is further weakened by reduced genetic potential. Overall, these patterns suggest a decline in evolutionary potential under directional natural selection in the progeny of ageing parents.

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