LETTER

Silver-spoon upbringing improves early-life fitness but promotes reproductive ageing in a wild bird

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Abstract

Early-life conditions can have long-lasting effects and organisms that experience a poor start in life are often expected to age at a faster rate. Alternatively, individuals raised in high-quality environments can overinvest in early-reproduction resulting in rapid ageing. Here we use a long-term experimental manipulation of early-life conditions in a natural population of collared flycatchers (*Ficedula albicollis*), to show that females raised in a low-competition environment (artificially reduced broods) have higher early-life reproduction but lower late-life reproduction than females raised in high-competition environment (artificially increased broods). Reproductive success of high-competition females peaked in late-life, when low-competition females were already in steep reproductive decline and suffered from a higher mortality rate. Our results demonstrate that 'silver-spoon' natal conditions increase female early-life performance at the cost of faster reproductive ageing and increased late-life mortality. These findings demonstrate experimentally that natal environment shapes individual variation in reproductive and actuarial ageing in nature.

Keywords

Ageing, brood size manipulation, condition dependence, disposable soma theory, early-life conditions, senescence, 'silver-spoon' theory.

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INTRODUCTION

Ageing, a physiological deterioration with advancing age, is a complex and highly variable trait that is widespread across the tree of life (Jones *et al.* 2014; Shefferson *et al.* 2017) and affects organismal fitness in nature (Bouwhuis *et al.* 2012; Nussey *et al.* 2013). Theory maintains that ageing evolves because selection gradients on traits decline with age (Medawar 1952; Williams 1957; Hamilton 1966), favouring alleles that increase early-life fitness even at the cost of negative pleiotropic fitness effects in late-life. While a large body of evidence supports these theoretical predictions on the population level, very little is known about the individual variation in ageing rates. There is a general agreement in the field that early-life environmental conditions can shape the life-history of an adult organism and explain much of the individual variation in ageing rates (Monaghan 2008; Marshall *et al.* 2017; Cooper & Kruuk 2018), but there is little consensus regarding the direction of the effect. Good environmental conditions during development can result in lifelong positive effects on physiology, reproduction and longevity (Lindström 1999; van de Pol *et al.* 2006a; Nussey *et al.* 2007; Hayward *et al.* 2013; Cooper & Kruuk 2018) ('silver-spoon' hypothesis – Grafen 1990). Alternatively, organisms raised in good natal environments can invest heavily in growth and early-life reproduction, resulting in accelerated senescence in late-life (Hunt *et al.*

2004; Bonduriansky & Brassil 2005; Hooper *et al.* 2017; Marshall *et al.* 2017) ('live-fast die-young' hypothesis – Promislow & Harvey 1990). These studies suggest that organisms can allocate resources to reproduction and somatic maintenance depending on their early-life environmental conditions, and that high latent costs of increased early-life growth and reproduction, under favourable conditions, can result in accelerated ageing (Adler *et al.* 2016; Hooper *et al.* 2017).

Previous studies emphasised the role of sexual selection in the evolution of resource allocation strategies that can result in rapid ageing under favourable natal conditions. Males, for example are predicted to invest in costly secondary sexual traits and risky behaviours, trading-off with investment in somatic maintenance and late-life performance (Hunt *et al.* 2004; Bonduriansky & Brassil 2005; Nussey *et al.* 2007; Hooper *et al.* 2017). However, recent studies also show that early-life investment in reproduction trades-off with late-life fitness in females in natural populations (reviewed in Lemaître *et al.* 2015) and in the laboratory (Travers *et al.* 2015). This suggests that 'silver-spoon' females could allocate more resources to early-life reproduction and suffer from faster senescence later in life, whereas females experiencing early-life stress could reduce their investment in early-life reproduction and delay senescence. Interestingly, mild stress results in reduced early but improved late breeding performance in captive female Zebra Finches (*Taeniopygia guttata*) (Marasco *et al.*

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2018). Overall, it is possible that two related processes shape the expression of age-specific life histories in response to differences in early-life conditions. First, poor conditions in early-life constrain the amount of resources that can be allocated to reproduction, which delays the reproductive peak. Second, the cumulative effect of investment in early-life reproduction under good early-life conditions accelerates senescence. For example a recent model specifically suggested that the cost of reproduction decreases the variance in lifetime reproductive success and organisms that start to reproduce earlier will also senesce earlier (Coste *et al.* 2017). However, whether females do indeed make similar reproductive decisions under natural conditions remains largely unknown.

We studied the long-lasting effects of natal environment on age-specific survival and reproduction in female Collared Flycatchers (*Ficedula albicollis*) from a natural population on the Swedish island of Gotland. Competition is an important aspect of early environment and earlier studies have shown that experimental increase in brood size results in reduced body mass at fledgling and increased offspring mortality (Alatalo *et al.* 1990; Tinbergen & Boerlijst 1990; de Kogel 1997; Sendecka *et al.* 2007). We experimentally manipulated the brood size of hundreds of nesting pairs across 26 breeding seasons (i.e. 1983–2009) to create either artificially high-competition (increased) or low-competition (reduced) broods. The nests were paired by matching hatching dates and two nestlings were transferred from a nest to the other for the reduced and enlarged broods or exchanged between nests for the control broods. Individuals from nests with a reduced brood size experience a 'silver-spoon' upbringing and grow to a larger body size, a proxy for body condition, at fledgling (Voillemot *et al.* 2012), allowing us to test directly whether experimentally improved developmental conditions decelerate or accelerate reproductive and actuarial ageing. To our knowledge, this is the first study that directly investigates the consequences of individual heterogeneity in early-life condition for ageing in an experimentally manipulated natural population.

METHODS

Study species and study area

Collared flycatchers (*Ficedula albicollis*) are small (*c.* 13 g), migratory, insectivorous, cavity-nesting passerines, that breed in deciduous and mixed forests of eastern and central Europe (and southwestern Asia). Females incubate four to eight eggs (mean ± SD = 6.3 ± 0.65) during a 14-day period and rear a single brood each year. Juveniles are fed by both parents and fledge 14–18 days after hatching (for further details on population and methods see (Gustafsson 1989)).

Isolated from their main breeding grounds, additional breeding populations have been established on the Swedish islands of Gotland and Öland in the Baltic Sea. The collared flycatcher population on the island of Gotland (57° 10´, 18° 20´) has been well studied since 1980. Each year, new individuals (i.e. both adults and offspring) are measured and ringed, to allow individual identification throughout their lifetime and enable the assessment of yearly reproductive success. The females are caught at the nest during incubation or the chick provisioning period and the males only during the chick provisioning period. Further details of the breeding region, and ecology have been collected using standard methods (Gustafsson 1989; Pärt & Gustafsson 1989).

Collared flycatchers in general and the long-term studied Gotland population in specific, are particularly suitable for the study of life-history trade-offs in nature due to their high degree of site fidelity, limited dispersal and preference for nest boxes over natural cavities, which allows accurate age and survival estimation (Pärt & Gustafsson 1989).

### Treatments and data selection

Since 1983, multiple brood size manipulation experiments have been conducted in the Gotland population (Gustafsson & Sutherland 1988; Nordling *et al.* 1998; Doligez *et al.* 2002; Sendecka *et al.* 2007), to examine various aspects of parental investment, the costs of increased reproductive effort and the trade-off between reproductive investment and immune function. To investigate the effects of early-life natal conditions on individual life histories, we collected data from individuals raised in manipulated nests, where brood size was increased or reduced by two offspring. In addition, we accounted for possible translocation effects by including a control treatment, in which offspring were swapped between nests without alteration of the initial total number (Fig. 1). The distribution of brood size after treatment was flatter but the number of nestlings remained in the range of the natural variation. The average brood size (mean ± SE) for reduced broods was 4.33 ± 0.05, for enlarged broods was 8.31 ± 0.04 and for control broods was 6.18 ± 0.05. We monitored the entire life-history of 943 collared flycatchers, which were raised in such manipulated nests, but eventually focused on females, due to the degree of male extra-pair paternity (*c.* 15% − Sheldon & Ellegren 1999) that makes the process of estimating male reproductive values challenging.

The experimental reduction of the brood size resulted in increased body mass at fledgling (15.12 ± 0.12, $\chi^2 = 10.69$, $P < 0.01$, Fig. 2a), whereas there was no effect of body size measured as tarsus length (19.56 ± 0.05, $\chi^2 = 0.52$, $P = 0.77$, Fig. S1). This suggests that brood reduction indeed improved natal environment and body condition of the offspring, rather than simply their structural size. In support of these findings, previous studies showed that experimental increase in brood size results in reduced body mass at fledgling and increases offspring mortality (Alatalo *et al.* 1990; Tinbergen & Boerlijst 1990; de Kogel 1997; Sendecka *et al.* 2007).

Reproductive and survival data were collected for 514 females born between 1983 and 2009. 122 females were raised in nests with reduced broods ('low competition'), 215 females were raised in controlled nests ('control') and 177 females were raised in nests with increased broods ('high competition'). For each female, we recorded their reproductive success, as the number of recruits (i.e. offspring that returned to Gotland to breed the following years, after overwintering in Africa; Fig. 1) during each reproductive season. Females
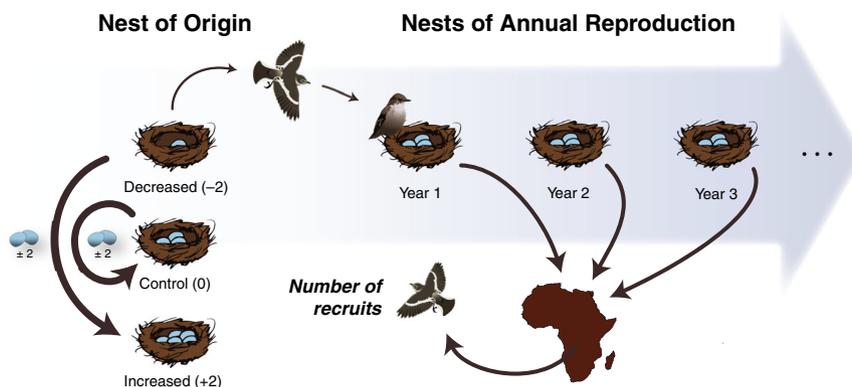
Figure 1 The graphical outline of the experimental design. The experimental brood size manipulation of the nest of origin is illustrated on the left and the age-specific reproductive data (i.e. number of recruits) collected from the nests of annual reproduction are illustrated on the right.
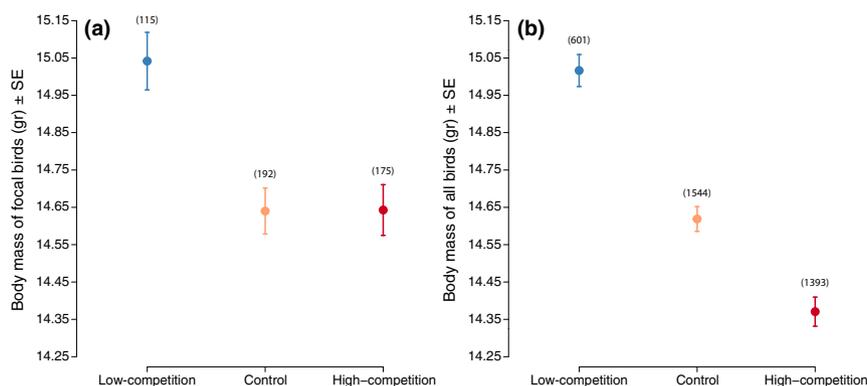
Figure 2 (a) Body mass at fledgling (means ± SE) of the focal birds raised in the three manipulated natal environments. (b) Body mass at fledgling (means ± SE) of all the birds raised in the three manipulated natal environments. Blue lines represent the increased low-competition treatment, orange lines represent the control treatment and red lines represent the decreased high-competition treatment.

failing to breed on a given year were attributed a reproductive success of 0.

This individual-based monitoring provides access to age-specific reproductive success (annual number of recruits), lifetime reproductive success (sum of recruits produced over lifetime) and the rate-sensitive individual fitness λ_{ind}, which encompasses the number of offspring, as well as their timing (Brommer *et al.* 2002; Lind *et al.* 2016) and is analogous to the intrinsic rate of population growth (Stearns 1992).

Over the years, some of our focal females ($N = 205$) were involved in additional experiments addressing different questions (Nordling *et al.* 1998; Doligez *et al.* 2002; Pitala *et al.* 2009). We made the hypothesis that these experiments do not have a strong impact on survival, and kept all the females for survival analyses, so the data set included the 514 monitored females. In turn these experiments directly influence reproductive success. When investigating reproductive success, we excluded the current and/or future reproductive values of each affected bird, depending on the nature of these additional experimental manipulations and the effect they may have on the female's reproduction. Due to these experiments, the full data set for age-specific reproductive success included 783 observations of 435 females. The data set for lifetime reproductive success restricted to females that were never

manipulated during their lifetime included 534 observations of 307 females (i.e. we excluded 205 females that participated in the additional experiments and 2 more females that had missing values in their life histories).

The age-specific reproductive patterns were similar in both the full ($N_{females} = 435$) and the restricted data set $N_{females} = 307$), so we only present results for the full data set. When analysing lifetime reproductive success and the rate-sensitive individual fitness (λ_{ind}), any female who had been involved in an experiment affecting breeding success during her lifetime should be excluded from the data set, thus we only used the restricted data set.

Statistical analysis

Reproductive success analyses

Age-specific reproductive success. To analyse the effects of the brood size manipulation on the age-specific reproduction, we used both a frequentist and a Bayesian framework, which yielded similar results. Thus, we describe below the frequentist approach, but see Supplementary Methods for further details on the Bayesian approach. We constructed a generalised linear mixed-effects model (GLMM) with Poisson distribution and log link function. We accounted for the non-independence of

data by including individual identity, nest of origin identity and year of annual reproduction as random effects. Furthermore, we included an observation-level random effect to control for overdispersion (Harrison 2014). As fixed effects, we included the treatment (increased, reduced and control broods), as well as a linear and a quadratic term of age (Age and Age^2), to test for nonlinear variation of reproductive value with age. In addition, we included the Age of First Reproduction (AFR) and Age at Last Reproduction (ALR) in our models, to control for selective appearance and selective disappearance (van de Pol *et al.* 2006b). All continuous predictor variables were mean centred and standardised (mean = 0 and SD = 1). Centring to the mean allows to give a natural meaning to the intercept, because the intercept is the predicted value when all fixed effects are set to 0. Furthermore, scaling to unit variance allows to obtain estimates of coefficients that can be compared across variables, that is an interpretation of relative effect sizes. We initially run a complete model including all fixed effects and up to three-way interactions when biologically meaningful (Full model, eqn 1).

$$
\begin{aligned}
ARS_{ij} = {} & \mu + \alpha_1 T_i + \alpha_2 A_{ij} + \alpha_3 A_{ij}^2 + \alpha_4 ALR_i + \alpha_5 AFR_i + \alpha_6 T_i A_{ij} \\
& + \alpha_7 T_i A_{ij}^2 + \alpha_8 T_i ALR_i + \alpha_9 T_i AFR_i + \alpha_{10} A_{ij} ALR_i \\
& + \alpha_{11} A_{ij}^2 ALR_i + \alpha_{10} A_{ij} AFR_i + \alpha_{11} A_{ij}^2 AFR_i \\
& + \alpha_{12} T_i A_{ij} ALR_i + \alpha_{13} T_i A_{ij}^2 ALR_i + \alpha_{12} T_i A_{ij} AFR_i \\
& + ind_i + year_j + \varepsilon_{ij}
\end{aligned}
$$
$$(1)$$

where the breeding success of female i in year j is explained by several fixed effects: early life conditions (treatment, T_i), age A_{ij} and age-squared A_{ij}^2 of female i in year j, age at first (AFR_i) and last breeding (ALR_i) of female i and their interactions. To take into account the non-independence of data collected on the same female and the same year, we included female identity (ind_i) and year ($year_j$) as random effects. The final term in equation 1 is the residual error term ε_{ij}.

We then evaluated which interaction to retain based on the conservative Akaike Information Criterion for small sample sizes (AIC$_c$). Three of these models had the smallest AIC$_c$ values (with a difference of less than 2 points), indicating best model fit (models 17, 24 and 31; for models details and differences see Supplementary Material and Table S4). They all gave similar outcomes and we therefore only present here the most comprehensive model (model 17) that includes most variables, but see supplementary material for the output of the other two models (Table S5).

All analyses were performed in *R* version 3.3.1 (R Development Core Team 2017). Statistical models were fitted using the *lme4* package (v.1.1-14 – Bates *et al.* 2015) and the *MCMCglmm* package (v.2.23 – Hadfield 2010). For generalised linear mixed models (Poisson error structure), we ensured an appropriate fit using the *DHARMa* package (v. 0.1.2) for diagnostic tests of model residuals (Hartig 2016). We found no evidence of zero-inflation. We used the package *AICcmodavg* (v. 2.1-0 – Mazerolle 2016) to compute the Akaike's Information Criterion for small sample sizes (AICc). To test for significance of model terms we used the Anova

function of the *car* package (v. 2.1-3) with Type III Wald chi-square tests. *Post hoc* comparisons were performed with the *emmeans* function in the *emmeans* package (v. 1.4.4).

Lifetime reproductive success. For the lifetime number of recruits, we tested the treatment effect using a GLMM with Poisson distribution and log link function. We included nest of origin identity and year of birth as random effects to control for cohort effects (Table S2).

Finally, for the analysis of rate-sensitive individual fitness, we first estimated λ_{ind} by solving the Euler–Lotka equation of age-specific recruit number and survival for each individual using the *lambda* function in the *popbio* package (v 2.4.4 – Stubben & Milligan 2007). Then we tested for the effect of treatment on the log of λ_{ind} in a linear mixed model (LMM) including the nest of origin and year of birth were used as random factors to control for cohort effects (Table S3).

Survival analysis

To investigate how early-life developmental conditions affect mortality rates of female collared flycatchers, we performed capture-mark-recapture (CMR) survival analysis on our data (Lebreton *et al.* 1992). Age-specific mortality trends was inferred using the Bayesian Survival Trajectory Analysis framework (BaSTA package, v. 1.9.5) (Colchero *et al.* 2012). BaSTA is a powerful package to fit age-specific mortality function on CMR data where information about the exact age of death is missing (right-censored data) (Colchero & Clark 2012).

The survival analyses were performed on individual encounter histories. Models assume no dispersal, which could bias the effect of natal environment on actuarial senescence. Indeed, collared flycatchers show high site fidelity and very limited dispersal (Pärt & Gustafsson 1989). Given that the year of birth is known for all individuals, our CMR analyses correspond to the classical Comark–Jolly–Seber (CJS) framework (Lebreton *et al.* 1992). To ensure that the CJS assumptions about trap-dependency and transience are met and models adequately fit the data, we first performed a goodness of fit analysis with the U-care Program (Choquet *et al.* 2009). Then, we found the best model that describes the recapture rate (see Supplementary Methods for all details). The probability to recapture individuals in our data was found to be constant and equal to 60% (Table S10).

We explored actuarial age-specific mortality rates using Weibull, Gompertz and Logistic functions, with either a simple shape or one of the more complex Makeham (Pletcher 1999) or bathtub (Siler 1979) shapes. The nine previous models were compared to a model without senescence (exponential model with a simple shape). The age-specific mortality curves were fitted starting from 1 year old, the minimum age of reproduction (Supplementary Methods). We performed four parallel simulations that each model for 1 200 000 iterations, used a burn-in of 200 000 iterations and took a model sample each 500 iterations. The model parameters likely converged on the most optimal mode and showed low serial autocorrelations (< 5%) and robust posterior distributions (with N = 2 000). For the model comparison, we used the lowest deviance

information criterion (DIC), which is similar to the Akaike Information Criterion (AIC) in non-Bayesian models. Comparison of the DIC values (Millar 2009) revealed that the Logistic model with the bathtub shape (eqn 2) is the most appropriate mortality function (Table S9). Mortality rate:

$$\mu_0(x|\boldsymbol{b},\boldsymbol{a},c) = e^{a_0-a_1x} + c + \frac{e^{b_0+b_1x}}{1+b_2\frac{e^{b_0}}{b_1}(e^{b_1x}-1)} \tag{2}$$

The bathtub structure used in the BaSTA package adds a declining Gompertz function and a constant to the basic logistic mortality function. Therefore, the two alpha parameters (a_0 and a_1) describe the exponential decline that may be observed very early in life and the constant (c) describe the lowest point of the mortality function. The beta parameters (b_0, b_1 and b_2) describe different parts of the logistic increase in mortality rates with age (i.e. initial exponential increase in mortality that decelerates until it reaches a plateau). More specifically, b_0 is the age-independent or baseline mortality, b_1 describes the initial exponential increase in mortality with age and b_2 describes the degree of deceleration in mortality with age and the level of the asymptote.

To assess the differences among the posterior distributions of our brood size manipulation treatments we used the Kullback–Leibler divergence calibration (KLDC) (Kullback & Leibler 1951; McCulloch 1989; Karabatsos 2006; Boonekamp et al. 2014; Atema et al. 2016; Rodríguez-Muñoz et al. 2019), which show the mean Kullback–Leibler discrepancies (KL) between two distributions. Values closer to 0.5 imply that there is minimal difference among the distributions and values closer to 1 imply major differences (Kullback & Leibler 1951; McCulloch 1989). We considered a KLDC value> 0.85 to

indicate a substantial difference between the two posterior distributions that are compared (Fig. 5).

RESULTS

While the high-competition treatment decreased body mass at fledgling when considering all fledglings (Voillemot et al. 2012, overall: 15.02 ± 0.09, $\chi^2 = 36.35$, $P < 0.01$, high-competition vs. control: post hoc Tukey: $P = 0.015$; Fig. 2b), this effect disappears when restricting the analysis to recruited females (overall: 15.12 ± 0.12, $\chi^2 = 10.73$, $P < 0.01$, high-competition vs. control: post hoc Tukey: $P = 0.989$, Fig. 2a).

Overall, the quality of the natal environment had no significant effect on lifetime reproduction ($\chi^2 = 0.99$, $P = 0.610$; Fig. 3c, Table S2) or individual fitness λ_{ind} ($\chi^2 = 1.07$, $P = 0.585$; Fig. 3d, Table S3) in this study. In terms of age-specific reproductive success, females from the low-competition treatment had a high success during the early years of their life, reaching a peak in their second year of breeding (Fig. 3a and b, Table 1). In contrast, females raised in the high-competition treatment started with a lower reproductive success, but steadily increased their performance reaching a peak in their third year and enjoyed a later onset of senescence (Fig. 3a and b, Table 1). More specifically, while the raw data suggest that individuals from high-competition nests maintained high reproductive performance in their fifth year (Fig. 3a), taking into account selective appearance (measured as age at first reproduction) and disappearance (measured as age at last reproduction) reveals a reproductive decline after year four (Fig. 3b). Nevertheless, high-competition females showed a delayed reproductive peak and started to senesce
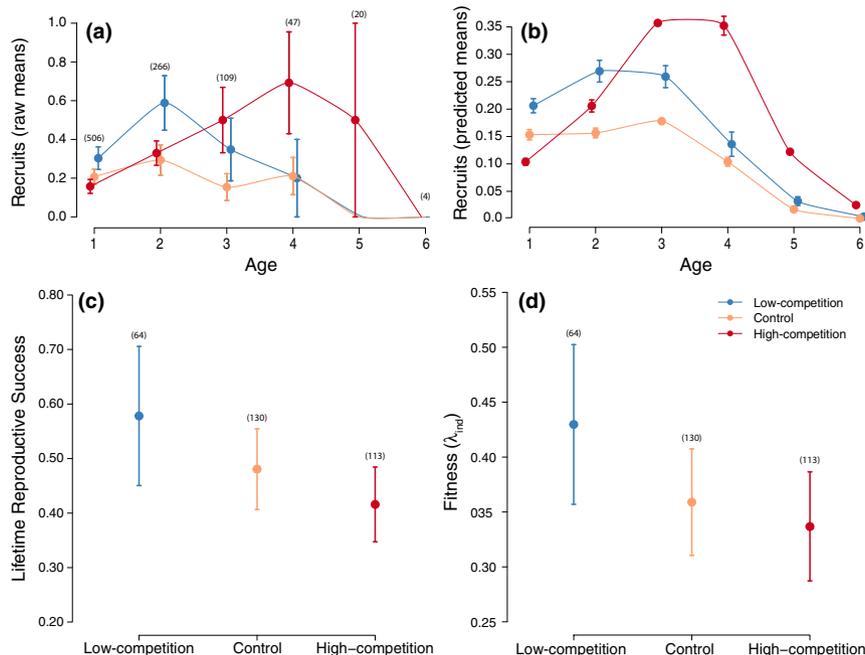
Figure 3 Reproduction of the three manipulated natal environments (Means ± SE). (a) Age-specific reproduction from the raw data. The sample sizes per age class are indicated with numbers in parentheses (Table S1); (b) Age-specific reproduction from the model predictions based on all random and fixed effects included in model 17 (Table 1); (c) Lifetime Reproductive Success; (d) Individual fitness (λ_{ind}). Blue lines represent the decreased low-competition treatment, orange lines represent the control treatment and red lines represent the increased high-competition treatment.

Table 1 Generalised linear mixed effects model examining the effect of early life environmental conditions on age-specific reproduction in 434 females (N_{obs} = 781)

Variable	Estimate ± SE	Z	P-value (Z)	χ^2	d.f.	P-value (χ^2)	Variance
Model 17							
Intercept	**−1.69 ± 0.26**	**−6.43**	**< 0.001**	**41.3**	**1**	**< 0.001**	
Age	**0.66 ± 0.27**	**2.49**	**0.012**	**6.20**	**1**	**0.013**	
Age^2	**−0.47 ± 0.13**	**−3.68**	**< 0.001**	**13.52**	**1**	**< 0.001**	
Age at first repr. (AFR)	**−0.89 ± 0.17**	**−5.34**	**< 0.001**	**28.49**	**1**	**< 0.001**	
Age at last repr. (ALR)	0.30 ± 0.19	1.60	0.111	2.53	1	0.111	
Nest treatment				3.65	2	0.161	
Control Trt	−0.34 ± 0.22	−1.59	0.120				
High-competition Trt	−0.37 ± 0.22	−1.74	0.082				
Interaction (Age × ALR)	0.14 ± 0.12	1.10	0.272				
Interaction (Age × AFR)	**0.89 ± 0.24**	**3.80**	**< 0.001**	**14.46**	**1**	**< 0.001**	
Interaction (Age^2 × AFR)	−0.19 ± 0.10	−1.84	0.065	3.40	1	0.065	
Interaction (Age × Trt)				**7.83**	**2**	**0.019**	
Age × Control Trt	0.23 ± 0.28	0.80	0.421				
Age × High-competition Trt	**0.74 ± 0.28**	**2.67**	**0.008**				
Interaction (ALR × Trt)				5.10	2	0.078	
ALR × Control Trt	**−0.54 ± 0.26**	**−2.08**	**0.037**				
ALR × High-competition Trt	−0.47 ± 0.26	−1.83	0.067				
Random effects							
Female ID							0.00
Nest ID							0.26
Year of annual repr.							0.42
Observation							0.19

The reference level for treatment is 'Low-competition'. Significant effects are in bold.

later than their counterparts from low-competition nests for whom senescence started a year earlier. Females raised in control nests started at intermediate level of reproduction, maintained relatively steady reproductive performance during the first 3 years of their life and declined at the fourth year.

Natal environment also had profound effects on age-specific mortality rates (Fig. 4, see Table S11 for age specific survival probabilities and reproductive rates). Low-competition females had substantially lower initial acceleration in mortality rate (smaller early-life acceleration parameter b_1 with KLDC > 0.85 – Fig. 4, Fig. 5 and Table S10) than both control and high-competition females, suggesting that 'silver-spoon' natal environment is beneficial for survival. However, while the mortality rates of control and high-competition females strongly decelerated in late-life and reached a plateau around 4 years of age, the mortality rate of low-competition females continued to increase and reached a higher plateau around the fifth year (smaller late-life deceleration parameter b_2 with KLDC > 0.85 – Figs 4 and 5 and Table S10).

DISCUSSION

We found that females experiencing a low-competition natal environment fledge at higher body mass, start to reproduce at a higher rate, have an earlier reproductive peak and have a lower acceleration of mortality rate than their counterparts raised in high-competition nests. However, low-competition females also show earlier signs of reproductive senescence and suffer from increased mortality rate in the last years of their lives. Thus, our results provide an experimental demonstration of condition-dependent ageing in a natural population. Specifically, these findings support the hypothesis that high-

condition females raised in 'silver-spoon' natal environment allocate their resources to early-life reproduction, but pay a cost in terms of accelerated ageing in late-life.

There has been a sustained interest in how resource availability affects age-specific life histories but, remarkably, both theoretical and empirical studies to date are focused exclusively on the role of sexual selection in driving rapid ageing in high-condition males (i.e. 'live-fast die-young' hypothesis) (Kokko 1998; Radwan & Bogacz 2000; Hunt *et al.* 2004; Bonduriansky & Brassil 2005; Bonduriansky *et al.* 2008; Preston *et al.* 2011; Adler *et al.* 2016; Hooper *et al.* 2017, 2018). This is, in part, because of the striking absence of studies showing that high-condition females may also trade-off early-life reproduction for accelerated ageing (but see Travers *et al.* 2015). Here, we used an experimental manipulation of early-life environment to show that 'silver-spoon' (i.e. low-competition) females indeed benefit from elevated early-life reproductive success, but suffer from an earlier onset of reproductive ageing and increased mortality late in life. Thus, our findings call for a re-appraisal of the idea that 'live-fast die-young' strategy applies primarily to males when faced with increased sexual competition (Bonduriansky *et al.* 2008; Hooper *et al.* 2018).

Furthermore, our results suggest, that we should look beyond a simple negative correlation between net reproduction and longevity when investigating the long-term fitness consequences of early-life conditions. Specifically, in this study 'silver-spoon' females did not live shorter, nor reproduced more, but rather had different age-specific mortality and reproduction rates. It is possible that stochastic natural environment masks the effect of our brood size manipulation on total reproduction of the recruits and that the addition of several more years of data collection could show that high-
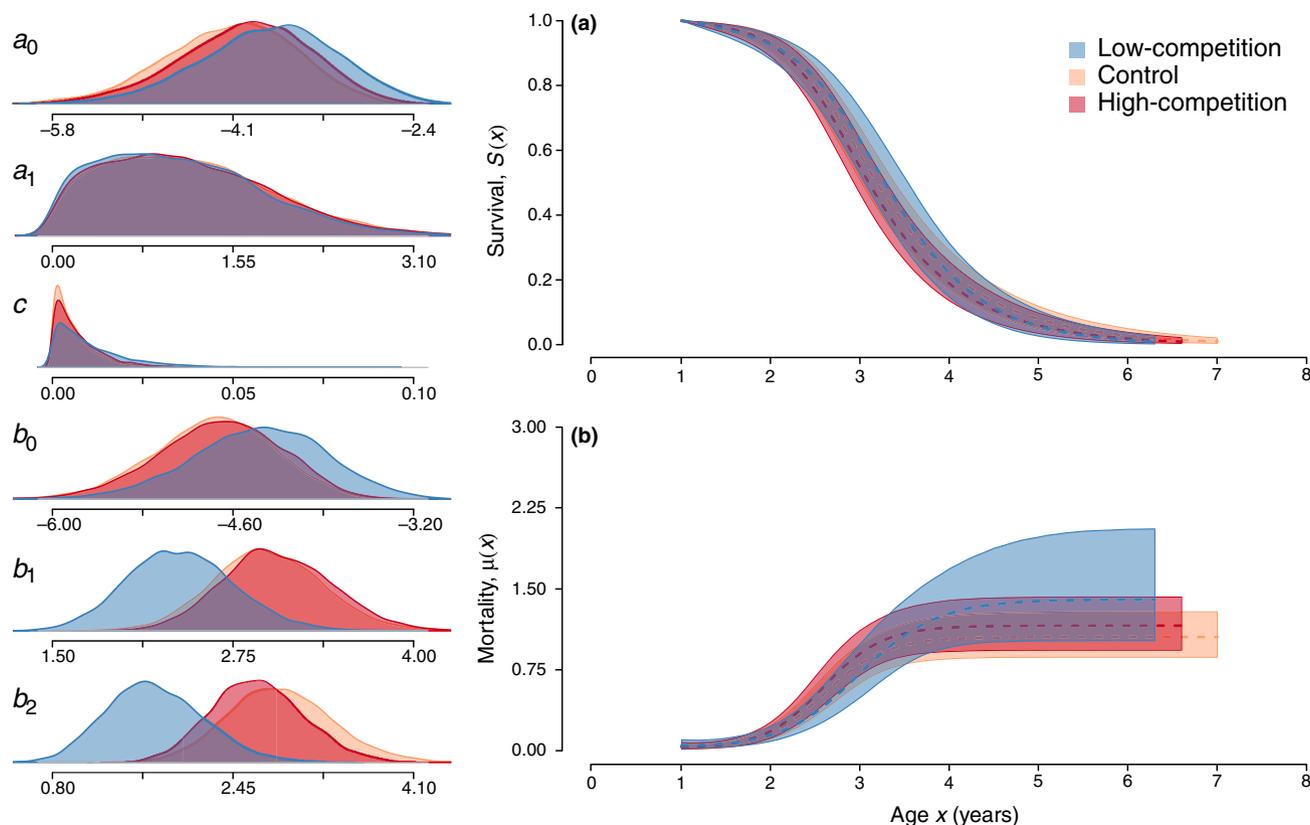
Figure 4 Survival (a) and Mortality (b) curves with 95% confidence intervals for each treatment as fitted by the bathtub Logistic mortality model. On the left are the posterior distributions of the six model parameters (see Methods for details). Blue lines and distributions represent the low-competition treatment, orange represent the control treatment and red represent the high-competition treatment.
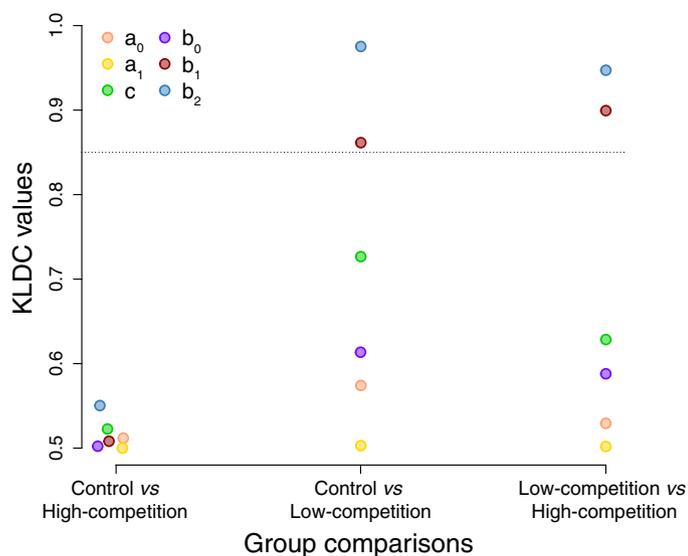
Figure 5 Values of the Kullback–Leibler divergence calibration (KLDC), which compare parameter posterior distributions of the bathtub Logistic mortality model, between the high-competition, the control and the low-competition treatments. The values that are above the dotted line (KLDC = 0.85) show parameters that are substantially different between the two treatments in each group comparison. The parameters a_0 and a_1 describe the exponential decline that may be observed very early in life and c describe the lowest point of the mortality function. b_0, b_1 and b_2 describe different parts of the logistic increase in mortality rates with age (see Methods for details)

condition females actually have higher fitness. At this point, however, we can only state that a 'silver-spoon' early-life environment results in increased early-life but reduced late-life performance. High-competition females fledge at lower body mass then low-competition birds and the control birds but those that survive to come back and reproduce have the same body mass as control birds. This suggests that high-competition treatment resulted in increased mortality of birds that had low body mass as fledglings.

Our results are in line with the 'disposable soma' theory of ageing, which maintains that early-life investment in growth and reproduction results in reduced late-life fitness due to an increased amount of unrepaired cellular damage (Kirkwood & Holliday 1979; Kirkwood 2002, 2017). However, such patterns can also emerge directly from upregulation of nutrient-sensing molecular signalling pathways in 'silver-spoon' females that received more food during development, resulting in increased cellular hypertrophy and accelerated cellular senescence in late life (Gems & Partridge 2013; Maklakov & Chapman 2019). Future research should focus on understanding the physiological and molecular underpinnings of the phenomenon that improved early-life conditions lead to detrimental effects in late-life.

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