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1 **Letter: Silver-spoon upbringing improves early-life fitness**
2 **but promotes reproductive ageing in a wild bird**

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15
16
17 **Running Title:** Good natal conditions fasten ageing in nature
18

19 **Authorship Statement**

20 L.G. collected the data. A.A.M, L.G. and F.S conceived the project. F.S., C.T. and
21 M.I.L. performed the reproductive data analysis. F.S and S.C. performed the survival
22 analysis. A.A.M. and L.G. contributed to data analysis and interpretation. F.S., C. T.
23 and A.A.M wrote the manuscript.

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28

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31

32 Data will be made available in DRYAD upon acceptance

33 **Abstract**

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

48

49 **Introduction**

50 Ageing, a physiological deterioration with advancing age, is a complex and highly
51 variable trait that is widespread across the tree of life (Jones *et al.* 2014; Shefferson
52 *et al.* 2017) and affects organismal fitness in nature (Bouwhuis *et al.* 2012; Nussey *et al.*
53 *et al.* 2013). Theory maintains that ageing evolves because selection gradients on traits
54 decline with age (Medawar 1952; Williams 1957; Hamilton 1966), favouring alleles
55 that increase early-life fitness even at the cost of negative pleiotropic fitness effects
56 in late-life. While a large body of evidence supports these theoretical predictions on
57 the population level, very little is known about the individual variation in ageing rates.
58 There is a general agreement in the field that early-life environmental conditions can
59 shape the life-history of an adult organism and explain much of the individual
60 variation in ageing rates (Monaghan 2008; Marshall *et al.* 2017; Cooper & Kruuk
61 2018), but there is little consensus regarding the direction of the effect. Good
62 environmental conditions during development can result in lifelong positive effects on
63 physiology, reproduction and longevity (Lindström 1999; van de Pol *et al.* 2006a;
64 Nussey *et al.* 2007; Hayward *et al.* 2013; Cooper & Kruuk 2018) (“silver-spoon”
65 hypothesis – Grafen 1990). Alternatively, organisms raised in good natal
66 environments can invest heavily in growth and early-life reproduction, resulting in
67 accelerated senescence in late-life (Hunt *et al.* 2004; Bonduriansky & Brassil 2005;
68 Hooper *et al.* 2017; Marshall *et al.* 2017) (“live-fast die-young” hypothesis –
69 Promislow & Harvey 1990). These studies suggest that organisms can allocate
70 resources to reproduction and somatic maintenance depending on their early-life
71 environmental conditions, and that high latent costs of increased early-life growth
72 and reproduction, under favourable conditions, can result in accelerated ageing
73 (Adler *et al.* 2016; Hooper *et al.* 2017).

74 Previous studies emphasised the role of sexual selection in the evolution of
75 resource allocation strategies that can result in rapid ageing under favourable natal
76 conditions. Males, for example, are predicted to invest in costly secondary sexual
77 traits and risky behaviours, trading-off with investment in somatic maintenance and
78 late-life performance (Hunt *et al.* 2004; Bonduriansky & Brassil 2005; Nussey *et al.*
79 2007; Hooper *et al.* 2017). However, recent studies also show that early-life
80 investment in reproduction trades-off with late-life fitness in females in natural
81 populations (reviewed in Lemaître *et al.* 2015) and in the laboratory (Travers *et al.*
82 2015). This suggests that “silver-spoon” females could allocate more resources to
83 early-life reproduction and suffer from faster senescence later in life, while females
84 experiencing early-life stress could reduce their investment in early-life reproduction
85 and delay senescence. Interestingly, mild stress results in reduced early but
86 improved late breeding performance in captive female Zebra Finches (*Taeniopygia*
87 *guttata*) (Marasco *et al.* 2018). Overall, it is possible that two related processes
88 shape the expression of age-specific life-histories in response to differences in early-
89 life conditions. First, poor conditions in early-life constrain the amount of resources
90 that can be allocated to reproduction, which delays the reproductive peak. Second,
91 the cumulative effect of investment in early-life reproduction under good early-life
92 conditions accelerates senescence. For example, a recent model specifically
93 suggested that the cost of reproduction decreases the variance in lifetime
94 reproductive success and organisms that start to reproduce earlier will also senesce
95 earlier (Coste *et al.* 2017). However, whether females do indeed make similar
96 reproductive decisions under natural conditions remains largely unknown.

97 We studied the long-lasting effects of natal environment on age-specific survival and
98 reproduction in female Collared Flycatchers (*Ficedula albicollis*) from a natural

99 population on the Swedish island of Gotland. Competition is an important aspect of
100 early environment and earlier studies have shown that experimental increase of
101 brood size results in reduced body mass at fledgling and increased offspring
102 mortality (Alatalo *et al.* 1990; Tinbergen & Boerlijst 1990; de Kogel 1997; Sendecka
103 *et al.* 2007). We experimentally manipulated the brood size of hundreds of nesting
104 pairs across 26 breeding seasons (i.e. 1983-2009) to create either artificially high-
105 competition (increased) or low-competition (reduced) broods. The nests were paired
106 by matching hatching dates and two nestlings were transferred from a nest to the
107 other for the reduced and enlarged broods or exchanged between nests for the
108 control broods. Individuals from nests with a reduced brood size experience a “silver-
109 spoon” upbringing and grow to a larger body size, a proxy for body condition, at
110 fledgling (Voillemot *et al.* 2012), allowing us to test directly whether experimentally
111 improved developmental conditions decelerate or accelerate reproductive and
112 actuarial ageing. To our knowledge, this is the first study that directly investigates the
113 consequences of individual heterogeneity in early-life condition for ageing in an
114 experimentally manipulated natural population.

115

116 **Methods**

117 **Study species and study area**

118 Collared flycatchers (*Ficedula albicollis*) are small (~ 13g), migratory, insectivorous,
119 cavity-nesting passerines, that breed in deciduous and mixed forests of eastern and
120 central Europe (and southwestern Asia). Females incubate four to eight eggs (mean
121 \pm sd = 6.3 ± 0.65) during a 14-day period and rear a single brood each year.

122 Juveniles are fed by both parents and fledge 14 to 18 days after hatching (for further
123 details on population and methods see (Gustafsson 1989)).

124

125 Isolated from their main breeding grounds, additional breeding populations have
126 been established on the Swedish islands of Gotland and Öland in the Baltic Sea.

127 The collared flycatcher population on the island of Gotland (57° 10', 18° 20') has
128 been well studied since 1980. Each year, new individuals (i.e. both adults and
129 offspring) are measured and ringed, to allow individual identification throughout their
130 lifetime and enable the assessment of yearly reproductive success. The females are
131 caught at the nest during incubation or the chick provisioning period and the males
132 only during the chick provisioning period. Further details of the breeding region, and
133 ecology have been collected using standard methods (Gustafsson 1989; Pärt &
134 Gustafsson 1989).

135

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

141 **Treatments and data selection**

142 Since 1983, multiple brood size manipulation experiments have been conducted in
143 the Gotland population (Gustafsson & Sutherland 1988; Nordling *et al.* 1998; Doligez
144 *et al.* 2002; Senddecka *et al.* 2007), to examine various aspects of parental
145 investment, the costs of increased reproductive effort and the trade-off between
146 reproductive investment and immune function. To investigate the effects of early-life

147 natal conditions on individual life histories, we collected data from individuals raised
148 in manipulated nests, where brood size was increased or reduced by two offspring.
149 In addition, we accounted for possible translocation effects by including a control
150 treatment, in which offspring were swapped between nests without alteration of the
151 initial total number (Fig. 1). The distribution of brood size after treatment was flatter
152 but the number of nestlings remained in the range of the natural variation. The
153 average brood size (mean \pm S.E.) for reduced broods was 4.33 ± 0.05 , for enlarged
154 broods was 8.31 ± 0.04 and for control broods was 6.18 ± 0.05 . We monitored the
155 entire life-history of 943 collared flycatchers, which were raised in such manipulated
156 nests, but eventually focused on females, due to the degree of male extra-pair
157 paternity (~15% – Sheldon & Ellegren 1999) that makes the process of estimating
158 male reproductive values challenging.

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

168 Reproductive and survival data were collected for 514 females born between
169 1983 and 2009. 122 females were raised in nests with reduced broods (“low
170 competition”), 215 females were raised in controlled nests (“control”) and 177
171 females were raised in nests with increased broods (“high competition”). For each

172 female, we recorded their reproductive success, as the number of recruits (i.e.
173 offspring that returned to Gotland to breed the following years, after overwintering in
174 Africa; Fig. 1) during each reproductive season. Females failing to breed on a given
175 year were attributed a reproductive success of 0.

176

177 This individual-based monitoring provides access to age-specific reproductive
178 success (annual number of recruits), lifetime reproductive success (sum of recruits
179 produced over lifetime) and the rate-sensitive individual fitness λ_{ind} , which
180 encompasses the number of offspring, as well as their timing (Brommer *et al.* 2002;
181 Lind *et al.* 2016) and is analogous to the intrinsic rate of population growth (Stearns
182 1992).

183

184 Over the years, some of our focal females (N = 205) were involved in additional
185 experiments addressing different questions (Nordling *et al.* 1998; Doligez *et al.* 2002;
186 Pitala *et al.* 2009). We made the hypothesis that these experiments do not have a
187 strong impact on survival, and kept all the females for survival analyses, so the data
188 set included the 514 monitored females. In turn these experiments directly influence
189 reproductive success. When investigating reproductive success, we excluded the
190 current and/or future reproductive values of each affected bird, depending on the
191 nature of these additional experimental manipulations and the effect they may have
192 on the female's reproduction. Due to these experiments, the full data set for age-
193 specific reproductive success included 783 observations of 435 females. The data
194 set for lifetime reproductive success restricted to females that were never
195 manipulated during their lifetime included 534 observations of 307 females (i.e. we

196 excluded 205 females that participated in the additional experiments and 2 more
197 females that had missing values in their life histories) .

198

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

205 **Statistical analysis**

206 *Reproductive success analysis:*

207 Age-specific reproductive success

208 To analyse the effects of the brood size manipulation on the age-specific
209 reproduction, we used both a frequentist and a Bayesian framework, which yielded
210 similar results. Thus, we describe below the frequentist approach, but see
211 Supplementary Methods for further details on the Bayesian approach. We
212 constructed a generalised linear mixed-effects model (GLMM) with Poisson
213 distribution and log link function. We accounted for the non-independence of data by
214 including individual identity, nest of origin identity and year of annual reproduction as
215 random effects. Furthermore, we included an observation-level random effect to
216 control for overdispersion (Harrison 2014). As fixed effects, we included the
217 treatment (increased, reduced and control broods), as well as a linear and a
218 quadratic term of age (Age and Age²), to test for non-linear variation of reproductive
219 value with age. In addition, we included the Age of First Reproduction (AFR) and
220 Age at Last Reproduction (ALR) in our models, to control for selective appearance

221 and selective disappearance (van de Pol *et al.* 2006b). All continuous predictor
 222 variables were mean centered and standardised (mean = 0 and SD = 1). Centering
 223 to the mean allows to give a natural meaning to the intercept, because the intercept
 224 is the predicted value when all fixed effects are set to 0. Furthermore, scaling to unit
 225 variance allows to obtain estimates of coefficients that can be compared across
 226 variables i.e. an interpretation of relative effect sizes. We initially run a complete
 227 model including all fixed effects and up to three-way interactions when biologically
 228 meaningful (Full model, Eq. 1).

229

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

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

239 We then evaluated which interaction to retain based on the conservative Akaike
 240 Information Criterion for small sample sizes (AIC_c). Three of these models had the
 241 smallest AIC_c values (with a difference of less than 2 points), indicating best model fit
 242 (models 17, 24 and 31; for models details and differences see Supplementary
 243 Material and Supplementary Table S4). They all gave similar outcomes and we
 244 therefore only present here the most comprehensive model (model 17) that includes

245 most variables, but see supplementary material for the output of the other two
246 models (Supplementary Table S5).

247

248 All analyses were performed in *R* version 3.3.1 (R Development Core Team
249 2017). Statistical models were fitted using the *lme4* package (v.1.1-14 – Bates *et al.*
250 2015) and the *MCMCglmm* package (v.2.23 – Hadfield 2010). For generalized linear
251 mixed models (Poisson error structure), we ensured an appropriate fit using the
252 *DHARMA* package (v. 0.1.2) for diagnostic tests of model residuals (Hartig 2016).
253 We found no evidence of zero-inflation. We used the package *AICcmodavg* (v. 2.1-0
254 – Mazerolle 2016) to compute the Akaike's Information Criterion for small sample
255 sizes (AICc). To test for significance of model terms we used the Anova function of
256 the *car* package (v. 2.1-3) with Type III Wald chi-square tests. Post-hoc comparisons
257 were performed with the *emmeans* function in the *emmeans* package (v. 1.4.4).

258

259 Lifetime reproductive success

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

263

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

270 *Survival analysis:*

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

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

289 We explored actuarial age-specific mortality rates using Weibull, Gompertz
290 and Logistic functions, with either a simple shape or one of the more complex
291 Makeham (Pletcher 1999) or bathtub (Siler 1979) shapes. The nine previous models
292 were compared to a model without senescence (exponential model with a simple
293 shape). The age-specific mortality curves were fitted starting from one year old, the

294 minimum age of reproduction (Supplementary Methods). We performed four parallel
 295 simulations that each model for 1 200 000 iterations, used a burn-in of 200 000
 296 iterations and took a model sample each 500 iterations. The model parameters likely
 297 converged on the most optimal mode and showed low serial autocorrelations (<5%)
 298 and robust posterior distributions (with N= 2 000). For the model comparison, we
 299 used the lowest deviance information criterion (DIC), which is similar to the Akaike
 300 Information Criterion (AIC) in non-Bayesian models. Comparison of the DIC values
 301 (Millar 2009) revealed that the Logistic model with the bathtub shape (Eq. 2) is the
 302 most appropriate mortality function (Supplementary Table S9). Mortality rate:

$$303 \quad \mu_0(x|\mathbf{b}, \mathbf{a}, c) = e^{a_0 - a_1 x} + c + \frac{e^{b_0 + b_1 x}}{1 + b_2 \frac{e^{b_0}}{b_1} (e^{b_1 x} - 1)} \quad (2)$$

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

314

315 To assess the differences among the posterior distributions of our brood size
 316 manipulation treatments we used the Kullback-Leibler divergence calibration (KLDC)
 317 (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).

324

325 **Results**

326 While the high-competition treatment decreased body mass at fledgling when
327 considering all fledglings (Voillemot *et al.* 2012, overall: 15.02 ± 0.09 , $\chi^2 = 36.35$, $P <$
328 0.01 , high-competition vs control: post hoc Tukey: $P = 0.015$; Fig. 2B), this effect
329 disappears when restricting the analysis to recruited females (overall: 15.12 ± 0.12 ,
330 $\chi^2 = 10.73$, $P < 0.01$, high-competition vs control: post hoc Tukey: $P = 0.989$, Fig 2A).

331

332 Overall, the quality of the natal environment had no significant effect on
333 lifetime reproduction ($\chi^2 = 0.99$, $P = 0.610$; Fig. 3C, Supplementary Table S2) or
334 individual fitness λ_{ind} ($\chi^2 = 1.07$, $P = 0.585$; Fig. 3D, Supplementary Table S3) in this
335 study. In terms of age-specific reproductive success, females from the low-
336 competition treatment had a high success during the early years of their life,
337 reaching a peak in their second year of breeding (Fig. 3A-B, Table 1). In contrast,
338 females raised in the high-competition treatment started with a lower reproductive
339 success, but steadily increased their performance reaching a peak in their third year
340 and enjoyed a later onset of senescence (Fig. 3A-B, Table 1). More specifically,
341 while the raw data suggest that individuals from high-competition nests maintained

342 high reproductive performance in their fifth year (Fig. 3A), taking into account
343 selective appearance (measured as age at first reproduction) and disappearance
344 (measured as age at last reproduction) reveals a reproductive decline after year four
345 (Fig. 3B). Nevertheless, high-competition females showed a delayed reproductive
346 peak and started to senesce later than their counterparts from low-competition nests
347 for whom senescence started a year earlier. Females raised in control nests started
348 at intermediate level of reproduction, maintained relatively steady reproductive
349 performance during the first three years of their life and declined at the fourth year.

350

351 Natal environment also had profound effects on age-specific mortality rates
352 (Fig. 4, see Table S11 for age specific survival probabilities and reproductive rates).
353 Low-competition females had substantially lower initial acceleration in mortality rate
354 (smaller early-life acceleration parameter b_1 with KLDC > 0.85 – Fig. 4, Fig. 5 and
355 Supplementary Table S10) than both control and high-competition females,
356 suggesting that “silver-spoon” natal environment is beneficial for survival. However,
357 while the mortality rates of control and high-competition females strongly decelerated
358 in late-life and reached a plateau around four years of age, the mortality rate of low-
359 competition females continued to increase and reached a higher plateau around the
360 fifth year (smaller late-life deceleration parameter b_2 with KLDC > 0.85 – Fig. 4, Fig.
361 5 and Supplementary Table S10).

362

363 **Discussion**

364 We found that females experiencing a low-competition natal environment fledge at
365 higher body mass, start to reproduce at a higher rate, have an earlier reproductive

366 peak and have a lower acceleration of mortality rate than their counterparts raised in
367 high-competition nests. However, low-competition females also show earlier signs of
368 reproductive senescence and suffer from increased mortality rate in the last years of
369 their lives. Thus, our results provide an experimental demonstration of condition-
370 dependent ageing in a natural population. Specifically, these findings support the
371 hypothesis that high-condition females raised in “silver-spoon” natal environment
372 allocate their resources to early-life reproduction, but pay a cost in terms of
373 accelerated ageing in late-life.

374

375 There has been a sustained interest in how resource availability affects age-
376 specific life histories but, remarkably, both theoretical and empirical studies to date
377 are focused exclusively on the role of sexual selection in driving rapid ageing in high-
378 condition males (i.e. “live-fast die-young” hypothesis) (Kokko 1998; Radwan &
379 Bogacz 2000; Hunt *et al.* 2004; Bonduriansky & Brassil 2005; Bonduriansky *et al.*
380 2008; Preston *et al.* 2011; Adler *et al.* 2016; Hooper *et al.* 2017, 2018). This is, in
381 part, because of the striking absence of studies showing that high-condition females
382 may also trade-off early-life reproduction for accelerated ageing (but see Travers *et*
383 *al.* 2015). Here, we used an experimental manipulation of early-life environment to
384 show that “silver-spoon” (i.e. low-competition) females indeed benefit from elevated
385 early-life reproductive success, but suffer from an earlier onset of reproductive
386 ageing and increased mortality late in life. Thus, our findings call for a re-appraisal of
387 the idea that “live-fast die-young” strategy applies primarily to males when faced with
388 increased sexual competition (Bonduriansky *et al.* 2008; Hooper *et al.* 2018).

389 Furthermore, our results suggest, that we should look beyond a simple
390 negative correlation between net reproduction and longevity when investigating the
391 long-term fitness consequences of early-life conditions. Specifically, in this study
392 “silver-spoon” females did not live shorter, nor reproduced more, but rather had
393 different age-specific mortality and reproduction rates. It is possible that stochastic
394 natural environment masks the effect of our brood size manipulation on total
395 reproduction of the recruits and that the addition of several more years of data
396 collection could show that high-condition females actually have higher fitness. At this
397 point, however, we can only state that a “silver-spoon” early-life environment results
398 in increased early-life but reduced late-life performance. High-competition females
399 fledge at lower body mass than low-competition birds and the control birds but those
400 that survive to come back and reproduce have the same body mass as control birds.
401 This suggests that high-competition treatment resulted in increased mortality of birds
402 that had low body mass as fledglings.

403

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

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420

421 **Competing Interests statement**

422 The authors declare no competing interests.

423

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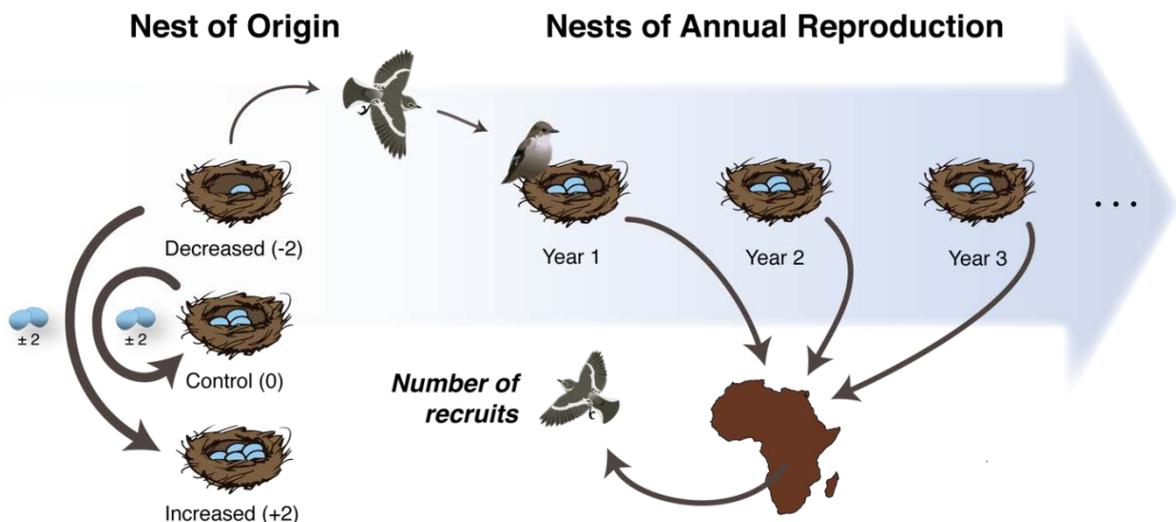
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591 **Figure 1.** The graphical outline of the experimental design. The experimental brood size
592 manipulation of the nest of origin is illustrated on the left and the age-specific reproductive
593 data (i.e. number of recruits) collected from the nests of annual reproduction are illustrated
594 on the right.

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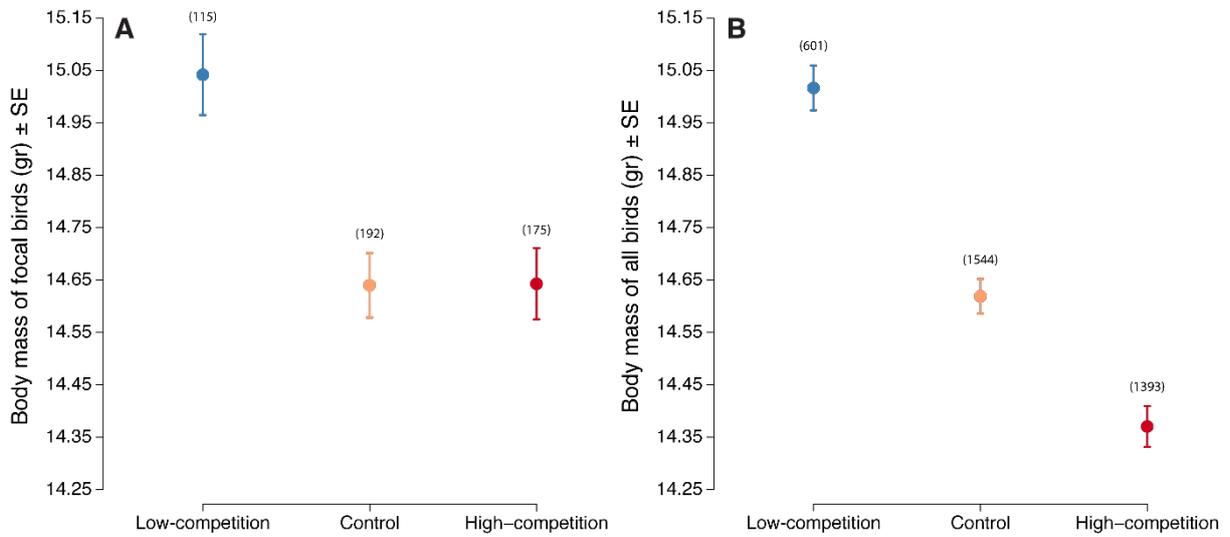


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599 **Figure 2.** A) Body mass at fledgling (means \pm SE) of the focal birds raised in the three
600 manipulated natal environments. B) Body mass at fledgling (means \pm SE) of all the birds
601 raised in the three manipulated natal environments. Blue lines represent the increased low-
602 competition treatment, orange lines represent the control treatment and red lines represent
603 the decreased high-competition treatment.

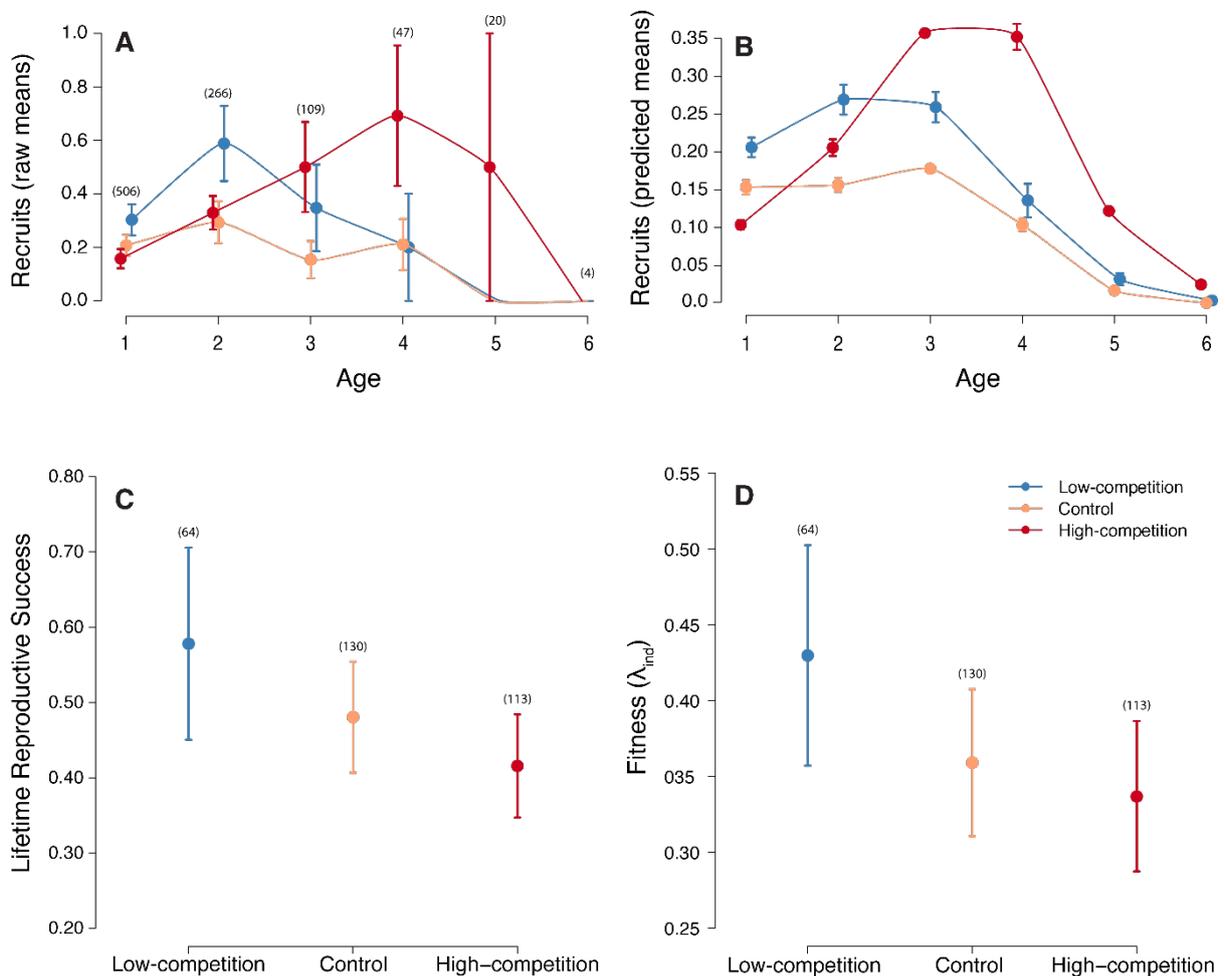
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610 **Figure 3.** Reproduction of the three manipulated natal environments (Means \pm SE). A) Age-
 611 specific reproduction from the raw data. The sample sizes per age class are indicated with
 612 numbers in parentheses (Table S1); B) Age-specific reproduction from the model predictions
 613 based on all random and fixed effects included in model 17 (Table 1); C) Lifetime
 614 Reproductive Success; D) Individual fitness (λ_{ind}). Blue lines represent the decreased low-
 615 competition treatment, orange lines represent the control treatment and red lines represent
 616 the increased high-competition treatment.

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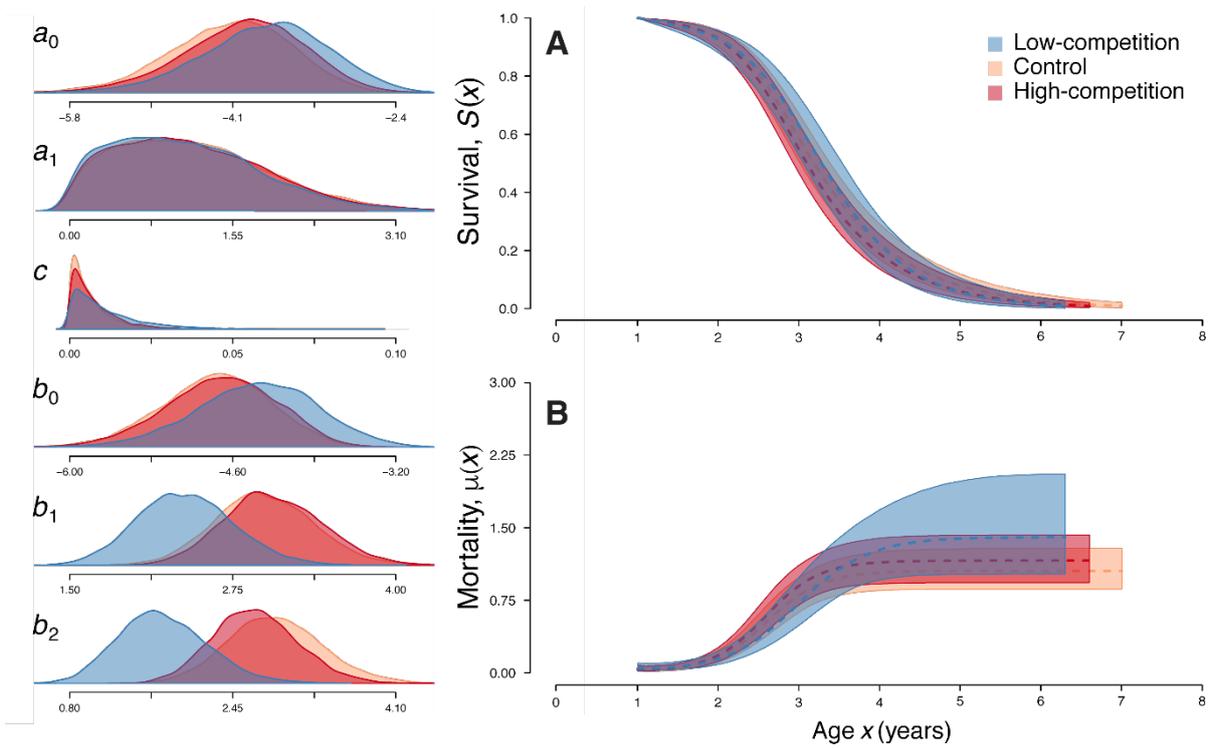
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620 **Figure 4.** Survival (A) and Mortality (B) curves with 95% confidence intervals for each
621 treatment as fitted by the bathtub Logistic mortality model. On the left are the posterior
622 distributions of the six model parameters (see Methods for details). Blue lines and
623 distributions represent the low-competition treatment, orange represent the control treatment
624 and red represent the high-competition treatment.

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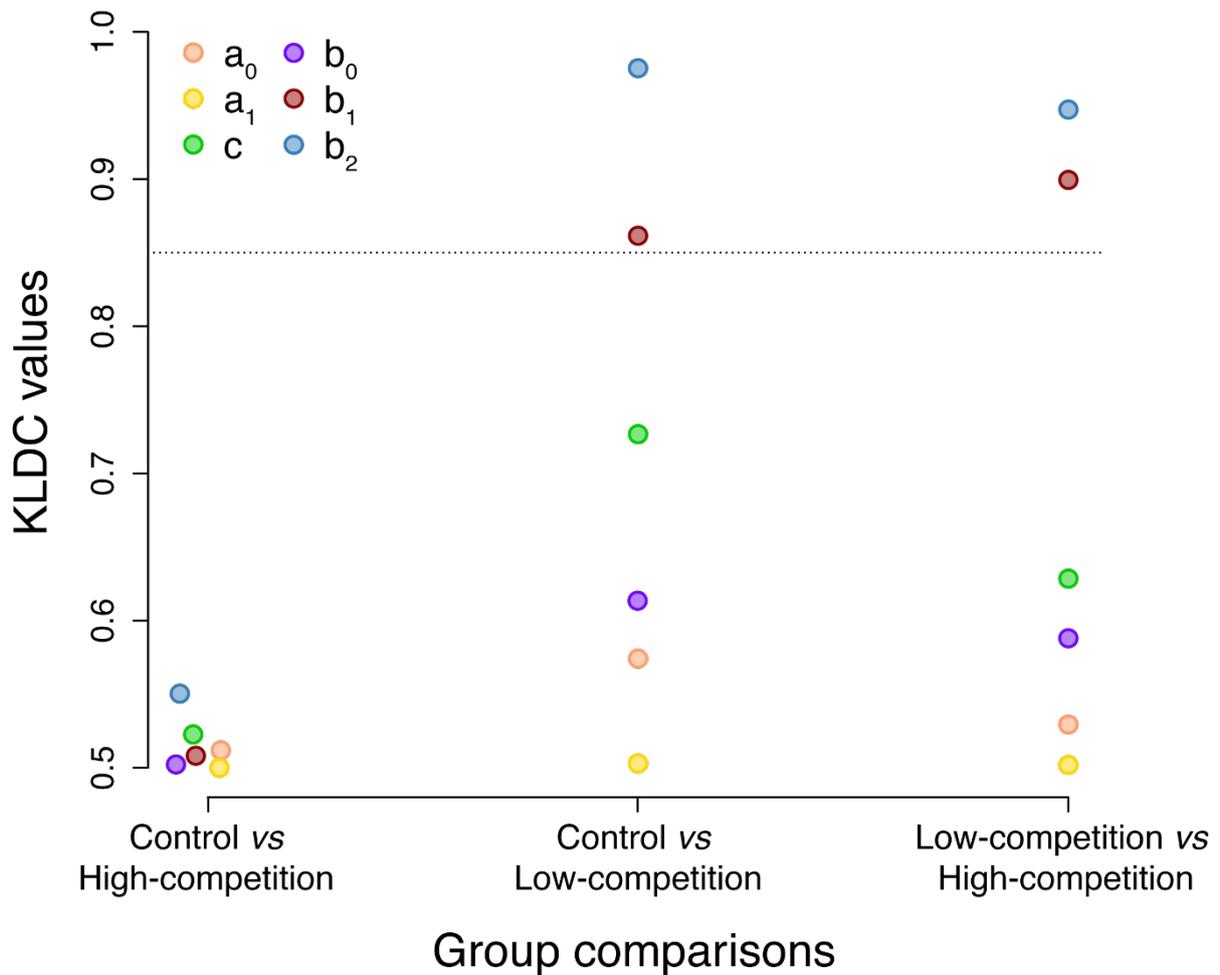


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629 **Figure 5.** Values of the Kullback-Leibler divergence calibration (KLDC), which compare
 630 parameter posterior distributions of the bathtub Logistic mortality model, between the high-
 631 competition, the control and the low-competition treatments. The values that are above the
 632 dotted line (KLDC = 0.85) show parameters that are substantially different between the two
 633 treatments in each group comparison. The parameters a_0 and a_1 describe the exponential
 634 decline that may be observed very early in life and c describe the lowest point of the
 635 mortality function. b_0 , b_1 and b_2 describe different parts of the logistic increase in mortality
 636 rates with age (see Methods for details).

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641 **Table 1.** Generalized linear mixed effects model examining the effect of early life environmental
 642 conditions on age-specific reproduction in 434 females ($N_{\text{obs}} = 781$). The reference level for
 643 treatment is “Low-competition”. Significant effects are in bold.

Variable	Estimate ± S.E.	Z	P-value (Z)	χ^2	df	P-value (χ^2)	Variance
<i>Model 17</i>							
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	
Age²	-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

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