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HAL Authorization

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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30 conditions, senescence, “silver-spoon” theory, brood size manipulation  
31

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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 =  $6.3 \pm 0.65$ ) 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  $4.33 \pm 0.05$ , for enlarged  
154 broods was  $8.31 \pm 0.04$  and for control broods was  $6.18 \pm 0.05$ . 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 ( $15.12 \pm 0.12$ ,  $\chi^2 = 10.69$ ,  $P < 0.01$ , Fig. 2A), while there was no effect of  
161 body size measured as tarsus length ( $19.56 \pm 0.05$ ,  $\chi^2 = 0.52$ ,  $P = 0.77$ ,  
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  $\lambda_{\text{ind}}$ , 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 ( $\lambda_{\text{ind}}$ ), 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 Age<sup>2</sup>), 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  $\varepsilon_{ij}$ .

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  $\lambda_{\text{ind}}$   
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  $\lambda_{\text{ind}}$  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.*

318 2014; Atema *et al.* 2016; Rodríguez-Muñoz *et al.* 2019), which show the mean  
319 Kullback-Leibler discrepancies (KL) between two distributions. Values closer to 0.5  
320 imply that there is minimal difference among the distributions and values closer to 1  
321 imply major differences (Kullback & Leibler 1951; McCulloch 1989). We considered a  
322 KLDC value > 0.85 to indicate a substantial difference between the two posterior  
323 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:  $15.02 \pm 0.09$ ,  $\chi^2 = 36.35$ ,  $P <$   
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:  $15.12 \pm 0.12$ ,  
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  $\lambda_{\text{ind}}$  ( $\chi^2 = 1.07$ ,  $P = 0.585$ ; 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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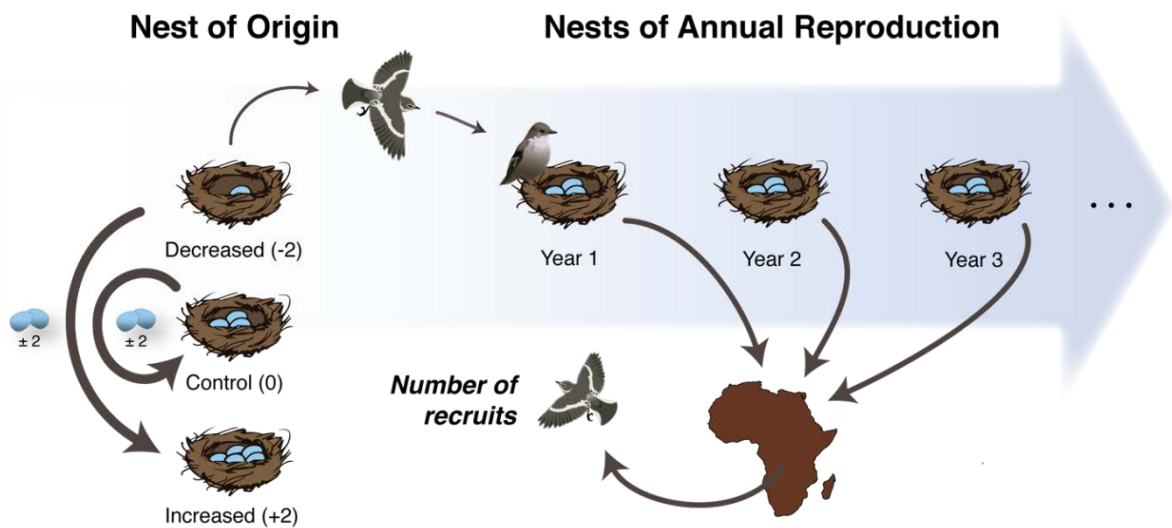
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591 **Figure 1.** The graphical outline of the experimental design. The experimental brood size  
592 manipulation of the nest of origin is illustrated on the left and the age-specific reproductive  
593 data (i.e. number of recruits) collected from the nests of annual reproduction are illustrated  
594 on the right.

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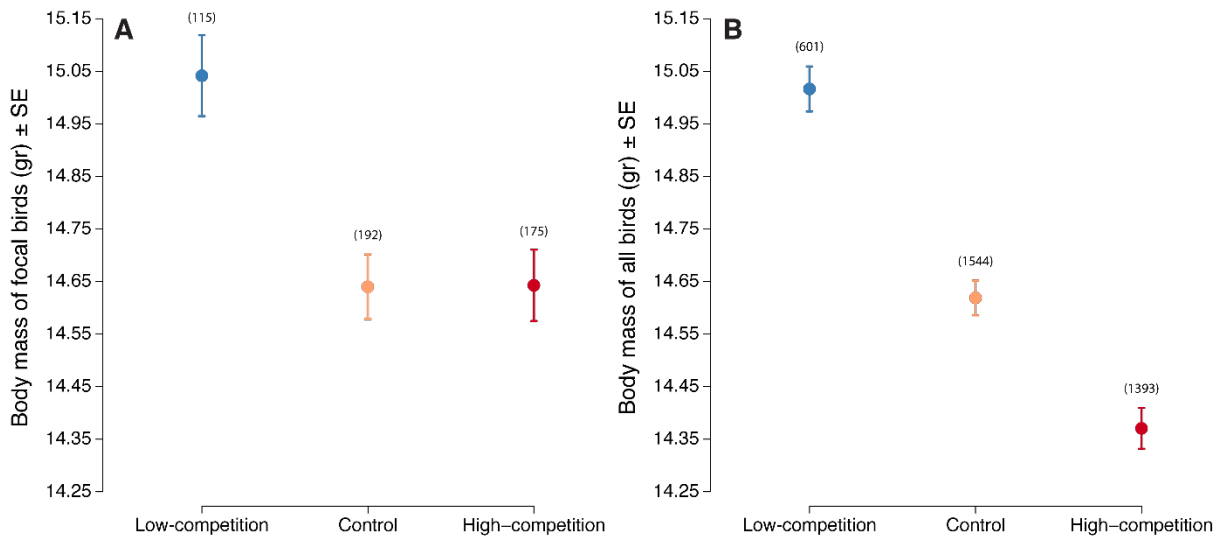


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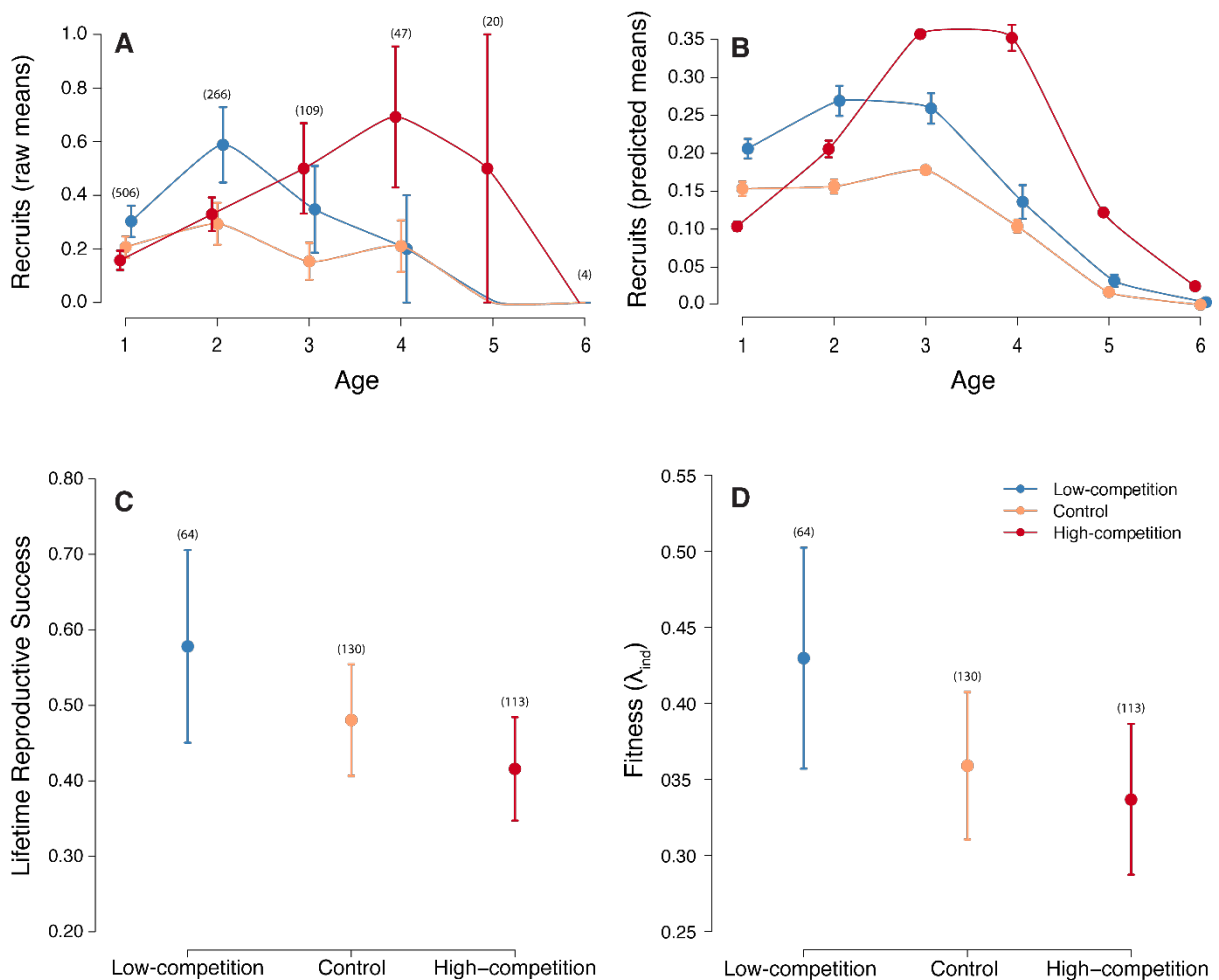
599 **Figure 2.** A) Body mass at fledgling (means  $\pm$  SE) of the focal birds raised in the three  
600 manipulated natal environments. B) Body mass at fledgling (means  $\pm$  SE) of all the birds  
601 raised in the three manipulated natal environments. Blue lines represent the increased low-  
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 ( $\lambda_{ind}$ ). 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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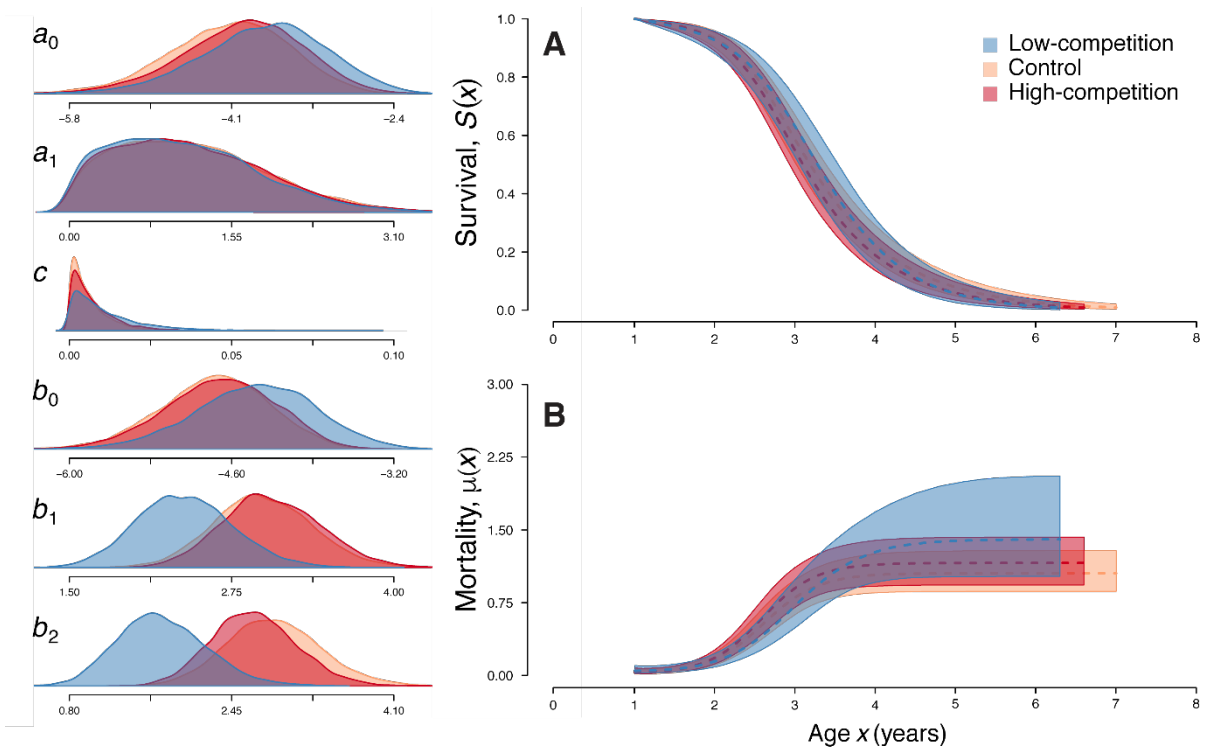
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620 **Figure 4.** Survival (A) and Mortality (B) curves with 95% confidence intervals for each  
 621 treatment as fitted by the bathtub Logistic mortality model. On the left are the posterior  
 622 distributions of the six model parameters (see Methods 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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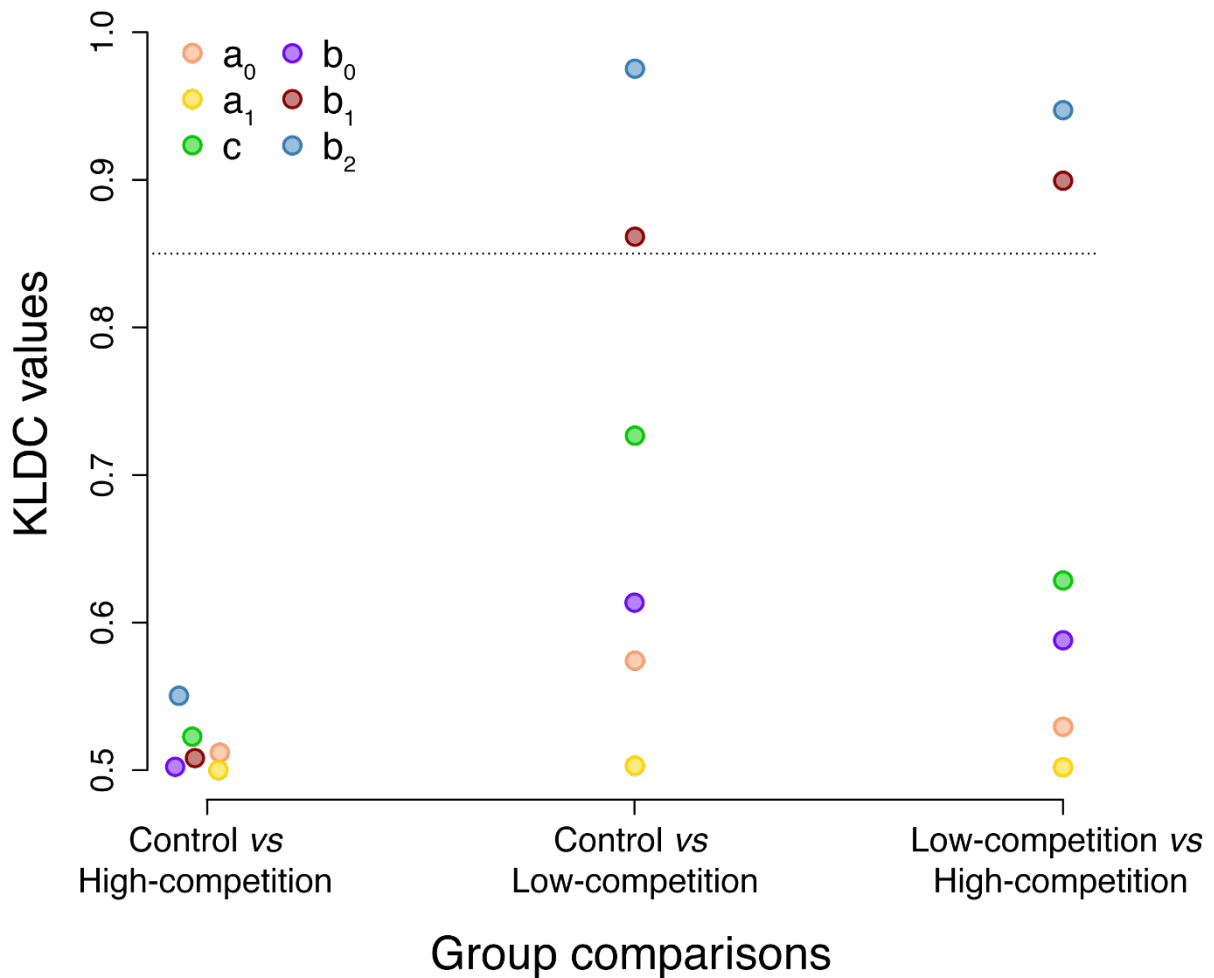


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629 **Figure 5.** Values of the Kullback-Leibler divergence calibration (KLDC), which compare  
 630 parameter posterior distributions of the bathtub Logistic mortality model, between the high-  
 631 competition, the control and the low-competition 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)	$\chi^2$	df	P-value ( $\chi^2$ )	Variance
<i>Model 17</i>							
<b>Intercept</b>	<b>-1.69 ± 0.26</b>	<b>-6.43</b>	<b>&lt; 0.001</b>	<b>41.3</b>	<b>1</b>	<b>&lt; 0.001</b>	
<b>Age</b>	<b>0.66 ± 0.27</b>	<b>2.49</b>	<b>0.012</b>	<b>6.20</b>	<b>1</b>	<b>0.013</b>	
<b>Age<sup>2</sup></b>	<b>-0.47 ± 0.13</b>	<b>-3.68</b>	<b>&lt; 0.001</b>	<b>13.52</b>	<b>1</b>	<b>&lt; 0.001</b>	
<b>Age at First Repr. (AFR)</b>	<b>-0.89 ± 0.17</b>	<b>-5.34</b>	<b>&lt; 0.001</b>	<b>28.49</b>	<b>1</b>	<b>&lt; 0.001</b>	
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				
<b>Interaction (Age × AFR)</b>	<b>0.89 ± 0.24</b>	<b>3.80</b>	<b>&lt; 0.001</b>	<b>14.46</b>	<b>1</b>	<b>&lt; 0.001</b>	
Interaction (Age <sup>2</sup> × AFR)	-0.19 ± 0.10	-1.84	0.065	3.40	1	0.065	
<b>Interaction (Age × Trt)</b>				<b>7.83</b>	<b>2</b>	<b>0.019</b>	
Age × Control Trt	0.23 ± 0.28	0.80	0.421				
<b>Age × High-competition Trt</b>	<b>0.74 ± 0.28</b>	<b>2.67</b>	<b>0.008</b>				
Interaction (ALR × Trt)				5.10	2	0.078	
<b>ALR × Control Trt</b>	<b>-0.54 ± 0.26</b>	<b>-2.08</b>	<b>0.037</b>				
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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