Parental age at reproduction accelerates offspring pace of life in *Gryllus bimaculatus*.

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Key words: senescence; life history; parental effects; slow-fast continuum, phenotypic plasticity; adaptation; constraint; selective disappearance; viability selection; trade-offs

Open research statement

Data and code are not yet provided but will be made openly and freely available on request during the review process. Data will be uploaded to the Dryad data repository and code will be uploaded to Zenodo open repository on acceptance of the manuscript.

Abstract

Across invertebrates, it is widely known that older parents produce offspring with abbreviated lifespans (i.e., the Lansing effect). Yet the ecological and evolutionary ramifications of such nongenetic parental age effects remain unclear. Two demographic processes could culminate in a Lansing effect, with different fitness implications: (i) Later-conceived offspring may display greater mortality from conception, constraining fitness from birth. (ii) Alternatively, they may exhibit an increased age-specific mortality (i.e., accelerated senescence), with negative effects that manifest after maturity. If parental age accelerates offspring senescence without affecting initial mortality rate, then later-conceived offspring may maintain fitness by shifting to a faster pace-of-life. We exposed *Gryllus bimaculatus* parents to one of three experimental temperatures to generate environmental variation in parent mortality. Using a longitudinal within-parent study, we tested whether parental age influenced offspring longevity via increased initial mortality or accelerated senescence, and whether these effects depended on the parents' thermal environment. Contrary to our predictions, we found no evidence that parental age reduced offspring longevity. Instead, parental age mediated offspring pace-of-life. Irrespective of the parents' thermal environment, the earliest- and latest-conceived offspring were demographically distinct. Later-conceived offspring exhibited accelerated development, a smaller adult mass, and improved reproductive success. Unexpectedly, they also showed extended adult lifespans. This increased adult lifespan did not arise from reduced initial mortality, but from a delayed onset and slower progression of actuarial senescence. The slowed senescence in later-conceived offspring is unlikely to reflect a direct parental age effect, potentially arising indirectly through causal effects on adult size. Our results challenge the universality of the Lansing effect and contribute to a growing recognition that parental age has important life-history consequences. Accelerated life-histories in later-conceived offspring may be adaptive, enabling these individuals to compete over dwindling seasonal resources or overcome seasonal time constraints. Determining whether these demographic parental age effects accumulate across generations, or persist under natural environments, will be essential for establishing their ecological consequences and proximate role in the evolution of ageing.

Introduction

Classical theories of ageing have implicitly assumed that senescence, the progressive increase to mortality and reduction in reproduction and somatic maintenance with increasing chronological age, is an exclusively within-individual phenomenon (Hamilton, 1966; Abrams, 1991; Monaghan et al. 2008; Nussey et al. 2013). Here, senescent damage is restricted to the soma, while the germline remains protected, or passes through rejuvenating mechanisms, that prevent the unregulated accumulation of senescent damage across generations (Kirkwood & Holliday, 1979; Rose, 1984; Kern et al. 2001; Monaghan & Metcalfe, 2019). Yet growing evidence suggests senescence can be transmitted intergenerationally to have penetrant detrimental effects on offspring quality (i.e., the Lansing effect or parental effect senescence; Lansing, 1947; Monaghan et al. 2020). It is crucial to understand the importance of these nongenetic parental age effects in both the evolution of senescence and species life-histories, as their adaptive role has previously been understated (Moorad & Nussey, 2016; Van Den Heuvel et al. 2016; Barks & Laird, 2020; Hernández et al. 2020).

Although evidence for negative effects of parental age at reproduction on offspring traits, hereafter 'parental effect senescence, continues to accumulate from both captive and natural populations (see: Kern et al. 2001; Priest et al. 2002; Schroeder et al. 2015; Lippens et al. 2017; Noguera et al. 2018; Monoghan et al. 2020; Reichert et al. 2020; Noguera et al. 2021; Angell et al. 2022), there is limited agreement over its universality and the underlying mechanisms involved (King, 1983; Anderson et al. 2022; Ivimey-Cook et al. 2023). Some of the earliest evidence for parental effect senescence comes from Lansing (1947), who demonstrated that old-aged lines of the

76 parthenogenic rotifer *Philodina citrina* produced offspring with abbreviated lifespans
77 (i.e., the Lansing effect), with this effect propagating across generations. More
78 recently, a meta-analysis investigating the general tendency for the Lansing effect by
79 Ivemy-Cook et al. (2023) demonstrated that a one-unit increase in maternal age was
80 associated with a 17 to 22% reduction in the offspring's adult lifespan, on average.
81 Alongside adult longevity, detrimental parental age effects have also been
82 demonstrated on embryo viability (Bloch Qazi et al. 2017; Noguera, 2021), juvenile
83 survival (Kern et al. 2001; Reid et al. 2010; Torres et al. 2011; Ivimey-Cook & Moorad,
84 2020), development and growth rate (Fox & Dingle, 1994; Singh & Omkar, 2009; Lind
85 et al. 2015), size at birth and maturity (Opit & Throne, 2007; Vega-Trejo et al. 2018),
86 and reproductive performance (Hercus & Hoffmann, 2000; Schroeder et al. 2015; Van
87 Daalen et al. 2022). However, there is a growing uncertainty over the ubiquity of this
88 phenomenon; studies frequently find conflicting results, even between laboratory
89 strains (Priest et al. 2002; Anderson et al. 2022), including neutral (Ivimey-Cook &
90 Moorad, 2018; Travers et al. 2021) or positive parental age effects on offspring
91 longevity (an "inverse Lansing effect"; Marshall et al. 2010; Anderson et al. 2022).
92 This lack of consistency raises challenges in identifying the physiological pathways,
93 biological factors, and environments that are important for parental effect
94 senescence (Priest et al. 2002; Monaghan et al. 2020).

95 Despite an extensive history of research, numerous issues plague the current study
96 designs, which could perturb, or spuriously create, the observed parental age effects.
97 First, most studies remain cross-sectional (see: Priest et al. 2002; Plaistow et al.
98 2015; Bloch Qazi et al. 2017; Bock et al. 2019; Wylde et al. 2019; Angell et al. 2022),

99 and cannot control for the differential survival of parental phenotypes (i.e., selective
100 disappearance: Nussey et al. 2008; Reid et al. 2010; Ivimey-Cook & Moorad, 2018;
101 Marasco et al. 2025). Second, studies into parental age effects are often
102 correlational, and there is a growing necessity for experimental manipulations of
103 parent longevity (both genetic and environmental) to tease apart the causal effects of
104 parental senescence on offspring performance (Kern et al. 2001; Bock et al. 2019).
105 Previous work has demonstrated that parental effect senescence can exhibit
106 genotypic heterogeneity, differing between strains artificially selected for early- or
107 late-life performance (Kern et al. 2001; Priest et al. 2002; Anderson et al. 2022).
108 However, there exists limited research on whether environmental variation in parent
109 mortality could also modulate the expression of parental age effects, which could
110 have important implications for understanding the intergenerational impacts of health
111 and lifestyle. Evidence suggests that adverse parental age effects may be context-
112 dependent, being influenced by the parents' diet and population density, though the
113 proximate role of parents' thermal environment has received little attention, despite
114 the known importance of temperature in modulating the lifespan of ectothermic
115 organisms (Fox & Dingle, 1994; Plaistow & Benton, 2009; Gribble et al. 2014; Burraco
116 et al. 2020; Morimoto, 2022). The role of temperature in dictating longevity in
117 ectothermic insects is complex, with chronic exposure to warm temperatures either
118 raising baseline mortality (i.e., temperature induced frailty) or accelerating
119 senescence, which may have different implications for offspring fitness (Mair et al.
120 2003; Burraco et al. 2020; Ma et al. 2021. Third, few have considered how the
121 variability of offspring phenotypes changes with parental age. It could be that

122 parental effect senescence not only governs the offspring's average trait value, but
123 also generates greater variation in offspring phenotypes, as has been similarly
124 observed for other parental environmental stressors (e.g., increasing temperature; De
125 Jong, 1995; O'Dea et al. 2016). Any change in the variability of offspring phenotypes
126 may be adaptive (i.e., bet-hedging; Crean & Marshall, 2009) or reflect physiological
127 constraints (Debat & David, 2001; Olsson & Uller, 2002), such as the senescent
128 instability of the parents' reproductive systems (Monaghan & Metcalfe, 2019). Finally,
129 despite the evolutionary importance of parental age effects in understanding
130 senescence, most studies have tested for their isolated effects on offspring longevity –
131 the end point of senescence (Promislow et al. 2022). However, this precludes any
132 effects of parental age on offspring age-specific mortality (i.e., *how* the offspring
133 senesce) and reproductive performance. Assessing the joint effects of parental age on
134 offspring mortality and reproduction is a necessary step in establishing the fitness,
135 demographic, and evolutionary consequences of parental effect senescence
136 (Kirkpatrick & Lande, 1989; Nussey et al., 2013; Bloch Qazi et al. 2017; Monaghan &
137 Metcalfe, 2019).

138 Monaghan et al. (2020) identified that increasing parental age could reduce offspring
139 longevity through two non-exclusive demographic processes. First, offspring from old-
140 age parents may be conceived with an increased frailty. Here, their mortality is
141 higher from conception as they initiate life with either the senescent damage (e.g.,
142 shortened telomeres, increased point mutation rates, abnormal DNA methylation
143 patterns, heightened mitochondrial dysfunction) accumulated by their aged parents
144 (Díaz & Esponda, 2004; Kong et al. 2012; Monaghan & Metcalfe, 2019; Dutt & Laird,

145 2024), or experience age-dependent reductions in either pre- or post-natal parental
146 provisioning (Bouwhuis et al. 2010; Lemaître & Gaillard, 2017; Muller et al. 2017;
147 Ivimey-Cook & Moorad, 2018). Here, this increased frailty persists, but does not
148 necessarily worsen, as the offspring age. Alternatively, offspring from old-aged
149 parents may exhibit increased age-specific mortality (i.e., accelerated senescence),
150 with the negative effects of their accumulated senescent damage only manifesting
151 after maturity, being buffered against during the developmental period (Monaghan et
152 al. 2020). Despite the breadth of studies demonstrating parental effect senescence,
153 few have assessed its demographic underpinnings. Indeed, those that have reported
154 conflicting results, with either increased frailty (Dutt & Laird, 2024), accelerated
155 senescence (Plaistow et al. 2015) or both (Bock et al. 2019), being implicated in
156 reducing offspring longevity. Although both demographic processes reduce survival,
157 this may not necessarily culminate in reducing offspring fitness. Rather, the two
158 processes can have different consequences for offspring life-histories. If offspring
159 exhibit increased frailty, then their fitness is likely constrained from birth.
160 Conversely, if parental age accelerates offspring senescence, but has no effect on
161 frailty, then offspring may undertake a phenotypically plastic switch to a fast pace-of-
162 life strategy. In this latter scenario, offspring may accelerate development to reach
163 reproductive maturity earlier in life, increasing investment in their early life fitness
164 contributions prior to their hastened onset of senescence (King, 1983; Bouwhuis et al.
165 2010; Plaistow et al. 2015; Monaghan et al. 2020). Thus, although increasing parental
166 age may have no effect on offspring fitness, the offspring produced at the extreme

167 ends of the parent's life could be demographically distinct (Van Noordwijk & De Jong,
168 1986; Travers et al. 2021; Anderson et al. 2022).

169 Using the two-spotted cricket (*Gryllus bimaculatus*), and an experimental,
170 longitudinal within-individual study design, we investigated how parental age affected
171 offspring mortality trajectories and life-history traits. *G.bimaculatus* has previously
172 been shown to display negative effects of parental age on offspring traits, including
173 longevity. However, this was with a cross-sectional study in which group housed
174 offspring of old and young parents were compared, rather than through any direct
175 impact of *within* parent senescence on individually housed offspring unaffected by
176 group competition effects (Noguera, 2021). In this study, we first addressed a key
177 conceptual issue: does experimentally induced environmental heterogeneity in parent
178 mortality mediate the expression of parental effect senescence? To this end, we
179 manipulated the temperature parents experienced over their adult lifespan, creating
180 variation in parent longevity. We then investigated whether parental age effects
181 manifested differently depending on the parents' temperature treatment, predicting
182 that any adverse parental age effects would be exacerbated under a warm
183 environment. Then, we aimed to disentangle which demographic process (frailty or
184 actuarial senescence) was implicated in reducing offspring longevity. Here, we
185 confine our inferences of senescence to the offspring's adult, rather than total,
186 lifespan. Adult eclosion marks the initiation point from which senescence progresses
187 in *Gryllus spp.*, when the individual has shifted resources from growth to reproduction
188 (Rodríguez-Muñoz et al. 2019). After determining how parental effect senescence
189 arises, we asked whether the following life-history traits were affected by increasing

190 parental age, in line with an expected switch to a fast pace-of-life strategy: post-
191 natal development time, adult size, and offspring reproductive performance. Finally,
192 for these listed traits, not only did we estimate the effect of parental age on their
193 average value, but we also aimed to establish how their variability changed over the
194 parents' lifespan.

195 Methods

196 2.1 Laboratory conditions

197 We conducted the study outlined below from September 2023 to September 2024,
198 using an outbred (<8 generation descendants from the wild) laboratory-reared
199 population of *G.bimaculatus*, located at the Scottish Centre for Ecology and the
200 Natural Environment (SCENE), University of Glasgow. All animals were individually
201 housed in plastic containers (75H × 125W × 185Lmm for the parent generation,
202 75H × 108W × 108Lmm for the offspring generation), containing *ad-libitum* rabbit
203 pellets (Supreme Petfoods Science Selective Adult Rabbit food) a cotton-soaked water
204 tube, and an egg carton for shelter. We individually housed individuals to remove any
205 effects that intra-specific competition may have on life-history traits (Simmons,
206 1987). Individuals were kept in incubators set to a 14:10 hour day: night cycle with a
207 relative humidity (RH) of ~50%. We continuously monitored temperature and relative
208 humidity through the experimental period with loggers (Elitech RC 5+, RS-191A
209 Temperature Loggers + DOOMAY mini hygrometer). We rotated all individuals amongst
210 the incubators weekly, ensuring animals were alternated between incubators and
211 shelve locations.

212 ## 2.2 Creating the parent generation

213 Colony animals (12 pairs) were mated opportunistically, avoiding pairing related
214 animals, to minimise inbreeding. Eggs were collected ad-hoc from the oviposition
215 substrate (damp cotton) before being plated and incubated in a climate room for nine
216 days at 28.5°C (14:10 hour day: night cycle; RH of 40-60%), after which we initiated
217 daily hatch checks. Once hatched, the parent generation was housed individually in
218 incubators (set temperature 25.0-26.5°C; *equally experienced by all offspring*). From
219 January 2024, we monitored late-stage instars daily to record the exact date of adult
220 eclosion. As adults can be damaged shortly after eclosing, we waited at least 24 hours
221 after observed emergence to measure their mass using a VWR SMG4i precision balance
222 (readability ±0.01mg). We photographed all individuals (Panasonic Lumix DC-FZ82
223 camera + Raynox DCR-250 macro lens (focal length: 60mm) + set to *"autofocus"*
224 mode) to measure pronotum width/length (mm). To prevent individuals from moving
225 during photography, we placed them in a petri dish filled with cotton (100mm x
226 15mm), with a scale attached. We measured pronotum width and length (in mm) from
227 the images with the line tool in IMAGEJ (Schneider et al. 2012). Images were analysed
228 by a single observer (Mark D. Pitt; MDP) to eliminate any between-observer
229 measurement bias. Pronotum width and length are assumed to be more fixed
230 indicators of structural size than adult mass, as daily variation in hydration, satiation,
231 and oviposition can cause mass to have low within-individual repeatability (Gershman
232 et al. 2022). However, we observed reasonably tight correlations between all three
233 measures of adult body size (pronotum width ~ pronotum length, Pearson's correlation
234 coefficient r [95% CI] = 0.856 [0.760, 0.916]; pronotum width ~ adult body mass,

235 Pearson's correlation coefficient *r* [95% CI] = 0.758 [0.610, 0.855]; pronotum length ~
236 adult body mass, Pearson's correlation coefficient *r* [95% CI] = 0.808 [0.685, 0.886],
237 using a subset of photographs, N = 51 animals). Therefore, we selected adult body
238 mass as our only proxy of adult size, assuming this measurement captured relevant
239 biological variation. Survival for the parent generation was monitored weekly, at
240 which point we also undertook husbandry (e.g. replaced food, water, and cleaned
241 housing).

242 2.3 Parent mating design

243 We paired parent animals pseudo-randomly, such that inbreeding was minimised, and,
244 where possible, members of a pair were age matched. Most parents were mated in
245 their second week of adult life, with an average adult age of 1.4 ± 0.67 weeks (mean
246 ± SD) at first mating. This allowed time for the oocytes in the females to mature,
247 which usually occurs in the initial 10 days post adult eclosion (Lorenz, 2007). During
248 the staged matings, the parent pair were placed into a plastic container
249 (75H × 125W × 185Lmm) and allowed to interact for 15 minutes. Pairs were watched
250 continuously, and mating was assumed to be successful after visual confirmation of
251 spermatophore transfer. We then immediately assigned pairs to be continuously
252 maintained under one of the three temperature treatments (25.5°C, 28.0°C, or
253 30.5°C). Previous experimental work has revealed that the survival of *Gryllus spp.* is
254 sensitive to experimental temperature manipulations, both constant and fluctuating,
255 over their adult lifespan (Centeno Filho et al. 2023). Additionally, we have
256 demonstrated that the strength of reproductive senescence is temperature dependent
257 in *G.bimaculatus*, with females exposed to the highest temperatures exhibiting the

258 steepest age-dependent reductions to fecundity and hatching success (*in prep*). Our
259 temperature manipulations successfully created experimental variation in parent
260 longevity, with the Kaplan-Meier curve demonstrating that parents experiencing the
261 warmer temperature treatments exhibited reduced survival relative those maintained
262 at 25.5°C (*Appendix S1: Section S1)*.

263 All parent animals were housed individually between mating attempts. We mated
264 pairs weekly, with offspring being collected and raised from the parents first eight
265 mating attempts. At the last mating from which offspring were housed, parents had
266 an average adult age of 8.52 ± 0.80 weeks (mean ± SD). The parents were repeatedly
267 mated with the same partner to reduce the likelihood of stored sperm senescence
268 confounding our results (Ribou & Reinhardt, 2012; Monaghan et al. 2020). We did not
269 directly observe any subsequent mating attempts after the first mating attempt.
270 Instead, pairs were housed together for three hours, before being separated. For four
271 pairs, we observed that one member of the pair had died after the staged mating,
272 though we were unable to identify if this was a natural death or cannibalism. Not all
273 parents produced hatchlings from all eight mating attempts, with some pairs ceasing
274 to produce viable eggs earlier than others. Due to capacity constraints, parents were
275 removed from their temperature treatment to a climate room (28.5°C; 14:10 hour
276 day: night cycle; RH of 40-60%) following three consecutive mating attempts that
277 resulted in no viable hatchlings (*Appendix S1: Section S1*). Our final sample size
278 consisted of 78 pairs (156 animals), with these representing animals that produced
279 viable eggs from their first mating attempt onwards. The sample sizes for each

temperature treatment were as follows: 27 pairs at 25.5°C, 23 pairs at 28°C, and 28 pairs at 30.5°C.

2.4 Creating the offspring generation

After each staged mating, we allowed females 15 hours to lay, providing them with a petri dish (60mm x 13mm) of sand (Children's Play Sand), covered with a damp cotton pad for laying. As laying effort may vary daily, we provided females with sand for three days consecutively. Where possible, we only collected and incubated eggs from the first and third batches of eggs. All eggs were photographed before being incubated on a petri dish (100mmx 15mm; filled with damp cotton) in the climate room. Eggs were inspected daily and, if hatchlings were observed, they were photographed (Panasonic Lumix DC-FZ82 camera, focal length: 35-40mm; *for another study*) as a group before being housed individually (*details below*). We defined a successful hatching as any nymph that had completely removed itself from its egg casing. Hatching was asynchronous, and if there were still unhatched eggs present, we returned the remaining eggs to the climate room. Hatching checks continued daily until all eggs on a dish had hatched or up to 14-days after first incubation, after which remaining eggs were assumed to be non-viable.

From each mating attempt, we individually housed up to three offspring per parent pair. Offspring were inspected weekly for any adult emergence or mortality. As per the parent generation, we waited two days after adult emergence to measure adult mass. Overall, our sample size consists of 1317 individually housed offspring from 78 parent pairs, while complete life-history data was collected from 1309 offspring. Eight

302 offspring had uncertainty surrounding their adult emergence dates and were excluded
303 from analyses on offspring development time, adult lifespan, and adult mass. A
304 further two animals had missing mass measurements and were thus removed from any
305 size analysis.

306 2.5 Offspring reproductive performance

307 Once the offspring generation reached adulthood, we mated a subset of individuals to
308 determine how parental age affected offspring reproductive success. We collected
309 and incubated offspring eggs from a single mating, at which offspring had a mean
310 adult age of 4.60 ± 1.44 weeks (mean ± SD), assuming this window represented an
311 intermediate adult age (e.g., neither young nor old). To increase the diversity of
312 offspring that could be paired, we binned parental age into two-levels, producing a
313 subset of individuals from either "early" or "late" aged parents. Offspring from the
314 "early" cohort descended from the first two parental mating attempts, while the
315 "late" cohort of offspring descended from parental mating attempts five through to
316 eight. We mated the offspring within the levels of parental age and parental
317 temperature. As per the parent generation, we paired offspring opportunistically,
318 minimising inbreeding.

319 We performed staged matings, egg collection, photography, and incubation, and
320 hatching checks as per the parent generation. We counted eggs and hatchlings using
321 the cell count tool in IMAGEJ, with these counts being conducted by a single observer
322 (MDP). For ease of analysis, we pooled measures of fecundity and hatching success
323 across all egg batches. Our sample size consists of eggs laid by 83 offspring pairs

descending from 45 parent pairs ("early" = 47 offspring descending from 32 parent pairs, "late" = 36 offspring descending from 26 parent pairs). Meanwhile, a single pair was removed from the hatching success analysis due to unreliable count data (many nymphs had died at the time of the hatchling counts), leaving 82 egg batches for the offspring hatching success analysis.

2.6 Data Analysis

2.6.1. General procedure for modelling life-history traits in brms

Model workflow

We undertook all statistical analysis and data visualisation in *RStudio v.2025.05.1+513*, using *R v.4.3.3* (R Core Team, 2024). We used the package *brms v.2.21.0* (Bürkner, 2017), implementing *rstan* v.2.32.2 (Gelman et al. 2015; Stan Development Team, 2024) as a backend, to create Bayesian generalised linear mixed models (GLMMs). We follow the location-scale approach to regression modelling, where both first-order (i.e., the mean/location (μ)) and second-order (i.e., the variance/scale (σ *or* ϕ)) traits are estimated (O'Dea et al. 2022; Nakagawa et al. 2025). This location-scale modelling approach permits heteroskedasticity, where differences in error variance exist across the predictors (Barreda & Silbert, 2023). Additionally, in models containing a zero-inflation component (z), we also directly estimated z to identify whether any predictors were associated with zero outcomes in the response.

We included both fixed (β) and random (u) terms in the location submodel, while only fixed effects were included in the scale and zero-inflation components. This single-

346 hierarchical framework ensured that the distributional parameters remained
347 identifiable (Nakagawa et al. 2025). Where possible, we included the parents' age as
348 a random slope, allowing the effect of parental age to differ between parent pairs.
349 We performed model simplification within the scale and zero-inflation component by
350 removing predictors exhibiting no effect or those that were weakly identifiable to
351 reduce the risk of model overparametrisation (Barreda & Silbert, 2023). We always
352 retained parental age in the scale and zero-inflation components, being central to our
353 underlying question (i.e., whether parental age affects the *variability* of offspring
354 traits). In the location component, we did not perform model simplification beyond
355 the removal of non-meaningful interactions (including quadratic terms) to ease the
356 interpretation of single-effect predictors. All continuous covariates were z-
357 transformed (mean = 0, standard deviation (SD) = 1), to facilitate prior specification.

358 For each fixed effect, we show the posterior mean estimates alongside the 95%
359 credible intervals (CI's) from the *brms* output. Additionally, we assessed the existence
360 and importance of the fixed effects with both the probability of direction (pd) and
361 Region of Practical Equivalence (ROPE), as estimated from *bayestestR v.0.17.0*
362 (Makowski et al. 2019). We used *pd* to describe the proportion of posterior estimates
363 in the same direction as the posterior mean, to assess whether the effect existed. We
364 assumed fixed effects with a pd ≥ 98% were real (i.e., differed from zero; Makowski et
365 al. 2019). However, the pd only indicates directionality, not the practical importance
366 nor magnitude of effect, which is better described by the ROPE statistic. ROPE
367 defines an area around the null value effectively equivalent to zero, and of negligible
368 interest (Kruschke, 2018). In this study, we set ROPE to the equivalent of $\pm 0.1 * SD_y$

(Kruschke, 2018). This range was consistently applied across model families, with appropriate transformation to the log-link scale when necessary. Meanwhile for models with the logit link (i.e., Bernoulli & beta-binomial) we set ROPE to the default (±0.18). We inferred that parameter posterior distributions with > 95% of estimates in ROPE were negligible, while those with <5% within the ROPE had a substantial effect (Makowski et al. 2019; Schwaferts & Augustin, 2020). We interpreted cases between these thresholds with caution, weighing the magnitude of the estimate (ROPE) with the directional evidence (pd). We provide all pd and ROPE statistics, together with model summaries, in the supplementary results (*Appendix S1: Table S3 – S20*). We did not apply ROPE to the scale or zero-inflation components, as defining areas of negligible effect for these parameters lacked interpretability.

For post-hoc inferences, we used the package *emmeans v.1.10.1* (Lenth, 2024) to report posterior medians and 95% HDI's (Highest density intervals) on the response scale. Additionally, as a visual measure of effect size, we then converted these post-hoc medians to a standardised mean difference (SMD), by dividing the pairwise difference by the raw standard deviation of the response. Using the package *bayesplot v.1.11.1* (Gabry & Mahr, 2025), we also plotted posterior distributions for fixed-effect predictors, plotting the posterior medians ± 95% CI's. We used the package *ggplot2 v.4.0.0* (Wickham, 2016) to visualise effect sizes, the raw data, and model predictions for the trait mean and variance. To visualize the entire distribution of offspring traits (i.e., variance), we plotted the probability mass for discrete outcomes (*p(x)*), the probability density for continuous outcomes (*f(x)*), and the

391 cumulative incidence for time-to-event outcomes (*F(x)*). The plotted predictions were
392 based on the median estimate ± 95% CI across posterior draws.

393 *Parental age: within-subject centring and selective disappearance*

394 We used within-subject centring to control for between-individual heterogeneity in
395 parental age (Van De Pol & Wright, 2009). First, we described the between-parent
396 effect with the *parents' average age at reproduction* ($\overline{age}$), calculated as the average
397 age of the mother across mating attempts resulting in successfully hatched eggs.
398 Second, the within-parent effect was captured by the *parents' delta age at*
399 *reproduction (Δage*), calculated as the difference between the parents' average age
400 and the mother's age at the current mating attempt. Here, we concluded that the
401 effect of *Δage* on each offspring trait represented the true within-individual parental
402 age effect. Across all models, a unit increase in *Δage* corresponds to a single standard
403 deviation increase in the parents' age at reproduction, or 2.06 weeks on the original
404 scale. We could not include the father's age at reproduction as an additional
405 covariate, as this was tightly correlated with maternal age (Pearson's correlation
406 coefficient *r* [95% CI] = 0.98 [0.97, 0.98]).

407 As a test for selective disappearance, we calculated the difference between the
408 slopes of these two parental age terms (using the *hypothesis ()* function from *brms*). If
409 the slopes for $\overline{age}$ and *Δage* matched (i.e., did not differ from zero), then we assumed
410 the between-individual parental age effect equaled the within-individual effect (Van
411 De Pol & Wright, 2009). If the Posterior probability (*Post.prob*) for the Δslopes
412 between the two terms had an estimate <0.98, we inferred there was uncertain
413 evidence for selective disappearance. The parents' delta age was initially included as

414 a quadratic, before the quadratic term was removed to ease interpretation of the
415 linear slope and simplify tests of selective disappearance (Fay et al. 2022), as its
416 inclusion did not improve Out-of-sample predictive performance (*details below*). We
417 aimed to include the parents' cumulative number of successful mating attempts (i.e.,
418 mating attempts from which eggs hatched, and offspring were subsequently housed)
419 as an additional covariate, to tease apart any variation in offspring phenotypes
420 explained by accumulated reproductive effort. However, this term was tightly
421 correlated with *Δage* owing to our longitudinal study design (cumulative number of
422 mating attempts ~ *Δage*, Pearson's correlation coefficient *r* [95% CI] = 0.94 [0.92,
423 0.95]; *Appendix S1: Figure S2*) and was not included in our modelling framework.

424 *Model settings*

425 Unless otherwise stated, we used the following settings for our fitted models: four
426 Hamiltonian Monte Carlo (HMC) parallel chains, 5000 iterations each (2500 warmup
427 chains which were discarded), and a thinning of one. This resulted in 10,000 post-
428 warmup posterior samples per parameter. We followed standard practice and
429 monitored model convergence and the effective sample sizes as recommended by
430 Gelman & Shalizi (2013). We used the potential scale reduction statistic ($\hat{R}$) supplied
431 by the model output to compare between and within-chain estimates for the model
432 parameters. We assumed that chains had sufficiently mixed when $\hat{R}$ <1.01 (Gelman &
433 Rubin, 1992; Vehtari et al. 2021). Model convergence was said to be achieved when
434 the effective sample sizes (both bulk and tail) for model parameters were >1,000
435 (Gelman & Rubin, 1992; Vats & Knudson, 2021).

Model diagnostics

To assess goodness-of-fit, we inspected the Leave-One-Out Probability Integral Transform (LOO-PIT) plots from *bayesplot*, ensuring the LOO-PIT integrals were uniformly distributed (Vehtari et al. 2017; Säilynoja et al. 2022; Nguyen et al. 2025). Additionally, we used the *ppc_loo_intervals()* function to determine how well our model predicted each observation. We also visualised how well the empirical distribution of the data (*y*) fitted to the distributions of simulated data (*yrep*) from the posterior predictive distribution, using the *pp_check()* function (Gabry et al. 2019; Winter & Bürkner, 2021). Additionally, we checked for any multicollinearity between predictors (VIF) using the package *car v.3.1.2 (*Fox & Weisberg, 2019*)*.

Model predictive performance

To assess the Out-of-sample predictive performance of each model, we used the expected log pointwise predictive density (*elpd*), calculated with Pareto smoothed importance sampling Leave-One-Out cross-validation (PSIS-LOO; Vehtari et al. 2015, 2017). For a series of models fitted to the same data, a higher *elpd* value indicates greater predictive performance (Vehtari et al. 2016). We assumed that models had a similar predictive ability if the difference in between their *elpd* (*Δelpd*) was < four (Sivula et al. 2020). When the predictive ability of the compared models was comparable, we selected the simplest model (with fewest predictors/no interactions).

Choice of prior distribution

For each trait, we conducted prior sensitivity analysis by fitting models with progressively tighter priors. These ranged from the default flat priors provided by

brms to normally distributed constraining priors that assumed no effect of the fixed effects on the response (Gelman et al., 2008, 2017). Unless otherwise stated, we fitted our final models with weakly informative priors, using $\beta_1 \sim normal\ (mean = 0,\ SD = 1\ SD_y)$ priors for fixed effects, accounting for the scale of the response and the model's link function (Lemoine et al. 2016; Lemoine, 2019). This weakly informative prior assumes a one standard deviation increase in the predictor leads to an approximately one standard deviation change in the response, and rarely any larger scale changes (Møller & Jennions, 2002; Lemoine, 2019). For a detailed overview of our prior specification process, including exact prior values for each model parameter and their justification, see *Appendix S1: Section S3*.

2.6.2 General procedure for survival analyses

Choice of mortality distribution

We used Bayesian survival trajectory analysis (*BaSTA v.2.0.1*; Colchero et al. 2012) to build parametric survival models explaining variation in offspring mortality. *BaSTA* uses a Monte Carlo Markov chains (MCMC) algorithm, describing mortality, the instantaneous risk of death given survival to age x, as $\mu(|\theta)$, where x is age and θ are the model parameters (Colchero & Clark, 2012). We fitted all possible mortality distributions parameterised by *BaSTA* (exponential, Gompertz, Weibull, and logistic) which we extended with the Makeham (*c;* Makeham, 1867) and bathtub (Siler, 1979) terms. The predictive performance of all the models was compared using their deviance information criterian (DIC) value. DIC describes the predictive power of each model by approximating the expected predictive deviance and penalises for model complexity, with a lower DIC indicating that the model has improved predictive

ability (Spiegelhalter et al. 2002; Millar, 2009). We selected the model with the lowest DIC, with a model said to be the best-performing when its ΔDIC < 3 from alternative candidate models. If models had comparable DIC values, we selected the simplest model (with the fewest predictors/no interactions). Out of all mortality distributions, we found the Weibull-Makeham model had the best predictive performance (*Appendix S1: Tables S9 and S21*).

Weibull Makeham model parameters

The Weibull-Makeham model (Pinder et al. 1978) assumes that mortality risk changes as a power function of age, describing mortality as:

$$\mu_b(x|b0, b1) = c + b0b1^{b0}x^{b0-1}.$$

Under the three-parameter Weibull-Makeham model, *b0* represents the shape parameter, *b1* is the scale parameter, and *c* is the Makeham parameter, while x denotes the individual's age.

The shape parameter (*b0*) describes the pattern mortality follows through time. When *b0* equals one, mortality is held constant, while when *b0* equals two, mortality risk increases linearly with age, and when *b0* is greater than two, mortality exhibits an accelerating increase over time. Meanwhile, *b1* determines the characteristic scale of failure times (i.e., when 63.2% of individuals have died, which we interpret as the age of onset for senescence) of the offspring (Gómez et al. 2023). *BaSTA* inverses the scale parameter (*1/b1*), with larger values of *b1* corresponding to an earlier characteristic failure time. Taken together, *b0* and *b1* describe age-specific mortality; *b1* scales the relationship between the age of the individual (x) and the shape of the

503 curve (*b0*), setting the inflection point for when the mortality increase occurs.
504 Finally, *c* is the age-independent baseline mortality risk (i.e., background mortality).
505 Here, we estimated the effect of our predictors on each mortality parameter. In
506 *BaSTA*, both *c* and *b1* have units in years, while *b0* is unitless.

507 *Covariate structure and model settings*

508 We only included categorical predictors in our models (using the *"fused"* covariate
509 structure) for which *BaSTA* provides Kullback-Leibler discrepancy calibration values
510 (KLDC; Kullback & Leibler, 1951). Between a pair of predictors, KLDC provides a
511 measure of the distance between their posterior distributions for each parameter. A
512 value of 0.5 indicates that posterior distributions overlap completely, while a KLDC of
513 one indicates that the posterior distributions exhibit no overlap (McCulloch, 1989).
514 When KLDC >0.8, we concluded that there was a substantial difference between the
515 compared predictors in their effect on the mortality parameter, assuming this
516 indicated a genuine biological effect (Karabatsos, 2006; Rodríguez-Muñoz et al. 2019).

517 Our final posterior distributions were based on four MCMC chains with 80,000
518 iterations (burn-in of 1001), and a thinning interval of 50, resulting in n = 6320 post-
519 burn-in posterior samples per parameter. We assessed the convergence of the
520 parameter estimates using $\hat{R}$. Despite reducing the length of the burn-in sequence,
521 the simulations in all our models rapidly converged, mixing well when stabilising
522 towards the target distribution ($\hat{R}$ <1.002), and serial autocorrelation was low (<5%).
523 We assessed model fit visually by overlaying the parametric survival curves over the
524 Kaplan-Meier plots of observed survival (*Appendix S1: Figures S10 and S18*). For each

525 parameter, we report the posterior mean along with the 95% CI's. We used *ggplot2* to
526 visualise parameter posterior distributions alongside the offspring's cumulative
527 survival curves and instantaneous hazard rates (i.e., mortality). In the final plots, we
528 show the full CI's for the posterior distributions for each mortality parameter, while
529 model predictions for the survival and hazard curves reflect the median ± 95% CI
530 estimates across posterior draws.

531 *Prior sensitivity analysis*

532 To assess the sensitivity of the mortality parameters to our choice of prior structure,
533 we conducted the analysis using three prior distributions (default, weakly-
534 informative, and moderately-informative), before selecting normally distributed
535 weakly informative priors for covariates, as recommended by Lemoine (2019).

536 Specifically, we set the prior for *c* and b0 to the weakly-informative *BaSTA* defaults
537 (c~normal [0,1], *b0*~normal [1.5, 1]). For *b1,* we adapted the default prior (normal [0,
538 1]) to a scale that was appropriate for the short lifespan of *G.bimaculatus*. Given
539 *BaSTA* was developed for long-lived wild vertebrates, the default *b1* prior is centred
540 incorrectly for our study species. To prevent inappropriate prior artifacts from
541 affecting posterior estimates, we scaled the *b1* prior to the raw population mean for
542 total or adult lifespan (*details below*). All priors were truncated to be ≥ 0, given the
543 Weibull model's parameterisation. We also evaluated tighter moderately informative
544 priors, with the same mean, but ½ the SD of the weakly informative priors.

2.6.3. Statistical models

Approach to modelling offspring longevity

We initially fitted a bathtub term to the Weibull model to capture the steep early-life mortality of our study population. However, inspection of the Kaplan-Meier curve against the parametric survival curve indicated the bathtub shape was too shallow to estimate early-life mortality (*Appendix S1*: *Figure S3*). Therefore, we used a mixed modelling approach to describe offspring survival. We first modelled the offspring's probability of early-life survival (from birth to week four of life; used as a proxy of frailty) using a GLMM. Here, the response represented whether an individual offspring survived their first four weeks of life (i.e., probability of survival: survived = 1, died = 0). We also modelled offspring juvenile survival (from week four post-hatching until adulthood), where the response represented whether an individual offspring survived the remainder of the juvenile period (survived =1, died = 0).

Then, conditional on the offspring surviving their first four weeks, we modelled their total lifespan and mortality using a dual Weibull modelling approach. First, we modelled mean offspring longevity using a GLMM, with the *brms* Weibull model allowing for the inclusion of multiple covariates, random terms, and tests of selective disappearance. However, under *brms,* we could not place covariates on the shape parameter (*k; analogous to b0*) as this quickly inflated model complexity, with the model failing to converge as indicated by low ESS values (<100) and $\hat{R}$ values > 1.5. Here, we only estimated the effect of the model covariates on the trait mean (μ). Subsequently, we then used the Weibull-Makeham model parameterised by *BaSTA* to

567 describe variation in the offspring's baseline (*c*) and age-specific mortality (*b1* and
568 *b0*).

569 *GLMMS on offspring traits*

570 Models on each offspring trait used different likelihoods and subsets of available data
571 (*Table 1*), but the covariate structure remained comparable across GLMMs on the
572 following: early-life survival, juvenile survival, total lifespan, development time,
573 adult mass, and adult lifespan. Here, we included, as fixed-effects: *Δage*, $\overline{age}$, and
574 the parents' temperature treatment (*Temp*: three-level factor; "25.5", "28", and
575 "30.5°C"). For development time, adult mass, and adult lifespan, we also included
576 offspring sex (two-level factor: "Male", "Female") as a fixed effect. Meanwhile, for
577 adult mass, the mother's adult mass (grams) was additionally fitted as a fixed effect.
578 Our global model on offspring total lifespan did not include offspring sex, to allow for
579 the inclusion of unsexed nymphs that died before adulthood. Therefore, we
580 conducted a subset analysis on the sexed adults, enabling offspring sex to predict
581 variation in their total lifespan. In all models, we included parent pair ID as a random
582 intercept and fitted *Δage* as a random slope.

583 Where possible, in the location component, we tested support for the three-way
584 interaction between *offspring sex x Temp x Δage*, including all lower-order
585 interactions between these terms. We then fitted two additional models including
586 either the two-way interaction between *Temp x Δage* or *offspring sex x Δage*, to test
587 whether parental age effects differed among temperature treatments or between
588 offspring sexes. For the scale component, to prevent model overparmaterisation, we
589 only tested the two-way interactions between *Temp x Δage* or *offspring sex x Δage*.

590 All interactions were removed from our final inference, as they did not improve
591 predictive performance. However, we always report estimates for the *Temp x Δage*
592 interaction from the location component, as this was central to answering whether
593 parental age effects exhibited any environmental heterogeneity.

594 For the GLMMs on offspring fecundity and hatching success, we only included the
595 following fixed-effects: the parents' age category (two-level factor: "Early-aged",
596 "Late-aged") and the parents' temperature treatment. Additionally, we tested
597 support for the two-way interaction between these two-terms, before we dropped the
598 interaction from the final model to ease the interpretation of single effect predictors.
599 Here, we never included interactive terms in the scale component to retain model
600 identifiability, as these models only used a small subset of individuals. In likelihoods
601 containing a variance or zero-inflation component (σ, ϕ, or z), we initially placed
602 both *Δage* (or the parents' age category for reproductive traits) and *Temp* as fixed
603 effects. However, *Temp* was subsequently removed from all scale and zero-inflation
604 models, as its effect on trait variance was weakly estimated.

605 *Offspring mortality models*

606 We used Weibull-Makeham models specified by *BaSTA* to describe variation in
607 offspring mortality, across either the offsprings total lifespan (total mortality;
608 estimated for offspring that survived their first four weeks of life) and adult lifespan
609 (adult mortality; from adult eclosion until death; *Table 1*). We first included the
610 isolated effects of parental age (three-level factor: "Early-aged", "Middle-aged", or
611 "Late-aged") or *Temp* as single-effect predictors on each mortality parameter (*c*, *b1*,

612 and *b0*), before assessing their joint effect (two-way *parental age* x *parental*
613 *temperature* interaction). To improve model convergence, parental age was binned
614 into a three-level factor ("Early-aged": parent mating attempts one & two, "Middle-
615 aged": mating attempts three to five, and "Late-aged": mating attempts six to eight).
616 For the subset of sexed offspring, we also tested support for the interaction between
617 *the parents' age x offspring sex*. We then compared each candidate model to the null
618 model, which contained no predictors. If the DIC of the null model was lower than the
619 alternative candidate models with covariates, we concluded that the predictors did
620 not explain variation in offspring mortality.

621 We used a weakly-informative prior structure, placing the default priors on *b0* and *c*,
622 while we scaled the *b1* prior. For total lifespan, (raw mean ± SD lifespan of 18.31 ±
623 3.97 weeks), this conversion resulted in a truncated *b1* ~ half-normal (2.57, 0.5) prior,
624 permitting characteristic lifespans of between 10 and 40 weeks. Meanwhile, for adult
625 lifespan, (raw mean ± SD adult lifespan = 9.53 ± 3.16 weeks), we used a truncated
626 *b1*~half-normal (4.94, 1) prior, permitting characteristic adult lifespans of between
627 five and 24 weeks. Prior sensitivity analysis confirmed these priors remained weakly
628 informative and did not change our posterior inferences when compared to the
629 default prior structure in *BaSTA* (*Appendix S1: Table S9* and *Table S21*).

Table 1. Error structures, parameters, link-functions, and sample sizes used by models on each offspring trait.

Offspring trait *(response)*	Likelihood & Package	Parameters & link	*n*	Corresponding Appendices
Early-life survival *(survived =yes/no)*	Bernoulli *(brms)*	*μ (logit)*	1317 offspring 78 parent pairs	***Section S4*** *Model selection:* Table S2 *Estimates:* Table S3
Juvenile survival *(survived =yes/no)*	Bernoulli *(brms)*	*μ (logit)*	987 offspring 77 parent pairs	***Section S5*** *Model selection:* Table S4 *Estimates:* Table S5
Total lifespan *(weeks)*	Weibull *(brms)*	*μ (log)* *k (identity)*	987 offspring 77 parent pairs sexed subset: 947	***Section S6.1*** *Model selection:* Table S6 *Estimates:* Table S7, S8
Total mortality *(c & b1 = years b0 = unitless)*	Weibull-Makeham *(BaSTA)*	*c, b0, b1 (identity)*	987 offspring 77 parent pairs sexed subset: 947	***Section S6.2*** *Model selection:* Table S9 *Estimates:* Table S10
Development time *(weeks)*	Lognormal *(brms)*	*μ (log)* *σ (log)*	939 offspring 77 parent pairs	***Section S7*** *Model selection:* Table S11 *Estimates:* Table S12
Adult mass *(grams)*	Gaussian *(brms)*	*μ (identity)* *σ (log)*	937 offspring 77 parent pairs	***Section S8*** *Model selection:* Table S13 *Estimates:* Table S14
Fecundity *(No. eggs laid)*	Negative binomial *(brms)*	*μ (log)* *ϕ (log)*	83 offspring 45 parent pairs	***Section S9*** *Model selection:* Table S15 *Estimates:* Table S16
Hatching success *(No. hatched eggs per trial)*	Zero-inflated beta-binomial *(brms)*	*μ (logit)* *ϕ (log)* *z (logit)*	82 offspring 45 parent pairs	***Section S10*** *Model selection:* Table S17 *Estimates:* Table S18
Adult lifespan *(weeks)*	Weibull *(brms)*	*μ (log)* *k (identity)*	938 offspring 77 parent pairs	***Section S11.1*** *Model selection:* Table S19 *Estimates:* Table S20
Adult mortality *(c & b1 = years b0 = unitless)*	Weibull-Makeham *(BaSTA)*	*c, b0, b1 (identity)*	939 offspring 77 parent pairs	***Section S11.2*** *Model selection:* Table S21 *Estimates:* Table S22

Note: Sample sizes describe the number of offspring from our starting pool (n = 1317 individuals) included in each model. Corresponding supplements refer to appendix sections detailing the model selection process (e.g., choice of likelihood, evidence for interactions) and parameter estimates.

Abbreviations: μ = location parameter; k = shape parameter (Weibull model in brms); c = Makeham term; b0, b1 = age-specific mortality parameters; σ = residual standard deviation; ϕ = dispersion parameter (Negative binomial/Zero-inflated beta-binomial); z = zero-inflation parameter.

641 # 3. Results

642 We report estimates for fixed effects on either the mean trait outcome (location
643 component (μ); β_1), on trait variability (scale component (σ/ϕ); γ_1), and on the
644 probability of zero outcomes (zero-inflation component (z); δ_1). We also provide the
645 probability of direction (pd), which indicates the direction of the effect, and the
646 percentage of posterior estimates in the region of practical equivalence (ROPE),
647 indicating the practical magnitude of the effect.

648

649 ## 3.1 Offspring early-life and juvenile survival

650 We found no evidence that parental age affected offspring early-life survival ($\beta_{\Delta age}$ = -
651 0.01, 95% CI [-0.18, 0.15], pd = 54.95%, 100% in ROPE*; Figure 1B, Appendix S1: Figure*
652 *S4*) and uncertain evidence for a slight positive effect on juvenile survival ($\beta_{\Delta age}$ =
653 0.30, 95% CI [-0.04, 0.65], pd = 95.62%, 23.44% in ROPE; *Figure 1B, Appendix S1:*
654 *Figure S5*). Additionally, there was limited evidence for an interactive effect of
655 parental age and temperature on either early-life survival ($\beta_{\Delta age:Temp\ (28.0^\circ C)}$ = 0.07, 95%
656 CI [-0.33, 0.47], pd = 62.47, 63.53% in ROPE; $\beta_{\Delta age:Temp\ (30.5^\circ C)}$ = 0.01, 95% CI [-0.39,
657 0.40], pd = 52.81%, , 66.53% in ROPE) or juvenile survival ($\beta_{\Delta age:Temp\ (28.0^\circ C)}$ = 0.03, 95%
658 CI [-0.72, 0.80], pd = 52.22%, 38.11% in ROPE; $\beta_{\Delta age:\mathrm{Temp}\ (30.5^\circ C)}$ = -0.30, 95% CI [-1.06,
659 0.44], pd = 78.03%, 30.20% in ROPE).

660

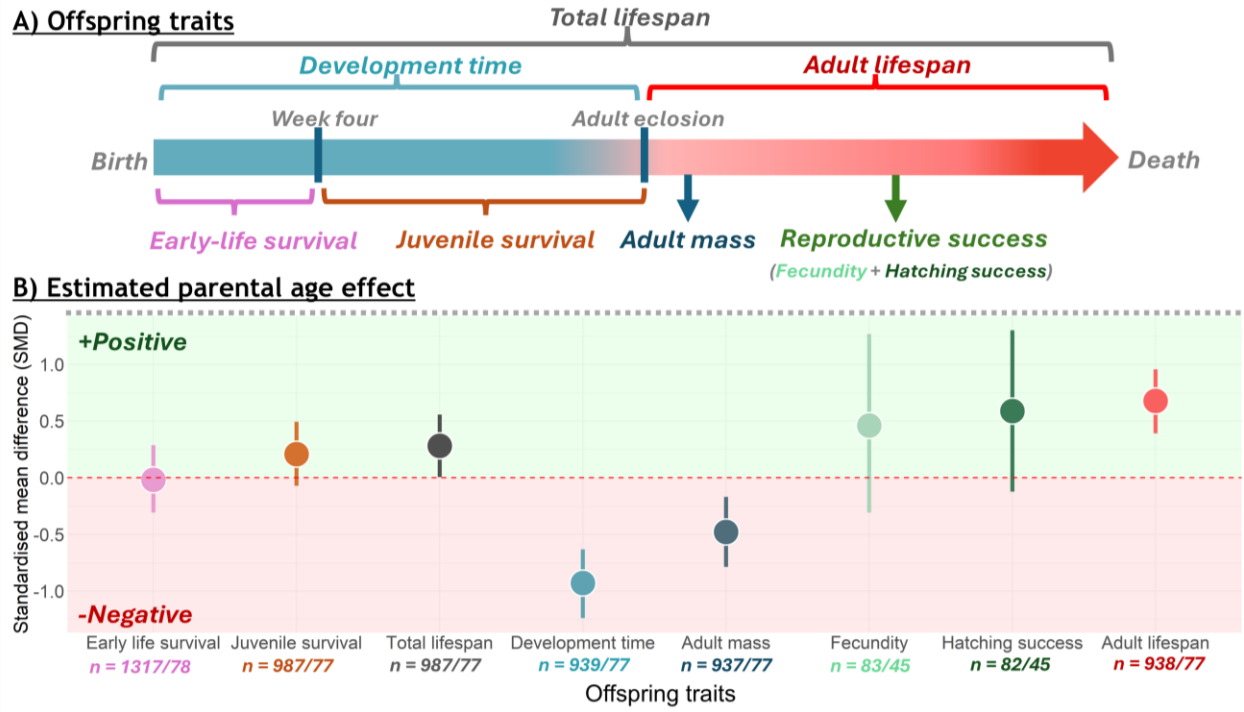


Figure 1. A) Overview of measured offspring traits. B) Standardised mean difference (i.e., effect size) of the parents' *Δage* on each offspring trait. Points represent the standardised mean difference and error bars show the 95% credible intervals. Samples sizes are shown as the number of offspring/number of parent pairs. Pairwise comparisons are based on the predicted difference in the average trait value (μ) between early-conceived (-1.96 SDs from the mean parental age) and late-conceived (+1.96 SDs from the mean parental age) offspring; a difference in parental age of ~8 weeks. For fecundity and hatching success, pairwise comparisons are based on the mean difference between the two categorical parental age groups ("early-aged" versus "late-aged"). All effects were standardised by the standard deviation of the response. For traits modelled on the log-odds scale (i.e., hatching success), the credible intervals are shown on the response scale and appear wider due to the non-

674 linear back-transformation. Effects are strong when the credible intervals are further
675 away from zero, while intervals overlapping zero show greater uncertainty.

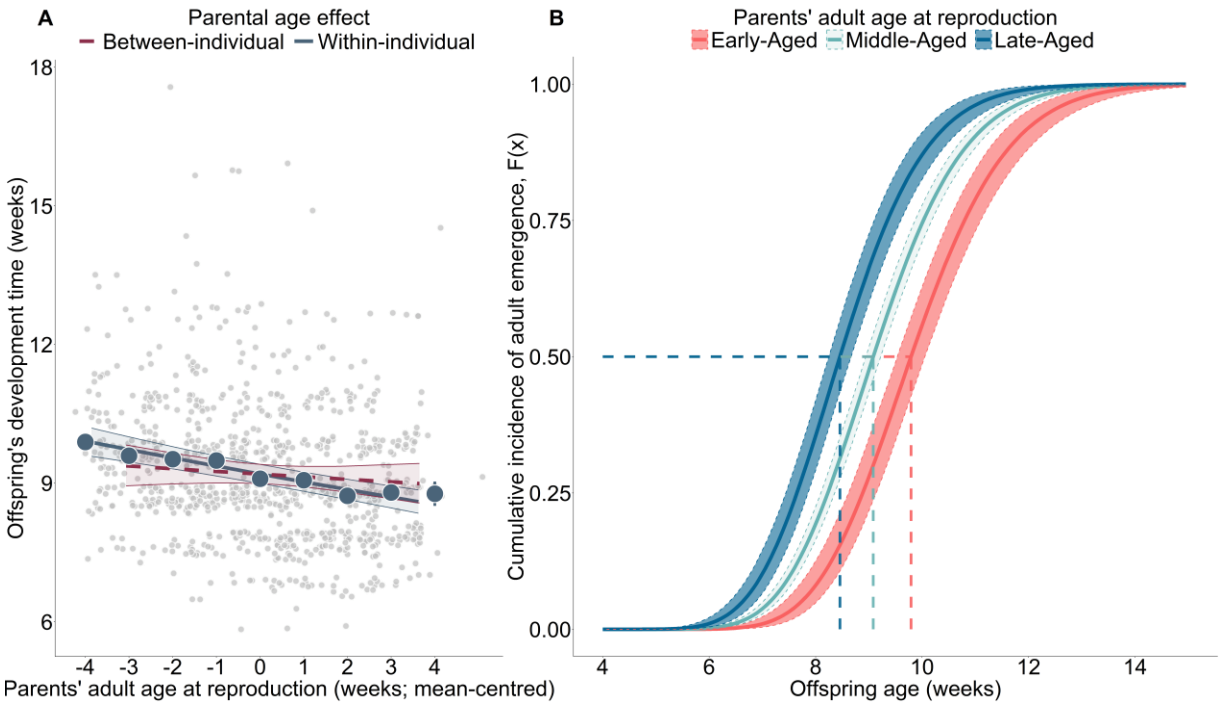
676 3.2 Offspring total lifespan and mortality

677 There was uncertain evidence for a positive parental age effect on the offspring's
678 total lifespan ($\beta_{\Delta age}$ = 0.02, 95% CI [0.00, 0.03]; *Appendix S1: Figure S6*). Although this
679 increase was well supported (pd = 98.84%), the effect was mostly negligible (96.19% in
680 ROPE; 0.03 log-weeks per SD increase in parental age). Post-hoc comparisons revealed
681 little difference in total lifespan, with the latest-conceived offspring (+1.96 SDs from
682 mean parental age) surviving for a median 18.80 weeks (95% HDI [18.30, 19.40
683 weeks]), compared with 17.70 weeks (95% HDI [17.20, 18.30 weeks]) for the earliest-
684 conceived offspring (-1.96 SDs from mean parental age; *Figure 1B*). There was limited
685 evidence this parental age effect differed across temperature treatments ($\beta_{\Delta age:Temp}$
686 $_{(28.0^\circ C)}$ = -0.03, 95% CI [-0.06, 0.01], pd = 93.60%, 51.39% in ROPE; $\beta_{\Delta age:Temp\ (30.5^\circ C)}$ = -
687 0.03, 95% CI [-0.06, 0.00], pd = 96.47%, 43.83% in ROPE). These findings were further
688 supported by our mortality models, which found only weak positive effects of parental
689 age on both offspring baseline (*c*) and age-specific mortality (*b0* and *b1; Appendix S1:*
690 *Section S6.2)*.

691 3.3 Offspring development time

692 Offspring development time decreased with increasing parental age ($\beta_{\Delta age}$ = -0.04, 95%
693 CI [-0.05, -0.03]; *Figure 2A, Appendix S1: Figure S11*), with this effect being strongly
694 supported (pd = 100% and 0% in ROPE; ±0.01 log weeks per SD increase in parental
695 age). The latest-conceived offspring (+1.96 SD parental age) eclosed at 8.43 weeks on

696 average (95% HDI [8.23, 8.64 weeks]) compared with 9.81 weeks (95% HDI [9.58, 10.07
697 weeks]) for the earliest-conceived offspring (-1.96 SD parental age; *Figure 1B, Figure*
698 *2B*). This parental age effect was likely independent of the parents' temperature
699 treatment ($\beta_{\Delta age:Temp\ (28.0^{\circ}C)}$ = -0.01, 95% CI [-0.04, 0.02], pd = 77.67%, 44.35% in ROPE;
700 $\beta_{\Delta age:Temp\ (30.5^{\circ}C)}$ = 0.00, 95% CI [-0.03, 0.02], pd = 57.75%, 56.34% in ROPE).



701

702 **Figure 2. A) The effect of parent's age on offspring's mean development time.** On
703 the x-axis is the parents' age at reproduction (mean-centred) and on the y-axis is
704 offspring development time (weeks). Small dots are raw data while large dots are raw
705 means ± SE. The lines and ribbons represent the posterior mean ± 95% CI. The colours
706 represent the parental age effect: red dashed line = between-parent, blue solid line =
707 within-parent. **B) The effect of the parents' age at reproduction on the cumulative**
708 **incidence of offspring adult emergence.** The x-axis depicts offspring age (weeks),
709 while the y-axis displays the cumulative incidence of adult emergence, *F(x)*. Lines and

710 ribbons are the posterior predictions for *F(x)*. The colours represent within-parent
711 deviations in age relative to the parent's mean age: red = "early-aged" (-1.96 SDs
712 from the mean parental age), green = "middle-aged" (the mean parental age), blue =
713 "late-aged" (+1.96 SDs from the mean parental age). The dashed lines represent the
714 posterior median estimate for development time per parental age category. N = 939
715 offspring from 77 parent pairs.

716 3.4 Offspring adult mass

717 Not only did offspring from older parents accelerate development; they may have
718 done so at the expense of adult mass ($\beta_{\Delta age}$ = -0.02, 95% CI [-0.04, -0.01]; *Figure 3A*,
719 *Appendix S1: Figure S12*). Although consistently negative (pd = 99.99%), the strength
720 of this effect was uncertain (24.66% in ROPE; ±0.02g per SD increase in parental age).
721 Post-hoc comparisons revealed the latest-conceived offspring (+1.96 SD parental age)
722 were the smallest at 0.71 g (95% HDI [0.69, 0.74 g]), while earliest-conceived
723 offspring (-1.96 SD parental age) weighed, on average, 0.80 g (95% HDI [0.77, 0.83 g]),
724 a mean difference of 11.77% (95% HDI [5.60, 18.01%]; *Figure 1B*). We found limited
725 evidence that this parental age effect depended on the parents' temperature
726 treatment ($\beta_{\Delta age:Temp\ (28.0^{\circ}C)}$ = -0.02, 95% CI [-0.05, 0.01], pd = 78.03%, 55.54% in ROPE;
727 $\beta_{\Delta age:Temp\ (30.5^{\circ}C)}$ = -0.02, 95% CI [-0.05, 0.00], pd = 57.27%, 33.87% in ROPE).

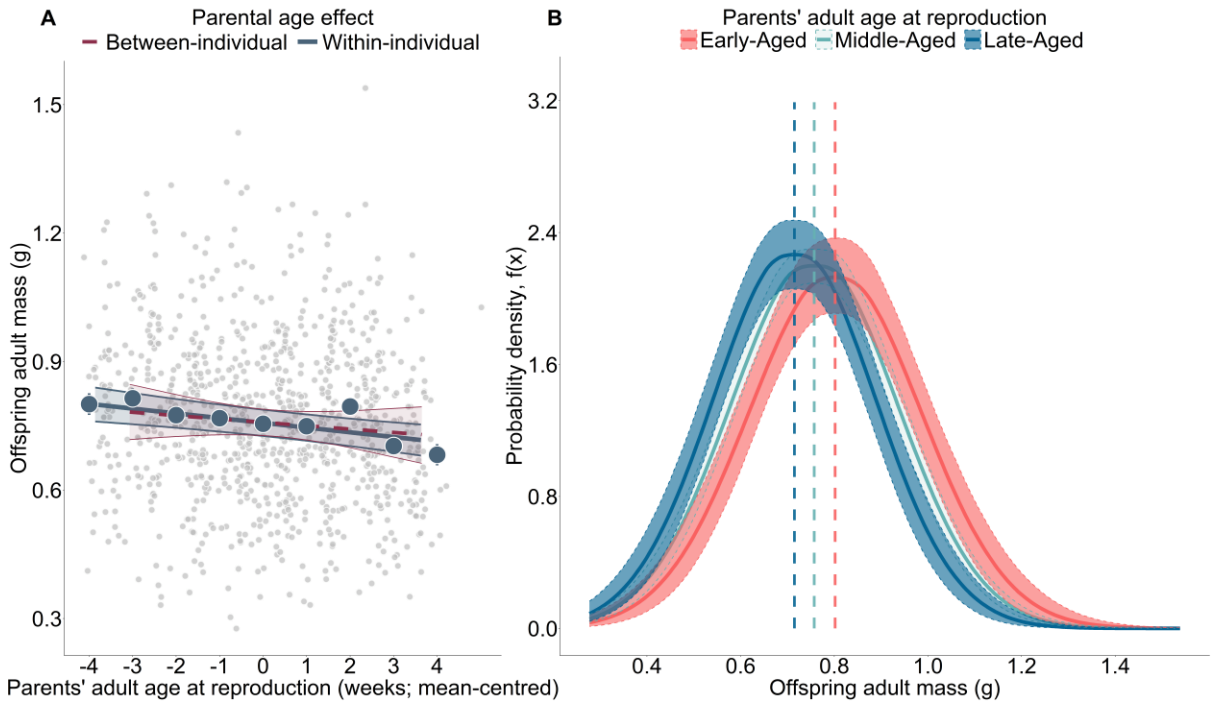


Figure 3. A) The effect of parent's age on offspring adult mass. On the x-axis is the parents' age at reproduction (mean-centred) and on the y-axis is offspring adult mass (g). Small dots represent raw data, large dots are raw means ± SE. Lines and ribbons represent model predictions for the posterior mean ± 95% CI. The colours reflect the type of parental age effect: red dashed line = between-parent, blue solid line = within-parent. **B) The effect of the parents' age at reproduction on the distribution of offspring adult mass (probability density).** On the x-axis is offspring adult mass (g), while the y-axis shows probability density, *f(x)*. Lines and ribbons represent posterior predictions for *f(x)*. The colours represent within-parent deviations in age relative to the parent's mean age: red = "early-aged" (-1.96 SDs from their mean parental age), green = "middle-aged" (their mean parental age), blue = "late-aged" (+1.96 SDs from their mean parental age). The dashed lines represent the posterior

741 median estimate for adult mass per parental age category. N = 937 offspring from 77
742 parent pairs.

743 3.5 Offspring reproductive success

744 *3.5.1 Offspring fecundity*

745 Increasing parental age may have improved offspring fecundity, although this effect
746 carried broad posterior uncertainty ($\beta_{parental\ age\ (late\text{-}aged)}$ = 0.36, 95% CI [-0.04, 0.75];
747 *Figure 4A*, *Appendix S1: Figure S13*). Even though the direction of the effect was
748 uncertain (pd = 95.79%, *Figure 1B*), the effect may be moderate (11.99% in ROPE;
749 ±0.13 log-eggs). Post-hoc comparisons revealed that offspring from late-aged parents
750 laid a median 342 eggs (95% HDI [246, 449 eggs]), compared with the 243 eggs (95%
751 HDI [177, 317 eggs]) produced by offspring from early-aged parents. There was no
752 evidence for a differential effect of parental age between the parent's temperature
753 treatments ($\beta_{parental\ age\ (late\text{-}aged):\ Temp\ (28.0^{\circ}C)}$ = 0.25, 95% CI [-0.42, 0.91], pd = 76.87%,
754 24.83% in ROPE; $\beta_{parental\ age\ (late\text{-}aged):\ Temp\ (30.5^{\circ}C)}$ = -0.25, 95% CI [-0.93, 0.44], pd =
755 76.27%, 23.80% in ROPE).

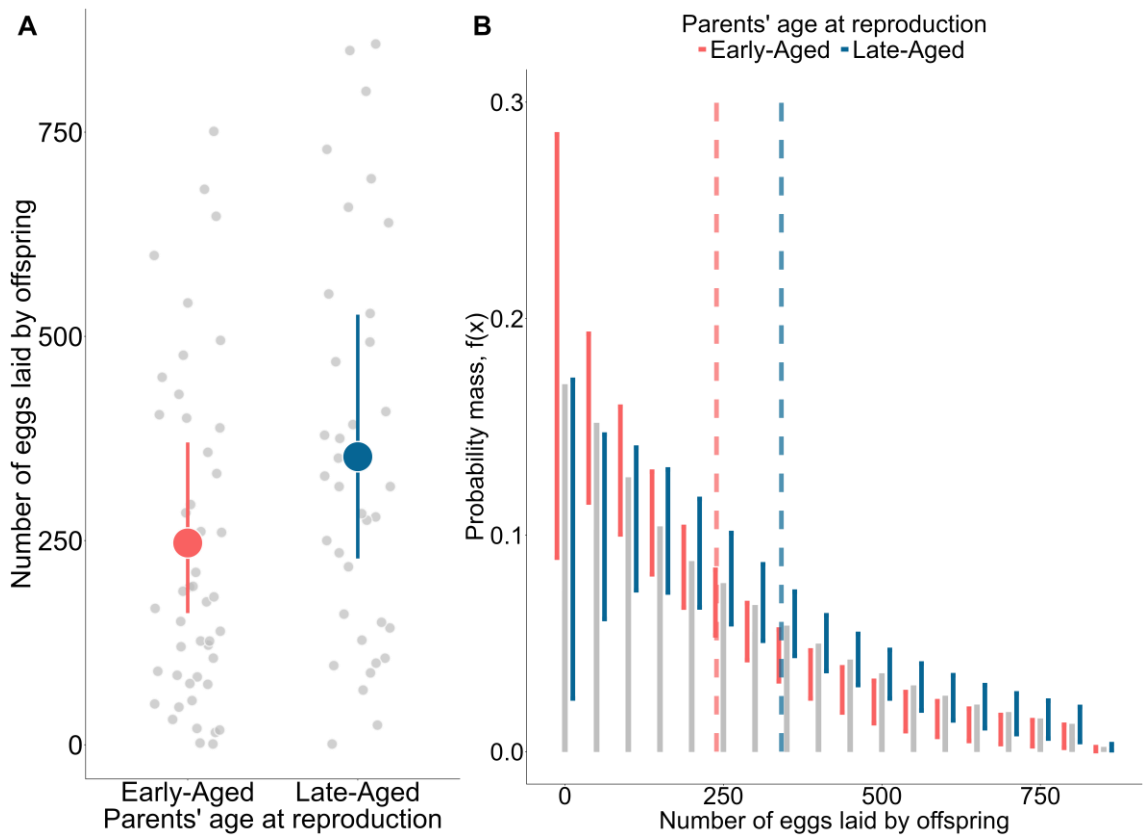


Figure 4. A) The effect of parental age on the mean number of eggs laid by the offspring. On the x-axis is the parents' age category, and on the y-axis is number of eggs laid by the offspring. Large dots represent model predictions ± 95% CI for mean offspring fecundity. Small dots are raw data. **B) The effect of parental age on the distribution of offspring fecundity (probability mass, *p(x)*).** Grey bars represent the median probability mass that the number of eggs laid by the offspring falls within each bin (binwidth = 50-eggs). The vertical-coloured bars are the 95% CI's around the probability mass, coloured by parental age (red = early-aged, blue = late-aged). Dashed lines represent the posterior median for offspring fecundity in each parental age group. Eggs from n = 83 offspring descending from 45 parent pairs.

767 ### *3.5.2 Offspring hatching success*

768 Parental age was associated with higher offspring hatching success ($\beta_{parental\ age\ (late\text{-}aged)}$
769 = 0.74, 95% CI [0.17, 1.33]; *Appendix S1: Figure S14*). This increase received strong
770 support (pd = 99.42% and 0.42% in ROPE; ±0.18), despite exhibiting broad posterior
771 uncertainty. The eggs laid by the latest conceived offspring had a median hatching
772 probability of 0.25 (95% HDI [0.18, 0.33]), compared with 0.14 (95% HDI [0.08, 0.21])
773 for earlier-conceived offspring. However, this estimate does not account for the zero-
774 inflation component of the model (i.e., complete reproductive failure). Although
775 parental age showed little association with complete hatching failure ($\delta_{parental\ age\ (late\text{-}}$
776 $_{aged)}$ = -0.06, 95% CI [-1.17, 1.23], pd = 56.44%), inclusion of this component reduced
777 the predicted difference in median hatching success between the two groups. Here,
778 the average hatching probability dropped to 0.20 (95% CI [0.12, 0.29]) for later-
779 conceived offspring and to 0.11 (95% CI [0.05, 0.18]) for earlier-conceived offspring
780 (*Figure 5A)*. When combining hatching probability with the total number of eggs laid,
781 later-conceived offspring may have hatched a greater number of eggs, with an
782 average of 56.80 hatched eggs (95% CI [33.50, 84.10]), compared to 31.10 eggs (95%
783 CI [15.90, 52.00 eggs]) hatched by earlier-conceived offspring (*Figure 5B*). Finally, the
784 observed parental age effect was broadly consistent across the parent's temperature
785 treatments ($\beta_{parental\ age\ (late\text{-}aged):\ Temp\ (28.0^{\circ}C)}$ = 0.46, 95% CI [-0.60, 1.49], pd = 79.96%,
786 19.51% in ROPE; $\beta_{parental\ age\ (late\text{-}aged):\ Temp\ (30.5^{\circ}C)}$ = -0.32, 95% CI [-0.70, 1.33], pd =
787 73.58%, 73.58% in ROPE).

788

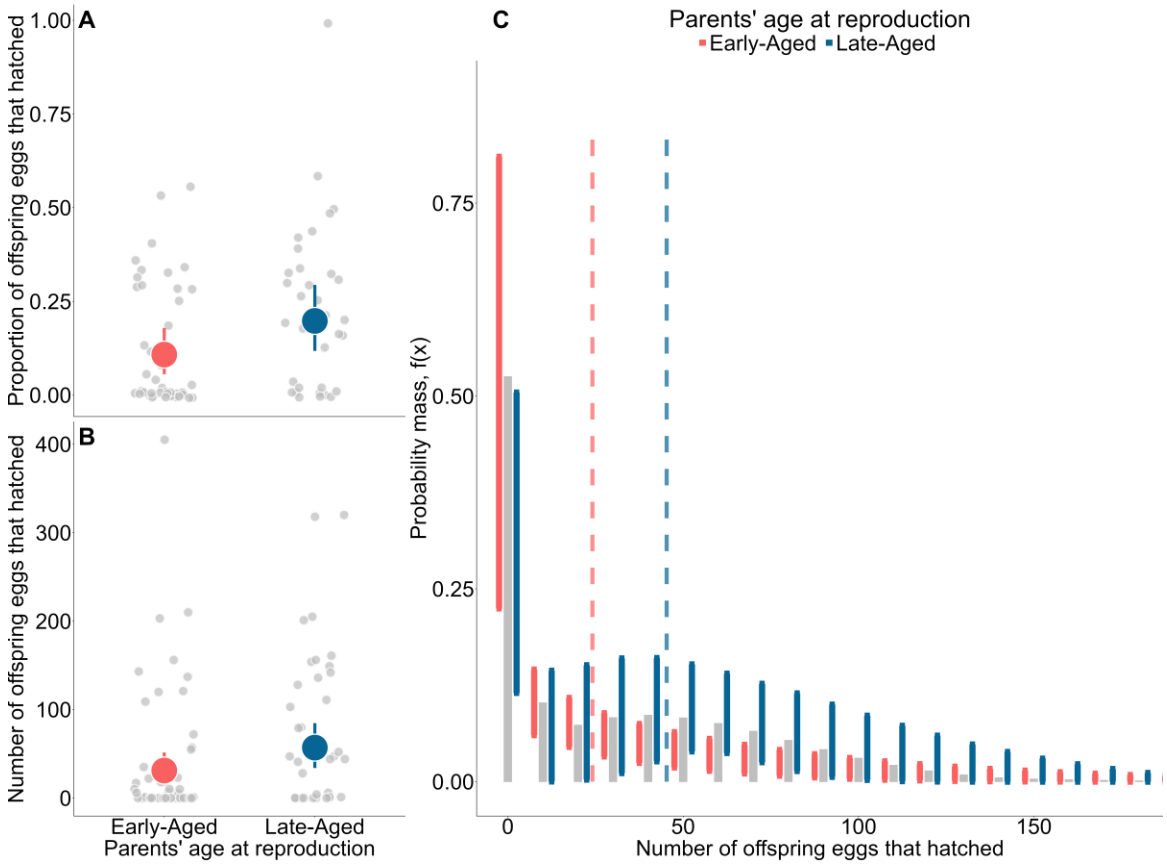


Figure 5. A) The effect of parental age on the proportion of eggs hatched by the offspring. On the x-axis is the parents' age, and on the y-axis is the proportion of eggs that the offspring hatched (accounting for the zero-inflation component). Large dots represent the posterior mean ± 95% CI for hatching success. Small dots are raw data. **B) The effect of parental age on the number of eggs hatched by the offspring**. On the x-axis is the parents' age category, while on the y-axis is the number of eggs that hatched. Here, we present the posterior mean predictions for number of eggs hatched, estimated as a combination of the total number of eggs laid by offspring in each group and their hatching probability. **C) The effect of parental age on the distribution of hatching success (probability mass, *p(x)*).** Grey bars represent the median probability that the number of eggs hatched by the offspring

801 falls within each bin (binwidth = 20-eggs). The vertical-coloured bars are the 95% CI's
802 around the probability mass, coloured by parental age (red = early-aged, blue = late-
803 aged). Dashed lines represent the median number of eggs hatched by offspring in each
804 parental age group. Model predictions for f(x) are visualised at the population median
805 clutch size (240 eggs). Eggs from n = 82 offspring descending from 45 parent pairs.

806 **3.6 Offspring adult lifespan and age-dependent mortality**

807 Given that increasing parental age has limited effect on the offspring's total lifespan
808 (*Figure 1B*), but accelerates their development time (*Figure 2A*), we would expect
809 later-conceived offspring to exhibit prolonged adult lifespans, opposite of a Lansing
810 effect. We found that increasing parental age extended the offspring's mean adult
811 lifespan ($\beta_{\Delta age}$ = 0.06, 95% CI [0.04, 0.08]; *Appendix S1: Figure S15*), with this increase
812 receiving strong support (pd = 100% and 5.27% in ROPE; ±0.04 log-weeks per SD
813 increase in parental age). Post-hoc comparisons revealed that the latest-conceived
814 offspring (+1.96 SD parental age) displayed a median adult lifespan of 10.60
815 weeks (95% HDI [10.17, 11.15 weeks]) compared to 8.51 weeks (95% HDI [8.12, 8.90
816 weeks]) for the earliest-conceived offspring (-1.96 SD parental age; *Figure 1B*). This
817 positive parental age effect was likely independent of the parents' temperature
818 ($\beta_{\Delta age:Temp\ (28.0^{\circ}C)}$ = -0.03, 95% CI [-0.08, 0.02]; pd = 86.27%, 72.52% in ROPE; $\beta_{\Delta age:Temp}$
819 $_{(30.5^{\circ}C)}$ = -0.04, 95% CI [-0.09, 0.01]; pd = 93.82%, 57.19% in ROPE). However, there was
820 some uncertain evidence that this inverse Lansing effect may be stronger in daughters
821 than in sons ($\beta_{\Delta age:\ offspring\ sex\ (male)}$ = -0.04, 95% CI [-0.11, 0.04]; pd = 97.84%, 53.19% in
822 ROPE).

823 For *BaSTA* models on the offspring's adult age-specific mortality rates, the best-
824 supported model included the two-way interaction between parental age and
825 offspring sex (DIC = -2628.82), with this performing better than the model with
826 isolated effects of parental age (DIC = -2617.67) and the null model (DIC = -2597.37).
827 However, evidence for this sex-specific inverse Lansing effect was weak, with the
828 predictive performance of this model likely benefitting from the added inclusion of
829 offspring sex *(Appendix S1: Figure S17*). The null model had improved performance
830 over both the parental temperature model (DIC = -2592.04) and the model including
831 the two-way interaction between *parental age x parental temperature* (DIC = -
832 2595.79; *Appendix S1: Figure S16*). For simplicity, we report mortality estimates from
833 the model with isolated effects of parental age (*Table 2*). Offspring from early and
834 late-aged parents entered adulthood with a similar baseline mortality (*c*). However,
835 those from late-aged parents displayed both a later onset (*b1*) and slowed rate (*b0*) of
836 actuarial senescence over their adult lifespan (*Table 2; Figure 6*).

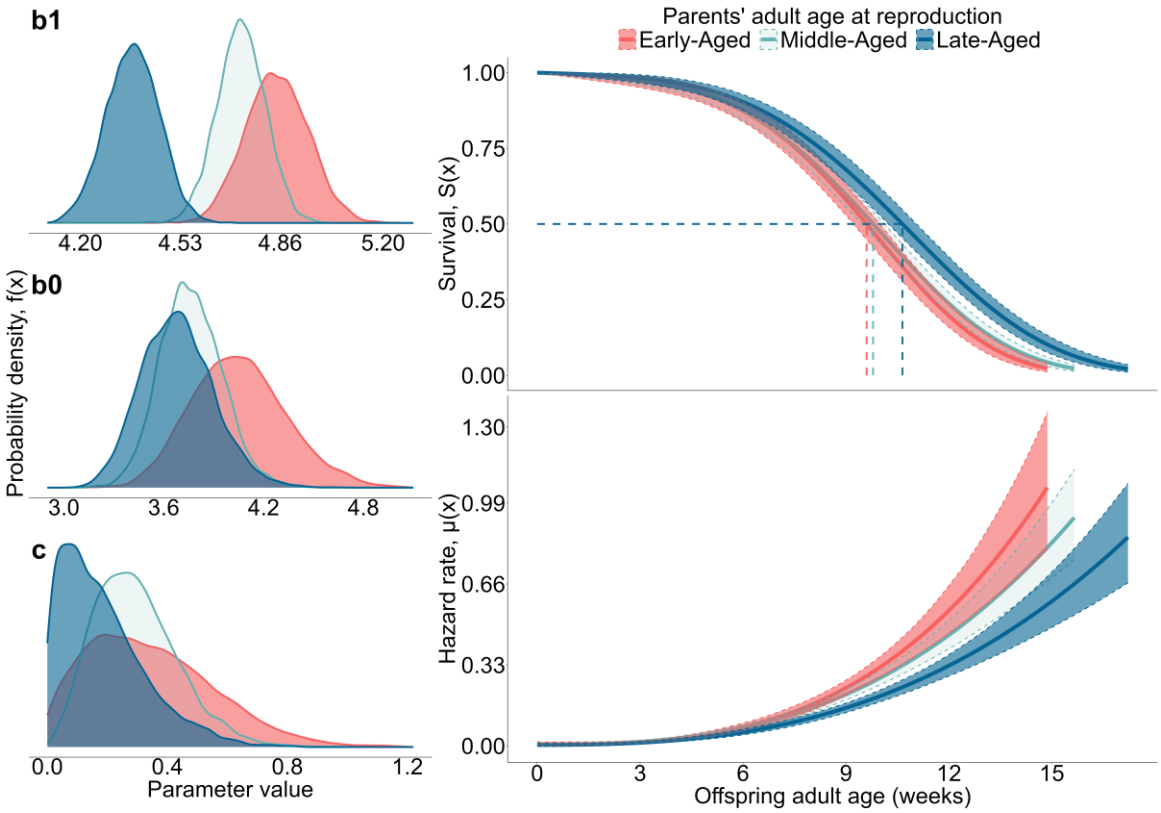


Figure 6. The effect of parental age on offspring adult survival and mortality. Shown on the right is the cumulative adult survival probability (top), with the median adult lifespan for offspring from each parental age group (dashed lines; as estimated from *BaSTA*), and the instantaneous adult hazard rate (bottom) against the offspring's adult age (weeks). Lines and ribbons represent model predictions for adult survival and mortality ± 95% CI, estimated from the posterior draws. For visualisation, we transformed the hazard from the units 1/year to 1/week. The panels on the left show the full posterior distributions of *b1*, *b0*, and *c*. Here, *b1* takes units 1/year, *c* is in years, while *b0* is unitless. The colours describe the different parental age groups: red = "early-aged", green = "middle-aged", and blue = "late-aged". N = 939 offspring from 77 parent pairs.

849 **Table 2. Posterior estimates for the effect of parental age on adult mortality.**

Mortality parameters	*Estimate ± SE*	*95% CI*
Makeham parameter (*c*)		
Parental age: Early-Aged	0.34 ± 0.21	0.03 - 0.81
Parental age: Middle-Aged	0.30 ± 0.14	0.07 – 0.60
Parental age: Late-Aged	0.18 ± 0.14	0.01 – 0.53
Scale parameter (*b1*)		
Parental age: Early-Aged	4.85 ± 0.10	4.65 – 5.05
Parental age: Middle-Aged	4.73 ± 0.07	4.57 – 4.88
Parental age: Late-Aged	4.37 ± 0.09	4.20 – 4.54
Shape parameter (*b0*)		
Parental age: Early-Aged	4.05 ± 0.28	3.54 – 4.65
Parental age: Middle-Aged	3.76 ± 0.18	3.42– 4.14
Parental age: Late-Aged	3.68 ± 0.22	3.29 – 4.13

KLDC pairwise comparisons

Parental age comparison	*c*	*b1*	*b0*
Early-aged vs. Late-aged	0.73	**1.00**	**0.85**
Middle-aged vs. Early-aged	0.59	**0.80**	**0.82**
Late-aged vs. Middle-aged	0.61	**0.99**	0.55

850 *Note: Shown for each mortality parameter is the estimate (posterior mean), the standard*
851 *errors, and the 95% CI. We also show the KLDC values for each mortality parameter to*
852 *describe the distance in the posterior distributions between each parental age category,*
853 *with estimates > 0.80 indicating genuine biological effects highlighted in bold. N = 939*
854 *offspring from 77 parent pairs.*

855 *Abbreviations: KLDC = Kullback-Leibler discrepancy calibration values; SE = Standard error;*
856 *95% CI = 95% Credible intervals*

857

3.6 Variability of offspring traits

We found limited evidence that parental age affected the variability of the following offspring traits: development time (σ: $\gamma_{\Delta age}$ = 0.01, 95% CI [-0.05, 0.06], pd = 60.74%; *Figure 2B*), adult mass (σ: $\gamma_{\Delta age}$ = -0.02, 95% CI [-0.07, 0.03], pd = 76.63%; *Figure 3B*), or fecundity (ϕ: $\gamma_{\text{parental age (late-aged)}}$ = 0.27, 95% CI [-0.34, 0.88], pd = 81.22%; *Figure 4B*). However, offspring from late-aged parents may have exhibited increased precision (ϕ: *i.e., reduced variability*) in their hatching success. Here, most of their hatchling counts clustered towards the posterior mean, although the 95% CI's overlapped zero ($\gamma_{\text{parental age (late-aged)}}$ = 1.04, 95% CI [-0.01, 2.23], pd = 97.44%; *Figure 5C*, *Appendix S1: Figure S14*).

3.7 Selective disappearance of parent phenotypes

We found evidence of selective disappearance for the following offspring traits: juvenile survival ($\Delta\text{slope}_{\overline{age}-\Delta age}$= -0.66, 95% CI [-1.06, -0.26], *Post.Prob* = 1.00), development time ($\Delta\text{slope}_{\overline{age}-\Delta age}$= 0.03, 95% CI [0.02, 0.05], *Post.Prob* = 1.00; *Figure 2A*), and adult lifespan ($\Delta\text{slope}_{\overline{age}-\Delta age}$= -0.04, 95% CI [-0.06, -0.01], *Post.Prob* = 0.99). Here, the slope for the between-individual age effect progressed in the opposite direction (e.g., juvenile survival; *Appendix S1*: *Table S5*) or was considerably flatter (e.g., development time and adult lifespan; *Appendix S1: Table S12 and Table S20*) than the within-individual age estimate. Thus, if we did not control between-individual heterogeneity in the parents mean age at reproduction, then this could have masked the within-parent trajectories we observed.

879 # Discussion

880 Our results question the universality of parental effect senescence and contribute to
881 the growing knowledge that later-conceived offspring exhibit accelerated life-
882 histories (Bouwhuis et al. 2010; Plaistow et al. 2015; Kroeger et al. 2020; Travers et
883 al. 2021; Anderson et al. 2022). Irrespective of their parents' thermal environment,
884 which induced experimental variation in parental mortality rate, the earliest- and
885 latest-conceived offspring appeared demographically distinct. Offspring from old
886 parents accelerated development, eclosed with a smaller adult mass, potentially
887 exhibited improved reproductive investment, and, crucially, displayed an extended
888 adult lifespan, opposite of a Lansing effect. This inverse Lansing effect arose not from
889 a reduced initial frailty, but from a delayed onset and slowed progression of actuarial
890 senescence. Below, we first discuss the lack of temperature induced environmental
891 heterogeneity in our parental age effects, before outlining the following alternate
892 explanations for our findings: do our results indicate constraints imposed by parental
893 senescence or an adaptive late-life shift in the parents' reproductive strategy?

894 **Environmental heterogeneity and parental age effects**

895 Although it was clear that increasing temperatures exacerbated parent mortality, we
896 found no convincing evidence that the parents' thermal environment mediated our
897 parental age effects. According to the metabolic theory of ecology (Brown *et al.*,
898 2004), warming temperatures accelerate metabolic processes in ectotherms, resulting
899 in the excessive generation of damaging reactive oxygen species (ROS; Munch &
900 Salinas, 2009; Burraco et al. 2020). Under chronic exposure to high temperatures, this

901 could have accelerated the parent's rate of actuarial senescence through increased
902 ROS damage to lipids, proteins, and telomeres (Mair et al. 2003; Conti, 2008; Flouris
903 & Piantoni, 2015; Keil et al. 2015; Friesen et al. 2022). Alternatively, temperature
904 may have directly increased the parents' baseline mortality, without accelerating
905 senescence (Bowler & Terblanche, 2008). In the first scenario, we would expect some
906 component of the senescent physiology of old parents to be transferred to the
907 offspring, inducing detrimental age effects (such as the Lansing effect). Here, we
908 should have found evidence for a temperature by parental age interaction on
909 offspring adult lifespan, which is not supported by our data. However, if the Lansing
910 effect is best understood as a manifestation of accelerated actuarial senescence
911 (Kern, 2001), and if increasing temperatures only directly affected the parents
912 baseline mortality, independently of senescence, then this could explain the lack of
913 any environmental variation in our parental age effects. Hence, we believe the most
914 parsimonious explanation for the lack of a temperature mediated parental age effect
915 is that temperature did not increase parental mortality through physiologically
916 accelerated ageing, but rather through a direct environmental effect on age-
917 independent baseline-mortality

918 **Parental age as a constraint: Egg provisioning and resource allocation trade-offs**

919 Our inverse Lansing effect may have arisen from late-life improvements to pre-natal
920 provisioning. Older mothers could have displayed a terminal investment strategy
921 (Clutton-Brock, 1984; Velando et al. 2006; Jehan et al. 2021) or improved in body
922 condition with age (Stahlschmidt et al. 2013). Under this scenario, older mothers
923 would provision their eggs with high concentrations of macro and micronutrients

924 (e.g., lipids, carbohydrates, proteins, vitamins), thereby improving offspring survival
925 (Clutton-Brock, 1984; Mousseau & Fox, 1998; Gilbert & Manica, 2010). However, this
926 explanation appears unlikely given the substantial evidence for reproductive
927 senescence across insects (Begon & Parker, 1986; Fox & Czesak, 2000; Rivero et al.
928 2001; Fox et al. 2003; Bloch Qazi et al. 2017; Muller et al. 2017; Noguera, 2021), and
929 from our own system *(in prep)*. Even in instances where maternal provisioning
930 increases with age, the Lansing effect often remains (Plaistow et al. 2015; Anderson
931 et al. 2022), suggesting any age-dependent improvements in maternal provisioning do
932 not necessarily alleviate, and may even exacerbate, offspring senescence.

933 It is entirely possible that the Lansing effect (or inverse thereof) can arise from the
934 offspring's response to age-dependent changes in maternal pre-natal provisioning,
935 rather than from the inheritance of their old parents' senescent phenotype (Monaghan
936 et al. 2020). In this scenario, parental provisioning modulates how offspring resolve
937 existing life-history trade-offs between the competing demands of growth,
938 reproduction, and somatic maintenance (Stearns, 2000; Anderson et al. 2022).
939 Previous research in *Daphnia pulex* found that later-conceived offspring exhibited a
940 truncated lifespan, yet this was correlated with an increased body size, accelerated
941 growth rate, and improved early-life fecundity (Plaistow et al. 2015). Similar parental
942 age effects on body size, development, and reproductive scheduling have been
943 demonstrated in *Caenorhabditis elegans* and *Daphnia magna* (Travers et al. 2021;
944 Anderson et al. 2022), being attributed to an offspring response to late-life
945 improvements in the mothers' egg quality. This could indicate that increased maternal
946 provisioning initiates an alternate offspring growth trajectory in the latest-conceived

947 offspring, accelerating their growth and increasing their early-life productivity at the
948 consequence of reducing survival (Boersma & Wit, 1997; Metcalfe, 2003; Plaistow et
949 al. 2015). Increased investment in growth or reproduction is well-known to
950 exacerbate senescence, being linked to reduced antioxidant defences (Blount et al.
951 2003, 2016), increased telomere attrition (Hall et al. 2004; Monaghan & Haussmann,
952 2006; Houben et al. 2008; Monaghan, 2014), and heightened oxidative stress
953 responses (Smith et al. 2016; Janssens & Stoks, 2020). Additionally, there may be
954 maintenance costs that scale with size, such as an increased physiological cost of
955 moulting (Hessen & Alstad Rukke, 2000). Yet there is a major caveat to interpreting
956 these studies relative to our findings; the species used display indeterministic growth,
957 with maternal age and adult size being tightly correlated (Bolanowski et al. 1981;
958 Glazier, 1992; Plaistow et al. 2015; Travers et al. 2021). Any improvements in pre-
959 natal provisioning could reflect the larger size of older mothers, rather than late-life
960 changes to provisioning.

961 Within our study we found an inverse pattern; later-conceived offspring exhibited
962 slowed senescence and a smaller adult mass. Could this reflect reductions in pre-natal
963 provisioning by older mothers? Anderson et al. (2022) showed that the Lansing effect
964 can operate in both directions between clones of *D.magna* depending on how
965 maternal provisioning changes with age, with the inverse Lansing effect fully
966 explained by reduced maternal lipid provisioning of the neonates. Brief periods of
967 dietary restriction have been widely demonstrated to extend lifespan, by slowing
968 metabolic processes, decelerating growth rates, and reducing body size (Masoro,
969 2005, 2006; Spindler, 2010; Bock et al. 2019). Thus, our inverse Lansing effect may

970 not arise from parental senescence *per se*; meaning we should expect to find similar
971 patterns in younger mothers if their egg provisioning was somehow experimentally
972 reduced (e.g., through caloric restriction; Gribble et al. 2014; Hibshman et al. 2016).
973 However, under this explanation, we would also expect later-conceived offspring to
974 display both reduced reproductive performance and prolonged development, with this
975 relationship being well established across insects (Zhang et al. 2019). For example,
976 reduced egg provisioning has been frequently demonstrated to delay maturity in
977 *Callosobruchus maculatus* (Fox, 1994; Fox et al. 2003; Lind et al. 2015). We found
978 that increasing parental age did not constrain offspring fecundity (there was even
979 limited evidence for an improvement in hatching success), and the later-conceived
980 offspring accelerated, rather than slowed, their development. Finally, and crucially,
981 this increased caloric restriction does not necessarily explain the ameliorated
982 senescence of later-conceived offspring. For example, in *Drosphilla melanogaster*,
983 dietary restriction does not slow the accumulation of senescent damage, instead
984 extending longevity by lowering the immediate risk of death (i.e., reduced baseline
985 mortality), with individuals carrying no memory of their past restricted feeding when
986 switched to a normal diet (Mair et al. 2003). Thus, it appears unlikely that reduced
987 pre-natal provisioning explains the accelerated life-histories and slowed senescence
988 we observed in latest-conceived offspring.

989 Alternatively, given we did not measure the offspring's initial size at hatching, or
990 subsequent growth rates, it remains possible that offspring from old parents exhibited
991 both slowed growth rates and an earlier adult emergence by eclosing at a smaller
992 body size (Vega-Trejo et al. 2018), a pattern supported by our data (*Figure 3*).

993 Furthermore, we were unable to tease apart the correlation between reproductive
994 history/experience and parental age, which covaried in our experimental design
995 (*Appendix S1: Figure S2*). Given the known importance of reproductive history in
996 influencing physiological condition and future reproductive investment (Bell, 1980;
997 Gershman, 2008; Stiver & Alonzo, 2009), it is possible that any deteriorations in pre-
998 natal provisioning reflect the cost of accumulated reproductive effort rather than an
999 effect of parental age *per se* (Monaghan et al. 2020). Finally, if mothers did
1000 display late-life reductions in pre-natal provisioning, then our inverse Lansing effect
1001 could have instead manifested from the selective disappearance of offspring
1002 phenotypes *within* families (Fox et al. 2003). In this situation, later-conceived
1003 offspring experience heightened mortality during embryogenesis, consequently
1004 leaving a non-random subset of high-quality or long-lived offspring phenotypes in the
1005 hatchling pool (*viability selection hypothesis*; Hadfield, 2008; Mojica & Kelly, 2010).
1006 However, we believe viability selection is unlikely to explain our findings as the initial
1007 mortality in the first four weeks after hatching was independent of parental age
1008 (*Figure 1B*), suggesting no increased mortality rate in frail individuals conceived by
1009 old parents.

1010 **Indirect parental age effects**

1011 It is plausible to speculate that the Lansing effect (and our inverse Lansing effect)
1012 manifests indirectly, through effects of parental age on offspring adult size. This is
1013 supported given the numerous studies finding that parental age is either positively or
1014 negatively correlated with offspring size, yet so far these effects on size are
1015 consistently in the opposite direction to those on adult lifespan (Plaistow et al. 2015;

Noguera, 2021; Anderson et al. 2022). Likewise, those finding that the offspring's adult lifespan is independent of parental age also tend to find the joint absence of any parental age effects on offspring adult size (Tregenza et al. 2026). This relationship has previously been described by Gershman et al. 2022. This suggests that parental age could either directly affect multiple offspring phenotypic traits, including adult size, independently of each other (*Figure 7A*) or operate entirely indirectly, entirely through a direct parental age effect on offspring development time (*Figure 7B*).

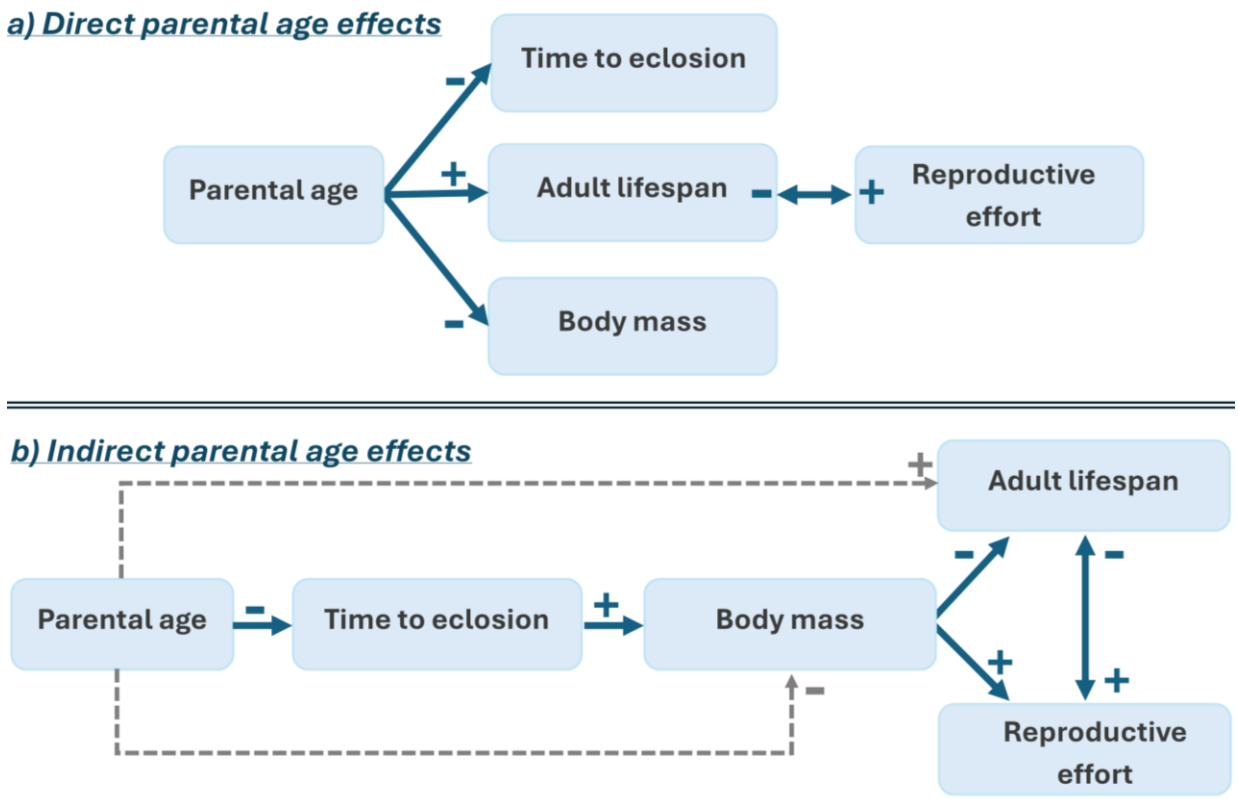


Figure 7. The non-mutually exclusive causal relationships between parental age, offspring development time, and offspring adult lifespan (Cause → Effect). Direct effects are shown as solid blue lines, while indirect effects are shown as grey dashed

1028 lines. The sign describes the direction of the relationship: + = positive relationship, - =
1029 negative relationship. **A) Direct parental age effects.** Under this scenario, parental
1030 age directly affects all offspring phenotypic traits independently. There also exists a
1031 trade-off between reproduction and adult lifespan. **B) Indirect parental age effects**
1032 **on offspring adult size and lifespan, arising from causal effects of parental age on**
1033 **offspring development time.** Here, any effect of parental age on offspring adult size
1034 and adult lifespan operates entirely indirectly, through the direct parental age effect
1035 on the time to eclosion. Diagrams adapted from Gershman et al. (2022).

1036 The pathway to causality in our system may have progressed as follows: first, parental
1037 age could directly accelerate offspring development times, with it being adaptive for
1038 later-conceived offspring to exhibit an earlier adult emergence (e.g., due to seasonal
1039 time constraints; *discussed below*). Second, this accelerated development limits the
1040 time available for somatic growth, with the latest-produced offspring being smaller
1041 on reaching adulthood. Indeed, this positive correlation between development time
1042 and adult size has been well-established across insects (Mousseau & Dingle, 1991;
1043 Roff, 2000). Finally, as later-conceived offspring may eclose with a smaller adult
1044 mass, they could have incurred less damage from ROS during development, thereby
1045 slowing senescence (*growth-lifespan trade-off*; Metcalfe & Monaghan, 2001; Metcalfe,
1046 2003). Here, our inverse Lansing effect arises indirectly, through causal parental age
1047 effects on offspring development time (Gershman et al. 2022). Alternatively, parental
1048 age may have direct detrimental effects on offspring adult longevity, but this Lansing
1049 effect is masked by both direct and indirect (i.e., through development time)
1050 negative effects of parental age on offspring adult size. Although such a causal

pathway seems intuitive, the observed negative parental age effect on adult mass was small. Approximately 20% of the posterior distribution fell within the region of practical equivalence (ROPE). As such, a meaningful proportion of posterior estimates corresponded to a negligible change in adult mass.

It is possible that, alongside their earlier adult emergence, later-conceived offspring improved their early-life fecundity, as is being increasingly evidenced (Bouwhuis et al. 2010; Plaistow et al. 2015; Travers et al. 2021; Dutt & Laird, 2024), but such improvements were partially offset by their smaller adult mass. Across invertebrates, a smaller soma constrains reproduction, owing to how the ovarian tissues scale with somatic size (Honěk & Honek, 1993; Berger et al. 2012; Sturm, 2016). Although we found limited evidence for a positive parental age effect on offspring fecundity, eggs laid by later-conceived offspring may have exhibited improved hatching success. Crucially, we cannot infer whether the reproductive scheduling of later-conceived offspring was shifted to increase early-life productivity, as we only measured reproductive effort from a single mating attempt at an intermediate adult age of ~5 weeks, e.g. when the offspring were neither young nor old. At the very least, increasing parental age did not appear to constrain offspring fecundity and may even improve the offspring's *per-capita* reproductive investment.

Regardless of the causal pathway, our explanation here assumes a direct effect of parental age on offspring development time and early-life fecundity. Therefore, is it adaptive for later-conceived offspring to accelerate their life-histories?

1072 **Adaptive parental age effects: *shifts to a fast pace-of life***

1073 Collectively, our results suggest that later-conceived offspring exhibit a fast pace-of-
1074 life strategy. This phenomenon appears taxonomically widespread, being observed in
1075 duckweed (*Lemna minor;* Dutt & Laird, 2024), black rockfish (*Sebastes melanops*;
1076 Berkeley et al. 2004), great tits (*Parus major;* Bouwhuis et al. 2010), yellow-bellied
1077 marmots (*Marmota flaviventer;* Kroger et al. 2020), Daphnia (*D. pulex* and *D.magna;*
1078 Plaistow et al. 2015; Anderson et al. 2022), Nematodes (*C.elegans;* Travers et al.
1079 2021), crickets (*Gryllus vocalis;* Gershamn et al. 2022*),* milkweed bugs (*Oncopeltus*
1080 *fasciatus;* Phelan & Frumhoff, 1991*),* zig-zag ladybirds (*Menochilus sexmaculatus;*
1081 Singh et al. 2021), and even from Lansing's original findings in *Philodina citrina*
1082 (Lansing, 1947; King, 1983), yet despite this evidence, the predominant perception
1083 remains that parental age negatively affects offspring adult longevity. We argue that
1084 parental age effects on the pace-of-life could be adaptive in the special case of
1085 seasonal insects with a definite environmental end point to reproduction.

1086 For example, it may be adaptive for old parents to produce offspring with accelerated
1087 life-histories if they are more prevalent late in the season (Phelan & Frumhoff, 1991;
1088 Zehnder et al. 2007; Gershman et al. 2022). In this circumstance, seasonal
1089 deteriorations in environmental conditions and increasing resource scarcity limits the
1090 time available for older parents to complete reproduction *("time-stress"* hypothesis;
1091 Gotthard, 2008; Metcalfe et al. 2002). In response to these time-constraints, old
1092 parents may influence offspring phenotypes through one of two pathways; they may
1093 conceive offspring that undergo diapause (Bigelow, 1962; Mousseau & Roff, 1989;
1094 Mousseau & Dingle, 1991) or produce offspring with uninterrupted development, that

exhibit an earlier adult emergence and increased early-life productivity, with these offspring seeing some fitness returns before the season deteriorates (Roff, 1980; Phelan & Frumhoff, 1991; Zehnder et al. 2007). The latter strategy is common within species which survive through the winter as adults rather than arresting development and diapausing as nymphs. Uninterrupted development carries substantial costs; for every directly-developing generation initiated before winter, the length of the season suitable for growth and reproduction increasingly narrows (Nylin et al. 2009). Thus, later-conceived offspring may experience strong selection to accelerate development and redirect investment from growth to current reproduction, resulting in them eclosing at a less-than-optimal size (Roff, 1980; Nylin et al. 1989; Abrams et al., 1996; Pfenning et al. 2007; Larsdotter Mellström et al. 2010). Alternatively, later-conceived offspring may encounter heightened intra-specific competition from their earlier-conceived siblings, who express an age-related competitive advantage (Plaistow et al. 2007; Benton et al. 2008; Travers et al. 2021). The earlier emergence of offspring from young parents creates a growth race among later-conceived offspring, who are then forced to eclose as adults earlier in life (Stearns, 2000; Pásztor et al. 2022). These time constraints on growth may be avoided in species that diapause as nymphs, as they could synchronise their growth in the following year. For example, recent evidence from a wild population of *G.campestris* suggests that increasing parental age has no effect on the offspring's adult size and, subsequently, adult lifespan (Tregenza et al. 2026). However, despite being a close relative of *G.bimaculatus* (hybrids between the two remain fertile; Tyler et al. 2013), *G.campestris* is univoltine, with the nymphs undergoing an obligate diapause at their penultimate instar (Martínez-

1118 Viejo et al. 2024), and the later-conceived offspring here may not experience the
1119 same time limitations on growth and reproduction as in our uninterrupted developing
1120 multivoltine *G.bimaculatus*.

1121 If development time is under strong selection, we would expect to see two patterns;
1122 not just a reduction in mean development time but also reduced variability in
1123 development time. However, despite strong effects of parental age on the average
1124 development time, there was limited evidence that *variation* in offspring
1125 development time changed with parental age. Rather, we found substantial variability
1126 in development time regardless of parental age. This lack of canalisation could
1127 suggest there is little selection for later-conceived offspring to accelerate
1128 development, or, inversely, there may be strong selection on offspring development
1129 time across all parental ages. It is also conceivable that selection actively maintains
1130 variability in development time, irrespective of parental age, especially if the
1131 seasonal environment varies unpredictably (Philippi & Seger, 1989; Simons &
1132 Johnston, 2006; Crean & Marshall, 2009).

1133 **Scope for future research**

1134 Producing offspring with accelerated life-histories late in the season could be
1135 adaptive given that *G.bimaculatus* is a multivoltine species, enabling another fully
1136 functional generation to be realised before conditions deteriorate. From an
1137 evolutionary perspective, an increase in the number of generations per year
1138 facilitates rapid population growth and a greater potential for adaptation (Altermatt,
1139 2010). What remains is in understanding whether these observed non-genetic effects
1140 of parental age can accumulate over multiple generations to influence grandoffspring

1141 phenotypes. Previous evidence for the transgenerational inheritance of parental age
1142 effects remains ambiguous, with some finding immediate parental age effects while,
1143 in others, the effects persist across multiple generations (Bloch Qazi et al. 2017;
1144 Wylde et al. 2019; Travers et al. 2021; Bleu et al. 2022). Evolutionary theory predicts
1145 that if parental age effects do accumulate, then they may alter selection gradients on
1146 late-life reproduction (Kern et al. 2001; Plaistow et al. 2007; Moorad & Nussey, 2016;
1147 Barks & Laird, 2020). Additionally, given our emphasis on the adaptive importance of
1148 parental age effects in life-history evolution, it would be necessary to perform a
1149 similar longitudinal study under a natural environment. Our study population was
1150 maintained under captive conditions, where no predators were present and food was
1151 provided *ad libitum*, so that two of the main drivers of life history variation were
1152 controlled for but not directly examined. The resolution of these life history trade-
1153 offs might work in different, or even opposite, ways when predators and food
1154 limitation are introduced into the system (Zajitschek et al. 2020; Promislow et al.
1155 2022). Currently, little is known on the importance of parental age effects for wild
1156 insect populations, and the two studies that have investigated such effects are
1157 limited in how much they can conclude (Angell et al. 2022; Tregenza et al. 2026).
1158 Both these studies were unable to control potential sources of viability selection
1159 (including effects of parental age on early-life mortality) and only investigated
1160 parental age effects over a single generation. Establishing whether demographic
1161 parental age effects can accumulate across generations, or persist under natural
1162 environments, will be a necessary next step for establishing their ecological
1163 importance and role in the evolution of ageing rates.

Conflict of Interest Statement

The authors declare no conflicts of interest.

Author Contributions

Mark D. Pitt (MDP), Brendan O'Connor (BC), Timothy D. Sheen (TS), Tom Tregenza (TT), and Jelle J. Boonekamp (JJB) conceived and designed the study. MDP, BC, TDS, and JJB collected the data. MDP, BC, and JJB conducted the data analysis, while MDP and JJB wrote the first draft of the manuscript. All authors contributed extensively to revise the manuscript.

Acknowledgements

We would like to thank the Scottish Centre for Ecology and the Natural Environment (SCENE) for facilitating our research. Additionally, we are incredibly grateful to Lucy Winder, Phoebe Kaiser-Wilks, Maddie Inglis, Margot Gibert, Joseph Roy, and Manon Quentin for covering husbandry when additional help was required. This study was supported by the Natural Environment Research Council (NE/X012018/1) and by the Leverhulme Trust (RPG-2024-207).

Ethics Statement

All work involving housing, husbandry, and care of the animals involved in this study was performed at the highest standard possible. Animals were cleaned and fed

weekly, and housed individually from conception, removing welfare concerns regarding overcrowding.

Use of AI statement

Chat GPT-5.3 was used to correct syntax errors in the R-scripts and to help develop code for plotting the model outputs. The corresponding author MDP takes full responsibility over the code and has ensured it remains accurate. No AI was used in any written aspect of this manuscript.

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