Environmental variation mediates the evolution of anticipatory parental effects

Martin I. Lind [1,*], Martyna K. Zwoinska [1], Johan Andersson [1], Hanne Carlsson [1,2], Therese Krieg [1], Tuuli Larva [1] & Alexei A. Maklakov [1,2]

[1] Animal Ecology, Department of Ecology and Genetics, Uppsala University, 752 36 Uppsala, Sweden

[2] School of Biological Sciences, University of East Anglia, Norwich Research Park, Norwich NR4 7TJ, UK

Martin I. Lind - martin.i.lind@gmail.com *Corresponding author

Statement of authorship. MIL and AAM designed the experiment, MIL, JA, HC, TK and TL performed experimental evolution, MIL, MKZ and JA performed phenotypic assays, MIL analysed the data. MIL drafted the manuscript together with AAM. All authors contributed to the revision of the manuscript.

Abstract

Environments vary over time and if this variation is predictable, environments that are similar across generations should favour evolution of anticipatory parental effects to benefit offspring. In contrast, the absence of correlation between parental and offspring environments should select against parental effects. However, experimental evidence is scarce. We investigated the evolution of maternal effects using experimental evolution. Populations of the nematode *Caenorhabditis remanei*, adapted to 20°C, were exposed to a novel temperature (25°C) for 30 generations with either positive or zero correlation between parent and offspring temperature. We found that populations evolving in environments with positive correlations had a positive maternal effect, since they required maternal exposure to 25°C to achieve maximum reproduction and fitness in 25°C. In contrast, populations evolving under zero correlation had lost this positive maternal effect. This shows that parental effects can aid population viability in warming environments. Correspondingly, ill-fitting parental effects can be rapidly lost.

Key words: *Caenorhabditis*, Environmental heterogeneity, Maternal effects, Reproduction, Temperature, Transgenerational plasticity

Introduction

The role of environmental variation in the adaptive expression of phenotypes has gathered considerable interest (Chevin *et al.* 2010; Hollander *et al.* 2015; Donelson *et al.* 2017; Lind & Spagopoulou 2018). Not only is heterogeneity common, it is also predicted to change evolutionary outcomes. While stable environments generally should select for genetic specialisation, environmental heterogeneity can select for environmental input on this process. Variable environments is expected to select for phenotypic plasticity (if environmental cues are reliable) or bet-hedging (if the cues are not reliable) (Moran 1992; Simons 2011). However, a developing organism may not be able to acquire and/or interpret the environmental cues itself; therefore, the parental environment can also function as a developmental cue (Marshall & Uller 2007; Leimar & McNamara 2015). Consequently, recent theory maintains that high environmental correlation between generations can select for adaptive parental effects and/or epigenetic inheritance of an environmentally induced phenotype (Lachmann & Jablonka 1996; Bonduriansky & Day 2009; Rivoire & Leibler 2014; Kuijper & Hoyle 2015; Leimar & McNamara 2015; Uller *et al.* 2015; Proulx & Teotónio 2017), mechanisms collectively referred to as transgenerational plasticity (Donelson *et al.* 2017). The sign of the environmental correlation is expected to result in a similar sign of the parental effect, so that parents can prepare their offspring for the same environment as they are themselves experiencing (positive correlation), the opposite environment (negative correlation) or does not influence the phenotype of their offspring (zero correlation between parent and offspring environments). The exception is constant (perfectly correlated) environments, where genetic specialisation is predicted to evolve (Leimar & McNamara 2015) together with a negative trans-

68 generational effect in order to reduce phenotypic variance (Hoyle & Ezard 2012;
69 Kuijper & Hoyle 2015).
70
71 However, while the theory is well developed, the empirical evidence is mixed. In its
72 most basic form, the theory of anticipatory parental effects predicts that offspring
73 should have higher performance if parental and offspring environments are matching
74 (Marshall & Uller 2007; Burgess & Marshall 2014). There are some striking
75 examples of this (e.g. Gustafsson *et al.* 2005; Galloway & Etterson 2007; Jensen *et al.*
76 2014; Kishimoto *et al.* 2017; Ryu *et al.* 2018; Toyota *et al.* 2019), but across studies
77 there is only weak support for this prediction in natural systems, and the effects are
78 small compared to the direct effects of offspring environment (reviewed in Uller *et al.*
79 2013). Since environmental predictability across generations is seldom quantified, one
80 reason for the scarcity of clear examples of adaptive parental effects is that
81 environments may not often be correlated across generations, and, therefore, provide
82 little opportunity for selection for such anticipatory effects (Burgess & Marshall
83 2014). Even if natural environments are correlated, the evidence for stronger parental
84 effects in more stable environments is mixed, and varies between traits (Walsh *et al.*
85 2016). Thus, there is a call for studies estimating parent and offspring fitness when the
86 environmental predictability between generations are well known, and maternal
87 effects are expected to be under selection (Burgess & Marshall 2014).
88
89 Direct experimental evidence for the evolution of positive anticipatory parental effects
90 of an environmentally induced parental phenotype is currently lacking. On the other
91 hand, if parents and offspring live in negatively correlated environments, the parental
92 phenotype should not be inherited, but the parents can still anticipate the offspring

93 environment. As such, the evolution of a negative parental effect could be adaptive, a
94 prediction that has recently received experimental support in a study by Dey *et al.*
95 (Dey *et al.* 2016), who found that populations of the nematode *Caenorhabditis*
96 *elegans* that evolved under strictly alternating hypoxia-normoxia conditions in every
97 generation evolved a negative maternal provisioning effect. This suggests an adaptive
98 benefit of maternal effects when the maternal environment is a strong cue for the
99 offspring environment (in this case a perfect negative correlation), as predicted by
100 theory (Uller *et al.* 2015; Proulx & Teotónio 2017). More generally, theory also
101 predicts that positive trans-generational correlations would result in the evolution of a
102 positive parental effect and, importantly, if the environmental state is uncorrelated
103 across generations, parental effects would be maladaptive and selected against
104 (Lachmann & Jablonka 1996; Kuijper & Hoyle 2015; Leimar & McNamara 2015;
105 Uller *et al.* 2015). The latter is considered a reason why adaptive parental effects are
106 generally weak (Uller *et al.* 2013). However, these scenarios have not been
107 investigated experimentally. Moreover, previous studies on evolution of parental
108 effects under environmental heterogeneity have investigated only the case of non-
109 overlapping generations (Dey *et al.* 2016). Most natural populations have however
110 overlapping generations and age structure, which can influence evolution in both
111 stable and heterogeneous environments, especially with respect to the evolution of life
112 history strategies (Cotto & Ronce 2014).
113
114 Taken together, environmental heterogeneity and environmental correlations over
115 generations predict the adaptive value of parental effects (Lachmann & Jablonka
116 1996; Kuijper & Hoyle 2015; Uller *et al.* 2015). We set out to test this using
117 experimental evolution in the dioecious free-living nematode *Caenorhabditis*

118 *remanei*, adapting to different temperature regimes. Genetically heterogeneous
119 populations, previously adapted to 20°C, were evolving for 30 generations in control
120 conditions or adapting to 25°C, in either constant 25°C, increased warming to 25°C or
121 a heterogeneous environment with fluctuating temperatures. We found positive
122 anticipatory maternal effects on reproduction in populations evolving in environments
123 that were positively correlated across generations. Moreover, we found the evolution
124 of a reduced maternal effect on reproduction in heterogeneous environments where
125 parent and offspring environments were not correlated during experimental evolution.
126

127 **Material and methods**
128

129 *Experimental evolution*
130 As founder population, we used the wild-type SP8 strain of *C. remanei*, obtained from
131 N. Timmermeyer at the Department of Biology, University of Tübingen, Germany.
132 This strain was created by crossing three wild-type isolates of *C. remanei* (SB146,
133 MY31, and PB206), harbour substantial standing genetic variation for life-history
134 traits (Chen & Maklakov 2012; Lind *et al.* 2017), and has been lab-adapted to 20°C
135 for 15 generations prior to setup of experimental evolution.

136 Experimental evolution was conducted for 30 generations in three climate cabinets;
137 one set to 20°C, one to 25°C and one with a slowly increasing temperature (see
138 below). Five selection regimes were used. *Control 20°C* was experiencing 20°C for
139 30 generations, and *Warm 25°C* was experiencing 25°C for 30 generations. *Increased*
140 *warming* started in 20°C, the cabinet temperature was then raised by 0.1°C every 2.13
141 day (rounded to whole days) to reach 25°C the last day of selection. *Slow temperature*
142 *cycles* spend their first 10 generations in 20°C, were then moved to the 25°C cabinet

143 for 10 generations, to finish the last 10 generations in the 20°C cabinet. Finally, the
144 *Fast temperature cycles* regime were moved between 20°C and 25°C every second
145 generation, thus experiencing 15 temperature shifts.

146 Generation time in 20°C and 25°C was defined as the average difference in age
147 between parents and offspring (Charlesworth 1994) and was calculated from the life-
148 table of age-specific reproduction and survival by solving the Euler-Lotka equation
149 (Stearns 1992; Charlesworth 1994) with trial data from the SP8 lines, and was 4.0
150 days in 20°C and 3.4 days in 25°C. This resulted 120 days of selection for *Control*
151 *20°C*, 114 days for *Slow temperature cycles*, 110 days for *Increased warming* and
152 *Fast temperature cycles* and 101 days for *Warm 25°C*. For the two temperature cycle
153 treatments, the worms spent shorter chronological time in 25°C than in 20°C, because
154 of the faster generation time in 25°C. This ensured equal exposure to the two
155 temperatures over biological time.

156 Experimental evolution was conducted on 92 mm NGM-plates (Stiernagle 2006) and
157 to combat infections the agar and bacterial LB also contained the antibiotics
158 kanamycin and streptomycin, and the fungicide nystatin (Lionaki & Tavernarakis
159 2013; Lind *et al.* 2016). The plates were seeded with 2mL of the antibiotic resistant *E.*
160 *coli* strain OP50-1 (pUC4K) obtained from J. Ewbank at the Centre d'Immunologie
161 de Marseille- Luminy, France. To keep worm populations age-structured in
162 overlapping generations, the lines were always kept in experimental growth face by
163 cutting out a bit of agar containing 150 individuals of mixed ages and transferring this
164 to freshly seeded plates. Transfer was conducted when needed (every 1-2 day), always
165 before food was finished. Six independent replicate populations of each selection
166 treatment were set up, resulting in a total of 30 populations. All populations were

167 expanded for two generations and frozen after 30 generations.

168

169 *Phenotypic assays*

170 Before assays, worms were recover from freezing and grown 2 generations in
171 common garden, each generation synchronized by bleaching, a standard procedure
172 that kills all life-stages but eggs (Stiernagle 2006). The common garden temperature
173 was 20°C or 25°C (see below).

174

175 Fitness assays were performed to test for local adaptation to the selective regime and
176 the evolution of adaptive trans-generational effects. We therefore carried out three
177 assays, by varying parental temperature (the 2 generations of common garden after
178 defrosting the lines) and testing temperature for offspring. The *20°C - 20°C assay* had
179 both common garden and testing in 20°C. This is the environment the *Control 20°C*
180 lines have experienced. Likewise, in the *25°C - 25°C assay* both parents and testing
181 worms experience 25°C, which is the selective environment for *Warm 25°C* and very
182 close to the final environment for *Increased warming*. Finally, the *20°C - 25°C assay*
183 have 20°C as parental temperature, while the testing worms have their whole
184 development in 25°C. This assay represents strong temperature fluctuations between
185 generations, which is the selective environments for the *Fast temperature cycle* lines,
186 and by comparing this assay to the *25°C - 25°C assay* we can estimate the importance
187 of trans-generational effects on fitness when adapting to a novel environment.

188

189 The assays were initiated by synchronised egg-laying in the testing temperature by 40
190 females of each population. After 5h, females were killed by bleaching, and setup of

L4 larvae was initiated 39h later (in 25°C) or 50h later (in 20°C), due to temperature-specific development time. The setup consisted of 8 testing females per plate, together with the same number of background males from the SP8 line. Sex ratio was kept 1:1 throughout the experiment by adjusting the number of males to match the number of females alive. Age-specific fecundity was measured by each day allowing the females 3h of egg-laying on an empty plate, where after the females were returned to a new plate (together with the males) and the number of hatched offspring on the egg-laying plate were counted 2 days later. The exact time the females were added to and removed from each plate was noted, and the number of offspring was corrected by exact number of minutes available for egg laying, and the number of females alive. Thus, we did not collect individual level data on total reproduction, but daily snapshot, in order to increase the number of individuals assayed and improve the reproduction estimate of each population. Daily reproduction was collected until all reproduction had ceased. Four replicate plates of each population was set up, and for the *20°C - 20°C* and *20°C - 25°C* assays the replicates were evenly split between two climate cabinets per temperature. However, for logistical reasons, the *20°C - 25°C assay* was reduced. We excluded the *Slow temperature cycle* treatment from this assay, and unfortunately we lost two *Warm 25°C* populations during common garden (due to overcrowding and subsequent starving, which is known to induce epigenetic effects (Rechavi *et al.* 2014) and therefore these populations were excluded), leaving us with 4 replicate population of this treatment. This resulted in 30 replicate populations and 960 female worms for the *20°C - 20°C* and *20°C - 25°C* assays, and 23 replicate populations and 736 female worms for the *25°C - 25°C assay*.

Statistical analyses

The age-specific reproduction data was analysed as rate-sensitive individual fitness λ_{ind} (Brommer *et al.* 2002) as well as the total reproduction of each replicate plate, adjusted to the number of females present each day. Individual fitness encompasses the timing and number of offspring and is analogous to the intrinsic rate of population growth (Stearns 1992) and was calculated by solving the Euler-Lotka equation for each replicate plate. Individual fitness and total reproduction in 20°C was analysed in separate mixed effect models with election treatment as fixed effect and population and cabinet as random effects. Both response variables were log-transformed before analysis. In 25°C, individual fitness and total reproduction was analysed with selection treatment and parental temperature as crossed fixed effects, and replicate population as random effect. Since the *25°C - 25°C assay* was conducted in only one cabinet, and moreover the *Slow temperature cycle* treatment was not run, the random effect of cabinet was excluded from the models, as was the *Slow temperature cycle* treatment.

Results

For individual fitness (λ_{ind}) in 25°C, we found a significant selection regime × parental temperature interaction (Selection regime: $\chi^2 = 1.302$, df = 3, p = 0.729; Parental temperature: $\chi^2 = 0.114$, df = 1, p = 0.736, Selection regime × Parental temperature: $\chi^2 = 15.562$, df = 3, p = 0.001). This interaction was caused by significantly opposite slope for *Fast temperature cycles* compared to *Increased warming* and *Warm 25°C*, with highest fitness in 25°C for *Fast temperature cycles*

241 when parents were grown in 20°C, while highest fitness in 25°C for *Increased*
242 *warming* and *Warm 25°C* was achieved when their parents were also grown in 25°C
243 (Figure 1A).
244
245 A significant selection regime × parental temperature interaction was also found for
246 total reproduction in 25°C (Selection regime: $\chi^2 = 0.995$, df = 3, p = 0.802; Parental
247 temperature: $\chi^2 = 41.373$, df = 1, p < 0.001, Selection regime × Parental temperature:
248 $\chi^2 = 10.747$, df = 3, p = 0.013), caused by a much weaker positive effect of parental
249 exposure to 25°C for *Fast temperature cycles* than for the other selection regimes
250 (Figure 1B). Finally, we found a fitness cost of adaptation, since all lines had reduced
251 individual fitness (λ_{ind}) in 20°C relative to the *Control 20°C* lines ($\chi^2 = 19.799$, df = 4,
252 p < 0.001, Figure 2). The cost was however not detected in total reproduction ($\chi^2 =$
253 2.238, df = 4, p = 0.692, Supplementary figure 1).
254
255
256 **Discussion**
257
258 The degree of environmental variation can influence the expression of phenotypes
259 (Moran 1992; Uller *et al.* 2015; Proulx & Teotónio 2017). In addition to genetic
260 specialisation, the phenotype can match the environment either by phenotypic
261 plasticity, where the offspring matches its development to the current environment, or
262 by anticipatory parental effects, where the offspring non-genetically inherit the
263 parents phenotype to match its environment, thus improving offspring performance if
264 parent and offspring environments are matching (Marshall & Uller 2007; Donelson *et*
265 *al.* 2017). While within-generation phenotypic plasticity is common (DeWitt &

Scheiner 2004), parental effects seem, in contrast, to be generally weak (Uller *et al.* 2013), despite some well known examples (Gustafsson *et al.* 2005; Galloway & Etterson 2007; Jensen *et al.* 2014; Kishimoto *et al.* 2017; Ryu *et al.* 2018; Toyota *et al.* 2019). Since trans-generational effects should evolve only if there is a strong correlation between parent and offspring environments (Kuijper & Hoyle 2015; Leimar & McNamara 2015; Uller *et al.* 2015), it is possible that environments generally are not highly correlated between generations, thus explaining why such anticipatory effects are uncommon (Uller *et al.* 2013). However, the environmental predictability is seldom investigated in studies of parental effects (Burgess & Marshall 2014). Therefore, we investigated whether the degree of temporal environmental variation, as well as the correlation between parent and offspring environment, influenced the evolution of maternal effects.

We found that the presence of environmental variation mediated the evolution of maternal effects on reproduction and individual fitness (λ_{ind}) in *C. remanei* nematode worms adapting to a novel and stressful warm temperature (25°C) for 30 generations (see predictions and findings summarized in Supplementary table 1). For all populations evolving in stable or slowly increasing temperature (*Control 20°C*, *Warm 25°C*, *Increased warming*), a strong positive maternal effect on reproductive output resulted in an increased offspring production in 25°C when the parents were also cultured in 25°C and not in 20°C. Since these populations have evolved in environments with high and positive environmental correlations over generations, trans-generational effects are adaptive and predicted by theory (Mousseau & Fox 1998; Kuijper & Hoyle 2015; Leimar & McNamara 2015; Uller *et al.* 2015). This result is also in agreement with a recent study by Dey *et al.* (2016) who found the

291 evolution of an anticipatory negative maternal effect in *C. elegans* evolving in
292 perfectly negatively correlated environments. Our finding of positive anticipatory
293 maternal effects in positively correlated environments highlights the importance of
294 experimental evolution studies with known environmental correlations to study the
295 evolution of adaptive trans-generational effects.
296
297 In contrast to environments with high positive correlations, the *Fast temperature*
298 *cycles* populations evolved in a fluctuating environment where the temperature
299 changed every second generation. Thus, the next generation would with equal
300 likelihood be exposed to the same or a different temperature as the parents, resulting
301 in zero correlation between parent and offspring environments. In this environment,
302 trans-generational effects are not considered adaptive (Mousseau & Fox 1998; Uller
303 *et al.* 2015), and, in agreement with the theory, we found a loss of the positive
304 maternal effect, since the reproductive output in 25°C of these lines was independent
305 of the environment their parents. The adaptive value of these differences in parental
306 effects is illustrated in the individual fitness (λ_{ind}) of the different selection treatments.
307 *Fast temperature cycles* populations had highest λ_{ind} in 25°C only when the parents
308 were cultured for two generations in 20°C, a situation mimicking the fluctuating
309 environments they were exposed to during evolution. Although adaptive, this should
310 be defined as negative maternal on λ_{ind}, which is not predicted by theory. Thus,
311 maternal effects on reproduction and λ_{ind} does not follow the same pattern for the *Fast*
312 *temperature cycles*, but importantly, none of the measures show the positive maternal
313 effects present in lines from more positively correlated environments (figure 1). In
314 contrast, populations adapting to more or less stable warm temperatures (*Warm 25°C*
315 and *Increased warming*) improved λ_{ind} when parents were also cultured in 25°C,

316 which mimics stable temperatures over generations and a positive maternal effect on
317 λ_{ind}. Whether the negative maternal effect on fitness in the *Fast temperature cycles* is
318 a result of the fact that the assays with parents and offspring in 25°C represent a
319 stability not experienced evolutionary by the *Fast temperature cycles* populations
320 (two generations common garden for the parents and one generation offspring testing
321 equals to three generations in the same environment), or is a general response when
322 parent and offspring environment is not matching is unknown.
323
324 In addition, we also found a fitness cost of adaptation, since all populations evolving
325 in new environments had lower λ_{ind} in the original environment (20°C), compared to
326 the *Control 20°C* populations. Moreover, although *Control 20°C* showed a positive
327 maternal effect on reproduction, parental exposure to 25°C did not improve their
328 individual fitness, suggesting that an evolutionary history in 25°C is needed for
329 maximum fitness in this temperature.
330
331 While the positive maternal effect present in both stable (*Control 20°C*, *Warm 25°C*)
332 and slowly increasing (*Increased warming*) temperature regimes is anticipated when
333 there is positive autocorrelation between parent and offspring environments
334 (Lachmann & Jablonka 1996; Uller *et al.* 2015), perfectly stable environments are
335 actually predicted to select for negative trans-generational effects (Hoyle & Ezard
336 2012; Kuijper & Hoyle 2015). When a population is well adapted to a stable
337 optimum, a negative maternal effect reduces phenotypic variance between
338 generations. However, we find no support for this prediction, since both treatments in
339 stable environments (*Control 20°C*, *Warm 25°C*) showed a positive maternal effect,
340 even after 30 generations in stable conditions. It is however possible that these lines

still show transient dynamics, since a positive trans-generational effect is predicted to evolve as a transient response when experiencing a novel environment (Kuijper & Hoyle 2015), in a similar way to how phenotypic plasticity is predicted to aid adaptation to new environments (Price *et al.* 2003; Lande 2009; Chevin *et al.* 2010; Coulson *et al.* 2017). It could possibly be argued that the positive trans-generational effect is non-adaptive, caused by the lines being maladapted in 20°C and therefore producing low-quality offspring in this temperature. However, the fact that the *Control 20°C* lines, who have highest fitness in 20°C and who have not experienced 25°C for at least 45 generations show positive maternal effects on reproduction of parental exposure to 25°C argues against a non-adaptive explanation and instead reinforces the view that all lines from stable and slowly changing environment has an adaptive positive maternal effect. In agreement with this, exposing a laboratory strain of *C. elegans* to 25°C (Schott *et al.* 2014) or to starvation (Rechavi *et al.* 2014) results in heritable changes in gene expression, directed by inherited small RNA (Rechavi *et al.* 2014; Schott *et al.* 2014; Houri-Zeevi & Rechavi 2017), which further suggests that transgenerational plasticity such as maternal effects and epigenetic inheritance is important in *Caenorhabditis* nematodes.

We found that the environmental correlation between generations is driving the evolution of anticipatory maternal effects, and the experimental design assured low within-generation heterogeneity. While stable and slowly changing environments select for positive anticipatory maternal effects, environments that fluctuate with no correlation between generations select against parental influence on offspring phenotype. This is the first empirical study that investigates the evolutionary loss of anticipatory maternal effect, which follow theoretical predictions (Kuijper & Hoyle

366 2015; Uller *et al.* 2015) and suggest that one reason for the weak effects of parent
367 environment on offspring phenotype in natural systems (Uller *et al.* 2013) could be
368 that natural environments are not always temporally correlated across generations.
369 While most examples of positive parental effects comes from systems with short life-
370 cycles such as nematodes (Kishimoto *et al.* 2017), daphniids (Gustafsson *et al.* 2005;
371 Toyota *et al.* 2019), annelids (Jensen *et al.* 2014) and herbs (Galloway & Etterson
372 2007), it is also been found in fish where the generation time span years (Ryu *et al.*
373 2018). However, when investigating maternal effects in *Daphnia* from natural
374 populations with different degree of variation in predation intensity, Walsh *et al.*
375 (2016) found some support for stronger positive maternal effects in population from
376 more stable environments, but the effect differed between traits and no effect was
377 found on reproduction. Nevertheless, these types of studies where the environmental
378 predictability is known are vital for our understanding of the selection pressures
379 resulting in the presence or absence of adaptive maternal effects in natural populations
380 (Burgess & Marshall 2014), where deconstruction of environmental predictability to
381 seasonality and environmental colour noise (following Marshall & Burgess 2015) is a
382 very promising approach.
383
384 On-going climate change is not only resulting in warmer temperatures, but also
385 increased temperature variation, which can impact both the ecology and evolution of
386 populations and species (Vasseur *et al.* 2014; Vázquez *et al.* 2017), and trans-
387 generational acclimation can be an important response to deal with a warming climate
388 (Donelson *et al.* 2017; Ryu *et al.* 2018). We show that environmental heterogeneity
389 drives the evolution of maternal effects, and support the theoretical predictions
390 (Lachmann & Jablonka 1996; Uller *et al.* 2015) that the correlation between parent

391 and offspring environment is the driver of the evolution of transgenerational
392 plasticity.
393
394 **Acknowledgement**
395 This work was funded by the Swedish Research Council [grant number 2016-05195
396 to MIL, grant number 621-2013-4828 to AAM] and the European Research Council
397 [grants AGINGSEXDIFF and GERMLINEAGEINGSOMA to AAM]. We thank
398 Frank Johansson, Anssi Laurila, Irja Ida Ratikainen and Claus Rüffler for discussions.
399
400 **Competing interests**
401 The authors report no competing interests.
402
403 **Materials & Correspondence**
404 Requests for material and correspondence should be addressed to MIL.
405
406 **References**
407 Bonduriansky, R. & Day, T. (2009). Nongenetic inheritance and its evolutionary
408 implications. *Annu. Rev. Ecol. Evol. Syst.*, 40, 103–125.
409 Brommer, J.E., Merilä, J. & Kokko, H. (2002). Reproductive timing and individual
410 fitness. *Ecol. Lett.*, 5, 802–810.
411 Burgess, S.C. & Marshall, D.J. (2014). Adaptive parental effects: the importance of
412 estimating environmental predictability and offspring fitness appropriately.
413 *Oikos*, 123, 769–776.
414 Charlesworth, B. (1994). *Evolution in age-structured populations*. 2nd edn.
415 Cambridge University Press, New York, NY, USA.

416 Chen, H. & Maklakov, A.A. (2012). Longer life span evolves under high rates of
417 condition-dependent mortality. *Curr. Biol.*, 22, 2140–2143.
418 Chevin, L.-M., Lande, R. & Mace, G.M. (2010). Adaptation, plasticity, and extinction
419 in a changing environment: towards a predictive theory. *PLoS Biol*, 8,
420 e1000357.
421 Cotto, O. & Ronce, O. (2014). Maladaptation as a source of senescence in habitats
422 variable in space and time. *Evolution*, 68, 2481–2493.
423 Coulson, T., Kendall, B.E., Barthold, J., Plard, F., Schindler, S., Ozgul, A., *et al.*
424 (2017). Modeling adaptive and nonadaptive responses of populations to
425 environmental change. *Am. Nat.*, 190, 313–336.
426 DeWitt, T.J. & Scheiner, S.M. (2004). *Phenotypic plasticity: functional and*
427 *conceptual approaches*. Oxford University Press, New York, NY, USA.
428 Dey, S., Proulx, S.R. & Teotónio, H. (2016). Adaptation to temporally fluctuating
429 environments by the evolution of maternal effects. *PLoS Biol.*, 14, e1002388.
430 Donelson, J.M., Salinas, S., Munday, P.L. & Shama, L.N.S. (2017). Transgenerational
431 plasticity and climate change experiments: Where do we go from here? *Glob.*
432 *Change Biol.*, 24, 13–34.
433 Galloway, L.F. & Etterson, J.R. (2007). Transgenerational plasticity is adaptive in the
434 wild. *Science*, 318, 1134–1136.
435 Gustafsson, S., Rengefors, K. & Hansson, L.-A. (2005). Increased consumer fitness
436 following transfer of toxin tolerance to offspring via maternal effects. *Ecology*,
437 86, 2561–2567.
438 Hollander, J., Snell-Rood, E. & Foster, S. (2015). New frontiers in phenotypic
439 plasticity and evolution. *Heredity*, 115, 273–275.

Houri-Zeevi, L. & Rechavi, O. (2017). A matter of time: small RNAs regulate the duration of epigenetic inheritance. *Trends Genet.*, 33, 46–57.

Hoyle, R.B. & Ezard, T.H.G. (2012). The benefits of maternal effects in novel and in stable environments. *J. R. Soc. Interface*, 9, 2403–2413.

Jensen, N., Allen, R.M. & Marshall, D.J. (2014). Adaptive maternal and paternal effects: gamete plasticity in response to parental stress. *Funct. Ecol.*, 28, 724–733.

Kishimoto, S., Uno, M., Okabe, E., Nono, M. & Nishida, E. (2017). Environmental stresses induce transgenerationally inheritable survival advantages via germline-to-soma communication in *Caenorhabditis elegans*. *Nat. Commun.*, 8, 14031.

Kuijper, B. & Hoyle, R.B. (2015). When to rely on maternal effects and when on phenotypic plasticity? *Evolution*, 69, 950–968.

Lachmann, M. & Jablonka, E. (1996). The inheritance of phenotypes: an adaptation to fluctuating environments. *J. Theor. Biol.*, 181, 1–9.

Lande, R. (2009). Adaptation to an extraordinary environment by evolution of phenotypic plasticity and genetic assimilation. *J. Evol. Biol.*, 22, 1435–1446.

Leimar, O. & McNamara, J.M. (2015). The evolution of transgenerational integration of information in heterogeneous environments. *Am. Nat.*, 185, E55–E69.

Lind, M.I., Chen, H., Meurling, S., Guevara Gil, A.C., Carlsson, H., Zwoinska, M.K., *et al.* (2017). Slow development as an evolutionary cost of long life. *Funct. Ecol.*, 31, 1252–1261.

Lind, M.I. & Spagopoulou, F. (2018). Evolutionary consequences of epigenetic inheritance. *Heredity*, 121, 205–209.

464 Lind, M.I., Zwoinska, M.K., Meurling, S., Carlsson, H. & Maklakov, A.A. (2016).
465 Sex-specific trade-offs with growth and fitness following lifespan extension
466 by rapamycin in an outcrossing nematode, *Caenorhabditis remanei*. *J.*
467 *Gerontol. A. Biol. Sci. Med. Sci.*, 71, 882–890.
468 Lionaki, E. & Tavernarakis, N. (2013). Assessing aging and senescent decline in
469 *Caenorhabditis elegans*: cohort survival analysis. In: *Cell Senescence*,
470 Methods in Molecular Biology (eds. Galluzzi, L., Vitale, I., Kepp, O. &
471 Kroemer, G.). Humana Press, New York, pp. 473–484.
472 Marshall, D.J. & Burgess, S.C. (2015). Deconstructing environmental predictability:
473 seasonality, environmental colour and the biogeography of marine life
474 histories. *Ecol. Lett.*, 18, 174–181.
475 Marshall, D.J. & Uller, T. (2007). When is a maternal effect adaptive. *Oikos*, 116,
476 1957–1963.
477 Moran, N.A. (1992). The evolutionary maintenance of alternative phenotypes. *Am.*
478 *Nat.*, 139, 971–989.
479 Mousseau, T.A. & Fox, C.W. (1998). The adaptive significance of maternal effects.
480 *Trends Ecol. Evol.*, 13, 403–407.
481 Price, T.D., Qvarnström, A. & Irwin, D.E. (2003). The role of phenotypic plasticity in
482 driving genetic evolution. *Proc. R. Soc. Lond. Ser. B-Biol. Sci.*, 270, 1433–
483 1440.
484 Proulx, S.R. & Teotónio, H. (2017). What kind of maternal effects can be selected for
485 in fluctuating environments? *Am. Nat.*, 189, E118–E137.
486 Rechavi, O., Houri-Ze'evi, L., Anava, S., Goh, W.S.S., Kerk, S.Y., Hannon, G.J., *et*
487 *al.* (2014). Starvation-induced transgenerational inheritance of small RNAs in
488 C. elegans. *Cell*, 158, 277–287.

489 Rivoire, O. & Leibler, S. (2014). A model for the generation and transmission of
490 variations in evolution. *Proc. Natl. Acad. Sci.*, 111, E1940–E1949.
491 Ryu, T., Veilleux, H.D., Donelson, J.M., Munday, P.L. & Ravasi, T. (2018). The
492 epigenetic landscape of transgenerational acclimation to ocean warming. *Nat.*
493 *Clim. Change*, 8, 504–509.
494 Schott, D., Yanai, I. & Hunter, C.P. (2014). Natural RNA interference directs a
495 heritable response to the environment. *Sci. Rep.*, 4, 7387.
496 Simons, A.M. (2011). Modes of response to environmental change and the elusive
497 empirical evidence for bet hedging. *Proc. R. Soc. Lond. B Biol. Sci.*, 278,
498 1601–1609.
499 Stearns, S.C. (1992). *The evolution of life histories*. Oxford University Press, New
500 York, NY, USA.
501 Stiernagle, T. (2006). Maintenance of C. elegans. *WormBook Online Rev. C Elegans*
502 *Biol.*
503 Toyota, K., Cuenca, M.C., Dhandapani, V., Suppa, A., Rossi, V., Colbourne, J.K., *et*
504 *al.* (2019). Transgenerational response to early spring warming in *Daphnia*.
505 *Sci. Rep.*, 9, 4449.
506 Uller, T., English, S. & Pen, I. (2015). When is incomplete epigenetic resetting in
507 germ cells favoured by natural selection? *Proc R Soc B*, 282, 20150682.
508 Uller, T., Nakagawa, S. & English, S. (2013). Weak evidence for anticipatory parental
509 effects in plants and animals. *J. Evol. Biol.*, 26, 2161–2170.
510 Vasseur, D.A., DeLong, J.P., Gilbert, B., Greig, H.S., Harley, C.D.G., McCann, K.S.,
511 *et al.* (2014). Increased temperature variation poses a greater risk to species
512 than climate warming. *Proc. R. Soc. B Biol. Sci.*, 281, 20132612.

513 Vázquez, D.P., Gianoli, E., Morris, W.F. & Bozinovic, F. (2017). Ecological and
514 evolutionary impacts of changing climatic variability. *Biol. Rev.*, 92, 22–42.
515 Walsh, M.R., Castoe, T., Holmes, J., Packer, M., Biles, K., Walsh, M., *et al.* (2016).
516 Local adaptation in transgenerational responses to predators. *Proc R Soc B*,
517 283, 20152271.
518
519

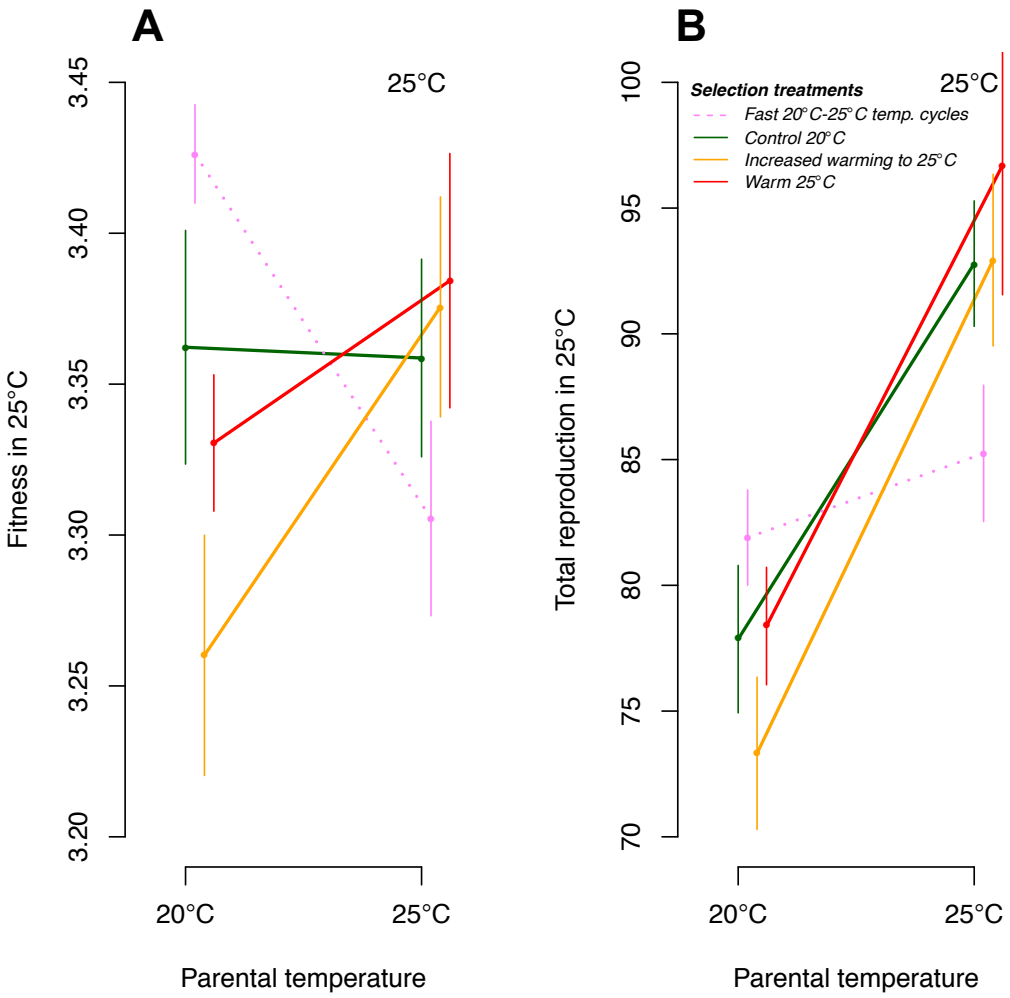
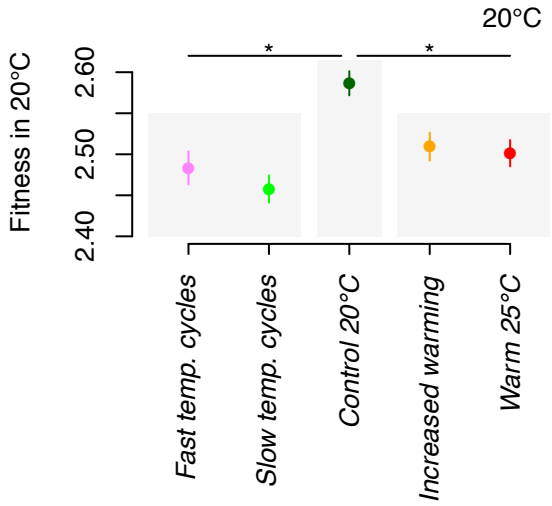


Figure 1. The effect of parental temperature on fitness and reproduction in 25°C. Individual fitness λ_{ind} (A) and total reproduction (B) in 25°C when parent were raised for 2 generations in either 20°C or 25°C. Symbols represent selection treatments (mean ± SE). *Control 20°C* and *Warm 25°C* have spent 30 generations in 20°C or 25°C, respectively. *Increased warming* has been subjected to slowly increased temperatures, starting in 20°C and reaching 25°C at generation 30. *Fast temperature* cycles have spent two generations in 20°C, followed by two generations in 25°C, this cycle has then been repeated for 30 generations.

531



532

533 **Figure 2. Fitness in original environment.** Individual fitness λ_{ind} in 20°C when

534 parents are also grown for two generations in 20°C. Symbols represent selection

535 treatments (mean ± SE). Grey symbolises treatments groups that differ significantly in

536 fitness.

537