Environmental variation mediates the evolution of anticipatory parental effects

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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

Introduction

The role of environmental variation in the adaptive expression of phenotypes has gathered considerable interest [1–4]. 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) [5,6]. 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 [7]. 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 [7–11], mechanisms collectively referred to as transgenerational plasticity [3]. 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 [7] together with a negative trans-generational effect in order to reduce phenotypic variance [10,12].

However, while the theory is well developed, the empirical evidence is mixed. In its most basic form, the theory of anticipatory parental effects predicts that offspring

should have higher performance if parental and offspring environments are matching. There are some striking examples of this (e.g. [13–17]), but across studies there is only weak support for this prediction in natural systems, and the effects are small compared to the direct effects of offspring environment (reviewed in [18]). One reason for the scarcity of clear examples of adaptive parental effects is that environments may not often be correlated across generations, and, therefore, provide little opportunity for selection for such anticipatory effects. Even if natural environments are correlated, the evidence for stronger parental effects in more stable environments is mixed, and varies between traits [19].

Direct experimental evidence for the evolution of positive anticipatory parental effects of an environmentally induced parental phenotype is currently lacking. On the other hand, if parents and offspring live in negatively correlated environments, the parental phenotype should not be inherited, but the parents can still anticipate the offspring environment. As such, the evolution of a negative parental effect could be adaptive, a prediction that has recently received experimental support in a study by Dey *et al.* [20], who found that populations of the nematode *Caenorhabditis elegans* that evolved under strictly alternating hypoxia-normoxia conditions in every generation evolved a negative maternal provisioning effect. This suggests an adaptive benefit of maternal effects when the maternal environment is a strong cue for the offspring environment (in this case a perfect negative correlation), as predicted by theory [11,21]. More generally, theory also predicts that positive trans-generational correlations would result in the evolution of a positive parental effect and, importantly, if the environmental state is uncorrelated across generations, parental effects would be maladaptive and selected against [7,8,10,11]. The latter is considered a reason why

adaptive parental effects effects are generally weak [18]. However, these scenarios have not been investigated experimentally. Moreover, previous studies on evolution of parental effects under environmental heterogeneity have investigated only the case of non-overlapping generations [20]. Most natural populations have however overlapping generations and age structure, which can influence evolution in both stable and heterogeneous environments, especially with respect to the evolution of life history strategies [22].

Taken together, environmental heterogeneity and environmental correlations over generations predict the adaptive value of parental effects [8,10,11]. We set out to test this using experimental evolution in the dioecious free-living nematode *Caenorhabditis remanei*, adapting to different temperature regimes. Genetically heterogeneous populations, previously adapted to 20°C, were evolving for 30 generations in control conditions or adapting to 25°C, in either constant 25°C, increased warming to 25°C or a heterogeneous environment with fluctuating temperatures. We found positive anticipatory maternal effects on reproduction in populations evolving in environments that were positively correlated across generations. Moreover, we found the evolution of a reduced maternal effect on reproduction in heterogeneous environments where parent and offspring environments were not correlated during experimental evolution.

Methodology

Experimental evolution

As founder population, we used the wild-type SP8 strain of *C. remanei*, obtained from N. Timmermeyer at the Department of Biology, University of Tübingen, Germany.

This strain was created by crossing three wild-type isolates of *C. remanei* (SB146, MY31, and PB206), harbour substantial standing genetic variation for life-history traits [23,24], and has been lab-adapted to 20°C for 15 generations prior to setup of experimental evolution.

Experimental evolution was conducted for 30 generations in three climate cabinets; one set to 20°C, one to 25°C and one with a slowly increasing temperature (see below). Five selection regimes were used. *Control 20°C* was experiencing 20°C for 30 generations, and *Warm 25°C* was experiencing 25°C for 30 generations. *Increased warming* started in 20°C, the cabinet temperature was then raised by 0.1°C every 2.13 day (rounded to whole days) to reach 25°C the last day of selection. *Slow temperature cycles* spend their first 10 generations in 20°C, were then moved to the 25°C cabinet for 10 generations, to finish the last 10 generations in the 20°C cabinet. Finally, the *Fast temperature cycles* regime were moved between 20°C and 25°C every second generation, thus experiencing 15 temperature shifts.

Generation time in 20°C and 25°C was defined as the average difference in age between parents and offspring [25] and was calculated from the life-table of age-specific reproduction and survival by solving the Euler-Lotka equation [25,26] with trial data from the SP8 lines, and was 4.0 days in 20°C and 3.4 days in 25°C. This resulted 120 days of selection for *Control 20°C*, 114 days for *Slow temperature cycles*, 110 days for *Increased warming* and *Fast temperature cycles* and 101 days for *Warm 25°C*. For the two temperature cycle treatments, the worms spent shorter chronological time in 25°C than in 20°C, because of the faster generation time in 25°C. This ensured equal exposure to the two temperatures over biological time.

Experimental evolution was conducted on 92 mm NGM-plates [27] and to combat

infections the agar and bacterial LB also contained the antibiotics kanamycin and streptomycin, and the fungicide nystatin [28,29]. The plates were seeded with 2mL of the antibiotic resistant *E. coli* strain OP50-1 (pUC4K) obtained from J. Ewbank at the Centre d'Immunologie de Marseille- Luminy, France. To keep worm populations age-structured in overlapping generations, the lines were always kept in experimental growth face by cutting out a bit of agar containing 150 individuals of mixed ages and transferring this to freshly seeded plates. Transfer was conducted when needed (every 1-2 day), always before food was finished. Six independent replicate populations of each selection treatment were set up, resulting in a total of 30 populations. All populations were expanded for two generations and frozen after 30 generations.

Phenotypic assays

Before assays, worms were recover from freezing and grown 2 generations in common garden, each generation synchronized by bleaching, a standard procedure that kills all life-stages but eggs [27]. The common garden temperature was 20°C or 25°C (see below).

Fitness assays were performed to test for local adaptation to the selective regime and the evolution of adaptive trans-generational effects. We therefore carried out three assays, by varying parental temperature (the 2 generations of common garden after defrosting the lines) and testing temperature for offspring. The *20°C - 20°C assay* had both common garden and testing in 20°C. This is the environment the *Control 20°C* lines have experienced. Likewise, in the *25°C - 25°C assay* both parents and testing worms experience 25°C, which is the selective environment for *Warm 25°C* and very

close to the final environment for *Increased warming*. Finally, the *20°C - 25°C assay* have 20°C as parental temperature, while the testing worms have their whole development in 25°C. This assay represents strong temperature fluctuations between generations, which is the selective environments for the *Fast temperature cycle* lines, and by comparing this assay to the *25°C - 25°C assay* we can estimate the importance of trans-generational effects on fitness when adapting to a novel environment.

The assays were initiated by synchronised egg-laying in the testing temperature by 40 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 [30] 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} [31] 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 [26] 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* when parents were grown in 20°C, while highest fitness in 25°C for *Increased warming* and *Warm 25°C* was achieved when their parents were also grown in 25°C (Figure 1A).

A significant selection regime × parental temperature interaction was also found for total reproduction in 25°C (Selection regime: $\chi^2 = 0.995$, df = 3, p = 0.802; Parental temperature: $\chi^2 = 41.373$, df = 1, p < 0.001, Selection regime × Parental temperature: $\chi^2 = 10.747$, df = 3, p = 0.013), caused by a much weaker positive effect of parental exposure to 25°C for *Fast temperature cycles* than for the other selection regimes (Figure 1B). Finally, we found a fitness cost of adaptation, since all lines had reduced individual fitness (λ_{ind}) in 20°C relative to the *Control 20°C* lines ($\chi^2 = 19.799$, df = 4, p < 0.001, Figure 2). The cost was however not detected in total reproduction ($\chi^2 = 2.238$, df = 4, p = 0.692, Supplementary figure 1).

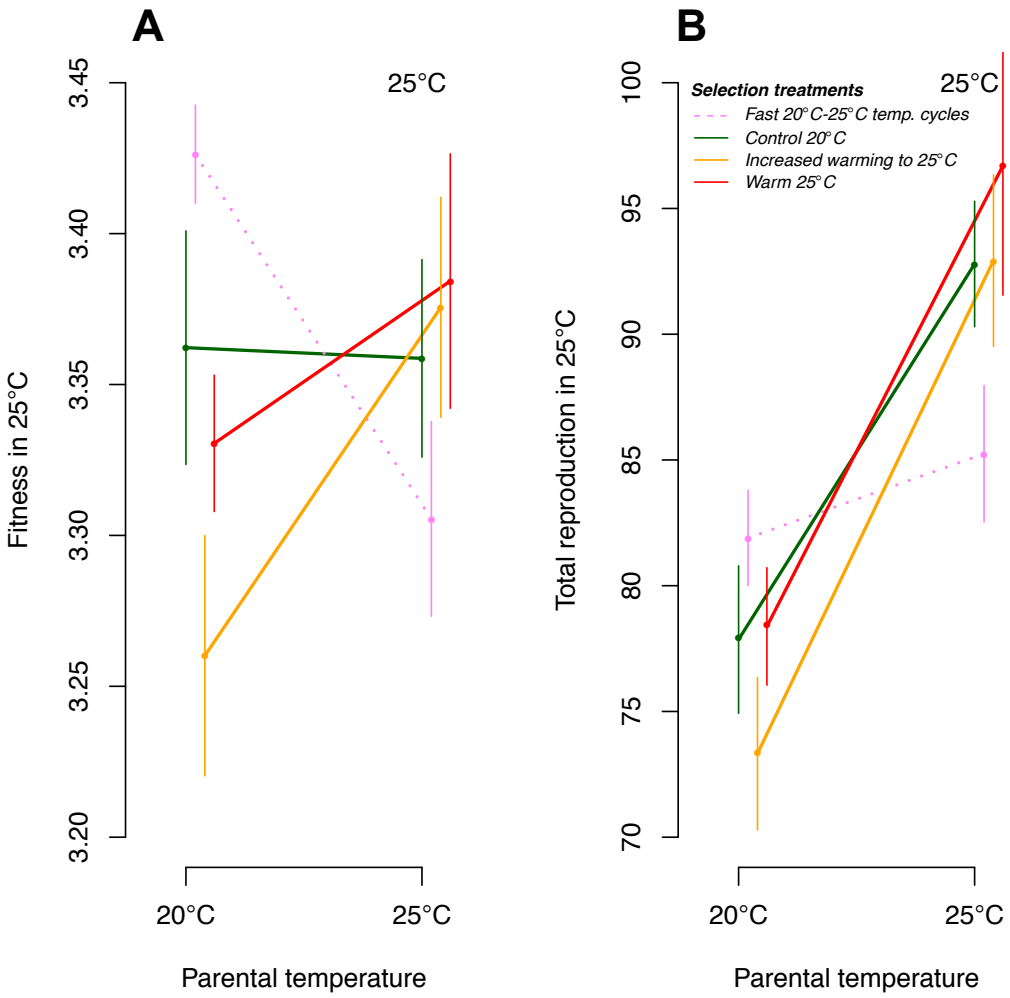

Figure 1. Individual fitness λ_{ind} (A) and total reproduction (B) in 25°C when parent were raised for 2 generations in either 20°C or 25°C. Symbols represent selection treatments (mean ± SE). *Control 20°C* and *Warm 25°C* have spent 30 generations in 20°C or 25°C, respectively. *Increased warming* 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.

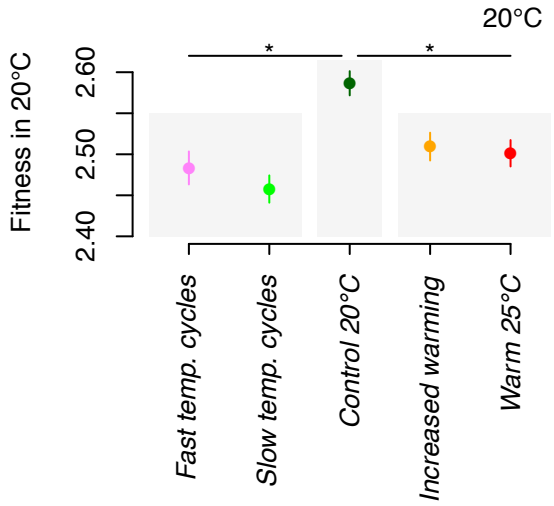

Figure 2. Individual fitness λ_{ind} in 20°C when parents are also grown for two generations in 20°C. Symbols represent selection treatments (mean ± SE).

Discussion

The degree of environmental variation can influence the expression of phenotypes [5,11,21]. In addition to genetic specialisation, the phenotype can match the environment either by phenotypic plasticity, where the offspring matches its development to the current environment, or by anticipatory parental effects, where the offspring non-genetically inherit the parents phenotype to match its environment, thus improving offspring performance if parent and offspring environments are matching [3]. While within-generation phenotypic plasticity is common [32], parental effects seem, in contrast, to be generally weak [18], despite some well known examples [13–17]. Since trans-generational effects should evolve only if there is a strong correlation between

parent and offspring environments [7,10,11], it is possible that environments generally are not highly correlated between generations, thus explaining why such anticipatory effects are uncommon [18]. 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 [7,10,11,33]. This result is also in agreement with a recent study by Dey *et al.* [20] who found the evolution of an anticipatory negative maternal effect in *C. elegans* evolving in perfectly negatively correlated environments. Our finding of positive anticipatory maternal effects in positively correlated environments highlights the importance of experimental evolution studies with known environmental correlations to study the evolution of adaptive trans-generational effects.

In contrast to environments with high positive correlations, the *Fast temperature cycles* populations evolved in a fluctuating environment where the temperature

changed every second generation. Thus, the next generation would with equal likelihood be exposed to the same or a different temperature as the parents, resulting in zero correlation between parent and offspring environments. In this environment, trans-generational effects are not considered adaptive [11,33], and, in agreement with the theory, we found a loss of the positive maternal effect, since the reproductive output in 25°C of these lines was independent of the environment their parents. The adaptive value of these differences in parental effects is illustrated in the individual fitness (λ_{ind}) of the different selection treatments. *Fast temperature cycles* populations had highest λ_{ind} in 25°C only when the parents were cultured for two generations in 20°C, a situation mimicking the fluctuating environments they were exposed to during evolution. Although adaptive, this should be defined as negative maternal on λ_{ind}, which is not predicted by theory. Thus, maternal effects on reproduction and λ_{ind} does not follow the same pattern for the *Fast temperature cycles*, but importantly, none of the measures show the positive maternal effects present in lines from more positively correlated environments (figure 1). In contrast, populations adapting to more or less stable warm temperatures (*Warm 25°C* and *Increased warming*) improved λ_{ind} when parents were also cultured in 25°C, which mimics stable temperatures over generations and a positive maternal effect on λ_{ind}. Whether the negative maternal effect on fitness in the *Fast temperature cycles* is a result of the fact that the assays with parents and offspring in 25°C represent a stability not experienced evolutionary by the *Fast temperature cycles* populations (two generations common garden for the parents and one generation offspring testing equals to three generations in the same environment), or is a general response when parent and offspring environment is not matching is unknown.

In addition, we also found a fitness cost of adaptation, since all populations evolving in new environments had lower λ_{ind} in the original environment (20°C), compared to the *Control 20°C* populations. Moreover, although *Control 20°C* showed a positive maternal effect on reproduction, parental exposure to 25°C did not improve their individual fitness, suggesting that an evolutionary history in 25°C is needed for maximum fitness in this temperature.

While the positive maternal effect present in both stable (*Control 20°C*, *Warm 25°C*) and slowly increasing (*Increased warming*) temperature regimes is anticipated when there is positive autocorrelation between parent and offspring environments [8,11], perfectly stable environments are actually predicted to select for negative trans-generational effects [10,12]. When a population is well adapted to a stable optimum, a negative maternal effect reduces phenotypic variance between generations. However, we find no support for this prediction, since both treatments in stable environments (*Control 20°C*, *Warm 25°C*) showed a positive maternal effect, 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 [10], in a similar way to how phenotypic plasticity is predicted to aid adaptation to new environments [1,34–36]. 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.

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 [10,11] and suggest that one reason for the weak effects of parent environment on offspring phenotype in natural systems [18] could be that natural environments are not always temporally correlated across generations. While most examples of positive parental effects comes from systems with short life-cycles such as nematodes [16], daphniids [13,17] and herbs [14], it is also been found in fish where the generation time span years [15]. However, when investigating maternal effects in *Daphnia* from natural populations with different degree of variation in predation intensity, Walsh et al. [19] found some support for stronger positive maternal effects in population from more stable environments, but the effect differed between traits and no effect was found on reproduction. Nevertheless, these types of studies are vital for our understanding of the selection pressures resulting in the presence or absence of adaptive maternal effects in natural populations.

On-going climate change is not only resulting in warmer temperatures, but also increased temperature variation, which can impact both the ecology and evolution of

populations and species [37,38], and trans-generational acclimation can be an important response to deal with a warming climate [3,15]. We show that environmental heterogeneity drives the evolution of maternal effects, and support the theoretical predictions [8,11] that the correlation between parent and offspring environment is the driver of the evolution of transgenerational plasticity.

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Author contributions

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.

References

1. Chevin, L.-M., Lande, R. & Mace, G. M. Adaptation, plasticity, and extinction in a changing environment: towards a predictive theory. *PLoS Biol* **8**, e1000357 (2010).

2. Hollander, J., Snell-Rood, E. & Foster, S. New frontiers in phenotypic plasticity and evolution. *Heredity* **115**, 273–275 (2015).
3. Donelson, J. M., Salinas, S., Munday, P. L. & Shama, L. N. S. Transgenerational plasticity and climate change experiments: Where do we go from here? *Glob. Change Biol.* **24**, 13–34 (2017).
4. Lind, M. I. & Spagopoulou, F. Evolutionary consequences of epigenetic inheritance. *Heredity* **121**, 205–209 (2018).
5. Moran, N. A. The evolutionary maintenance of alternative phenotypes. *Am. Nat.* **139**, 971–989 (1992).
6. Simons, A. M. Modes of response to environmental change and the elusive empirical evidence for bet hedging. *Proc. R. Soc. Lond. B Biol. Sci.* **278**, 1601–1609 (2011).
7. Leimar, O. & McNamara, J. M. The evolution of transgenerational integration of information in heterogeneous environments. *Am. Nat.* **185**, E55–E69 (2015).
8. Lachmann, M. & Jablonka, E. The inheritance of phenotypes: an adaptation to fluctuating environments. *J. Theor. Biol.* **181**, 1–9 (1996).
9. Rivoire, O. & Leibler, S. A model for the generation and transmission of variations in evolution. *Proc. Natl. Acad. Sci.* **111**, E1940–E1949 (2014).
10. Kuijper, B. & Hoyle, R. B. When to rely on maternal effects and when on phenotypic plasticity? *Evolution* **69**, 950–968 (2015).
11. Uller, T., English, S. & Pen, I. When is incomplete epigenetic resetting in germ cells favoured by natural selection? *Proc R Soc B* **282**, 20150682 (2015).
12. Hoyle, R. B. & Ezard, T. H. G. The benefits of maternal effects in novel and in stable environments. *J. R. Soc. Interface* **9**, 2403–2413 (2012).

13. Gustafsson, S., Rengefors, K. & Hansson, L.-A. Increased consumer fitness following transfer of toxin tolerance to offspring via maternal effects. *Ecology* **86**, 2561–2567 (2005).

14. Galloway, L. F. & Etterson, J. R. Transgenerational plasticity is adaptive in the wild. *Science* **318**, 1134–1136 (2007).

15. Ryu, T., Veilleux, H. D., Donelson, J. M., Munday, P. L. & Ravasi, T. The epigenetic landscape of transgenerational acclimation to ocean warming. *Nat. Clim. Change* **8**, 504–509 (2018).

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

17. Toyota, K. *et al.* Transgenerational response to early spring warming in Daphnia. *Sci. Rep.* **9**, 4449 (2019).

18. Uller, T., Nakagawa, S. & English, S. Weak evidence for anticipatory parental effects in plants and animals. *J. Evol. Biol.* **26**, 2161–2170 (2013).

19. Walsh, M. R. *et al.* Local adaptation in transgenerational responses to predators. *Proc R Soc B* **283**, 20152271 (2016).

20. Dey, S., Proulx, S. R. & Teotónio, H. Adaptation to temporally fluctuating environments by the evolution of maternal effects. *PLoS Biol.* **14**, e1002388 (2016).

21. Proulx, S. R., Teotónio, H., Bonduriansky, R. & Michalakis, Y. What kind of maternal effects can be selected for in fluctuating environments? *Am. Nat.* **189**, E118–E137 (2017).

22. Cotto, O. & Ronce, O. Maladaptation as a source of senescence in habitats variable in space and time. *Evolution* **68**, 2481–2493 (2014).

23. Chen, H. & Maklakov, A. A. Longer life span evolves under high rates of condition-dependent mortality. *Curr. Biol.* **22**, 2140–2143 (2012).

24. Lind, M. I. *et al.* Slow development as an evolutionary cost of long life. *Funct. Ecol.* **31**, 1252–1261 (2017).

25. Charlesworth, B. *Evolution in age-structured populations*. (Cambridge University Press, 1994).

26. Stearns, S. C. *The evolution of life histories*. (Oxford University Press, 1992).

27. Stiernagle, T. Maintenance of C. elegans. *WormBook Online Rev. C Elegans Biol.* (2006). doi:10.1895/wormbook.1.101.1

28. Lionaki, E. & Tavernarakis, N. Assessing aging and senescent decline in Caenorhabditis elegans: cohort survival analysis. in *Cell Senescence* (eds. Galluzzi, L., Vitale, I., Kepp, O. & Kroemer, G.) 473–484 (Humana Press, 2013).

29. Lind, M. I., Zwoinska, M. K., Meurling, S., Carlsson, H. & Maklakov, A. A. Sex-specific trade-offs with growth and fitness following lifespan extension by rapamycin in an outcrossing nematode, Caenorhabditis remanei. *J. Gerontol. A. Biol. Sci. Med. Sci.* **71**, 882–890 (2016).

30. Rechavi, O. *et al.* Starvation-induced transgenerational inheritance of small RNAs in C. elegans. *Cell* **158**, 277–287 (2014).

31. Brommer, J. E., Merilä, J. & Kokko, H. Reproductive timing and individual fitness. *Ecol. Lett.* **5**, 802–810 (2002).

32. DeWitt, T. J. & Scheiner, S. M. *Phenotypic plasticity: functional and conceptual approaches*. (Oxford University Press, 2004).

33. Mousseau, T. A. & Fox, C. W. The adaptive significance of maternal effects. *Trends Ecol. Evol.* **13**, 403–407 (1998).

34. Price, T. D., Qvarnström, A. & Irwin, D. E. The role of phenotypic plasticity in driving genetic evolution. *Proc. R. Soc. Lond. Ser. B-Biol. Sci.* **270**, 1433–1440 (2003).

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

36. Coulson, T. *et al.* Modeling adaptive and nonadaptive responses of populations to environmental change. *Am. Nat.* **190**, 313–336 (2017).

37. Vasseur, D. A. *et al.* Increased temperature variation poses a greater risk to species than climate warming. *Proc. R. Soc. B Biol. Sci.* **281**, 20132612 (2014).

38. Vázquez, D. P., Gianoli, E., Morris, W. F. & Bozinovic, F. Ecological and evolutionary impacts of changing climatic variability. *Biol. Rev.* **92**, 22–42 (2017).