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How is epigenetics predicted to contribute to climate change adaptation? What evidence do we need?

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Transgenerational effects that are interpreted in terms of epigenetics have become an important research focus at a time when rapid environmental changes are occurring. These effects are usually interpreted as enhancing fitness extremely rapidly, without depending on the slower process of natural selection changing DNA-encoded (fixed) genetic variants in populations. Supporting evidence comes from a variety of sources, including environmental associations with epialleles, cross-generation responses of clonal material exposed to different environmental conditions, and altered patterns of methylation or frequency changes in epialleles across time. Transgenerational environmental effects have been postulated to be larger than those associated with DNA-encoded genetic changes, based on (for instance) stronger associations between epialleles and environmental conditions. Yet environmental associations for fixed genetic differences may always be weak under polygenic models where multiple combinations of alleles can lead to the same evolutionary outcome. The ultimate currency of adaptation is fitness, and few transgenerational studies have robustly determined fitness effects, particularly when compared to fixed genetic variants. Not all transgenerational modifications triggered by climate change will increase fitness: stressful conditions often trigger negative fitness effects across generations that can eliminate benefits. Epigenetic responses and other transgenerational effects will undoubtedly play a role in climate change adaptation, but further, well-designed, studies are required to test their importance relative to DNA-encoded changes.

This article is part of the theme issue 'How does epigenetics influence the course of evolution?'

1. Introduction

Global change is rapidly exposing natural populations to increasingly stressful environmental conditions. Populations are already responding by shifting their distributions and their timing of growth and reproduction [1,2]. However, the response of many populations/species may be inadequate to counter the speed and magnitude of environmental change, leaving them vulnerable to extinction [3]. Avoiding extinction depends on the rate at which organisms can mitigate stressful conditions via phenotypic plasticity or evolutionary adaptation. A prerequisite for evolutionary adaptation is adequate genetic variation in the trait(s) under selection, with adaptation occurring via standing genetic variation or new mutations [4,5]. Standing genetic variation is ubiquitous in natural populations [5,6] but there are exceptions, which may include classes of traits relevant to climate change, such as heat resistance [7].

Despite the evidence for rapid evolution in nature [8–10], most likely resulting from standing genetic variation [5,10], many question whether standing genetic variation will be sufficient to enable adaptation to rapid environmental change (e.g. [11,12]. Population declines due to environmental mismatch and

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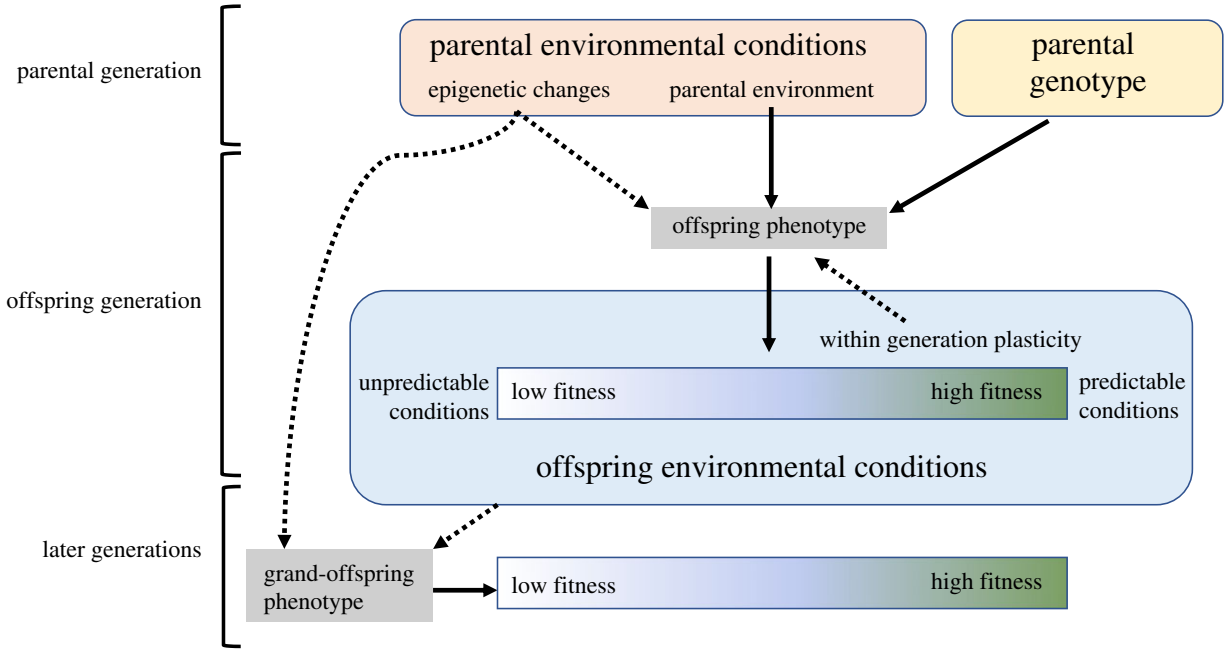

Figure 1. Challenges in assessing epigenetic effects on fitness. Parental environment can influence offspring phenotype directly (e.g. via body condition) or indirectly via epigenetic changes (dashed arrows). The impact of such environmentally induced parental effects on offspring fitness depends on whether parental environmental conditions predict the environment experienced by offspring (grand-offspring, etc.).

stress are exacerbated by habitat loss, resulting in lower levels of gene flow and lower genetic variation [13]. In this context, increasing attention is being given to the potential for transgenerational effects, and in particular epigenetic changes, to contribute to evolutionary responses to global change and rescue populations from extinction (e.g. [11,12,14]). Yet questions remain about the extent to which epigenetic changes will contribute to adaptive responses to global warming.

Here we focus on two main points that are crucial to demonstrating the evolutionary significance of epigenetics in the context of adaptation to environmental change: (i) the relative contribution of genetic and epigenetic variation and (ii) the fitness consequences of transgenerational epigenetic effects. First, we establish the theoretical predictions of the contribution epigenetics could make to climate change adaptation, then we consider the empirical evidence currently available, and what further evidence is required. Our intention here is not to provide a comprehensive review of transgenerational effects and epigenetics, which have been covered elsewhere (e.g. [13–17]); rather we begin with the presumption that environment induces epigenetic change, which is then transmitted across generations, and we then seek to determine the role this variation plays in adaptation.

2. Theoretical frameworks for studying the evolutionary significance of transgenerational effects/epigenetics

While much of the recent interest in transgenerational effects has focused on epigenetics [14], transgenerational effects have been considered in the broader context of parental and grandparental effects (e.g. [16–18]) for decades. Parental effects are ubiquitous in nature in a wide range of traits and taxa (overviews in [19–23]) and can be environmentally induced (transgenerational plasticity; figure 1) or genetic (parental genetic effects). Theoretical frameworks that explicitly examine the evolutionary significance of parental effects have been developed (e.g. [22,24–27]). When linked to the empirical literature, these frameworks have provided insight into the impact of parental effects on the evolutionary change that occurs over ecological timescales (years, decades) [22]. Theory and empirical studies indicate that parental genetic affects and the correlation between direct and parental genetic effects can influence the phenotypic expression of traits [28] and their subsequent evolution [25,27]. Importantly, what emerges from this work is that evolution in response to environmental change will be dependent on genetic and non-genetic factors that interact both within and across generations [28,29].

Since epigenetic effects represent one mechanism for parental environmental effects to extend across multiple generations [17], consideration of their evolutionary significance should take place within the broader context of the existing literature on parental effects and transgenerational plasticity. Theoretical frameworks for understanding the evolutionary significance of epigenetic effects are provided elsewhere (e.g. [30,31]) and have emerged from two approaches. In the first, Day & Bonduriansky [29] expanded upon the Price Equation to develop a theory of evolutionary change that explicitly considers genetic and non-genetic (including epigenetic) effects, and their interactions. In the second, which builds on the extension of population genetic theory ([31,32] and references therein), the focus is on how epigenetic mechanisms might evolve due to environmental heterogeneity [33] and variation in the direction of selection between two habitats [31] under the assumption that epigenetic variation is induced by the environment and that a single or a few loci are involved [31,34–36]. The latter has also been extended to understand when de novo epigenetic variation (mutations) across many loci should contribute to

adaptation [32]. Regardless of the theoretical approaches used, these studies emphasize that any assessment of theory depends on assessing the fitness effects of epigenetic and genetic effects within relevant environmental contexts (figure 1).

The adaptive significance of parental effects and transgenerational effects generally (including epigenetic effects) will depend on the nature of environmental variation [17,37,38]: in predictable environments, with high autocorrelation between parental and offspring environments, the parental trait/value is a good predictor of the fittest trait value in the current generation, and so beneficial parental effects are expected to evolve and facilitate adaptive shifts [25,37]. However, when the environment becomes less predictable from one generation to the next, theory suggests that parental effects may in fact become deleterious and thus impede adaptation [25]. For epigenetic mechanisms, the situation becomes more complicated because it depends on how the environment affects the persistence versus reversibility of epigenetic marks. Theoretical predictions arising from the phenotypic plasticity literature also suggest epigenetic effects should be selectively favoured when environments vary, but the parental environment predicts the offspring environment [39]. However, when epigenetic marks persist across more than a generation, even if offspring fitness effects are predictable from parental effects, the carry-over of information about prior environments could be maladaptive.

Rapid environmental change will likely lead to reduced environmental autocorrelation and increased mismatch between parental environment/cues and offspring selection environment, one aspect of the more general erosion of reliable cues by environment change [40]. It is therefore likely that transgenerational effects will generate inappropriate (maladaptive) responses under novel and increasingly unpredictable, environmental conditions [17]. Temporal and spatial variation in the autocorrelation of environmental cues is also important [41,42], perhaps increasingly so with rapid environmental change. This could result in transgenerational effects that are highly variable in nature, depending on site-specific patterns of environmental variability; environmental factors might interact across space and time to also influence the cues that favour adaptive transgenerational effects [12,41]. Finally, the importance of spatial and temporal variation in environmental cues will vary across developmental stages, with two life stages likely to be key—early development and prior to and during reproduction—although such effects will be complex [12]. Perhaps transgenerational effects may be beneficial if a rapid environmental change results in environments that are stressful to parents and offspring for different reasons that nonetheless trigger a general (beneficial) stress response [17]. However transgenerational responses to the types of environmental stressors increasingly being experienced in nature may often have negative rather than positive fitness consequences [43].

Where environmental conditions are increasingly unpredictable, bet-hedging may be favoured [17,39], where phenotypic diversity among offspring may lead to at least some individuals within each generation having high fitness under prevailing conditions. Evidence for bet-hedging comes from a wide variety of taxa [17,39,44]. Epigenetic effects may theoretically help produce the type of variability associated with bet-hedging [34], particularly when epigenetic effects are reversible between generations.

The above considerations highlight that empirical studies of the contribution of epigenetics to climate change adaptation require assessments of the fitness consequences of any transgenerational/epigenetic effects in an environmental context that spans across multiple generations. However, it is also necessary for empirical studies to clearly distinguish between genetic, epigenetic and other sources of transgenerational variation. We expand on these points below, considering the current empirical evidence of the role of epigenetics in adaptation, and identifying what further evidence we need to determine the expected extent of the contribution of epigenetics to climate adaptation. Our aim is to highlight the experimental approaches currently being used to study the ecological and evolutionary consequences of epigenetic effects, their strengths, their limitations and some of the most exciting opportunities for future research (table 1).

3. Empirical investigations of the genetic and epigenetic contributions to phenotypic variation and adaptation

Early work on the evolutionary potential of epigenetic variation focused on the relationship between epigenomic changes and phenotypes and/or environments. Genetically identical replicates (e.g. clones) provide ideal systems for this research, allowing unequivocal attribution to epigenetic, not genetic, causes of variation and also allowing researchers to begin to unravel the puzzle of how genetically depauperate taxa adapt (e.g. [45,46]). Extending this idea to consider whether taxa with low genetic variation might also typically be able to compensate with elevated epigenetic variation, several studies have reported a negative correlation between genetic and epigenetic variation (e.g. [47]). However, other studies have not provided strong support for this conjecture (e.g. [48]), leaving open the question of whether the ability of organisms to trigger epigenetic variation might evolve in response to low standing genetic variation.

For genetically variable taxa, several analyses of wild populations suggest weak or no genetic variation among environments, but strong epigenetic environmental differentiation (e.g. [49,50]), implying that epigenetic variation facilitates local adaptation even in the presence of genetic variation. While intriguing, some caution is required in interpreting these observations. Notably, studies considering only genetic variation have reported strong genotype–environment associations (reviewed in [51,52]). This apparent discrepancy might reflect biases where epigenetic studies focus on taxa *a priori* expected to exhibit epigenetic variation, while genetic studies focus on taxa *a priori* expected to be genetically differentiated. Alternatively, the different outcomes might reflect methodological issues. Single nucleotide polymorphisms (SNPs) are typically considered in genetic studies of adaptation (reviewed in [51,52]), while markers such as copy number variants (CNVs) (e.g. [53]) or amplified fragment length polymorphisms (AFLPs) (e.g. [49]) are commonly used in epigenetic studies. Further, while many epigenetic studies have equivalent potential to detect similar numbers of variable genetic and epigenetic markers (e.g. [49]), other studies survey far fewer potentially variable genetic than epigenetic loci (e.g. [53,54]). Nonetheless, current data provide an intriguing suggestion that

Table 1. Approaches to investigating how epigenetics can contribute to climate adaptation. For each of the two general approaches, we identify questions that they have addressed as well as future research opportunities. In addition to identifying some general outcomes from published studies, we highlight some strengths and weaknesses of each approach.

approach	questions addressed	outcomes	strengths	challenges and weaknesses	future research
Correlational: Sample wild individuals experiencing or adapted to divergent environmental conditions.	1. Does epigenetic variation vary with environmental conditions that might reflect selection? 2. How strong are epigenetic patterns versus those observed in the fixed component of the genome?	1. Across a range of different taxa and habitats, epigenetic variation (globally and at specific loci) is associated with environment /climate variation. 2. More epigenetic than genetic variation within and among populations.	1. Observe outcomes of long-term selection on epigenetic patterns. 2. Make links to landscape processes that might influence epigenetic responses.	1. Ensuring equal opportunity to sample genetic and epigenetic variation (i.e. equivalent genomic coverage of each). 2. Demonstrating that epigenetic variation is not a consequence of genetic adaptation (particularly challenging when different polygenic responses might lead to the same evolved phenotype).	1. Theoretical predictions and null models for the evolution of epigenetic variation under different assumptions about selection regime, mutation rate, mutational effects, and the coincident contribution of standing genetic variation. This would emulate the wealth of simulation studies leading to development of our understanding of the evolution of allele frequencies under divergent selection and various demographic histories. 2. Manipulative experimental tests of predictions from correlational data, assessing the importance of identified environmental variables in more controlled settings. 3. Climatic hindcasting linking environmental variability experienced across generations and how these might favour/disfavour epigenetic versus fixed genetic responses.

(*Continued*.)

Table 1. (*Continued.*)

approach	questions addressed	outcomes	strengths	challenges and weaknesses	future research
Manipulative: Expose individuals from the same population(s) to different environments that vary in stress levels and/or levels of variability. Either determine responses over the short term or allow populations to adapt over multiple generations (experimental evolution).	1. Does the population mean (phenotype and/or epigenome) differ between environmental conditions? 2. Does parental (grand-parental etc) environment have carry-over effects on the offspring epigenome and/or the phenotype of offspring? 3. Do epigenetic effects on fitness vary among taxa with low levels of genetic diversity (e.g. clonal taxa) versus taxa with substantial standing genetic variation? 4. Does the epigenetic response increase fitness in the generation of initial exposure or subsequent generations?	1. Epigenetic variation is triggered by particular conditions and persists across multiple generations. 2. Epigenetic variation can increase/decrease fitness.	1. Infer causality of that specific environmental effect on the observed epigenetic response (but further experiments required to determine the effect of specific epigenetic markers on phenotype). 2. Can distinguish epigenetic from genetic and maternal responses to the environmental manipulation. 3. Can partition the beneficial and deleterious fitness effects of epigenetic variation within and across generations exposed to different environments.	1. Logistical limits leading to low sampling of environmental space and low replication, predicted to lead to unrepeatable results. 2. Sampling of few genotypes (parental individuals) to form the population is predicted to cause founder effects, and non-representative, unrepeatable results. 3. Selection under stressful environments causes genetic evolution (genotype-specific mortality or infertility), confounding this with epigenetic responses. 4. Individual relative fitness in the natural environment can only be approximated by life-history trait values under laboratory conditions.	1. Characterization of the fitness consequences of induced responses under multiple environmental conditions, using experimental designs that account for genotype-specific variation in representation across multiple generations (i.e. genetic responses to selection). 2. Consider the role of environmental unpredictability experimentally in favouring/disfavouring epigenetic responses. 3. Examine the effects on population persistence in stressful environments of artificially reducing the epigenetic response (through inhibitors). 4. Determine the effect size (on fitness and traits) of environmentally induced and spontaneous epimutations relative to the effect sizes of genetic variants. 5. Characterize the rate of change in the frequency of environmentally induced epialleles over generations (i.e. their evolution), and compare to those observed in fixed DNA.

epialleles might have a different spectrum of effect sizes and/or frequencies than standing genetic variation.

Although empirical attention has predominantly focused on among-population variation, the role of epigenetic variation in adaptation needs to be assessed by examining how it contributes to relative fitness variation among individuals within a population, i.e. to the variation that selection acts on. Further, understanding the genetic and environmental causes of that phenotype-altering epigenetic variation is important for defining the evolutionary role of epigenetics. Correlational (observational) studies in clonal taxa have demonstrated that within-population epigenetic variation correlates with within-population phenotypic variation, independent of any genetic contribution (e.g. [55]), but they cannot determine whether the epigenetic (and hence phenotypic) variation is environmentally induced or spontaneous. Manipulative studies of epigenetic responses can expose the same genotypes to different environmental conditions, demonstrating the role of the environment in inducing consistent epigenetic (and phenotypic) differences among individuals (e.g. [56]). However, several papers have reported idiosyncratic responses of the same genotype to the same environmental stress (e.g. [57,58]), pointing to a need to consider both mean response and also the variation around that response. Furthermore, a critical missing link is our knowledge of the natural spatio-temporal scale of environmental variation, and thus how much epigenetic variation might typically be induced within populations of individuals occupying the same habitat and experiencing the same selection.

In genetically variable taxa, genotypes can vary markedly in their phenotypic fitness or epigenetic responses to the same environmental conditions (e.g. [59]). Alvarez *et al*. [60] found significant among-genotype variation in epigenetic responses to a common environment in *Arabidopsis*; however, there appeared to be little shared genetic variation between the initial response to the environment, and how that response was transmitted across generations [60]. Such studies, demonstrating genotype by environment interaction effects on the epigenome, highlight the expected complex interaction between epigenome and genome during adaptation, and the necessity of jointly studying these sources of heritable variation in fitness.

Manipulative investigations of the role of epigenetics in adaptation have tended to have low replication generally and, for genetically variable taxa, the number of genotypes sampled in particular has typically been very small ($\ll$20). Both founder effects among treatments and the evolutionary process itself will generate variation among independent replicates experiencing the same environment [61–63]. Experimental populations are often exposed to harsh conditions, which may induce mortality or infertility (i.e. apply selection) in a genotype-specific manner, but such effects will be statistically difficult to detect in small experiments. Fakheran *et al*. [63] subjected a population of 19 *Arabidopsis* genotypes to selection for dispersal, where after five generations, 16 genotypes had been completely eliminated and two genotypes contributed 90% of the population. Notably, in small populations, Mendelian randomization ensures drift and selection can drive genetic evolution both within (i.e. among siblings) and among families [64]. Well-replicated experiments are required to ensure that conclusions about genotype-independent environmental effects on the epigenome and/or phenotype are robust.

The nature of standing genetic variation itself is likely to challenge both identification of genetic loci controlling epigenetic responses to the environment and dissection of genetic and epigenetic contributions to adaptation. In particular, investigations of heritable variation in human traits have implicated strong contributions from rare alleles, requiring very large datasets for detection [65]. Further, polygenic adaptation is theoretically predicted to be driven by very small (statistically undetectable) allele frequency changes across many loci [66]. Genetic redundancy, inherent in polygenic architecture, is also expected to result in non-parallel allele frequency changes among populations evolving in response to the same selection pressures [62,67] and turnover in alleles as adaptation proceeds [68]. These complexities of the genotype:phenotype map will make it difficult to detect causal genetic variants, or definitively demonstrate that genetic evolution has not occurred. Thus, the lack of statistical support for adaptive genetic changes does not provide robust indirect evidence that phenotypic change was due to epigenetic causes. Adaptive changes in allele frequencies across multiple loci could underpin fitness-increasing epigenetic responses to the local environment [69].

Theoretical simulations have been beneficial in developing expectations for genetic evolution in response to selection and the analytical approaches to detect signals of selection in the genome (e.g. [68,70]). Extending such studies to incorporate epigenetic effects will allow us to develop robust approaches to investigate how genetic and epigenetic variation interact during adaptation, particularly as empirical data on epimutations accumulates, supplying relevant parameter values to simulate. Simulations should place epigenetic variation within the genomic context where it arises, exploring how selection on the genetic background (synergistic or antagonistic) affects the evolutionary trajectories of epialleles, independent of the direct selection operating on them. Using individual-based evolutionary simulations, Kronholm & Collins [32] determined that the contribution of epigenetic variation to adaptation depended on the epiallele effect sizes relative to genetic variants, accelerating evolution when their effects were small, but slowing adaptation when their effects were relatively large.

One potentially powerful empirical avenue for understanding the epigenetic and genetic responses to environment change, including any coevolutionary dynamics, is experimental evolution coupled with genome and epigenome sequencing through time. Kronholm *et al*. [71] experimentally adapted unicellular green alga to different biotic environments, demonstrating that chemical or genetic inhibition of epigenetic responses slowed adaptation. Evolve re-sequence studies have been conducted in several genetically variable eukaryotes (e.g. [67]) and could consider epigenetic responses alongside genetic responses.

4. Determining the fitness consequences of epigenetic effects

Epigenetic variation is associated with the environment in a wide range of organisms (e.g. [49,72,73]), suggesting that local fitness effects of the epigenome have led to the evolution of these patterns. However, among-population divergence does not directly indicate the fitness consequences of epigenetically generated phenotypic variation within populations,

and hence the potential contribution of epigenetic variation to future or ongoing adaptation. By contrast, while providing limited insight into naturally occurring epigenetic variation or the variability of selection environments, manipulative experiments, where individuals are exposed to specific environmental conditions and their (or their offspring's) responses measured, can indicate how the environment can generate epigenetic variation and the potential fitness effects of the induced variation under specific conditions.

Individual fitness is difficult to measure, and studies typically measure components of fitness, or traits that have been shown to affect fitness under at least some conditions. For example, the offspring of drought-stressed parent plants grow longer roots, which is predicted to enhance fitness under drought conditions [74]. In general, meta-analyses are consistent with fitness benefits of parental or transgenerational effects, where offspring had elevated fitness in their parental environment ([75], but see [76,77]). Nonetheless, many studies, including ones in which individuals were exposed to simulated future climate conditions, have found evidence of reduced offspring fitness in the parental (inducing) environment, inconsistent with an epigenetic contribution to adaptation (e.g. [78]). Further investigation is required to understand the extent to which contradictory results reflect biologically interesting differences in responses among populations/taxa or in the effects of different environments [41,75]. It will be important to consider both evidence for environmental autocorrelation [79] and the robustness of experimental designs (e.g. sample size, correct identification of fitness traits; table 1).

Fitness in populations under natural conditions is extremely complex, where, for instance, the selection environment can alter trade-offs among fitness components (e.g. [80]), novel climate conditions might generate opposing density-independent and density-dependent selection [81,82], and the direction of selection can change throughout life [83]. Manipulative studies typically consider few (one or two) axes of environmental variation, few levels along each axis and few components of fitness or episodes of selection. Studies considering more complex, multi-factorial environments have documented complex effects on phenotypes, including responses with apparent antagonistic fitness effects among environmental axes (e.g. [84,85]). These considerations highlight both the potential for experimental studies to miss true fitness benefits from epigenetic responses (e.g. they occur at a different life stage to the one surveyed) or to identify a fitness benefit, but fail to detect a fitness cost (e.g. paid at a different life stage) that outweighs the benefit.

Again, experimental evolution, where among-individual variation in relative fitness under the experimental conditions causes selection, is a potentially powerful tool for investigating the total fitness consequences of epigenetic variation. Working with populations experimentally adapted to different levels of habitat disturbance, Schmid *et al*. [86] demonstrated reduced epigenetic variation associated with the response to selection, suggesting that selection eliminated low-fitness epialleles, fixing high-fitness epialleles. Other experimental evolution studies have focused not on the mean response of populations/treatments to specific environmental conditions, but on the heterogeneity of responses, i.e. bet-hedging. You *et al*. [87] demonstrated that a methyltransferase gene regulates inter-cellular variation in yeast, where stressful environmental conditions cause a decrease in the methyltransferase and an increase in 'noise' of protein production among cells; this bet-hedging (i.e. heterogeneity among cells) was associated with increased fitness under variable conditions. At the level of population fitness, Latzel *et al*. [88] demonstrated greater fitness in epigenetically diverse then epigenetically homogeneous populations of *Arabidopsis thaliana*. Given that climate change is expected to decrease the predictability of environments, further work should seek to determine the role of epigenetics in generating variation, and the fitness benefits accruing to epigenomes that generate phenotypic variation among individuals.

Manipulative reduction (elimination) of genome-wide methylation is a powerful approach for demonstrating that methylation causes phenotypic variation. Some investigated phenotypes (e.g. growth rate), expected to contribute to total fitness, are depressed by de-methylation (e.g. [89]), while others are depressed by elevated methylation levels (e.g. [54]). Populations with induced genetic mutation load also exhibit depressed fitness (reviewed by [90]): should we interpret the fitness consequences of genome-wide changes in methylation levels to be indicative that epimutation load, like genetic mutation load, depresses fitness? Molecular genetic studies are another empirical avenue for understanding the distribution of fitness effects of epimutations. For example, the loss of function DNA mutations (or gene knockout manipulations) cause greater phenotypic effect than other mutations; do epimutations that turn on/off genes have greater phenotypic (fitness) effects than epimutations with quantitative effects on expression?

5. Where to from here?

Despite decades of empirical studies of epigenetic effects, we are still lacking a clear understanding of the adaptive significance of epigenetics in response to the types of environmental shifts that are currently driving rapid global change. As we have highlighted, to define the contribution of epigenetics to climate change adaptation, future empirical studies must elucidate the fitness consequences of epigenetic effects and the extent to which such effects are independent of genetic variation and other sources of transgenerational variation. Empirical studies must be robust to sampling error, and they must investigate experimental conditions that reflect the types of environmental variation that is likely to occur with ongoing change in nature, such as changes in environmental variability, autocorrelation, cue reliability and novel conditions [17,41]. Assessing the fitness consequences throughout the lifecycle and over multiple generations is also necessary [12]. Consideration should be given to the evolutionary history of the taxa under study—does their environmental history suggest that they will have evolved an adaptive epigenetic response to environmental variation?

Finally, the extent to which epigenetic/transgenerational effects may contribute to the evolutionary rescue of populations/species threatened by rapid and increasingly stressful environmental change requires not just consideration of the fitness effects of genetic and non-genetic factors and their interactions, but also the consideration of the evolutionary processes that the populations undergo [10]. Population size, dispersal and density regulation of population growth will also play a key role in determining how populations respond to climate change [10].

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