PROCEEDINGS B

royalsocietypublishing.org/journal/rspb

Research

Cite this article: Acasuso-Rivero C, Murren CJ, Schlichting CD, Steiner UK. 2019 Adaptive phenotypic plasticity for life-history and less fitness-related traits. *Proc. R. Soc. B* **286**: 20190653.
http://dx.doi.org/10.1098/rspb.2019.0653

Received: 20 March 2019
Accepted: 12 May 2019

Subject Category:
Evolution

Subject Areas:
evolution, theoretical biology, ecology

Keywords:
fitness, demographic buffering, life-history, evolution, environmental variation, (non-) adaptive plasticity

Author for correspondence:
Cristina Acasuso-Rivero
e-mail: acasusa@gmail.com

†Present address: Department of Veterinary Medicine, University of Cambridge, Cambridge, UK.

Electronic supplementary material is available online at http://dx.doi.org/10.6084/m9.figshare.c.4508615.

THE ROYAL SOCIETY PUBLISHING

Adaptive phenotypic plasticity for life-history and less fitness-related traits

Cristina Acasuso-Rivero[1,†], Courtney J. Murren[2], Carl D. Schlichting[3] and Ulrich K. Steiner[1,4]

[1]INSERM U1001, Université Paris Descartes, Sorbonne Paris Cité, Faculté Cochin, 75014 Paris, France
[2]Department of Biology, College of Charleston, Charleston, SC 29424, USA
[3]Department of Ecology and Evolutionary Biology, University of Connecticut, Storrs, CT 06269, USA
[4]Department of Biology, Center of Population Dynamics, University of Southern Denmark, 5230 Odense, Denmark

CA-A, 0000-0002-1002-4917; CJM, 0000-0003-0361-1790; CDS, 0000-0001-7093-7913; UKS, 0000-0002-1778-5989

Organisms are faced with variable environments and one of the most common solutions to cope with such variability is phenotypic plasticity, a modification of the phenotype to the environment. These modifications are commonly modelled in evolutionary theories as adaptive, influencing ecological and evolutionary processes. If plasticity is adaptive, we would predict that the closer to fitness a trait is, the less plastic it would be. To test this hypothesis, we conducted a meta-analysis of 213 studies and measured the plasticity of each reported trait as a coefficient of variation. Traits were categorized as closer to fitness—life-history traits including reproduction and survival related traits, and farther from fitness—non-life-history traits including traits related to development, metabolism and physiology, morphology and behaviour. Our results showed, unexpectedly, that although traits differed in their amounts of plasticity, trait plasticity was not related to its proximity to fitness. These findings were independent of taxonomic groups or environmental types assessed. We caution against general expectations that plasticity is adaptive, as assumed by many models of its evolution. More studies are needed that test the adaptive nature of plasticity, and additional theoretical explorations on adaptive and non-adaptive plasticity are encouraged.

1. Introduction

Adaptation to varying environments has long been a central question in ecology and evolution [1]. In times of global change and increased habitat fragmentation owing to anthropogenic activities, individuals and populations experience novel environments more frequently than in the recent past. Therefore, they are confronted with increased environmental variability and less predictable spatial and temporal environments. Populations can deal with such varying conditions by either local adaptation or phenotypic plasticity [2,3]. Local adaptation involves genetic differentiation specific to each environment, whereas phenotypic plasticity allows single genotypes to express different phenotypes under diverse environmental conditions.

Adaptive plasticity evolves when the environmental cues are reliable and environments vary frequently, i.e. selection for plasticity is strong. Fixed genetically determined traits (environmental phenotypic canalization) evolve and may be selected for, if environments vary rarely or unpredictably [2]. To this end, selection acts to optimize the trait expression in each environment to maximize fitness in that specific environment or across environments [4]. The tracking of environmental change via plasticity can be achieved with or without genetic differentiation [2,3]. Phenotypically plastic traits evolve as do other quantitative traits, by natural selection acting on genetic variation among genotypes; here genotypes vary in their reaction norms, the level of phenotypic plastic responses across environments [5].

(a) Plasticity in life-history and non-life-history traits

Because of trait plasticity evolves as any other trait, plastic traits more closely related to fitness are predicted to be under stronger selection for genetic canalization, both within and across environments [6–10]. Genetic canalization refers to individuals of the same genotype that have different genetic backgrounds; and should not be confused with environmental canalization that denotes individuals from similar genotypes with a fairly invariant phenotype in response to environmental variations [11]. So, genetic variability in reaction norms should erode faster in life-history traits (LHt; here considered as reproduction and survivorship) compared to non-life-history traits (N-LHt; all other traits that are less directly related to fitness) [6–8]. However, the difference in selection strength for genetic canalization does not predict whether LHt should exhibit more or less plasticity than N-LHt. Such a prediction can be derived from evolutionary theories of plasticity and demographic theory [12,13] related to similar arguments discussed in previous studies [14,15].

(b) Evolutionary theories of plasticity

A genotype that matches the fitness optimum in only one environment, a specialist, will have the highest fitness in that environment and not other environments [5,16]. An adaptive plastic genotype, a generalist, will express a closer match and higher fitness in various environments but not as high as the specialist in any particular environment [5,16]. This theory leads to the expectation that fitness across environments should vary more for specialists that lack adaptive plasticity in N-LHt compared to generalists, when phenotypic (fitness) optima differ among environments. Because of trait integration, trade-offs and optimization of fitness across traits and environments, N-LHt are expected to moderate and buffer—through their plasticity—the effect of environmental variation on LHt.

Related arguments of organismal response to environments rooted in demographic theory have been formulated by Caswell [14]. He extended previously developed models of homoeostasis [17] to a demographic setting [18,19]. The population growth rate λ serves as fitness and Caswell argued that plasticity in N-LHt may reduce variance in λ, as long as LHt are not negatively covarying. Extensions of these foundational demographic theories expand aspects of these arguments, e.g. the demographic buffering hypothesis which states that vital rates (mortality and fertility) of population projection models with higher sensitivities (with respect to λ) should be negatively correlated to variability in these vital rates across environments. Empirical evidence for the demographic buffering hypothesis is mixed. In the context of our study, it is crucial to note that the demographic buffering hypothesis does not predict whether one trait type is expected to express higher plasticity. This lack of prediction is likely because the theoretical focus and empirical tests of the demographic hypothesis have been on LHt, i.e. age-specific survival or reproduction [20], and not on N-LHt.

The environmental canalization hypothesis states that the potential demographic impact of fitness components and their temporal variability (such as plasticity) should be negatively correlated. This hypothesis is based on investigations from age-structured populations [21], and has been empirically tested by comparing variance in survival among juvenile versus adult individuals where it is supported only for longer-lived mammals [21].

The underlying arguments of both the demographic buffering and the environmental canalization hypotheses are derived from stochastic population theory [22,23]. This theory illustrates how stochastic population growth rate, λ_s, is reduced by high variability in vital rates (i.e. reproduction and survival, the LHt we discuss above) among different environments experienced across time. Following from this, an increase in fitness can be achieved simply by reducing variability in reproduction and survival across time or environments, without increasing either mean survival or reproduction. Adaptive phenotypic plasticity is predicted to do exactly this: high plasticity of N-LHt buffer the environmental effects on fitness, leading to reduced variance in traits closely related to fitness (or LHt) [14,15].

(c) The controversies

The role that plasticity plays for ecological and evolutionary processes is controversial in the context of speciation and diversification, adaptation to novel environments, population viability, population management and the invasiveness of species [24–26]. These controversies arise in part because on one hand, phenotypic plasticity is a potential mechanism for responding to environmental challenges [24,27,28], but on the other hand, might also hinder adaptation to novel environmental conditions [29].

Many of these controversies are contingent upon the expectation that phenotypic plasticity should be adaptive [28,30,31], though neutral and maladaptive plasticity have been shown [32]. Theoretical predictions of how maladaptive plasticity should evolve or can be maintained are lacking, beyond predictions related to novel environments [33]. Despite the large amount of high-quality studies on phenotypic plasticity and expositions of its costs and limits [34,35], our understanding of the mechanics of its evolution is still limited. Whether plasticity is adaptive depends on traits, species and environments. For example, generalizations of the adaptive capacity of phenotypic plasticity have been challenged by postulating differences in the costs and limits of plasticity between sessile and free-moving organisms [36]. Previous approaches to assessing the frequency of adaptive plasticity have been based on analyses of reciprocal transplant studies [37], transcriptome and proteome analyses (see references in [36]), or meta-analyses comparing plastic and canalized responses, particularly in plants [32].

Fitness is frequently defined through LHt, and different traits may respond in similar ways to environmental changes, So, fitness may be correlated, and thus confounded, with environmental effects [32,38]. For instance, larger body size is often correlated with higher fitness, but is also commonly enabled by nutrient rich environments. Typically, we lack data on what the optimal size in such a rich environment would be and if such an optimum is realized [39,40]. Even powerful approaches such as reciprocal transplant experiments do not circumvent the challenge of determining optimal phenotypes.

(d) The predictions

Here, we tested two hypotheses. First, we tested whether plasticity is adaptive by comparing the plastic responses between LHt and N-LHt and predict that LHt will show

lower levels of plasticity. We performed this test from two perspectives, by comparing plastic responses of LHt and N-LHt among studies, and then by restricting comparisons to within studies. Our second hypothesis is that LHt will be genetically more canalized. We evaluated this by comparing canalization of the different trait types in relation to their distance to fitness.

2. Methods

We used an indirect approach to evaluate these hypotheses by reviewing 24 years of research publications exploring phenotypic plasticity, covering a wide range of environmental conditions and spanning taxonomic groups. Our meta-analysis approach provides power to the study of phenotypic plasticity by generalizing the hypotheses and comparing information from a great variety of sources.

(a) Adaptive plasticity

In order to test whether LHt exhibit lower plasticity than N-LHt, we conducted a meta-analysis on studies reporting reaction norms in different trait types across environments and species. We selected publications employing the keywords 'life history & morphology & plasticity' between 1991 and 2006 from all databases in *Web of Knowledge (WoK-ISI)* and between 2007 and 2011 from *Web of Science (ISI)*. We identified 583 papers in total, and added a few studies published between 1987 and 2011 known to us that also met the criteria. From this initial set, we extracted data from 213 studies reporting reaction norms as a measure for plasticity. The data extracted were the mean values for each environment evaluated in each study and the independent sample sizes. Quantification of the reaction norms was achieved through the computation of the coefficient of variation (CV) of the trait expression across environments, a dimensionless parameter allowing the evaluation of proportional responses as a mean-standardized measure [41]. We computed 5885 CVs for 211 species exposed to 2–11 environment levels. As a visual validation for reporting and publication bias [42], we plot the number of studies through time and a funnel plot in the electronic supplementary material, figures S1 and S2.

Traits were categorized as: LHt: survival and reproduction; and N-LHt: behaviour, morphology, metabolism and physiology, and development. When available, we extracted data directly from tables or the text; alternatively, we extracted quantitative information from figures using the software ImageJ [43]. Environmental variation was grouped into six categories: (i) environment quality, (ii) interspecific interactions, (iii) intraspecific interactions, (iv) intrinsic resources, (v) photoperiod and light; and (vi) temperature. When genotypes or families were included, taxonomic groups were classified based on species identity (ID) as defined by the NCBI taxonomy database [44]. We clustered organisms according to NCBI at the taxonomic level of Phylum (referred as taxa in table 1). Electronic supplementary material, S1 and table S1 show details of the categorization.

We analysed the data by generalized mixed-effect models in software R ([45]; R Core team, version 3.2.3, package lme4.) CVs were log transformed and used as the response variable. Reference ID was included as a random effect accounting for confounding effects within the same publication. We also included the number of data points retrieved from a single study—Repetitions (electronic supplementary material, table S1)—as weights to assess potential bias towards a few studies or species. The focus of our study was the evaluation of the explanatory variables trait type, environment type and taxonomic group (electronic supplementary material, table S1). The models tested if plasticity was better explained by (i) the trait type, by (ii) the type of environment, by (iii) the taxonomic group or interactions among those. Other factors, such as the location of the experiments, breeding conditions, or others listed in electronic supplementary material, table S1, were explored. All of these other factors contributed marginally to the observed variance (analyses not shown). We used information theoretical approaches (Akaike information criterion; AIC) [46] to select among our candidate models and interpreted a model to be better supported for any $\Delta AIC \geq 2$. This approach allowed us to test among multiple competing hypotheses (models) that included additive and interactive effects among taxa, environment type and trait type.

To ensure that the CV captured plasticity for any number of tested environments, we included the number of environments as a fixed effect. Also, we re-analysed the data in a collapsed dataset that included the average CV for all means reported in the same publication, that reported the same species, environment, environment type, location, measurement, number of environments and breeding (see factors in the electronic supplementary material, table S1). Because variability in sample size and error structure might bias our results, we examined the residuals from the selected models in both the original and collapsed datasets. When comparing these models, we did not find any quantitative difference, which suggests that our results are robust regarding potential sample size bias and biased estimates related to number of environments evaluated.

To gain a better quantitative understanding of how frequently our prediction was met, we computed, for each pair of LHt and N-LHt, the differences in CV within studies ($n = 3939$), limiting our analysis to the 103 studies that measured both categories of traits. Negative values indicate that the N-LH trait showed higher plasticity, which would support our first hypothesis, and positive values reveal the opposite.

(b) Genetic canalization

Finally, we tested the prediction that LHt are more genetically canalized compared to N-LHt [8–10]. Unfortunately, reaction norm plots generally do not indicate genotypes with unique labelling, preventing distinguishing replicates from truly different genotypes (including strains, families, populations). For the genetic canalization analysis, we excluded studies that compared groups (genotypes) that are probably genetically not very distinct. For example, studies that examined workers and drones in bees, experimental evolution studies, studies that compared same genotypes or populations over time (season, years), or studies where sex was the only genotypic difference recorded. We included distinct populations, different strains and subspecies that shared the same NCBI identity but were noted as dissimilar by the authors. Using this subset of data, we estimated the mean CV within each study, for each genotype and each trait type, i.e. mean plasticity for each genotype/strain, assuring that each genotype is equally weighted. From the mean CVs, we estimated the variance among genotypes in their reaction norms. To test if LHt is genetically more canalized than N-LHt, we used a generalized linear model with a gamma error structure (lme4) to distinguish variance in CV between LHt and N-LHt. This analysis was supported by evaluations on the CV variations from populations reported as genetically similar and by a correlation analysis between plasticity and genetic variance.

3. Results

The 5885 CVs quantifying the plastic responses to environmental variation across 211 eukaryote species, comprised 59% invertebrates, 34% chordates, 6% plants and 1% green algae. Details on data and taxonomic identities are shown in the electronic supplementary material, table S2 and

Table 1. Competing linear models selected based on AIC. (Models used as response variable the natural logarithm from the coefficient of variation (CV, as a proxy for scaled plasticity). Combinations explored include trait type, environment and taxa as fixed effects. Reference study was considered as a random effect. Repetitions (sample size) were the weighting factor.)

	model	AIC	ΔAIC
null model	intercept only	19494	546.41
1	trait type	19111.41	163.82
2	environment	19463.77	516.18
3	taxa	19476.06	528.47
4	trait type + environment	19076.17	128.58
5	trait type + taxa	19083.74	136.15
6	trait type × environment	18970.24	22.65
7	trait type × taxa	19044.05	96.46
8	trait type + environment + taxa	19051.65	104.06
9	trait type × environment + taxa	18947.59	0

figure S3 (see the Dryad Digital Repository: https://doi.org/10.5061/dryad.72s8g4j [47]).

(a) Adaptive plasticity

Substantial variation in plasticity (CV) was revealed within groups and among groups of trait types, taxonomic groups and environment type (table 1 and figure 1). The plastic response to environmental variation (CV) when only a single factor was assessed, was best explained by trait type rather than environment type or taxonomic group. A null model (intercept only) did not perform better than any of the single-factor variable models. The differences in plasticity among trait types are not correlated to how closely traits are associated with fitness (i.e. trait types are ordered with relation to relative distance to fitness in figure 1). Adding taxa as an additional variable to the model with trait type explained more variation than adding environment type (table 1). Interaction effects between trait types and environment type or taxonomic group did further improve the model fit (table 1) but are challenging to interpret biologically. In contrast to our hypothesis, none of these models showed a clear correlation between plasticity (CV) and how closely traits are connected to fitness (figure 1). Hence, we show that the phenotypic response to environmental variation is dependent on the interaction between trait and environment type, but with no relation to how close the trait is to fitness.

Plants expressed high plasticity (CV) and they showed limited evidence of increased CV of LHt compared to N-LHt, contradicting our main hypothesis (figure 1). Also, plants are the taxonomic group that drove most of the difference among the taxa. Analyses that excluded the plant data, found no difference between taxonomic groups, an interaction between trait type and environment, and no relationship to how closely a trait is related to fitness (electronic supplementary material, S5).

The average sample size reported from the 5885 CVs was 119.15. The collapsed dataset comprised 1868 CV averages. When testing those two as independent variables, we found that the best-fit models corresponded to the models selected from the original dataset (data not shown). In both cases, we did not find any major biases in the residuals of the models (electronic supplementary material, figure S3). Including the number of environments worsened the models in all three cases. Together, these results support the robustness of the data provided by the extraction of the trait means from previous publications—we are confident that the CV is capturing plasticity even when accounting for only two environments.

Our results were robust to explorations of other factors—the type of experiment (laboratory or field), whether natural populations or laboratory lines were used, how many environments per study were explored, the number of data points per study, the study year or whether only LHt, only N-LHt or both types of traits were reported within a study (electronic supplementary material, table S2). None of these changed the results qualitatively or had much quantitative influence. Also, the means and standard deviations from the CVs correlated for each trait type (data not shown) discarding potential scaling artefacts. A lack of influence of these other factors suggests that our sample size is sufficiently large, and that our results are robust and general across different types of study systems

(b) Genetic canalization

When we compare pairs of LHt and N-LHt within studies, i.e. estimating the difference in CV between an LHt and an N-LHt, we see that our hypothesis was supported as often as it was rejected (figure 2). Negative values, supporting our expectation of adaptive plasticity (i.e. N-LHt exhibiting higher plasticity), were equally common as positive values (interpreted as revealing non-adaptive plasticity). For most comparisons, differences in CV were close to zero (figure 2), indicating that LHt and N-LHt within studies show similar plasticity, even though we see general differences in plasticity among trait types (figure 1). Behavioural traits tend to express more non-adaptive plasticity shown by the positive differences like metabolism and behaviour with reproduction. Morphological traits tend to express more adaptive plasticity shown by the negative differences like metabolism and survival.

Even though LHt did not show lower plasticity (figure 1), we still expected that LHt should be genetically more canalized than N-LHt. In contrast to our expectation, LHt survival and reproduction, did not show lower levels of

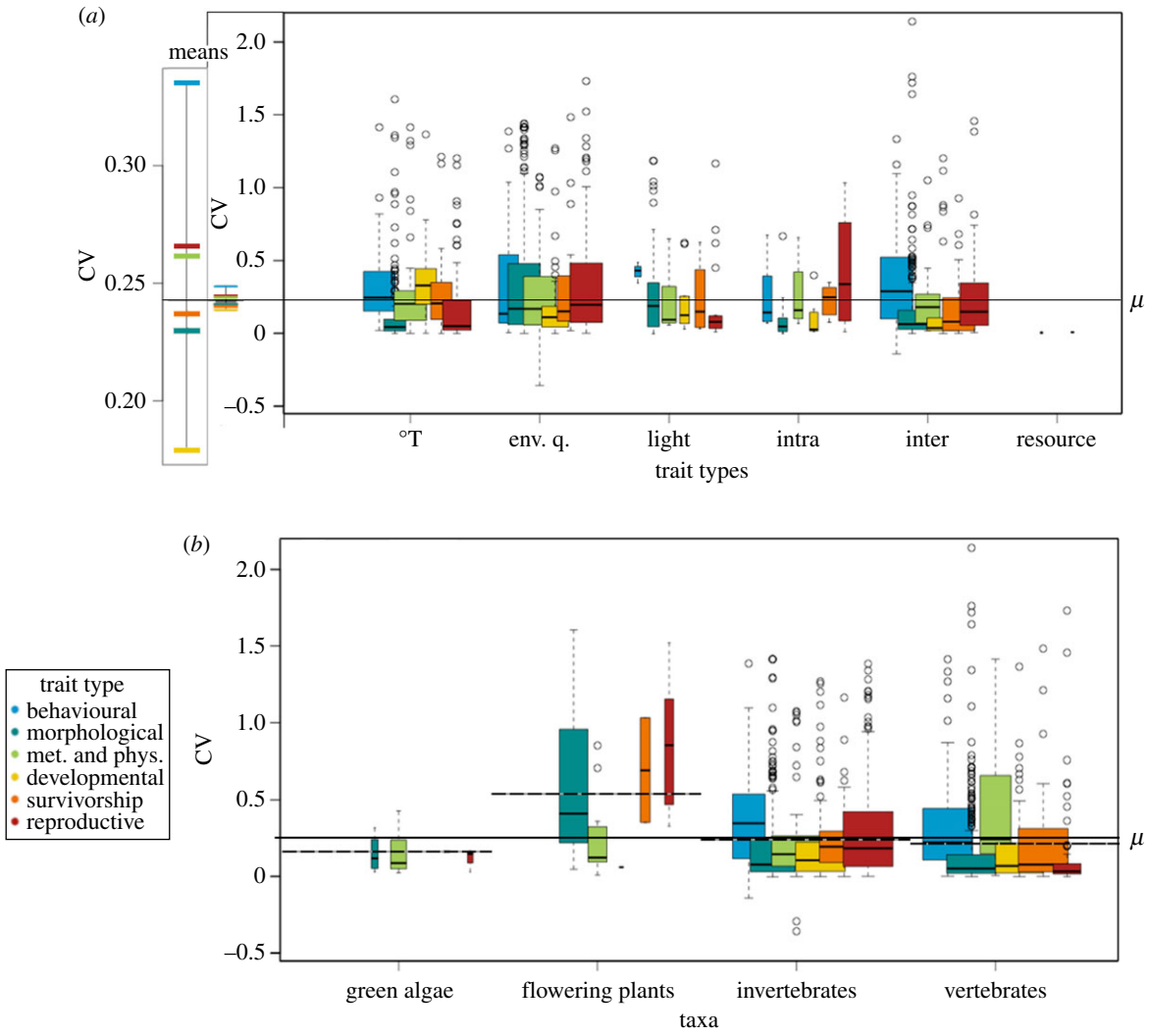

Figure 1. Plasticity (coefficients of variation; CV) among different trait types, environment types and taxa. Trait types are ordered roughly in accordance with the relative distance to fitness. We categorized life-history traits (LHt) into survivorship and reproductive; and non-life-history traits (N-LHt) into behavioural, morphological, metabolism and physiology, and developmental. The top left panel shows the trait means across all environments on an expanded scale. Directly to its right are these means shown at the same scale as the boxplot. Top, far right, CVs for each environment and trait type (colour coded). The widths of the boxes are relative to the amount of data; whiskers represent the standard deviations. The grand mean (μ) is denoted by the black line across all graphs. °T, temperature; env. q., environmental quality; light, photoperiods and light; intra, intraspecific interactions; inter, interspecific interactions; resource, intrinsic resources. (*b*) CVs among taxa and trait types. The widths of the boxes are relative to sample size; whiskers represent the standard deviations. The dotted lines denote the means per taxa. For green algae and plants, no behavioural or developmental data are available.

variability in reaction norms among genotypes, compared to N-LHt (figure 3). We found significant differences in the variance among genotypes in their reaction norms, with behavioural traits showing the highest variance among genotypes, but developmental, physiological and morphological traits showed less variance in their reaction norms compared to the LHt. Thus, there is no evidence for increased genetic canalization for LHt compared with N-LHt.

Note that the mean level of plasticity exhibited (figure 1) does not appear to be related to variability in genetic canalization (figure 3; no direct comparison possible because figure 3 is based on only a subset of the data).

We report a negative correlation between the overall CV of each trait type (figure 1) as a measure of plasticity, and the CV among genotypes (figure 3) as an inverted measure of genetic canalization ($r_s = 0.77$, Spearman; figure 4). This suggests that less plastic traits will also be the ones that are fixed faster in the populations. However, as illustrated by figure 4, the relationship between canalization and plasticity is not related to the distance from fitness. We found, as expected, that behavioural traits were more plastic and the least canalized. But we also found that morphological and developmental-related traits are the least plastic and more canalized than the other trait types. Our results also illustrate that reproductive and metabolic-related traits showed the least canalization and exhibited only a mid-plastic response when compared to other trait types. Traits related to survival showed an intermediate plastic response as well as an intermediate canalization.

4. Discussion

(a) Adaptive plasticity

Our results show that trait types did not correlate, as predicted, with how closely the traits were related to fitness but did differ in their amount of plasticity. Taken together, LHt are not less plastic than N-LHt, nor are LHt buffered

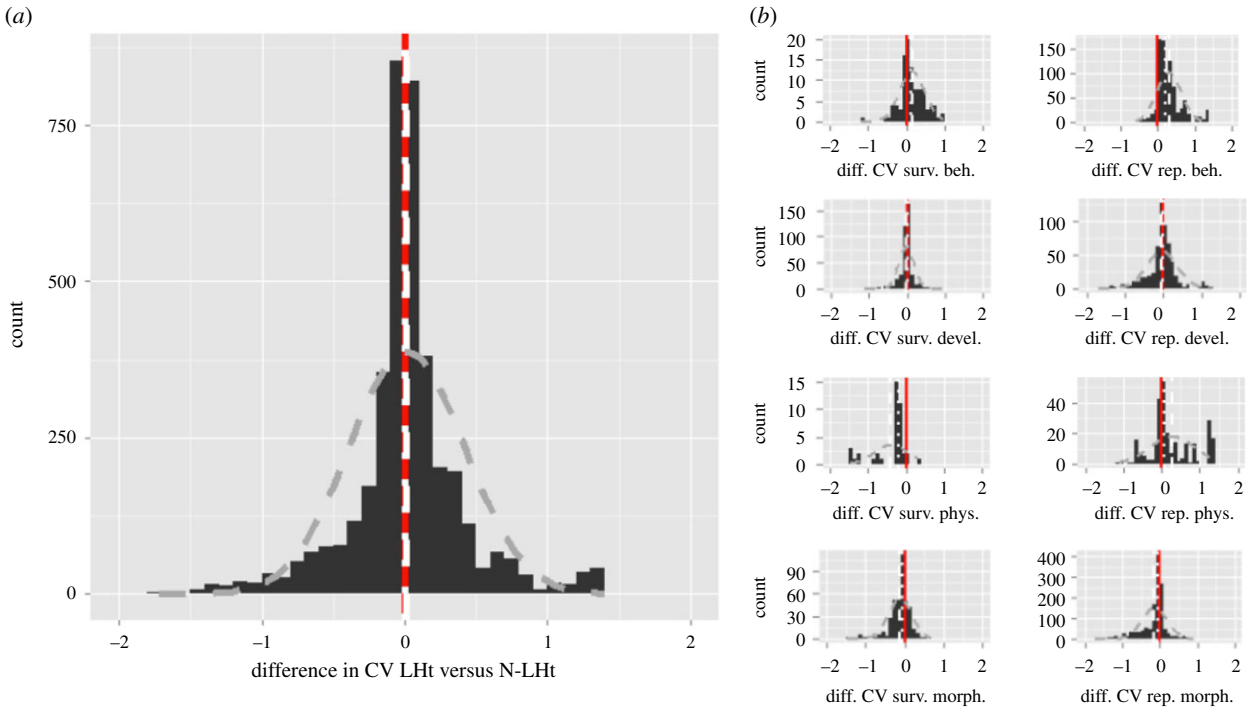

Figure 2. Distribution of differences in CV between life-history traits (LHt) and non-life-history traits (N-LHt), large panel. Small panels illustrate distributions of differences in CV between survivorship and N-LHt (behavioural, developmental, metabolism and physiology, and morphological) left column of panels, and reproductive and N-LHt (right column of panels). Red solid line depicts no difference in CV (0), white dashed line depicts the mean and dotted line depicts the median. The grey dashed line depicts a normal distribution based on the observed mean and variance. (Online version in colour.)

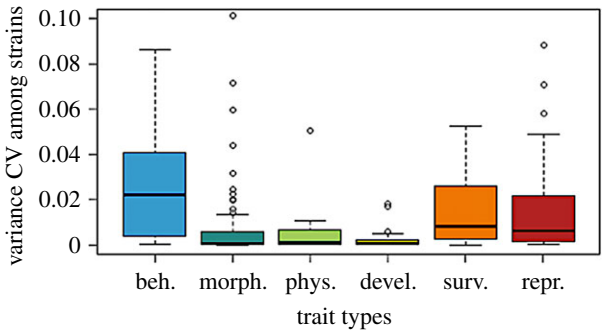

Figure 3. Variance in CV among genotypes within the same study and trait, plotted for the different trait types: beh., behaviour; morph., morphology; phys., metabolism and physiology; devel., development; surv., survival; repr., reproduction. Note *y*-axis is limited to values less than 0.1 for better visibility, i.e. not all outliers are shown. Model selection for generalized linear models with a gamma error structure and variance of CV as response variable: AIC for model with trait types (−1426.6), null model (intercept only model) AIC (−1422.0). (Online version in colour.)

against environmental variation by N-LHt. These results indicate that non-adaptive or potentially maladaptive responses in plasticity might be quite common [48]. The meta-analytical techniques we used, allowed us to aggregate information producing robust findings independent of the larger taxonomic groups, study conditions and types, and excluding the influence of potential publication biases (electronic supplementary material, figure S2). Hence, our results were robust and seem general. Our results are in line with previous studies raising the question of the relationship between plasticity and fitness and highlight the challenges in differentiating between adaptive and non-adaptive plasticity [32,49]. Studies targeting few species and few traits continue to grow evidence that challenges the generalization of adaptive plasticity. For example, by evaluating intrinsic resources and metabolic traits [50], temperature [51,52] and light [53]. The patterns we reveal suggest that identifying adaptive signals from phenotypic plasticity studies may be beyond the reach of studies that focus only on one or few traits or one or a few species or populations. We stress a need for caution related to the expectation that plasticity is adaptive and suggest a re-evaluation of the generality of conceptual work based on the assumption that most plasticity is adaptive. We also encourage further explorations about the potential role of non-adaptive plasticity.

To critically reflect on our results that plasticity is not necessarily adaptive, we revisit the arguments behind our hypothesis. Our central argument is derived from basic evolutionary theories which suggest that the strength of selection varies with trait type, such that different selective forces should lead to different evolutionary outcomes. Our first assumption is that survivorship and reproductive traits are more closely related to fitness than other traits. Fitness, the population growth rate λ, is made up of fitness components, and both, the traits that we define as LHt as well as the other traits, can be seen as such components. Although N-LHt probably also influence fitness indirectly through their influence on reproduction and survival, the influence of LHt is more direct and therefore stronger [54]. For example, body size influences both survival and reproduction in many systems [55,56], but is not perfectly correlated with either [57], and is thus less closely related to fitness.

(b) Genetic canalization

Following from the proximity to fitness of LHt, selection for genetic canalization is expected to be stronger, and thus should reduce variability in plasticity among genotypes [8–10]; we did not find such patterns (figure 3). It could be

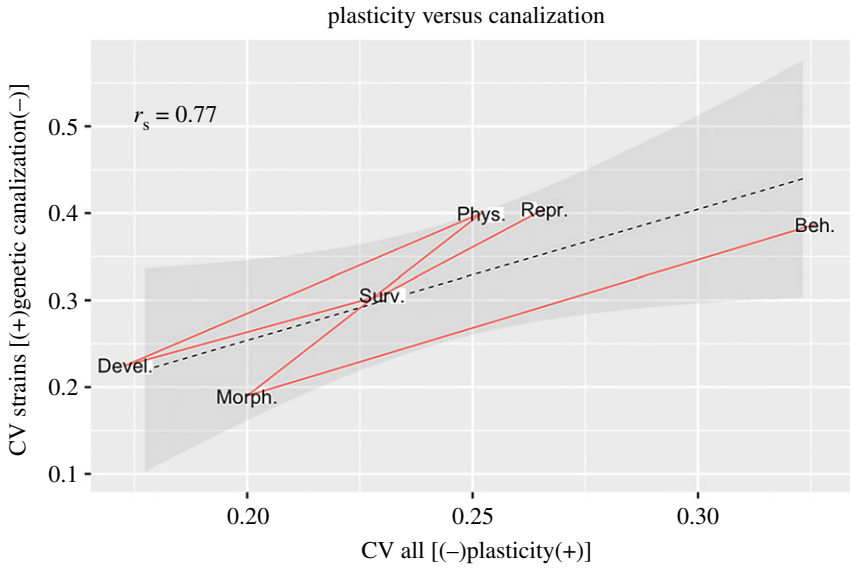

Figure 4. The correlation between plasticity and genetic variance (a reciprocal measure for genetic canalization) is significant ($r_s = 0.77$, Spearman). However, the order of the traits did not correspond to their proximity to fitness. The black dashed line illustrates the linear relationship between the trait types if ordered as follows: developmental, morphological, survivorship, metabolism and physiology, reproduction, and behaviour. The red (solid) lines link the traits in order as defined previously in relation to their distance to fitness: beh.-morph.-phys.-devel.-surv.-rep. If the distance to fitness was a main determining factor to the relationship between plasticity and genetic canalization, the lines should coincide. (Online version in colour.)

that higher genetic canalization might be observed within studies where all aspects of the experiment are considered together, but a comparison of levels of canalization of LHt and N-LHt within studies finds no more frequent lower variances in CV of LHt (results not shown).

The strong relationship between trait plasticity and canalization suggests the more plastic a trait, the less canalized it will be, supporting previous theories. However, we found that the relationship to fitness is not necessarily what determines the level of canalization. The lack of association indicates that the difference in selective forces for genetic canalization does not determine whether traits closely related to fitness (LHt) exhibit more or less plasticity compared with N-LHt. According to our categorization, the traits were more fixed and less plastic resulting in the following order: developmental, morphological, survivorship, metabolism and physiology, reproduction, and behaviour. Interestingly, survival traits fell out in the middle of both the plastic and genetic canalization responses, which provide some support for some kind of buffering role among traits.

(c) Evolutionary theories of plasticity

We are by no means suggesting that there are no trade-offs between traits—we are questioning direct trade-offs with respect to their relationship to fitness. Theories of life-history evolution rely heavily on trade-offs among LHt [58]. If LHt negatively covary with each other, they could also buffer each other against environmental variation and thereby weaken the expected environmental buffering of N-LHt on LHt [14]. Supporting previous studies, we do not find negative correlations among LHt (electronic supplementary material, figure S4).

Most studies in an evolutionary ecological framework aim at conditions that are comparable to natural conditions. Maladaptive plastic responses are predicted to occur under rare or novel environments when cryptic genetic variation is released [59,60]. While our results suggest that non-adaptive or potentially maladaptive plastic responses might be common (figure 2), we cannot evaluate to what extent the different studies employed novel environments. From our own empirical work and from other meta-analyses on plasticity [61], the inclusion of novel environments in plasticity studies is frequent but by no means universal. The more studies that included novel environments, the more likely a bias against our hypothesis. But because we tested our hypothesis with several subsets of data and found similar results, this is most probably not the case. A more general challenge that we face, similar to other studies on plasticity, is not knowing what the optimal plasticity is in a given set of environments. For example, a plastic response that overshoots an intermediate optimum will also be seen as maladaptive, and such overshooting in an N-LHt would support our hypothesis that N-LHt are more plastic. This becomes even more challenging to quantify empirically as natural environmental variation is changing rapidly through climate and land use change.

(d) Plants

Previous investigations in plants have suggested that sessile and free-moving organisms might have different costs and limits of plasticity [36]. Our results show that plants show the highest level of plasticity which could be related to them being largely sessile, lacking some types of opportunity to avoid environmental variation through habitat selection. Such a limitation might lead to particularly strong selective forces for plasticity [62]. However, if this high level of plasticity is adaptive, we would expect that plants would exhibit less plasticity in LHt compared to N-LHt, but the reverse was observed (figure 1*b*). A meta-analysis of results of plant reciprocal transplant experiments also found no differences in the relative plasticity of LHt versus morphological traits [32]. Their results suggest however that, for those traits that are plastic, putatively adaptive plasticity is more common than non-adaptive responses. Whether this pattern holds for other

sessile organisms (e.g. certain fungi or marine invertebrates) is worthy of future-targeted investigations.

(e) Study limitations

We realize there is, as for most studies, a potential bias caused by the selected methodology. For instance, the scaled plasticity (CV) could introduce noise, but it is an accepted method to standardize variability among traits with fundamentally different units [63]. Our sample size should be sufficiently large for generalizing our overall findings, supported by the absence of a major publication bias (electronic supplementary material, figure S1), although relevance of findings for specific groups might suffer from low amount of data (e.g. green algae), and trait types might differ in their measuring error. Trait types are also expected to differ in their variance owing to biological characteristics, which could influence CVs. For example, behavioural traits might be measured with less accuracy and precision compared to morphological traits. However, there was no general pattern of difference in the errors between trait types. The results are robust to a number of other factors and do not depend on specific types of studies. These arguments give us confidence in our results, despite the highly variable and diverse patterns of plasticity detected. Certainly, we encourage others to follow up on these general understandings and patterns gained from our meta-analysis and continue to perform empirical studies of species for which we know that adaptive, non-adaptive (neutral) and maladaptive plasticity have been found.

5. Conclusion

Our results support growing evidence [32,64–66] that much plasticity might be neutral, or even maladaptive. Our study does not reveal the causes that prevent the evolution of adaptive plasticity, but there are many that can be posited: variability in environmental conditions is high enough that environmental cues might not be as reliable as assumed [15]; environmental frequencies do not select for plasticity as argued by others [67] or do not reflect selective histories; or costs and limits of phenotypes inhibit plasticity evolution [34]. We are not suggesting that plasticity is necessarily non-adaptive, and our results do not suggest so, but we call for caution about the generalizing expectations about adaptive plasticity. Our findings also relate to the larger question of how genetic variation in LHt and fitness components in natural populations is maintained [9,68].

Data accessibility. Data available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.72s8g4j [47].

Competing interests. We declare we have no competing interests.

Funding. C.M.M. and C.D.S. were supported by a grant from the National Evolutionary Synthesis Center (NESCent; NSF no. EF-0423641), and by the National Science Foundation (C.J.M.: IOS-1146977; C.D.S.: DEB-1046328).

Acknowledgements. We would like to thank the Bettencourt Foundation for its support, including the fellowship appointed to C.M.M. Special thanks to Alice Demarez, Josh Van Buskirk, Josh Auld, Anurag Agrawal, and Rick Relyea. Miguel Tejedo, Federico Marangoni, Cino Pertoldi, Alex Ritcher-Boix, Germán Orizaola, Alfredo G. Nicieza, David Álvarez, Iván Gómez-Mestre, Christer Brönmark, Thomas Lakowitz, Johan Hollander, Wilco C.E.P. Verberk, David T. Bilton, Anssi Laurila, Beatrice Lindgren, Ane T. Laugen, Marjan De Block, Mark A. McPeek and Robby Stoks for their direct contributions to the database.

References

1. Levin SA. 1992 The problem of pattern and scale in ecology: The Robert H. MacArthur Award Lecture. *Ecol.* **73**, 1943–1967. (doi:10.2307/1941447)
2. Whitman DW, Agrawal AA. 2009 What is phenotypic plasticity and why is it important. In *Phenotypic plasticity of insects: mechanisms and consequences* (eds DW Whitman, TN Ananthakrishnan), pp. 1–63. Enfield, NH: CRC Press.
3. Stoks R, Govaert L, Pauwels K, Jansen B, De Meester L. 2016 Resurrecting complexity: the interplay of plasticity and rapid evolution in the multiple trait response to strong changes in predation pressure in the water flea *Daphnia magna*. *Ecol. Lett.* **19**, 180–190. (doi:10.1111/ele.12551)
4. Sultan SE. 2015 *Organism and environment: ecological development, niche construction, and adaptation*. Oxford, UK: Oxford University Press.
5. Via S, Lande R. 1985 Genotype-environment interaction and the evolution of phenotypic plasticity. *Evolution* **39**, 505. (doi:10.2307/2408649)
6. Fisher RA. 1930 *The genetical theory of natural selection*. Oxford, UK: Clarendon Press.
7. Wright S. 1931 Evolution in Mendelian populations. *Genetics* **16**, 97–159.
8. Stearns SC, Kawecki TJ. 1994 Fitness sensitivity and the canalization of life-history traits. *Evolution* **48**, 1438. (doi:10.2307/2410238)
9. Kingsolver JG, Diamond SE, Siepielski AM, Carlson SM. 2012 Synthetic analyses of phenotypic selection in natural populations: lessons, limitations and future directions. *Evol. Ecol.* **26**, 1101–1118. (doi:10.1007/s10682-012-9563-5)
10. Waddington CH. 1942 Canalization of development and the inheritance of acquired characters. *Nature* **150**, 563–565. (doi:10.1038/150563a0)
11. Flatt T. 2005 The evolutionary genetics of canalization. *Q. Rev. Biol.* **80**, 287–316.
12. Schlichting CD, Pigliucci M. 1998 *Phenotypic evolution: a reaction norm perspective*. Sunderland, MA: Sinauer Associates Incorporated.
13. Pigliucci M. 2001 *Phenotypic plasticity: beyond nature and nurture*. Baltimore, MD: Johns Hopkins University Press.
14. Caswell H. 1983 Phenotypic plasticity in life-history traits: demographic effects and evolutionary consequences. *Am. Zool.* **23**, 35–46. (doi:10.1093/icb/23.1.35)
15. Reed TE, Waples RS, Schindler DE, Hard JJ, Kinnison MT. 2010 Phenotypic plasticity and population viability: the importance of environmental predictability. *Proc. R. Soc. B* **277**, 3391–3400. (doi:10.1098/rspb.2010.0771)
16. van Tienderen P. 1991 Evolution of generalists and specialist in spatially heterogeneous environments. *Evolution* **45**, 1317–1331. (doi:10.2307/2409882)
17. Ashby WR. 1956 *An introduction to cybernetics*. New York, NY: Wiley.
18. Bateson G. 1963 The role of somatic change in evolution. *Evolution* **17**, 529–539. (doi:10.1111/j.1558-5646.1963.tb03310.x)
19. Bateson G. 1979 *Mind and nature: a necessary unity*. New York, NY: Dutton, E.P.
20. Caswell, H., 2001. *Matrix population models: construction, analysis, and interpretation*. Sunderland, MA: Sinauer Associates.
21. Gaillard J-M, Yoccoz NG. 2003 Temporal variation in survival of mammals: a case of environmental canalization? *Ecology* **84**, 3294–3306. (doi:10.1890/02-0409)
22. Tuljapurkar SD. 1982 Population dynamics in variable environments. II. Correlated environments, sensitivity analysis and dynamics. *Theor. Popul. Biol.* **21**, 114–140. (doi:10.1016/0040-5809(82)90009-0)
23. Tuljapurkar S. 1990 *Population dynamics in variable environments*. New York, NY: Springer. (http://www.popline.org/node/376960).
24. Lande R. 2009 Adaptation to an extraordinary environment by evolution of phenotypic plasticity and genetic assimilation. *J. Evol. Biol.* **22**, 1435–1446. (doi:10.1111/j.1420-9101.2009.01754.x)
25. Hoffmann AA, Sgrò CM. 2011 Climate change and evolutionary adaptation. *Nature* **470**, 479–485. (doi:10.1038/nature09670)

26. Schlichting CD, Wund MA. 2014 Phenotypic plasticity and epigenetic marking: an assessment of evidence for genetic accommodation. *Evolution* **68**, 656–672. (doi:10.1111/evo.12348)
27. Charmantier A, McCleery RH, Cole LR, Perrins C, Kruuk LEB, Sheldon BC. 2008 Adaptive phenotypic plasticity in response to climate change in a wild bird population. *Science* **320**, 800–803. (doi:10.1126/science.1157174)
28. Valladares F *et al*. 2014 The effects of phenotypic plasticity and local adaptation on forecasts of species range shifts under climate change. *Ecol. Lett.* **17**, 1351–1364. (doi:10.1111/ele.12348)
29. Pfennig DW, Wund MA, Snell-Rood EC, Cruickshank T, Schlichting CD, Moczek AP. 2010 Phenotypic plasticity's impacts on diversification and speciation. *Trends Ecol. Evol.* **25**, 459–467. (doi:10.1016/j.tree.2010.05.006)
30. Schlichting C. 1986 The evolution of phenotypic plasticity in plants. *Annu. Rev. Ecol. Syst.* **17**, 667–693. (doi:10.1146/annurev.ecolsys.17.1.667)
31. Chevin L-M, Lande R, Mace GM. 2010 Adaptation, plasticity, and extinction in a changing environment: towards a predictive theory. *PLoS Biol.* **8**, e1000357. (doi:10.1371/journal.pbio.1000357)
32. Palacio-López K, Beckage B, Scheiner S, Molofsky J. 2015 The ubiquity of phenotypic plasticity in plants: a synthesis. *Ecol. Evol.* **5**, 3389–3400. (doi:10.1002/ece3.1603)
33. Chevin L-M, Hoffmann AA. 2017 Evolution of phenotypic plasticity in extreme environments. *Phil. Trans. R. Soc. B* **372**, 20160138. (doi:10.1098/rstb.2016.0138)
34. Callahan HS, Maughan H, Steiner UK. 2008 Phenotypic plasticity, costs of phenotypes, and costs of plasticity: toward an integrative view. *Ann. N. Y. Acad. Sci.* **1133**, 44–66. (doi:10.1196/annals.1438.008)
35. DeWitt TJ, Sih A, Wilson DS. 1998 Costs and limits of phenotypic plasticity. *Trends Ecol. Evol.* **13**, 77–81. (doi:10.1016/S0169-5347(97)01274-3)
36. van Kleunen M, Fischer M. 2005 Constraints on the evolution of adaptive phenotypic plasticity in plants. *New Phytol.* **166**, 49–60. (doi:10.1111/j.1469-8137.2004.01296.x)
37. Dorn LA, Hammond Pyle E, Schmitt J. 2000 Plasticity to light cues and resources in *Arabidopsis thaliana*: testing for adaptive value and costs. *Evolution* **54**, 1982–1994. (doi:10.1554/0014-3820(2000)054[1982:PTLCAR]2.0.CO;2)
38. Sultan SE. 2000 Phenotypic plasticity for plant development, function and life history. *Trends Plant Sci.* **5**, 537–542. (doi:10.1016/S1360-1385(00)01797-0)
39. Blanckenhorn WU. 2009 Causes and consequences of phenotypic plasticity in body size: the case of the yellow dung fly *Scathophaga stercoraria* (Diptera: Scathophagidae). In *Phenotypic plasticity of insects: mechanism and consequences*. (eds DW Whitman, TN Ananthakrishnan), pp. 369–422. Enfield, NH: CRC Press.
40. Blanckenhorn WU. 1998 Adaptive phenotypic plasticity in growth, development, and body size in the yellow dung fly. *Evolution* **52**, 1394. (doi:10.2307/2411309)
41. Houle D. 1992 Comparing evolvability and variability of quantitative traits. *Genetics* **130**, 195–204.
42. Palmer AR. 2000 Quasi-replication and the contract of error: lessons from sex ratios, heritabilities and fluctuating asymmetry. *Annu. Rev. Ecol. Syst.* **31**, 441–480. (doi:10.1146/annurev.ecolsys.31.1.441)
43. Schneider CA, Rasband WS, Eliceiri KW. 2012 NIH Image to ImageJ: 25 years of image analysis. *Nat. Methods* **9**, 671–675. (doi:10.1038/nmeth.2089)
44. Sayers EW *et al*. 2012 Database resources of the National Center for Biotechnology Information. *Nucleic Acids Res.* **40**, D13–D25. (doi:10.1093/nar/gkr1184)
45. Bates D, Mächler M, Bolker B, Walker S. 2014 Fitting linear mixed-effects models using lme4. *J. Stat. Softw.* **67**, 1–48. (doi:10.18637/jss.v067.i01)
46. Burnham KP, Anderson DR (eds). 2004 *Model selection and multimodel inference*. New York, NY: Springer.
47. Acasuso-Rivero C, Murren CJ, Schlichting CD, Steiner UK. 2019 Data from: Adaptive phenotypic plasticity for life-history and less fitness-related traits. Dryad Digital Repository. (https://doi.org/10.5061/dryad.72s8g4j/1)
48. Price TD, Qvarnstrom A, Irwin DE. 2003 The role of phenotypic plasticity in driving genetic evolution. *Proc. R. Soc. Lond. B* **270**, 1433–1440. (doi:10.1098/rspb.2003.2372)
49. Arnold PA, Nicotra AB, Kruuk LEB. 2019 Sparse evidence for selection on phenotypic plasticity in response to temperature. *Phil. Trans. R. Soc. B* **374**, 20180185. (doi:10.1098/rstb.2018.0185)
50. Norin T, Metcalfe NB. 2019 Ecological and evolutionary consequences of metabolic rate plasticity in response to environmental change. *Phil. Trans. R. Soc. B* **374**, 20180180. (doi:10.1098/rstb.2018.0180)
51. Jarrold MD, Chakravarti LJ, Gibbin EM, Christen F, Massamba-N'Siala G, Blier PU, Calosi P. 2019 Life-history trade-offs and limitations associated with phenotypic adaptation under future ocean warming and elevated salinity. *Phil. Trans. R. Soc. B* **374**, 20180428. (doi:10.1098/rstb.2018.0428)
52. Salinas S, Irvine SE, Schertzing CL, Golden SQ, Munch SB. 2019 Trait variation in extreme thermal environments under constant and fluctuating temperatures. *Phil. Trans. R. Soc. B* **374**, 20180177. (doi:10.1098/rstb.2018.0177)
53. Baker BH, Sultan SE, Lopez-Ichikawa M, Waterman R. 2019 Transgenerational effects of parental light environment on progeny competitive performance and lifetime fitness. *Phil. Trans. R. Soc. B* **374**, 20180182. (doi:10.1098/rstb.2018.0182)
54. Metcalf CJE, Pavard S. 2007 Why evolutionary biologists should be demographers. *Trends Ecol. Evol.* **22**, 205–212. (doi:10.1016/j.tree.2006.12.001)
55. Promislow DEL, Harvey PH. 1991 Mortality-rates and the evolution of mammal life histories. *Acta Oecologica* **12**, 119–137.
56. Shine R, Charnov EL. 1992 Patterns of survival, growth, and maturation in snakes and lizards. *Am. Nat.* **139**, 1257. (doi:10.1086/285385)
57. Coulson T, Tuljapurkar S, Childs DZ. 2010 Using evolutionary demography to link life history theory, quantitative genetics and population ecology. *J. Anim. Ecol.* **79**, 1226–1240. (doi:10.1111/j.1365-2656.2010.01734.x)
58. Stearns SC. 1992 *The evolution of life histories*. Oxford, UK: Oxford University Press. (http://www.ulb.tu-darmstadt.de/tocs/2418442X.pdf)
59. Ghalambor CK, McKAY JK, Carroll SP, Reznick DN. 2007 Adaptive versus non-adaptive phenotypic plasticity and the potential for contemporary adaptation in new environments. *Funct. Ecol.* **21**, 394–407. (doi:10.1111/j.1365-2435.2007.01283.x)
60. Scheiner SM. 2013 The genetics of phenotypic plasticity. XII. Temporal and spatial heterogeneity. *Ecol. Evol.* **3**, 4596–4609. (doi:10.1002/ece3.792)
61. Murren CJ *et al*. 2014 Evolutionary change in continuous reaction norms. *Am. Nat.* **183**, 453–467. (doi:10.1086/675302)
62. Bradshaw AD. 1972 Some of the evolutionary consequences of being a plant. *Evol. Biol.* **5**, 25–47.
63. Sokal R, Rohlf J. 1995 *Biometry: principles and practice of statistics in biological research*, 3rd edn. New York, NY: W. H. Freeman and Co.
64. Donohue K, Dorn L, Griffith C, Kim E, Aguilera A, Polisetty CR, Schmitt J, Galloway L. 2005 The evolutionary ecology of seed germination of *Arabidopsis thaliana*: variable natural selection on germination timing. *Evolution* **59**, 758–770. (doi:10.1554/04-418)
65. Caruso CM, Maherali H, Sherrard M. 2006 Plasticity of physiology in *Lobelia*: testing for adaptation and constraint. *Evolution* **60**, 980–990. (doi:10.1554/06-050.1)
66. Ghalambor CK, Hoke KL, Ruell EW, Fischer EK, Reznick DN, Hughes KA. 2015 Non-adaptive plasticity potentiates rapid adaptive evolution of gene expression in nature. *Nature* **525**, 372–375. (doi:10.1038/nature15256)
67. Sultan SE, Spencer HG. 2002 Metapopulation structure favors plasticity over local adaptation. *Am. Nat.* **160**, 271–283. (doi:10.1086/341015)
68. Merilä S. 2000 Lifetime reproductive success and heritability in nature. *Am. Nat.* **155**, 301–310. (doi:10.1086/303330)