

Causes of variation in genotype $\times$ environment interaction

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ABSTRACT

Questions: What sustains genetic variation in plasticity (genotype $\times$ environment interaction, $G \times E$) under selection across environments? What explains variation among traits and species in the expression of $G \times E$?

Hypotheses and methods: I review two hypotheses that seek to explain variation in the expression of $G \times E$. The grain-size hypothesis attempts to elucidate when selection can erode $G \times E$, as it shapes patterns of phenotypic plasticity across environments. The developmental architecture hypothesis identifies developmental features that make traits vary in the propensity to express $G \times E$. I also review studies that have addressed patterns of geographic variation in $G \times E$, and discuss metrics of the strength of $G \times E$ that will facilitate future comparisons across traits and taxa.

Conclusions: There is tentative support for both the grain-size and the developmental architecture hypotheses, but further work is required to explain the maintenance of $G \times E$ and variation in its expression. I describe scenarios in which, on the one hand, $G \times E$ may promote the maintenance of genetic variation and hinder sexual selection, versus on the other, promote evolutionary divergence under natural and sexual selection.

Keywords: genotype $\times$ environment interaction, maintenance of variation.

INTRODUCTION

The conditions that prevail in natural populations determine the course of evolutionary events. For instance, multiple causes of variation and adaptation combine to produce the patterns of geographic variation that we observe in nature (Carroll and Corneli, 1999; Thompson, 1999). Since these patterns constitute the ‘initial conditions’ for further ecological or evolutionary change, predicting the consequences of a change in the selection regime or of colonizing a new habitat requires taking into account the features of the populations involved. For example, consider two discoveries about natural populations that have drastically changed biologists’ expectations for the course of evolution. Over the last few decades, it has become

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clear that natural populations often contain reservoirs of genetic variation in fitness-related traits (Roff, 1997; Stirling *et al.*, 2002), and that such traits very often have the ability to express phenotypic plasticity (West-Eberhard, 2003). These two advances suggest that natural populations may be more likely to respond rapidly to changes in selection or in the environment than biologists have anticipated (West-Eberhard, 2003; Le Rouzic and Carlborg, 2007; Barrett and Schluter, 2008; Hahn, 2008). Thus, our growing understanding of the features of natural populations is refining our expectations for the course of evolution, as well as requiring evolutionary theory to be based on the patterns of variation on which selection will act – we need a natural history of the patterns of variation that occur in nature, and of their underlying causes.

Here, I attempt a natural history of a widespread form of variation that offers not only solutions to key problems in evolutionary theory but also novel challenges and implications for how we view the process of divergence. This form of variation is genetic variation in phenotypic plasticity, or genotype $\times$ environment interaction ($G \times E$). $G \times E$ is best conceptualized as variation in reaction norms. A reaction norm describes the phenotypes that a given genotype expresses in different environments. Figure 1 shows an example of strong $G \times E$, where reaction norms vary so much that they frequently cross from one environment to another. The extent of this reaction norm crossover corresponds to the strength of $G \times E$.

$G \times E$ plays a variety of roles in evolutionary thought. At the most basic level, it offers a potential solution to the problem of the maintenance of variation under selection: With strong $G \times E$ (as in Fig. 1c), the fitness ranking of genotypes varies from one environment to another in correspondence to the reaction norm crossovers, and selection favours different genotypes across environments. Thus, with sufficient migration among environments and/or generation overlap, $G \times E$ can help maintain genetic variation (Lynch and Walsh, 1998; Radwan, 2008).

The $G \times E$ solution to the problem of the maintenance of genetic variation also presents novel evolutionary problems. One of these is analogous to the problem of the maintenance of variation: genetic variation should be eroded by selection within environments, but it often is not, because of mechanisms such as $G \times E$ (see above). Similarly, $G \times E$ itself should be eroded by selection acting across environments (Via and Lande, 1985; Roff, 2002). However, it often appears not to be eroded. Instead, $G \times E$ seems to be common in traits of all kinds, including behavioural, morphological, and life-history traits and even strongly selected traits such as sexual ornaments (for reviews, see Greenfield and Rodríguez, 2004; Bussière *et al.*, 2008; Ingleby *et al.*, 2010). $G \times E$ has also been documented in scaling relationships between behavioural and morphological traits and body size [i.e. in allometric relationships (Rodríguez and Al-Wathiqui, 2012)]. In short, $G \times E$ appears to be maintained under selection at least sometimes, and this requires explanation. A related problem arises from the observation that, although widespread, $G \times E$ is not universally present – it has sometimes been shown to be absent or weak. Furthermore, when present, it varies considerably between traits within studies and among species (Ingleby *et al.*, 2010; Rodríguez and Al-Wathiqui, 2011; Rodríguez, 2012). Thus, variation in the presence and strength of $G \times E$ requires explanation.

I review two hypotheses that seek to explain variation in the presence and strength of the expression of $G \times E$. One hypothesis posits an explanation based on variation in the ability of selection to act on reaction norms. The other hypothesis contemplates developmental influences on the expression of $G \times E$. There is tentative support for both hypotheses, and I make suggestions that may help us arrive at a comprehensive view of the evolution of $G \times E$.

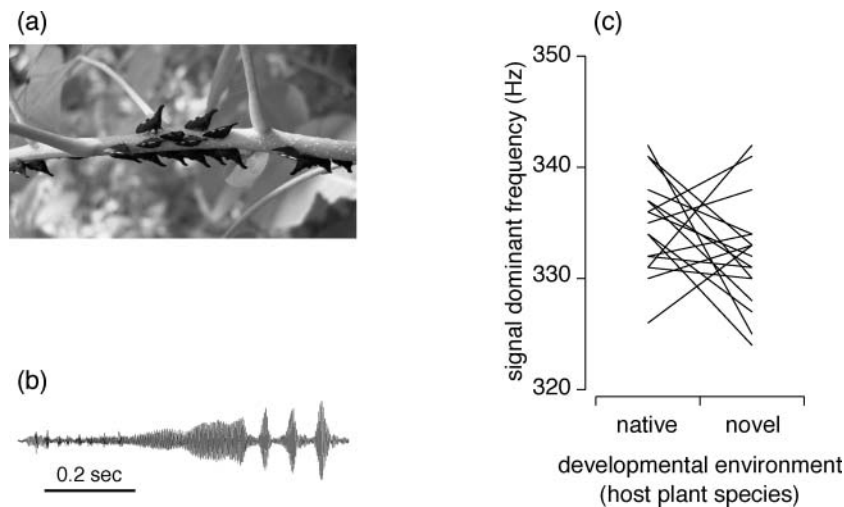


Fig. 1. Example of strong $G \times E$ in the mating signals of an insect. (a) This study focused on a member of the *Enchenopa binotata* species complex of treehoppers (Hemiptera: Membracidae). The picture shows an aggregation of the treehoppers on a stem of their host plant species, *Ptelea trifoliata* (Rutaceae). (b) These treehoppers communicate with signals that are transmitted along the tissues of the host plant. A male's signal consists of a near pure tone structured in one long initial component, followed by a series of shorter pulses. (c) Reaction norms for the dominant frequency (pitch) of male mating signals. Each reaction norm shows the mean signal frequency for a full-sibling family of the treehoppers, reared on two environments: their native host plant (*P. trifoliata*) and a novel host (that of another member of the complex: *Robinia pseudoacacia*, Fabaceae). Full-sib families serve as the proxy for genotype differences. This example shows considerable variation in reaction norms, indicating strong genetic variation in developmental plasticity (i.e. strong $G \times E$). Panel (c) modified from Rodríguez *et al.* (2008). Photo: G. Höbel.

WHEN CAN SELECTION ERODE $G \times E$? THE GRAIN-SIZE HYPOTHESIS

The first hypothesis is framed in terms of the ability of selection to favour adaptive plasticity and thereby erode genetic variation in plasticity, or $G \times E$ (Rodríguez, 2012). That is, this hypothesis addresses the ability of selection to favour some reaction norms and disfavour others, decreasing variation in reaction norms. The hypothesis posits that this ability of selection will vary as a function of the 'grain' of the environment, which describes the scale at which populations experience environmental heterogeneity in relation to generation time. At fine grains, individuals from any generation encounter various types of environment; at coarser grains, any generation encounters fewer types of environment (Gillespie, 1974). Thinking in terms of grain size, therefore, explicitly links the environmental factors influencing the expression of $G \times E$ to the scales at which selection can be effective (Fig. 2). I therefore refer to this hypothesis as the 'grain-size hypothesis' (Rodríguez, 2012). This hypothesis is, in turn, an adaptation of the 'grain-size model' for the evolution of adaptive plasticity (Levins, 1968; Hollander, 2008; Baythavong, 2011). The grain-size model states that adaptive plasticity can evolve in fine-grained environments, where environmental

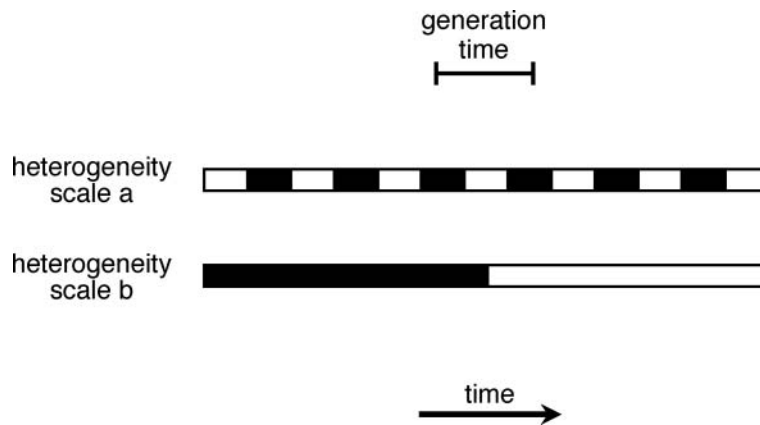


Fig. 2. The grain size of the environment is a function of the scale of environmental heterogeneity and generation time. In other words, the grain size of the environment describes the scale at which a population experiences environmental heterogeneity, from the point of view of the population's generation time. The horizontal bars show two possible rates of change in the environment over time, fast and slow. For a species with a generation time of the span shown, the scale of environmental heterogeneity a would yield a fine-grained environment, because each generation is likely to experience heterogeneity. In contrast, for the same species with the same generation time, the scale of environmental heterogeneity b would yield a coarse environment, because several generations are likely to occur before the environment changes.

heterogeneity occurs within the window of operation of selection; at coarser grains, selection loses the power to shape reaction norms.

The rationale for applying the grain-size model to the expression of $G \times E$ is that $G \times E$ represents genetic variation in reaction norms. It should therefore be possible to understand variation in $G \times E$ in terms of when selection can be expected to shape plasticity and thus erode variation in reaction norms (i.e. erode $G \times E$; Fig. 3). Specifically, the grain-size hypothesis posits that selection should be able to shape reaction norms (and thereby erode $G \times E$) in fine-grained environments, but not at coarser grains. Accordingly, this hypothesis predicts that $G \times E$ should be stronger or more frequently detected at coarser grains, and weaker or less often detected at finer grains. That is, the strength of $G \times E$ will increase with grain size.

I performed a preliminary test of this hypothesis with a survey of studies of $G \times E$ in sexual traits (Rodríguez, 2012). This survey included studies of $G \times E$ in sexual traits that met the following requirements: (1) it was possible to estimate the strength [effect size (Nakagawa and Cuthill, 2007)] of $G \times E$ from the P -value of the interaction terms in statistical models (Hunt *et al.*, 2004); and (2) either $G \times E$ was detected or, if not, genetic variation was detected – this second condition with the purpose of excluding cases where $G \times E$ may have been absent or weak not because of lack of genetic variation in plasticity *per se*, but because of lack of overall genetic variation in the trait concerned. These criteria admitted 17 studies on 12 different species into the analysis. I then categorized the grain at which studies assessed $G \times E$ as fine ($\leq$ generation time), medium (few generations), coarse (many generations), and very coarse (thousands of generations). The crucial increase in the strength of $G \times E$ should occur between fine and medium grains – the transition that mostly affects the power of selection to shape reaction norms. In addition, because of instances of

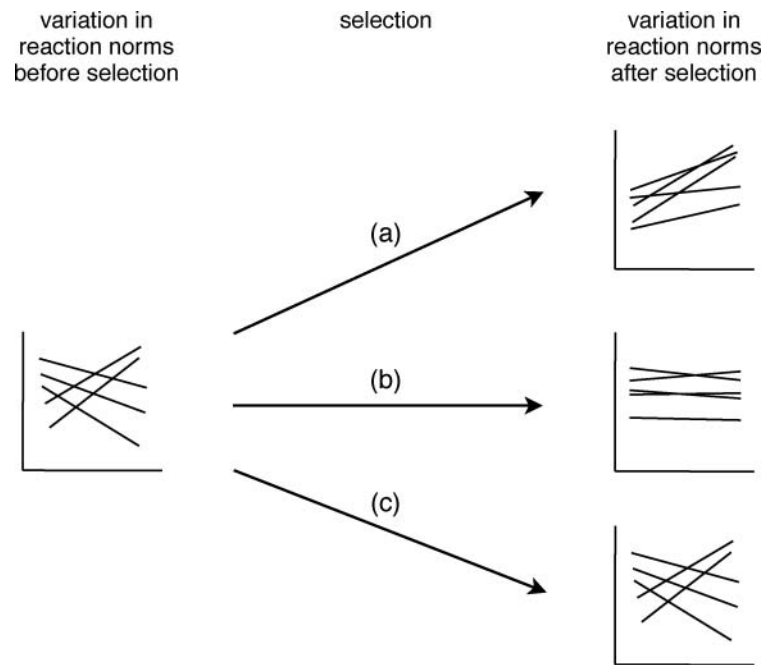


Fig. 3. Illustration of the potential outcomes of selection on reaction norms. The starting point is strong $G \times E$, or abundant genetic variation in plasticity. (a) Selection favouring adaptive plasticity would have the effect of reducing variation in the forms of plasticity (i.e. erode $G \times E$) and approximating the optimal type of reaction norm. (b) Selection disfavoring any form of plasticity would also erode $G \times E$ but with the effect of approximating 'flat' reaction norms. (c) Alternatively, $G \times E$ may be sustained under selection – it is my purpose here to begin to elucidate when, if ever, this may occur.

the evolution of adaptive plasticity in medium grains (e.g. Simpson *et al.*, 2011), a further increase in the strength of $G \times E$ from medium to coarse grains can also be predicted.

The survey detected both of the predicted increases in the strength of $G \times E$ with grain size: $G \times E$ was weaker in fine-grained environments, intermediate in strength at medium grains, and strongest at coarser grains (Fig. 4). This pattern lends support to the notion that selection can erode $G \times E$ in fine-grained environments, but that at coarser grains the scale of the expression of $G \times E$ (i.e. the scale of heterogeneity in the environmental variables that influence the expression of $G \times E$) is beyond the window of operation of selection. This offers a potential explanation for why we would observe $G \times E$ being maintained on some occasions but not others.

For future work on the grain-size hypothesis

The above results are encouraging, but more rigorous tests of the grain-size hypothesis are required, if nothing else because the sample of studies of $G \times E$ in sexual traits is relatively small. A more important limitation is that it is currently possible to relate the strength of $G \times E$ to grain size in a correlational analysis, but not to estimate the effect of grain size on the strength of $G \times E$, because no $G \times E$ study has manipulated grain size experimentally. Ideally, tests would use experimental manipulation of the grain size at which $G \times E$ is

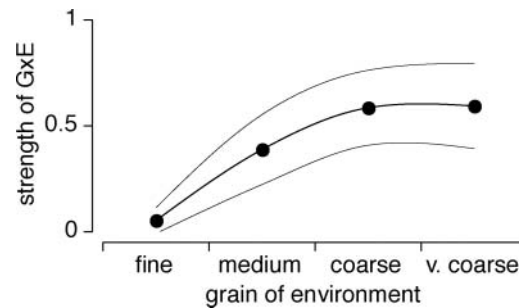


Fig. 4. In a survey of studies of $G \times E$ in sexual traits, the strength of $G \times E$ varied with the grain size of the environments across which $G \times E$ was evaluated in each study. Across four categories for grain size, each subsuming fairly broad ranges of variation in the scale of environmental heterogeneity, $G \times E$ was expressed more strongly at coarser and coarser grains. The figure shows the least square means ± 1 standard error extracted from the statistical models used to analyse the survey data (see text). Although in this analysis the x-axis corresponds to a categorical description of grain size, I have plotted spline functions to emphasize the continuous nature of this variable. Modified from Rodríguez (2012).

expressed. In order to conduct experimental tests of this hypothesis, it will be crucial to obtain accurate and adequate quantifications of grain size. Experimental tests of the hypothesis will need to determine the range of environmental conditions that constitute different grain sizes for the study species, and use the natural history of the species to design experimental grain size treatments.

Two recent studies designed the treatment environments used to assess $G \times E$ on the basis of quantification of the range of conditions experienced in the field by the source populations (Zhou *et al.*, 2008; Greenfield *et al.*, 2012). This is an extremely valuable approach, and the kind of information that it affords is exactly what is required to ‘calibrate’ the ranges of environmental conditions that represent different grain sizes for the species. For instance, fine grain sizes could be defined as the range of conditions that the species experiences regularly and can be expected to be adapted to, whereas coarser grain sizes could be defined as ranges of conditions that the species encounter less often and cannot be expected to be adapted to.

Geographic variation in $G \times E$

Another important contribution of the above two studies was to explore geographic variation in $G \times E$, by comparing patterns among sites (Zhou *et al.*, 2008; Greenfield *et al.*, 2012) and over time (Greenfield *et al.*, 2012). In both studies, the strength of $G \times E$ (quantified as the proportion of reaction norms that crossed over with other reaction norms between environments) was comparable among sites. However, a geographic variation approach suggests a refinement for testing the grain-size hypothesis: across a large enough latitudinal gradient (e.g. tropical vs. temperate sites), any one range of environments should represent a fine grain for some sites and a coarse grain for other sites (cf. Janzen, 1967; Tewksbury *et al.*, 2008). For example, for a study using two rearing temperatures (say, 15°C and 25°C), this difference of 10°C would represent a coarse grain for most lowland tropical species, but a fine grain for most temperate species. I think that such experiments would offer particularly robust predictions

to test the grain-size hypothesis. Yet additional leverage could come from assessing $G \times E$ across a variety of environmental variables (Bussière *et al.*, 2008; Zhou *et al.*, 2008; Greenfield *et al.*, 2012). It may be, for instance, that some variables are more stable than others in some sites (e.g. temperature is more constant than humidity in tropical sites) and vice versa in other sites (humidity more constant than temperature in temperate sites). This would offer extra within- and among-site contrasts for fine and coarse grains.

The survey used to test the grain-size hypothesis (Rodríguez, 2012) found considerable variation in the strength of $G \times E$ not only across grain-size categories but also within grain-size categories: for any one species, different traits often varied (sometimes quite dramatically) in the strength of $G \times E$ they expressed across the same set of environments. This points to additional factors that influence the expression of $G \times E$. The next hypothesis deals with one such factor.

WHY DO SOME TRAITS EXPRESS STRONGER $G \times E$ THAN OTHERS? THE DEVELOPMENTAL ARCHITECTURE HYPOTHESIS

This hypothesis addresses the influence of the developmental architecture of traits on the strength with which they express $G \times E$ (Rodríguez and Al-Wathiqui, 2011). Its rationale is that architectures evolved under selection in a given set of environments may also influence trait expression in other environments. In other words, trait architectures that have been shaped by selection in the range of conditions commonly encountered by a population (those representing fine grains) may still influence trait expression when the population encounters rare or novel conditions. Trait architectures would be the outcome of selection, but their influence on $G \times E$ expressed across familiar and novel environments would be unselected. Thus, this hypothesis represents the influence of proximate byproducts of evolved developmental processes.

The developmental architecture hypothesis focuses on two related features of the regulation of trait expression: canalization and condition-dependence. Canalization describes the resistance of phenotype expression against genetic or environmental perturbations (Flatt, 2005; Hansen, 2006). The rationale is that traits that have evolved to be highly canalized within a given set of environments may also prove to be canalized when expressed at a broader range of environments. Consequently, $G \times E$ expressed across environments representing coarse grains may be weak because of selection on canalization at the finer grains. Condition-dependence refers to the degree to which trait expression is influenced by an individual's available resources and/or by the overall health of its metabolism (Hunt *et al.*, 2004; Hill, 2011). Here, the rationale is that increased condition-dependence would increase the amount of overall developmental plasticity that a trait can express, thereby increasing its potential to express $G \times E$. In contrast, decreases in condition-dependence would limit how much overall plasticity a trait can express, and thus reduce its ability to express $G \times E$. Consequently, more strongly condition-dependent traits may express stronger $G \times E$.

In actuality, canalization and condition-dependence are likely correlated (negatively) with traits that are more highly canalized showing lower condition-dependence. Accordingly, I treat them as a single variable in a test of the developmental architecture (see below). Nevertheless, it is important to note that a trait can be both highly canalized and highly condition-dependent. For example, selection may favour a pattern of adaptive plasticity whereby a trait's form could vary dramatically with individual condition [as in the horns of

horned beetles, for example (Emlen, 2000)] yet nevertheless each of the forms could be tightly regulated. It is thus prudent to separate canalization and condition-dependence in the process of outlining this hypothesis.

An ideal test of the developmental architecture hypothesis would involve a comparative analysis or meta-analysis of the strength of $G \times E$ across studies and species involving traits varying in canalization/condition-dependence. To my knowledge, this is currently not possible, because we lack information about these trait features in studies of $G \times E$. An additional limitation is that, although it is possible to categorize traits according to *expectations* regarding canalization and condition-dependence (e.g. there are classes of traits that are likely to be especially highly canalized and less condition-dependent, such as genitalia – see below), there is nevertheless a scarcity of studies of $G \times E$ across such trait types. For example, in the above survey of studies of $G \times E$ in sexual traits (Rodríguez, 2012), only one study dealt with genitalia.

Owing to the above limitations, tests of the developmental architecture hypothesis have to begin with within-species comparisons of the strength of $G \times E$ across different types of traits. I conducted such a preliminary test (Rodríguez and Al-Wathiqui, 2011). Working with one species, I identified traits differing in expected levels of canalization/condition-dependence, and compared the strength of $G \times E$ they expressed across native and novel environments – with the native environment representing conditions likely to have played a role in shaping trait developmental architecture, and the novel environment offering the lever to test the consequences of those architectures for the expression of $G \times E$. The key to this test was using the evolutionary history of the clade to which the study species belongs – the *Enchenopa binotata* complex of treehoppers (Hemiptera: Membracidae) – to help inform expectations about differences in trait developmental architecture. Speciation in this clade of plant-feeding insects has involved a strong pattern of divergence in communication systems associated with colonizing and adapting to novel environments (host plants) (Cocroft *et al.*, 2008, 2010). This history of divergence offers an extremely convenient identifier for developmental environments that may be considered native (the host plant species used by a given member of the complex) versus novel (the host plant used by a different member of the complex). Furthermore, the pattern of divergence of the *E. binotata* complex also offers a useful lever for comparison between different types of traits, according to the following rationale: male mating signals are the most divergent traits among the phenotypes of adult members of the complex (Cocroft *et al.*, 2010). In contrast, there has been very little divergence in adult body morphology and negligible divergence in genitalic morphology (Pratt and Wood, 1993; Cocroft *et al.*, 2008; Hamilton and Cocroft, 2009). This pattern – of strong and recent divergence in signals at one extreme and apparent stasis and stabilizing selection on genitalia at the other extreme – offers the expectation of greater canalization in genitalia than in body traits and signals.

On the basis of the above reasoning, the developmental architecture hypothesis makes the following prediction for the *E. binotata* complex: $G \times E$ should be weakest in genitalia and strongest in signals. Testing this prediction requires controlling for the overall amount of genetic variation present in different traits. This is because the prediction requires weak $G \times E$ to represent little genetic variation *in plasticity*, not little genetic variation overall in the trait. I therefore compared the strength of $G \times E$ across a sample of traits measured from male mating signals, male body morphology, and male genitalic morphology, including trait broad-sense heritability as a covariate in the analysis (Rodríguez and Al-Wathiqui, 2011).

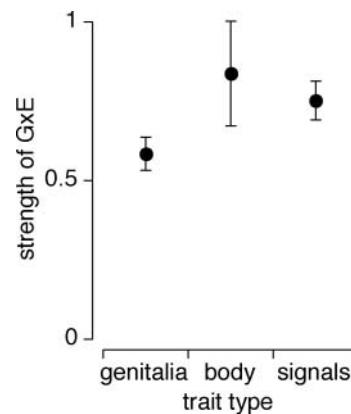


Fig. 5. Variation in the strength of $G \times E$ across three trait types in the males of a member of the *Enchenopa binotata* species complex of treehoppers (Hemiptera: Membracidae). The trait types (genitalia, body, and mating signals) represent expected differences in developmental architecture, with genitalia being the most canalized and least condition-dependent (see text). The figure shows least square means ± 1 standard error extracted from an analysis that took into account variation in the heritability of different traits. This analysis revealed small but significant differences in the expression of $G \times E$ across trait types, with genitalia expressing weaker $G \times E$. Note that $G \times E$ was in all cases relatively strong, which may reflect the fact that it was expressed across a very coarse grain of environments (native and novel host plant species; cf. grain-size hypothesis). Modified from Rodríguez and Al-Wathiqui (2011).

The results of the above comparison were as follows: genitalia did show the weakest levels of $G \times E$, as predicted, but signals did not show stronger $G \times E$ than body traits (Fig. 5). This pattern thus lends support for the developmental architecture hypothesis: there are differences among types of traits in the strength of $G \times E$ that they express.

For future work on the developmental architecture hypothesis

The above results should be taken as preliminary, for various reasons. To begin with, the test involved only a comparison among trait types for a single species. In addition, it did not measure the level of canalization/condition-dependence of the different traits. Instead, it categorized them according to expectations derived from the evolutionary history of the species complex. Ideally, future tests of this hypothesis will be able to conduct meta-analyses involving more rigorous categorization of the level of canalization and condition-dependence of different kinds of traits varying in their expression of $G \times E$.

One reason to be hopeful about the feasibility of broader and more refined tests of the developmental architecture hypothesis is that there is a wealth of evidence that strongly suggests that genitalia are generally highly condition-independent and highly canalized (Arnqvist and Thornhill, 1998; House and Simmons, 2007). For instance, genitalia scale very poorly with body size (Eberhard *et al.*, 1998, 2009; Eberhard, 2009). Thus, the prediction that genitalia should express relatively weak $G \times E$ should be broadly testable across a wide variety of animal species once $G \times E$ in genitalia has been assessed for a sample of species.

DISCUSSION

Improving our understanding of the evolution of $G \times E$ will require comparing the expression of $G \times E$ across species or populations and traits. The studies reviewed here (Rodríguez and Al-Wathiqui, 2011; Rodríguez, 2012) compared the overall strength of $G \times E$ with effect size estimates calculated from the genotype $\times$ environment interaction term in statistical models. This seemed to afford the widest possible sampling among studies of $G \times E$, but other metrics might be just as good if not better. For instance, variation in reaction norms is best described in terms of differences in the ‘slope’ of reaction norms, and stronger $G \times E$ means that reaction norms are less parallel and cross more among environments. Thus, the strength of $G \times E$ can be quantified as the proportion of reaction norms that cross other reaction norms (e.g. Zhou *et al.*, 2008; Greenfield *et al.*, 2012). It might be well for future studies to report both the statistics associated with the genotype $\times$ environment interaction terms from statistical models as well as the incidence of reaction norm crossover.

It will take considerable effort to describe and assemble a synthesis of the patterns of variation in the expression of $G \times E$ that occur in nature. I think the payoffs of such efforts will be large and varied. Understanding the causes of variation in $G \times E$ will have many useful applications to evolutionary theory. One advance that is beginning to take shape involves taking the grain of the environment into account when considering the consequences of $G \times E$ for the theory of sexual selection and its role in divergence.

The role of $G \times E$ in divergence by means of sexual and natural selection

$G \times E$ has an especially important role in the theory of sexual selection, where the maintenance of genetic variation under selection is particularly challenging because of the strength and constancy of sexual selection, and because of the rapid evolution it generates (West-Eberhard, 1983; Siepielski *et al.*, 2011). Witness the name often given to the problem of the maintenance of genetic variation under selection: the ‘lek paradox’. As discussed above, $G \times E$ offers a potential solution to this problem: when the relative attractiveness of genotypes changes among environments (e.g. Fig. 1c), overall genetic variation may be sustained. This brings an additional complication within the context of sexual selection: the change in relative attractiveness across environments that is caused by $G \times E$ will disrupt the patterns of assortative mating that would otherwise be established within each environment – indeed, this is how $G \times E$ can help maintain genetic variation in sexual traits. But disruption of the patterns of assortative mating may hinder the basic mechanism of sexual selection – that is, Fisherian selection, wherein assortative mating and genetic variation in sexual traits establish sexual genetic covariance that sustains and promotes mate choice (Fisher, 1958; Mead and Arnold, 2004; Prum, 2010). If this process is disrupted, mate choice may be disfavoured or mate preferences may be selected to attend to ornament traits not expressing $G \times E$, and sexual selection may weaken or cease (Greenfield and Rodríguez, 2004; Kokko and Heubel, 2008; Higginson and Reader, 2009).

This is where the considerations presented in this paper reveal a range of scenarios that may arise from $G \times E$ ’s disruption of patterns of assortative mating. Consider, for example, the potential role of the grain of the environments across which $G \times E$ is expressed. At fine grains, mate choice should indeed be disfavoured and sexual selection hindered. But at coarser grains, selection could not disfavour mate choice, because $G \times E$ and disruption of

patterns of assortative mating would occur at scales beyond the window of operation of selection – as discussed above (Rodríguez, 2012). That is, mate preferences could not be selected to avoid ornament traits exhibiting $G \times E$, because selection is ineffective at such coarse grains. Instead, sexual selection would continue after the disruption caused by the (relatively rare) expression of $G \times E$, but with altered patterns of assortative mating and sexual covariance in each of the different environments. This means that the dynamics of sexual selection would change between environments because of the expression of $G \times E$, potentially promoting divergence by means of sexual selection (Rodríguez and Greenfield, 2003; Rodríguez *et al.*, 2008). In effect, disruption of Fisherian selection may amount to a change in the regime of sexual selection and promote divergence among environments. Even if disruption events are rare, they can be influential, as with the colonization of novel environments (Schluter, 2001; Rundle and Nosil, 2005; Cocroft *et al.*, 2008; Nosil, 2012). Note that these consequences for divergence by sexual selection would arise ‘spontaneously’ – that is, they would arise from the simple yet unavoidable facts of environmental change and how it influences the expression of existing pools of genetic variation in natural populations. Consequently, this represents a potential ‘default’ mechanism for how divergence may begin, regardless of whether other mechanisms are at play.

As another example, consider how differences in trait developmental architecture may interact with the above effects. If there are specific kinds of traits that are especially unlikely to express $G \times E$ (e.g. genitalia, potentially), then the course of sexual selection is likely to be more stable for those traits than for others, and environmental change is less likely to promote divergent sexual selection for those trait types. Conversely, for trait types more prone to express $G \times E$, the course of sexual selection may be more unstable or more often halted, but environmental change may be more likely to promote divergence by means of sexual selection.

The divergence-promoting consequences of $G \times E$ may not be limited to the genetic dynamics of sexual selection; instead, they may also arise from a more general effect of the expression of $G \times E$ on the response to either sexual or natural selection (Rodríguez *et al.*, 2008). One of the main ways in which $G \times E$ is expressed is as among-environment changes in the genetic correlations between traits (Stearns *et al.*, 1991; Rodríguez *et al.*, 2008). Such changes may also entail the scaling (i.e. allometric) relationships between traits (Rodríguez and Al-Wathiqui, 2011). $G \times E$ thus sets the conditions for phenotypic differences to appear among environments, with two main consequences: First, even if selection does not vary among environments, the response to selection may differ because populations follow different trajectories according to the newly-expressed patterns of genetic correlations. Second, as phenotypic differences appear, the underlying developmental mechanisms may become differentially exposed to selection in the different environments, thereby promoting evolutionary change (cf. West-Eberhard, 2003).

CONCLUSION

In this paper I have reviewed two hypotheses seeking to explain variation in the expression of $G \times E$ (the grain-size and developmental architecture hypotheses), and I have discussed preliminary tests that yield support for both hypotheses. My goal has been to suggest these hypotheses and considerations as ideas in need of formal modelling and empirical testing. The payoff of engaging in this work will be to increase our ability to predict sizeable aspects of the course of evolution for whole classes of traits according to the patterns of variation

that prevail in natural populations and how they are influenced by environmental variation and change.

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