**Adaptive variance in labile traits and its expected evolutionary change**

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**Abstract**

Closer integration between behavioral ecology and quantitative genetics has a resulted in an increase in studies quantifying among- and within-individual sources of variation in labile traits. Consistent individual differences are commonly documented, and their existence is generally explained using adaptive arguments. However, the lack of a quantitative null model predicting the expected non-adaptive behavioral variation around an optimal phenotypic value makes it difficult to assess the adaptive nature of the observed patterns. We argue that estimating expected evolutionary change in trait variances across generations provides a way forward to test adaptive theory concerning behavioral variation. We describe an extension of the Price equation and the breeder’s equation that allows estimating the expected evolutionary change in the additive genetic, developmental and within-individual variance. This framework provides a multi-level approach to the study of adaptive evolutionary changes in trait variance for labile traits.

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**Introduction**

Evolutionary ecologists have traditionally been concerned with the evolution of the optimal mean phenotype in a population under a given a set of ecological conditions (Davies et al. 2017). Understanding the adaptive evolution of trait variances in response to ecological pressures has received far less theoretical and empirical attention (Bulmer 1981; Bull 1987). Recent calls for closer integration between behavioral ecology and quantitative genetics has resulted in an increased focus on the evolutionary ecology of behavioral variation within populations (Westneat & Fox 2010; Dingemanse & Dochtermann 2013), especially in the form of ‘animal personality’ (Réale et al. 2010; Carere & Maestripieri 2013). A crucial but underexamined claim that has arisen from these studies is that consistent individual differences in behavior are adaptive (Bergmüller & Taborsky 2010; Dall et al. 2012; Dingemanse & Réale 2013), implying that among- and within-individual behavioral variance has been shaped by selection in a similar way as the mean behavior. However, few studies have estimated the expected evolutionary change in behavioral variance in response to selective pressures.

The mere existence of non-zero among-individual differences in behavior or any other labile trait does not necessarily imply that this phenotypic variation is adaptive. The infinitesimal model from quantitative genetics provides the basis for a null expectation for the variance in a trait. First introduced by Fisher (1918), the infinitesimal model has proven extremely useful for estimating the additive genetic variance of phenotypes and predict evolutionary change in a wide array of study systems, particularly in breeding programs (Barton et al 2017). It states that additive genetic variance in a quantitative trait can be quantified assuming that its expression is determined by an infinitely large number of genes and environmental factors each with very small effects (Fisher 1918). Such a combination of genetic and (short- and long-term) environmental effects could explain most observed individual differences in behavior without invoking any adaptive explanation. However, behavioral variance likely results from a combination of non-adaptive and adaptive process operating at different time scales.

Variation in quantitative traits is ubiquitous, and there has been extensive analysis and discussion as to what maintains genetic variation. This requires some balance between the input from mutation and loss by drift and by most, if not all, selective forces acting

directly on the trait itself or through pleiotropic effects. There is not yet, however, an unequivocal conclusion as to how the typical levels of genetic variance are maintained (e.g. Bürger, 2000 ; Johnson & Barton,2005 ; Zhang & Hill, 2005 a ; Hill, 2010). Much less attention has been paid to factorsaccounting for the magnitude of the V P , V E or the CV. Understanding the forces that determine the magnitude of the non-genetic component of phenotypic variance is a broad question in evolutionary biology.

Since the genotype for the magnitude of environmental variation can be regarded as a quantitative trait, it is assumed to be determined by the actions and interactions of multiple genes. Much of the standard methodology of quantitative genetics can then be invoked. Assuming that the environmental components of the phenotype have a genetic basis thus implies that the repeatability of the trait can evolve.

Various non-adaptive processes, such as immigration and mutation, are thought to increase or maintain the amount of additive genetic variation in traits experiencing directional or stabilizing selection (see Lande 1992; Armbruster & Schwaegerle 1996; Whitlock et al. 2002). In the absence of immigration and mutation, sustained directional or stabilizing selection for an optimum phenotypic value is intuitively expected to erode additive genetic variation. However, under the infinitesimal model, reductions in genetic variance associated with selection are expected to be mostly temporary (Bulmer 1981). This is because the extent of any permanent reduction in additive genetic variance due to selection decreases as the number of loci involved increases. In traits influenced by many loci, permanent reductions will therefore be small compared to temporary ones. Most of the reduction in genetic variance is expected to be temporary because it is caused by selection inducing correlations between pairs of loci leading to linkage disequilibrium. When selection ceases, the joint equilibrium between pairs of loci will be reestablished and the additive genetic variance will return to its baseline value. Therefore, even under strong selection, we always expect a certain amount of genetic variance in populations and thus some among individual variation if the average phenotype in the population is close to or fluctuates around the optimal phenotypic value.

Phenotpic variation among-individuals within the same population will partly reflect plastic responses to environmental variation during development (see West-Eberhard 2003). If the optimal phenotype differs depending on the developmental environment, adaptive plasticity allows individuals to adjust their phenotype to match the optimum value for that environment, given the costs of plasticity and if the environment is sufficiently predictable (see Botero et al. 2015; Tufto 2015). Adaptive among-indivdiual variance can thus be driven by plasticity moving individuals closer to the optimum phenotype given their developmental environment. However, not all non-genetic variation in behavior involves the predictable effects of adaptive plasticity. Non-adaptive random phenotypic variation can be caused by developmental instability because of unpredictable stochastic environmental effects (Falconer & Mackay 1996). While the evolution of adaptive plasticity has been the focus of much theoretical and empirical research, how selection shapes developmental instability causing among-individual phenotypic variation has received far less attention.

Changes in developmental variance can be caused by the evolution of population sensitivity to the environment via individual abilities to buffer phenotypic expression from stochastic environmental noise (Fig.1). We expect some level of adaptive environmental canalization if developmental instability moves phenotypes away from their optimal mean value (Waddington 1942) or optimal reaction norm. At least whenever possible given the expected diminishing returns of any phenotypic accuracy (Hansen et al. 2006).

In a similar fashion as developmental plasticity, reversible phenotypic plasticity allows individuals to get as close as possible to the optimal phenotypic expression by adjusting to its “current” environment. However, there is substantial within individual variation in phenotypic expression that does not seem to be associated to adaptive reversible plasticity. We expect environmental canalization to minimize any within-individual deviations in phenotypic expressions of behavior from the optimal reaction norm of each individual (see Westneat et al. 2015). In the context of adaptive consistent individual differences in behavior, the optimum level of within-individual behavioral variance may also depend upon the relative amount of among-individual behavioral variance. For instance, individual repeatability can have important implications for reliable communication concerning individual attributes (see Bradbury & Vehrencamp 2011), leading to hypothesis about the signaling advantages of consistent individual differences in signaling within social groups (Dall et al. 2012).

Unfortunately, in most natural populations we have no quantitative predictions of the level of among- or within individual variation we expect to see around a mean phenotypic value under stabilizing selection, making it difficult to determine how much of the observed behavioral variation has an adaptive origin. In this paper we suggest studying the adaptive evolution of variation, by explicitly quantifying the expected evolutionary changes of the different sources of variation underpinning labile traits. We describe extensions to the Price equation (Price 1970) and the breeder’s equation that can be used to study evolutionary changes in trait variance at the among- and within-individual levels. These equations allow predicting the expected evolutionary change in the variance of labile traits across generations when stochastic perturbations have moved the population away from the optimal variance or when the population is evolving towards a new optimal level of trait variation. We use individual based simulations to describe processes that are hypothesized to result in adaptive changes in variation and show how to empirically parametrize the presented models.

**Evolutionary Change in Trait Variances**

Evolutionary quantitative genetics has mostly focused on the evolutionary dynamics of the mean phenotype () of a population:

, (1)



where is a vector of phenotypic values of a population with individuals.

The Price equation (Price 1970) can be used in the context of a quantitative genetics framework to describe evolutionary changes in mean phenotype from one time step to the next :



(2)



where the first term on the right-hand side represents natural selection as the covariance between fitness () and the individual trait value (), standardized by the mean fitness in the population (), while the second term represents transmission bias as a fitness-weighted average of between-generation (i.e. parent-offspring) deviations in trait values. Here, our focus is on the variance in a phenotypic trait:



. (3)



We can thus express changes in phenotypic variance using the Price equation (see Lehtonen 2018) as:

(4)



where we consider as a trait of the individual reflecting an absolute deviation from the population mean. Changes in the mean value of this new trait ) will result in changes in the variance of trait () in the population, because the variance in a population is by definition the average squared deviation from the mean (eq 3). This is a similar expression of stabilizing or disruptive selection acting a phenotypic trait, generally presented in its multiple regression form (see below).



We can expand equation 4 based on this decomposition to formulate a multi-level expression of the Price equation predicting changes in phenotypic trait variance:

(6)



where reflects selection associated to an individual’s genetic (squared) deviation from the population mean value, selection on the susceptibility to environmental effects deviating (negatively or positively) an individual’s phenotype from the mean phenotype in the population, and selection on the susceptibility to environmental variation causing deviations of the different phenotypic expressions within an individual from its own mean.



For more convenient empirical use, we can express the causal processes affecting the evolution of phenotypic variance using a multiple regression equation (Lande & Arnold 1983), as:

(7)



where is the linear selection gradient relating the phenotype () of an individual to its relative fitness (, describing the strength of directional selection on the mean phenotype. The second term reflects the strength of selection on the squared deviation of an individual’s average phenotypic expression from the population mean, where is the selection gradient associated to the effects on fitness of (). This second term is thus equivalent to a quadratic selection gradient, with trait values being mean centered on the population mean in each selection episode (e.g., year). When this second term is negative, it reflects stabilizing selection, which it is expected to decrease variance. When it is positive it reflects disruptive selection, which is expected to increase variance. Following the approach above, we can expand this second term to give:



(8)



The expected evolution of the among- and within-individual variance components is a function of the selection gradients and , respectively. These coefficients thereby allow empirically linking individual fitness to the individual’s average deviation from the population mean () and the average deviation of each expression from that individual’s own mean (. While theoretical models often use gaussian fitness functions in their evolutionary predictions and patterns of non-linear selection have been the focus of many studies, their expected effect on the different components of phenotypic variance within populations has generally been overlooked, especially in empirical studies.



The expected change in the direct genetic variance affecting a trait () can be estimated based on the estimates of (non-linear) quadratic selection and additive genetic variance in the trait (). The variance of a vector of squared values centered around zero equals two times the squared variance of the values, therefore the additive genetic variance in the squared deviation of an individual’s average trait from the population mean associated to direct genetic effects () equals 2 times the squared additive genetic variance of the trait (). Multiplying the quadratic selection gradient by two times the square of the additive genetic variances provides the expected change in additive genetic variance from one generation to the next:



(9)



Note that the estimate of quadratic effects needs to be divided by two to give the strength of stabilizing selection.

Predicting the expected change in developmental and within-individual variance is trickier. Although the two environmental components of the phenotype ( and ) are not themselves heritable, the susceptibility of an individual to such environmental effects is a property that may have a genetic basis (Bull 1987). For instance, when there is additive genetic variation in the sensitivity to the environment. To clarify this, we could express as a function of the trait sensitivity to developmental environment () and its developmental environment (), so . Sensitivity to the environment could be adaptive in the form of adaptive plasticity in response to changes in the environment or non-adaptive in the sense that individuals that are able to buffer the “negative” effects of environmental variation are favored. However, measuring this trait () directly and all the environmental variables () that may influence the phenotype of individuals is difficult. We can circumvent this problem by directly estimating the amount of additive genetic variance underpinning the squared deviation of an individual’s average phenotype from the population mean phenotype (). This additive genetic variance will have a component associated to direct genetic effects on the phenotype () and due to genetic variation on the susceptibility to environmental variation during development (,

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The genetic variance underpinning the susceptibility to environmental effects is therefore,

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and the expected change in permanent environmental variance due to selection is

Finally, the expected change in within individual variance due to selection can be predicted using the additive genetic variance in an individual’s average phenotypic deviations from its own mean () and the estimated relation of this value with fitness . Additive genetic variance in the within individual variance has already been estimated in a few studies, accompanied by the appropriate selection gradient we can estimate the expected change in within individual variance as

Importantly, this set of equations provide a quantitative genetics framework to study evolutionary changes in trait variance at multiple levels of biological organization.

**Individual based simulation**

We designed an individual based simulation to show how selection on an individual’s mean phenotype and its sensitivity to the “current environment” can shape the among and within-individual variance of a labile trait. We analyzed the data generated with the individual based simulation using the framework detailed above and show how to estimate the expected change in among- and within-individual variance from one generation to the next to compare this with the observed changes in variance after selection. We simulated a population with two sexes and non-overlapping generations. Individuals mate randomly for *g* generations, and then at generation *g,* the population experience non-linear selection on the mean phenotype and direct selection on its sensitivity to the “current” environment.

We specify the genetic variance in the mean phenotype of individuals by simulating the breeding values (**)** of the founder population. The breeding values are simulated from a normal distribution with mean of zero and variance . We also simulated environmental effects caused by the developmental environment and the environment when the phenotype was expressed, i.e current environment . These values were generated by simulating an individual’s sensitivity to the developmental environment () and to its “current” environment () for the founder population assuming a normal distribution with mean of 1 variance and , respectively. In this way the environment had almost no effect in some individuals (values closer to 0), whereas for some others the environmental effects had a stronger effect on their phenotype. Environmental values (and ) were simulated from a normal distribution with mean of zero and variance and respectively. Where and **.** The equation describing phenotypic expression is,

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Where **z** is a vector describing the observed phenotype. **, ,** andvary among individuals, while varies within individuals. We simulated 100 individuals for 5 generations, and we measured the expressed phenotype 10 times for each individual. Therefore, the pedigree consisted of 500 individuals and the total number of phenotypic observations was 5000. Following quantitative genetic theory, the breeding values for the mean phenotype and for the sensitivities to the developmental and current environment, of the new individuals in each generation was the average of the two parents plus the variance due mendelian inheritance, , and , respectively.

We then proceeded to impose selection and simulated the fitness of generation . Individual fitness was simulated as a function of an individual’s average phenotype () linear and quadratic effects on fitness, and/or its sensitivity to the current environment (), such that

Where w represents fitness in a latent scale that was then rescaled such that in each generation the average fitness in the population was always one, and thus the population is always at its equilibrium size. The fitness of individuals was then used as the mean of a Poisson process that generates the number of offspring produce by each individual.

The among- and within- individual variance in the new generation is expected to change due to the non-linear selection imposed on the mean phenotype and the linear selection imposed on the deviations from an individual’s mean phenotype.

We simulated a scenario in which the patterns of selection were expected to generate an increase in the among-individual variance in the trait () and a decrease in the within individual variance in the trait (). This was done by inducing disruptive selection on the mean trait () but selection against sensitivity to the current environment (. This may reflect a scenario of reliable identity communication where individuals need to advertise their accurately advertise their identity. In this biological scenario is good to be different.

**Statistical analyzes**

The statistical approach we advocate to study the expected adaptive evolutionary change on the among- and/or within-individual phenotypic variance in a population, hinges upon accurate estimates of additive genetic variation and selection gradients of the squared deviations of an individual’s mean behavior from the population mean (), and the average deviations of repeated behavioral expressions from the individual’s own mean behavior (). If not properly modelled, inaccurate empirical estimates of mean individual behavior will underestimate the among individual variance in these quantities with cascading consequences on the estimates of selection gradients and additive genetic variance in the squared deviations of the average phenotype of an individual form the population mean and the average squared deviation from the repeated expressions from the individual’s own mean. These problems have been documented when using within-subject centering techniques (Westneat et al. 2020), and also when studying non-linear selection gradients (Dingemanse et al. 2021). To overcome these problems, we advocate the use of error-in-variables models, as they provide a flexible and robust method for estimating accurate estimates of additive genetic variance and selection coefficients (Ponzi 2018).



Using error in variable models and analyzed the data in two steps (see supplementary material for R code). We first quantified the additive genetic variation in the simulated trait and the deviations from the mean of the population and the deviations of an individual’s own mean. To this end we performed three animal models. The first one, estimated the additive genetic variation in the trait (z). In the second we estimated the additive genetic variation in the squared of the individual’s mean trait deviation from the population mean . The third model consisted of an animal model estimating the additive genetic variance in average deviation of the repeated expressions from each individual’s own mean phenotype (). This last model was parametrized as an animal model estimating additive genetic variance underpinning individual specific within-individual variance.

We then proceeded to estimate the non-linear selection gradient on the mean phenotype, and linear selection on an individual’s sensitivity to variation in the “current” environment. We did this only for individuals breeding in generation g, as we imposed selection only on this generation. This was aimed to represent environmental change disrupting the equilibrium variance in the population and its subsequent return to equilibrium driven by the selective pressure. It is important to keep in mind that the equations presented above are designed to study expected evolutionary changes from one time step to the next from one time period to the next, and we need to carefully consider the specific hypothesis we are supposed to be testing if we simultaneously include multiple episodes of selection within the same statistical model.

**Results**

When comparing the observed change in variance associated to selection resulting from the individual based simulations with the predicted changes based on the estimates derived from the statistical models we find that the there is substantial dispersion across simulations on both the observed and predicted changes in the among and within individual variance. This suggests that studying changes in variance might be challenging (Fig 2). However, the average predicted and observed change across simulations are very much in agreement- i.e. black and red solid circle are close together in figure 2. The expected change in among individual variance is underestimated by 20%, while the expected change in within individual variance is underestimated by 18%.

It is also important to note that the predicted changes using the statistical models have the same sign as expected changes based on the inputted simulation values. For the among individual variance the predicted changes are always positive, while for the within individual variance the predicted changes are always negative. This is the case despite the observed changes in among individual variance sometimes being negative, despite the imposed disruptive selection. While the changes in within-individual variance being positive despite the imposde negative selection on an individual’s sensitivity to environmental variation. These results thus imply that it is possible to study the ecological pressures expected to produce changes in the among- and within-individual variance.

**Chart, scatter chart

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**Figure 2**. Comparison between the observed changes in among and within individual variance. The x axis represents the predicted change in variance estimated from the statistical models, and the y axis, the observed changes in variance in the individual based simulation. The open circles are values for each simulation and the solid black circle are the average across all the simulations. Red solid circle represents the expected values given the simulation parameters.

**Discussion**

Wright (1932) introduced the concept of adaptive topography in evolutionary biology by demonstrating that evolution of gene frequencies in a constant environment will maximize the mean fitness of the individuals in the population. Natural selection will cause changes gene frequencies moving populations upward on the adaptive topography, resulting in an increase in the mean fitness of the population. The formulation of such an adaptive topography has provided a theoretical foundation for analyses of many problems in evolutionary biology (Gavrilets 2004; Svensson and Calsbeek 2012). Lande (1976, 1979, 1982) and Lande and Arnold (1983) provided important conceptual and statistical advancements by developing a quantitative genetic theory for selection of the mean phenotype to study the adaptive topography for a normally distributed characters and estimate the expected change in mean phenotype of multivariate phenotypes. Here we describe the extension of this framework to incorporate the variance of a phenotype as part of its adaptive topography.

Phenotypic variation on phenotypes under stabilizing selection is expected to decrease population mean fitness (Lande & Shannon 1996). Therefore, selection should minimize any phenotypic variance, especially in traits that are closely linked to fitness. A key test of the adaptive nature of behavioural variation is thus to quantify when larger values of phenotypic variance will lead to higher mean fitness in a population. Focusing on the evolutionary changes in phenotypic variance can provide some insights on the shape of the adaptive topography of populations.

Certain hypotheses concerning the evolution of phenotypic variance cannot be addressed by focusing on changes during single episodes of selection or from one generation to the next, because they operate over much longer evolutionary timescales. For instance, diversification bet-hedging favors genotypes that produce an array of different phenotypes at some short-term cost per generation of producing suboptimal phenotypes, because this reduces the long-term risk of a catastrophic loss of genotypic fitness from failing to have any phenotypes that match the environment in any one generation (see Simons 2011; Starrfelt & Kokko 2012). However, the fitness benefits of such long-term strategies may not always be apparent when focusing on changes in phenotypic variance from one generation to the next.

Limitations

There are many reasons why we expect that the predicted changes in variance will not match the observed changes in natural populations. However, this also a feature of evolutionary predictions for changes in the mean phenotype of the population. Changes in the in the variance in the environment will also result in changes in the phenotypic variance without any selection shaping the mechanisms underpinning phenotypic variation. Genetic correlations with other traits, in a similar way as they affect the evolution of the mean phenotype, they are expected to alter the evolutionary changes in trait variance. However, it is important to highlight that the mismatches between the predicted changes in the different components of phenotipyc variation and the change we may observed in natural population will provide useful insights in the biological processes shaping phenotypic distributions in natural populations. Furthermore, as we shown in the simulations it is possible to detect a signal of selection expected to change phenotypic variance across generations, even if the realized changes do not align with the predictions.

Conclusion

Formulating a null hypothesis for the expected levels of genetic or environmental variance in behavioral phenotypes within a population is often not possible. Therefore, exploring the selective pressures expected to produce adaptive short-term evolutionary changes in behavioral variation, both among and within individuals, is key to understanding the adaptive nature of consistent individual differences in behavior. We hope that the ideas outlined in this paper encourage behavioral ecologists to estimate the various selection gradients and the variance components necessary to quantify the expected evolutionary change in behavioral variation within populations. This will further our overall understanding of the ecological contexts expected to increase or decrease the different components of phenotypic variation in labile traits.

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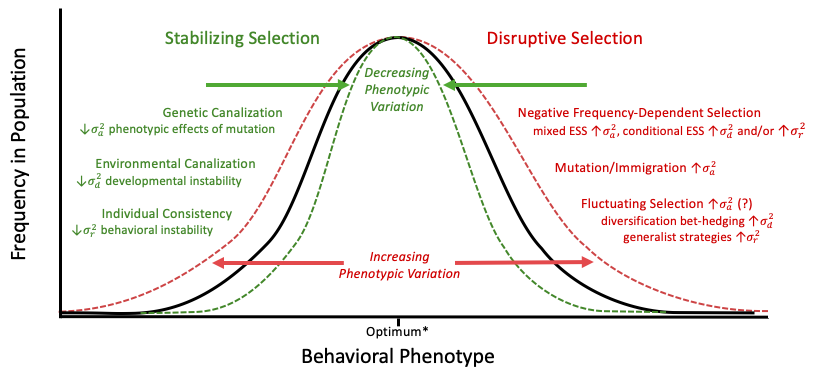
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**Figure 1**. Hypothetical representation of a distribution in behavioral phenotypes expected from the infinitesimal model (black bold line), listing various adaptive and non-adaptive effects increasing phenotypic variation (in red) versus effects decreasing phenotypic variation (in green), as a result of changes in additive genetic (), permanent environmental () and/or within-individual () variance – see text for further explanation.



We do, however, have to recognize that the variances are unlikely to be normally (Gaussian) distributed, that there are inevitable problems of scale when considering the correlation or covariance of rait mean and variance and that natural selection acts

on the individual phenotype, not on the ‘ variation trait’.