The biology hidden inside residual within-individual phenotypic variation

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ABSTRACT

Phenotypes vary hierarchically among taxa and populations, among genotypes within populations, among individuals within genotypes, and also within individuals for repeatedly expressed labile phenotypic traits. This hierarchy produces some fundamental challenges to clearly defining biological phenomena and constructing a consistent explanatory framework. We

use a heuristic statistical model to explore two consequences of this hierarchy. First, although the variation existing among individuals within populations has long been of interest to evolutionary biologists, within-individual variation has been much less emphasized. Within-individual variance occurs when labile phenotypes (behaviour, physiology, and sometimes morphology) exhibit phenotypic plasticity or deviate from a norm-of-reaction within the same individual. A statistical partitioning of phenotypic variance leads us to explore an array of ideas about residual within-individual variation. We use this approach to draw attention to additional processes that may influence within-individual phenotypic variance, including interactions among environmental factors, ecological effects on fitness consequences of plasticity, and various types of adaptive variance. Second, our framework for investigating "variance of variance" reveals that interactions between levels of the hierarchy form the preconditions for the evolution of all types of plasticity, and we extend this idea to the residual level within individuals, where both adaptive plasticity in residuals and canalization-like processes (stability) can evolve. With the statistical tools now available to examine heterogeneous residual variance, an array of novel questions linking phenotype to environment can be usefully addressed.

Key words: plasticity, canalization, variance sensitivity, gene–environment interaction, phenotypic stability, bet-hedging, reaction norm.

CONTENTS

I. INTRODUCTION

Phenotypic variance shows a distinctly hierarchical pattern, with variance existing among taxa, among populations within species, and among individuals within populations (Fig. 1). Among-species and among-individual phenotypic variation have been a central focus of evolutionary thinking since Darwin and Wallace connected the two through the process of natural selection. Many traits, such as behaviour, physiology, and some morphological characteristics, are

expressed at different instances multiple times within the lifetime of an individual (Fig. 1). Such traits also exhibit within-individual variation. Increasingly, within-individual variation is being integrated into evolutionary theory (e.g. Nussey, Wilson & Brommer, 2007; Dingemanse & Dochtermann, 2013), but major gaps exist in our knowledge of processes affecting this level in the hierarchy of variance. This is surprising given that genetic differences in within-individual phenotypic variance are necessary for the evolution of any mechanism for an individual to respond flexibly to the environment. Such mechanisms range from gene regulation within individual cells to whole nervous systems. Conversely, organisms are also under selection to maintain phenotypic integrity and reduce within-individual variance across environmental conditions that may fluctuate within the lifetime of the individual (e.g. Cannon, 1929). These fundamental attributes of organisms that control phenotypic expression arise out of patterns of within-individual variation.

(1) A statistical framework

A hierarchical structure to phenotypic variance, as shown schematically in Fig. 1, is well suited to descriptions using statistical models. We pursue this idea with four general messages in mind. First, a complete description of the hierarchy will aid biological understanding of phenotypic variance. Second, hierarchical descriptions of phenotypic variance highlight the fact that patterns at one level in the hierarchy are often non-independent from processes acting at other levels. Natural selection leading to evolution is the clearest example of this; variance among individuals is necessary for selection, and this within-population process leads to variance among units at higher levels (e.g. populations, species). A complete partitioning of variance at levels within the individual may reveal other potential examples of cross-level effects. Third,

another improvement to understanding arises because being explicit about hierarchical variance and the patterns produced raises challenges for current definitions of a variety of phenomena, including plasticity, developmental stability and canalization. While we do not focus on those issues directly herein, we will point out a few of the important implications that some variance terms have for these concepts. Finally, and perhaps most importantly, fully partitioning variance reveals patterns that demand explanation, and this can lead to new hypotheses about biological processes. Our review begins to identify some potential patterns and some of the intriguing hypotheses that may explain them.

Because within-individual phenotypic change constitutes a major subtype of phenotypic plasticity, much is known about particular aspects of the biology of within-individual variance. We suggest, however, that there is an additional level to phenotypic variance that exists inside within-individual variance. This is residual within-individual variance, or unexplained within-individual variance (see Glossary in Table 1). This variance is not well integrated into evolutionary theory, leading to recent calls for more attention to be paid to this variance component (e.g. Cleasby & Nakagawa, 2011; Stamps, Briffa & Biro, 2012; Nicolaus *et al*., 2013). Thus, besides the general goals outlined above, herein we specifically explore three ideas related to residual within-individual variance: (1) patterns of apparently unexplained within-individual variance can provide clues to the existence of several important but possibly hidden biological processes; (2) this component of variance may itself evolve from several interesting types of adaptive processes; and (3) because within-individual variance is a distinct level in the hierarchical structuring of phenotypic variance, interactions with other levels are likely integral to many biological processes linking phenotype to environment. We review what is known about

the processes affecting within-individual variance and draw connections between previously poorly linked ideas.

A full model of the hierarchy shown in Fig. 1 would be cumbersome, so here we focus first on the among- and within-individual levels within a single population of the same species. We begin with a statistical description of an observed phenotype. One common approach is to partition sources of variation using the quantitative genetics equations where variance in phenotype (V_P) is parceled into variance due to genetics (V_G) *versus* environment and error (V_E). Many patterns of phenotypic variation have been explored using versions of this equation (e.g. Lynch & Walsh, 1998; Moore, Brodie & Wolf, 1997; Tonsor, Elnaccash & Scheiner, 2013). Here, we use the related 'phenotypic equation' (Nussey *et al*., 2007; Dingemanse *et al*., 2010) that describes the component parts of each observation of the phenotype, Y. We consider observations taken from a population across a sample that includes replication within each individual, assuming that Y is a continuous character measured for instance i of individual j:

$$Y_{ij} = (\beta_0 + ind_{0j}) + (\beta_1 + ind_{1j})E_{ij} + e_{0ij} \quad (1)$$

where β_0 is the population-mean phenotype [at the position where the value of the mean-centred environmental gradient (E_{ij}) equals zero; *cf*. Dingemanse & Dochtermann, 2013]; ind_{0j} represents the deviation from that mean for the j^{th} individual, β_1 the population-mean slope with respect to E_{ij}, ind_{1j} the deviation in slope of the j^{th} individual from the population-mean slope, and e_{0ij} the residual deviation of the i^{th} instance from individual j's estimated reaction norm. The term e_{0ij} represents the focus of this paper: unexplained deviations in phenotype within individuals. We thus explicitly distinguish between among-individual variation (the differences in average value

between individuals), within-individual variation (differences between observations of the same individual) and residual variation, which herein we will refer to explicitly as unexplained within-individual variation (see also Table 1).

Equation (1) describes a linear mixed-effect model (or "random regression"). This equation is commonly used to investigate phenotypic plasticity (Nussey *et al*., 2007), defined as the effect of an environmental factor on the phenotype with β_0 and β_1 describing the intercept and slope of the population mean norm-of-reaction (*sensu* Woltereck, 1909). The concept of plasticity cuts across two levels of phenotypic variance: (1) within-genotype among-individual variance, which we will call 'developmental plasticity' because this variance is caused by environmental effects during development (Table 1); and (2) within-individual variance. In equation (1), β_1 refers to population average within-individual plasticity (see also the glossary in Table 1 for synonyms). The term ind_{0j} is determined by including 'random intercepts' for individual identity into the model, and variation among individuals in intercepts (V_{ind0j}) is consequently estimated. This variance component may reflect either genetic variance or environmental factors that have carry-over effects from one instance of expression to another (e.g. developmental plasticity: Lynch & Walsh, 1998; Wilson *et al*., 2008; Dingemanse & Wolf, 2013; Snell-Rood, 2013). The other individual term, ind_{1j}, is similarly determined by including a random effect (on the slope) arising from an interaction between individual and the environmental variable (E_{ij}). This is individual plasticity and the associated estimate of variance among slopes (V_{ind1j}) captures differences between individuals in how they change their phenotype in response to changes in the environment they experience. Individual plasticity could also have genetic variance (e.g. $V_{G\times E}$) or also arise from carry-over effects of other environmental factors (e.g. V_{PExE} or V_{GxPExE}, where PE indicates permanent environmental

effects; Schaeffer, 2004; Nussey *et al*., 2007; Dingemanse *et al.*, 2010). Genetic variance in patterns of within-individual variance is an important element of hypotheses about the evolution of within-individual variance.

We focus here on the residual deviation (e_{0ij}) in equation (1): the deviation of observation *i* from individual *j*'s reaction norm. Residual variance (variance in e_{0ij}, or V_{e_0}) is thus the amount of within-individual variance not explained by other terms in the model. Residual variance is important statistically because it forms the basis for testing whether sufficient evidence exists to reject a statistical null hypothesis about included terms (Cleasby & Nakagawa, 2011). Most statistical tests assume that residual variance is distributed normally and uniformly (i.e. residual variance should not differ between individuals or along the environmental gradient). However, because residual variance is never actually random and contains overlooked biology, this assumption of homogeneity may often be false (Dutilleul & Potvin, 1995; Cleasby & Nakagawa, 2011) and this can have some important effects (Nicolaus *et al*., 2013).

We argue that residual variance is of interest well beyond the question of whether the appropriate statistical model was used to test hypotheses about other terms in the model. Residual within-individual variation often amounts to the largest component of variation for many labile traits, sometimes as much as 60% (e.g. Bell, Hankison & Laskowski, 2009; Westneat *et al.*, 2011; Tonsor, Elnaccash & Scheiner, 2013). An appreciation for the processes that cause residual within-individual phenotypic variance, and particularly heterogeneity in residuals, will generate empirical advances and stimulate new conceptual or theoretical insights. Indeed, because differences between individuals in sources of within-individual variance are required for the evolution of mechanisms for both phenotypic stability and flexibility, most biological phenomena are linked in some way to heterogeneous residual within-individual

variances. We explore the causes of this type of variance in more detail, and review the ideas on processes acting at this level of phenotypic variance and the empirical work focused on those ideas.

II. EXPLANATIONS FOR HETEROGENEITY IN RESIDUAL WITHIN-INDIVIDUAL VARIANCE

Consider some hypothetical data collected from a single individual across an environmental gradient (Fig. 2). A simplification of equation (1) yields:

$$Y_{i1} = \beta_{01} + \beta_{11}E_{i1} + e_{0i1} \quad (2)$$

where β_{01} is the focal individual's mean (i.e. $\beta_0 + ind_{01}$ from equation 1), β_{11} is its slope with respect to E_{i1} (i.e., $\beta_1 + ind_{11}$ from equation 1), and e_{0i1} represents the deviation of the i^{th} value from the reaction norm of individual j=1. It is clear from Fig. 2 that e_{0i1} is not homogenous, because the values tend to deviate to a greater extent from the individual's reaction norm (i.e. the fitted line) at higher values of E_{i1}. There are a number of possible explanations for such heterogeneous residuals. In order to be complete, we first consider non-biological explanations, but we will focus on interesting but relatively unexplored biological explanations for heterogeneous residual within-individual variance.

(1) Sampling or measurement error and the influence of bias

Sampling and measurement error are inevitable consequences of empirical data collection. We make two brief points about this source of residual variance. First, not all residual variance is due to sampling or measurement error, and it is these other sources that we explore in more detail below. Second, sampling and measurement error may not be homogenous. Measurement error

can depend on the magnitude of the measured variable or on the conditions under which it is measured (Viswanathan, 2005). Consider, for example, measures of parental care in which the load of food brought by a parent on each visit to the nest is measured. When nestlings are small, load sizes are small, and measurement error is typically less than the mean load size. When nestlings are older, load sizes can be 4–5 times the size seen at the earlier age, and the measurement error is often several times greater in magnitude than that at the earlier age. Such examples of differences in measurement error across an environmental gradient often have a biological explanation and should be accounted for.

(2) The inaccurate or incomplete model hypothesis

A second common interpretation of residuals in a statistical model is that they include the effects of variables the investigator has not included in the model. This will likely be the case in both field and laboratory studies (see Dingemanse & Dochtermann 2014; Niemelä & Dingemanse 2014), and if the research is testing specific, hypothesized influences then lumping everything else into the residual variance is sufficient to proceed. However, an alternative goal may be to seek new explanations, in which case attending to the residual variance may be valuable. Heterogeneity in residual within-individual variance may provide hints concerning the existence of various additional and potentially interesting biological processes that might be affecting phenotypic expression. We discuss here a number of key candidates.

(*a*) *Non-linear reaction norms*

Within-individual heterogeneous residuals might occur when reaction norm slopes are modelled as linear but individuals vary in the extent of non-linearity (Fig. 3A). Non-linear reaction norms may exist when there are thresholds for shifting between one phenotype and another (e.g.

Moczek *et al*., 2002), such as in Atlantic salmon (*Salmo salar*) that exhibit genetic variation for maturation thresholds affecting alternative reproductive tactics (Piche, Hutchings & Blanchard, 2008). Most studies of thresholds have investigated non-labile traits. Thresholds in labile traits also exist and show individual variation. For example, humans differ in the threshold at which a skin irritant elicits a behavioural response (Smith *et al*., 2004) and the threshold time to process a perceptual task (e.g. Brock, Xu & Brooks, 2011).

Non-linear but continuous (e.g. parabolic) reaction norms may also be common. For example, Brommer, Rattiste & Wilson (2010) found that annual reproductive success in a long-lived gull increased and then declined with age. Provisioning behaviour of parent birds also exhibits non-linearity with respect to offspring age (Westneat *et al.*, 2011). However, both of these examples illustrate the shape of population mean reaction norms; little is known about individual variation in non-linear reaction norms, especially parabolic ones, or the underlying mechanisms that produce them. Individual variation in non-linearity may thus be of considerable biological interest.

(*b*) *Slope–intercept covariance*

Heterogeneous residual within-individual variance may also arise if there is covariance between individual intercepts (ind_{0j}) and slopes (ind_{1j}), and the slope and covariance terms are not included in the phenotypic equation (Fig. 3B) — a frequent practice when a common reaction norm is assumed for all individuals. Only a few studies have documented covariances between intercepts and slopes (Mathot *et al.*, 2012), and in no case is the cause fully understood. Kontiainen *et al.* (2009) found that nest defence intensity of Ural owls (*Strix uralensis*) varied among individuals and yet was plastic with respect to the abundance of voles. Individual aggressiveness varied in how responsive it was to vole abundance, and more aggressive

individuals were more plastic (positive covariance between intercept and slope). Slope–intercept covariance of this sort may reflect important biological processes. Mathot *et al.* (2012) suggest that such relationships may arise due to specific adaptions to environmental uncertainty, which cause the magnitude of the intercept (e.g. in sampling effort or fat stores) strategically to predetermine any associated responsiveness in adaptive plasticity to environmental change. Alternatively, such covariances may arise from other types of constraints. For example, the aggressiveness of Ural owls may be state dependent (Kontiainen *et al*., 2009), and state may change with vole abundance, possibly non-linearly. Parent house sparrows (*Passer domesticus*) cannot feed very young nestlings at a high rate perhaps because of nestling digestive constraints, hence either differences in peak provisioning rates (variation in intercept) or in the ability to assess changing offspring need (variation in slope) could drive a positive covariance between them (Westneat *et al*., 2011). Thus positive (or negative) covariance between intercept and slopes, which can be buried in the residual variance, could potentially be driven by some interesting, yet relatively unknown, biology (e.g. Dingemanse *et al.* 2012).

(*c*) *Multidimensional reaction norms*

Interactions among environmental factors affecting plastic phenotypes can also create heterogeneous residuals if not included in the phenotypic equation. Organisms live in environments that vary in many ways, and phenotypes could be a function of more than one environmental factor simultaneously. For example, herbivory and competition for light influence growth and changes in defensive compounds in plants (e.g. *Arabidopsis thaliana*; Cipollini, 2004) and temperature interacts with food type to influence growth rate in larval insects (e.g. Kingsolver *et al.*, 2006; Stillwell *et al*., 2007). These examples involve effects causing between-individual differences *via* developmental plasticity, but multiple environmental factors can

obviously also affect within-individual variance in phenotype, and hence the residual variance in equation (1). For example, in house sparrows, breeding attempt order and date in the season interact to affect clutch size (Westneat, Stewart & Hatch, 2009), and nestling age and brood size interact to affect parental feeding rate (Westneat *et al*., 2011).

We label reaction norms that occur in response to more than one environmental factor "multidimensional" norms of reaction (Westneat *et al*., 2009). Multidimensionality can produce heterogeneous residual within-individual variance in two ways. First, sensitivity of the phenotype to additive effects of two or more environmental variables can produce this type of heterogeneity if individuals experience only subsets of both environments. All individuals in a population might share the same reaction norm that is responsive additively to two environmental factors (E_1 and E_2). If E_1 is more variable at some values of E_2, such as if territories with good food supplies also had more stable temperatures, then individuals on good territories might be less variable than those on poor territories (e.g. Charmantier & Garant, 2008). While some of these effects could be fixed by better sampling by the researcher, the case of territory effects illustrates the more interesting possibility that expression of one phenotype could alter the environments experienced (a phenotype–environment correlation) and thereby affect expression of another phenotype; in this way multidimensionality combined with a phenotype–environment correlation may be the underlying cause of heterogeneous residuals.

Second, the phenotype may be sensitive to a non-additive (i.e. interactive) effect of two or more environmental factors. This interaction can create unequal variances across one of the environmental gradients (e.g. Fig. 4). For datasets of repeatedly expressed traits, multidimensionality can easily be incorporated in the phenotypic equation by constructing

models with more than one environmental gradient (e.g. E_1 and E_2 instead of just E) plus their interactions (e.g. parental provisioning rates: Westneat *et al*., 2011).

Non-additive effects have implications for understanding both the ecology of plasticity and the organismal mechanisms producing it. An interaction effect may arise because of some constraint to a process involved in the trait of interest. For example, an influence of host plant diet on the thermal reaction norm of insects may arise in part because phenolic compounds present in some diets are harder to process at cooler temperatures (e.g. Diamond & Kingsolver, 2012). Alternatively, interaction terms may arise because environmental variables affect fitness trade-offs in ways that produce multiple fitness peaks. For example, within-individual variance in clutch size in sparrows is influenced by an interaction between date in the season and nesting attempt order (Westneat *et al*., 2009). This appears consistent with life-history theory that incorporates a seasonal decline in offspring quality (Rowe, Ludwig & Schluter, 1994). In this model, multiple breeding episodes create separate adaptive ridges with respect to date for each nesting attempt, producing multidimensional reaction norms affected by interactions between date and nesting attempt order. Such circumstances could select for the integration of multiple environmental cues.

Multidimensionality in reaction norms affects interpretations about tests of theory. For example, evolutionary theory on pleiotropic effects leading to senescence suggests that genetic variation in fitness should increase at older ages. Brommer *et al*. (2010) analysed declines with age in reproductive performance (annual fitness) in common gulls (*Larus canus*), showing among-individual variance, but little additive genetic variance, in slope with respect to age. Yet, residual variance increased with age. This heterogeneity in residuals suggests the possibility of multidimensionality — that is, as individuals age they may be increasingly susceptible to the

294 impact of other environmental factors, possibly in non-additive ways. Multidimensionality would
295 explain the change in residual variance with age, and the expected impact of pleiotropy on
296 genetic variance in fitness might be resurrected if there was genetic variation in the interaction
297 between age and environment on performance. Evidence supports the idea that genetic variance
298 for such interactions exists (e.g. Kingsolver *et al*., 2006; Stillwell *et al*., 2007), although we
299 know of no studies demonstrating such effects on within-individual variation. In general, the
300 biology of multidimensional reaction norms is likely to be quite important both for evolutionary
301 hypotheses regarding plasticity and for understanding underlying mechanisms of phenotypic
302 development, with non-additivity raising challenging questions about the mechanisms by which
303 variance in environment produces phenotypic variance.

304 **(3) Organismal error**

305 Many reaction norms arise from some mechanism of assessing an environmental factor (called
306 "active plasticity"; Scheiner, 2006). Errors in assessment (e.g. Reeve, 1989; Wiley, 1994;
307 Sherman, Reeve & Pfennig, 1997; DeWitt, Sih & Wilson, 1998; Auld, Agrawal & Relyea, 2010)
308 can produce phenotypes that deviate from the correct one. In other words, the deviations from
309 the line in Fig. 2 occur horizontally, and they arise from the organism misidentifying the cue to
310 the environment on the *x*-axis and producing a phenotype that would be better suited to a
311 different environment. Such errors in plasticity occur at both the among-individual and within-
312 individual levels; we refer to this phenomenon as "organismal error" (see glossary in Table 1 for
313 synonyms) simply to separate these errors from researcher measurement error. These types of
314 error contribute to limited plasticity (Moran, 1992; Getty, 1996; DeWitt *et al*., 1998; Auld *et al*.,
315 2010), and occur whenever assessment mechanisms (broadly defined) are involved in phenotype

316 production. For example, misidentification of self as non-self by the immune system leads to
317 inappropriate activation of the immune system producing auto-immune disorders (Golub &
318 Green, 1991). Errors in growth processes during development may underlie fluctuations in the
319 symmetry of paired attributes (e.g. Van Valen 1962; Hansen, Carter & Pélarbon, 2006). Finally,
320 inappropriate behaviour may arise because of inadequacies of assessment at the sensory level
321 (e.g. Wollerman & Wiley, 2002) or the ways in which information is integrated as is exhibited
322 by increased error when attention is divided (e.g. Dukas, 1998).

323 Organismal errors could be heterogeneous for several reasons. First, organismal error is
324 likely proportional to the cue's scale (known as Weber's Law; Ross & Murray, 1996). This
325 effect is known in all sensory modalities and impacts many types of cues, including assessment
326 of time (e.g. Gibbon, 1977). Heterogeneity might also arise because multidimensionality leads to
327 problems with integration. For example, the inappropriate activation (response to internal
328 environment) of the immune system that leads to autoimmune disease can be exacerbated by
329 exposure to some bacterial pathogens, a second environment due to an external invader (Playfair,
330 1995). Phenotypic imprecision due to error may itself be influenced by developmental processes.
331 Deviations from a target phenotype can be compensated for in some cases (e.g. Kellner &
332 Alford, 2003), and therefore the magnitude of such noise may vary through ontogeny. Finally,
333 heterogeneous phenotypic expression could also reflect heterogeneous selection if either the
334 fitness consequences of the inaccurate phenotype or the costs to improving precision differ along
335 the range of the environment gradient. For example, in birds, brood parasites produce
336 circumstances in which errors in egg recognition by hosts reduce host fitness. As the rate of
337 parasitism increases, the costs of acceptance increase, and indeed, acceptance rates decline with
338 increases in parasitism (e.g. Lindholm & Thomas, 2000; Stokke *et al*., 2008). Preventing

parasitism appears costly because acceptance increases when parasitism levels decline (Brooke, Davies & Noble, 1998). Acceptance errors may be influenced by learning (e.g. Rothstein, 1978), and so they can exhibit within-individual plasticity (Lotem, Nakamura & Zahavi, 1995), but it is not known if variance in either the costs of accepting a parasite egg or the cost of discriminating among eggs influences acceptance errors within an individual.

(4) **Random residual within-individual variance**

A final possibility is that phenotypes vary due to truly random processes. We describe two major ways this could occur, which differ in the mechanism that connects the environment with the phenotypic effect.

(*a*) *Passive plasticity*

The phenotype could exhibit "passive" plasticity (Scheiner, 2006) in which purely physical processes create phenotypic variation. Fluctuations in body temperature in ectotherms due to the physics of heat transfer and changes in ambient air temperature could be seen as one example of passive plasticity. More convincingly, foraging success, measured as the time to find the next food item, might exhibit passive plasticity as a result of changes in the density or distribution of prey items. Thus food intake rate will have a component of variation that arises from the physical constraint that food cannot be ingested before it is found, and the time taken to find the next prey item will show some unpredictable variance because the location of a particular prey item is usually not known by the forager when they start foraging. In both cases, some portion of the phenotypic variance arises due to passive plasticity and the environmental factor causing this may be unpredictable. The phenotype thus contains some stochastic variation that, if not otherwise accounted for, would be present in the residual phenotypic variance. Because the

environmental factor causing passive plasticity may be associated with other factors, then, as may occur with multidimensional reaction norms, this could create heterogeneity of residual variance (e.g. Stearns & Kawecki, 1994).

Residual variance caused by unpredictable passive plasticity could have fitness consequences creating stabilizing selection on reaction norms. This could produce a process akin to canalization, occurring at the within-individual level, whereby the phenotype is stabilized around the optimal reaction norm (Stearns & Kawecki, 1994). However, there are also circumstances when increased residual variance may be favoured. Variance-prone foraging is one example (Stephens, 1981). Encounters with prey may be unpredictable, but if the variance in encounter times can be assessed by foragers, then individuals can make decisions to experience either more or less unpredictable passive plasticity in the instantaneous food-capture rate (Shafir, 2000). Certain state variables, such as energy reserves, are predicted to create selection favouring either variance-averse or variance-prone behaviour (Caraco, Martindale & Whittam, 1980; Stephens, 1981). Studies of this idea in captivity have produced some equivocal results that may be resolved by accounting for the scale of environmental variance (reviewed in Shafir, 2000), but there remains a lack of studies conducted in the wild that properly test for such effects of ecologically relevant state variables (but see Ratikainen, Wright & Kazem, 2010).

(*b*) *Adaptive residual within-individual variation*

Adaptive residual phenotypic variation could be induced by mechanisms incorporating stochasticity into phenotype expression. When this occurs at the level of among-individual but within-genotype variation, it can be a mechanism for adaptive phenotypic polymorphisms (phenotype switching, e.g. Kussel & Leibler, 2005), polyphenisms (Mayr, 1963; Van Dooren, 2001) and diversification bet-hedging (Gillespie, 1973; Frank & Slatkin, 1990; Simons, 2011).

Residual within-individual variation in a wide variety of traits could be adaptive (Table 2). Some phenotypes emerge from processes that initially generate (possibly random) variation and then involve mechanisms that reduce this variation within individuals (Frank, 1997). In the vertebrate immune system, clonal selection within a highly diverse population of B-cells is a central process for adaptive immunity (Golub & Green, 1991). Similar processes may occur at the cellular level for epidermal or neural tissues (e.g. Changeux & Danchin, 1976; Kagan, Novoplansky & Sachs, 1992). Likewise, some learning processes such as trial-and-error learning may involve generating variation followed by a mechanism of sorting among options within the individual (Frank, 1997). For example, in jumping spiders, individuals produce a large array of signals oriented toward potential prey (other spiders); appropriate feedback from the prey then leads to repetition of the effective signal (Jackson & Wilcox, 1993). Such mechanisms would produce heterogeneous residuals in phenotypic variation across either time or specific environmental gradients, and could evolve through differential selection among genotypes producing different patterns of within-individual variation.

Reduced residual within-individual variance may be adaptive. Theory suggests that the presence of conspecific observers during contests can favour predictable levels of aggression (e.g. Johnstone, 2001; Nesse, 2001), which would reduce within-individual variance in aggression. In complex social groups, particular social niches may exist, and individuals taking on those roles may behave less variably (e.g. Bergmüller & Taborsky, 2010). Similarly, Schuett, Tregenza & Dall (2010) hypothesize that sexual selection on male behaviour might produce sexual dimorphism in within-individual variance in behaviour.

Alternatively, unpredictability *per se* may be favoured. Being unpredictable could in some conditions lead to higher rates of winning in contests (e.g. Whiten & Byrne, 1997).

Variable display intensity by individual combatants is favoured in war-of-attrition contests (Maynard Smith, 1974). Similarly, variable waiting times for both predator and prey, possibly at both the among- and within-individual levels, are favoured when the prey is in refuge and the predator waits for them to emerge (Hugie, 2003). Briffa (2013), for example, found that residual variation in a startle response, after controlling for individual identity and mean plasticity, increased in the presence of additional cues to a predator. This type of unpredictable behaviour might also increase as individuals become familiar with individual predators (Stamps *et al.*, 2012).

An intriguing example of potentially adaptive stochastic variance may occur at the sub-cellular level. There is growing evidence that a number of molecular events, including gene regulation, are subject to stochastic variation from cell to cell within the individual (e.g. Eldar & Elowitz, 2010). This may arise because of relatively small copy numbers of some key molecules (e.g. DNA, some large regulatory proteins) within the cell that produce differences in rates of chemical contacts from cell to cell. Some of this variation might be considered passive plasticity, but in some cases it may be adaptive. For example, in yeast a suite of genes is regulated by calcium, and the main transcription factor Crz1 exhibits apparently stochastic bursts of up-regulation. Cai, Dalal & Elowitz (2008) show that stochastic bursting, which varies in response to calcium, produces more uniform co-regulation across an array of downstream genes because of proportional control — that is, the proportion of time Crz1 is bursting produces stronger correlations between downstream products than would more modulated amplitudes or durations of individual bursts. Hence within-individual residual variation in Crz1 activation oddly results in more coordinated control of other genes than would less stochastic Crz1 regulation. Stochastic bursts of up-regulation may also be the underlying molecular explanation for the generation of

variable populations of cells involved in internal selection during development (Losick & Desplan, 2008).

In summary, adaptive residual within-individual phenotypic variation may exist across several levels in organismal organization, from sub-cellular to organismal. We have described some hypotheses that might explain such variation, but the array of studies that have directly focused on these is remarkably small.

III. INTERACTIONS ACROSS HIERARCHICAL LEVELS: GENOTYPIC AND INDIVIDUAL DIFFERENCES IN RESIDUAL WITHIN-INDIVIDUAL VARIATION

We have emphasized that residual within-individual variance occurs in the context of a hierarchy of variances, ranging from the within-individual level on up to higher levels of taxonomic organization (Fig. 1). Thus, instances of phenotypic expression are nested within individuals, individuals within genotypes, genotypes within populations, populations within species, and so forth. A fascinating feature of this structure is that interactions between levels occur, and they have far-reaching consequences.

One well-known example involves the genotype by environment interactions (G×E) depicted in quantitative genetics theory concerning the evolution of phenotypic plasticity (Via & Lande, 1985; Gomulkiewicz & Kirkpatrick 1992). This is typically viewed as an interaction between the among-genotype-within-a-population level (multiple genotypes exist within a population) and the among-individuals-within-genotype level (multiple environments can be experienced by different individuals with the same genotype, producing "permanent environment", PE, variation). But, since individuals can experience different environments in their lifetimes, G×E also captures an interaction between the among-genotype level and the

452 within-individual level, leading some explicitly to distinguish between these two types of gene
453 by environment interaction (G×PE *versus* G×E; Nussey *et al*., 2007; Dingemanse *et al*., 2010). A
454 potentially interesting possibility is multidimensionality across levels, with an interaction
455 between an environmental effect at the within-genotype level (E_{1j}) and another factor at the
456 within-individual level (E_{2ij}) (e.g. Weinig & Delph, 2001). A suitable modification of equation
457 (1) to include the genotypic level would account for developmental plasticity in behavioural
458 flexibility (Piersma & Drent, 2003; Stamps & Groothuis, 2010; Dingemanse *et al*., 2010), and
459 this can be extended to include effects of environmental variables that interact but do so across
460 different timescales. For example, in birds, variation in maternal androgens present in the yolk of
461 eggs may affect the mean level of aggressive behaviour by these individuals as adults (Gil, 2008;
462 Müller *et al*., 2012). Levels of aggression in any particular interaction are also influenced by the
463 value of a food resource (e.g. Chancellor & Isbell, 2008). An interesting but untested possibility
464 is that yolk androgens influence the way in which food value influences aggression (i.e. the slope
465 of a reversibly plastic response), producing a between-individual by within-individual
466 multidimensional reaction norm (PE×E), with the possibility of there being genetic variance for
467 this (e.g. G×PE×E). We note that the distinction between environments that have developmental
468 and those that have only activational effects is often more subtle than typically portrayed; some
469 activational environmental effects (cue to a predator) can also have carryover effects through
470 processes such as learning (Dingemanse & Wolf, 2013), potentially producing complexities not
471 captured by current variance equations.

472 The hierarchical phenotypic variance structure may produce interactions or covariances
473 between elements of residual within-individual variance and the among-individual or the among-

genotype levels. To illustrate, we take the phenotype equation (1) and expand the residual within-individual deviations (e_{0ij}) into its own equation:

$$\sigma_{eij} = (\beta_{\sigma 0} + ind_{\sigma 0j}) + (\beta_{\sigma 1} + ind_{\sigma 1j})E_{ij} \quad (3)$$

where σ_{eij} describes the residual variance (Ve_{0i}) as having a population mean variance ($\beta_{\sigma 0}$), an individual-specific deviation in variance from the mean ($ind_{\sigma 0j}$), and an effect of both population and individual effects of environment on the variance ($\beta_{\sigma 1} + ind_{\sigma 1j}$). These latter terms capture the heterogeneous nature of residual variance due to, in many cases, factors that influence the phenotypic sensitivity to environmental factors.

This double equation, with equation (1) describing effects on means and the simultaneous equation (3) capturing patterns in variances, has several important consequences. One is that there may be interactions between elements of the residual variance (equation 3) and terms present in the mean portion (equation 1). Equation (3) already includes one such interaction — residual within-individual variance could vary among individuals. Equation (3) could be expanded to include between-genotype, between-population, and between-species differences in residual within-individual variation. Such effects would make the residual variance in a particular trait behave as if it is a trait itself (Biro & Adriaenssens, 2013).

A second implication of the double equation is that there are new potential covariances between terms within and between the two linked equations that are, as we detail below, of biological interest. Some of these are evident in Fig. 5; we describe two in more detail here.

Cov (ind_{0j}, $ind_{\sigma 0j}$): the magnitude of an individual's reaction norm intercept could covary with the magnitude of an individual's residual variance. Either positive or negative covariances are possible; Fig. 5 depicts a negative covariance. This covariance seems likely to have a biological basis since the magnitude of a phenotype and tight control over its variance in expression may be

linked. For example, aggressive individuals might exhibit less residual variance because they may be less sensitive to extraneous stimuli (e.g. Natarajan *et al*., 2009). In general terms, processes involved in changing residual variance (e.g. canalization or behavioural stability) may be integrated with processes producing mean phenotypes. A review of genetic variation in environmental variance reports a handful of studies that have measured a genetic correlation between mean phenotype and variance in phenotype, the majority of which are negative (Hill & Mulder, 2010).

Cov (ind_{1j}, $ind_{\sigma 0j}$): the magnitude of an individual's reaction norm slope covaries with its within-individual residual variance; also shown as negative in Fig. 5. Several potential examples of this covariance exist; a positive covariance could perhaps be due to increases in plasticity making the phenotype more sensitive to organismal error or the impact of other environmental factors. This covariance is similar to one suggested for a relationship between developmental plasticity and developmental instability (e.g. Hansen *et al*., 2006; Tonsor *et al.*, 2013), which is a covariance between a genotype's intercept and within-genotype among-individual deviations from the genotype's reaction norm. Alternatively, an individual with a strong reaction to a particular environmental gradient might be less sensitive to stochastic influences of other cues (e.g. attentional focus; Dukas, 1998).

Other covariances with elements of stochastic residual within-individual variance are possible, especially if other hierarchical levels of phenotypic variance are included. We also expect interactions with other levels. For example, if residual within-individual variation itself is to evolve, as we suspect it might, then there must be genetic variation for residual deviations. Indeed, studies have uncovered evidence of genetic variation for environmental variance (e.g. Hill & Mulder, 2010). Often these have lumped together many of the processes acting within the

individual (plasticity, all of the sources of heterogeneous residual variance discussed above). It is not clear in any case that the genetic variance of any specific cause of heterogeneity in residuals has been estimated. Hill & Mulder (2010) review a variety of methodological approaches and some of the problems with each. Here, we note that an important implication of our treatment is that attending to different potential sources of unexplained residual within-individual variance and being able to assess the genetic variance in specific causes would fine-tune tests of hypotheses about the evolution of phenotypic variance.

IV. DISCUSSION

Labile phenotypes, especially behavioural and physiological characters, exhibit substantial within-individual variation. We emphasize that the presence of this variation is a large, mostly untapped, opportunity to understand better the ecology of selection and evolution. The basic logic here is powerful: if variance in phenotype within an individual has fitness consequences and differences in within-individual variance exist between genotypes, then patterns of within-individual variance can evolve. Presumably, it is exactly this process that has driven the variety of mechanisms for assessing environments and producing adaptive reversible, or irreversible, phenotypic plasticity that we now see in most organisms. Although within-individual plasticity may be one of the most widespread of biological phenomena, it has usually been studied indirectly and is not well integrated conceptually. More importantly for the purposes of this review, variation in residual within-individual variance (that not explained by active plasticity) also likely underlies how individuals maintain consistent phenotypes in the face of considerable environmental variance. Such stability is an example of within-individual canalization, and has also been understudied from the perspective of the evolution of reaction norms. Finally, we

542 expect residual within-individual residual variation to differ between individuals and genotypes,
543 and if so, heterogeneous residual within-individual variance may be as common as genetic
544 variance itself.

545 Residual within-individual variance in the phenotype is neither "noise" nor "random"
546 variance, despite the labels given it from the statistical assumptions needed for hypothesis
547 testing. It is, in fact, a rich source of clues about the biology of phenotypes. It is likely to be
548 heterogeneous for many reasons, and so it should be the explicit focus of investigation more so
549 than it currently is. Residual within-individual variance is often the largest component of
550 phenotypic variance for some phenotypes, such as behavioural traits (e.g. Bell *et al.*, 2009).
551 Clues as to its underlying biology can be gained by statistically exploring the structure of
552 residual variance, particularly for patterns of heterogeneity. To that end, several statistical
553 approaches have been developed to account appropriately for heterogeneous within-individual
554 residual variance in tests of hypotheses about other terms in a model, but they can be adapted to
555 explore patterning in residual variance directly (e.g. Breusch & Pagan, 1979; White, 1980; Lee &
556 Nelder, 1996; Smyth & Verbyla, 1999; Cleasby & Nakagawa, 2011; Westneat, Schofield &
557 Wright, 2013). Of critical importance here is that residual within-individual variance is modelled
558 analogously to means. Because residual within-individual variance can vary simultaneously with
559 respect to several variables (including individual identity), a mixed-model structure that accounts
560 for influences on both mean effects and residual variances within a single model has the most
561 potential to uncover new patterns. Recent techniques appear to accomplish this (Lee & Nelder,
562 1996; Smyth & Verbyla, 1999; Westneat *et al.*, 2013) and can be applied to datasets containing
563 repeated measures of phenotypes within individuals.

564 Models of mixed effects for both means and variances require large datasets. For
565 example, good estimates of variance terms in the mean portion of the model need 1000 data
566 points or more (e.g. Martin *et al*., 2011; van de Pol, 2012). While applying our approach to some
567 rarely expressed traits may be a challenge, there are many morphological, physiological, and
568 behavioural traits that are expressed quite often. Consider, for example, feathers on a bird that
569 moults twice a year, leaves on a plant, eggs per spawn in a fish, or tendency to attack an
570 opponent in crickets. These traits are expressed dozens to hundreds of times in each individual,
571 and so massive datasets can be relatively easily collected. Empirical studies have detected
572 heterogeneous residuals in several different traits (reviewed by Nicolaus *et al*., 2013) and in
573 some cases from modest-sized datasets collected for other purposes (e.g. Westneat *et al*., 2013).
574 We think the phenotypic equation combined with other conceptual and empirical tools has the
575 potential to lead to a variety of novel hypotheses and experiments for many types of traits.
576 Our review emphasizes that the nature of residual within-individual variance is not
577 merely an empirical issue; several potentially important conceptual ideas have emerged from
578 considering the underlying reasons for residual phenotypic variance and the impact that such
579 variance might have on the evolutionary process. For example, our examination of residual
580 within-individual variance intersects with concepts of phenotypic plasticity, canalization, and
581 developmental stability. The specific relationships between these terms are often confusing and
582 there appears to be no general agreement on definitions (Dworkin, 2005). Some authors, for
583 example, view plasticity and canalization as opposites (e.g. Gibson & Wagner, 2000; Debat &
584 David, 2002; Nijhout & Davidowitz, 2003; Ghalambor, Angeloni & Carroll, 2010), whereas
585 others treat them as potentially independent phenomena (e.g. Stearns & Kawecki, 1994) although
586 they may be correlated (e.g. Tonsor *et al.*, 2013). Our focus on the phenotypic equation and our

587 treatment of within-individual residual variance as a component of variance within a hierarchy of
588 variances favours distinct but overlapping definitions. We do not have the space here to explore
589 all the nuances, but a brief example illustrates our point that the concepts of plasticity and
590 canalization can cut across several levels of phenotypic variance. Selection could act on a
591 particular trait to reduce environmentally induced variation in phenotype among individuals
592 within a genotype. We might call this within-genotype canalization of intercepts.
593 Simultaneously, selection might favour a more flexible within-individual phenotypic response to
594 the environment. A possible by-product of this might be higher within-individual residual
595 variance due to organismal error, meaning that at the within-individual level the organism is
596 simultaneously more plastic (steeper slope) and less canalized (higher residual variance), even
597 though the genotype is more canalized developmentally around the intercept. Improved clarity
598 about concepts and processes may be achieved by taking a more statistical approach to such
599 definitions and attending to the full hierarchical structure of variance, including residual within-
600 individual variance.

601 We also claim that residual within-individual variance deserves more attention because it
602 would bring renewed focus on the ecology of phenotypes. Molecular and quantitative genetics
603 have contributed major new insights into the genetics of phenotypes. Yet, our focus on reaction
604 norms and residual within-individual variance rests on how environments affect phenotypes.
605 Genotypes can interact with the environment at two levels — among individuals within genotype
606 and within individuals. The environment also has effects on within-individual phenotypic
607 variation in three distinct ways: (*i*) via within-individual plasticity, (*ii*) through several possible
608 impacts on within-individual residual variance, and (*iii*) due to effects on developmental
609 plasticity that change either within-individual plasticity or the nature of residual variance.

610 Finally, the environment influences the fitness consequences of phenotypic variation at each of
611 these levels. These influences of ecology have important ramifications, and while we have made
612 great strides in understanding the interface between ecology and phenotypic diversity, our
613 analysis here suggests that we could gain even more by attending to the ecology of individual
614 phenotypes in greater detail. This may be especially important in this time of rapid ecological
615 change.

616 Another emergent conclusion is that statistical models are more than a means to evaluate
617 particular biological hypotheses. As we have done here, the phenotypic equation can clearly also
618 be used to generate biological hypotheses. It is effective precisely because it is
619 phenomenological — it is a description of pattern in phenotype. Too often in biology we conflate
620 pattern and process in our terminology. Statistical descriptions allow for clearer definitions of
621 pattern, which then demand explanation. Phenotypic variation is an unusual blend of processes
622 that mimic statistical properties and those that actually incorporate variance, all combined in a
623 hierarchical structure (from individual to phylogeny) that is especially well suited for statistical
624 modelling.

625 Thus, the phenotypic equation may be viewed as a biological hypothesis in itself. It
626 models a hierarchical structure, and so thereby constitutes a hypothesis about the hierarchical
627 nature of phenotypic variance. This draws attention to each term in the equation and leads to
628 hypotheses regarding its potential biological importance. In this context, the residual term
629 becomes as important as the population mean. Moreover, we suggest that extensions of the
630 phenotypic equation can integrate patterns of phenotypic variance from within the individual up
631 to among taxa. Employing the phenotypic equation fully might catalyse a new integration of
632 micro and macro evolutionary processes, overcoming some of the problems with such

integration (e.g. Martin, Ton & Niklison, 2013). It could also provide the structure for assessing the role of ecology on multiple scales (e.g. within individuals, among individuals, among populations) simultaneously. Such considerations go beyond understanding the biology underlying residual within-individual variance, but our systematic exploration of this one element of the phenotypic equation is illustrative of the potential value of more fully integrating statistical thinking into biology (e.g. Bolker *et al*., 2009).

V. CONCLUSIONS

1. The hierarchical structure of phenotypic variance is especially amenable to hierarchical statistical models, and applying such models highlights the potential importance of within-individual residual variance. This variance term is more than "error", and could contain interesting patterns, such as heterogeneous residual variance. We review hypotheses that may explain heterogeneity in within-individual residual variance in phenotype.
2. Our review reveals many relatively poorly studied phenomena that have potential theoretical importance, including non-linear reaction norms, intercept-slope covariance, multidimensional phenotypic plasticity, various forms of passive plasticity, and several types of adaptive variance.
3. We find that the biology of within-individual residual variance cuts across multiple levels of biological organization, from gene regulation within cells, to whole organism traits such as physiology and behavior. Our investigation of heterogeneous residual variation also links concepts from multiple fields. For example, canalization in developmental biology and variance sensitivity in behavioral ecology have elements in common. Moreover, explicitly considering the causes of phenotypic variance in a hierarchical framework reveals multiple

scales at which particular processes may occur, with some seemingly opposite processes (e.g., canalization and plasticity) occurring simultaneously but at different levels in the hierarchy.

4. By embedding within-individual residual variance at its appropriate level in the hierarchy of phenotypic variance, we establish that residual variance can evolve. It is nested several levels down from genotypic variance, and so may evolve in ways that are linked to individual plasticity (within-individual level), developmental plasticity (among-individual within-genotype level), and mean phenotype (among-genotype level). Such interactions may have important implications for the ecology of selection and the process of evolution.
5. Methods are available to assess within-individual residual variance in a variety of repeatedly expressed traits and statistically explore pattern in these residuals. With these tools, new understanding of the ecology of phenotypes can be obtained.

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933 Table 1. Glossary of terms used in the text, a short definition, and related terms with the same or
934 similar meaning.

935

Term	Definition	Similar terms
Active plasticity	**Phenotypic plasticity** in which the phenotype responds to environmental cues through a biological mechanism (*sensu* Scheiner, 2006)	Adaptive plasticity
Among-individual phenotypic variance	Variance among individuals in average phenotype in a specified environment	-
Canalization	The reduction of **residual phenotypic variance** at either the within-genotype-among-individual or within-individual levels	Developmental stability; behavioural stability; individual stability (Dingemanse *et al.*, 2010; Stamps & Groothuis, 2010)
Developmental plasticity	**Phenotypic plasticity** occurring earlier in the lifetime that has long-lasting effects on the phenotype	Permanent environmental effect; irreversible plasticity (West-Eberhard, 2003)
Heterogeneous residual within-individual variance	Differences in **residual within-individual variance** across any terms in a model of phenotypic variance	Non-normal residual variance
Measurement error	Variance in phenotypic measures due to the way the trait is measured	Observer error
Multidimensional reaction norm	A function relating a phenotype to two or more environmental factors	
Organismal error	Variance in phenotype due to mismeasures of the environment by the subject	Phenotype–environment mismatching (DeWitt *et al*., 1998); developmental instability (Waddington, 1942; Markow, 1995; Tonsor *et al*., 2013); recognition error (Sherman *et al*., 1997) or imprecision (Hansen *et al*., 2006)
Passive plasticity	**Phenotypic plasticity** in which the effect of the environment can	Non-adaptive plasticity

	be explained by non-biological processes (*sensu* Scheiner, 2006)	
Phenotypic plasticity	A change in the phenotype expressed by a genotype or individual with respect to a difference in environment, either **passive** or **active plasticity**	Plasticity; flexibility
Residual within-individual variance	Amount of **within-individual variance** not explained in a specific statistical model (i.e. the average squared deviations of observations from an individual's reaction norm), averaged over a sample of individuals	Unexplained within-individual variance
Within-genotype among-individual variance	Variance in mean phenotype among individuals of a given genotype, measured in a specified environment	**Among-individual variation**
Within-individual plasticity	Variation in an individual's phenotype with respect to variation in the environment. Quantified at the individual level or averaged across individuals ("population average")	Reversible plasticity; behavioural flexibility (Piersma & Drent, 2003); activational plasticity (Snell-Rood, 2013); labile phenotype
Within-individual variance	Amount of phenotypic variance among instances of phenotypic expression of an individual. Quantified at the individual level or averaged across individuals ("population average")	Intra-individual variation (Stamps *et al*., 2012)

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937

938 Table 2. Examples of traits exhibiting patterns of residual variance that differ from that expected
939 under passive plasticity. Such deviations have been suggested to be adaptive *via* the listed
940 selective agent.

Trait exhibiting adaptive residual variance	Selective agent	Reference
Gene expression	Stochasticity leads to more efficient coregulation	Cai *et al*. (2008)
B-cells (antibody types)	Diversity followed by internal selection leads to more effective adaptive immunity	Golub & Green (1991)
Components of neural networks	Diversity followed by self-selection leads to more finely tuned neural processing	Changeux & Danchin (1976); Kagan *et al*. (1992)
Homeostatic temperature control	Multiple mechanisms across endotherms and ectotherms reduce variation leading to more effective physiological functions	
Task roles	Reduced variance leads to more effective output of social group	Bergmüller & Taborsky (2010)
Male courtship	Stereotyped and predictable courtship may be favoured through female preference	Schuett *et al*. (2010)
Prey responses	Variable and unpredictable emergence from refuge reduces predation	Hugie (2003); Briffa (2013)
Aggression levels in consecutive contests	Reduced variability increases ability to assess outcome and reduce costs to both contestants	Johnstone (2001)
Food intake rate (individual or provisioning parent)	Reduced variability beneficial to forager in high condition; increased variability beneficial to forager in poor condition	Stephens (1981); Ydenberg (1994)
Trial-and-error learning	Increased diversity of solutions, followed by self-selection, may lead to novel solutions to common problems	Frank (1997)

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943 **Fig. 1**. Schematic representation of the hierarchical organization of phenotypic variance, with
944 directional arrows indicating that replicates of the next level (e.g. populations within species,
945 individuals within genotypes) are nested within the upper level. Variance in trait expression
946 among instances (i.e., within-individual variance) is relatively poorly studied, and so we focus on
947 phenotypes that have multiple instances of expression within an individual. We explore
948 processes that produce patterns of variance among instances. We also emphasize that variation in
949 patterns of variation can occur due to the hierarchical structure. That is, patterns of variation in
950 expression among instances can vary among individuals, genotypes, populations, etc.
951
952 **Fig. 2**. Plot of phenotypic measures (Y_{i1}) taken from a single individual (j=1) across an
953 environmental gradient (E_{i1}). The mean phenotype (β_{01}) is the elevation and is appropriately
954 taken at the mean-centred environment, and the slope (β_{11}) describes the individual's plasticity,
955 with elevation and slope together producing a norm of reaction. In this case there is
956 heterogeneous residual variance, with confidence limits indicated by the dashed lines that 'fan
957 out' over the gradient.

958 **Fig. 3.** Two examples of incomplete models producing heterogeneous within-individual residual
959 variance. (A) Modelling a phenotype with a linear reaction norm (solid line) produces
960 heterogeneous residuals when the reaction norm is actually non-linear (dashed line). (B)
961 Individuals (one in red, the other in blue) vary in how they respond to changes in the
962 environmental gradient (e.g. I×E) and slope covaries with intercept. Omission of these terms
963 from the model will produce heterogeneous residual within-individual variance (if each is

964 assumed to have the average reaction norm, black dotted line). The vertical line indicates the
965 mean environment for E_1.

966 **Fig. 4**. Multidimensional reaction norm depicted in two dimensions: gradient E_{1i1} (x-axis)
967 interacts with gradient E_{2i1} (indicated by colour) to affect the phenotype of an individual. This
968 non-additive effect of two different environmental parameters creates heterogeneous residual
969 within-individual variance if it is not modelled.

970 **Fig. 5**. Graphical depiction of the extended phenotypic equation applied to hypothetical data
971 from two individuals. The solid black line represents the population-average reaction norm. The
972 two individuals deviate from the population intercept (blue = ind_{01} and red = ind_{02}) and they
973 differ in slopes (blue line, ind_{11} < red line, ind_{12}). Individual 1, with the larger intercept, also has
974 a shallower slope, hinting at a negative covariance between intercept and slope. The two
975 individuals also differ in residual variance ($ind_{\sigma 01} < ind_{\sigma 02}$), indicated by the spread of points at
976 the intercept. Finally, the residual variance changes with E_{ij} differently for the two individuals
977 ($ind_{\sigma 11} < ind_{\sigma 12}$) and the change is positively correlated with individual residual variance
978 [Cov(ind_{0j}, $ind_{\sigma 1j}$) > 0]. Moreover, the individual with the smaller intercept has the larger residual
979 variance, indicating a negative covariance across levels [Cov(ind_{0j}, $ind_{\sigma 0j}$) < 0].

Figure 1.

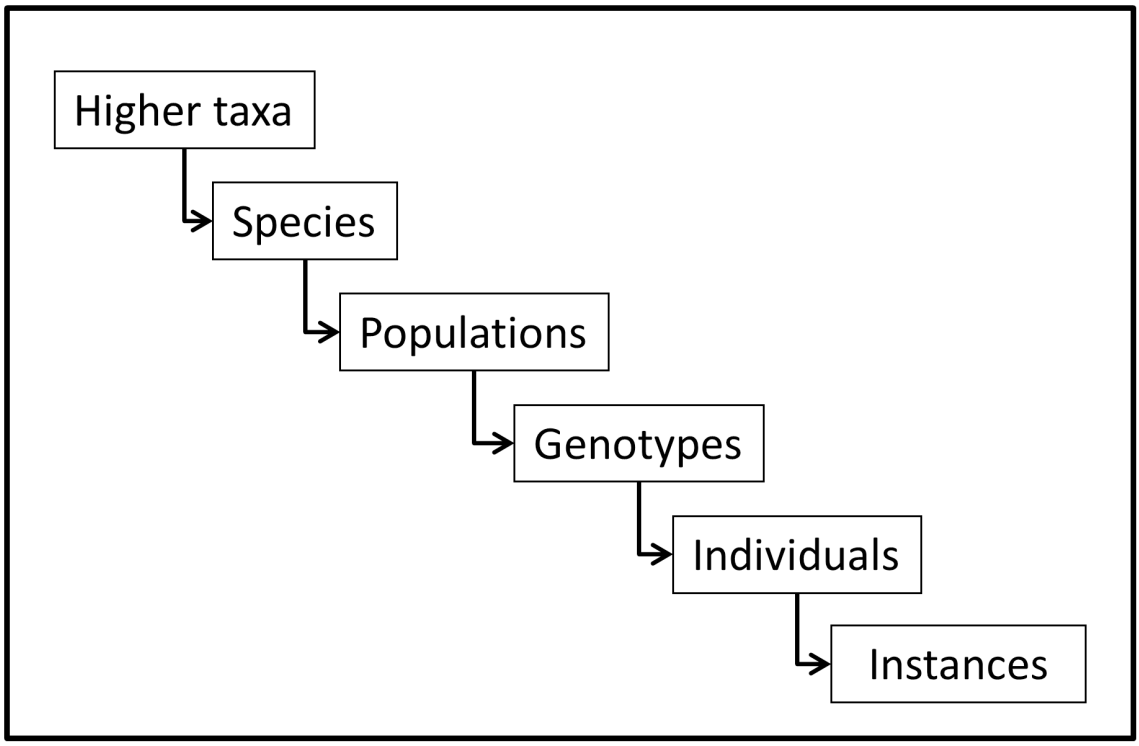
Higher taxa
Species
Populations
Genotypes
Individuals
Instances

Figure 2.

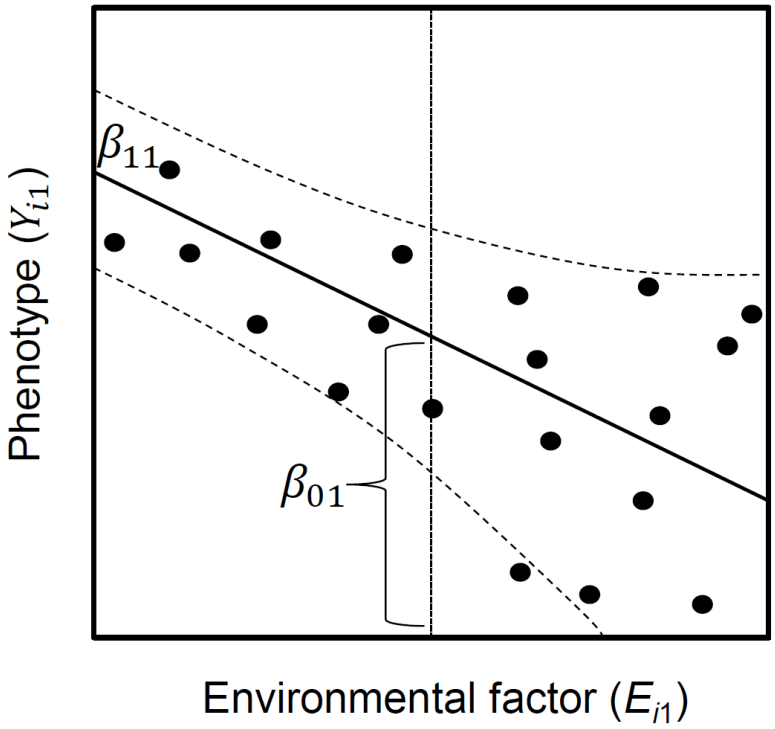

Figure 3.

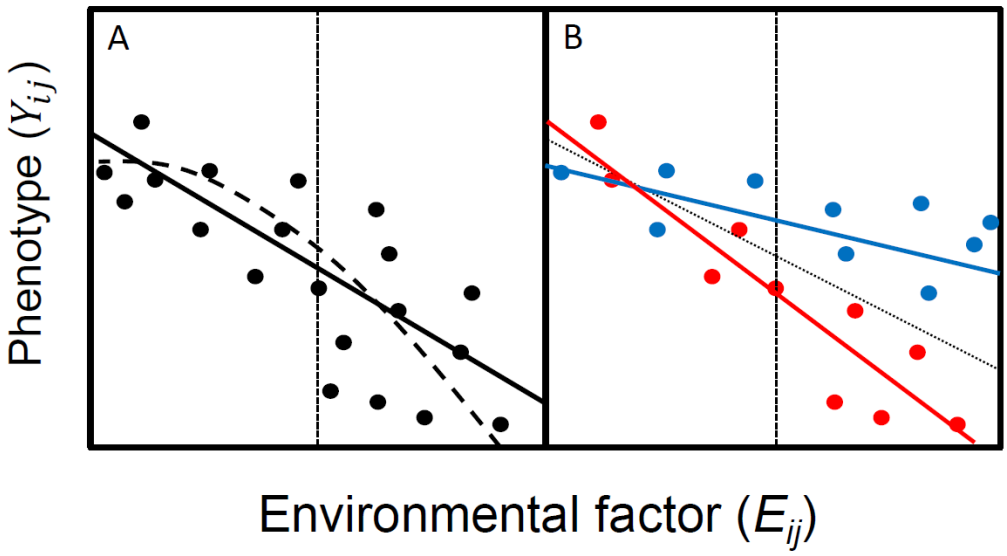

Figure 4.

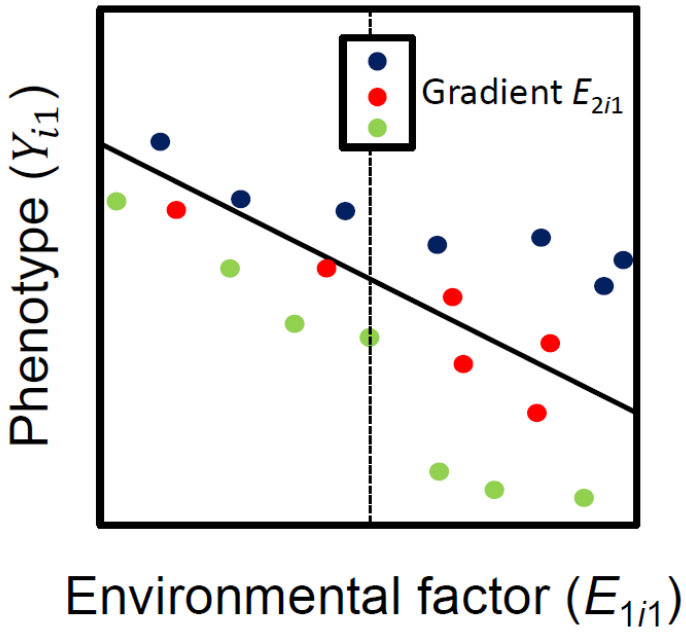

Figure 5.