

# **The biology hidden inside residual within-individual phenotypic variation**

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## 1 ABSTRACT

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

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

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

21

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## 41 I. INTRODUCTION

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

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

### 59 **(1) A statistical framework**

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

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

77         Because within-individual phenotypic change constitutes a major subtype of phenotypic  
78 plasticity, much is known about particular aspects of the biology of within-individual variance.  
79 We suggest, however, that there is an additional level to phenotypic variance that exists inside  
80 within-individual variance. This is residual within-individual variance, or unexplained within-  
81 individual variance (see Glossary in Table 1). This variance is not well integrated into  
82 evolutionary theory, leading to recent calls for more attention to be paid to this variance  
83 component (e.g. Cleasby & Nakagawa, 2011; Stamps, Briffa & Biro, 2012; Nicolaus *et al.*,  
84 2013). Thus, besides the general goals outlined above, herein we specifically explore three ideas  
85 related to residual within-individual variance: (1) patterns of apparently unexplained within-  
86 individual variance can provide clues to the existence of several important but possibly hidden  
87 biological processes; (2) this component of variance may itself evolve from several interesting  
88 types of adaptive processes; and (3) because within-individual variance is a distinct level in the  
89 hierarchical structuring of phenotypic variance, interactions with other levels are likely integral  
90 to many biological processes linking phenotype to environment. We review what is known about

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

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

104

$$105 \quad Y_{ij} = (\beta_0 + ind_{0j}) + (\beta_1 + ind_{1j})E_{ij} + e_{0ij} \quad (1)$$

106

107 where  $\beta_0$  is the population-mean phenotype [at the position where the value of the mean-centred  
108 environmental gradient ( $E_{ij}$ ) equals zero; *cf.* Dingemanse & Dochtermann, 2013];  $ind_{0j}$  represents  
109 the deviation from that mean for the  $j^{\text{th}}$  individual,  $\beta_1$  the population-mean slope with respect to  
110  $E_{ij}$ ,  $ind_{1j}$  the deviation in slope of the  $j^{\text{th}}$  individual from the population-mean slope, and  $e_{0ij}$  the  
111 residual deviation of the  $i^{\text{th}}$  instance from individual  $j$ ’s estimated reaction norm. The term  $e_{0ij}$   
112 represents the focus of this paper: unexplained deviations in phenotype within individuals. We  
113 thus explicitly distinguish between among-individual variation (the differences in average value

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

117 Equation (1) describes a linear mixed-effect model (or “random regression”). This  
118 equation is commonly used to investigate phenotypic plasticity (Nussey *et al.*, 2007), defined as  
119 the effect of an environmental factor on the phenotype with  $\beta_0$  and  $\beta_1$  describing the intercept and  
120 slope of the population mean norm-of-reaction (*sensu* Woltereck, 1909). The concept of  
121 plasticity cuts across two levels of phenotypic variance: (1) within-genotype among-individual  
122 variance, which we will call ‘developmental plasticity’ because this variance is caused by  
123 environmental effects during development (Table 1); and (2) within-individual variance. In  
124 equation (1),  $\beta_1$  refers to population average within-individual plasticity (see also the glossary in  
125 Table 1 for synonyms). The term  $ind_{0j}$  is determined by including ‘random intercepts’ for  
126 individual identity into the model, and variation among individuals in intercepts ( $V_{ind_{0j}}$ ) is  
127 consequently estimated. This variance component may reflect either genetic variance or  
128 environmental factors that have carry-over effects from one instance of expression to another  
129 (e.g. developmental plasticity: Lynch & Walsh, 1998; Wilson *et al.*, 2008; Dingemanse & Wolf,  
130 2013; Snell-Rood, 2013). The other individual term,  $ind_{1j}$ , is similarly determined by including a  
131 random effect (on the slope) arising from an interaction between individual and the  
132 environmental variable ( $E_{ij}$ ). This is individual plasticity and the associated estimate of variance  
133 among slopes ( $V_{ind_{1j}}$ ) captures differences between individuals in how they change their  
134 phenotype in response to changes in the environment they experience. Individual plasticity could  
135 also have genetic variance (e.g.  $V_{G \times E}$ ) or also arise from carry-over effects of other  
136 environmental factors (e.g.  $V_{PE \times E}$  or  $V_{G \times PE \times E}$ , where PE indicates permanent environmental

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

140 We focus here on the residual deviation ( $e_{0ij}$ ) in equation (1): the deviation of observation  
141  $i$  from individual  $j$ 's reaction norm. Residual variance (variance in  $e_{0ij}$ , or  $V_{e_0}$ ) is thus the amount  
142 of within-individual variance not explained by other terms in the model. Residual variance is  
143 important statistically because it forms the basis for testing whether sufficient evidence exists to  
144 reject a statistical null hypothesis about included terms (Cleasby & Nakagawa, 2011). Most  
145 statistical tests assume that residual variance is distributed normally and uniformly (i.e. residual  
146 variance should not differ between individuals or along the environmental gradient). However,  
147 because residual variance is never actually random and contains overlooked biology, this  
148 assumption of homogeneity may often be false (Dutilleul & Potvin, 1995; Cleasby & Nakagawa,  
149 2011) and this can have some important effects (Nicolaus *et al.*, 2013).

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



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

## 163 **II. EXPLANATIONS FOR HETEROGENEITY IN RESIDUAL WITHIN-** 164 **INDIVIDUAL VARIANCE**

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

$$167 \quad Y_{i1} = \beta_{01} + \beta_{11}E_{i1} + e_{0i1} \quad (2)$$

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

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

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

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

## 189 **(2) The inaccurate or incomplete model hypothesis**

190 A second common interpretation of residuals in a statistical model is that they include the effects  
191 of variables the investigator has not included in the model. This will likely be the case in both  
192 field and laboratory studies (see Dingemanse & Dochtermann 2014; Niemelä & Dingemanse  
193 2014), and if the research is testing specific, hypothesized influences then lumping everything  
194 else into the residual variance is sufficient to proceed. However, an alternative goal may be to  
195 seek new explanations, in which case attending to the residual variance may be valuable.  
196 Heterogeneity in residual within-individual variance may provide hints concerning the existence  
197 of various additional and potentially interesting biological processes that might be affecting  
198 phenotypic expression. We discuss here a number of key candidates.

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

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

203 Moczek *et al.*, 2002), such as in Atlantic salmon (*Salmo salar*) that exhibit genetic variation for  
204 maturation thresholds affecting alternative reproductive tactics (Piche, Hutchings & Blanchard,  
205 2008). Most studies of thresholds have investigated non-labile traits. Thresholds in labile traits  
206 also exist and show individual variation. For example, humans differ in the threshold at which a  
207 skin irritant elicits a behavioural response (Smith *et al.*, 2004) and the threshold time to process a  
208 perceptual task (e.g. Brock, Xu & Brooks, 2011).

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

### 217 (b) Slope–intercept covariance

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

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

#### 240 (c) *Multidimensional reaction norms*

241 Interactions among environmental factors affecting plastic phenotypes can also create  
242 heterogeneous residuals if not included in the phenotypic equation. Organisms live in  
243 environments that vary in many ways, and phenotypes could be a function of more than one  
244 environmental factor simultaneously. For example, herbivory and competition for light influence  
245 growth and changes in defensive compounds in plants (e.g. *Arabidopsis thaliana*; Cipollini,  
246 2004) and temperature interacts with food type to influence growth rate in larval insects (e.g.  
247 Kingsolver *et al.*, 2006; Stillwell *et al.*, 2007). These examples involve effects causing between-  
248 individual differences *via* developmental plasticity, but multiple environmental factors can

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

253         We label reaction norms that occur in response to more than one environmental factor  
254 “multidimensional” norms of reaction (Westneat *et al.*, 2009). Multidimensionality can produce  
255 heterogeneous residual within-individual variance in two ways. First, sensitivity of the  
256 phenotype to additive effects of two or more environmental variables can produce this type of  
257 heterogeneity if individuals experience only subsets of both environments. All individuals in a  
258 population might share the same reaction norm that is responsive additively to two  
259 environmental factors ( $E_1$  and  $E_2$ ). If  $E_1$  is more variable at some values of  $E_2$ , such as if  
260 territories with good food supplies also had more stable temperatures, then individuals on good  
261 territories might be less variable than those on poor territories (e.g. Charmantier & Garant,  
262 2008). While some of these effects could be fixed by better sampling by the researcher, the case  
263 of territory effects illustrates the more interesting possibility that expression of one phenotype  
264 could alter the environments experienced (a phenotype–environment correlation) and thereby  
265 affect expression of another phenotype; in this way multidimensionality combined with a  
266 phenotype–environment correlation may be the underlying cause of heterogeneous residuals.

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

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

273       Non-additive effects have implications for understanding both the ecology of plasticity  
274 and the organismal mechanisms producing it. An interaction effect may arise because of some  
275 constraint to a process involved in the trait of interest. For example, an influence of host plant  
276 diet on the thermal reaction norm of insects may arise in part because phenolic compounds  
277 present in some diets are harder to process at cooler temperatures (e.g. Diamond & Kingsolver,  
278 2012). Alternatively, interaction terms may arise because environmental variables affect fitness  
279 trade-offs in ways that produce multiple fitness peaks. For example, within-individual variance  
280 in clutch size in sparrows is influenced by an interaction between date in the season and nesting  
281 attempt order (Westneat *et al.*, 2009). This appears consistent with life-history theory that  
282 incorporates a seasonal decline in offspring quality (Rowe, Ludwig & Schluter, 1994). In this  
283 model, multiple breeding episodes create separate adaptive ridges with respect to date for each  
284 nesting attempt, producing multidimensional reaction norms affected by interactions between  
285 date and nesting attempt order. Such circumstances could select for the integration of multiple  
286 environmental cues.

287       Multidimensionality in reaction norms affects interpretations about tests of theory. For  
288 example, evolutionary theory on pleiotropic effects leading to senescence suggests that genetic  
289 variation in fitness should increase at older ages. Brommer *et al.* (2010) analysed declines with  
290 age in reproductive performance (annual fitness) in common gulls (*Larus canus*), showing  
291 among-individual variance, but little additive genetic variance, in slope with respect to age. Yet,  
292 residual variance increased with age. This heterogeneity in residuals suggests the possibility of  
293 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



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

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

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

##### 348 (a) *Passive plasticity*

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

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

364 Residual variance caused by unpredictable passive plasticity could have fitness  
365 consequences creating stabilizing selection on reaction norms. This could produce a process akin  
366 to canalization, occurring at the within-individual level, whereby the phenotype is stabilized  
367 around the optimal reaction norm (Stearns & Kawecki, 1994). However, there are also  
368 circumstances when increased residual variance may be favoured. Variance-prone foraging is  
369 one example (Stephens, 1981). Encounters with prey may be unpredictable, but if the variance in  
370 encounter times can be assessed by foragers, then individuals can make decisions to experience  
371 either more or less unpredictable passive plasticity in the instantaneous food-capture rate (Shafir,  
372 2000). Certain state variables, such as energy reserves, are predicted to create selection favouring  
373 either variance-averse or variance-prone behaviour (Caraco, Martindale & Whittam, 1980;  
374 Stephens, 1981). Studies of this idea in captivity have produced some equivocal results that may  
375 be resolved by accounting for the scale of environmental variance (reviewed in Shafir, 2000), but  
376 there remains a lack of studies conducted in the wild that properly test for such effects of  
377 ecologically relevant state variables (but see Ratikainen, Wright & Kazem, 2010).

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

379 Adaptive residual phenotypic variation could be induced by mechanisms incorporating  
380 stochasticity into phenotype expression. When this occurs at the level of among-individual but  
381 within-genotype variation, it can be a mechanism for adaptive phenotypic polymorphisms  
382 (phenotype switching, e.g. Kussel & Leibler, 2005), polyphenisms (Mayr, 1963; Van Dooren,  
383 2001) and diversification bet-hedging (Gillespie, 1973; Frank & Slatkin, 1990; Simons, 2011).

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

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

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

407 Variable display intensity by individual combatants is favoured in war-of-attrition contests  
408 (Maynard Smith, 1974). Similarly, variable waiting times for both predator and prey, possibly at  
409 both the among- and within-individual levels, are favoured when the prey is in refuge and the  
410 predator waits for them to emerge (Hugie, 2003). Briffa (2013), for example, found that residual  
411 variation in a startle response, after controlling for individual identity and mean plasticity,  
412 increased in the presence of additional cues to a predator. This type of unpredictable behaviour  
413 might also increase as individuals become familiar with individual predators (Stamps *et al.*,  
414 2012).

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

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

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

### 436 **III. INTERACTIONS ACROSS HIERARCHICAL LEVELS: GENOTYPIC AND** 437 **INDIVIDUAL DIFFERENCES IN RESIDUAL WITHIN-INDIVIDUAL VARIATION**

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

444 One well-known example involves the genotype by environment interactions ( $G \times E$ )  
445 depicted in quantitative genetics theory concerning the evolution of phenotypic plasticity (Via &  
446 Lande, 1985; Gomulkiewicz & Kirkpatrick 1992). This is typically viewed as an interaction  
447 between the among-genotype-within-a-population level (multiple genotypes exist within a  
448 population) and the among-individuals-within-genotype level (multiple environments can be  
449 experienced by different individuals with the same genotype, producing “permanent  
450 environment”, PE, variation). But, since individuals can experience different environments in  
451 their lifetimes,  $G \times E$  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 ( $G \times PE$  versus  $G \times E$ ; 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 ( $PE \times E$ ), with the possibility of there being genetic variance for  
467 this (e.g.  $G \times PE \times E$ ). 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-

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

$$476 \quad \sigma_{eij} = (\beta_{\sigma 0} + ind_{\sigma 0j}) + (\beta_{\sigma 1} + ind_{\sigma 1j})E_{ij} \quad (3)$$

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

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

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

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

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

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

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



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

#### 527 **IV. DISCUSSION**

528 Labile phenotypes, especially behavioural and physiological characters, exhibit substantial  
529 within-individual variation. We emphasize that the presence of this variation is a large, mostly  
530 untapped, opportunity to understand better the ecology of selection and evolution. The basic  
531 logic here is powerful: if variance in phenotype within an individual has fitness consequences  
532 and differences in within-individual variance exist between genotypes, then patterns of within-  
533 individual variance can evolve. Presumably, it is exactly this process that has driven the variety  
534 of mechanisms for assessing environments and producing adaptive reversible, or irreversible,  
535 phenotypic plasticity that we now see in most organisms. Although within-individual plasticity  
536 may be one of the most widespread of biological phenomena, it has usually been studied  
537 indirectly and is not well integrated conceptually. More importantly for the purposes of this  
538 review, variation in residual within-individual variance (that not explained by active plasticity)  
539 also likely underlies how individuals maintain consistent phenotypes in the face of considerable  
540 environmental variance. Such stability is an example of within-individual canalization, and has  
541 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

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

639

## 640 V. CONCLUSIONS

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

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

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

668

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## 675 **VII. REFERENCES**

676 AULD, J. R., AGRAWAL, A. A. & RELYEA, R. A. (2010). Re-evaluating the costs and limits of  
677 adaptive phenotypic plasticity. *Proceedings of the Royal Society of London B*, **277**, 503–11.

- 678 BELL, A. M., HANKISON, S. L. & LASKOWSKI, K. L. (2009) The repeatability of behaviour: a  
679 meta-analysis. *Animal Behaviour*, **77**, 771–783.
- 680 BERGMÜLLER, R. & TABORSKY, M. (2010) Animal personality due to social niche specialisation.  
681 *Trends in Ecology and Evolution*, **9**, 504–511.
- 682 BIRO, P. A. & ADRIAENSSENS, B. (2013). Predictability as a personality trait: consistent  
683 differences in intra-individual behavioral variation. *The American Naturalist*, **182**, 621–629
- 684 BOLKER, B. M., BROOKS, M. E., CLARK, C. J., GEANGE, S. W., POULSEN, J. R., STEVENS, M. H. H.  
685 & WHITE, J. S. S. (2009). Generalized linear mixed models: a practical guide for ecology and  
686 evolution. *Trends in Ecology and Evolution*, **24**, 127–135.
- 687 BREUSCH, T.S. & PAGAN, A.R. (1979). Simple test for heteroscedasticity and random coefficient  
688 variation. *Econometrica*, **47**, 1287–1294.
- 689 BRIFFA, M. (2013). Plastic proteans: reduced predictability in the face of predation risk in hermit  
690 crabs. *Biology Letters*, **9**, 20130592.
- 691 BROCK, J., XU, J. Y. & BROOKS, K. R. (2011). Individual differences in visual search: relationship  
692 to autistic traits, discrimination thresholds, and speed of processing. *Perception*, **40**, 739–742.
- 693 BROMMER, J. E., RATTISTE, K. & WILSON, A. (2010). The rate of ageing in a long-lived bird is  
694 not heritable. *Heredity*, **104**, 363–70.
- 695 BROOKE, M. D., DAVIES, N.B. & NOBLE, D. G. (1998). Rapid decline of host defences in  
696 response to reduced cuckoo parasitism: behavioural flexibility of reed warblers in a changing  
697 world. *Proceedings of the Royal Society of London B*, **265**, 1277–1282.
- 698 CAI, L., DALAL, C. K. & ELOWITZ, M. B. (2008). Frequency-modulated nuclear localization  
699 bursts coordinate gene regulation. *Nature*, **455**, 485–490.



- 700 CANNON, W. B. (1929) Organization for physiological homeostasis. *Physiological Reviews*, **9**,  
701 399-431.
- 702 CARACO, T. MARTINDALE, S. & WHITTAM, T. S. (1980). An empirical demonstration of risk  
703 sensitive foraging preferences. *Animal Behaviour*, **28**, 820-830.
- 704 CHANCELLOR, R. L. & ISBELL, L. A. (2008). Punishment and competition over food in captive  
705 rhesus macaques, *Macaca mulatta*. *Animal Behaviour*, **75**, 1939-1947.
- 706 CHANGEUX, J. P. & DANCHIN, A. (1976). Selective stabilization of developing synapses as a  
707 mechanism for specification of neuronal networks. *Nature*, **264**, 705-712.
- 708 CHARMANTIER, A. & GARANT, D. (2008). Environmental quality and evolutionary potential:  
709 lessons from wild populations. *Proceedings of the Royal Society of London B*, **272**, 1415–  
710 1425.
- 711 CIPOLLINI, D. (2004). Stretching the limits of plasticity: can a plant defend against both  
712 competitors and herbivores? *Ecology*, **85**, 28–37.
- 713 CLEASBY, I. R. & NAKAGAWA, S. (2011). Neglected biological patterns in the residuals.  
714 *Behavioral Ecology and Sociobiology*, **65**, 2361–2372.
- 715 DEBAT, V. & DAVID, P. (2002). Mapping phenotypes: canalization, plasticity and developmental  
716 stability. *Trends in Ecology and Evolution*, **16**, 555-561.
- 717 DEWITT, T. J., SIH, A. & WILSON, D. S. (1998). Costs and limits of phenotypic plasticity. *Trends*  
718 *in Ecology and Evolution*, 13,77–81.
- 719 DIAMOND, S. E. & KINGSOLVER, J. G. (2012). Host plant adaptation and the evolution of thermal  
720 reaction norms. *Oecologia*, **169**, 353–60.

- 721 DINGEMANSE, N. J., BARBER, I., WRIGHT, J. & BROMMER, J. E. (2012). Quantitative genetics of  
722 behavioural reaction norms: genetic correlations between personality and behavioural  
723 plasticity vary across stickleback populations. *Journal of Evolutionary Biology*, **25**, 485-496.
- 724 DINGEMANSE, N. J. & DOCHTERMANN, N. A. (2013). Quantifying individual variation in  
725 behaviour: mixed-effect modelling approaches. *Journal of Animal Ecology*, **82**, 39-54.
- 726 DINGEMANSE, N. J. & DOCHTERMANN, N. A. (2014). Individual behaviour: Behavioural ecology  
727 meets quantitative genetics. In: *Quantitative Genetics in the Wild* (eds. A. CHARMANTIER, D.  
728 GARANT & L. E. B. KRUK). In press, Oxford University Press, Oxford, UK.
- 729 DINGEMANSE, N. J., KAZEM, A. J. N., RÉALE, D. & WRIGHT, J. (2010). Behavioural reaction  
730 norms: animal personality meets individual plasticity. *Trends in Ecology and Evolution*, **25**,  
731 81–89.
- 732 DINGEMANSE, N. J. & WOLF M. (2013). Between-individual differences in behavioural plasticity  
733 within populations: causes and consequences. *Animal Behaviour*, **85**, 1031-1039
- 734 DUKAS, R. (1998). Constraints on information processing and their effects on behavior. In:  
735 *Cognitive Ecology: The Evolutionary Ecology of Information Processing and Decision-*  
736 *Making* (ed. R. DUKAS), pp. 89–128. University of Chicago Press, Chicago, IL.
- 737 DUTILLEUL, P. & POTVIN, C. (1995). Among-environment heteroscedasticity and genetic  
738 autocorrelation: implications for the study of phenotypic plasticity. *Genetics*, **139**, 1815–  
739 1829.
- 740 DWORKIN, I. (2005). Canalization, cryptic variation, and developmental buffering. In: *Variation:*  
741 *A Central Concept in Biology* (eds. B. HALGRIMSSON & B. K. HALL), pp. 131-158. Academic  
742 Press, New York, NY.

- 743 ELДАР, A. & ELOWITZ M. B. (2010). Functional roles for noise in genetic circuits. *Nature*, **467**,  
744 167-173.
- 745 FRANK, S. A. (1997). The design of adaptive systems: optimal parameters for variation and  
746 selection in learning and development. *Journal of Theoretical Biology*, **184**, 31-39.
- 747 FRANK, S. A. & SLATKIN, M. (1990). Evolution in a variable environment. *The American*  
748 *Naturalist*, **136**, 244–260.
- 749 GETTY, T. (1996). The maintenance of phenotypic plasticity as a signal detection problem. *The*  
750 *American Naturalist*, **148**, 378–385.
- 751 GHALAMBOR, C. K., ANGELONI, L. M. & CARROLL S. P. (2010). Behavior as phenotypic  
752 plasticity. In: *Evolutionary Behavioral Ecology* (eds. D. F. WESTNEAT & C. W. FOX), pp 90-  
753 107. Oxford University Press, New York, NY.
- 754 GIBBON, J. (1977). Scalar expectancy theory and Weber's law in animal timing. *Psychology*  
755 *Reviews*, **84**, 279–325.
- 756 GIBSON, G. & WAGNER, G. (2000). Canalization in evolutionary genetics: A stabilizing theory?  
757 *Bioessays*, **22**, 372-380.
- 758 GIL, D. (2008). Hormones in avian eggs: physiology, ecology and behavior. *Advances in the*  
759 *Study of Behavior*, **38**, 337–398
- 760 GILLESPIE, J. H. (1973). Polymorphism in random environments. *Theoretical Population*  
761 *Biology*, **4**, 193–195.
- 762 GOLUB, E. S. & GREEN, D. R. (1991). *Immunology: A Synthesis*, 2<sup>nd</sup> ed. Sinauer, Sunderland,  
763 MA.
- 764 GOMULKEIWICZ, R. & KIRKPATRICK, M. (1992). Quantitative genetics and the evolution of  
765 reaction norms. *Evolution*, **46**, 390-411.

- 766 HANSEN, T. F., CARTER, A. J. R. & PÉLABON, C. (2006). On adaptive accuracy and precision in  
767 natural populations. *The American Naturalist*, **168**, 168–81.
- 768 HILL, W. G. & MULDER, H. A. (2010). Genetic analysis of environmental variation. *Genetics*  
769 *Research*, **92**, 381-395.
- 770 HUGIE, D. M. (2003). The waiting game: a "battle of waits" between predator and prey.  
771 *Behavioral Ecology*, **14**, 807-817.
- 772 JACKSON, R. R. & WILCOX, R. S. (1993). Spider flexibly chooses aggressive mimicry signals for  
773 different prey by trial and error. *Behaviour*, **127**, 21-36.
- 774 JOHNSTONE, R. A. (2001). Eavesdropping and animal conflict. *Proceedings of the National*  
775 *Academy of Sciences USA*, **98**, 9177–9180.
- 776 KAGAN, M. L., NOVOPLANSKY, N. & SACHS, T. (1992). Variable cell lineages from the functional  
777 pea epidermis. *Annals of Botany*, **69**, 303-312.
- 778 KELLNER, J. R. & ALFORD, R. A. (2003). The ontogeny of fluctuating asymmetry. *The American*  
779 *Naturalist*, **161**, 931-947.
- 780 KINGSOLVER, J. G., SHLICHTA, J. G., RAGLAND, G. J. & MASSIE, K. R. (2006). Thermal reaction  
781 norms for caterpillar growth depend on diet. *Evolutionary Ecology Research*, **8**, 703–715.
- 782 KONTIAINEN, P., PIETIÄINEN, H., HUTTUNEN, K., KARELL, P., KOLUNEN, H. & BROMMER, J. E.  
783 (2009). Aggressive Ural owl mothers recruit more offspring. *Behavioral Ecology*, **20**, 789-  
784 796.
- 785 KUSSEL, E. & LEIBLER, S. (2005). Phenotypic diversity, population growth, and information in  
786 fluctuating environments. *Science*, **309**, 2075–2078.
- 787 LEE, Y. & NELDER, J. A. (1996). Hierarchical generalized linear models (with Discussion).  
788 *Journal of the Royal Statistical Society B*, **58**, 619-678.

- 789 LINDHOLM, A. & THOMAS, R. (2000). Differences between populations of reed warblers in  
790 defences against brood parasitism. *Behaviour*, **137**, 25–42.
- 791 LOSICK, R. & DESPLAN, C. (2008). Stochasticity and cell fate. *Science*, **320**, 65–68.
- 792 LOTEM, A., NAKAMURA, H. & ZAHAVI, A. (1995). Constraints on egg discrimination and cuckoo-  
793 host co-evolution. *Animal Behaviour*, **49**, 1185-1209.
- 794 LYNCH, M. & WALSH, B. (1998). *Genetics and Analysis of Quantitative Traits*. Sinauer Press,  
795 Sunderland, MA.
- 796 MARKOW, T. A. (1995). Evolutionary ecology and developmental instability. *Annual Reviews of*  
797 *Entomology*, **40**, 105-120.
- 798 MARTIN, T. M., TON, R. & NIKLISON, A. (2013). Intrinsic versus extrinsic influences on life  
799 history expression: metabolism and parentally induced temperature influences on embryo  
800 development rate. *Ecology Letters*, **16**, 738-745.
- 801 MARTIN, J. G. A., NUSSEY, D., WILSON, A. & RÉALE, D. (2011). Measuring individual differences  
802 in reaction norms in field and experimental studies: a power analysis of random regression  
803 models. *Methods in Ecology and Evolution*, **2**, 362-374.
- 804 MATHOT, K. J., WRIGHT, J., KEMPENAERS, B. & DINGEMANSE, N. J. (2012). Adaptive strategies  
805 for managing uncertainty may explain personality-related differences in behavioural  
806 plasticity. *Oikos*, **121**, 1009–1020.
- 807 MAYNARD SMITH, J. (1974). Theory of games and the evolution of animal contests. *Journal of*  
808 *Theoretical Biology*, **47**, 209-221.
- 809 MAYR, E. (1963). *Animal species and evolution*. Belknap Press/Harvard University Press,  
810 Cambridge, MA.

- 811 MOCZEK, A. P., HUNT, J., EMLEN, D. J. & SIMMONS, L. W. (2002). Threshold evolution in exotic  
812 populations of a polyphonic beetle. *Evolutionary Ecology Research*, **4**, 587–601.
- 813 MOORE, A. J., BRODIE III, E. D. & WOLF, J. B. (1997). Interacting phenotypes and the  
814 evolutionary process: I. Direct and indirect genetic effects of social interactions. *Evolution*,  
815 **51**, 1352-1362.
- 816 MORAN, N. A. (1992). The evolutionary maintenance of alternative phenotypes. *The American*  
817 *Naturalist*, **139**, 971–989.
- 818 MÜLLER, M. S., ROELOFS, Y., ERIKSTAD, K. E. & GROOTHUIS, T. G. G. (2012). Maternal  
819 androgens increase sibling aggression, dominance, and competitive ability in the siblicidal  
820 black-legged kittiwake (*Rissa tridactyla*). *PLoS ONE*, 7(10), e47763.  
821 doi:10.1371/journal.pone.0047763
- 822 NATARAJAN, D., DEVRIES, H., SAALTINK, D. J., DE BOER, S. F. & KOOLHAAS, J. (2009).  
823 Delineation of violence from functional aggression in mice: an ethological approach.  
824 *Behavioral Genetics*, **39**, 73-90.
- 825 NESSE, R. M. (2001). *Evolution and the Capacity for Commitment*. Russell Sage Foundation,  
826 New York, NY.
- 827 NICOLAUS, M., BROMMER, J. E., UBELS, R., TINBERGEN, J. M. & DINGEMANSE, N. J. (2013).  
828 Exploring patterns of variation in clutch size-density reaction norms in a wild passerine bird.  
829 *Journal of Evolutionary Biology*, **26**, 2031-2043.
- 830 NIEMELÄ, P. T. & DINGEMANSE, N. J. (2014). Artificial environments and the study of "adaptive"  
831 personalities. *Trends in Ecology and Evolution*, **29**, 245-247.

- 832 NIJHOUT, H. F. & DAVIDOWITZ, G. (2003). Developmental perspectives on phenotypic plasticity,  
833 canalization, and fluctuating asymmetry. In: *Developmental Instability: Causes and*  
834 *Consequences* (ed. M. POLAK) , pp. 3-13. MIT Press, Boston, MA.
- 835 NUSSEY, D. H., WILSON, A. J. & BROMMER, J. E. (2007). The evolutionary ecology of individual  
836 phenotypic plasticity in wild populations. *Journal of Evolutionary Biology*, **20**, 831–44.
- 837 PICHE, J., HUTCHINGS, J. A. & BLANCHARD, W. (2008). Genetic variation in threshold reaction  
838 norms for alternative reproductive tactics in male Atlantic salmon, *Salmo salar*. *Proceedings*  
839 *of the Royal Society of London B*, **275**, 1571-1575.
- 840 PIERSMA, T. & DRENT, J. (2003). Phenotypic flexibility and the evolution of organismal design.  
841 *Trends in Ecology and Evolution*, **18**, 228–233.
- 842 PLAYFAIR, J. H. L. (1995). *Infection and Immunity*, 2<sup>nd</sup> ed. Oxford University Press, New York,  
843 NY.
- 844 RATIKAINEN, I. I., WRIGHT, J. & KAZEM, A. J. N. (2010). Social class influences degree of  
845 variance sensitivity in wild Siberian jays. *Behavioral Ecology*, **21**, 1067-1072.
- 846 REEVE, H. K. (1989). The evolution of conspecific acceptance thresholds. *The American*  
847 *Naturalist*, **133**, 407–435.
- 848 ROSS, H. E. & MURRAY, D. J. (1996). *E. H. Weber on the tactile senses*, 2<sup>nd</sup> ed. Erlbaum, Taylor  
849 & Francis, Hove, UK.
- 850 ROTHSTEIN, S. I. (1978). Mechanisms of avian egg-recognition: Additional evidence for learned  
851 components. *Animal Behaviour*, **26**, 671–677.
- 852 ROWE, L., LUDWIG, D. & SCHLUTER, D. (1994). Time, condition, and the seasonal decline of  
853 avian clutch size. *The American Naturalist*, **143**, 698–722.

- 854 SCHAEFFER, L. R. (2004). Application of random regression models in animal breeding.  
855 *Livestock Production Science*, **86**, 35–45.
- 856 SCHEINER, S. M. (2006). Genotype-environment interactions and evolution. In: *Evolutionary*  
857 *genetics: concepts and case studies* (eds. C. W. FOX & J. B. WOLF) , pp. 326–338. Oxford  
858 University Press, New York, NY.
- 859 SCHUETT, W., TREGENZA, T. & DALL, S. R. X. (2010). Sexual selection and animal personality.  
860 *Biological Reviews*, **85**, 217-246.
- 861 SHAFIR, S. (2000). Risk-sensitive foraging: the effect of relative variability. *Oikos*, **88**, 663–669.
- 862 SHERMAN, P. W., REEVE, H. K. & PFENNIG, D. W. (1997). Recognition systems. In: *Behavioural*  
863 *Ecology: An Evolutionary Approach*. Fourth edition. (eds J. R. KREBS & N. B. DAVIES), pp.  
864 69–96. Blackwell Scientific, Oxford, UK.
- 865 SIMONS, A. M. (2011). Modes of response to environmental change and the elusive empirical  
866 evidence for bet-hedging. *Proceedings of the Royal Society of London B*, **278**, 1601-1609.
- 867 SMITH, H. R., ROWSON, M., BASKETTER, D. A. & MCFADDEN, J. P. (2004). Intra-individual  
868 variation of irritant threshold and relationship to trans-epidermal water loss measurement of  
869 skin irritation. *Contact Dermatitis*, **51**, 26-29.
- 870 SMYTH, G. K. & VERBYLA, A. P. (1999). Adjusted likelihood methods for modeling dispersion in  
871 generalized linear models. *Environmetrics*, **10**, 695-710.
- 872 SNELL-ROOD, E. C. (2013). An overview of the evolutionary causes and consequences of  
873 behavioural plasticity. *Animal Behaviour*, **85**, 1004-1011.
- 874 STAMPS, J. A., BRIFFA, M. & BIRO, P. A. (2012). Unpredictable animals: individual differences in  
875 intraindividual variability (IIV). *Animal Behaviour*, **83**, 1325–1334.



- 876 STAMPS, J. A. & GROOTHUIS, T. G. G. (2010). Developmental perspectives on personality:  
877 implications for ecological and evolutionary studies of individual differences. *Philosophical*  
878 *Transactions of the Royal Society B*, **365**, 4029-4041.
- 879 STEARNS, S. C. & KAWECKI, T. J. (1994). Fitness sensitivity and the canalization of life-history  
880 traits. *Evolution*, **48**, 1438-1450.
- 881 STEPHENS, D. W. (1981). The logic of risk-sensitive foraging preferences. *Animal Behaviour*, **29**,  
882 628–629.
- 883 STILLWELL, R. C., WALLIN, W. G., HITCHCOCK, L. J. & FOX, C. W. (2007). Phenotypic plasticity  
884 in a complex world: interactive effects of food and temperature on fitness components of a  
885 seed beetle. *Oecologia*, **153**, 309–321.
- 886 STOKKE, B. G., HAFSTAD, I., RUDOLFSEN, G., MOKSNES, A., MØLLER, A. P., ROSKRAFT, E. &  
887 SOLER, M. (2008). Predictors of resistance to brood parasitism within and among reed  
888 warbler populations. *Behavioral Ecology*, **19**, 612–620.
- 889 TONSOR, S. J., ELNACCASH, T. W. & SCHEINER, S. M. (2013). Developmental instability is  
890 genetically correlated with phenotypic plasticity, constraining heritability, and fitness.  
891 *Evolution*, **67**, 2923-2935.
- 892 VAN DE POL, M. (2012). Quantifying individual variation in reaction norms: how study design  
893 affects the accuracy, precision and power of random regression models. *Methods in Ecology*  
894 *and Evolution*, **3**, 268-280.
- 895 VAN DOOREN, T. J. M. (2001). Reaction norms with bifurcations shaped by evolution.  
896 *Proceedings of the Royal Society of London B*, **268**, 279–287.
- 897 VAN VALEN, L. (1962). A study of fluctuating asymmetry. *Evolution*, **16**, 125–142.

- 898 VIA, S. & LANDE, R. (1985). Genotype-environment interaction and the evolution of phenotypic  
899 plasticity. *Evolution*, **39**, 505-522.
- 900 VISWANATHAN, M. (2005). *Measurement error and research design*. Sage Publications,  
901 Thousand Oaks, CA.
- 902 WADDINGTON, C. H. (1942). Canalization of development and the inheritance of acquired  
903 characters. *Nature*, **150**, 563-565
- 904 WEINIG C. & DELPH, L. F. (2001). Phenotypic plasticity early in life constrains developmental  
905 responses later. *Evolution*, **55**, 930-936.
- 906 WEST-EBERHARD, M. J. (2003). *Developmental plasticity and evolution*. Oxford University  
907 Press, Oxford, UK.
- 908 WESTNEAT, D. F., HATCH, M. I., WETZEL, D. P. & ENSMINGER, A. L. (2011). Individual variation  
909 in parental care reaction norms: integration of personality and plasticity. *The American*  
910 *Naturalist*, **178**, 652–667.
- 911 WESTNEAT, D. F., SCHOFIELD, M. & WRIGHT, J. (2013). Parental behavior exhibits between-  
912 individual variance, plasticity and heterogeneous residual variance. *Behavioral Ecology*, **24**,  
913 598-604.
- 914 WESTNEAT, D. F., STEWART, I. R. K. & HATCH, M. I. (2009). Complex interactions among  
915 temporal variables affect the plasticity of clutch size in a multi-brooded bird. *Ecology*, **90**,  
916 1162–1174.
- 917 WHITE, H. (1980). A heteroskedasticity-consistent covariance matrix estimator and a direct test  
918 for heteroskedasticity. *Econometrica*, **48**, 817–838.
- 919 WHITEN, A. & BYRNE, R.W. (1997). *Machiavellian Intelligence II*. Cambridge University Press,  
920 Cambridge, UK.

- 921 WILEY, R. H. (1994). Errors, exaggeration and deception in animal communication. In:  
922 *Behavioural Mechanisms in Evolutionary Ecology* (ed. L. A. REAL), pp. 157–189. University  
923 of Chicago Press, Chicago, IL.
- 924 WILSON, A. J., RÉALE, D., CLEMENTS, M. N., MORRISSEY, M. M., POSTMA, E., WALLING, C. A.,  
925 KRUIK, L. E. B. & NUSSEY, D. H. (2008). An ecologist's guide to the animal model. *Journal*  
926 *of Animal Ecology*, **79**, 13–26.
- 927 WOLLERMAN, L. & WILEY, R. H. (2002). Background noise from a natural chorus alters female  
928 discrimination of male calls in a Neotropical frog. *Animal Behaviour*, **63**, 15–22.
- 929 WOLTERECK, R. (1909). Weitere experimentelle Untersuchungen uber Artveränderung, speziell  
930 uber das Wesen quantitativer Artunterschiede bei Daphniden. *Versuche Deutsche Zoologische*  
931 *Gesellschaft*, **19**, 110–172.
- 932 YDENBERG, R. C. (1994). The behavioral ecology of provisioning in birds. *Ecoscience*, **1**, 1-14.

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

935

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

	be explained by non-biological processes ( <i>sensu</i> Scheiner, 2006)	
<b>Phenotypic plasticity</b>	A change in the phenotype expressed by a genotype or individual with respect to a difference in environment, either <b>passive</b> or <b>active plasticity</b>	Plasticity; flexibility
<b>Residual within-individual variance</b>	Amount of <b>within-individual variance</b> 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
<b>Within-genotype among-individual variance</b>	Variance in mean phenotype among individuals of a given genotype, measured in a specified environment	<b>Among-individual variation</b>
<b>Within-individual plasticity</b>	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
<b>Within-individual variance</b>	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 <i>et al.</i> , 2012)

936

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 <i>et al.</i> (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 <i>et al.</i> (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 <i>et al.</i> (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)

941

942

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 ( $\beta_{01}$ ) is the elevation and is appropriately  
954 taken at the mean-centred environment, and the slope ( $\beta_{11}$ ) 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.

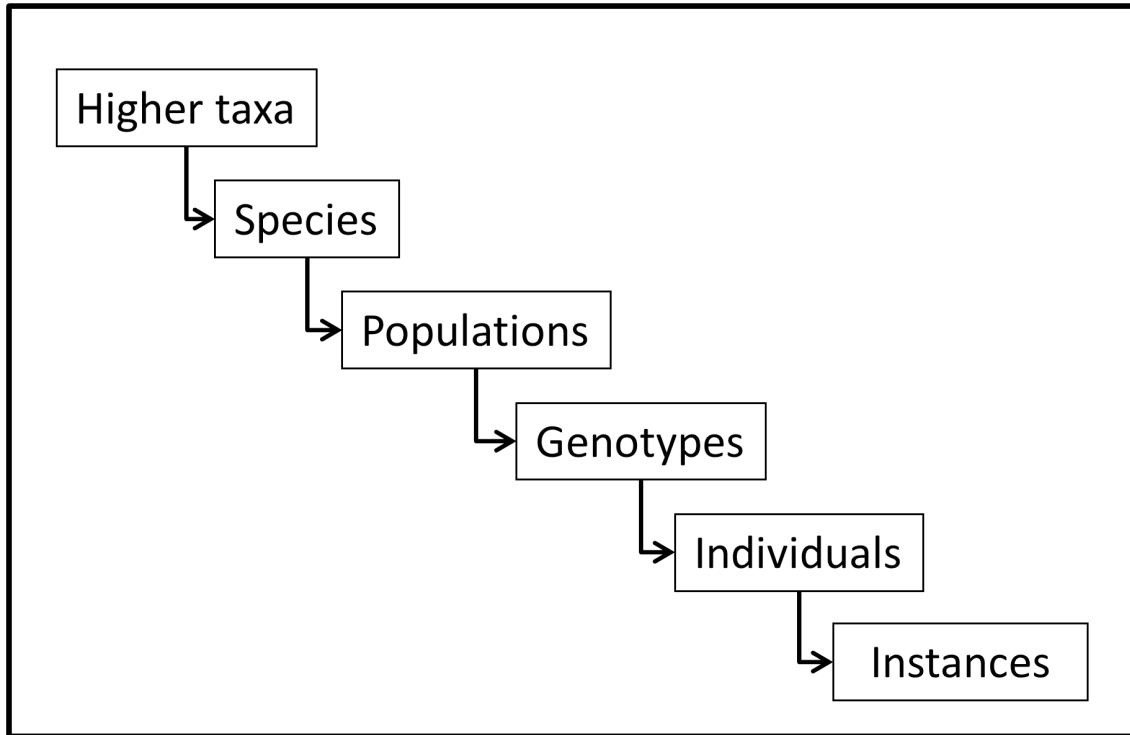


Figure 2.

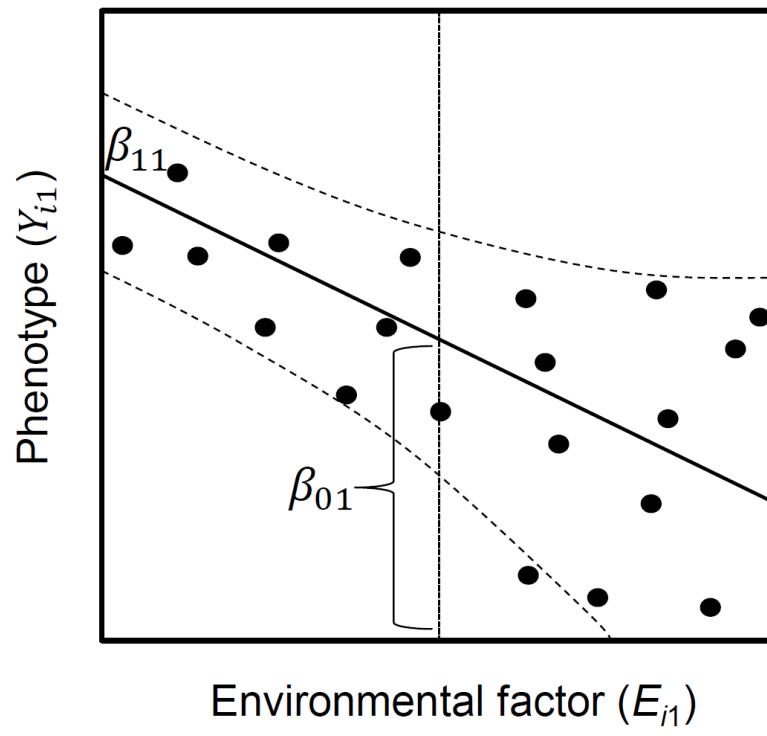


Figure 3.

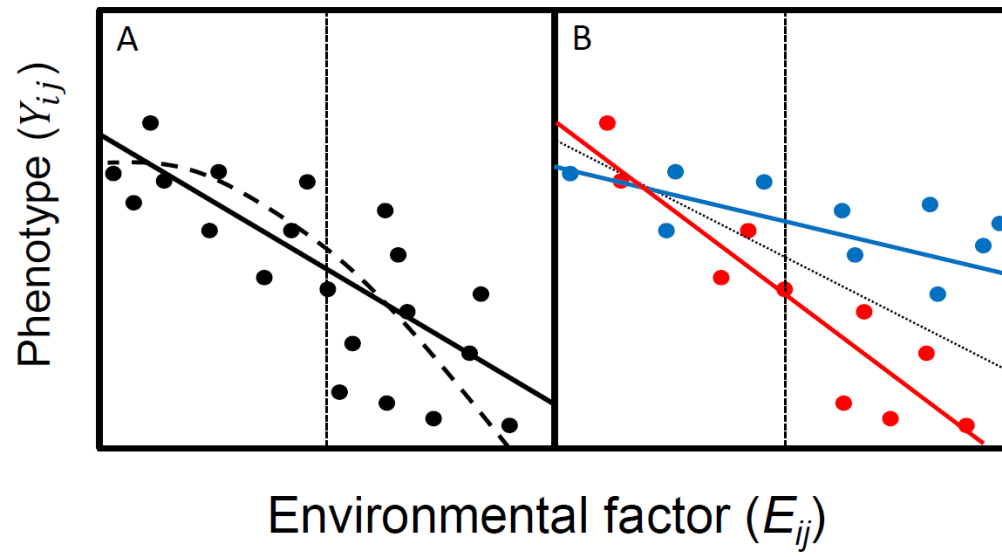


Figure 4.

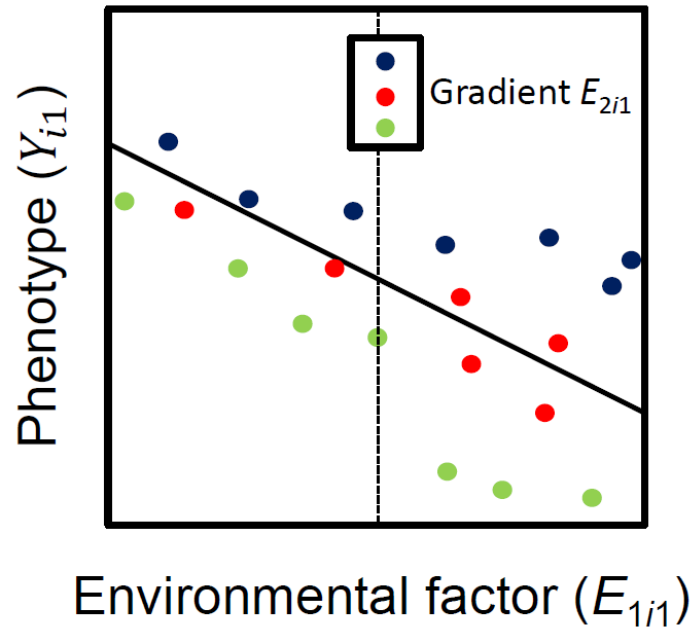


Figure 5.

