Unifying individual differences in personality, predictability, and plasticity: a practical guide

Authors

Rose E. O'Dea[1,2*], Daniel W.A. Noble[3], Shinichi Nakagawa[1,2]

Affiliations

[1]Evolution & Ecology Research Centre, School of Biological and Environmental Sciences, University of New South Wales, Sydney, NSW, 2052, Australia.

[2]Diabetes and Metabolism Division, Garvan Institute of Medical Research, 384 Victoria Street, Darlinghurst, Sydney, NSW 2010, Australia

[3]Division of Ecology and Evolution, Research School of Biology, The Australian National University, Canberra, Australia.

**Corresponding author:* rose.eleanor.o.dea@gmail.com

Author contributions statement

Rose O'Dea: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Software; Visualisation; Writing – original draft; Writing – reviewing & editing.

Daniel Noble: Conceptualization; Funding acquisition; Investigation; Methodology; Resources; Writing – reviewing & editing.

Shinichi Nakagawa: Conceptualization; Funding acquisition; Investigation; Methodology; Resources; Software; Supervision; Writing – reviewing & editing.

Data availability statement

All data, code, and model objects required to reproduce the results presented in this manuscript are available from a dedicated repository at: http://doi.org/10.17605/OSF.IO/V3QAX

Supplementary information

Available to download from https://osf.io/mws98/

Abstract

1. Many animal species show individual differences in behaviour that are partially consistent across repeated measurements. Commonly referred to as personality traits, differences in average behaviours are often correlated across individuals, forming 'behavioural syndromes' (e.g. individuals who are more aggressive are also bolder).

2. Generally, differences in the average behaviour of individuals explains less than half the variation in behavioural traits. To explain the rest, we need to consider how individuals themselves vary both plastically and unpredictably.

3. Here, we integrate the study of multiple individual differences. With a reproducible worked example based on zebrafish (*Danio rerio*) behaviours, we give guidance for measuring individual differences in average phenotypes (i.e. personality), responses to an environmental or biological context (i.e. plasticity), and intrinsic variability across time (i.e. predictability, or intra-individual variability).

4. Individuality is multi-faceted. By modelling personality, plasticity, and predictability simultaneously, empiricists can quantify how these traits covary across individuals, and test theoretical ideas about phenotypic integration. We provide detailed descriptions and resources for measuring behavioural syndromes, plasticity syndromes, predictability syndromes, personality-plasticity associations, personality-predictability associations, and plasticity-predictability associations. These methods can be extended to incorporate plastic changes in predictability (termed 'stochastic malleability').

5. Overall, we showcase the unfulfilled potential of existing statistical tools. Empiricists can use these methods to test more holistic and nuanced questions about the evolution, function, and maintenance of phenotypic variation, for any trait that is repeatedly expressed.

Introduction

Over the past decade, behavioural ecologists have studied how behaviours vary across environments, and between individuals within populations (Allegue et al., 2017). Across environments, the average behaviour of a population can show phenotypic plasticity, either as an adaptive response to predictable environmental change, or a maladaptive consequence of environmental stress (Snell-Rood, 2013). Within a population, individuals can vary both in their average behavioural response to a given environment, and in their plastic response to environmental change; these individual differences can be measured with multilevel models (Dingemanse & Dochtermann, 2013; Dingemanse et al., 2010b). Most commonly, researchers use multilevel models to measure how consistently individuals differ from each other in a stable environment, for one or more behavioural traits. These 'animal personality' studies have identified 'behavioural syndromes', whereby individual differences are correlated across behaviours (e.g. some individuals are consistently more risk-averse) (Bell, 2007; Dingemanse et al., 2010a; Dochtermann, 2010; Sih et al., 2004). While the 'consistency' of individual differences in behaviour has received much attention, in general most behavioural variation is driven not by differences between individuals, but instead by residual variation (meta-analysis of repeatability: Bell et al., 2009).

Residual variation is a composite of both biologically meaningful variation (e.g. within-individual variation), and variation that is inherent to the method of measurement. To estimate intra-individual variability from residual variance, we could quantify the precision of our measurements, and manually subtract the portion of residual variance that is attributable to measurement error. More simply, by using a measurement method that is as precise as possible, we explicitly assume that residual variance approximately represents within-individual variance. Regardless of the proportion of residual variance that we take to represent intra-individual variability, we hereafter refer to magnitude of within-individual variance as 'predictability' (Cleasby et al., 2015).

Standard multilevel models assume that each individual has the same level of predictability (i.e. assume homogeneity of residual variances). The homogeneity assumption is violated when some individuals are more variable than others across time (Ramakers et al., 2020), and this 'heteroscedasticity' could represent interesting non-adaptive deviations from an optimal phenotype (e.g. maladaptive imprecision; Hansen et al., 2006), or adaptive variation between individuals in their level of predictability (e.g. alternative strategies; Wolf et al., 2007). The underlying mechanisms that dictate predictability are likely to be shared across different phenotypic traits. Such 'phenotypic

integration' could lead to a trade-off that constrains or maintains phenotypic variance for a given trait, where individuals are more predictable than optimal for some traits, and less predictable than optimal for others (Pigliucci, 2003; Viney & Reece, 2013; Willmore et al., 2007).

Statistical methods for studying individual differences in labile (i.e. repeatedly expressed) traits will be most powerful when multiple individual differences are considered together. The field of animal personality has spent over a decade quantifying individual differences in intercepts (personalities) and their between-individual correlations (behavioural syndromes). We can take the same approach to the study of two other types of individual differences (Fig. 1): slopes for environmental variables (plasticity) and within-individual variances (predictability). In addition to behavioural syndromes, below we describe and quantify five other biologically meaningful correlations. This major extension will allow empiricists to study factors contributing to the maintenance of within-individual variation. Quantifying phenotypic integration among personality, plasticity, and predictability is within the reach of many, however without a guide, navigating the statistical methods could discourage the uninitiated. Here, we lower the barrier to entry for quantifying various types of individual differences, and their correlations, for multiple phenotypic traits. Using a reproducible worked example with zebrafish behavioural data, we demonstrate how researchers can use these statistical tools to tackle biological questions.

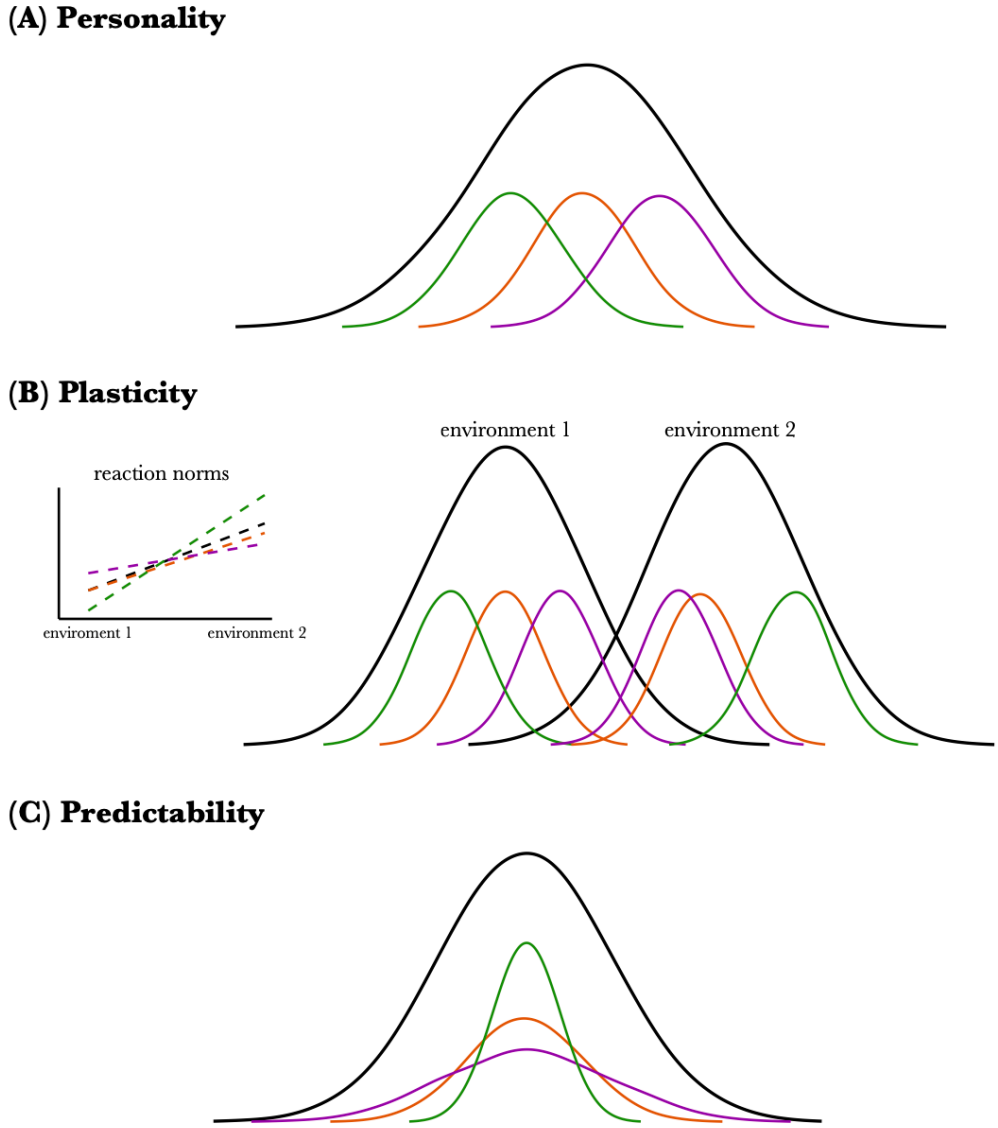

Figure 1

Conceptual illustration of three types of individual differences for a labile trait (in this case, behaviour). In each panel, black curves represent the normal distribution of a phenotypic trait in a population. Smaller, coloured curves represent the distribution of phenotypes expressed by an individual within that population. (**A**) 'Personality': individual differences in average behaviour. (**B**) 'Plasticity' due to a change in the environment. In environment 2, compared with environment 1, the average phenotype of the population increases, as shown by the black distribution shifting to the right. Individuals' averages (distributions) shift to varying extents (i.e. variation in reaction norm slopes). (**C**) 'Predictability': individuals' level of variability (the breadth of individual distributions). Even individuals with the same 'personality' can show differences in predictability.

Table 1: Mathematical notation describing statistical models

Throughout this paper we assume that we are modelling behavioural traits in a multilevel model

framework, and we are interested in the biological variables of sex, age, and individual identity. Note that when presenting square matrices, the bottom triangle elements are omitted for simplicity (as they are identical to the upper triangle).

Notation	Definition
y_{ij}	Response variable (i.e., a behavioural trait): the measured phenotypic value of trait *y* for the *j*th individual at instance *i*.
^{t1}	Superscript is used for bivariate models, to indicate model parameters for trait 1 (^{t1}) and trait 2 (^{t2}).
e_{ij}	Residual error: difference between the predicted and fitted value for the *j*th individual at instance *i*.
σ_e^2	Residual variance for single hierarchical models ('mean' model only).
$\sigma_{e_{ij}}^2$	Residual variance for double hierarchical models ('mean' and 'dispersion' models): unique value for each individual and instance.
x_{1j}	Categorical input variable for the 'sex' of individual *j* ($x_{1j} = 0$ for female, and 1 for male).
x_{2ij}	Continuous input variable for the *z*-transformed 'age' of individual *j* at instance *i* ($x_{2ij} = 0$ is the average age of the population).
β_{m0}	Population intercept for the mean model. Average value of *y* when all other input variables are set to zero (females of average age).
$\beta_{\mathrm{v0,exp}}$	Population intercept for the dispersion (variance) model. Average value of $\ln(\sigma_{e_{ij}}^2)$when all other input variables are set to zero (females of average age). Estimated on the natural logarithm (ln) scale.
β_{m1}	Population slope for the female-male contrast for the mean model.
$\beta_{\mathrm{v1,exp}}$	Population slope for the female-male contrast for the dispersion model). Estimated on the ln scale.
β_{m2}	Population slope. Average value of phenotypic plasticity (reaction norm) for x_{2ij} = *z*-scaled age, for the mean model.
$\beta_{\mathrm{v2,exp}}$	Population slope. Average value of phenotypic plasticity (reaction norm) for x_{2ij} = *z*-scaled age, for the dispersion model). Estimated on the ln scale.
$\mathrm{ID}_{\mathrm{m0}j}$	Deviation between the population intercept β_{m0} and the random intercept for individual *j* for the mean model.

Table continued on next page

Table 1 — *continued*

Notation	Definition
$\mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}$	Deviation between the population intercept β_{v0} and the random intercept for individual *j* for the dispersion model. Estimated on the ln scale.
$\mathrm{ID}_{\mathrm{m2}j}$	Deviation between the population slope β_{m2} and the random slope for individual *j* for the mean model.
$\lvert\beta_{\mathrm{m2}} + \mathrm{ID}_{\mathrm{m2}j}\rvert$	Absolute value of the (age) slope for individual *j* for the mean model. Describes the magnitude of individuals' average plasticity.
$\sigma^2_{\mathrm{ID}_{\mathrm{m0}}}$	Between-individual variance for the individual intercepts for the mean model.
$\sigma^2_{\mathrm{ID}_{\mathrm{m2}}}$	Between-individual variance for the individual slopes for the mean model.
$\sigma^2_{\mathrm{ID}_{\mathrm{v0,exp}}}$	Between-individual variance for the individual intercepts for the dispersion model, on the ln scale.
$\sigma^2_{\mathrm{fixed}_{\mathrm{m}}}$	Variance due to fixed effects for the mean model.
$\sigma^2_{\mathrm{fixed}_{\mathrm{v,exp}}}$	Variance due to fixed effects for the dispersion model. Estimated on the ln scale.
$\mathrm{var}(\mathrm{a} + \mathrm{b})$	Variance of the sum of random variables (vectors) a and b.
$\rho(\mathrm{a}, \mathrm{b})$	Correlation between two random variables a and b.
σ_{ab}	Covariance between two random variables a and b.

First individual difference: Personality

Animal behaviours are deemed 'personality traits' when, after measuring the same behaviour two or more times for multiple individuals, the differences among individuals are consistent across time and contexts (i.e. non-zero between-individual variance) (Bell, 2007; Sih et al., 2004). Individual differences can be quantified with a random intercept for each individual, using a multilevel model. Other sources of variation can be modelled as fixed effects (and, if necessary, additional random effects). Throughout this paper, we will present Gaussian multilevel models containing two fixed effects: the first for sex (i.e. a fixed effect with two categories, female and male), and the second for age (i.e. a continuous fixed effect). Age is mean-centred, so that the overall intercept of the model represents the average phenotype of females at the average age of the population. Notation for all equations are explained in Table 1 (note that the same principles can be applied to non-Gaussian data too; Nakagawa & Schielzeth, 2010). To measure differences in personalities, our basic model can be written as:

$$y_{ij} = \left(\beta_{\mathrm{m0}} + \mathrm{ID}_{\mathrm{m0}j}\right) + \beta_{\mathrm{m1}} x_{1j} + \beta_{\mathrm{m2}} x_{2ij} + e_{ij}, \qquad \text{eqn 1}$$

$$e_{ij} \sim (0, \sigma_e^2), \quad \text{eqn 2}$$

$$\mathrm{ID}_{\mathrm{m}0j} \sim \left(0, \sigma^2_{\mathrm{ID}_{\mathrm{m}0}}\right), \quad \text{eqn 3}$$

$$\sigma^2_{\mathrm{fixed}_{\mathrm{m}}} = \mathrm{var}\left(\beta_{\mathrm{m}1} x_{1j} + \beta_{\mathrm{m}2} x_{2ij}\right). \quad \text{eqn 4}$$

The model described by equations 1-3 assume homoscedasticity, meaning we model differences in individuals' average behaviour, but not variability in behaviour (Fig. 1A). The spread of individual averages allows us to estimate the between-individual variance in behaviour, which is used to quantify the strength of personality traits (with equations described in the Supplementary Information). When fixed effects represent biological variation (rather than experimental artefacts), it is recommended to add the fixed effect variance (calculated as in equation 4) back into the total variance (de Villemereuil et al., 2018) before calculating repeatability.

Second individual difference: Phenotypic plasticity

When we expect a predictable relationship between a phenotypic trait and an environmental or biological context (e.g. environmental temperature, or biological age), we can model this relationship with a function called a 'reaction norm' (Gavrilets & Scheiner, 1993; Gomulkiewicz & Kirkpatrick, 1992; Stearns & Koella, 1986). In the simplest case of a linear relationship (specified by an intercept and slope), the slope ($\beta_{\mathrm{m}2}$) describes the magnitude and direction of the population's average phenotypic plasticity. If the same individuals were measured multiple times across different contexts, we can use 'random regression' to estimate random slopes for each individual ($\beta_{\mathrm{m}2} + \mathrm{ID}_{\mathrm{m}2j}$). Individuals can vary in both intercepts (personality) and slopes (plasticity) (Fig. 1B). As a consequence, the magnitude of differences in average individual behaviour ($\sigma_{\mathrm{ID}_{\mathrm{m}0}}$) could depend upon the context at which the intercept is estimated (in this case, the value of $x_2 = 0$, which is usually set to be the 'average' environment). Whereas the model in equation 1 assumes that personality is fixed, this 'random slope' model allows for individuals to converge upon, or diverge from, the population mean in different environments:

$$y_{ij} = \left(\beta_{\mathrm{m}0} + \mathrm{ID}_{\mathrm{m}0j}\right) + \beta_{\mathrm{m}1} x_{1j} + \left(\beta_{\mathrm{m}2} + \mathrm{ID}_{\mathrm{m}2j}\right) x_{2ij} + e_{ij}, \quad \text{eqn 5}$$

$$e_{ij} \sim (0, \sigma_e^2), \quad \text{eqn 6}$$

$$\begin{bmatrix} \mathrm{ID}_{\mathrm{m}0j} \\ \mathrm{ID}_{\mathrm{m}2j} \end{bmatrix} \sim \mathrm{MVN}\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma^2_{\mathrm{ID}_{\mathrm{m}0}} & \rho\left(\mathrm{ID}_{\mathrm{m}0j}, \mathrm{ID}_{\mathrm{m}2j}\right)\sigma_{\mathrm{ID}_{\mathrm{m}0}}\sigma_{\mathrm{ID}_{\mathrm{m}2}} \\ \ldots & \sigma^2_{\mathrm{ID}_{\mathrm{m}2}} \end{bmatrix}\right). \quad \text{eqn 7}$$

Multiple individual differences are modelled together using the multivariate normal distribution (MVN), which estimates the covariance between the random intercepts and slopes across

individuals. This covariance is written (in the upper triangle of equation 7) as the product of the correlation between the intercepts and slopes [$\rho(\mathrm{ID}_{\mathrm{m0}j}, \mathrm{ID}_{\mathrm{m2}j})$], the standard deviation for the intercepts ($\sigma_{\mathrm{ID}_{\mathrm{m0}}}$), and standard deviation for the slopes ($\sigma_{\mathrm{ID}_{\mathrm{m2}}}$).

Personality-plasticity associations: Correlation between personality types and plasticity

Theoretically, individuals with different personalities might differ in their ability to accurately assess their environment or change their phenotype, and there are empirical observations of such 'personality-plasticity' associations (Bogacz et al., 2010; Sih & Del Giudice, 2012). For example, in a marine gastropod boldness was negatively correlated with plasticity in response to tidal and temperature changes (Cornwell et al., 2019), in sticklebacks, exploration was positively correlated with acclimation to a novel environment (Dingemanse et al., 2012), and in house sparrows, the level of parental care was shown to be correlated with plasticity in response to brood size, nestling age, precipitation, and the provisioning effort of the breeding partner (Westneat et al., 2011). Theoretically, Dubois (2019) predicted a negative correlation between proactive personalities and plasticity, based on the assumption that proactive individuals are less capable of accurately assessing their environment, due to higher cognitive demands of proactivity. A positive correlation, meanwhile, could represent a "rich get richer" scenario, whereby individuals in better condition are more proactive *and* better able to bear the costs associated with plasticity (DeWitt et al., 1998; Reznick et al., 2000). Alternatively, phenotypic plasticity can represent a maladaptive change in the phenotype (e.g. due to environmental stress), and therefore personality types that show reduced plasticity might be more resilient to environmental change (Ghalambor et al., 2007).

There are two possible types of personality-plasticity associations. First, from the multivariate normal distribution in equation 7, we can ask whether individual differences in intercepts are correlated with individual differences in slopes. The correlation provided by the model is the rank correlation between individual deviations (i.e. the best linear unbiased predictions: BLUPs) from the average population intercept (β_{m0}) and the average population slope (β_{m2}). This correlation represents the covariance between the random intercepts and slopes ($\sigma_{\mathrm{ID}_{\mathrm{m0}}\mathrm{ID}_{\mathrm{m2}}}$), divided by the product of their standard deviations:

$$\rho\left(\mathrm{ID}_{\mathrm{m0}j}, \mathrm{ID}_{\mathrm{m2}j}\right) = \frac{\sigma_{\mathrm{ID}_{\mathrm{m0}}\mathrm{ID}_{\mathrm{m2}}}}{\sigma_{\mathrm{ID}_{\mathrm{m0}}}\sigma_{\mathrm{ID}_{\mathrm{m2}}}}. \qquad \text{eqn 8}$$

To understand this rank correlation, we need to consider the direction of plasticity — increasing phenotypes, decreasing phenotypes, or both — by plotting individual's reaction norms. The

importance of considering the direction of phenotypic change is shown in Fig. 2 (and was recognised by Stamps & Biro, 2016), because different scenarios can produce the same correlations between intercepts and slope deviations. When individual reaction norms 'fan out' (or in), personalities that are above (or below) the population average have a more positive slope, and individuals that are below (or above) the population average have a more negative slope. A conceptual model of 'fanning' is presented by Sih et al. (2015) as a result from within-individual feedback loops. Fanning can also occur when adaptive plasticity is condition-dependent, and only high-quality individuals can express adaptive plasticity. Individuals in poor condition might express maladaptive plasticity in the opposite direction to the adaptive response. Regardless of the cause of these patterns, in a full fan scenario, the ranking of individual intercepts does not correlate with their magnitude of phenotypic plasticity. Contrasting with a full fan pattern, often we might expect all individuals in a population to respond to an environmental change with a plastic response in the *same* direction. In Fig. 2, we call these scenarios 'positive fans' (when all phenotypes increase or stay the same) and 'negative fans' (when all phenotypes decrease or stay the same). For example, in a warmer environment, members of an ectothermic population might all show increased activity levels, and reduced developmental times.

The direction of plastic responses is central to many research questions, but there will often be scenarios where we care more about the magnitude of plasticity, irrespective of the direction (e.g. in comparative analyses). In these cases, we propose a second type of personality-plasticity association, which is calculated from the magnitude of each individual's reaction norm (i.e. the sum of the population slope, and individuals' BLUP deviation; $|\beta_{\mathrm{m2}} + \mathrm{ID}_{\mathrm{m2}j}|$). This personality-plasticity association,

$$\rho\left(\mathrm{ID}_{\mathrm{m0}j}, |\beta_{\mathrm{m2}} + \mathrm{ID}_{\mathrm{m2}j}|\right) = \frac{\sigma_{\mathrm{ID_{m0}}|\beta_{\mathrm{m2}}+\mathrm{ID_{m2}}|}}{\sigma_{\mathrm{ID_{m0}}}\sigma_{|\beta_{\mathrm{m2}}+\mathrm{ID_{m2}}|}}, \qquad \text{eqn 9}$$

can be calculated from the posterior distributions of individual differences, and the population slope. Here (as with all calculations involving BLUPs), we should use posterior distributions of estimated parameters (Hadfield et al., 2010; Postma, 2006) (which can be obtained using Bayesian methods; the worked example, below, includes code for the calculations). Posterior distributions retain uncertainty from each estimate, and carry those uncertainties forward in each calculation. This method therefore retains uncertainty in $\rho(\mathrm{ID}_{\mathrm{m0}j}, |\beta_{\mathrm{m2}} + \mathrm{ID}_{\mathrm{m2}j}|)$, which we would lose if the correlation was obtained from point estimates alone, making it easy to present credible intervals. While bootstrapping methods could be used to estimate uncertainty from frequentist (likelihood-based) models (cf. Stoffel et al., 2017) these methods would become very difficult when predictability

is incorporated into the model structure (using double hierarchical models, discussed below).

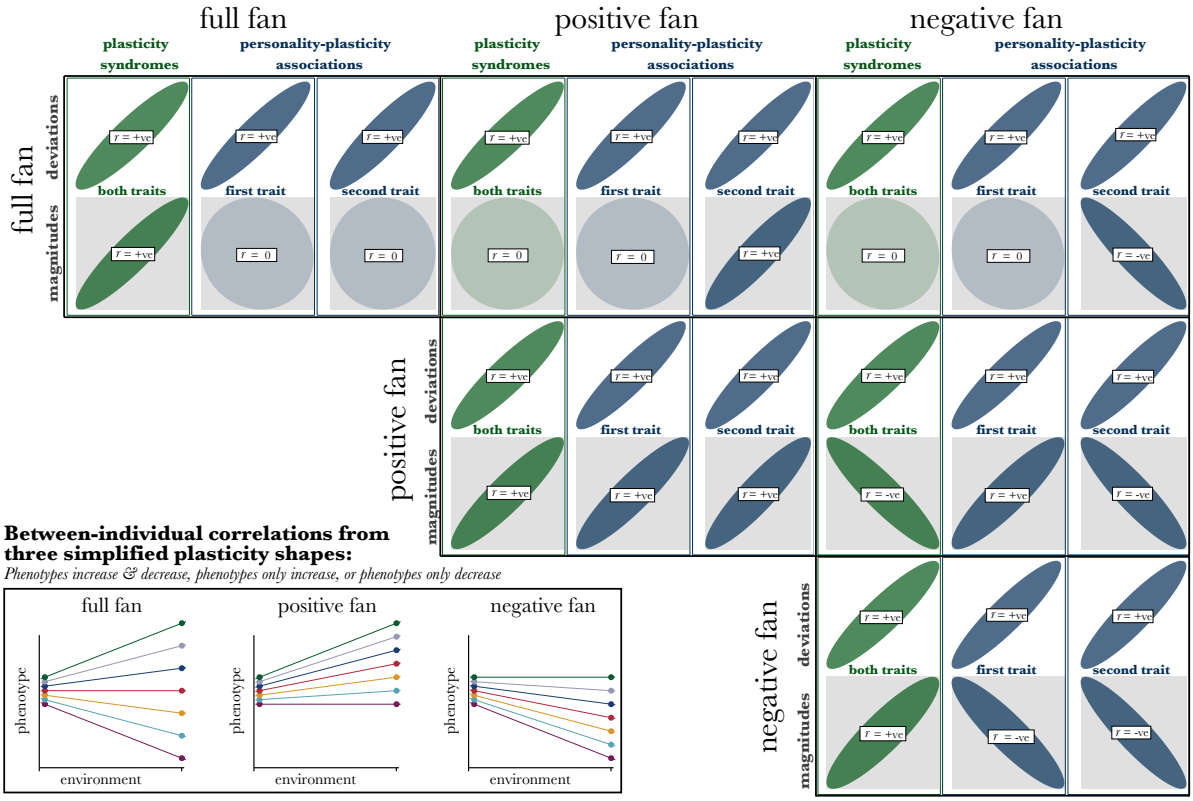

Figure 2

The influence of three 'shapes' of population plasticity on the direction of plasticity syndromes and personality-plasticity associations, measured with either BLUPs (deviations from the average slope) or the absolute value of individual slopes (magnitudes). The **bottom-left box** illustrates three shapes of population plasticity, for a hypothetical population measured in two environments. Each coloured point represents a different individual measured in one environment. The coloured lines that connect two points depict a reaction norm, with the direction and magnitude of phenotypic plasticity shown by the direction and steepness of each slope. The three plasticity shapes are: (1) **Full fan**: individuals vary in both the magnitude and direction of their slopes, such that some phenotypes increase in the changed environment, while others decrease; (2) **Positive fan**: individual slopes have a lower-bound at zero, such that phenotypes always increase or stay the same in the changed environment; (3) **Negative fan**: individual slopes have an upper-bound at zero, such that phenotypes always decrease or stay the same in the changed environment. The **grid boxes** are arranged to depict each possible combination of plasticity shapes and their resulting between-individual correlations. In each box, correlations involving slope **deviations** (BLUPs) are shown in the upper row, whereas correlations involving slope **magnitudes** (absolute slopes) are

shown in the lower row, with grey shading. **Ellipses** depict positive and negative correlations, whereas **circles** depict null correlations. **Plasticity syndromes** (shown in green) are the correlations between slopes (deviations, equation 15, or magnitudes, equation 16) from reaction norms from two different traits. **Personality-plasticity associations** (shown in blue) are the correlations between intercepts and slopes (deviations, equation 8, or magnitudes, equation 9) from reaction norms measured for one trait.

Behavioural and plasticity syndromes

When two different traits are measured repeatedly for the same individuals, we can use a bivariate model to estimate the covariances (and therefore correlations) between individual differences in intercepts and slopes for these traits (shown in equation 13, below). Between-individual correlations that span across distinct traits might reflect biologically interesting dependence, such as genetic correlations (e.g. due to linkage disequilibrium) or developmental constraints (Sih et al., 2012). Such phenotypic integration can prevent phenotypic traits from evolving independently (Fawcett et al., 2012; Pigliucci, 2003). Trait correlations could also reflect correlated selective pressures, where a change in one trait encourages an adaptive change in the other. In theory, multivariate models can estimate the dependence between many traits at once, but here, for ease of presentation, we focus on the simplest scenario of two traits ('t1' and 't2'). The bivariate model can be written as:

$$y_{ij}^{\mathrm{t1}} = \left(\beta_{\mathrm{m0}}^{\mathrm{t1}} + \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t1}}\right) + \beta_{\mathrm{m1}}^{\mathrm{t1}} x_{1j}^{\mathrm{t1}} + \left(\beta_{\mathrm{m2}}^{\mathrm{t1}} + \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}}\right) x_{2ij}^{\mathrm{t1}} + e_{ij}^{\mathrm{t1}}, \qquad \text{eqn 10}$$

$$y_{ij}^{\mathrm{t2}} = \left(\beta_{\mathrm{m0}}^{\mathrm{t2}} + \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}}\right) + \beta_{\mathrm{m1}}^{\mathrm{t2}} x_{1j}^{\mathrm{t2}} + \left(\beta_{\mathrm{m2}}^{\mathrm{t2}} + \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}}\right) x_{2ij}^{\mathrm{t2}} + e_{ij}^{\mathrm{t2}}, \qquad \text{eqn 11}$$

$$\begin{bmatrix} e^{\mathrm{t1}} \\ e^{\mathrm{t2}} \end{bmatrix} \sim \mathrm{MVN}\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_{e^{\mathrm{t1}}}^2 & \rho(e^{\mathrm{t1}}, e^{\mathrm{t2}})\sigma_{e^{\mathrm{t1}}}\sigma_{e^{\mathrm{t2}}} \\ \cdots & \sigma_{e^{\mathrm{t2}}}^2 \end{bmatrix}\right), \qquad \text{eqn 12}$$

$$\begin{bmatrix} \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t1}} \\ \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}} \\ \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}} \\ \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}} \end{bmatrix} \sim \mathrm{MVN}\left(\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t1}}}^2 & \rho(\mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}})\sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t1}}}\sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t1}}} & \rho(\mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}})\sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t1}}}\sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t2}}} & \rho(\mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}})\sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t1}}}\sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t2}}} \\ \cdots & \sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t1}}}^2 & \rho(\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}})\sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t1}}}\sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t2}}} & \rho(\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}})\sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t1}}}\sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t2}}} \\ \cdots & \cdots & \sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t2}}}^2 & \rho(\mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}}, \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}})\sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t2}}}\sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t2}}} \\ \cdots & \cdots & \cdots & \sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t2}}}^2 \end{bmatrix}\right). \qquad \text{eqn 13}$$

Dependence between residual errors for different traits is modelled using the multivariate normal distribution (MVN) in equation 12. Similarly, in equation 13, the covariance matrix to describe the relationship between individual differences has been expanded to include correlations both within and between traits.

Behavioural syndrome: Correlation across trait intercepts

Bivariate models can quantify the relationship between different types of personality traits (equations 10-11). When individual intercepts in behavioural traits are correlated (i.e. between-individual correlation between personality traits), those traits are said to exhibit a 'behavioural

syndrome' (Dingemanse et al., 2010a), which we can estimate as:

$$\rho\left(\mathrm{ID}^{\mathrm{t1}}_{\mathrm{m0}j}, \mathrm{ID}^{\mathrm{t2}}_{\mathrm{m0}j}\right) = \frac{\sigma_{\mathrm{ID}^{\mathrm{t1}}_{\mathrm{m0}}\mathrm{ID}^{\mathrm{t2}}_{\mathrm{m0}}}}{\sigma_{\mathrm{ID}^{\mathrm{t1}}_{\mathrm{m0}}}\sigma_{\mathrm{ID}^{\mathrm{t2}}_{\mathrm{m0}}}}. \qquad \text{eqn 14}$$

While many empirical papers purport to have found these syndromes, far fewer have done so following the recommended method of decomposing total phenotypic variance into its among- and within- individual components (Dingemanse & Dochtermann, 2013; Moirón et al., 2020; Niemelä & Dingemanse, 2018). Combining both levels of the phenotypic correlation can be misleading, as selection should occur at the between-individual level, and the strength and direction of this correlation can be different from the within-individual level (i.e. violating the 'individual gambit'; Brommer, 2013).

Plasticity syndrome: Correlation across trait slopes

Correlations between the reaction norm slopes for the same individuals can be measured for multiple traits, or multiple environmental manipulations. Positive correlations might be common, due to shared mechanisms in the maintenance of plasticity. Plasticity that shows phenotypic integration, or modularity, has been of enduring interest to plant scientists (Gianoli & Palacio-Lopez, 2009; Mallitt et al., 2010; Pigliucci, 2002; Schlichting, 1989). Alternatively, a negative correlation in the magnitude of plasticity could reflect trade-offs due to associated costs (DeWitt et al., 1998), while the absence of a correlation suggests the traits have been selected to be decoupled, or face independent selective pressures.

'Plasticity syndromes' are more challenging to interpret than behavioural syndromes, due to the rank order of individual differences in slopes not necessarily being correlated with the magnitude of individuals' plasticity. As with personality-plasticity associations, plasticity syndromes can be estimated in two different ways (which are compared in Fig. 2, above). Taken directly from the model, the correlation between individual slope deviations,

$$\rho\left(\mathrm{ID}^{\mathrm{t1}}_{\mathrm{m2}j}, \mathrm{ID}^{\mathrm{t2}}_{\mathrm{m2}j}\right) = \frac{\sigma_{\mathrm{ID}^{\mathrm{t1}}_{\mathrm{m2}}\mathrm{ID}^{\mathrm{t2}}_{\mathrm{m2}}}}{\sigma_{\mathrm{ID}^{\mathrm{t1}}_{\mathrm{m2}}}\sigma_{\mathrm{ID}^{\mathrm{t2}}_{\mathrm{m2}}}}, \qquad \text{eqn 15}$$

describes whether the 'rank order' of slopes is maintained between the two traits. That is, a rank correlation from equation 15 indicates that individuals whose slopes are more positive than average in trait 1 tend to *also* be more positive than average in trait 2. This rank correlation could be useful for certain patterns of plasticity (see Fig. 2). However, when we care about the total slope (rather than its deviation), we should consider the slope's magnitude rather than direction. In this case, a 'plasticity syndrome' (equation 16) is calculated as the correlation between the absolute magnitude of individuals' reaction norms, such that:

$$\rho\left(\left|\beta_{\mathrm{m2}}^{\mathrm{t1}}+\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}}\right|,\left|\beta_{\mathrm{m2}}^{\mathrm{t2}}+\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}}\right|\right)=\frac{\sigma_{\left|\beta_{\mathrm{m2}}^{\mathrm{t1}}+\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}}\right|\left|\beta_{\mathrm{m2}}^{\mathrm{t2}}+\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}}\right|}}{\sigma_{\left|\beta_{\mathrm{m2}}^{\mathrm{t1}}+\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}}\right|}\sigma_{\left|\beta_{\mathrm{m2}}^{\mathrm{t2}}+\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}}\right|}}. \quad \text{Eqn 16}$$

As with equation 9, this correlation of magnitudes can be calculated from the posterior distributions of model estimates (with code provided in materials accompanying the worked example)

Summary of personality and plasticity

Individuals can have different personalities, as measured by differences in average behaviour (intercepts), and differences in plasticity, as measured by differences in their average change in behaviour across a covariate (slopes). The covariate can be an external variable such as temperature, or an internal variable such as developmental history. Between-individual variation in plasticity has implications for estimates of personality (and related correlations), because the magnitude of between-individual variation can depend upon the point at which the 'intercept' in behaviour is measured. From individual differences in two BLUPs (personality and plasticity) we can measure three types of biologically relevant correlations: first, personality-plasticity associations are a correlation between reaction norm intercepts and slopes (either deviations or magnitudes); second, behavioural syndromes are a correlation between individual intercepts for more than one trait; third, plasticity syndromes are a correlation between the deviations or magnitudes of individual slopes for more than one trait, or the same trait measured across more than one covariate. When interpreting rank correlations involving slopes, which have both a direction and magnitude, researchers should plot each individual's reaction norm to consider the 'shape' of phenotypic plasticity. In circumstances where we care more about the magnitude of plasticity than its direction, researchers can perform additional calculations to capture the absolute value of individual slopes, rather than individuals' deviations from the average slope. Performing vector calculations on posterior distributions (from a Bayesian model) ensures that uncertainty in model estimates is carried forward.

Third individual difference: Predictability

The impact that animals have on their surroundings depends not only on their average behaviour, but also on how their behaviour fluctuates through time. Individual differences can be consistent yet small, and these might not have a material impact on fitness (and therefore might not respond to selection). Despite the variability of individuals' behaviour being biologically important, it is currently rare for animal behaviour studies to distinguish between individuals who are very consistent through time, and those whose behaviour fluctuates enormously (an early example is seen in Westneat et al., 2013). Individual differences in within-individual variance (i.e. heteroscedasticity)

can be modelled with a Double Hierarchical Generalized Linear Model (DHGLM; Cleasby et al., 2015). The 'double' in DHGLM refers to a random effect being included in both the mean model, and the residual variance model. In the social and medical sciences, DHGLMs are also known as location-scale regression models (with 'location' indicating the mean, and 'scale' indicating the variance; e.g. Lin et al., 2018; Rast et al., 2012). The residual variance model, hereafter referred to as the 'dispersion' model, is usually estimated on the natural logarithm scale. Fitting a random intercept for individual identity at both levels of the model allows individuals to vary in both their average behaviour, and residual variance (equations 17-18, below). We can therefore consider a third individual difference alongside personality and plasticity: predictability.

Modelling personality, plasticity, and predictability simultaneously

For labile traits, by allowing individuals to vary in both personality (Fig. 1A) and predictability (Fig. 1C), we effectively estimate a different distribution for each individual. Extending the univariate model shown in equations 5-8, we can write the double hierarchical model as:

$$y_{ij} = \left(\beta_{\mathrm{m0}} + \mathrm{ID}_{\mathrm{m0}j}\right) + \beta_{\mathrm{m1}}x_{1j} + \left(\beta_{\mathrm{m2}} + \mathrm{ID}_{\mathrm{m2}j}\right)x_{2ij} + e_{ij}, \quad \text{eqn 17}$$

$$\ln\left(\sigma^2_{e_{ij}}\right) = \left(\beta_{\mathrm{v0,exp}} + \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}\right) + \beta_{\mathrm{v1,exp}}x_{1j} + \beta_{\mathrm{v2,exp}}x_{2ij}, \quad \text{eqn 18}$$

$$e_{ij} \sim N(0, \sigma^2_{e_{ij}}) \quad \text{eqn 19}$$

$$\begin{bmatrix} \mathrm{ID}_{\mathrm{m0}j,} \\ \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}} \\ \mathrm{ID}_{\mathrm{m2}j} \end{bmatrix} \sim MVN\left(\begin{bmatrix}0\\0\\0\end{bmatrix}, \begin{bmatrix} \sigma^2_{\mathrm{ID_{m0}}} & \rho\left(\mathrm{ID}_{\mathrm{m0}j}, \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}\right)\sigma_{\mathrm{ID_{m0}}}\sigma_{\mathrm{ID_{v0,exp}}} & \rho\left(\mathrm{ID}_{\mathrm{m0}j}, \mathrm{ID}_{\mathrm{m2}j}\right)\sigma_{\mathrm{ID_{m0}}}\sigma_{\mathrm{ID_{m2}}} \\ \dots & \sigma^2_{\mathrm{ID_{v0,exp}}} & \rho\left(\mathrm{ID}_{\mathrm{v0}j}, \mathrm{ID}_{\mathrm{m2}j}\right)\sigma_{\mathrm{ID_{v0},exp}}\sigma_{\mathrm{ID_{m2}}} \\ \dots & \dots & \sigma^2_{\mathrm{ID_{m2}}} \end{bmatrix}\right). \quad \text{eqn 20}$$

Estimating individual variances requires many repeated measurements at the individual level, which is relatively uncommon in animal personality studies (sample size recommendations depend on the number of individuals and the magnitude of heteroscedasticity, which is explored in Cleasby et al., 2015). Note that equations 17-20 vary from equations 19-24 in Cleasby et al. (2015), as the dispersion model is based on residual variances, rather than residual standard deviations (which has some benefits for summarising the magnitude of individual differences, which we elaborate on in the Supplementary Information).

Personality-predictability association: Correlation between personality types and individual consistency

From the correlation between individual intercepts in both the mean and dispersion models, we can estimate whether some personality types are more prone to being unpredictable than others. From

the multivariate distribution in equation 20, we have:

$$\rho\left(\mathrm{ID}_{\mathrm{m0}j}, \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}\right) = \frac{\sigma_{\mathrm{ID}_{\mathrm{m0}}\mathrm{ID}_{\mathrm{v0,exp}}}}{\sigma_{\mathrm{ID}_{\mathrm{m0}}}\sigma_{\mathrm{ID}_{\mathrm{v0,exp}}}}. \quad \text{eqn 21}$$

Interpreting equation 21 is somewhat unintuitive; remember that an individual having more residual variance is likely to be less predictable (i.e. because they have more within-individual variance). Therefore, a positive correlation between mean and dispersion intercepts represents a negative correlation between personality and predictability. When presenting results, we therefore recommend multiplying correlations involving dispersion intercepts by minus 1, to make their interpretation intuitive (e.g. a positive correlation signifies a bolder individual is more predictable, with a smaller residual variance), such that:

$$\rho\left(\mathrm{ID}_{\mathrm{m0}j}, -1 \times \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}\right) = -\frac{\sigma_{\mathrm{ID}_{\mathrm{m0}}\mathrm{ID}_{\mathrm{v0,exp}}}}{\sigma_{\mathrm{ID}_{\mathrm{m0}}}\sigma_{\mathrm{ID}_{\mathrm{v0,exp}}}}. \quad \text{eqn 22}$$

The worked example below presents this sign-reversed correlation for personality-predictability associations. Although little theory exits on the personality-predictability association, we might expect risker personality types to also be less predictable; being more variable can be a risky strategy. Alternatively, riskier individuals could be closer to a hypothetical 'ceiling', whereby a fluctuation beyond that point would be fatal to the individual. Riskier individuals might therefore show greater precision around their mean phenotype, to avoid crossing some point of no return (a similar idea around stability of more 'extreme' personalities is discussed in Stamps & Groothuis, 2010).

Plasticity-predictability association: Correlation between plasticity and individual consistency

Broadly, plasticity is the expression of different phenotypes by the same genotype in a different environment (Stamps, 2015). The environment will always be slightly different each time an individual expresses a labile trait because of variation in endogenous variables (internal and developmental), and uncontrolled fluctuations in the external environment (Flatt, 2005; Hansen et al., 2006). Therefore, predictability is a special type of 'unpredictable' plasticity, because there are stochastic changes in internal and external environments that prevent us from knowing exactly which phenotype will be expressed at any point in time. From the slope in the mean model and the intercept in the dispersion model, we can estimate whether individual differences in predictable and unpredictable plasticity are correlated. There is theoretical interest in whether different types of plasticity (or 'flexibility' or 'responsiveness') are related to each other (e.g. through shared mechanisms), but to date this type of question has received little empirical attention (Stamps & Biro,

2016). Less predictable individuals express a larger range of behaviours, which could imply a greater scope for showing an average plastic response to an environmental change. Predictability and plasticity could therefore be positively correlated. The rank correlation between individual deviations from mean slopes, and dispersion intercepts,

$$\rho\left(\mathrm{ID}_{\mathrm{m2}j}, \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}\right) = \frac{\sigma_{\mathrm{ID}_{\mathrm{m2}}\mathrm{ID}_{\mathrm{v0,exp}}}}{\sigma_{\mathrm{ID}_{\mathrm{m2}}}\sigma_{\mathrm{ID}_{\mathrm{v0,exp}}}}, \quad \text{eqn 23}$$

measures whether individuals that are further away from the average level of plasticity are more or less predictable than average. The correlation between the magnitudes of mean slopes and dispersion intercepts,

$$\rho\left(\left|\beta_{\mathrm{m2}} + \mathrm{ID}_{\mathrm{m2}j}\right|, -1 \times \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}\right) = -\frac{\sigma_{|\beta_{\mathrm{m2}}+\mathrm{ID}_{\mathrm{m2}}|\mathrm{ID}_{\mathrm{v0,exp}}}}{\sigma_{|\beta_{\mathrm{m2}}+\mathrm{ID}_{\mathrm{m2}}|}\sigma_{\mathrm{ID}_{\mathrm{v0,exp}}}}, \quad \text{eqn 24}$$

estimates whether individuals who are more plastic (in either direction) are more or less predictable. Multiplying by minus 1 makes this correlation interpretable is a 'plasticity-predictability association'.

Multivariate DHGLM: personality, plasticity, and predictability

Up to this point, we have discussed fives types of correlations: behavioural syndromes (Fig. 3A); plasticity syndromes (Fig. 3B); personality-plasticity associations (Fig. 3D); personality-predictability associations (Fig. 3E); and plasticity-predictability associations (Fig. 3F). Given sufficient data, one bivariate with three individual differences can estimate all of these correlations, plus a sixth: predictability syndromes (Fig. 3C), which we describe below. The bivariate model can be written as:

$$y_{ij}^{t1} = \left(\beta_{m0}^{t1} + ID_{m0j}^{t1}\right) + \beta_{m1}^{t1} x_{1j}^{t1} + \left(\beta_{m2}^{t1} + ID_{m2j}^{t1}\right) x_{2ij}^{t1} + e_{ij}^{t1}, \quad \text{eqn 25}$$

$$y_{ij}^{\mathrm{t2}} = \left(\beta_{\mathrm{m0}}^{\mathrm{t2}} + \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}}\right) + \beta_{\mathrm{m1}}^{\mathrm{t2}} x_{1j}^{\mathrm{t2}} + \left(\beta_{\mathrm{m2}}^{\mathrm{t2}} x_{2ij}^{\mathrm{t2}} + \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}}\right) + e_{ij}^{\mathrm{t2}}, \quad \text{eqn 26}$$

$$\ln\left(\sigma_{e_{ij}^{\mathrm{t1}}}^2\right) = \left(\beta_{\mathrm{v0}}^{\mathrm{t1}} + \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}^{\mathrm{t1}}\right) + \beta_{\mathrm{v1}}^{\mathrm{t1}} x_{1j}^{\mathrm{t1}} + \beta_{\mathrm{v2}}^{\mathrm{t1}} x_{2ij}^{\mathrm{t1}}, \quad \text{eqn 27}$$

$$\ln\left(\sigma_{e_{ij}^{\mathrm{t2}}}^2\right) = \left(\beta_{\mathrm{v0}}^{\mathrm{t2}} + \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}^{\mathrm{t2}}\right) + \beta_{\mathrm{v1}}^{\mathrm{t2}} x_{1j}^{\mathrm{t2}} + thes\beta_{\mathrm{v2}}^{\mathrm{t2}} x_{2ij}^{\mathrm{t2}}, \quad \text{eqn 28}$$

$$\begin{bmatrix} e_{ij}^{\mathrm{t1}} \\ e_{ij}^{\mathrm{t2}} \end{bmatrix} \sim \mathrm{MVN}\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_{e_{ij}^{\mathrm{t1}}}^2 & \rho\left(e_{ij}^{\mathrm{t1}}, e_{ij}^{\mathrm{t2}}\right)\sigma_{e_{ij}^{\mathrm{t1}}}\sigma_{e_{ij}^{\mathrm{t2}}} \\ \ldots & \sigma_{e_{ij}^{\mathrm{t2}}}^2 \end{bmatrix}\right), \quad \text{eqn 29}$$

$$\begin{bmatrix} \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t1}} \\ \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}^{\mathrm{t1}} \\ \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}} \\ \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}} \\ \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}^{\mathrm{t2}} \\ \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}} \end{bmatrix} \sim \mathrm{MVN}\left(\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t1}}}^2 & \rho\left(\mathbf{ID}_{\mathbf{m0}j}^{\mathbf{t1}}, \mathbf{ID}_{\mathbf{v0}j,\mathbf{exp}}^{\mathbf{t1}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m0}}}^{\mathbf{t1}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{v0,exp}}}^{\mathbf{t1}} & \rho\left(\mathbf{ID}_{\mathbf{m0}j}^{\mathbf{t1}}, \mathbf{ID}_{\mathbf{m2}j}^{\mathbf{t1}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m0}}}^{\mathbf{t1}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m2}}}^{\mathbf{t1}} & \rho\left(\mathbf{ID}_{\mathbf{m0}j}^{\mathbf{t1}}, \mathbf{ID}_{\mathbf{v0}j,\mathbf{exp}}^{\mathbf{t2}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m0}}}^{\mathbf{t1}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m0}}}^{\mathbf{2}} & \rho\left(\mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}^{\mathrm{t2}}\right)\sigma_{\mathrm{ID}_{\mathrm{m0}}}^{\mathrm{t1}}\sigma_{\mathrm{ID}_{\mathrm{v0,exp}}}^{2} & \rho\left(\mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}}\right)\sigma_{\mathrm{ID}_{\mathrm{m0}}}^{\mathrm{t1}}\sigma_{\mathrm{ID}_{\mathrm{m2}}}^{2} \\ \ldots & \sigma_{\mathrm{ID}_{\mathrm{v0,exp}}^{\mathrm{t1}}}^2 & \rho\left(\mathbf{ID}_{\mathbf{v0}j,\mathbf{exp}}^{\mathbf{t1}}, \mathbf{ID}_{\mathbf{m2}j}^{\mathbf{t1}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{v0,exp}}}^{\mathbf{t1}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m2}}}^{\mathbf{t1}} & \rho\left(\mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}}\right)\sigma_{\mathrm{ID}_{\mathrm{v0,exp}}}^{\mathrm{t1}}\sigma_{\mathrm{ID}_{\mathrm{m0}}}^{2} & \rho\left(\mathbf{ID}_{\mathbf{v0}j,\mathbf{exp}}^{\mathbf{t1}}, \mathbf{ID}_{\mathbf{v0}j,\mathbf{exp}}^{\mathbf{t2}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{v0,exp}}}^{\mathbf{t1}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{v0,exp}}}^{\mathbf{2}} & \rho\left(\mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t2}}\right)\sigma_{\mathrm{ID}_{\mathrm{v0,exp}}}^{\mathrm{t1}}\sigma_{\mathrm{ID}_{\mathrm{m2}}}^{2} \\ \ldots & \ldots & \sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t1}}}^2 & \rho\left(\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{m0}j}^{\mathrm{t2}}\right)\sigma_{\mathrm{ID}_{\mathrm{m2}}}^{\mathrm{t1}}\sigma_{\mathrm{ID}_{\mathrm{m0}}}^{2} & \rho\left(\mathrm{ID}_{\mathrm{m2}j}^{\mathrm{t1}}, \mathrm{ID}_{\mathrm{v0}j,\mathrm{exp}}^{\mathrm{t2}}\right)\sigma_{\mathrm{ID}_{\mathrm{m2}}}^{\mathrm{t1}}\sigma_{\mathrm{ID}_{\mathrm{v0,exp}}}^{2} & \boldsymbol{\rho}\left(\mathbf{ID}_{\mathbf{m2}j}^{\mathbf{t1}}, \mathbf{ID}_{\mathbf{m2}j}^{\mathbf{t2}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m2}}}^{\mathbf{t1}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m2}}}^{\mathbf{2}} \\ \ldots & \ldots & \ldots & \sigma_{\mathrm{ID}_{\mathrm{m0}}^{\mathrm{t2}}}^2 & \boldsymbol{\rho}\left(\mathbf{ID}_{\mathbf{m0}j}^{\mathbf{t2}}, \mathbf{ID}_{\mathbf{v0}j,\mathbf{exp}}^{\mathbf{t2}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m0}}}^{\mathbf{2}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{v0,exp}}}^{\mathbf{2}} & \boldsymbol{\rho}\left(\mathbf{ID}_{\mathbf{m2}j}^{\mathbf{t2}}, \mathbf{ID}_{\mathbf{v0}j,\mathbf{exp}}^{\mathbf{t2}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m2}}}^{\mathbf{2}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{v0,exp}}}^{\mathbf{2}} \\ \ldots & \ldots & \ldots & \ldots & \sigma_{\mathrm{ID}_{\mathrm{v0,exp}}^{\mathrm{t2}}}^2 & \boldsymbol{\rho}\left(\mathbf{ID}_{\mathbf{m2}j}^{\mathbf{t2}}, \mathbf{ID}_{\mathbf{v0}j,\mathbf{exp}}^{\mathbf{t2}}\right)\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{m2}}}^{\mathbf{2}}\boldsymbol{\sigma}_{\mathbf{ID}_{\mathbf{v0,exp}}}^{\mathbf{2}} \\ \ldots & \ldots & \ldots & \ldots & \ldots & \sigma_{\mathrm{ID}_{\mathrm{m2}}^{\mathrm{t2}}}^2 \end{bmatrix}\right). \quad \text{eqn 30}$$

The variance-covariance matrix in equation 30 emphasises, in bold, the off-diagonal elements that comprise the six types of correlations we are interested in (shown in Fig. 3).

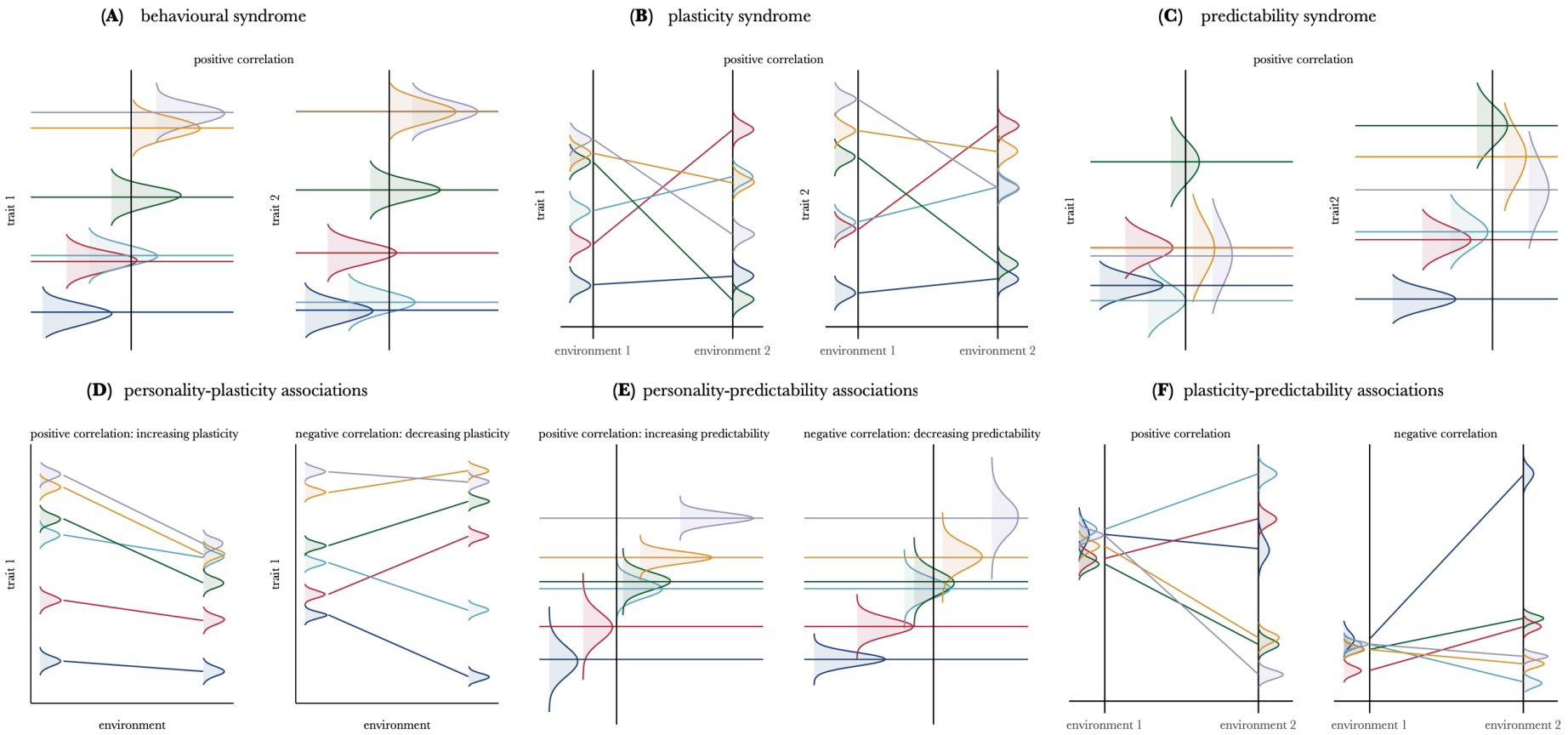

Figure 3: *Legend on following page*

Figure 3

Conceptual illustration of six types of correlations, from three types of individual differences (personality, individual differences in plasticity, and individual differences in predictability). Each coloured line and distribution represents a different individual from the same population. The top row shows positive between-trait correlations ('syndromes'), where individual differences are correlated with each other for multiple traits. The bottom row shows within-trait correlations between pairs of individual differences. (**A**) Behavioural syndrome: individual differences in average behaviour (measured by random intercepts) are positively correlated between two traits, meaning that the 'rank order' of intercepts is maintained (equation 14). (**B**) Plasticity syndrome: the magnitudes of random slopes are positively correlated (equation 16). (**C**) Predictability syndrome: individuals that are less predictable in one trait (shown by a wider distribution) are less predictable in the second trait (equation 31). (**D**) Personality-plasticity association: individuals with a higher ranking in average behaviour (intercept) have larger absolute slopes (equation 9). (**E**) Personality-predictability association: individuals' intercepts are correlated with the (reversed) magnitude of within-individual variance (equation 22).
(**F**) Plasticity-predictability syndrome: the magnitude of individual slopes correlates with the ranking of (reversed) individual variances (equation 24).

Predictability syndrome: Correlation across trait predictabilities

Given sufficient data for two phenotypic traits, we can measure whether individuals who are less (or more) predictable in one trait are also less (or more) predictable in another trait (i.e., a positive, or negative, correlation between within-individual variances), such that:

$$\rho\left(\mathrm{ID}^{\mathrm{t1}}_{\mathrm{v0}j,\mathrm{exp}}, \mathrm{ID}^{\mathrm{t2}}_{\mathrm{v0}j,\mathrm{exp}}\right) = \frac{\sigma_{\mathrm{ID}^{\mathrm{t1}}_{\mathrm{v0,exp}}\mathrm{ID}^{\mathrm{t2}}_{\mathrm{v0,exp}}}}{\sigma_{\mathrm{ID}^{\mathrm{t1}}_{\mathrm{v0,exp}}}\sigma_{\mathrm{ID}^{\mathrm{t2}}_{\mathrm{v0,exp}}}}. \qquad \text{eqn 31}$$

As with behavioural syndromes, the presence of a 'predictability syndrome' implies some modularity, or phenotypic integration (which can represent correlated selective pressures, or genetic correlations; Pigliucci, 2003). The absence of a predictability syndrome implies that different types of traits might be selected to have different levels of predictability.

Summary of incorporating predictability into multilevel models

With two individual differences — a random intercept and slope in the mean model — we can look at three correlations: two types of syndromes (between traits; Fig. 3A and Fig. 3B) and one intercept-slope association (within trait; Fig. 3D). Modelling predictability adds a third individual difference — a random intercept in the dispersion model. Using a bivariate (multivariate) model, we can

simultaneously model these three individual differences in two (or more) types of traits (equations 25-30), and estimate three additional correlations: (1) a predictability syndrome (between traits; Fig. 3C); (2) an association (within traits) between personality and predictability (Fig. 3E); and (3) an association between plasticity and predictability (Fig. 3F). Given that most of the variation in behaviour is contained within residual variation — the lowest level of the phenotypic hierarchy — we cannot meaningfully explain important biological variation without considering the variability of individuals.

Summary statistics for individual differences in personality and predictability

The Supplementary Information includes equations for quantifying the magnitude of individual differences in personality and predictability with two different summary statistics: repeatability (Rp), which is variance-standardised, and the coefficient of individual variation (CV_{ID}), which is mean-standardised. Both statistics are used to summarise results in the worked example, below. When standardising variance estimates is important to consider the scale of measurement, mean-variance relationships, and any transformations that were performed prior to analysis (formulas for common back-transformations are also provided in the supplement). An accessible summary of the limitations of coefficients of variation is provided by Pélabon et al. (2020). For ratio-scale data, both repeatability and the coefficient of individual variation are phenotypic analogues for statistics relating to evolutionary potential (the utility of which are, themselves, debatable; Hansen et al., 2011). Repeatability roughly sets the upper limit on narrow-sense heritability (but see: Dohm, 2002), whereas the coefficient of individual variation is analogous to the coefficient of additive genetic variance, CV_A (Dochtermann & Royauté, 2019; Holtmann et al., 2017; Houle, 1992). Notably, by definition (detailed in the Supplementary Information), a repeatability estimate from the dispersion model will always be smaller than its counterpart from the mean model, whereas estimates of the coefficient of individual variation for means and variances are comparable to each other.

Worked example: Zebrafish behaviour

Abridged descriptions of data collection and analyses are presented here, with further details available in the Supplementary Information.

Data description

To demonstrate the models and calculations described here and in the main text, we use behaviour data from a laboratory population of 248 adult zebrafish (*Danio rerio*) following protocols developed by Fangmeier et al. (2018). We presented individual zebrafish with stimuli on computer tablets, and used automated tracking software to record five behavioural traits: (1) aggression: time spent within 5 cm of an aggressor stimulus; (2) sociability: time spent within 5 cm of a shoal stimulus; (3) neophilia: time spent within 10 cm of a novel object stimulus; (4) boldness: time spent within 10 cm of a predator stimulus; and (5) activity: the average distance travelled without stimulus present. Stimuli lasted for three minutes each, with four minutes of no stimulus separating them. Individuals were measured a maximum of 12 times each, with around two weeks separating each measurement.

Data analysis

Data and code to reproduce results from this worked example are available from http://doi.org/10.17605/OSF.IO/V3QAX (O'Dea & Nakagawa, 2020). All analyses were conducted in the *R* computing environment (v. 4.0.2; R Core Team, 2020), and Bayesian models were run using Stan, accessed through the *R* environment using the '*stan*' function from the '*RStan*' package (v. 2.21.2; Stan Development Team, 2020). Neophilia and boldness were strongly positively skewed, due to most fish spending little to no time within 10 cm of the stimulus (reflecting aversive behavioural responses to the 'risky' stimulus zone). We therefore analysed neophilia and boldness on the square-root scale, which greatly improved the normality of residuals. In addition, to help model fitting, the five response variables were *z*-transformed prior to analysis (to put them all in standard deviation units, with a mean of zero). For biological interpretation of model estimates, the unscaled descriptive statistics were used for back-transformation from the *z*-scale (back-transformations are described in the Supplementary Information, and shown in Fig. 4, below).

For illustrative purposes, we specified six different models: (1) random intercept in mean model only (personality; single hierarchical, as in equations 1-4); (2) random intercept and random slope in mean model (personality and plasticity; single hierarchical, as in equations 5-7); (3) bivariate model, random intercept and random slope in mean model only (personality and plasticity syndromes and associations; single hierarchical, as in equations 10-13); (4) random intercept in both the mean and

dispersion models (personality and predictability; double hierarchical); (5) random intercept in both the mean and dispersion models, random slope in the mean model only (personality, plasticity, and predictability; double hierarchical, as in equations 17-20); (6) bivariate model, random intercept in both the mean and dispersion models, random slope in the mean model only (personality, plasticity, and predictability syndromes and associations; double hierarchical, as in equations 25-30). Estimates of repeatability (Rp_m and Rp_v) and the coefficient of individual variation (CV_{IDm} and CV_{IDv}) were made using the formulas presented in the Supplementary Information (with small modifications for single hierarchical models, available in the supplementary script 'Step 3 - Process Models.R').

Results and discussion: worked example

Full results are available in Supplementary Tables S1, S2, and S3, and a selection of diagnostic plots are shown in Supplementary Figures S01-S14. Statistical significance was determined from 95% credible intervals not crossing zero. Descriptive statistics

($\mu \pm \sigma$) for the five behavioural traits were: (1) aggression = 79.3 $\pm$ 49.3 seconds spent within 5 cm of aggressor video; (2) sociability = 109.0 $\pm$ 54.1 seconds spent within 5 cm of shoal video; (3) neophilia (square-root transformed) = 4.75 $\pm$ 3.48 seconds spent within 10 cm of novel object animation; (4) boldness (square-root transformed) = 6.16 $\pm$ 3.18 seconds spent within 10 cm of predator animation; (5) activity = 0.559 $\pm$ 0.217 cm moved in 1 second.

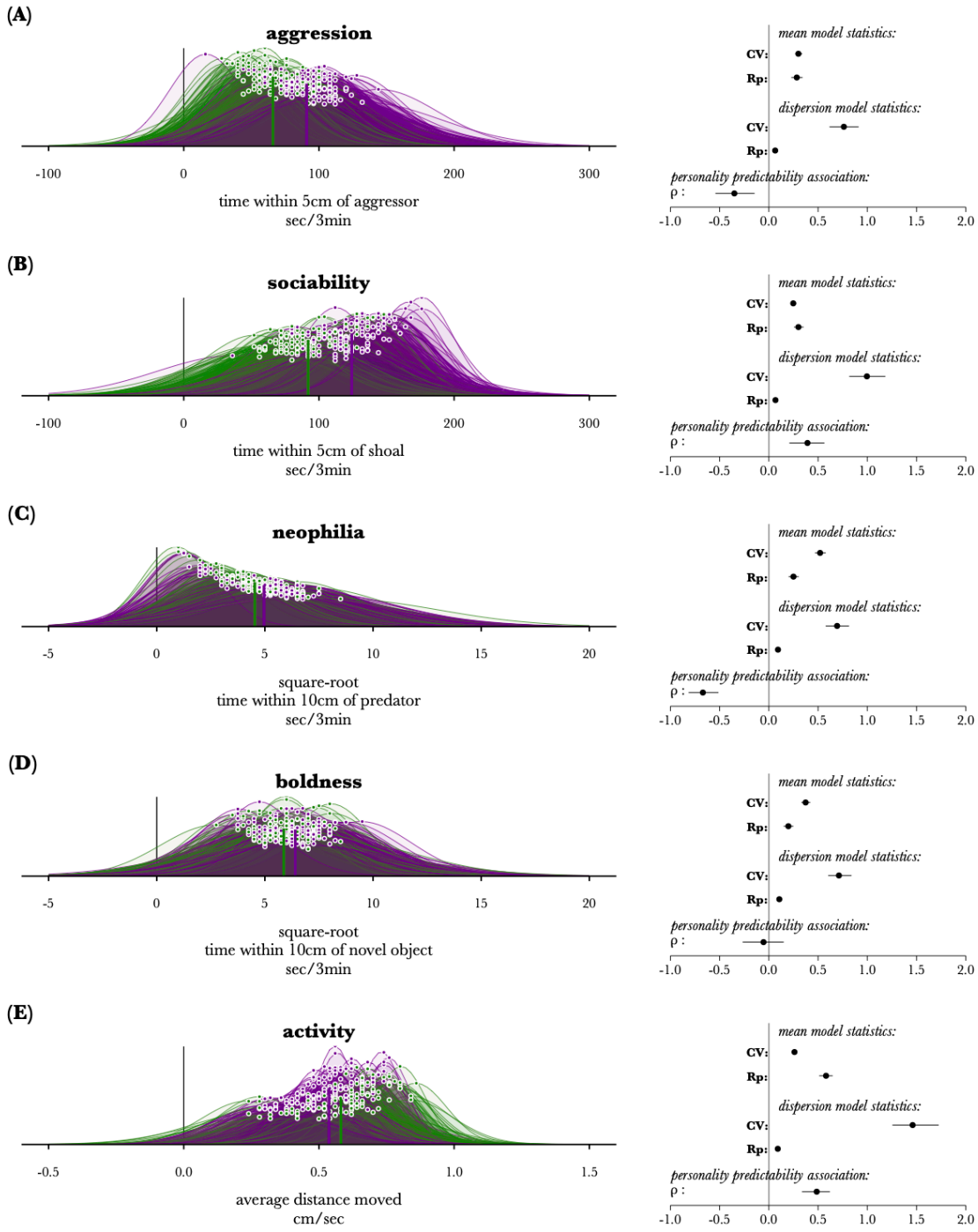

Figure 4

Individual differences, summary statistics, and sex differences in five behaviours, estimated from univariate double hierarchical linear models, with a random intercept in mean and dispersion (i.e. personality and predictability), and a random slope for the effect of age (i.e. plasticity). **Left-hand panels** show simulated distributions for the behaviour of N_{ID} = 248 individuals. Females = **purple**; males = **green**. Purple and green vertical lines show the population averages for female and male zebrafish, respectively. Filled points indicate the mean and peak of each individual's

distribution; taller peaks indicate a narrower distribution, and therefore a smaller variance (i.e. greater predictability). Each individual's distribution is simulated from the posterior mean estimate of their phenotypic mean and variance, which were back-transformed from the *z*-scale to the raw scale (within-individual variances were first converted back from the ln- scale by taking the exponent). **Right-hand panels** show summary statistics (posterior means and 95% credible intervals) for the magnitude of individual differences in both the mean and dispersion models (CV = coefficient of individual variation; Rp = repeatability; see Supplementary Information), and the personality predictability association for each behaviour (i.e. the sign-reversed correlation between individual intercepts in the mean and dispersion model; equation 22).

Individual differences in personalities

The average behaviour of individuals varied substantially, as can be seen by the spread of distributions in Fig. 4. From the coefficient of individual variation, relative to the population mean, neophilia and boldness showed the most individual differences in average behaviours ($\mathrm{CV_{IDm}} \approx 0.5$ and 0.4, respectively). Note that neophilia was positively skewed, even after the square-root transformation, which likely over-estimated $\mathrm{CV_{IDm}}$ (Fig. 4C; the importance of normality for DHGLMs is discussed in the Supplementary Information). While aggression, sociability, and activity had a similar magnitude of individual differences ($\mathrm{CV_{IDm}} \approx 0.3$), those differences were the most consistent for activity ($\mathrm{Rp_m} \approx 0.6$; Fig. 4E). The consistencies of individual differences were similar for sociability, aggression, neophilia, and boldness ($\mathrm{Rp_m} \approx 0.2$-$0.3$). Estimates of repeatability from the single hierarchical models were around 5-10% lower than those from DHGLMs, suggesting that when homoscedasticity is assumed, the total estimate of residual variance can be inflated, and therefore underestimate repeatability.

Males were significantly more active, less aggressive and social, and less bold and neophilic than females (Fig. 5; Table S1). Previous studies on zebrafish have also found male zebrafish to be more active (e.g. Dereje et al., 2012; Moretz et al., 2007; Mustafa et al., 2019), but males are also generally reported to be bolder than females (e.g. Dahlbom et al., 2011; Dereje et al., 2012; Kern et al., 2016). However, estimates of sex differences can depend on both the type of behavioural assays, and the zebrafish strain (Mustafa et al., 2019). Often measures of 'boldness' are conflated with activity levels (e.g. distanced moved in an open arena). Here, it is conceivable that female zebrafish, who were less active than males, happened to settle close to the stimulus screen, although across individuals more activity was correlated with more time near the predator stimulus (e.g. positive

activity-boldness correlation).

Zebrafish behavioural syndromes

While zebrafish studies have found mixed results for a total (i.e. un-partitioned) phenotypic correlation between aggression and boldness (Martins & Bhat, 2014; Norton & Bally-Cuif, 2012), the across-individual correlations we estimated here provide evidence for proactive behavioural syndromes. Statistically significant correlations between mean individual intercepts (equation 14) were all in the positive direction (Fig. 6). Strong correlations existed between neophilia and boldness, and between aggression and sociability. Moderate correlations were seen between neophilia and aggression, neophilia and sociability, and aggression and boldness. In addition to the moderate positive correlation between activity and boldness, there was a weaker negative correlation between activity and sociability (albeit this negative correlation was not statistically significant). Activity was not significantly correlated with aggression or neophilia.

Individual differences in predictability of behaviour

All measured behaviours showed considerable heteroscedasticity, which is depicted in Fig. 4 as variability in the heights of individual peaks. The magnitude of individual differences in predictability was most pronounced in activity ($\mathrm{CV_{IDv}} \approx 1.25$) and least pronounced for aggression, neophilia, and boldness ($\mathrm{CV_{IDv}} \approx 0.6$) (Table S2). For comparisons to studies that model individual differences in residual standard deviations, rather than variances, this translates into a range of $\mathrm{CV_{IDsd}}$ from 0.3 to 0.5 (the Supplementary Information and code contain conversion formulas), which is in the range of estimates seen over shorter timespans for guppies and zebrafish (Mitchell & Biro, 2017; Mitchell et al., 2016). As with mean differences, the behaviour with the greatest magnitude of individual differences in predictability did not have the greatest *consistency* of differences, as measured by repeatability. Individual differences in predictability were most consistent for boldness ($\mathrm{Rp_v} \approx 0.11$), although the other behaviours were not far behind ($\mathrm{Rp_v} \approx 0.06 – 0.09$).

Zebrafish personality-plasticity associations and plasticity syndromes

Over the course of the experiment zebrafish became, on average, less active, aggressive, and sociable, but tended to become more neophilic and bold (Fig. 5). The plastic effect of age could be driven by habituation (Biro, 2012). Plasticity in sociability (Fig. 5C) was the most integrated with other individual differences. Individuals with more active and more sociable personalities were less

plastic (moderate negative personality-plasticity associations; equation 9, Fig. 3D), which could provide some support for predictions by Dubois (2019). However, the magnitudes of plasticity in aggression, neophilia, and boldness were unrelated to individual personalities (Fig. 6, green diagonal: A,F,J,M,O). For plasticity syndromes (equation 16, Fig. 3B), individuals who were more plastic in sociability also tended to be more plastic in aggression, and less plastic in activity (moderately positive and negative plasticity syndromes, respectively; Fig. 6, blue boxes). No other combinations of traits showed statistically significant plasticity syndromes. Within the same system, therefore, we found all possible outcomes for plasticity syndromes — position, negative, and null relationships — suggesting individual differences in plasticity are underpinned by multiple mechanisms.

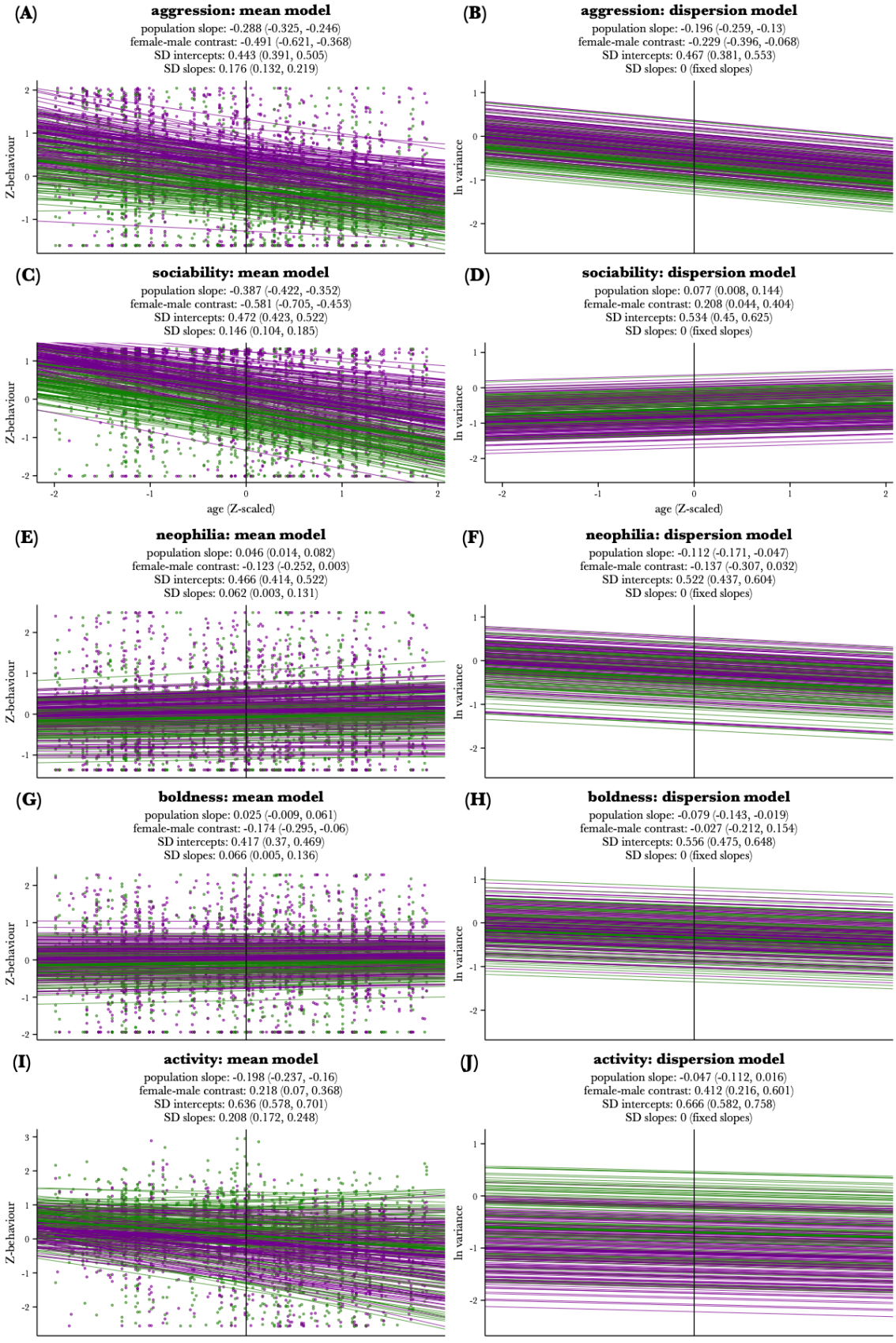

Figure 5

Individual differences in personality, plasticity, and predictability. **Purple** = female zebrafish, and

green = male zebrafish. In left-hand panels, points show the raw values of zebrafish behaviour across age. Population estimates for male and female zebrafish are shown as thick green and purple lines, respectively. **Personality**: the left-hand column shows individual differences in intercepts (i.e. the point at which the sloped lines cross the vertical line at zero). **Plasticity**: the left-hand column shows individual differences in slopes (i.e. the change in average behaviour with zebrafish age). **Predictability**: the right-hand column shows individual intercepts for within-individual variances, and the population-level change in predictability with time.

Zebrafish predictabilities and predictability syndromes

Male zebrafish were significantly less predictable than female zebrafish in activity and sociability (i.e. greater within-individual variance), and more predictable in aggression (i.e. smaller within-individual variance) (Fig. 5, right column; Supplementary Table S1). There were no statistically significant sex differences in predictability for neophilia (Fig. 5 F) or boldness (Fig. 5H). Predictability tended to increase with age for aggression (Fig. 5B), neophilia (Fig. 5F), and boldness (Fig. 5H), as seen by negative slopes for age against within-individual variance. In contrast, sociability became less predictable with time (Fig. 5D), and there was no change in the predictability of activity with time (Fig. 5J). From bivariate models, we found moderate-to-strong positive predictability syndromes for all combinations of traits (see blue boxes in Fig. 6, derived from equation 31; see also Fig. 3C and Table S3). In contrast to plasticity, therefore, predictability was strongly integrated among behavioural traits. Positive predictability syndromes could be indicative of mechanistic constraints, which limit the independent evolution of individual's predictability across traits, or correlated selective pressures, such that individuals are selected to be more or less predictable for a suite of related traits. Quantitative genetic methods can be incorporated into studies of predictability to determine the relative contribution of environmental and genetic effects on individual differences in predictability (e.g. Martin et al., 2017; Prentice et al., 2020).

Zebrafish personality-predictability associations

We found both moderately positive, weakly negative, negative, and null personality-predictability associations (Fig. 3E; Fig. 6, green diagonal; equation 22). More active and sociable zebrafish also tended to be more predictable, as measured by a narrow distribution in behaviours. In contrast, individuals who were more aggressive and neophilic tended to be less predictable in their behaviour. Boldness did not show a significant personality-predictability association (Fig. 6M). Therefore, there was no clear trend for riskier personality types to also be less predictable, or for more extreme personalities to show greater precision around their mean.

Zebrafish plasticity-predictability associations

Only activity and sociability showed an association between plasticity and predictability (equation 24; Fig. 3F). This pattern is similar to the personality-plasticity associations, except in this case sociability and activity showed correlations in opposite directions. Individuals who were more predictable in activity were less plastic (i.e. had a smaller change in activity as they aged), whereas individuals who were more predictable in sociability showed a greater change in sociability as they aged. The remaining behaviours (aggression, neophilia, and boldness) did not show significant plasticity-predictability associations (Fig. 6; Supplementary Table S3). Given that we found strong phenotypic integration of predictability (in positive predictability syndromes), but variable or null integration of plasticity (in positive, negative, and null predictability syndromes), the variable relationships between plasticity and predictability were likely driven by independence of plasticity between traits, rather than flexibility in predictability.

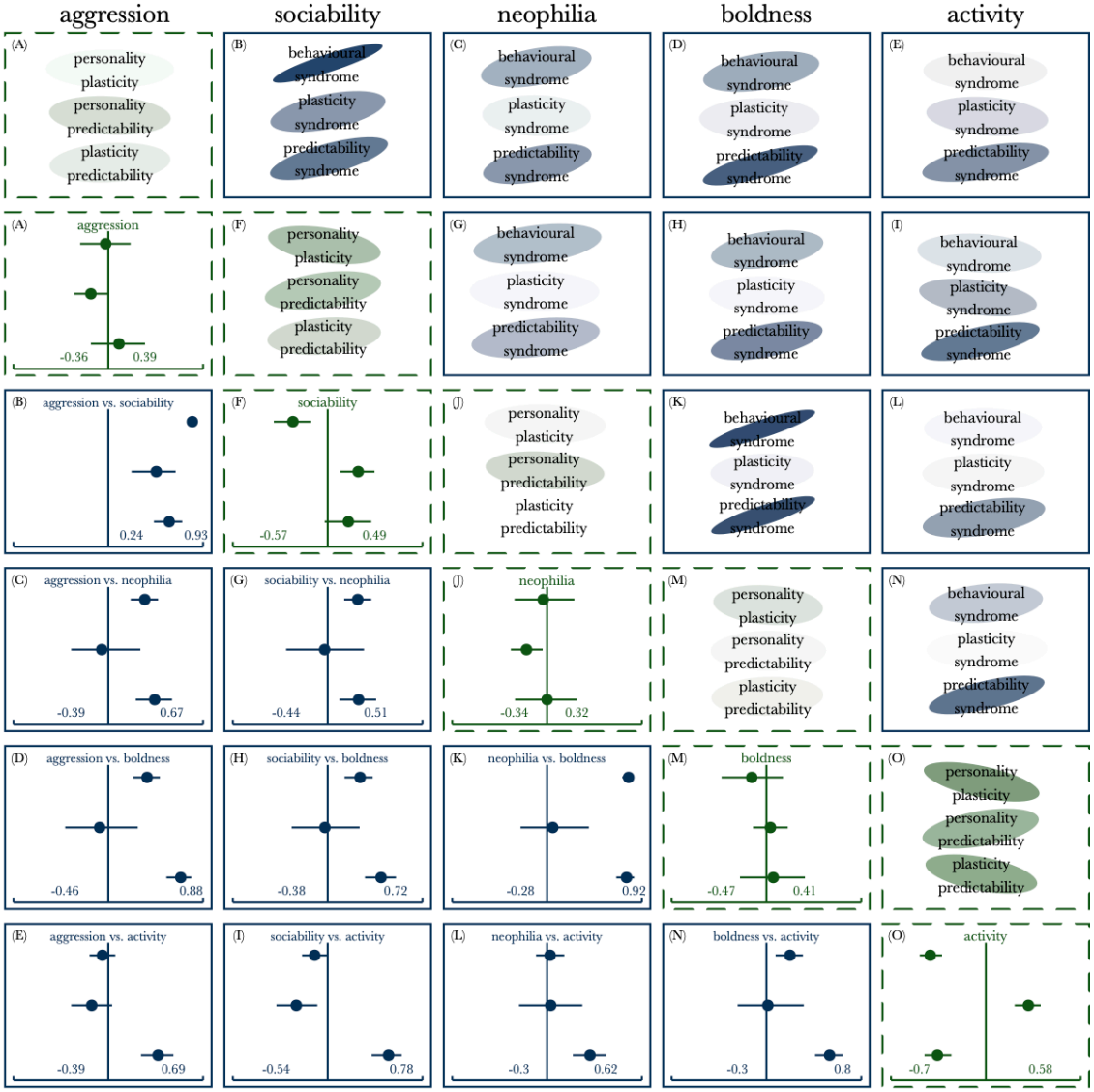

Figure 6

Six types of correlations between and within personality, plasticity, and predictability, estimated from bivariate double hierarchical linear models on five behavioural traits. Diagonal boxes, in green, show within-trait associations (averaged across the four bivariate models featuring each focal trait). Off-diagonal boxes, in blue, show between-trait correlations. In the **upper triangle**, each ellipsis represents a correlation; ellipses tilted to the right represent positive correlations (i.e. $r > 0$), and ellipses tilted to the left represent negative correlations (i.e. $r < 0$). Narrower ellipses, with more opaque shading, represent stronger correlations. Ellipses with no tilt, and more transparent colouration, represent little or no correlation (i.e. $r = 0$). The **lower triangle** shoes posterior mean estimates and 95% credible intervals for each correlation (bound between -1 and 0), with the range of lower and upper estimates shown on the *x*-axis. Personality-plasticity associations and plasticity syndromes were calculated from slope magnitudes (the absolute value of individual slopes; equations 9 and 16). Equivalent correlations for slope deviations (equations 8 and 15) are available from the supplementary code.

Discussion

Extending the animal personality framework

Incorporating predictability into studies of personality and plasticity creates an opportunity to test more nuanced questions about how phenotypic variation is maintained, or constrained. For some traits, it might be adaptive to be unpredictable, such as in predator-prey interactions (Briffa, 2013). For other traits, selection might act to minimise maladaptive imprecision around an optimal mean (Hansen et al., 2006). In the worked example, we showed phenotypic integration of predictability across multiple behavioural traits, and some integration of predictability with personality and plasticity. Phenotypic integration could hint at genetic integration too; other studies have found additive genetic variance in predictability (Martin et al., 2017; Prentice et al., 2020). Given that different traits might have different optimal levels of unpredictability, phenotypic integration of predictability could constrain variation in one trait (resulting in lower than optimal variability) and maintain variation in another (resulting in greater than optimal variability). Associations with personality and plasticity mean that variation in predictability — the lowest level of the phenotypic hierarchy — could have cascading effects upwards (Westneat et al., 2015). Empirical estimates of the strength of these associations can inform theoretical models on the simultaneous evolution of means and variances.

Beyond behaviour

We focussed this paper on animal behaviour (the field we are most familiar with), but the models are broadly adaptable. Individuals can show differences in predictability for any trait that is repeatedly expressed. For example, medical researchers might want to quantify the variability of patient's drug responses (Nettles et al., 2006), and selective breeders of plants might want to reduce individual variability in seed or fruit mass (Herrera, 2017). The review by Herrera (2017) discusses the overlooked importance of variability within the structures of an individual plant, including for plant-animal interactions. Given the large sample sizes required to estimate multiple individual differences, the most tractable tests of the synchronous evolution of means and variances could come from non-animal systems. Clonal species can also be used to estimate individual differences in predictability of non-labile traits.

Introducing a fourth individual difference: stochastic malleability

As a future extension to the models presented here, it is possible (given sufficient data) to include a random slope in the dispersion model (i.e. to add $\mathrm{ID}_{\mathrm{v2}j,\mathrm{exp}}$ into equation 18), to estimate individual differences in 'stochastic malleability' (i.e. plasticity in predictability, or simply 'malleability'). While it would require many repeated measurements across different contexts, a fourth type of individual difference, in malleability, could answer three additional questions about phenotypic integration (Fig. 7, below): (1) is the level of malleability integrated across traits (i.e. *malleability syndromes*), or can individuals be malleable in one trait and show fixed predictability in another? (2): do individuals with more flexibility in average phenotypes show more flexibility in variability (i.e. *plasticity-malleability associations*)? (3) are some personality types more or less likely to change their level of predictability in response to an environmental change (i.e. *personality-malleability associations*)? Stochastic malleability could be an important aspect of learning or adapting to novel conditions: naïve individuals (i.e. individuals who are young, or in an unfamiliar environment) might increase variability, to 'sample' a wider array of options. As individuals gain more experience, they might hone in upon the optimal phenotype, and therefore become more predictable (McNamara et al., 2006). An interesting avenue of future research, therefore, could be to incorporate individual differences in malleability into studies of learning or invasion biology (c.f. Chapple et al., 2012; Griffin et al., 2015).

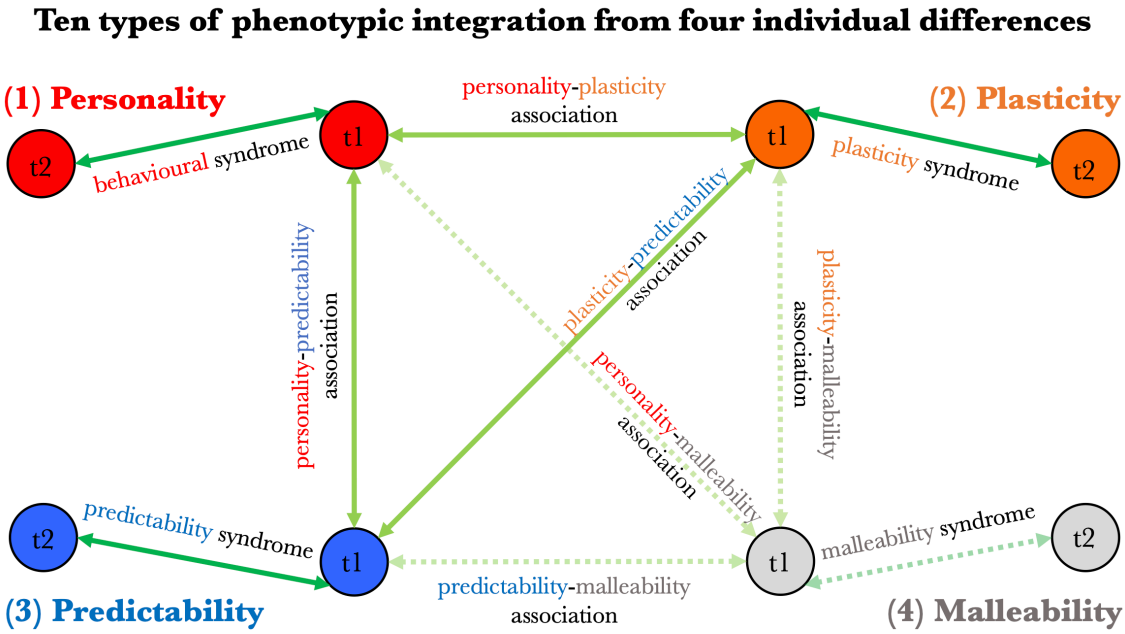

Figure 7

Ten types of between-individual correlations can be modelled in a bivariate DHGLM (t1 = trait 1, and t2 = trait 2; as in Table 1), containing four individual differences: (1) personality (random intercept in mean models); (2) plasticity (random slope in mean models); (3) predictability (random intercept in dispersion models); and (4) malleability (random slope in dispersion models). Solids lines indicate correlations that were modelled in the worked example; our dataset was not suitable to model the correlations shown by dashed lines.

Conclusion

While many studies quantify consistent individual differences in repeatedly expressed traits, such as behaviour, much of the mystery of phenotypic variation is obscured within residual variation. Individuals impact the world not only through their 'average' phenotype, but also through their extremes. Given that evolution can act on both averages and variances, to understand the evolution of labile traits, we need to measure both the magnitude and consistency of individual differences, as well as their phenotypic integration. Using the concepts and tools presented here, empiricists can chronicle the integration of multiple levels of phenotypic variation in diverse systems. In doing so we can improve our understanding of the factors promoting and constraining variability, as well as the evolution, and ecological consequences, of individuality.

Acknowledgements

This work was conducted with permission from the Garvan Institute Animal Ethics Committee (approval number 15/15) with funding support from UNSW. Daniel Hesselson provided access to the zebrafish population featured in the worked example, which were looked after by staff within the Garvan Institute of Medical Research. Data were collected with help from Malgorzata Lagisz, Melissa Fangmeier, Takuji Usui, Marissa Baptista, Alex Lincoln-Dodgson, Hamza Anwer, Harry Thomas, and Alexander Aloy. Joel Pick helped write the code for bivariate predictability models. RO was supported by an Australian Government Research Training Program scholarship, DN was supported by an ARC DECRA fellowship (DE150101774), SN was supported by an ARC Discovery grant (DP180100818), and all authors declare no conflicts of interest. We would like to thank Bob Wong and Alison Bell for their comments on an earlier version of this manuscript.

References

Allegue, H., Araya-Ajoy, Y. G., Dingemanse, N. J., Dochtermann, N. A., Garamszegi, L. Z., Nakagawa, S., et al. (2017). Statistical Quantification of Individual Differences (SQuID): An educational and statistical tool for understanding multilevel phenotypic data in linear mixed models. *Methods in Ecology and Evolution*, *8*(2), 257–267. http://doi.org/10.1111/2041-210X.12659

Bell, A. M. (2007). Future directions in behavioural syndromes research. *Proceedings of the Royal Society B: Biological Sciences*, *274*(1611), 755–761. http://doi.org/10.1098/rspb.2006.0199

Bell, A. M., Hankison, S. J., & Laskowski, K. L. (2009). The repeatability of behaviour: A meta-analysis. *Animal Behaviour*, *77*(4), 771–783. http://doi.org/10.1016/j.anbehav.2008.12.022

Biro, P. A. (2012). Do rapid assays predict repeatability in labile (behavioural) traits? Animal Behaviour, 83(5), 1295–1300. http://doi.org/10.1016/j.anbehav.2012.01.036

Bogacz, R., Wagenmakers, E.-J., Forstmann, B. U., & Nieuwenhuis, S. (2010). The neural basis of the speed–accuracy tradeoff. *Trends in Neurosciences, 33*(1), 10–16. http://doi.org/10.1016/j.tins.2009.09.002

Briffa, M. (2013). Plastic proteans: Reduced predictability in the face of predation risk in hermit crabs. *Biology Letters*, *9*(5), 20130592–4. http://doi.org/10.1098/rsbl.2013.0592

Brommer, J. E. (2013). On between-individual and residual (co)variances in the study of animal personality: Are you willing to take the "individual gambit"? *Behavioral Ecology and Sociobiology*, *67*(6), 1027–1032. http://doi.org/10.1007/s00265-013-1527-4

Chapple, D. G., Simmonds, S. M., & Wong, B. B. M. (2012). Can behavioral and personality traits influence the success of unintentional species introductions? *Trends in Ecology & Evolution*, *27*(1), 57–

64. http://doi.org/10.1016/j.tree.2011.09.010

Cleasby, I. R., Nakagawa, S., & Schielzeth, H. (2015). Quantifying the predictability of behaviour: Statistical approaches for the study of between-individual variation in the within-individual variance. *Methods in Ecology and Evolution*, *6*(1), 27–37. http://doi.org/10.1111/2041-210X.12281

Cornwell, T. O., McCarthy, I. D., Snyder, C. R. A., & Biro, P. A. (2019). The influence of environmental gradients on individual behaviour: Individual plasticity is consistent across risk and temperature gradients. *Journal of Animal Ecology*, *88*(4), 511–520. http://doi.org/10.1111/1365-2656.12935

Dahlbom, S. J., Lagman, D., Lundstedt-Enkel, K., Sundström, L. F., & Winberg, S. (2011). Boldness predicts social status in zebrafish (*Danio rerio*). *PLoS One*, *6*(8), e23565. http://doi.org/10.1371/journal.pone.0023565

de Villemereuil, P., Morrissey, M. B., Nakagawa, S., & Schielzeth, H. (2018). Fixed-effect variance and the estimation of repeatabilities and heritabilities: Issues and solutions. *Journal of Evolutionary Biology*, *31*(4), 621–632. http://doi.org/10.1111/jeb.13232

Dereje, S., Sawyer, S., Oxendine, S. E., Zhou, L., Kezios, Z. D., Wong, R. Y., et al. (2012). Comparing behavioral responses across multiple assays of stress and anxiety in zebrafish (*Danio rerio*). *Behaviour*, *149*(10-12), 1205–1240. http://doi.org/10.1163/1568539X-00003018

DeWitt, T. J., Sih, A., & Wilson, D. S. (1998). Costs and limits of phenotypic plasticity. *Trends in Ecology & Evolution*, *13*(2), 77–81. http://doi.org/10.1016/S0169-5347(97)01274-3

Dingemanse, N. J., & Dochtermann, N. A. (2013). Quantifying individual variation in behaviour: Mixed-effect modelling approaches. *Journal of Animal Ecology*, *82*(1), 39–54. http://doi.org/10.1111/1365-2656.12013

Dingemanse, N. J., Barber, I., Wright, J., & Brommer, J. E. (2012). Quantitative genetics of behavioural reaction norms: Genetic correlations between personality and behavioural plasticity vary across stickleback populations. *Journal of Evolutionary Biology*, *25*(3), 485–496. http://doi.org/10.1111/j.1420-9101.2011.02439.x

Dingemanse, N. J., Dochtermann, N., & Wright, J. (2010a). A method for exploring the structure of behavioural syndromes to allow formal comparison within and between data sets. *Animal Behaviour*, *79*(2), 439–450. http://doi.org/10.1016/j.anbehav.2009.11.024

Dingemanse, N. J., Kazem, A. J. N., Réale, D., & Wright, J. (2010b). Behavioural reaction norms: Animal personality meets individual plasticity. *Trends in Ecology & Evolution*, *25*(2), 81–89. http://doi.org/10.1016/j.tree.2009.07.013

Dochtermann, N. A. (2010). Behavioral syndromes: Carryover effects, false discovery rates, and a priori hypotheses. *Behavioral Ecology*, *21*(3), 437–439. http://doi.org/10.1093/beheco/arq021

Dochtermann, N. A., & Royauté, R. (2019). The mean matters: Going beyond repeatability to interpret

behavioural variation. *Animal Behaviour*, *153*, 147–150. http://doi.org/10.1016/j.anbehav.2019.05.012

Dohm, M. R. (2002). Repeatability estimates do not always set an upper limit to heritability. *Functional Ecology*, *16*(2), 273–280. http://doi.org/10.1046/j.1365-2435.2002.00621.x

Dubois, F. (2019). Why are some personalities less plastic? *Proceedings of the Royal Society B: Biological Sciences*, *286*(1908), 20191323–7. http://doi.org/10.1098/rspb.2019.1323

Fangmeier, M. L., Noble, D. W. A., O'Dea, R. E., Usui, T., Lagisz, M., Hesselson, D., & Nakagawa, S. (2018). Computer animation technology in behavioral sciences: A sequential, automatic, and high-throughput approach to quantifying personality in zebrafish (*Danio rerio*). *Zebrafish*, zeb.2017.1532–4. http://doi.org/10.1089/zeb.2017.1532

Fawcett, T. W., Hamblin, S., & Giraldeau, L. A. (2012). Exposing the behavioral gambit: The evolution of learning and decision rules. *Behavioral Ecology*, *24*(1), 2–11. http://doi.org/10.1093/beheco/ars085

Flatt, T. (2005). The evolutionary genetics of canalization. *The Quarterly Review of Biology*, *80*(3), 287–316. http://doi.org/10.1086/432265

Gavrilets, S., & Scheiner, S. M. (1993). The genetics of phenotypic plasticity. V. Evolution of reaction norm shape. *Journal of Evolutionary Biology*, *6*(1), 31–48. http://doi.org/10.1046/j.1420-9101.1993.6010031.x

Ghalambor, C. K., McKay, J. K., Carroll, S. P., & Reznick, D. N. (2007). Adaptive versus non-adaptive phenotypic plasticity and the potential for contemporary adaptation in new environments. *Functional Ecology*, *21*(3), 394–407. http://doi.org/10.1111/j.1365-2435.2007.01283.x

Gianoli, E., & Palacio-Lopez, K. (2009). Phenotypic integration may constrain phenotypic plasticity in plants. *Oikos*, *118*(12), 1924–1928. http://doi.org/10.1111/j.1600-0706.2009.17884.x

Gomulkiewicz, R., & Kirkpatrick, M. (1992). Quantitative genetics and the evolution of reaction norms. *Evolution*, *46*(2), 390–411. http://doi.org/10.1111/j.1558-5646.1992.tb02047.x

Griffin, A. S., Guillette, L. M., & Healy, S. D. (2015). Cognition and personality: An analysis of an emerging field. *Trends in Ecology & Evolution*, *30*(4), 207–214. http://doi.org/10.1016/j.tree.2015.01.012

Hadfield, J. D., Wilson, A. J., Garant, D., Sheldon, B. C., & Kruuk, L. E. B. (2010). The misuse of BLUP in ecology and evolution. *The American Naturalist*, *175*(1), 116–125. http://doi.org/10.1086/648604

Hansen, T. F., & Houle, D. (2008). Measuring and comparing evolvability and constraint in multivariate characters. *Journal of Evolutionary Biology*, *21*(5), 1201–1219. http://doi.org/10.1111/j.1420-9101.2008.01573.x

Hansen, T. F., Carter, A. J. R., & Pélabon, C. (2006). On adaptive accuracy and precision in natural

populations. *The American Naturalist*, *168*(2), 168–181. http://doi.org/10.1086/505768

Hansen, T. F., Pélabon, C., & Houle, D. (2011). Heritability is not Evolvability, *38*(3), 258–277. http://doi.org/10.1007/s11692-011-9127-6

Herrera, C. M. (2017). The ecology of subindividual variability in plants: Patterns, processes, and prospects. *Web Ecology*, *17*(2), 51–64. http://doi.org/10.5194/we-17-51-2017

Holtmann, B., Lagisz, M., & Nakagawa, S. (2017). Metabolic rates, and not hormone levels, are a likely mediator of between-individual differences in behaviour: A meta-analysis. *Functional Ecology*, *31*(3), 685–696. http://doi.org/10.1111/1365-2435.12779

Houle, D. (1992). Comparing evolvability and variability of quantitative traits. *Genetics*, *130*, 195–204.

Kern, E. M. A., Robinson, D., Gass, E., Godwin, J., & Langerhans, R. B. (2016). Correlated evolution of personality, morphology and performance. *Animal Behaviour*, *117*, 79–86. http://doi.org/10.1016/j.anbehav.2016.04.007

Lin, X., Mermelstein, R. J., & Hedeker, D. (2018). A 3-level Bayesian mixed effects location scale model with an application to ecological momentary assessment data. *Statistics in Medicine*, *37*(13), 2108–2119. http://doi.org/10.1002/sim.7627

Mallitt, K. L., Bonser, S. P., & Hunt, J. (2010). The plasticity of phenotypic integration in response to light and water availability in the pepper grass, *Lepidium bonariense*. *Evolutionary Ecology*, *24*(6), 1321–1337. http://doi.org/10.1007/s10682-010-9373-6

Martin, J. G. A., Pirotta, E., Petelle, M. B., & Blumstein, D. T. (2017). Genetic basis of between-individual and within-individual variance of docility. *Journal of Evolutionary Biology*, *30*(4), 796–805. http://doi.org/10.1111/jeb.13048

Martins, E. P., & Bhat, A. (2014). Population-level personalities in zebrafish: Aggression-boldness across but not within populations. *Behavioral Ecology*, *25*(2), 368–373. http://doi.org/10.1093/beheco/aru007

McNamara, J. M., Green, R. F., & Olsson, O. (2006). Bayes' theorem and its applications in animal behaviour. *Oikos*, *112*(2), 243–251. http://doi.org/10.1111/j.0030-1299.2006.14228.x

Mitchell, D. J., & Biro, P. A. (2017). Is behavioural plasticity consistent across different environmental gradients and through time? *Proceedings of the Royal Society B: Biological Sciences*, *284*(1860), 20170893. http://doi.org/10.1098/rspb.2017.0893

Mitchell, D. J., Fanson, B. G., Beckmann, C., & Biro, P. A. (2016). Towards powerful experimental and statistical approaches to study intraindividual variability in labile traits. *Royal Society Open Science*, *3*(10), 160352–10. http://doi.org/10.1098/rsos.160352

Moirón, M., Laskowski, K. L., & Niemelä, P. T. (2020). Individual differences in behaviour explain variation in survival: a meta-analysis. *Ecology Letters*, *23*(2), 399–408. http://doi.org/10.1111/ele.13438

Moretz, J. A., Martins, E. P., & Robison, B. D. (2007). Behavioral syndromes and the evolution of correlated behavior in zebrafish. *Behavioral Ecology*, *18*(3), 556–562. http://doi.org/10.1093/beheco/arm011

Mustafa, A., Roman, E., & Winberg, S. (2019). Boldness in male and female zebrafish (*Danio rerio*) is dependent on strain and test. *Frontiers in Behavioral Neuroscience*, *13*, 20150509. http://doi.org/10.3389/fnbeh.2019.00248

Nakagawa, S., & Schielzeth, H. (2010). Repeatability for Gaussian and non-Gaussian data: A practical guide for biologists. *Biological Reviews*, *85*(4), 935–956. http://doi.org/10.1111/j.1469-185X.2010.00141.x

Nettles, R. E., Kieffer, T. L., Parsons, T., Johnson, J., Cofrancesco, J., Gallant, J. E., et al. (2006). Marked intraindividual variability in antiretroviral concentrations may limit the utility of therapeutic drug monitoring. *Clinical Infectious Diseases*, *42*(8), 1189–1196. http://doi.org/10.1086/501458

Niemelä, P. T., & Dingemanse, N. J. (2018). On the usage of single measurements in behavioural ecology research on individual differences. *Animal Behaviour*, *145*, 99–105. http://doi.org/10.1016/j.anbehav.2018.09.012

Norton, W. H. J., & Bally-Cuif, L. (2012). Unravelling the proximate causes of the aggression-boldness behavioural syndrome in zebrafish. *Behaviour*, *149*(10-12), 1063–1079. http://doi.org/10.1163/1568539X-00003012

O'Dea, R.E., Nakagawa, S. (2020). Worked Example: Personality, Plasticity, and Predictability. *Open Science Framework.* http://doi.org/10.17605/OSF.IO/V3QAX

Pélabon, C., Hilde, C. H., Einum, S., & Gamelon, M. (2020). On the use of the coefficient of variation to quantify and compare trait variation. *Evolution Letters*, 4(3), 180–188. http://doi.org/10.1002/evl3.171

Pigliucci, M. (2002). Touchy and bushy: Phenotypic plasticity and integration in response to wind stimulation in *Arabidopsis thaliana*. *International Journal of Plant Sciences*, *163*(3), 399–408. http://doi.org/10.1086/339158

Pigliucci, M. (2003). Phenotypic integration: Studying the ecology and evolution of complex phenotypes. *Ecology Letters*, *6*(3), 265–272. http://doi.org/10.1046/j.1461-0248.2003.00428.x

Postma, E. (2006). Implications of the difference between true and predicted breeding values for the study of natural selection and micro-evolution. *Journal of Evolutionary Biology*, *19*(2), 309–320. http://doi.org/10.1111/j.1420-9101.2005.01007.x

Prentice, P. M., Houslay, T. M., Martin, J. G. A., & Wilson, A. J. (2020). Genetic variance for behavioural "predictability" of stress response. *Journal of Evolutionary Biology*, *39*(03), 473. http://doi.org/10.1111/jeb.13601

R Core Team. (2020). *R*: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from http://www.R-project.org.

Ramakers, J. J. C., Visser, M. E., & Gienapp, P. (2020). Quantifying individual variation in reaction norms: Mind the residual. *Journal of Evolutionary Biology*, *33*, 352-366. http://doi.org/10.1111/jeb.13571

Rast, P., Hofer, S. M., & Sparks, C. (2012). Modeling individual differences in within-person variation of negative and positive affect in a mixed effects location scale model using BUGS/JAGS. *Multivariate Behavioral Research*, *47*(2), 177–200. http://doi.org/10.1080/00273171.2012.658328

Reznick, D., Nunney, L., & Tessier, A. (2000). Big houses, big cars, superfleas and the costs of reproduction. *Trends in Ecology & Evolution*, *15*(10), 421–425. http://doi.org/10.1016/S0169-5347(00)01941-8

Schlichting, C. D. (1989). Phenotypic integration and environmental change. *BioScience*, *39*(7), 460–464. http://doi.org/10.2307/1311138

Sih, A., Bell, A., & Johnson, J. C. (2004). Behavioral syndromes: An ecological and evolutionary overview. *Trends in Ecology & Evolution*, *19*(7), 372–378. http://doi.org/10.1016/j.tree.2004.04.009

Sih, A., Cote, J., Evans, M., Fogarty, S., & Pruitt, J. (2012). Ecological implications of behavioural syndromes. *Ecology Letters*, *15*(3), 278–289. http://doi.org/10.1111/j.1461-0248.2011.01731.x

Sih, A., & Del Giudice, M. (2012). Linking behavioural syndromes and cognition: a behavioural ecology perspective. *Philosophical Transactions of the Royal Society B: Biological Sciences, 367*(1603), 2762–2772. http://doi.org/10.1098/rstb.2012.0216

Sih, A., Mathot, K. J., Moirón, M., Montiglio, P. O., Wolf, M., & Dingemanse, N. J. (2015). Animal personality and state–behaviour feedbacks: a review and guide for empiricists. *Trends in Ecology & Evolution*, 30(1), 50–60. http://doi.org/10.1016/j.tree.2014.11.004

Snell-Rood, E. C. (2013). An overview of the evolutionary causes and consequences of behavioural plasticity. *Animal Behaviour*, *85*(5), 1004–1011. http://doi.org/10.1016/j.anbehav.2012.12.031

Stamps, J. A. (2015). Individual differences in behavioural plasticities. *Biological Reviews*, *91*(2), 534–567. http://doi.org/10.1111/brv.12186

Stamps, J. A., & Biro, P. A. (2016). Personality and individual differences in plasticity. *Current Opinion in Behavioral Sciences*, *12*, 18–23. http://doi.org/10.1016/j.cobeha.2016.08.008

Stamps, J. A., & Groothuis, T. G. G. (2010). Developmental perspectives on personality: implications for ecological and evolutionary studies of individual differences. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *365*(1560), 4029–4041. http://doi.org/10.1098/rstb.2010.0218

Stan Development Team. (2020). *RStan*: the *R* interface to Stan. R package. Retrieved from http://mc-stan.org/

Stearns, S. C., & Koella, J. C. (1986). The evolution of phenotypic plasticity in life-history traits: Predictions of reaction norms for age and size at maturity. *Evolution*, *40*(5), 893–913. http://doi.org/10.1111/j.1558-5646.1986.tb00560.x

Stoffel, M. A., Nakagawa, S., & Schielzeth, H. (2017). *rptR*: Repeatability estimation and variance decomposition by generalized linear mixed-effects models. *Methods in Ecology and Evolution*, *8*(11), 1639–1644. http://doi.org/10.1111/2041-210X.12797

Viney, M., & Reece, S. E. (2013). Adaptive noise. *Proceedings of the Royal Society B: Biological Sciences*, *280*(20131104). http://doi.org/10.1098/rspb.2013.1104

Westneat, D. F., Hatch, M. I., Wetzel, D. P., & Ensminger, A. L. (2011). Individual variation in parental care reaction norms: Integration of personality and plasticity. *The American Naturalist*, *178*(5), 652–667. http://doi.org/10.1086/662173

Westneat, D. F., Schofield, M., & Wright, J. (2013). Parental behavior exhibits among-individual variance, plasticity, and heterogeneous residual variance. *Behavioral Ecology*, *24*(3), 598–604. http://doi.org/10.1093/beheco/ars207

Westneat, D. F., Wright, J., & Dingemanse, N. J. (2015). The biology hidden inside residual within-individual phenotypic variation. *Biological Reviews*, *90*(3), 729–743. http://doi.org/10.1111/brv.12131

Willmore, K. E., Young, N. M., & Richtsmeier, J. T. (2007). Phenotypic variability: Its components, measurement and underlying developmental processes. *Evolutionary Biology*, *34*(3-4), 99–120. http://doi.org/10.1007/s11692-007-9008-1

Wolf, M., van Doorn, G. S., Leimar, O., & Weissing, F. J. (2007). Life-history trade-offs favour the evolution of animal personalities. *Nature*, *447*(7144), 581–584. http://doi.org/10.1038/nature05835