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COMMENTARY

Defining behavioural syndromes and the role of 'syndrome deviation' in understanding their evolution

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Abstract This commentary highlights multivariate tools that have been used by evolutionary biologists in the study of syndromes and their evolution and discusses the insights that these methods provide into evolutionary processes relative to the metric 'syndrome deviation' that has recently been proposed by Herczeg and Garamszegi (Behav Ecol Sociobiol 66:161–169, 2012). We clarify that non-zero phenotypic correlations arise from the joint influences of within- and between-individual correlations, whereas only non-zero between-individual correlations represent behavioural syndromes, and discuss how acknowledgement of this subtle difference between phenotypic and between-individual correlations affects the applicability of syndrome deviation for the study of behavioural syndromes.

Keywords Animal personality · Behavioural syndrome · Correlational selection · Quantitative genetics · Mixed-effect modelling

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Introduction

In a recent paper published in *Behavioral Ecology and Sociobiology*, Herczeg and Garamszegi (2012) propose a new method for studying the evolutionary mechanisms shaping behavioural correlations, for example, between aggressiveness and boldness. Herczeg and Garamszegi postulate that phenotypic correlations less than |1| imply that individuals differ in how consistently they behave across contexts. The authors introduce a metric called 'syndrome deviation' that captures each individual's deviation from a perfect (i.e. $r=1$) correlation. The calculation of syndrome deviation requires that each behavioural trait is first rank transformed, and then each individual's value for syndrome deviation is calculated as the absolute difference between its ranks for the two behaviours. Syndrome deviation would thus equal zero for an individual that had the same rank for the two behaviours and equal, for example, three for an individual that ranked sixth on one and ninth on the other behavioural axis. The authors also propose that syndrome deviation might be a distinct phenotypic trait that can be heritable and thus shaped by natural selection. They further suggest that the study of selection acting on syndrome deviation would deepen our insight into the evolutionary mechanisms generating 'behavioural syndromes' (also called 'animal personality' in the recent behavioural ecology literature).

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In this commentary, we clarify that behavioural syndromes are not simply raw phenotypic correlations—which incorporate sources of within-individual variation, correlated measurement errors and other confounding factors (Roff 1997; Lynch and Walsh 1998). Instead, behavioural syndromes represent between-individual correlations (see Glossary in Dingemanse et al. 2010), which are solely due to between-individual effects (such as additive genetic, permanent environment or maternal correlations). Importantly, between-individual correlations can only be demonstrated by decomposing raw phenotypic correlations into between- and within-individual components, which we detail in this paper. We discuss how acknowledgement of this subtle difference between phenotypic and between-individual correlations affects the applicability of syndrome deviation for the study of behavioural syndromes. We also highlight multivariate tools that have been used by evolutionary biologists for decades in the study of syndromes (i.e. trait correlations) and their evolution and discuss the insights that these methods provide into evolutionary processes relative to the proposed alternative metric. Importantly, these multivariate tools are also accompanied by a wealth of complementing ecological and evolutionary theory that is extremely useful for behavioural syndrome research.

What is a behavioural syndrome?

In discussing Herczeg and Garamszegi's (2012) measure of syndrome deviation, it is important to first carefully and explicitly define what a behavioural syndrome actually represents. Herczeg and Garamszegi discuss behavioural syndromes in terms of raw phenotypic correlations and calculate syndrome deviation from the same basis. However, behavioural syndromes exist when the average phenotypes of individuals in one context/situation are correlated with the average phenotypes of the same individuals in a different context/situation such that populations harbour consistent individual variation in suites of correlated behaviours (Sih et al. 2004a, b; Réale et al. 2007). Statistically, behavioural syndromes, therefore, do not simply refer to the existence of a non-zero *phenotypic* correlation (contra Herczeg and Garamszegi 2012) but rather to a non-zero *between-individual* correlation (see Glossary in Dingemanse et al. 2010). We attribute this confusion between these two types of correlation to the intuitive—but erroneous—common assumption that, if a set of individuals is measured only once for a set of phenotypic traits, the trait correlations should also represent correlations between these individuals. This interpretation is typically not warranted for labile phenotypic traits, like behavioural ones. Instead, the relationship between the two types of correlation is given by (Eq. 1):

$$r_{P_y,P_z} = r_{ind_{0y},ind_{0z}} \sqrt{\left(\frac{v_{ind_{0y}}}{v_{ind_{0y}} + v_{e_{0y}}}\right)\left(\frac{v_{ind_{0z}}}{v_{ind_{0z}} + v_{e_{0z}}}\right)} + r_{e_{0y},e_{0z}} \sqrt{\left(\frac{v_{e_{0y}}}{v_{ind_{0y}} + v_{e_{0y}}}\right)\left(\frac{v_{e_{0z}}}{v_{ind_{0z}} + v_{e_{0z}}}\right)} \quad (1)$$

where r_{P_y,P_z}, $r_{ind_{0y},ind_{0z}}$ and $r_{e_{0y},e_{0z}}$ represent the phenotypic, between-individual and within-individual correlations, respectively; $v_{ind_{0y}}$ and $v_{ind_{0z}}$ are the between-individual variances, $v_{e_{0y}}$ and $v_{e_{0z}}$ represent the within-individual variances for behaviours y and z, respectively (cf. Snijders and Bosker 1999; see Appendix). Between-individual variances measure how much individuals differ from each other in their average phenotype, whereas within-individual variances measure how much repeated responses differ from each other within the same individual.

The different types of correlations, including the one that measures the strength of the behavioural syndrome $\left(r_{ind_{0y},ind_{0z}}\right)$, can be estimated whenever each individual within a sample is subjected to two or more trials, and whenever the behaviour of these individuals is measured in different contexts/situations within each of these trials (Box 3 in Dingemanse et al. 2010). For example, if each individual was subjected to an aggression and boldness test within each of two trials, the between- and within-individual variances (which are used for calculating repeatability; Nakagawa and Schielzeth 2010) can be estimated for both behaviours, as well as the phenotypic, between- and within-individual correlations (Eq. 1).

Biologically, between-individual correlations can only exist when both traits show between-individual variation; between-individual correlations have non-zero values when the average phenotypes of individuals for one trait (e.g. aggressiveness) are correlated with the average phenotypes of the same individuals for another trait (e.g. boldness). Equation 1 thus implies that a behavioural syndrome (i.e. $r_{ind_{0y},ind_{0z}} \neq 0$) can only exist when both behaviours show individual repeatability (i.e. $v_{ind_{0y}} > 0$ and $v_{ind_{0z}} > 0$) (see also Réale et al. 2007). Between-individual correlations are proximately underpinned by the effects of maternal, permanent-environment and genetic correlations between the traits (Dingemanse and Dochtermann 2012), the latter occurring either because of gene pleiotropy or linkage disequilibrium (Roff 1997). Similarly, within-individual correlations can only be present when both traits show within-individual variation; within-individual correlations have non-zero values when the change in behavioural expression in one trait (e.g. aggressiveness) correlates with the change in expression of another trait (e.g. boldness) within the same individual. This would, for

example, occur when the expression of both traits is underpinned by a common factor that varies within an individual, such as hunger level. Equation 1 thereby demonstrates that y and z can be phenotypically correlated in the absence of *any* between-individual (i.e. repeatable) variation in y and/or z strictly through the effects of a within-individual correlation. Hence, a simple phenotypic correlation does not necessarily provide insight into the presence of a behavioural syndrome.

Behavioural syndromes can readily be estimated by applying multivariate statistical tools (particularly multivariate mixed-effect models) to decompose phenotypic correlations into between- and within-individual components (Eq. 1) (for a worked example, see Wilson et al. 2011). With additional information about patterns of relatedness amongst individuals, the between-individual correlations that make up behavioural syndromes can be further broken up to explicitly estimate the contribution of additive genetic correlations, permanent environmental correlations and correlations due to maternal effects (e.g. Taylor et al. 2012). There is considerable empirical and theoretical research investigating these different components and, in particular, the evolutionary implications of additive genetic correlations (for an introduction into this literature, see Steppan et al. 2002).

Behavioural ecologists sometimes also infer behavioural syndromes from correlations between individual mean values of two phenotypic traits, the so-called 'phenotypic correlation of individual means' ($\bar{r}_{P_y,P_z}$) (cf. Snijders and Bosker 1999). However, in the Appendix, we demonstrate that $\bar{r}_{P_y,P_z}$, unfortunately, also represents a biased estimate of the between-individual correlation ($r_{ind_{0y}ind_{0z}}$) because it is also affected by the within-individual correlation ($r_{e_{0y},e_{0z}}$).We note further that methods have been proposed to infer $r_{ind_{0y},ind_{0z}}$ from the estimated values of $\bar{r}_{P_y,P_z}$ using information on trait-specific repeatabilities (Adolph and Hardin 2007), but those approaches are not generally applicable as they make the a priori assumption that $r_{e_{0y},e_{0z}}$ equals zero.

Calculating syndrome deviation

Having explicitly defined behavioural syndromes as between-individual correlations between behaviours, we discuss here how the metric syndrome deviation might be estimated empirically, while simultaneously introducing alternative approaches to facilitate the same objectives. Given that syndrome deviation is to be calculated with reference to a 'perfect' (Herczeg and Garamszegi 2012) between-individual (this paper) correlation, what one could do is estimate each individual's average phenotype for each phenotypic behaviour considered (e.g. by extracting each individual's best linear unbiased predictors; Henderson 1953) and thereby obtain an estimate of its multivariate behavioural type. Importantly, if such estimates were used for calculating an individual's value for syndrome deviation, one would have to account for the large uncertainty around each individual's estimated multivariate phenotype (see Hadfield et al. 2010 for a full discussion on this type of problem). Fortunately, modern statistical methods do allow for uncertainty to be taken forward into subsequent analyses, for example, by using Markov chain Monte Carlo methods and the posterior distributions of estimates (Ellison 2004). The calculation of syndrome deviation to study behavioural syndromes could be based on the mode and dispersion (i.e. uncertainty) of individual estimates of multivariate phenotypes from a Bayesian analysis. However, we are not aware of how the uncertainty around these estimates would be appropriately taken forward when syndrome deviation is based on rank-ordered variables. Taken together, the complex pattern of biological covariance that defines behavioural syndromes does not currently allow for the calculation of syndrome deviation.

Whereas syndrome deviation would be inherently difficult to calculate empirically, there is a suite of statistical approaches that might be useful in the context of the topic of what one might call 'behavioural canalisation' that the proposed metric by Herczeg and Garamszegi indirectly measures. For example, instead of asking how much individuals differ from a perfect correlation—a question lacking a clear biological rationale—one could instead ask how much individuals differ from the observed between-individual correlation. Deviation from the observed between-individual correlation has biological relevance, for example, because the relative level of cross-context behavioural consistency has been hypothesized to represent a fitness-indicator trait subject to sexual selection (Dall et al. 2004; Schuett et al. 2010). This could be done in the following way: first, one would apply the sampling design detailed above that would enable one to fit multivariate-mixed effect models such that the phenotypic variance–covariance matrix (**P**) would be partitioned into a between-individual (**I**) and within-individual (**R**) one (for guidelines on how to do so, see Dingemanse and Dochtermann 2012). Second, **I** could then be subjected to conventional matrix-rotation techniques (e.g. Schluter 1996; Houle 2001; Blows 2007), where the first eigenvector—which would reflect behavioural syndrome strength—would explain a major portion of the variation. An individual's score for the second eigenvector would represent its orthogonal deviation from the first one, which should very closely resemble how much the individual deviates from the observed behavioural syndrome structure. Importantly, behavioural researchers are already familiar with these approaches which are mathematically identical principal component extractions without post hoc rotations. As we have introduced above, modern statistical methods would

enable one to take uncertainty forward, such that each individual's estimate of syndrome deviation would have a corresponding confidence interval. Such an approach would notably very closely resemble those advocated by researchers studying phenotypic integration and canalisation (Houle 2001).

Multivariate selection and evolution

Herczeg and Garamszegi (2012) propose the use of syndrome deviation to study the evolution of behavioural syndromes, and that syndrome deviation itself might be an evolvable trait. With this suggestion, it becomes important to consider how the evolution of correlated traits and the evolution of correlations themselves have been addressed within evolutionary biology. Evolutionary biologists have long been interested in the adaptive nature of correlations between phenotypic traits, and a variety of methods have been proposed to study whether selection is multivariate—rather than univariate—in nature (reviewed by Brodie et al. 1995). The application of these approaches has greatly advanced our understanding of when natural selection favours associations between phenotypic traits (e.g. Sinervo and Svensson 2002). Evolutionary biologists typically quantify selection using the 'phenotypic selection approach' (Lande and Arnold 1983), which we illustrate here for a two-trait example by regressing relative fitness ($\hat{w}$) against two standardised traits ($\widetilde{y}$ and $\widetilde{z}$), as in Eq. 2 (e.g. Stinchcombe et al. 2008):

$$\widehat{w} = \beta_1 \widetilde{y} + \frac{1}{2}\gamma_{11}\widetilde{y}^2 + \beta_2 \widetilde{z} + \frac{1}{2}\gamma_{22}\widetilde{z}^2 + \gamma_{12}\widetilde{y}\widetilde{z} + c + e \qquad (2)$$

where β_1 and β_2 are direct linear selection gradients for traits 1 and 2, respectively; γ_{11} and γ_{22} are direct non-linear selection gradients (measuring quadratic effects for each trait separately, i.e. stabilising or disruptive forms of selection); γ_{12} is the direct non-linear selection gradient on combinations of the traits (therefore called 'correlational' selection gradient); c, the intercept, and e, the model's residual error. This classic approach continues to be used as it allows researchers to characterize many complex forms of selection, including single and multiple adaptive peaks and 'ridges of high fitness' in bivariate (or multivariate) phenotypic space (for graphical illustrations, see Brodie et al. 1995; Sinervo and Svensson 2002). For this reason, behavioural syndrome researchers have both suggested the use of the phenotypic selection approach (Dingemanse and Réale 2005; Réale and Dingemanse 2010) and applied it to study whether correlational selection (represented by γ_{12} in Eq. 2) favours suites of correlated behaviours (e.g. Eaves et al. 1990; Bell and Sih 2007; Adriaenssens and Johnsson 2012). In the context of behavioural syndrome research, such selection analyses should notably be modelled on **I** rather than **P**, where non-linear selection gradients at the between-individual level could readily be studied when using posterior estimates and distributions discussed above. Importantly, the phenotypic selection approach is firmly embedded in evolutionary biology because the resultant standardised estimates of the strength and shape of selection (so-called 'selection gradients') can subsequently be used to predict the evolutionary response to selection for traits with known heritability and genetic correlation structure (i.e. using the Lande and Arnold (1983) equation).

Herczeg and Garamszegi (2012) state that the study of an individual's deviation from a perfect correlation enables 'solid evolutionary inferences about correlated behaviours'. We have difficulty appreciating this strong statement for a number of reasons. First, what is the rationale underlying the interest in whether selection would act on the deviation from an arbitrary perfect correlation? As illustrated by Eq. 2, selection is typically modelled and viewed as acting directly on values of phenotypic traits that can, in some instances, have interacting effects on fitness. Furthermore, even if the true correlation was perfect (i.e. $r=1$), *estimates* of phenotypic correlations will always be less than one, because of extrinsic factors that are irrelevant to biological processes, such as measurement errors (Spearman 1904). Second, individuals that differ substantially in their bivariate (or multivariate) phenotype could have exactly the same value for syndrome deviation. This means that complex patterns of selection (e.g. saddle-shaped forms of selection on human personality traits; Eaves et al. 1990) would be completely missed when this metric was used for selection analysis. This would be problematic unless one assumes explicitly that syndrome deviation strictly represents a measure of behavioural canalisation (in which case, we suggest the application of the parametric variant that we introduced above). Third, heritable variation in syndrome deviation conflates heritable variation specific to one of the phenotypic traits (which would be due to trait-specific expression of genes) with heritable variation common to both phenotypic traits (which would be due to gene pleiotropy), making it difficult to infer how evolutionary change in this metric can be predicted or estimated. Fourth, the non-parametric nature of the metric hampers its implementation using quantitative evolutionary methods. Both the estimation of quantitative genetic parameters (e.g. additive genetic variance) and the quantification of the covariance between relative fitness and syndrome deviation require decomposition of 'phenotypic variance', information that is lost when using a non-parametric metric.

In short, we are of the opinion that the application of syndrome deviation would likely result in inferences that are difficult to interpret biologically. The laudable aim of Herczeg and Garamszegi to stimulate research into the question of whether selection acts on multivariate aspects of behavioural phenotypes, fortunately, can be facilitated by applying the mainstream evolutionary tools and modern statistical approaches detailed in this paper.

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Appendix: The relationships among phenotypic, between-individual and within-individual correlations

Using the symbols introduced in Eq. 1, the between-individual, within-individual and phenotypic correlations at the observation level are, respectively, defined as (Snijders and Bosker 1999; p. 204):

$$r_{ind_{0y},ind_{0z}} = \frac{cov_{ind_{0y},ind_{0z}}}{\sqrt{v_{ind_{0y}} v_{ind_{0z}}}},$$

$$r_{e_{0y},e_{0z}} = \frac{cov_{e_{0y},e_{0z}}}{\sqrt{v_{e_{0y}} v_{e_{0z}}}},$$

$$r_{P_y,P_z} = \frac{cov_{ind_{0y},ind_{0z}} + cov_{e_{0y},e_{0z}}}{\sqrt{(v_{ind_{0y}} + v_{e_{0y}})(v_{ind_{0z}} + v_{e_{0z}})}},$$

where $cov_{ind_{0y},ind_{0z}}$ and $cov_{e_{0y},e_{0z}}$ are the co-variances between the phenotypic attributes at the individual level (i.e. between individuals) and the observation level (i.e. within individuals), respectively. Therefore,

$$r_{P_y,P_z} = \frac{r_{ind_{0y},ind_{0z}} \sqrt{v_{ind_{0y}} v_{ind_{0z}}} + r_{e_{0y},e_{0z}} \sqrt{v_{e_{0y}} v_{e_{0z}}}}{\sqrt{(v_{ind_{0y}} + v_{e_{0y}})(v_{ind_{0z}} + v_{e_{0z}})}},$$

$$r_{P_y,P_z} = r_{ind_{0y},ind_{0z}} \frac{\sqrt{v_{ind_{0y}} v_{ind_{0z}}}}{\sqrt{(v_{ind_{0y}} + v_{e_{0y}})(v_{ind_{0z}} + v_{e_{0z}})}} + r_{e_{0y},e_{0z}} \frac{\sqrt{v_{e_{0y}} v_{e_{0z}}}}{\sqrt{(v_{ind_{0y}} + v_{e_{0y}})(v_{ind_{0z}} + v_{e_{0z}})}},$$

$$r_{P_y,P_z} = r_{ind_{0y},ind_{0z}} \sqrt{\left(\frac{v_{ind_{0y}}}{v_{ind_{0y}} + v_{e_{0y}}}\right)\left(\frac{v_{ind_{0z}}}{v_{ind_{0z}} + v_{e_{0z}}}\right)} + r_{e_{0y},e_{0z}} \sqrt{\left(\frac{v_{e_{0y}}}{v_{ind_{0y}} + v_{e_{0y}}}\right)\left(\frac{v_{e_{0z}}}{v_{ind_{0z}} + v_{e_{0z}}}\right)}$$

where the latter equation corresponds to Eq. 1.

The phenotypic correlation of individual means, which is also often used in behavioural syndrome research, can be approximated by the following (Snijders and Bosker 1999):

$$\bar{r}_{P_y,P_z} = \frac{cov_{ind_{0y},ind_{0z}} + cov_{e_{0y},e_{0z}}/n}{\sqrt{\left(v_{ind_{0y}} + \frac{v_{e_{0y}}}{n}\right)\left(v_{ind_{0z}} + \frac{v_{e_{0z}}}{n}\right)}},$$

$$\bar{r}_{P_y,P_z} = \frac{r_{ind_{0y},ind_{0z}} \sqrt{v_{ind_{0y}} v_{ind_{0z}}} + r_{e_{0y},e_{0z}} \sqrt{v_{e_{0y}} v_{e_{0z}}}/n}{\sqrt{\left(v_{ind_{0y}} + \frac{v_{e_{0y}}}{n}\right)\left(v_{ind_{0z}} + \frac{v_{e_{0z}}}{n}\right)}},$$

$$\bar{r}_{P_y,P_z} = r_{ind_{0y},ind_{0z}} \frac{\sqrt{v_{ind_{0y}} v_{ind_{0z}}}}{\sqrt{\left(v_{ind_{0y}} + \frac{v_{e_{0y}}}{n}\right)\left(v_{ind_{0z}} + \frac{v_{e_{0z}}}{n}\right)}} + \frac{r_{e_{0y},e_{0z}}}{n} \frac{\sqrt{v_{e_{0y}} v_{e_{0z}}}}{\sqrt{\left(v_{ind_{0y}} + \frac{v_{e_{0y}}}{n}\right)\left(v_{ind_{0z}} + \frac{v_{e_{0z}}}{n}\right)}},$$

$$\bar{r}_{P_y,P_z} = r_{ind_{0y}ind_{0z}} \sqrt{\left(\frac{v_{ind_{0y}}}{v_{ind_{0y}} + \frac{v_{e_{0y}}}{n}}\right)\left(\frac{v_{ind_{0z}}}{v_{ind_{0z}} + \frac{v_{e_{0z}}}{n}}\right)} + \frac{r_{e_{0y},e_{0z}}}{n} \sqrt{\left(\frac{v_{e_{0y}}}{v_{ind_{0y}} + \frac{v_{e_{0y}}}{n}}\right)\left(\frac{v_{e_{0z}}}{v_{ind_{0z}} + \frac{v_{e_{0z}}}{n}}\right)},$$

where the latter equation reveals that the phenotypic correlation of individual means is not only a function of the between-individual correlation ($r_{ind_{0y}ind_{0z}}$) but also of the within-individual correlation ($r_{e_{0y},e_{0z}}$). The extent to which $\bar{r}_{P_y,P_z}$ reflects $r_{ind_{0y}ind_{0z}}$ depends on the number of observations per individual (n). Typically, n is relatively low (2–5) in behavioural syndrome research, implying that $\bar{r}_{P_y,P_z}$ might often reflect $r_{e_{0y},e_{0z}}$ rather than the behavioural syndrome (i.e. $r_{ind_{0y}ind_{0z}}$) that was meant to be quantified by using this method.

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