bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

The role of trade-offs and feedbacks in shaping integrated plasticity and behavioral correlations

Ned A. Dochtermann
Department of Biological Sciences, North Dakota State University
ned.dochtermann@gmail.com

LAY SUMMARY
Correlations between behaviors are common but observed patterns of these correlations are, at least superficially, inconsistent with expectations of trade-offs. This mismatch is potentially resolved via feedbacks between behaviors and energy availability, suggesting important new research directions.

ABSTRACT
How behaviors vary among individuals and covary with other behaviors has been a major topic of interest over the last two decades. Unfortunately, proposed theoretical and conceptual frameworks explaining the seemingly ubiquitous observation of behavioral (co)variation have rarely successfully generalized. Two observations perhaps explain this failure: First, phenotypic correlations between behaviors are more strongly influenced by correlated and reversible plastic changes in behavior than by "behavioral syndromes". Second, while trait correlations are frequently assumed to arise via trade-offs, the observed pattern of correlations is not consistent with simple pair-wise trade-offs. A possible resolution to the apparent inconsistency between observed correlations and a role for trade-offs is provided by state-behavior feedbacks. This is critical because the inconsistency between data and theory represents a major failure in our understanding of behavioral evolution. These two primary observations emphasize the importance of an increased research focus on correlated reversible plasticity in behavior—frequently estimated and then disregarded as within-individual covariances.

KEY WORDS
behavioral syndromes, integrated plasticity, state dependence, trade-off, feed-back

DATA AND MODEL AVAILABILITY
Model code, as well as the data associated with Figures 2 & 3, are available at github.com/DochtermannLab/FeedbacksModel. Both code and data will be made available at Dryad if accepted.

ACKNOWLEDGMENTS
The author thanks B. Hedinger, R. Royauté, J. Saltz, and D. Westneat for helpful discussions. The author also thanks two anonymous for critical and helpful feedback. This feedback improved the clarity and accuracy of the manuscript. This work was funded by NSF-IOS 1557951

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

INTRODUCTION

Despite almost two decades of intensive research effort, overarching explanations for why behavioral responses are frequently correlated remain elusive. Here, I argue that this stems from two key empirical findings that have not been fully appreciated. First, behavioral correlations are more strongly influenced by within-individual correlations than they are by behavioral syndromes (Table 1A, Box1). Second, bivariate trade-offs are an insufficient explanation for—and inconsistent with—observed behavioral correlations. Instead, observed correlations are consistent with a combination of feedbacks (positive and negative) between behaviors and states.

1. Understanding how among and within individual variation contribute to behavioral correlations

Much of the research examining behavioral correlations over the last two decades has focused on among-individual correlations, i.e., "behavioral syndromes" (Figure 1A & 1B, Box 1; Sih et al. 2004a, Sih et al. 2004b, Dingemanse et al. 2010, Dingemanse et al. 2012). This interest has been reasonable because behavioral syndromes connect to both underlying genetic correlations and developmental processes. As such, behavior correlations can directly influence evolutionary outcomes (Dochtermann and Dingemanse 2013, Royauté et al. 2020). This clearly embeds behavioral syndrome research into the broader realms of evolutionary biology. Unfortunately, this focus on syndromes ignores the fact that behavioral correlations are, instead, more strongly influenced by within-individual correlations (Box 1, Figure 1, Table 1A). This has led to a disconnect between research effort and biology.

Within-individual correlations represent the occurrence of correlated *changes* in behaviors at the level of an individual. For example, a negative within-individual correlation between aggression and exploratory propensity would mean that when an individual increases its aggression, it also decreases its activity (Figure 1). This correlated change can occur even if, on average, more aggressive individuals are also more active (Figure 1).

That behavioral correlations at the phenotypic level are more strongly influenced by within-individual correlations than by behavioral syndromes is a necessary conclusion given observed behavioral repeatabilities. Due to the mathematical relationship of a phenotypic correlation to its constituent parts (Box 1), whenever repeatability is less than 0.5, the phenotypic correlation is more strongly influenced by within-individual correlations than among-individual correlations (Dingemanse et al. 2012, Dingemanse and Dochtermann 2013). Bell et al.'s (2009) meta-analysis of repeatabilities—a keystone

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

contribution to our understanding of behavioral variation—found that the average repeatability of behaviors was about 0.4. Consequently, behavioral correlations are, on average, most strongly influenced by within-individual correlations (Box 1).

Within-individual correlations include—but are not limited to (see Table 1B)—correlated plastic responses to temporary environmental variation; specifically, responses to unmeasured or unmodeled environmental variation. This correlated reversible plasticity is akin to "phenotypic flexibility" (Piersma and Van Gils 2011) but correlated across multiple behaviors. Despite more strongly influencing behavioral correlations than do behavioral syndromes (Box 1), within-individual correlations have received much less direct theoretical or empirical attention from behavioral researchers.

Box 1. The relationship of behavioral syndromes and reversible plasticity to behavioral correlations: the primacy of correlated reversible plasticity

Behavioral correlations at the population and phenotypic level (r_p) emerge from the joint contributions of among-individual correlations, i.e. behavioral syndromes (r_A), and within-individual correlations (r_w, Figure 1; (Dingemanse et al. 2012)). The relative influence of each of these on observed correlations is mediated by the repeatability of the behaviors of interest (τ_1 and τ_2):

$$r_p = r_A\sqrt{\tau_1\tau_2} + r_w\sqrt{(1-\tau_1)(1-\tau_2)}$$

r_A is the statistical definition of a behavioral syndrome (Dingemanse et al. 2012) and, as repeatability decreases, the contribution of behavioral syndromes to phenotypic correlations necessarily decreases. At the extreme, if τ for either behavior is 0, then r_p is entirely determined by the r_w. Moreover, r_w contributes more strongly to r_p than does r_A when geometric mean repeatability is less than 0.5 (Dingemanse and Dochtermann 2013). Based on meta-analysis (Bell et al. 2009) we know that average τs are around 0.4 and, consequently, r_w, on average influences r_p more strongly than does r_A. Put another way, within-individual correlations have an average 1.5 times greater influence on phenotypic correlations than do behavioral syndromes.

Importantly, r_w includes correlated reversible plasticity (Figure 1), though this plasticity will not necessarily be adaptive and errors both in the response to cues by organisms or in measurement also contribute to r_w (Table 1).

2. Interpreting patterns of correlations and indications, or lack thereof, of a role for trade-offs

A possible factor shaping correlated plastic responses are trade-offs in investment of energy (or other resources) into behaviors. For example, energy used during agonistic interactions is not available for exploring new areas. Accordingly, we would expect that as individuals increase investment in one behavior, they decrease investment in another (Figure 1A), generating negative within-individual correlations (Figure 1C). If individuals differ in their ability to acquire resources, this results in the familiar "big house, big car" scenario from life-history theory (Van Noordwijk and de Jong 1986, Reznick et al. 2000): high variation in acquisition (relative to variation in allocation) results in a positive among-individual correlation despite underlying trade-offs. However, because behavioral researchers frequently change the sign of behavioral measures to aid in the ecological

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

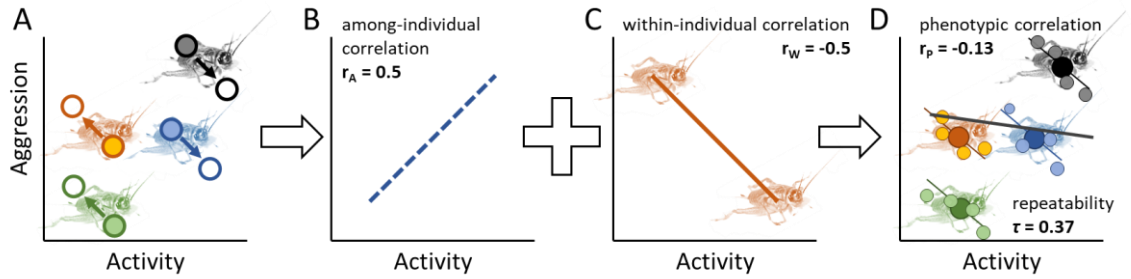

Figure 1. Example of how behavioral syndromes, i.e. among-individual correlations (r_A) and correlated plasticity, i.e. within-individual correlations (r_W), additively contribute to phenotypic correlations. A) Activity and aggressive behavior of four crickets are measured on two occasions (1 & 2). Crickets largely maintain their relative differences in each behavior (i.e. "personality") *and* their behavior changes in a correlated manner (correlated plasticity). B) The relationship *among* crickets is due to a positive among-individual correlation (dashed blue line, r_A). C) Behavioral plasticity is negatively correlated (within-individual correlation: solid orange line, r_W): if an *individual* increases its activity, it decreases its aggression. D) The among- and within-individual correlations additively determine average individual behaviors (dark circles) and how crickets vary from their averages (colored lines and smaller, lighter, circles). The influence of either r_A or r_W on the phenotypic correlation (solid black line in D) is determined by the repeatabilities of the behaviors. If repeatability is high (> 0.5), this phenotypic correlation is primarily determined by the behavioral syndrome, r_A (Box 1). Here, with a repeatability of 0.37 (Bell et al. 2009), the phenotypic correlation is more strongly influenced by the within-individual correlation than the among-individual correlation but is ultimately quite modest.

interpretation of behavioral assays, it is not the case that among-individual correlations are strictly expected to be positive and within-individual correlations be negative. Instead, if trade-offs underpin behavioral correlations, we would expect among-individual and within-individual correlations to be of *opposite signs* (Figure 1D; Downs and Dochtermann 2014).

Meta-analyses have examined behavioral correlations at the genetic, among-individual, and within-individual levels (Dochtermann 2011, Brommer and Class 2017). These analyses have demonstrated that correlations across levels are generally concordant as to their signs and magnitudes (Figure 2). As suggested above, this concordance of signs suggests either a *potential* lack of trade-offs in the expression of behaviors or that variation in acquisition is low relative to variation in allocation. Of course, it is also possible that many of the analyzed behaviors should not be expected to trade-off due to naming fallacies obscuring that the same behavior is actually being measured in different ways (Carter et al. 2013) or because limiting resources differ between behaviors. Moreover, because phenotypes consist of many, many behaviors and other traits, any trade-offs would not typically be expected to exist solely between two traits. Nonetheless, the results in Figure 2 suggest that bivariate trade-offs alone are *insufficient* to explain patterns of among- and within-individual correlations alone.

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

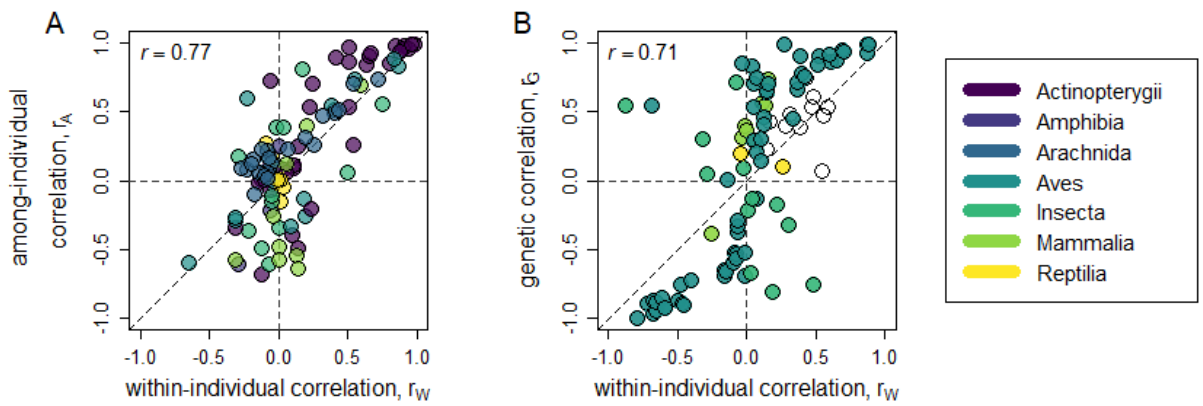

Figure 2. Relationship of within-individual behavioral correlations (r_W) with (A) among-individual correlations (r_A) and (B) genetic correlations (r_G). The sign and magnitude of r_A and r_G are highly concordant with r_W across behaviors and taxa. Diagonal lines indicate 1:1 relationship. Horizontal and vertical dashed lines divide plots into sign mismatches (top left, bottom right) and sign matches (top right, bottom left). Within-individual correlations were the same sign as among-individual and genetic correlations in 62% and 79% of cases respectively. Data in A are from Brommer and Class (2017). Data in B are from Dochtermann (2011).

3. Feedbacks as a potential component influencing behavioral correlations

An alternative mechanism for understanding behavioral correlations builds on a simple model of trade-offs by adding feedbacks between underlying state and behaviors. Sih et al. (2015) proposed that among-individual differences in behavior—i.e., "personality"—can be generated by feedbacks between behavior and underlying state. Specifically, Sih et al. (2015) proposed that positive feedbacks can explain the emergence of personality variation. This was supported by a model where the intensity of initial behavioral expression resulted in feedbacks that then shaped an individual's future behavioral expression.

To determine whether feedbacks similarly produce both behavioral syndromes and within-individual correlations, I extended the Sih et al. (2015) model to two traits following Houle's (1991) classic y-model (Figure 3A). The y-model is a simple representation of trade-offs where "state" or some other proxy for energy (S_1) is allocated to expression of one of two traits (B_1 or B_2, Figure 3A). Because energy allocated to one trait at a particular time ($TO \times S_{1,t}$) is not available for allocation to the other trait ($(1\text{-}TO) \times S_{2,t}$), a trade-off exists between them.

Following this framework, I modeled the expression of two behaviors (B_1 and B_2) as emerging from the investment of energy (based on "state") into each, with the amount of energy invested trading off between behaviors. In this model the two behaviors subsequently affect the gain of state/resources for use in the future expression of behavior

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

via feedbacks (λ, the proportion of energy added back to S_1, Figure 3A). Biologically, such feedbacks might arise if, for example, exploratory propensity is positively correlated with energy gain. In such a case, individuals with an initially high state can explore more, gaining more energy that can be invested in even greater exploration and energy gain in the future—a positive feedback.

I modeled varied magnitudes of feedbacks, both positive and negative, and analyzed modeled populations to determine resulting the among- and within-individual correlations (Figure 3B). At the start of the model, state differed among individuals due to stochastic variation. This seeded the population with variation in initial acquisition. In natural populations, individuals may similarly initially differ due to developmental stochasticity, parental investment, genetic differences, and other environmental factors.

In the absence of feedbacks ($\lambda_1 = \lambda_2 = 0$, Figure 3), this model reduces to the general structure of models developed by Houle (1991) and Van Noordwijk and de Jong (1986). Consequently, without feedbacks, the trade-off in allocation along with underlying

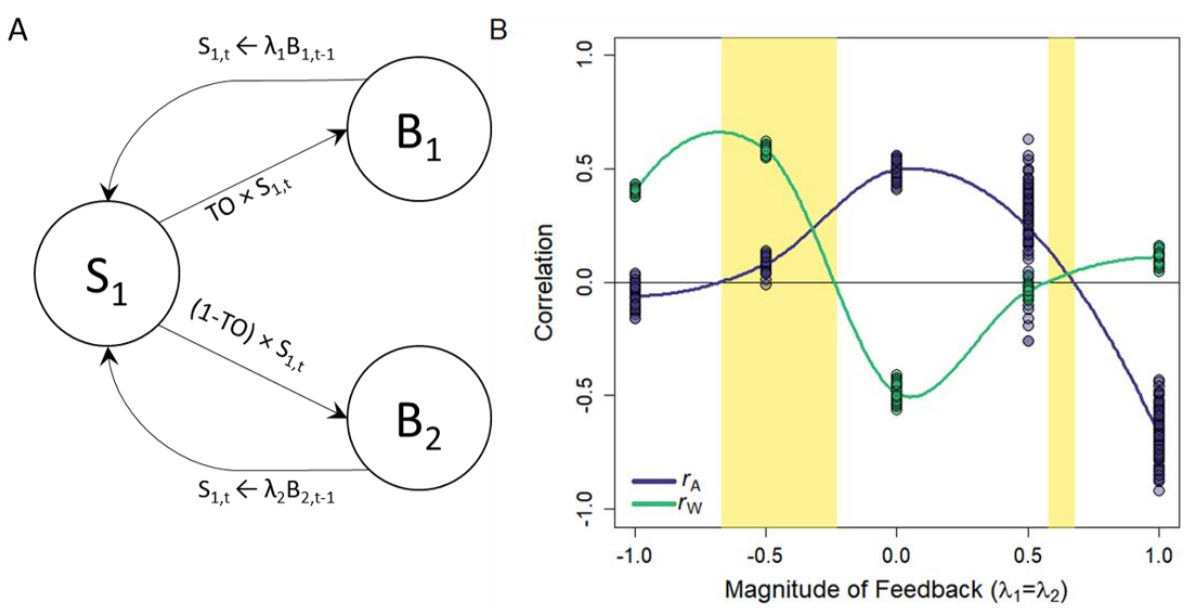

Figure 3. (A) Simple model structure combining Sih et al.'s (2015) feedback model with Houle's (1991) y-model to include feedbacks between behavior and state. TO (the trade-off parameter) is the proportion of state energy (S_1 at time t) allocated to behavior B_1, making 1-TO the energy allocated to B_2. λ is the feedback strength and the conversion rate of a behavior (B_1 or B_2) into energy that can be used at a later time. (B) Magnitude of among- (purple) and within-individual (green) correlations under feedbacks (λ) of different strength. Shaded regions indicate those feedback strengths that produce correlations of the same sign. Repeatability is not set *a priori* and is instead an emergent property of the model. Individuals within a population expressed behaviors according to A over ten time steps, with 250 individuals per population. Fifty populations were simulated at each of five levels of feebacks (λ in B). r_A and r_w were estimated using the `MCMCglmm` package in R (Hadfield 2010).

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

variation in initial acquisition results in positive among-individual (r_A) and negative within-individual correlations (r_w, Figure 3B), i.e., negatively correlated plasticity plus different signs for the two correlations. In this case, within-individual variation is generated by stochasticity in how much is invested in one behavior and thus how much is available for the other. For example, individuals might differentially allocate energy to growth versus activity at different periods of their life.

In contrast, with both negative and positive feedbacks (negative and positive values of λ respectively), the within-individual correlation becomes increasingly positive (Figure 3B). Simultaneously, negative and positive feedbacks lead to a reduction in among-individual correlations, with these correlations becoming negative and trade-offs becoming particularly apparent at the among-individual level with strong positive feedback. Importantly, this model identifies both positive and negative feedbacks in two disjunct ranges that result in among-individual correlations and integrated plasticity of the same sign (shaded regions of Figure 3B). This suggests that feedbacks might partially explain the patterns of correlations observed in Figure 2.

The potential for state-behavior feedbacks combined with trade-offs to explain observed patterns of correlations adds to several prior models that suggested state might generate consistent individual differences and phenotypic behavioral correlations (Dall 2004, Wolf et al. 2007, Luttbeg and Sih 2010, Dall et al. 2012). However, these prior models did not separately examine among- and within-individual correlations nor the relationship between them. State-behavior feedbacks are also implied in existing conceptual frameworks, such as the pace-of-life syndrome hypothesis (Réale et al. 2010), though support for such overarching explanations remains elusive (e.g. Niemelä and Dingemanse 2018, Royauté et al. 2018).

4. Conclusions

Given that within-individual correlations more strongly influence observed correlations between behaviors than do behavioral syndromes, the mismatch in effort to understand these correlations over the last fifteen years has hindered our general understanding of behavioral correlations. While considerably more investigation is required, the simple model presented here suggests that feedbacks might play a role in shaping both behavioral syndromes and integrated plasticity. This potential for feedbacks to shape correlations can be empirically addressed via manipulation of food availability (e.g. MacGregor et al. 2021), manipulating population densities, and longitudinal measurements of both state and behavior (Sih et al. 2015). Importantly, within-individual correlations can only be estimated with specific study designs (Dingemanse and

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

Dochtermann 2013). Regardless, whether focused on feedbacks or other potential causes, our understanding of behavioral correlations can only advance if more—and careful—attention is paid to within-individual correlations.

REFERENCES CITED

Bell, A. M., S. J. Hankson, and K. L. Laskowski. 2009. The repeatability of behaviour: a meta-analysis. Animal Behaviour **77**:771-783.

Berdal, M. A., and N. A. Dochtermann. 2019. Adaptive alignment of plasticity with genetic variation and selection. Journal of Heredity **110**.

Brommer, J. E., and B. Class. 2017. Phenotypic correlations capture between-individual correlations underlying behavioral syndromes. Behavioral Ecology and Sociobiology **71**:50.

Carter, A. J., W. E. Feeney, H. H. Marshall, G. Cowlishaw, and R. Heinsohn. 2013. Animal personality: what are behavioural ecologists measuring? Biological Reviews **88**:465-475.

Dall, S. R. X. 2004. Behavioural biology: Fortune favours bold and shy personalities. Current Biology **14**:R470-R472.

Dall, S. R. X., A. M. Bell, D. I. Bolnick, and F. L. W. Ratnieks. 2012. An evolutionary ecology of individual differences. Ecology Letters **15**:1189-1198.

Dingemanse, N. J., and N. A. Dochtermann. 2013. Quantifying individual variation in behaviour: mixed-effect modelling approaches. Journal of Animal Ecology **82**:39-54.

Dingemanse, N. J., and N. A. Dochtermann. 2014. Individual behaviour: behavioural ecology meets quantitative genetics.*in* A. Charmantier, D. Garant, and L. E. B. Kruuk, editors. Quantitative genetics in the wild. Oxford University Press.

Dingemanse, N. J., N. A. Dochtermann, and S. Nakagawa. 2012. Defining behavioural syndromes and the role of "syndrome" deviation in understanding their evolution. Behavioral Ecology and Sociobiology **66**:1543-1548.

Dingemanse, N. J., A. J. N. Kazem, D. Réale, and J. Wright. 2010. Behavioural reaction norms: animal personality meets individual plasticity. Trends in Ecology & Evolution **25**:81-89.

Dochtermann, N. A. 2011. Testing Cheverud's conjecture for behavioral correlations and behavioral syndromes. Evolution **65**:1814-1820.

Dochtermann, N. A., and N. J. Dingemanse. 2013. Behavioral syndromes as evolutionary constraints. Behavioral Ecology **24**:806-811.

Downs, C. J., and N. A. Dochtermann. 2014. Testing hypotheses in ecoimmunology using mixed models: disentangling hierarchical correlations. Integrative And Comparative Biology **54**:407-418.

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

Hadfield, J. D. 2010. MCMC Methods for Multi-Response Generalized Linear Mixed Models: The MCMCglmm R Package. Journal of Statistical Software **33**:1-22.

Houle, D. 1991. Genetic covariance of fitness correlates--what genetic correlations are made of and why it matters. Evolution **45**:630-648.

Luttbeg, B., and A. Sih. 2010. Risk, resources and state- dependent adaptive behavioural syndromes. Philosophical Transactions of the Royal Society B-Biological Sciences **365**:3977-3990.

MacGregor, H. E., A. Cottage, and C. C. Ioannou. 2021. Suppression of personality variation in boldness during foraging in three-spined sticklebacks. Behavioral Ecology and Sociobiology **75**:1-10.

Niemelä, P. T., and N. J. Dingemanse. 2018. Meta-analysis reveals weak associations between intrinsic state and personality. Proceedings of the Royal Society B: Biological Sciences **285**:20172823.

Piersma, T., and J. Drent. 2003. Phenotypic flexibility and the evolution of organismal design. Trends in Ecology & Evolution **18**:228-233.

Piersma, T., and J. A. Van Gils. 2011. The flexible phenotype: a body-centred integration of ecology, physiology, and behaviour. Oxford University Press.

Réale, D., D. Garant, M. M. Humphries, P. Bergeron, V. Careau, and P. O. Montiglio. 2010. Personality and the emergence of the pace-of-life syndrome concept at the population level. Philosophical Transactions of the Royal Society B-Biological Sciences **365**:4051-4063.

Reznick, D., L. Nunney, and A. Tessier. 2000. Big houses, big cars, superfleas and the costs of reproduction. Trends in Ecology & Evolution **15**:421-425.

Royauté, R., M. A. Berdal, C. R. Garrison, and N. A. Dochtermann. 2018. Paceless life? A meta-analysis of the pace-of-life syndrome hypothesis. Behavioral Ecology and Sociobiology **72**:64.

Royauté, R., A. Hedrick, and N. A. Dochtermann. 2020. Behavioural syndromes shape evolutionary trajectories via conserved genetic architecture. Proceedings of the Royal Society B **287**:20200183.

Sih, A., A. Bell, and J. C. Johnson. 2004a. Behavioral syndromes: an ecological and evolutionary overview. Trends in Ecology & Evolution **19**:372-378.

Sih, A., A. M. Bell, J. C. Johnson, and R. E. Ziemba. 2004b. Behavioral syndromes: An integrative overview. Quarterly Review of Biology **79**:241-277.

Sih, A., K. J. Mathot, M. Moirón, P.-O. Montiglio, M. Wolf, and N. J. Dingemanse. 2015. Animal personality and state–behaviour feedbacks: a review and guide for empiricists. Trends in Ecology & Evolution **30**:50-60.

Van Noordwijk, A. J., and G. de Jong. 1986. Acquisition and allocation of resources: their influence on variation in life history tactics. American Naturalist:137-142.

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

Westneat, D. F., J. Wright, and N. J. Dingemanse. 2015. The biology hidden inside residual within-individual phenotypic variation. Biological Reviews **90**:729-743.

Whitman, D., and A. Agrawal. 2009. What is phenotypic plasticity and why is it important? Phenotypic plasticity of insects: mechanisms and consequences:1-63.

Wolf, M., G. S. van Doorn, O. Leimar, and F. J. Weissing. 2007. Life-history trade-offs favour the evolution of animal personalities. Nature **447**:581-584.

bioRxiv preprint doi: https://doi.org/10.1101/2021.07.26.453877; this version posted January 24, 2023. The copyright holder for this preprint (which was not certified by peer review) is the author/funder, who has granted bioRxiv a license to display the preprint in perpetuity. It is made available under aCC-BY-NC-ND 4.0 International license.

Table 1. A. Parameters characterizing behavioral associations. B. Some possible contributors to within-individual behavioral correlations. If either active or passive plasticity are elicited in response to known and measured environmental parameters, they can be explicitly modeled. Otherwise, they will contribute to within-individual variation and covariation. Similar processes contribute to the irreversible plasticity (aka developmental plasticity), which contributes to among-individual correlations (Dingemanse and Dochtermann 2014). The contribution of each component of within-individual correlations can be determined by breaking r_w down into constituent parts, similar to how a phenotypic correlation was broken down in Box 1.

A.	Also known as	Definition	
Phenotypic correlations		The standardized covariance between two behaviors. Describes the strength and direction of association.	(Sih et al. 2004a, Sih et al. 2004b, Dingemanse et al. 2010)
Among-individual correlations	Behavioral syndromes	Correlations in an individual's behavioral responses	(Dingemanse et al. 2012, Dingemanse and Dochtermann 2013)
Within-individual correlations	Residual correlations	Correlations in *changes* in an individual's responses	
B.	**Definition**		
Active reversible plasticity	changes in an individual's behavior expressed in response to environmental cues indicative of selective pressures		(Piersma and Drent 2003, Piersma and Van Gils 2011)
Passive reversible plasticity	changes in response to environmental conditions rather than specific cues of selective pressures; includes passive responses to abiotic conditions, such as hypoxia		(Whitman and Agrawal 2009)
Reversible organismal error	changes to an organism's phenotype in response to an incorrectly processed cue		(Westneat et al. 2015)
Measurement error	error in quantification of behaviors; can be correlated due to bias an		(Westneat et al. 2015)

for a discussion of additional contributors to within-individual (co)variation see: (Piersma and Drent 2003, Whitman and Agrawal 2009, Piersma and Van Gils 2011, Westneat et al. 2015, Berdal and Dochtermann 2019)