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Raphaël Royauté, Monica Anderson Berdal, Courtney Garrison, Ned Dochtermann

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Paceless life? A meta-analysis of the pace-of-life syndrome hypothesis

Raphaël Royauté [a]

Monica Anderson Berdal [a]

Courtney R. Garrison [a]

Ned A. Dochtermann [a, b]

[a] Department of Biological Sciences; North Dakota State University

[b] corresponding author: ned.dochtermann@gmail.com

Abstract

The pace-of-life syndrome hypothesis predicts that individual differences in behaviour should integrate with morphological, physiological, and life-history traits along a slow to fast pace-of-life continuum. For example, individuals with a "slow" pace-of-life are expected to exhibit a slower growth rate, delayed reproduction, longer lifespans, have stronger immune responses, and are expected to avoid risky situations relative to "fast" individuals. If supported this hypothesis would help resolve ecological and evolutionary questions regarding the origin and maintenance of phenotypic variation. Support for the pace-of-life syndrome hypothesis has, however, been mixed. Here we conducted a meta-analysis of 42 articles and 179 estimates testing the pace-of-life syndrome hypothesis as it applies to the integration of behaviours with physiological or life-history traits. We found little overall support for the pace-of-life syndrome hypothesis with the mean support estimated as r = 0.06. Support for the pace-of-life syndrome hypothesis was significantly higher in invertebrates (r = 0.23) than vertebrates (r = 0.02) and significantly higher when based on phenotypic (r = 0.10) versus genetic correlations (r = -0.09). We also found that females exhibited correlations between behaviour and life-history and physiology that were opposite the predictions of the pace-of-life syndrome hypothesis (r = -0.16) and that these correlations significantly differed from those observed in males (r = 0.01) or males and females pooled (r = 0.12). It was also the case that there was little support for the hypothesis when life-history and physiological traits were independently analysed (behaviour × life-history: r = 0.12; behaviour × physiology: r = 0.04). Exploratory post-hoc analyses revealed that correlations of behaviour with growth rate and hormone levels were more likely to show support for the predictions of the pace-of-life syndrome hypothesis.

The lack of overall support found in our analyses suggests that general assertions regarding phenotypic integration due to “pace-of-life” and should be re-evaluated.

Significance Statement

The pace-of-life syndrome hypothesis has been proposed as an overall organizational framework for the integration of behavioural, life-history, and physiological traits. This hypothesis provides potentially profound insights into how and why phenotypic traits might covary and why phenotypic variation may be maintained within populations. Over the last seven years this organizational framework has been intensively investigated as it pertains to relationships between behaviour and other traits. Here we conducted an overall analysis of whether the hypothesis was supported. Despite considerable research investment across behavioural ecology, we did not find that available data supported the pace-of-life syndrome hypothesis. This suggests that either the hypothesis has been inappropriately tested or is not generally applicable.

Keywords

Personality, behavioural syndrome, covariation, phenotypic integration

1. Introduction

Explaining the emergence and maintenance of phenotypic variation is a major topic in evolutionary biology. However, understanding phenotypic variation in either regard is complex because this variation is expressed at multiple levels (individual, population, species) and bridges multiple phenotypic domains (e.g. life-history, physiology, behaviour). One of the most comprehensive conceptual models that integrates variation in life-history, physiology, and behaviour is the "pace-of-life" syndrome hypothesis (Ricklefs and Wikelski 2002). According to this hypothesis, individuals with a "slow" pace-of-life exhibit relatively slow growth rates, reach sexual maturity later in life and have long life spans. These life-history strategies are correlated with a set of physiological responses—including slower metabolic rates, slower stress reactivity and higher immune response—which in turn are correlated with behavioural responses. "Slow" individuals are predicted to minimize risk-taking and behave more cautiously when facing threats from predators or conspecifics (Réale et al. 2010). In contrast, individuals on a "fast" pace-of-life, are expected to adopt a "live fast-die young" strategy and are less risk averse due to higher metabolic rates and lower stress reactivity.

Originally proposed to explain variation across species or populations, this hypothesis has more recently been expanded to apply to variation among individuals *within* populations (Réale et al. 2010). Réale et al. (2010) proposed that the hypothesis might underlie the general observation that populations contain considerable behavioural variation and that individuals behave in predictably (i.e. consistently) different ways from one another. Extending the hypothesis to behaviour makes the implicit assumption that among-individual differences in behaviour (Réale et al. 2007) are correlated with among-

individual differences in physiology and life-history along a slow-fast pace-of-life axis. This integration across traits is expected because: 1) individuals allocate energy to different functions based on limited resources, thus generating trade-offs and resulting in trait integration (Ricklefs and Wikelski 2002), 2) correlational selection is common in nature and will maintain trait integration when combinations of trait values yield high fitness (Sinervo and Svensson 2002) and, 3) because common physiological pathways and pleiotropic genes are involved in the regulation and expression of life-history and behaviours (Sih et al. 2004).

One of the most appealing aspects of the pace-of-life syndrome hypothesis is that it produces explicit predictions regarding how behaviour should be organized given underlying life-history and physiology (see Fig. 1 in Réale et al. 2010). For example, aggressive or exploratory individuals are predicted to acquire resources at a faster rate, which may in turn select for faster growth rates (Réale et al. 2010). However, despite this intuitive conceptual framework, there have been few mathematical treatments of whether and how behaviours should covary with physiology and life-history (Wolf et al. 2007; Engqvist et al. 2015; reviewed in Mathot and Frankenhuis 2018). Further, despite the number of conceptual papers arguing for the pace-of-life syndrome hypothesis as an organizational basis for the integration of behaviour with life-history and physiology, empirical support for behaviour has been mixed (e.g. Careau et al. 2015; Mathot et al. 2015). While some studies have found full support for the predictions stemming from the pace-of-life syndrome hypothesis (Careau et al. 2010), others have found only partial support (Krams et al. 2014a), context dependent support (Mathot et al. 2015), no overall association among life-history, physiology and behavioural traits (Galliard et al. 2013;

Royauté et al. 2015a), or correlations in directions opposite those predicted (Adriaenssens and Johnsson 2010). This contradicting evidence leads to the general question of whether behaviour is indeed integrated with other phenotypic domains according to slow and fast pace-of-life strategies.

Here we tested whether overall support for the predictions of the pace-of-life syndrome hypothesis exists. Specifically we aimed to (i) estimate the global effect size for the pace-of-life syndrome hypothesis, (ii) test whether support for the pace-of-life syndrome hypothesis differed between males and females, between ectotherms and endotherms, invertebrates and vertebrates, laboratory and field studies, life-stages, or level of inference (genetic, among-individual, or phenotypic) and, (iii) perform exploratory tests of whether some categories of estimates within life-history or physiology had stronger associations with behaviours.

2. Methods

(a) Data collection

To test for overall support of the pace-of-life syndrome hypothesis, we gathered estimates of the relationship between life-history and behaviour or physiology and behaviour by searching 15 leading journals in behavioural and evolutionary ecology in ISI Web of Science on April 27th 2016, using the key words: 'pace-of-life' or 'pace of life'. The journals included in our search were: *American Naturalist, Behavioral Ecology, Behavioral Ecology and Sociobiology, Animal Behaviour, Behaviour, Ethology, Evolution, Journal of Evolutionary Biology, Journal of Animal Ecology, Ecology, Oikos, Oecologia, Proceedings of the Royal Society B* and *Ecology Letters*. We only included articles that were published after 1990. This search yielded a total of 41 articles. Four recently published articles from 2015 and

2016 that did not appear in our initial search were later added. We also included 23 articles from a broader search using the terms 'personality AND pace of life' or 'personality AND pace-of-life' and not restricted to any specific journal. We used this broader search because the Réale et al. (2010) framework was explicitly applied to the study of behavioural variation under the label of "personality variation". To these we added 14 additional articles identified by Royauté et al. (2015a), which included a short review of the empirical support for the pace-of-life syndrome hypothesis. Of this initial list of 82 articles, we excluded all articles that did not include at least one behavioural trait discussed in the initial article by Réale et al. (2010) or articles that included only comparisons across but not within species. This led to a final list of 46 articles and 184 estimates of the correlation between behaviour and life-history or behaviour and physiology that fit the above criteria (Fig. S1).

(b) Data analysis

All analyses were conducted in R version 3.4.2 using the `metafor` package for mixed effect models (Viechtbauer 2010). After converting published effect sizes to *r* (following Nakagawa and Cuthill 2007), we adjusted the sign of effect size estimates so that positive values indicated support for the pace-of-life syndrome hypothesis, while negative values indicated relationships in a direction opposite to that predicted by the model. Only five estimates did not contain enough information to convert to effect size and these were removed prior to analysis, leading to a final data set consisting of 179 estimates from 42 studies. Because correlation coefficients are not normally distributed, we then applied Fisher's *z*-transformation (*Zr*) prior to all analyses and back-transformed estimates to correlation coefficients (*r*) for interpretation.

We used a phylogenetic meta-regression model with an intercept but no moderators to estimate the global effect size for the pace-of-life syndrome hypothesis. Article identity and the species to which the study organism belonged were included as random effects and modelled evolutionary nonindependence based on a phylogenetic tree built using phyloT (Letunic 2015). We report the intercept as the global effect size for the pace-of-life syndrome hypothesis and base our statistical inference on the overlap of the 95% confidence interval with 0. We calculated the heterogeneity in the dataset (I^2) as the variance explained by all random effects (among- and within-article variance and species variance) over the total amount of variation, which included sampling variance (calculated after Nakagawa and Santos 2012). We repeated this procedure separately with the data divided into subsets for behaviour × life-history or behaviour × physiology correlations.

Next we tested whether support for the pace-of-life syndrome hypothesis differed among *a priori* selected biological categories (i.e., moderators). To do so, we conducted a series of meta-regressions this time testing for the effects of the following moderators: sex (male, females, or mixed), thermoregulation type (ectotherms or endotherms), correlation type (behaviour × life-history or behaviour × physiology), whether the study organism was an invertebrate or a vertebrate, whether the study was laboratory or field-based, life-stage of measured individuals (juveniles, adults or mixed), the level of inference (genetic, among-individual, or phenotypic), and sample size (centered around the median). As some combinations of *a priori* categories had few estimates and uneven sample sizes (Table 1), each moderator was tested in a separate meta-regression model following Vincze et al. (2017) rather than a single over-arching model with multiple moderators. The random effect structure and phylogenetic modelling in these separate meta-regression models was

154 specified as above. We assessed the significance of each moderator based on likelihood
155 ratio tests (Wald test *W*) and overlap of the 95 % confidence intervals with 0 for each
156 regression coefficient.
157 We then examined whether some specific trait categories (Table S1) exhibited
158 higher support for the pace-of-life syndrome hypothesis. To do so we analysed our data
159 separately by life history or physiological trait category (life-history: 4 categories, 53
160 estimates, 16 articles; physiology: 3 categories, 126 estimates, 30 articles) and behavioural
161 trait category (behaviour × life-history: 19 categories, behaviour × physiology: 18
162 categories). As above we used phylogenetic meta-regression models on each of these
163 datasets and included all random effects and moderators mentioned above (except
164 correlation type). For this analysis we again used likelihood ratio tests to assess the
165 statistical significance of the moderators. Because of the exploratory nature of this analysis
166 and the small sample sizes for particular behavioural categories (Table 1), we also
167 calculated 95% confidence intervals to assess support for these finer-scale categorizations.
168 As a final exploratory analysis, we conducted a meta-regression with species identity as a
169 moderator to test whether certain species showed stronger support for the pace-of-life
170 syndrome hypothesis.
171 We tested for the presence of publication bias in our data through visual inspection
172 of funnel plots and Egger's regression tests (Egger et al 1997) on our full dataset and
173 subsets including estimates for behaviour × life-history and behaviour × physiology
174 correlations. We also used a trim-and-fill method to estimate the number of studies
175 potentially absent from our dataset.
176

177 **3. Results**

178 **(a) Overall effect size for the pace-of-life syndrome hypothesis**

179 The global effect size for correlations among behavioural, physiological and life-history
180 traits—as predicted by the pace-of-life syndrome hypothesis —was 0.06 ± [-0.01; 0.14]
181 (estimate ± [95% CIs]), while the median effect size was of 0.02 (Fig. 1, Table S2). We also
182 did not find support for the pace-of-life syndrome hypothesis when focusing on either
183 behaviour × life-history estimates (r = 0.12 ± [-0.01; 0.26]) or behaviour × physiology
184 estimates (r = 0.04 ± [-0.05; 0.14]). Heterogeneity in the full dataset and when subsetted
185 into behaviour × life-history and behaviour × physiology datasets was equally high (I^2 > 99
186 %), thus justifying our investigation of the effects of moderators on the support for the
187 pace-of-life syndrome hypothesis. When more finely examining sources of variation in
188 effect sizes, we found the largest sources were at the residual level (i.e. within-article and
189 within species variation), accounting for 62 to 81 % of the variation depending on the
190 subset considered (Table 2). Species identity also showed substantial variation in effect
191 sizes and accounted for 15 to 37 % of the variation.

192

193 **(b) Effects of moderator on the support for the pace-of-life syndrome hypothesis**

194 In the full dataset, we found that the following moderators showed significant differences
195 in their support for the pace-of-life syndrome hypothesis: vertebrates versus invertebrates
196 (W = 5.23, df = 1, P = 0.02), sex (W = 12.76, df = 2, P = 0.002), levels of inference (W =
197 150.50, df = 2, P = 0.0001) and relationship type (W = 65.26, df = 1, P = 0.0001) (Fig. 2,
198 Table S2). Specifically, we found that support for the pace-of-life syndrome hypothesis was
199 stronger in invertebrates (r = 0.23 ± [0.07; 0.37]) compared to vertebrates (r = 0.02 ± [-

0.06; 0.10]), that females showed strong correlations in directions opposite to that predicted by the pace-of-life syndrome hypothesis (r = -0.16 ± [-0.30; 0.00] versus, for males: r = 0.01 ± [-0.13; 0.15]), and that estimates taken at the genetic (r = -0.09 ± [-0.17; -0.01]) and phenotypic levels (r = 0.10 ± [0.02; 0.18]) had opposite support for the pace-of-life syndrome hypothesis. In addition, we found that estimates of correlations for behaviour × life-history traits (r = 0.22 ± [0.13; 0.29]) were more likely to follow the predictions of the pace-of-life syndrome hypothesis compared to correlations of behaviour × physiology traits (r = -0.01 ± [-0.09; 0.07]).

When investigating the effect of these moderators within subsets of the data (Fig. 2), we did not find evidence of moderator effects for correlations between behaviour and life-history traits (P > 0.20, Fig. 2a, Table S3). For correlations between behaviour and physiological traits, data taken on females (r = -0.17 ± [-0.34; 0.00]; W = 12.95, df = 2, P = 0.002) and correlations estimated at the genetic level (r = -0.13 ± [-0.23; -0.03]; W = 167.57, df = 2, P = 0.0001) were both more likely to show correlations in directions opposite to that predicted by the pace-of-life syndrome hypothesis.

(c) Exploratory comparison by correlation category and species

Specific categories of behaviour × life-history (W = 27.13, df = 3, P = 0.0001, Table S3) and behaviour × physiology traits (W = 7.36, df = 2, P = 0.03, Table S4) exhibited stronger support for the pace-of-life syndrome hypothesis.

In particular, correlations involving growth rate (r = 0.23 ± [0.03; 0.41]) or hormones (r = 0.18 ± [0.04; 0.32]) showed moderate support for the pace-of-life. Note also that correlation estimates with life span showed similar effect size in support for the pace-

of-life but had 95 % confidence intervals overlapping 0 (r = 0.14 ± [-0.06; 0.34]). All other life-history and physiology categories showed no support (Fig .2, Table S3, S4).

When considering the specific behavioural traits within the data, we found that for correlations with life-history traits; docility × life-span (r = 0.61, n = 1), boldness × growth-rate (r = 0.37, n = 6), aggression × life-span (r = 0.24, n = 1) and aggression × growth-rate (r = 0.20, n = 2) had the highest median $|r|$ values. For correlations with physiological traits, we found that neophobia (r = 0.51, n = 2), exploration (r = 0.35, n = 8) and aggression (r = 0.29, n = 4) all had moderate to strong correlations with metabolism, and that the positive trend with hormonal traits was primarily driven by estimates taken on foraging behaviour (r = 0.23, n = 4) and on alarm calls (r = 0.61, n = 1, classified as "other") (Table 1, S1).

When we included species as a moderator in our meta-regression, we found strong amounts of variation among species (W = 48.39, df = 30, P = 0.02) (Fig. 3). Note, however, that most species were represented by only one article in our data, making interpretation of these trends difficult.

(d) Publication bias

Based on both graphical inspection of funnel plots (Fig. 4) and Eggert's regressions, we did not find evidence of publication bias in our full dataset (t = 0.05, df = 177, P = 0.96) nor in subsets of the data (behaviour × life-history: t =- 0.38, df = 51, P =0.70; behaviour × physiology: t = 0.09, df = 124, P = 0.93) (Fig. 4). In addition, trim-and-fill analysis did not suggest any evidence of missing estimates, which is consistent with the absence of publication bias. Generally, studies with high precision were as likely to indicate effect size in favour or against the predictions of the pace-of-life, as they were for smaller studies. This suggests that the overall lack of support for the pace-of-life syndrome hypothesis is

246 unlikely to be due to lack of statistical power to estimate correlations among behaviour,
247 life-history, and physiological traits.

248 **4. Discussion**

249 We found little support for the general predictions of the pace-of-life syndrome hypothesis.
250 Global effect sizes across the full data set and subsets were low and had confidence
251 intervals that overlapped zero. Thus behaviour, based on currently available data, does not
252 appear to be integrated with physiology or life-history in a manner that is consistent with
253 the pace-of-life syndrome hypothesis.

254 Despite the lack of general support for the pace-of-life syndrome hypothesis, we did
255 find that some combinations of traits were more likely to be associated in predicted
256 directions (Table 1, Fig. 2). Our post-hoc exploratory analysis found that hormonal levels
257 and growth rate were particularly likely to be linked to behavioural traits, and linked in the
258 direction predicted by Réale et al. (2010). Interestingly, we found that the pace-of-life
259 syndrome hypothesis predictions were least well supported for females. This reflects the
260 possibility that hypotheses regarding sex-differences in how traits should covary in females
261 versus males require further development (Hämäläinen et al. 2018). Importantly, while the
262 lack of general support for the pace-of-life syndrome hypothesis is robustly demonstrated
263 by our estimates of global effect sizes, some of the finer scale effects of moderators should
264 be interpreted with caution as narrow combinations of traits were often estimated in few
265 studies and species. This therefore creates the possibility that some analyses confound the
266 results of multiple differences. For example, the finding of stronger support in behaviour ×
267 life-history versus behaviour × physiology trait combinations is attributable to among-
268 species and among-article differences being conflated with moderator effects in the full

dataset (Table S5). Consistent with this, little support for the pace-of-life syndrome hypothesis was found when these subsets were analysed separately.

Our failure to detect general support for the pace-of-life syndrome hypothesis is particularly interesting given the considerable literature suggesting such links (Careau et al. 2008, 2009; Biro and Stamps 2010; Réale et al. 2010; Careau and Garland 2012). Importantly, our analysis pertains specifically to the role of behaviour as a link between life-history and physiology. The lack of general evidence that behaviour connects to either life-history or physiology as predicted does not preclude the possibility that the pace-of-life framework properly predicts connections between life-history and physiology, as identified in a number of studies (e.g. Wikelski et al. 2003; Tieleman et al. 2005; Wiersma et al. 2007; Tieleman 2009). The lack of detectable support may be attributable to three general and non-exclusive explanations: 1) the frequent violation of key assumptions of the pace-of-life syndrome hypothesis, 2) confusing terminology and methods for measuring behavioural traits , and 3) the importance played by the local environment in shaping and maintaining trait correlations (Montiglio et al. 2018; Salzman et al. 2018).

A first potential explanation for the observed lack of support for the pace-of-life syndrome hypothesis may be that its assumptions are frequently violated (see Montiglio et al. 2018 for a review of these assumptions and approaches to validating them). For example, an important assumption of the pace-of-life syndrome hypothesis is that the slow-fast pace-of-life axis is in part generated through trade-offs in allocation resulting from limited resources. Specifically, trade-offs arise due to conflict between investment in growth or mortality and between current and future reproduction (Rickleffs and Wikelski 2002; Biro and Stamps 2008; Réale et al. 2010; Montiglio et al. 2018). Problematically, 51%

of our estimates (i.e. 20 studies) originated from laboratory studies where *ad-lib* access to food is a typical practice. However, and very importantly, whether a study was conducted in the lab or field did not change support for the pace-of-life syndrome hypothesis (W = 0.96, df = 1, P = 0.33). Another implicit assumption for testing the pace-of-life syndrome hypothesis is that inferences should be drawn at the appropriate level of biological organization. Specifically, the pace-of-life syndrome hypothesis predicts that among-individual or genetic differences in suites of life-history, behavioural and physiological traits share a common axis of variation. This implies that the model should be tested at least at the level of among-individual correlations (*sensu* Dingemanse et al. 2012; Dingemanse and Dochtermann 2013) or based on additive genetic correlations. Unfortunately, only 32% of our estimates (i.e. 20 studies) were based on repeated measurements of at least one of the traits or were estimates of genetic correlations. This suggests that most purported tests of the pace-of-life syndrome hypothesis have violated this key assumption. Interestingly, however, we found that at the genetic level behaviour was negatively correlated with physiology, opposite predictions (Fig. 2B), with positive support at the phenotypic level (Fig. 1). This suggests that while different patterns may be manifested at different levels, as is generally expected for trade-offs (van Noordwijk and de Jong 1986), the pattern predicted by the pace-of-life was not supported at the level it should have been most clearly observable. Similarly, Santostefano et al. (2017) found that some correlative patterns differed between the phenotypic and genetic level in Mediterranean field crickets (*Gryllus bimaculatus*), though there was again a lack of support for pace-of-life syndrome hypotheses at the genetic level. Further, while among-individual support was positive its estimate was very low (Fig. 1) with confidence intervals

overlapping zero (Fig. 1). Importantly, this suggests that the pace-of-life syndrome primarily exhibits support at the within-individual level, contrary to predicted relationships. The lack of differences between laboratory and field studies and the finding that genetic correlations are in the opposite direction predicted suggests that our failure to find support for the pace-of-life syndrome hypothesis is not generally due to assumptions being violated.

Second, one difficulty in evaluating general support for the pace-of-life syndrome hypothesis involves determining how particular trait combinations should be correlated. Many of the behavioural traits which are expected to covary with either life-history or physiology are very broadly defined and can be estimated through multiple and sometimes conflicting assays. For example, according to Réale et al. (2010), thorough "explorers"—individuals that are less active but investigate their environment more thoroughly—are predicted to fall toward the "slow" portion of the pace-of-life axis. Yet, in many cases, exploration tests are conducted such that thorough explorers cannot be distinguished from fast ones. For example, exploration is often measured in open-field arenas where the number of unique zones visited within a fixed amount of time serves as a metric of exploratory propensity (Carducci and Jakob 2000; Montiglio et al. 2010; Royauté et al. 2015b). In such tests, individuals with higher exploration scores are typically considered "fast" explorers as they cover more distance in the test arena. However, these individuals could also be argued to be more thorough and thus "slow" explorers, as they cover a larger proportion of the space available for them to explore. Similarly, "boldness"—a measure of an individual's tendency to take risks—is often estimated through a variety of methods. These methods include the latency to emerge from a shelter (Niemelä et al. 2012), vigilance

behaviour (Montiglio et al. 2010), or response to the presence of predators or cues of predator presence (Krams et al. 2014b; Shearer and Pruitt 2014; Royauté and Dochtermann 2017). Whether any of these assays actually measures "boldness" and whether they are all equally likely to be consistent with pace-of-life predictions remains unclear. This problem extends broadly across the study of behaviour where naming fallacies and jingle-jangle fallacies are difficult to avoid (Uher 2011; Carter et al. 2013; Dochtermann and Nelson 2014). Besides naming and jingle-jangle fallacies, whether behavioural measurements taken in standardized laboratory environments are ecologically relevant is often unclear (e.g. Adriaenssenss and Johnsson 2009; Dammhahn and Almeling 2012). Nonetheless, given that we based whether a study exhibited effects as expected according to the pace-of-life syndrome hypothesis on the operational definitions of behaviours provided by authors, we do not consider this explanation sufficient to justify the lack of support found in our analysis.

Third, as Réale et al. (2010) themselves noted, the pace-of-life is not the sole source of "correlation and causation between physiology, personality and life-history" and several departures from the model's predictions were previously identified by Réale et al. (2010). This includes departures from predictions likely due to local environmental conditions. For example, predation presence/absence has been shown to shape the overall strength and direction of trait correlations in a number of studies (Bell 2005; Bell and Sih 2007; Dingemanse et al. 2007), and populations living in low predation have been observed to have slower pace-of-life characteristics (slow growth rate and reproduction) compared to populations heavily exposed to predation pressures (Reznick et al. 1996; Montiglio et al. 2018). Thus, the natural and evolutionary history of a particular species or population

might be more relevant for understanding and predicting connections between behaviour and life-history.

Besides these three explanations, a fourth alternative is simply that the pace-of-life syndrome hypothesis does not apply to the integration of behaviour with either physiology or life-history. While we do not currently have sufficient information to conclusively distinguish among these four explanations, the available data clearly suggests that the pace-of-life syndrome hypothesis is insufficient to explain observed patterns of how physiological or life-history traits correlate with behaviours at the within-species level. Therefore, based on this available data, the predictions of the pace-of-life syndrome hypothesis require further development and testing before being considered broadly applicable. Wikelski and Ricklefs (2002) originally proposed the pace-of-life hypothesis as an explanation for among-species covariation in physiology, life-history, and behaviour based on the presence of slow versus fast life-histories. The extension by Réale et al. (2010) to explain among-individual variation therefore first requires the demonstration of among-individual variation in life-history along a slow-fast continuum. If among-individual variation in life-history doesn't correspond to this continuum, it is not clear whether behaviours should be expected to covary with either physiology or life-history in the directions predicted by Réale et al. (2010). A way forward may be to derive predictions regarding phenotypic integration on a case-by-case basis given the natural history of species while considering the environmental conditions that may favour specific traits to be linked along a pace-of-life axis (Montiglio et al. 2018).

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Data availability: Analyses reported in this article can be reproduced using the data and statistical code provided by Royauté et al. (2018) and via provided supplemental information.

References

Adriaenssens B, Johnsson JI (2009) Personality and life-history productivity: consistent or variable association? Trends Ecol Evol 24:179-180

Adriaenssens B, Johnsson JI (2010) Shy trout grow faster: exploring links between personality and fitness-related traits in the wild. Behav Ecol 22:135-143

Bell AM (2005) Behavioural differences between individuals and two populations of stickleback (*Gasterosteus aculeatus*). J Evol Biol 18:464-473

Bell AM, Sih A (2007) Exposure to predation generates personality in threespined sticklebacks (*Gasterosteus aculeatus*). Ecol Lett 10:828-834

Biro PA, Stamps JA (2008) Are animal personality traits linked to life-history productivity?. Trends Ecol Evol 23:361-368

Biro PA, Stamps JA (2010) Do consistent individual differences in metabolic rate promote consistent individual differences in behavior? Trends Ecol Evol 25:653-659

404 Carducci JP, Jakob EM (2000) Rearing environment affects behaviour of jumping spiders.
405 Anim Behav 59:39-46
406 Careau V, Bininda-Emonds O, Thomas D, Réale D, Humphries M (2009) Exploration
407 strategies map along fast–slow metabolic and life-history continua in muroid
408 rodents. Funct Ecol 23:150-156
409 Careau V, Garland T (2012) Performance, personality, and energetics: correlation,
410 causation, and mechanism. Physiol Biochem Zool 85:543-571
411 Careau V, Montiglio P-O, Garant D, Pelletier F, Speakman JR, Humphries MM, Réale D
412 (2015) Energy expenditure and personality in wild chipmunks. Behav Ecol Sociobiol
413 69:653-661
414 Careau V, Réale D, Humphries MM, Thomas DW (2010) The pace of life under artificial
415 selection: personality, energy expenditure, and longevity are correlated in domestic
416 dogs. Am Nat 175:753-758
417 Careau V, Thomas D, Humphries MM, Réale D (2008) Energy metabolism and animal
418 personality. Oikos 117:641-653
419 Carter AJ, Feeney WE, Marshall HH, Cowlishaw G, Heinsohn R (2013) Animal personality:
420 what are behavioural ecologists measuring? Biol Rev 88:465-475
421 Dammhahn M, Almeling L (2012) Is risk taking during foraging a personality trait? A field
422 test for cross-context consistency in boldness. Anim Behav 84:1131-1139
423 Dingemanse NJ, Dochtermann NA (2013) Quantifying individual variation in behaviour:
424 mixed-effect modelling approaches. J Anim Ecol 82:39-54

425 Dingemanse NJ, Dochtermann NA, Nakagawa S (2012) Defining behavioural syndromes
426 and the role of 'syndrome deviation'in understanding their evolution. Behav Ecol
427 Sociobiol 66:1543-1548
428 Dingemanse NJ, Wright J, Kazem AJ, Thomas DK, Hickling R, Dawnay N (2007) Behavioural
429 syndromes differ predictably between 12 populations of three-spined stickleback. J
430 Anim Ecol 76:1128-1138
431 Dochtermann NA, Nelson AB (2014) Multiple facets of exploratory behavior in house
432 crickets (*Acheta domesticus*): split personalities or simply different behaviors?
433 Ethology 120:1110-1117
434 Engqvist L, Cordes N, Reinhold K (2015) Evolution of risk-taking during conspicuous
435 mating displays. Evolution 69:395-406
436 Galliard JF, Paquet M, Cisel M, Montes-Poloni L (2013) Personality and the pace-of-life
437 syndrome: variation and selection on exploration, metabolism and locomotor
438 performances. Funct Ecol 27:136-144
439 Hadfield J, Nakagawa S (2010) General quantitative genetic methods for comparative
440 biology: phylogenies, taxonomies and multi-trait models for continuous and
441 categorical characters. J Evol Biol 23:494-508
442 Hämäläinen A, Immonen E, Tarka M, Schuett W (2018) Evolution of sex-specific pace-of-life
443 syndromes. Behav Ecol Sociobiol: topical issue on Pace-of-life syndrome
444 Krams I, Kivleniece I, Kuusik A, Krama T, Freeberg TM, Mänd R, Sivacova L, Rantala MJ,
445 Mänd M (2014a) High repeatability of anti-predator responses and resting
446 metabolic rate in a beetle. J Insect Behav 27:57-66

Krams IA, Vrublevska J, Sepp T, Abolins-Abols M, Rantala MJ, Mierauskas P, Krama T (2014b) Sex-specific associations between nest defence, exploration and breathing rate in breeding pied flycatchers. Ethology 120:492-501

Letunic, I. (2015). *phyloT : Phylogenetic Tree Generator*. [online] Phylot.biobyte.de. Available at: http://phylot.biobyte.de/.

Mathot KJ, Frankenhuis WE (2018) Models of pace-of-life syndromes (POLS): a systematic review. Behav Ecol Sociobiol: topical issue on Pace-of-life syndrome

Mathot KJ, Nicolaus M, Araya-Ajoy YG, Dingemanse NJ, Kempenaers B (2015) Does metabolic rate predict risk-taking behaviour? A field experiment in a wild passerine bird. Funct Ecol 29:239-249

Montiglio PO, Dammhahn M, Dubuc Messier G, Réale D (2018) The pace-of-life syndrome hypothesis: evidence, limitations and future directions. Behav Ecol Sociobiol: topical issue on Pace-of-life syndrome

Montiglio P-O, Garant D, Thomas D, Réale D (2010) Individual variation in temporal activity patterns in open-field tests. Anim Behav 80:905-912

Nakagawa S, Cuthill IC (2007) Effect size, confidence interval and statistical significance: a practical guide for biologists. Biol Rev 82:591-605

Nakagawa S, Santos ES (2012) Methodological issues and advances in biological meta-analysis. Evol Ecol 26:1253-1274

Niemelä PT, Vainikka A, Hedrick AV, Kortet R (2012) Integrating behaviour with life history: boldness of the field cricket, *Gryllus integer*, during ontogeny. Funct Ecol 26:450-456

R Core Team (2016) R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria, https://www.R-project.org/

Réale D, Garant D, Humphries MM, Bergeron P, Careau V, Montiglio P-O (2010) Personality and the emergence of the pace-of-life syndrome concept at the population level. Philos T Roy Soc B 365:4051-4063

Réale D, Reader SM, Sol D, McDougall PT, Dingemanse NJ (2007) Integrating animal temperament within ecology and evolution. Biol Rev 82:291-318

Reznick DN, Butler IV MJ, Rodd FH, Ross P (1996) Life-history evolution in guppies (*Poecilia reticulata*) 6. Differential mortality as a mechanism for natural selection. Evolution 50:1651-1660

Ricklefs RE, Wikelski M (2002) The physiology/life-history nexus. Trends Ecol Evol 17:462-468

Royauté R, Berdal MA, Garrison CR, Dochtermann NA (2018) Data from: Paceless life? A meta-analysis of the pace-of-life syndrome hypothesis. Dryad Digital Repository, doi: **Pending**

Royauté R, Buddle CM, Vincent C (2015b) Under the influence: sublethal exposure to an insecticide affects personality expression in a jumping spider. Funct Ecol 29:962-970

Royauté R, Dochtermann NA (2017) When the mean no longer matters: developmental diet affects behavioral variation but not population averages in the house cricket (*Acheta domesticus*). Behav Ecol 28:337-345

490 Royauté R, Greenlee K, Baldwin M, Dochtermann NA (2015a) Behaviour, metabolism and
491 size: phenotypic modularity or integration in *Acheta domesticus*? Anim Behav
492 110:163-169
493 Salzman TC, McLaughlin AL, Westneat DF, Crowley PH (2018) Energetic trade-offs and
494 feedbacks between behavior and metabolism influence correlations between pace-
495 of-life attributes. Behav Ecol Sociobiol: topical issue on Pace-of-life syndrome
496 Santostefano F, Wilson AJ, Niemelä PT, Dingemanse NJ (2017) Behavioural mediators of
497 genetic life-history trade-offs: a test of the pace-of-life syndrome hypothesis in field
498 crickets. Proc R Soc B 284:20171567
499 Shearer TA, Pruitt JN (2014) Individual differences in boldness positively correlate with
500 heart rate in orb-weaving spiders of genus Larinioides. Curr Zool 60:387-391
501 Sih A, Bell AM, Johnson JC, Ziemba RE (2004) Behavioral syndromes: an intergrative
502 overiew. Q Rev Biol 79:241-277
503 Sinervo B, Svensson E (2002) Correlational selection and the evolution of genomic
504 architecture. Heredity 89:329-338
505 Tieleman BI (2009) High and low, fast or slow: the complementary contributions of altitude
506 and latitude to understand life-history variation. J Anim Ecol 78:293-295
507 Tieleman BI, Williams JB, Ricklefs RE, Klasing KC (2005) Constitutive innate immunity is a
508 component of the pace-of-life syndrome in tropical birds. Proc R Soc Lond B
509 272:1715-1720
510 Uher J (2011) Individual behavioral phenotypes: An integrative meta-theoretical
511 framework. Why “behavioral syndromes” are not analogs of “personality”. Dev
512 Psychobiol 53:521-548

van Noordwijk AJ, de Jong G (1986) Acquisition and allocation of resources: their influence on variation in life history tactics. Am Nat 128:137-142

Vincze E, Seress G, Lagisz M, Nakagawa S, Dingemanse N, Sprau P (2017) Does urbanization affect predation of bird nests? A meta-analysis. Front Ecol Evol 5:29

Wiersma P, Muñoz-Garcia A, Walker A, Williams JB (2007) Tropical birds have a slow pace of life. P Natl Acad Sci USA 104:9340-9345

Wikelski M, Spinney L, Schelsky W, Scheuerlein A, Gwinner E (2003) Slow pace of life in tropical sedentary birds: a common-garden experiment on four stonechat populations from different latitudes. Proc R Soc Lond B 270:2383-2388

Wolf M, van Doorn GS, Leimar O, Weissing FJ (2007) Life-history trade-offs favour the evolution of animal personalities. Nature 447:581-584

525 **Table 1** Median effect sizes (*r*) and associated lower (2.5 %) and upper (97. 5 %) quantiles for
526 the support of the pace-of-life syndrome hypothesis between behavior and life-history traits (a)
527 and physiological traits (b). Bold indicates non-overlap of the 95 % quantile with 0, values |*r*| >
528 0.4 are indicated in bold and italics

	Category		Behaviour	*r*	2.5 % quantile	97.5 % quantile	number of estimates
(a)	Growth-rate	×	Overall	**0.11**	**0.01**	**0.79**	15
			Activity	0.02	-0.38	0.69	3
			Aggression	**0.20**	**0.07**	**0.46**	2
			Boldness	**0.37**	**0.11**	**0.81**	6
			Exploration	-0.06			1
			Foraging	**0.08**	**0.06**	**0.11**	2
			Trainability	0.17			1
	Life-span	×	Overall	-0.02	-0.04	0.54	9
			Activity	**-0.13**	**-0.18**	**-0.01**	2
			Aggression	0.24			1
			Boldness	**-0.04**	**-0.04**	**-0.04**	2
			Exploration	-0.10			1
			Foraging	**0.01**	**0.00**	**0.05**	2
			Trainability	***0.61***			1
	Maturation rate	×	Overall	-0.04	-0.08	0.29	13
			Activity	**0.12**	**0.11**	**0.12**	2
			Boldness	-0.04	-0.06	0.03	7
			Exploration	0.09	-0.04	0.35	2
			Foraging	**-0.16**	**-0.18**	**-0.11**	2

	Productivity	×	Overall	0.16	-0.05	0.63	16
			Activity	0.06	-0.49	0.65	4
			Boldness	**0.16**	**0.14**	**0.33**	8
			Exploration	-0.04	-0.16	0.23	4
(b)	Hormones	×	Overall	0.05	-0.03	0.68	27
			Activity	0.04	-0.03	0.34	13
			Boldness	-0.03	-0.11	0.07	3
			Docility	0.06	-0.01	0.77	4
			Exploration	**0.08**	**0.02**	**0.20**	2
			Foraging	**0.23**	**0.01**	**0.42**	4
			Other	***0.61***			1
	Immunity	×	Overall	-0.08	-0.27	0.32	30
			Activity	-0.07	-0.24	0.44	11
			Boldness	-0.03	-0.10	0.22	6
			Docility	-0.10	-0.25	0.04	7
			Exploration	-0.38			1
			Foraging	-0.17	-0.25	0.11	4
			Other	0.03	-0.16	0.75	1
	Metabolism	×	Overall	0.02	-0.17	0.70	69
			Activity	-0.19	-0.22	0.75	21
			Aggression	**0.29**	**0.22**	**0.73**	4
			Boldness	-0.23	-0.40	0.10	15
			Docility	-0.15	-0.32	0.10	10
			Exploration	**0.35**	**0.19**	**0.65**	8
			Foraging	0.01	-0.06	0.15	8

	Neophobia	***0.51***			2
	Trainability	0.03	-0.16	0.75	1

529

Table 2 Sources of variation (i.e., heterogeneity) attributable to the random effects of article identity, species, residuals (i.e. within-article and within-species variance) and sampling error for the complete dataset and the subset containing correlations between behavior × life-history and behavior × physiology traits. I^2 represents the total heterogeneity in the data, calculated as the proportion of variation explained by all random effects excluding sampling error variance

Sources of variation (%)	All data	Behavior × Life history	Behavior × Physiology
Article	3.0	8.2 × 10-8	3.76
Species	21.3	37.1	14.3
Residuals	75.0	62.0	81.2
Sampling error	0.71	0.91	0.73
I^2	99.3	99.1	99.3

Figure Legends

Fig. 1 Forest plot of estimated effect sizes (*r* ± 95% CI) for all moderator categories based on moderator contrasts. Positive values indicate stronger support for the pace-of-life hypothesis. The effect size and confidence interval from the intercept only model (white diamond) indicates whether there is an overall support for the pace-of-life hypothesis. Point size is proportional to the sample size in the dataset

Fig. 2 Forest plots of estimated effect sizes (*r* ± 95% CI) of the pace-of-life hypothesis compared across (a) life-history traits and (b) physiological traits. Positive values indicate stronger support for the pace-of-life hypothesis. Point size is proportional to the sample size in the dataset

Fig. 3 Phylogenetic tree of the species present in the dataset (left side) and their associated mean effect sizes and 95 % confidence intervals calculated from a meta-regression with species as a moderator

Fig 4 Funnel plots used to estimate publication bias in the full dataset (a) and the subsets containing behaviour × life-history correlations (b) and behaviour × physiology correlations (c). Dashed lines are the meta-analytic means