



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

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Abstract

The pace-of-life syndrome hypothesis predicts that individual differences in behavior 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 behaviors 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 behavior 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 analyzed (behavior \times life-history: $r = 0.12$; behavior \times physiology: $r = 0.04$). Exploratory post hoc analyses revealed that correlations of behavior 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” should be re-evaluated.

Significance statement

The pace-of-life syndrome hypothesis has been proposed as an overall organizational framework for the integration of behavioral, 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 7 years, this organizational framework has been intensively investigated as it pertains to relationships between behavior and other traits. Here, we conducted an overall analysis of whether the hypothesis was supported. Despite considerable research investment across behavioral 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.

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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, behavior). One of the most comprehensive conceptual models that integrates variation in life-history, physiology, and behavior 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 behavioral 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 behavioral variation and that individuals behave in predictably (i.e., consistently) different ways from one another. Extending the hypothesis to behavior makes the implicit assumption that among-individual differences in behavior (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 behaviors (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 behavior 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 behaviors should covary with physiology and life-history (Wolf et al. 2007; Engqvist et al. 2015; reviewed in Mathot and Frankenhuis 2018, topical collection on Pace-of-life syndromes). Further, despite the number of conceptual papers arguing for the pace-of-life syndrome hypothesis as an organizational basis for the integration of behavior with life-history and physiology, empirical support for behavior 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 behavioral 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 behavior 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 behaviors.

Methods

Data collection

To test for overall support of the pace-of-life syndrome hypothesis, we gathered estimates of the relationship between life-history and behavior or physiology and behavior by searching 15 leading journals in behavioral and evolutionary ecology in ISI Web of Science on April 27, 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 behavioral 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 behavioral 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 behavior and life-history or behavior and physiology that fit the above criteria (Fig. S1).

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 modeled 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 behavior \times life-history or behavior \times 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 (behavior \times life-history or behavior \times 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 modeling in these separate meta-regression models was specified as above. We assessed the significance of each moderator based on likelihood ratio tests (Wald test W) and overlap of the 95% confidence intervals with 0 for each regression coefficient.

We then examined whether some specific trait categories (Table S1) exhibited higher support for the pace-of-life syndrome hypothesis. To do so, we analyzed our data separately by life-history or physiological trait category (life-history 4 categories, 53 estimates, 16 articles; physiology 3 categories, 126 estimates, 30 articles) and behavioral trait category (behavior \times life-history 19 categories, behavior \times physiology 18 categories). As above, we used a phylogenetic meta-regression models on each of these datasets and included all random effects and moderators mentioned above (except correlation type). For this analysis, we again used likelihood ratio tests to assess the statistical significance of the moderators. Because of the exploratory nature of this analysis and the small sample sizes for particular behavioral categories (Table 1), we also calculated 95% confidence intervals to assess support for these finer-scale categorizations. As a final exploratory analysis, we conducted a meta-regression with species identity as a moderator to test whether certain species showed stronger support for the pace-of-life syndrome hypothesis.

We tested for the presence of publication bias in our data through visual inspection of funnel plots and Egger's regression tests (Egger et al. 1997) on our full dataset and subsets including estimates for behavior \times life-history and behavior \times physiology correlations. We also used a trim-and-fill method to estimate the number of studies potentially absent from our dataset.

Results

Overall effect size for the pace-of-life syndrome hypothesis

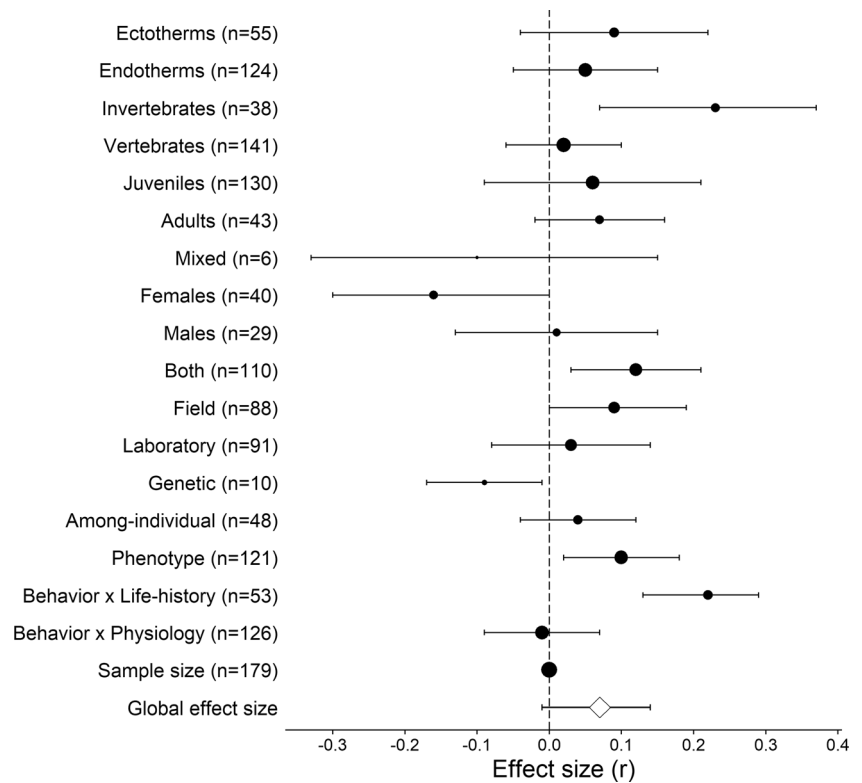
The global effect size for correlations among behavioral, physiological, and life-history traits—as predicted by the pace-of-life syndrome hypothesis—was $0.06 \pm [-0.01; 0.14]$ (estimate \pm (95% CIs)), while the median effect size was 0.02 (Fig. 1, Table S2). We also did not find support for the pace-of-life syndrome hypothesis when focusing on either behavior \times life-history estimates ($r = 0.12 \pm [-0.01; 0.26]$) or behavior \times physiology estimates ($r = 0.04 \pm [-0.05; 0.14]$). Heterogeneity in the full dataset and when subsetted into behavior \times life-history and behavior \times physiology datasets was equally high ($I^2 > 99\%$), thus justifying our investigation of the effects of moderators on the support for the pace-of-life syndrome hypothesis. When more finely examining sources

Table 1 Median effect sizes (r) and associated lower (2.5%) and upper (97.5%) quantiles for the support of the pace-of-life syndrome hypothesis between behavior and life-history traits (a) and physiological traits (b)

	Category		Behavior	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	<i>0.61</i>			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	
		Other	<i>0.61</i>			1	
(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	<i>0.61</i>			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			<i>0.51</i>			2	
Trainability			0.03	-0.16	0.75	1	

Bold indicates non-overlap of the 95% quantile with 0, values $|r| > 0.4$ are indicated in bold and italics

Fig. 1 Forest plot of estimated effect sizes ($r \pm 95\%$ 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



of variation in effect sizes, we found the largest sources were at the residual level (i.e., within-article and within species variation), accounting for 62 to 81% of the variation depending on the subset considered (Table 2). Species identity also showed substantial variation in effect sizes and accounted for 15 to 37% of the variation.

Effects of moderator on the support for the pace-of-life syndrome hypothesis

In the full dataset, we found that the following moderators showed significant differences in their support for the pace-of-life syndrome hypothesis: vertebrates versus invertebrates ($W = 5.23$, $df = 1$, $P = 0.02$), sex ($W = 12.76$, $df = 2$, $P = 0.002$), levels of inference ($W = 150.50$, $df = 2$, $P = 0.0001$),

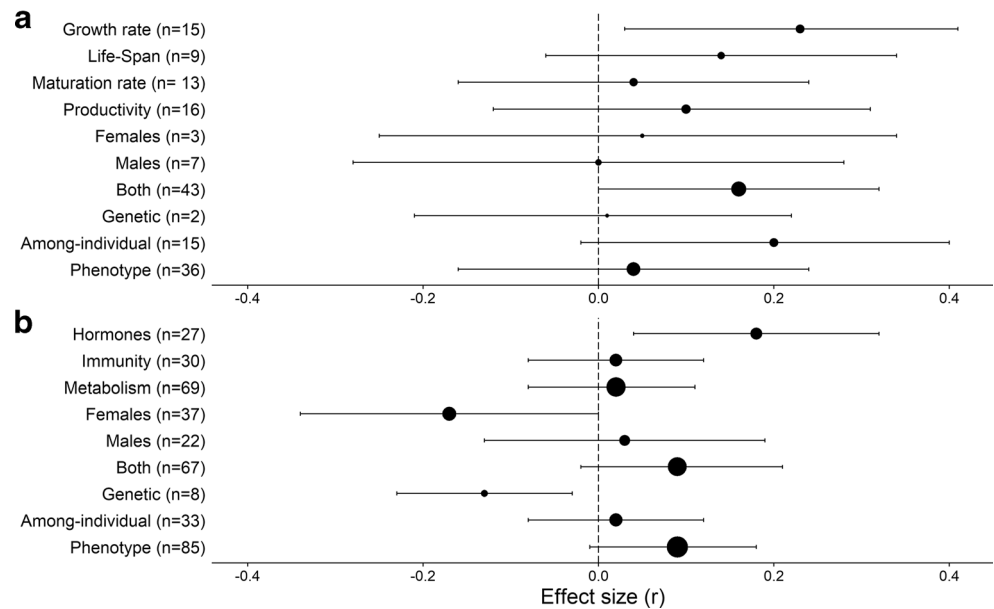
and relationship type ($W = 65.26$, $df = 1$, $P = 0.0001$) (Fig. 2, Table S2). Specifically, we found that support for the pace-of-life syndrome hypothesis was stronger in invertebrates ($r = 0.23 \pm [0.07; 0.37]$) compared to vertebrates ($r = 0.02 \pm [-0.06; 0.10]$), that females showed strong correlations in directions opposite to that predicted by the pace-of-life syndrome hypothesis ($r = -0.16 \pm [-0.30; 0.00]$) versus, for males: $r = 0.01 \pm [-0.13; 0.15]$, and that estimates taken at the genetic ($r = -0.09 \pm [-0.17; -0.01]$) and phenotypic levels ($r = 0.10 \pm [0.02; 0.18]$) had opposite support for the pace-of-life syndrome hypothesis. In addition, we found that estimates of correlations for behavior \times life-history traits ($r = 0.22 \pm [0.13; 0.29]$) were more likely to follow the predictions of the pace-of-life syndrome hypothesis compared to correlations of behavior \times physiology traits ($r = -0.01 \pm [-0.09; 0.07]$).

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 \times life-history and behavior \times physiology traits

Sources of variation (%)	All data	Behavior \times life-history	Behavior \times 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

I^2 represents the total heterogeneity in the data, calculated as the proportion of variation explained by all random effects excluding sampling error variance

Fig. 2 Forest plots of estimated effect sizes ($r \pm 95\%$ 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



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 behavior and life-history traits ($P > 0.20$, Fig. 2a, Table S3). For correlations between behavior and physiological traits, data taken on females ($r = -0.17 \pm [-0.34; 0.00]$; $W = 12.95$, $df = 2$, $P = 0.002$) and correlations estimated at the genetic level ($r = -0.13 \pm [-0.23; -0.03]$; $W = 167.57$, $df = 2$, $P = 0.0001$) were both more likely to show correlations in directions opposite to that predicted by the pace-of-life syndrome hypothesis.

Exploratory comparison by correlation category and species

Specific categories of behavior \times life-history ($W = 27.13$, $df = 3$, $P = 0.0001$, Table S3) and behavior \times 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 ($r = 0.23 \pm [0.03; 0.41]$) or hormones ($r = 0.18 \pm [0.04; 0.32]$) 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 ($r = 0.14 \pm [-0.06; 0.34]$). All other life-history and physiology categories showed no support (Fig. 2, Tables S3, S4).

When considering the specific behavioral traits within the data, we found that for correlations with life-history traits, docility \times life-span ($r = 0.61$, $n = 1$), boldness \times growth rate ($r = 0.37$, $n = 6$), aggression \times life span ($r = 0.24$, $n = 1$) and aggression \times growth rate ($r = 0.20$, $n = 2$) had the highest

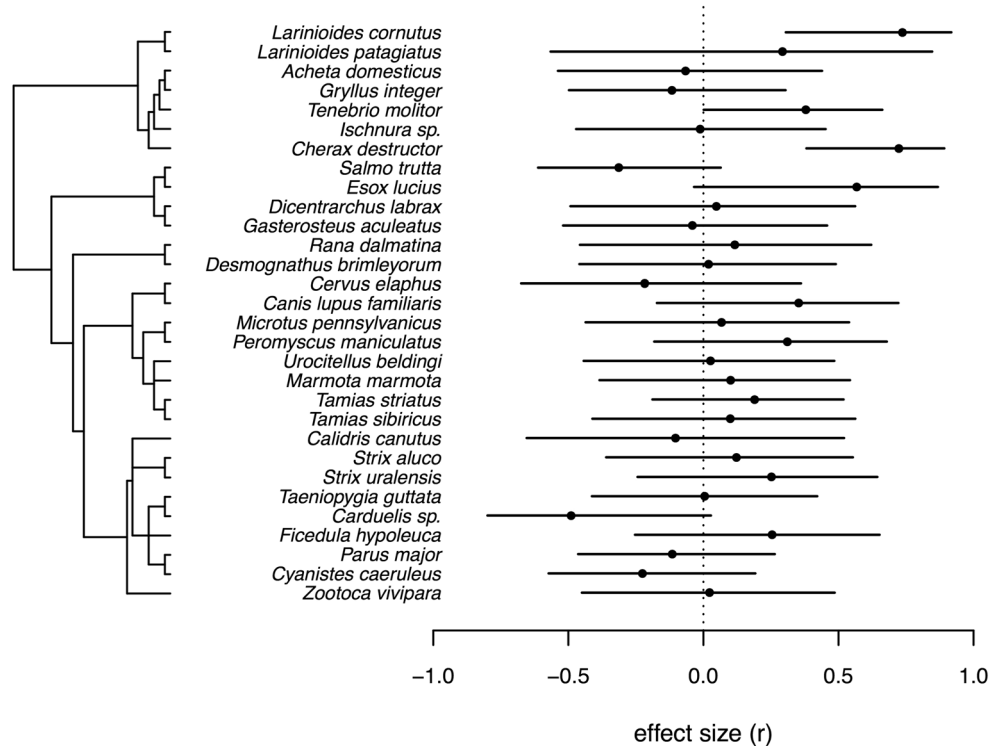
median $|r|$ values. For correlations with physiological traits, we found that neophobia ($r = 0.51$, $n = 2$), exploration ($r = 0.35$, $n = 8$), and aggression ($r = 0.29$, $n = 4$) all had moderate to strong correlations with metabolism, and that the positive trend with hormonal traits was primarily driven by estimates taken on foraging behavior ($r = 0.23$, $n = 4$) 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.

Publication bias

Based on both graphical inspections 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 (behavior \times life-history: $t = -0.38$, $df = 51$, $P = 0.70$; behavior \times 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 favor 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 unlikely to be due to lack of statistical power to estimate correlations among behavior, life-history, and physiological traits.

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



Discussion

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

Despite the lack of general support for the pace-of-life syndrome hypothesis, we did find that some combinations of traits were more likely to be associated in predicted directions (Table 1, Fig. 2). Our post hoc exploratory analysis found that hormonal levels and growth rate were particularly likely to be linked to behavioral traits, and linked in the direction predicted

by Réale et al. (2010). Interestingly, we found that the pace-of-life syndrome hypothesis predictions were least well supported for females. This reflects the possibility that hypotheses regarding sex-differences in how traits should covary in females versus males require further development (Hämäläinen et al. 2018, topical collection on Pace-of-life syndromes). Importantly, while the lack of general support for the pace-of-life syndrome hypothesis is robustly demonstrated by our estimates of global effect sizes, some of the finer scale effects of moderators should be interpreted with caution as narrow combinations of traits were often estimated in few studies and species. This therefore creates the possibility that some analyses confound the results of multiple differences. For example, the finding of stronger support in behavior × life-history versus behavior × physiology trait combinations is attributable to among-species and among-

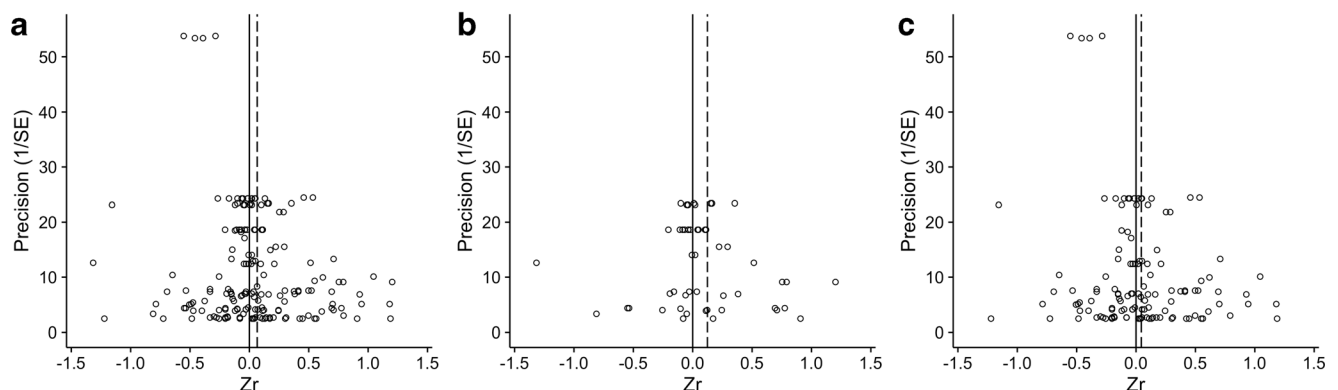


Fig. 4 Funnel plots used to estimate publication bias in the full dataset (a) and the subsets containing behavior × life-history correlations (b) and behavior × physiology correlations (c). Dashed lines are the meta-analytic means

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 analyzed 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 behavior as a link between life-history and physiology. The lack of general evidence that behavior 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 behavioral traits, and (3) the importance played by the local environment in shaping and maintaining trait correlations (Montiglio et al. 2018, topical collection on Pace-of-life syndromes; Salzman et al. 2018, topical collection on Pace-of-life syndromes).

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, topical collection on Pace-of-life syndromes 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 (Ricklefs and Wikelski 2002; Biro and Stamps 2008; Réale et al. 2010; Montiglio et al. 2018, topical collection on Pace-of-life syndromes). 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, behavioral, 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 Dingemans et al. 2012; Dingemans 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 behavior 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 behavioral 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 behavior (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 behavior 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 behavioral measurements taken in standardized laboratory environments are ecologically relevant is often unclear (e.g., Adriaenssens 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 behaviors 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, topical collection on Pace-of-life syndromes). Thus, the natural and evolutionary history of a particular species or population might be more relevant for understanding and predicting connections between behavior 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 behavior 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 behaviors 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. Ricklefs and Wikelski (2002) originally proposed the pace-of-life hypothesis as an explanation for among-species covariation in physiology, life-history, and behavior 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 does not correspond to this continuum, it is not clear whether behaviors 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 favor specific traits to be linked along a pace-of-life axis (Montiglio et al. 2018, topical collection on Pace-of-life syndromes).

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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), topical collection on Pace-of-life syndromes and via provided supplemental information.

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