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SYNTHESIS

What is the meta-analytic evidence for life-history trade-offs at the genetic level?

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Abstract

Understanding the evolutionary mechanisms underlying the maintenance of individual differences in behavior and physiology is a fundamental goal in ecology and evolution. The pace-of-life syndrome hypothesis is often invoked to explain the maintenance of such within-population variation. This hypothesis predicts that behavioral traits are part of a suite of correlated traits that collectively determine an individual's propensity to prioritize reproduction or survival. A key assumption of this hypothesis is that these traits are underpinned by genetic trade-offs among life-history traits: genetic variants that increase fertility, reproduction and growth might also reduce lifespan. We performed a systematic literature review and meta-analysis to summarize the evidence for the existence of genetic trade-offs between five key life-history traits: survival, growth rate, body size, maturation rate, and fertility. Counter to our predictions, we found an overall positive genetic correlation between survival and other life-history traits and no evidence for any genetic correlations between the non-survival life-history traits. This finding was generally consistent across pairs of life-history traits, sexes, life stages, lab vs. field studies, and narrow- vs. broad-sense correlation estimates. Our study highlights that genetic trade-offs may not be as common, or at least not as easily quantifiable, in animals as often assumed.

KEYWORDS
correlation, covariance, fertility, pace-of-life, personality, reproduction, survival

INTRODUCTION

Individual animals consistently differ in their behavioral and physiological traits, and these differences can have important fitness consequences. A fundamental goal in ecological and evolutionary research is to understand the mechanisms that maintain such phenotypic variation within populations. Life-history trade-offs have been central to explaining the maintenance of phenotypic variation (MacArthur & Wilson, 1967; Pianka, 1970; Stearns, 1989) and have been very successful at explaining variation present at the among-species level (Healy et al., 2019; Promislow & Harvey, 1990). This classic life-history theory predicts that species differ in their 'pace of life' due to differential resource allocation; correlational selection subsequently generates a suite of traits involved with a particular strategy. In the past 10–15 years this classic theory has been adapted to explain variation, particularly in behavioral traits, at the within-species level. The modern 'pace-of-life syndrome' (POLS) hypothesis predicts that individuals also differ in their 'paces-of-life' and those that have faster paces-of-life grow faster, have shorter lives, reproduce earlier, have faster metabolic rates, and also exhibit riskier behaviors, compared to individuals with slower paces-of-life (Montiglio et al., 2018; Réale et al., 2010; Wolf et al., 2007; Figure 1). Originally developed to explain

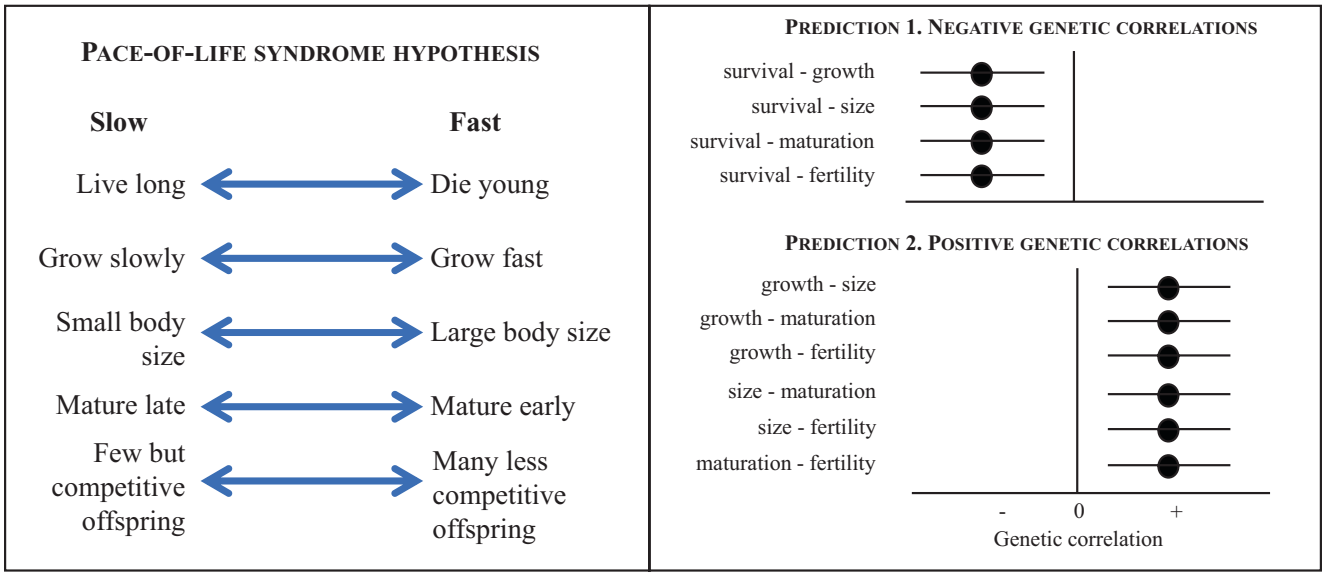

FIGURE 1 Predictions derived from the pace-of-life syndrome hypothesis for the direction of the genetic correlations between five key life-history traits.

variation at the among-species level, life-history trade-offs are thus also invoked as evolutionary explanations for the maintenance of individual variation in whole suites of traits including life-history, physiological, and behavioral traits at the within-species level.

A key assumption in explaining trade-offs among life-history traits is that individuals have limited resources, creating resource allocation compromises. Importantly, resolutions to these allocation challenges are predicted to be resolved at the genetic level: traits that allow individuals to invest more heavily in current fitness goals (e.g., higher growth rates) are predicted to come at the cost of future investments (e.g., lower future survival rate, resulting in a shorter lifespan). These negative correlations can come about through shifts in genetic architecture from antagonistic pleiotropy or linkage disequilibrium. Recent meta-analyses summarizing studies of phenotypic correlations between life-history and behavioral traits have, however, shown a lack of general agreement in the direction of these correlations (Moiron et al., 2020; Royauté et al., 2018). In fact, Haave-Audet et al.'s meta-analysis found a positive, instead of negative, overall phenotypic correlation between survival and reproduction (2022). While this may appear counter-intuitive, theory demonstrates that even if mechanistic trade-offs exist at the genetic level, correlations at the phenotypic level can appear as positive or zero if individuals have differential resource acquisition (van Noordwijk & de Jong, 1986). Increasing resource acquisition can allow some individuals to acquire more, or better quality, resources than others in absolute terms, allowing them to both grow faster and live longer than individuals with fewer or poorer overall resources (Laskowski et al., 2021; Reznick et al., 2000). This can lead to a positive correlation at the among-individual level, even if an allocation trade-off exists at the additive genetic level. Importantly, manipulating or controlling resource acquisition is rare in most empirical studies. It is largely impossible in most field studies, and under laboratory settings food resources are typically provided *ab libitum* meaning individuals may not be faced with limiting resources at all, further obscuring the apparent presence of functional allocation trade-offs. Therefore, the key assumption of the pace-of-life syndrome hypothesis relies on the presence of functional trade-offs among life-history traits, which is best tested at the genetic level.

Many studies have quantified genetic correlations among life-history traits; however, the magnitude and general direction of these correlations is not yet clear. The most recent meta-analysis on genetic correlations among life-history traits was performed in 1996 (Roff, 1996), and it showed that while the overall genetic correlation between life-history traits was positive, there was a greater proportion of correlations that were negative compared to correlations between other traits such as morphology or behavior, suggesting that genetic trade-offs may be more likely between life-history traits. Nearly 30 years later, our goal is to update and expand on this previous work to explicitly test whether key life-history traits exhibit genetic trade-offs, the key assumption of the pace-of-life syndrome hypothesis explaining maintenance of phenotypic variation at the within-species level and life-history theory more generally. We expect to see negative genetic correlations between traits related to survival and reproduction, and positive correlations between traits that contribute to similar fitness proxies such as between growth rates and rate of sexual maturation (i.e., faster growth will correlate positively with earlier sexual maturation; Figure 1).

METHODS

We compiled genetic correlations among life-history traits from studies published since 1995 as we assumed studies published before were included in Roff (1996). We focused on five key life-history traits: survival (e.g., longevity), growth rate (e.g., change in the body size between developmental intervals), body size, maturation rate (e.g., reversed age to maturation), and fertility (e.g., number of offspring). We recorded body size because it could reflect growth in some cases (e.g., higher growth rate leads to larger body size within the same time interval). We predicted an overall negative genetic correlation between survival and these other life-history traits such that increases in survival or longevity are associated with slower growth rates, slower rates of sexual maturation and lower fertility (prediction 1, Figure 1), and a positive genetic correlation between other life-history traits (prediction 2, Figure 1) such that faster growth rates, faster rates of sexual maturation and larger body sizes would all be associated positively with each other and with greater fertility. We also explored several moderators potentially influencing the magnitude and direction of the genetic correlations, including sex (i.e., male, female, both), life stage (i.e., adults, non-adults, cross), experimental design (i.e., family design, pedigrees, genetic lines), lab vs. field studies, and narrow- vs. broad-sense estimates. We included sex as a potential moderator because selection pressures often differ between males and females (Janicke et al., 2016; Winkler et al., 2021) though the predicted direction of these effects on the genetic correlations between life-history traits could be equivocal given that both sexes need to economize their resources to the same extent. On the one hand, we may expect stronger genetic correlations in females, if we consider that they invest more heavily in their reproduction through the production of larger gametes, but on the other hand, in some species, males invest heavily in secondary sexual characteristics and may thus show tighter trade-offs among life-history traits. We also tested for effects of life stage (juvenile vs. adult) as selection pressures may be stronger on juveniles before they have had a chance to reproduce. We included lab vs. field setting as a moderator because individuals might be exposed to different environments depending on the experimental conditions (e.g., presence of predators or more limiting resources in field studies). Finally, we also included experimental design and narrow- vs. broad-sense estimates as moderators to explore whether they may influence the magnitude of the genetic correlations and the uncertainty of the estimates.

Study selection, eligibility criteria, and data collection

We performed a systematic literature review following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines in ecology and evolutionary biology (O'Dea et al. 2021). We performed our search in *Scopus* and *Web of Science* in June 2021, and included articles published from 1995 on. In *Scopus*, we used the following search string: *TITLE-ABS-KEY("life-histor*" OR "life histor*") AND ("genetic" AND "correlate*" OR "covar*")*. We restricted subject area to Agricultural and Biological Sciences, Biochemistry, Genetics, and Molecular Biology, Environmental Science, and Neuroscience. In *Web of Science*, we covered the following databases: Science Citation Index Expanded—1945-present, Social Sciences Citation Index—1956-present, Arts &Humanities Citation Index—1975-present, Conference Proceedings Citation Index-Science—1990-present, Conference Proceedings Citation Index—Social Science & Humanities—1990-present, Book Citation Index—Science—2005-present, Book Citation Index—Social Sciences & Humanities—2005-present, and Emerging Sources Citation Index—2015-present; and our search string was: *TS = ("life-histor*" OR "life histor*") AND("genetic" AND "correlate*" OR "covar*")*. We restricted subject area to Ecology, Evolutionary Biology, Genetics heredity, Zoology, Marine freshwater biology, Biology, Fisheries, Behavioral sciences, Biodiversity Conservation, Environmental Sciences, Entomology, Ornithology, Physiology, Mathematical Computational Biology, Parasitology, Limnology, Developmental Biology, Toxicology, Demography, Endocrinology Metabolism, Neurosciences, Anatomy Morphology, Infectious Biseases, Paleontology, and Reproductive Biology. We limited our search to papers published in English.

The title and abstract of all studies ($n = 3490$) were independently screened for eligibility by three authors (K.L.L., M.M., and P.T.M.) using the software Rayyan (Ouzzani et al., 2016) and using the following inclusion/exclusion criteria: the study should (1) be empirical, (2) use non-domesticated animals (studies on humans were also excluded), (3) include at least one life-history trait at any life stage, e.g., survival, fertility, growth rate, body size, maturation rate, or any other fitness proxy, and (4) explicitly mention quantitative genetic components such as heritability or genetic variance, but excluding fixation index (FST), heterozygosity matrix, and SNP polymorphism. In addition, (5) we excluded studies that measured the genetic components at the population or species level. To increase the reproducibility and reliability of the process, three authors (K.L.L., M.M., and P.T.M.) screened the titles and abstracts of the same 100 studies to calibrate the agreement on the inclusion/exclusion criteria before proceeding with the screening of the remaining 3390 studies.

All studies that passed the title-and-abstract screening ($n = 433$) were full-text screened by one author (C.C.), but prior to that, three authors (C.C., K.L.L. and M.M.) calibrated the agreement on the full-text inclusion/exclusion criteria using 50 studies. For the full-text screening

we had an additional set of five inclusion/exclusion criteria in addition to the title-and-abstract ones (1–5). We excluded studies that: (6) only studied one life-history trait measurement or only multiple measurements on body size proxies, (7) did not report genetic correlations or covariances between life-history traits, (8) measured life-history traits under extreme conditions, such as extreme temperature or humidity, under starvation, or pathogen infection, because traits measured under extreme conditions might mostly reflect physiological responses to stress; and (9) used hybrid animals (e.g., mule). Lastly, (10) we excluded genetic correlations measured across environments or across sexes as it is unclear how we would expect the genetic correlation to change across contexts (e.g., Sgrò & Hoffmann, 2004). Data for all studies that passed the full-text screening ($n = 151$) were extracted by one author (C.C.), but only after three authors (K.L., M.M., and A.S-T) had double-checked 5 studies each to ensure the reliability of the data extraction procedure. The PRISMA flowchart showing the number of studies included and excluded, and the exclusion reasons at each stage of the systematic review are shown as Figure S1. The full list of included and excluded studies is available in Data S1. The checklist from PRISMA-EcoEvo is available in Data S2. The full dataset used in our analyses is available in Data S3 and S4 (meta-data). Note S1 includes the knit Rmarkdown file re-creating all results presented in the manuscript; Note S2 presents a sensitivity analysis (see section 'Calculation of effect sizes and sampling variances'). All these data are also deposited online at https://doi.org/10.5281/zenodo.8075879.

Data coding

Proxies and trait categorization

For each genetic correlation, we recorded the life-history traits involved and categorized them as: survival, growth rate, body size, maturation rate, or fertility (Table 1). We excluded measures that combined more than one life-history trait (e.g., survival and fertility combined in a principal component analysis). To make genetic correlations comparable across studies, their signs were coded so that a positive genetic correlation represented that a genetic basis with a positive effect on one life-history trait also has a positive effect on the other trait (i.e., survive longer, reproduce more, grow faster, mature earlier, and bigger body size), whereas a negative correlation represented that the genetic basis that benefits one trait has a cost to the other trait. For example, higher mortality means lower survival, thus, we reversed the sign of any genetic correlation between mortality and number of offspring, but not for those between longevity and number of offspring.

Field or lab

We recorded whether the experiment was conducted in the field or in the lab (including any artificial environments such as outdoor tanks and enclosures).

Experimental design

We categorized the experimental design of each study into three: genetic lines, family design, or pedigree. Genetic lines included studies using clones or genotypes, whereas family designs included half- and full-sib designs and parent-offspring pairs. We considered studies using individual information from a pedigree (e.g., relatedness matrix using data from parents and grandparents) as a pedigree design. Design was used to determine the unit of replication at which to calculate the sampling variance of each genetic correlation (see below).

Sample size

We recorded sample sizes at multiple levels if provided, including number of: (i) families/dams/sires, (ii) individuals or offspring, and (iii) genetic lines or clones. If only degrees of freedom were provided, we decided to assign sample size as the degrees of freedom plus one for all models regardless of model structure because it

TABLE 1 Categorization of life-history trait proxies.

Traits	Proxies
Survival	Longevity (e.g., days) and mortality (e.g., proportion of individuals who died at a certain time-point)
Growth rate	The change in body size or mass during a time interval (e.g., change in body size per day)
Body size	Body size or weight, or body condition (i.e., weight relative to size) at any life stage, as well as other proxies such as tarsus length in birds or thorax width in insects
Maturation rate	Rate to reach maturation, including development time, pre-adult duration, age at metamorphosis or maturity, and age at first reproduction
Fertility	Direct measures of reproduction, including number of eggs, hatchlings, recruits, and adult offspring, birth rate (e.g., per year), mating success, number of mating events, extra-pair reproduction, and within-pair paternity success We excluded measures that do not directly reflect fertility, such as reproductive tissue size, laying date, mate choice outcome, age at last reproduction, or rate of ageing

was often difficult to determine the exact sample size from degrees of freedom based on model structure (e.g., mixed-effects models).

Narrow- or broad sense

We recorded whether the genetic correlations were calculated as additive genetic correlations (narrow-sense) or broad-sense genetic correlations (additive and non-additive).

Sex

We recorded the sex of the measured individuals (i.e., female or male), using "both" when the authors either included individuals of both sexes or were unable to tell the sexes apart (e.g., measures taken before the individuals have reached adulthood). Note that contrary to the other life-history traits, fertility was mostly a female trait in our database (except for extra-pair and within-pair reproduction, sperm competitiveness, and mating success). In those cases where one of the life-history traits involved in the genetic correlation was measured for "both" sexes and the other trait measured for either females or males only, we used the latter to categorize the genetic correlation as "female" or "male", respectively. We excluded cross-sex (i.e., across males and females) genetic correlations.

Life stage

We recorded the life stage of the measured individuals (i.e., non-adult or adult), using "both" when authors either mixed individuals at both life stages or measured across life stages (from non-adult to adult). Note that the categorization of life stages is strongly linked to the life-history trait itself. For example, fertility can only be measured at the adult stage and maturation rates can only be measured at non-adult stages, whereas longevity proxies could be considered as either non-adult stages (e.g., larval viability) or "both" stages (e.g., longevity). In cases where the trait pairs were measured at different life stages, we assigned the genetic correlation as "cross" life stages. Note also that the life stage variable may be linked with sex; for example, non-adults are likely to be "both" sexes.

Genetic correlation or (co)variance

Our effect sizes of interest for the meta-analytic models were genetic correlations, which we preferentially extracted from the text and tables of the included studies. However, if the information was only provided in figures (e.g., barplots), we used the software WebPlotDigitizer (Rohatgi, 2022) to extract and calculate those genetic correlations. If the study only provided genetic (co)variances, we calculated their corresponding genetic correlations as

$$rG_{xy} = \frac{Cov_{xy}}{\sqrt{\sigma_x^2 \sigma_y^2}} \quad (1)$$

where rG_{xy} is the genetic correlation between life-history trait x and y, and Cov_{xy} is the genetic covariance between them. σ_x^2 and σ_y^2 are the genetic variances of the respective life-history traits.

Other variables

We recorded the year of publication of each study to test for decline effects. We also recorded the year when the experiments took place, the statistical approach used in each study to estimate each genetic correlation (i.e., animal model, family mean correlations, genetic line mean correlations or matrix 'by hand' calculations), and the geographical location.

Calculation of effect sizes and sampling variances

We transformed all genetic correlations (rG_{xy}) to Fisher's Zr (Hedges & Olkin, 1985), which, contrary to the correlations, is unbounded and normally distributed, following:

$$Zr = \frac{1}{2} ln \frac{(1 + rG)}{(1 - rG)} \quad (2)$$

Before applying the Fisher's Zr transformation, we excluded any $rG_{xy} \leq -1$ and ≥ 1 as well as genetic variances <0 from the analyses because (1) these estimates are likely unreliable and (2) the former cannot be transformed to Zr (see Equation (2)). A potential solution could have been to artificially change those ≤ -1 and ≥ 1 values to a value within the $-1<$value <1 bound; however, we decided against it because our choice of value would contribute to substantial noise in the dataset. For example, converting 1 to 0.9 yields a Zr value of 1.47, while converting 1–0.99 yields a Zr value 2.65.

The sampling variance in Zr (Hedges & Olkin, 1985) was calculated as

$$VZr = \frac{1}{(n - 3)} \quad (3)$$

where the sample size (n) was determined based on the type of experimental design (see section '*Design*' and '*Sample*

Size'): (1) For genetic line designs, we used the number of genetic lines as the sample size. When these studies used multiple genetic lines with several crossings within or between lines, we still used the number of genetic lines as the sample size because the genetic lines, instead of the number of families, best captures the amount of genetic variation in the study population that generates the variation among families. (2) For family designs, we used the number of full families as the sample size, but when this was not provided, we used the number of dams, which reflects the number of full families, or if that was not provided either, we used the number of sires. (3) For pedigree designs, we used the number of individuals as the sample size. In cases where a study provided a range for the sample size (e.g., 100–200 individuals), we use the smaller number (i.e., 100) for the analyses to err on the conservative side. Lastly, in cases where the sample sizes differed between the two life-history traits used to calculate the genetic correlation, we used the smaller number (e.g., in a genetic correlation between growth rate and survival, 200 individuals were used to measure growth rate, but only 100 individuals were used for survival, then 100 was used as the sample size for this genetic correlation). As the number of individuals in the pedigree designs tends to be much larger than the number of genetic lines or families, we conducted a sensitivity analysis where the sample sizes for the pedigree designs were natural-log transformed prior to calculating *VZr* (results were robust to this sensitivity analysis; see Supplementary Note).

Meta-analysis

All analyses were performed in R v.4.2.2 (R Core Team, 2021) using the R package 'metafor' v.3.4 (Viechtbauer, 2010). To test our predictions (Figure 1), we ran two sets of analyses, one for survival pairs (Figure 1, Prediction 1), and the other one for non-survival pairs (Figure 1, Prediction 2).

To estimate the overall mean effect size (i.e., the meta-analytic mean) for each prediction, we ran phylogenetic multilevel intercept-only models that included phylogeny, species, study identity, group identity, and a unit-level observation identity as random effects using the function rma.mv() from the R package 'metafor'. We extracted the phylogenetic information from the Open Tree of Life database using the R package 'rotl' v.3.0.11 (Michonneau et al. 2016). We computed branch lengths using the Grafen method with height set to 1 using the R package 'ape' v.5.4.1 (Paradis and Schliep 2019), and the phylogenetic variance–covariance matrix was then added as a random effect to all models. Figure S2 shows the phylogenetic relationship of species. Species was also added as a random effect because studies using the same species are likely to have similar estimates regardless of phylogeny (Cinar et al., 2022). Study identity was added as a random effect because some studies provided multiple genetic correlations. When a study provided multiple genetic correlations for different experiments (e.g., with different environmental conditions), we used group identity to account for such non-independence. Group identity was identical to study identity if the study only provided one genetic correlation for one pair of traits. We included a unit-level observation identity to model within-study or residual variance. For the intercept-only models, we provide Q as a measure of total absolute heterogeneity and I^2 as a measure of total relative heterogeneity, which we also partitioned for each random effect (Nakagawa & Santos, 2012). The 95% confidence intervals (CI) of I^2 were calculated using the function i2_ml() from the R package 'metaAidR' v.0.0.0.900 (Lagisz et al., 2022).

To investigate the sources of heterogeneity observed in the intercept-only models (see Results), we explored several moderators (i.e., variables extracted in the '*Data coding*' section: trait pairs, lab vs. field, experimental design, sexes, narrow- vs. broad-sense, life stages) by running phylogenetic multilevel meta-regressions with the same random effects structure as the intercept-only models. We ran separate meta-regressions for each moderator (i.e., uni-moderator meta-regressions). We did not run meta-regressions with multiple moderators because moderators were often correlated (but see section '*Publication bias*'). For these meta-regressions, we reported the percentage of variation explained by the moderator(s) as R^2_{marginal} (Nakagawa & Schielzeth, 2017), which was calculated using the function r2_ml() from the R package 'orchaRd' v.2.0 (Nakagawa et al., 2021). We performed post hoc tests for moderators having more than two levels using the function linearHypothesis() from the R package 'car' v.3.1.1 (Fox & Weisberg, 2019).

We plotted the results from all the models using the function orchard_plot() from the R package 'orchaRd' v.2.0 (Nakagawa et al., 2021), and reported the estimates with both their 95% CIs and their 95% prediction intervals (PIs). The latter incorporate heterogeneity to show the range of effect sizes to be expected for 95% of similar studies (IntHout et al., 2016).

Some studies calculated multiple genetic correlations from the same exact data using different methodologies (e.g., different analytical approaches). In these cases, we used only one estimate and selected it based on the following order of priority: (1) estimates from the model with the fewest number of variables (i.e., fixed and random effects) included whenever the study provided estimates from models with different model structures; (2) estimates from a model that partitioned genetic variances (i.e., animal models) over estimates solely based on correlations across family means or line means because the latter two could be biased by parental or permanent environmental effects; (3) estimates from the largest dataset provided if the study also provided estimates from subset(s); and (4) we arbitrarily selected the second set of estimates when we could not classify them based on the above criteria ($n = 6$ studies).

Publication bias

We tested for small-study and decline effects, i.e., reduction in effect size over time, by running a total of six meta-analytic models, three for the pairs of survival traits and three for the non-survival pairs. These included phylogenetic multilevel uni-moderator meta-regressions with either standard error (square root of *VZr*) or mean-centered year of publication as the only moderator (Nakagawa et al., 2022) for both survival and non-survival pairs. The random effect structure was identical to the models mentioned above. We also fit 'all-in' models following Nakagawa et al. (2022) which are models that simultaneously include all moderators (pair of traits, lab vs. field, sex, life stage, experimental design, narrow- vs. broad-sense, standard error, and mean-centered year of publication) and corrected for phylogeny to test whether evidence for publication bias remained after accounting for the heterogeneity explained by all our moderators combined.

RESULTS

Our final dataset comprised a total of 1356 genetic correlations from studies published since the seminal Roff (1996) paper.

Of these, 543 were for correlations between survival and other life-history traits, what we will call 'survival pairs' throughout. These estimates came from 58 studies across 37 species (11 classes, Table 2), with insects (k = 405, n = 39 studies) and particularly the fruit fly *Drosophila melanogaster* being the species most commonly studied (k = 153, n = 15 studies). There were a relatively small number of estimates for the genetic correlation between survival and growth (k = 30, n = 8 studies; Figure 2).

TABLE 2 Total number of correlations and studies (in parentheses) included within each animal taxon.

	Pairs of survival traits	Pairs of non-survival traits
Actinopterygii	32 (3)	132 (11)
Amphibia	—	50 (8)
Appendicularia	4 (1)	31 (1)
Aves	8 (3)	4 (1)
Bivalvia	20 (1)	13 (4)
Branchiopoda	10 (1)	17 (2)
Chromadorea	41 (4)	14 (3)
Collembola	1 (1)	10 (2)
Gastropoda	4 (1)	12 (2)
Insecta	405 (39)	528 (66)
Lepidosauria	2 (1)	6 (2)
Mammalia	16 (3)	24 (6)

Counter to the key assumption of the pace-of-life syndrome hypothesis, we did not find support for an overall negative genetic correlation between survival and other life-history traits, but instead, an overall positive genetic correlation (Zr = 0.19, 95% CI [0.06–0.31], 95% PI [−0.99–1.37], Figure 2a). However, both absolute and relative heterogeneity were high, with 7.6% being attributed to study, 8.7% attributed to experimental group, 17.9% attributed to species, and 64.5% attributed to residual/within-study variance; phylogeny did not account for any heterogeneity (Table 3). We did not detect statistically significant differences among different pairs of life-history traits (genetic correlation between: survival and fertility: 0.22, 95% CI [0.07–0.36]; survival and growth: 0.22, [−0.04–0.49]; survival and maturation: 0.12, [−0.03–0.28]; survival and size: 0.20, [0.04–0.35]; p > 0.34 in all post hoc analyses; Figure 2b, Table S1), and the variation explained by this moderator was negligible ($R^2_{marginal}$ = 0.4%).

The other 813 genetic correlations were estimated between the other life-history traits not including survival, what we will call 'non-survival pairs'. These correlations were collected from 108 studies across 82 species (12 classes, Table 2), with insects (k = 528, n = 66 studies) providing the most estimates. Interestingly, the rainbow trout *Oncorhynchus mykiss* also provided a large number of estimates (k = 97, n = 4 studies). There were relatively few genetic correlations between growth and fertility (k = 17, n = 5 studies; Figure 2). For non-survival life-history traits, we found that the overall genetic correlation between them did not statistically differ from zero (Zr = 0.11, 95% CI [−0.13–0.34], 95% PI [−1.16–1.38], Figure 2c). However, both absolute and relative heterogeneity were also high: 9.8% was attributed to phylogeny, 30.4% attributed to study, and 59.7% attributed to residual/within-study variance; there was no heterogeneity attributable to species or group identity (Table 3). Estimates among different pairs of non-survival life-history traits largely overlapped (correlation between fertility and size: 0.19, 95% CI [−0.01–0.39]; growth and fertility: 0.05, [−0.35–0.46]; growth and maturation: 0.36, [0.09–0.63]; growth and size: 0.16, [−0.08–0.39]; maturation and fertility: 0.19, [−0.02–0.40]; maturation and size: −0.03, [−0.22–0.16]; Figure 2d), although the following comparisons differed statistically: the correlation between fertility and size, maturation and fertility, growth and maturation, growth and size were all significantly larger than the correlation between maturation and size (p = 0.002, p = 0.004, p = 0.0004, and p = 0.03, respectively, Figure 2d, Table S2). The variation explained by the moderator "trait pairs" was relatively small ($R^2_{marginal}$ = 3.5%).

Furthermore, we explored several potential moderators that may explain the high levels of heterogeneity observed for both survival and non-survival pairs. Overall, results were generally consistent across moderator levels for genetic correlations between survival pairs (p > 0.14, Figure 3a; Table S3) and genetic correlations between

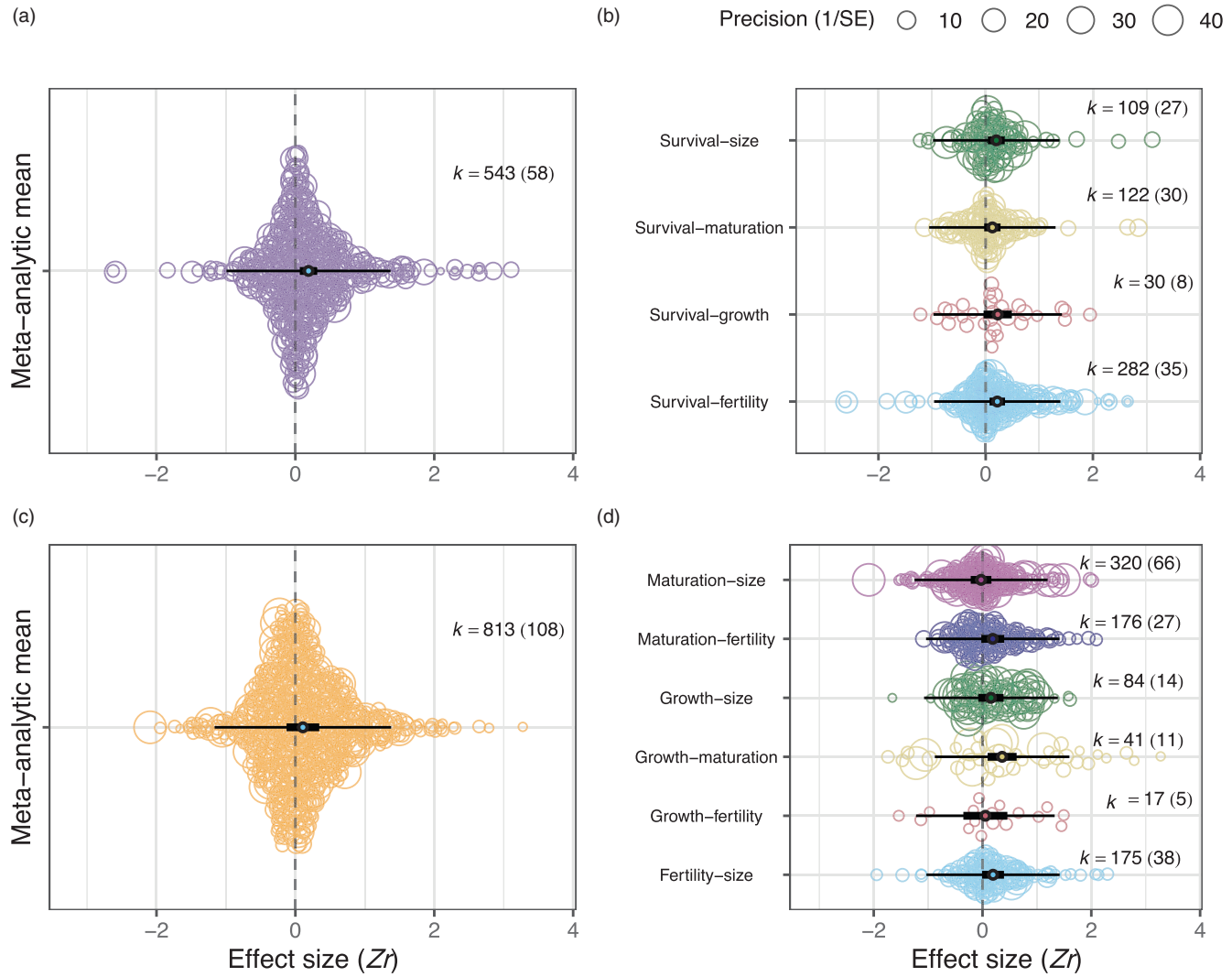

FIGURE 2 The overall genetic correlation between survival and other life-history traits was positive (a) and did not clearly differ among different pairs of traits (b). In contrast, the overall genetic correlation among pairs of non-survival life-history traits was not clearly different from zero (c) and, with a few exceptions, (d) did not clearly differ among the different pairs of traits (see section 'Results'). Orchard plots show the mean estimate, 95% CI (thick whisker) and 95% PI (thin whisker), with dot size being scaled by effect size's precision (i.e., 1/SE). *k* corresponds to the numbers of genetic correlations, with numbers of studies shown in parentheses.

TABLE 3 Absolute (Q) and relative heterogeneities (%, I^2) for the intercept-only models (see section "Methods"). Parentheses show 95% confidence intervals.

	Pairs of survival traits	Pairs of non-survival traits
Q	23,815, $p<0.0001$	430,354, $p<0.0001$
I^2 total	98.7 (98.5–98.8)	99.8 (99.8–99.8)
I^2 species	17.9 (11.4–25.3)	0 (0–0)
I^2 phylogeny	0 (0–0)	9.8 (7.2–12.8)
I^2 study identity	7.6 (5.1–10.5)	30.4 (24.5–36.7)
I^2 group identity	8.7 (6.8–10.8)	0 (0–0)
I^2 unit-level observation identity	64.5 (57.8–70.7)	59.7 (53.9–65.3)

non-survival pairs ($p>0.14$, except for the comparison between adult stages and cross stages [$p=0.02$]; the comparisons between females and males and between family and pedigree designs were marginal [$p=0.054$ and $p=0.08$, respectively], Figure 3b, Table S4). The moderators explained a relatively small amount of variation for survival pairs (lab vs. field: $R^2_{marginal}=1.4\%$; sex: $R^2_{marginal}=0.4\%$; life stage: $R^2_{marginal}=0.09\%$;

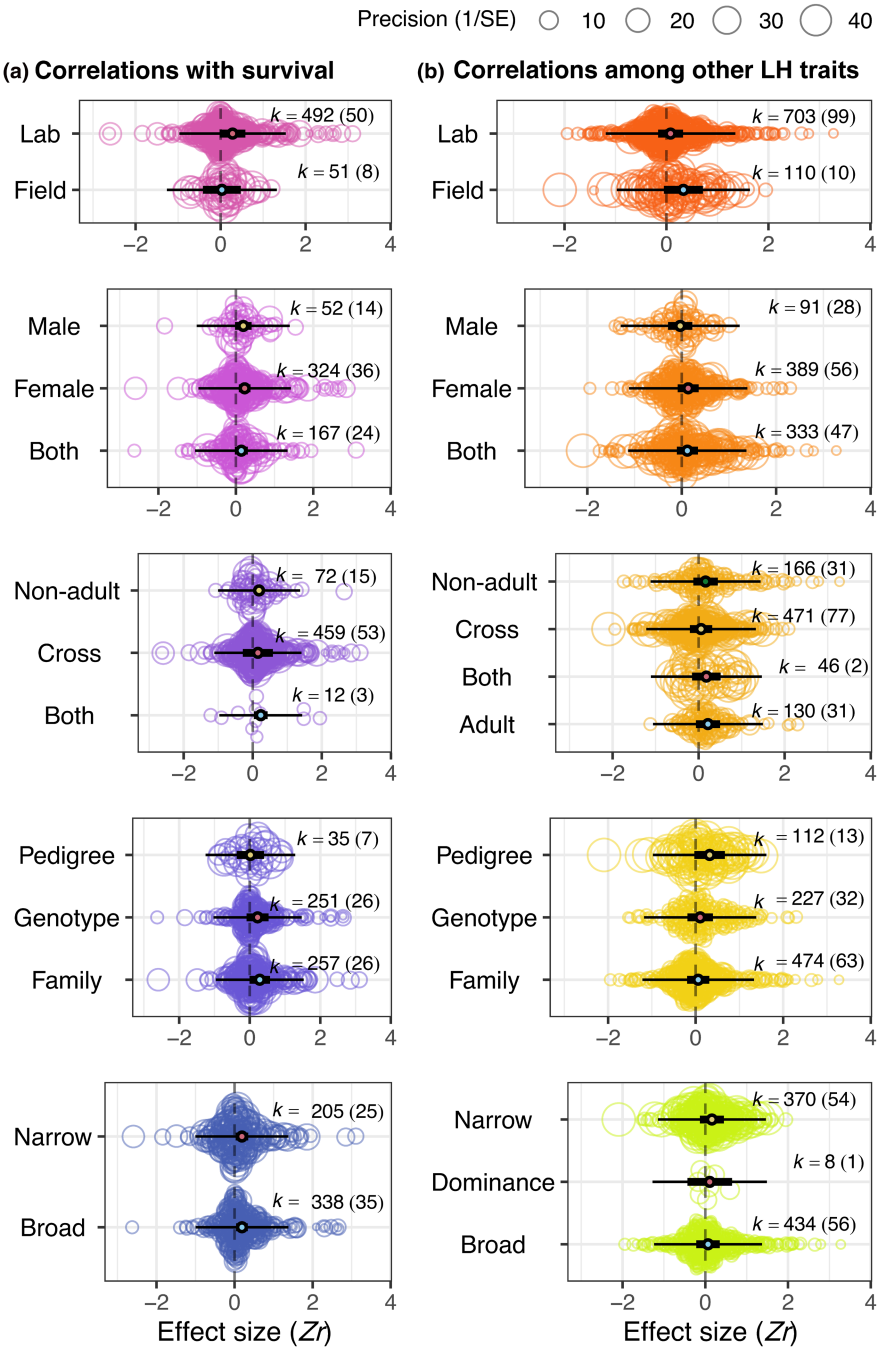

FIGURE 3 Genetic correlations between both survival and other life-history traits (a) and between non-survival life-history traits (b) were not strongly affected by moderators. Orchard plots show the mean estimates, and 95% CI (thick whisker), 95% PI (thin whisker), with dot size being scaled by effect size's precision (i.e., 1/SE). *k* corresponds to the numbers of genetic correlations, with numbers of studies shown in parentheses.

experimental design: $R^2_{marginal}$=1.1%; narrow- vs. broad sense: $R^2_{marginal}$<0.001), and non-survival pairs (lab vs. field: $R^2_{marginal}$=1.8%; sex: $R^2_{marginal}$=0.7%; life stage $R^2_{marginal}$=1.0%; experimental design: $R^2_{marginal}$=1.8%; narrow- vs. broad-sense: $R^2_{marginal}$=0.5%).

We detected little evidence of small-study effects in both survival pairs (slope of SE=0.42, 95% CI [−0.20–1.05]; overall meta-analytic mean=0.11, [−0.05–0.28]; p=0.19; $R^2_{marginal}$=1.1%; Figure 4a) and non-survival pairs (slope of SE=−0.45, [−1.06–0.16]; overall meta-analytic mean=0.19, [−0.09–0.48]; p=0.15; $R^2_{marginal}$=1.1%; Figure 4b). Evidence for an overall decline in the genetic correlation over time was also seemingly not present for survival pairs (slope of publication year=0.05, [−0.04–0.13]; overall meta-analytic mean=0.18, [0.05–0.31]; p=0.27; $R^2_{marginal}$=0.6%; Figure 4c) and non-survival pairs and (slope of publication year=0.06, [−0.02–0.15]; overall meta-analytic mean=0.1, [−0.11–0.31]; p=0.15;

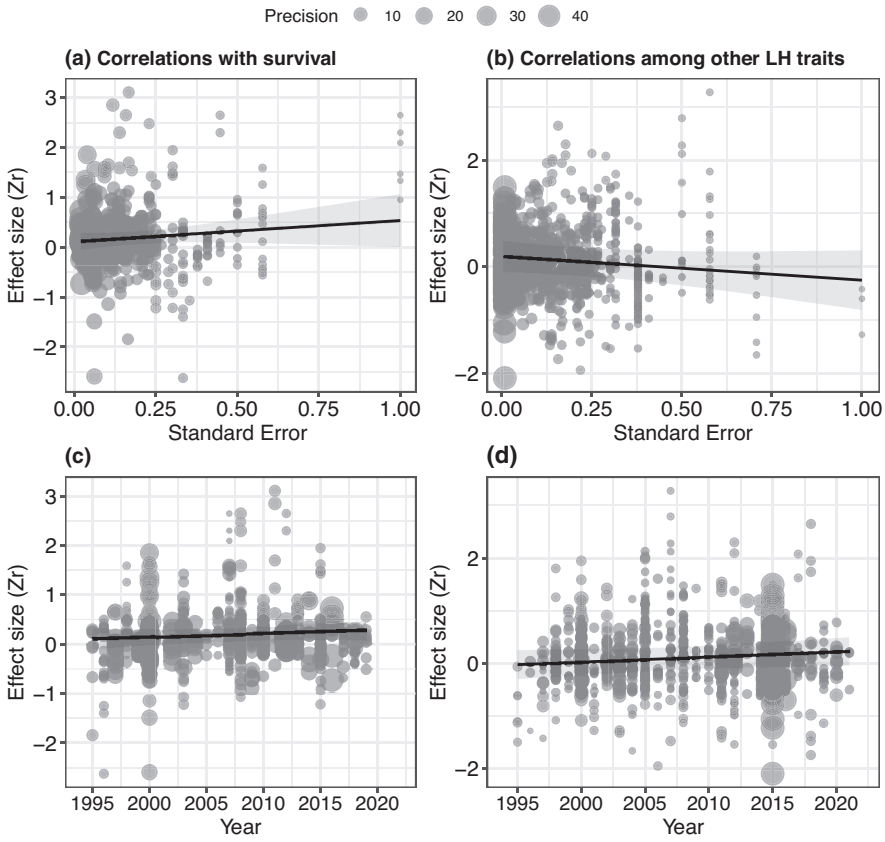

FIGURE 4 Genetic correlations for pairs of survival traits and pairs of non-survival traits were not clearly associated with their standard error (i.e., no clear evidence of small-study effects; a, b), and there was no clear evidence of effect sizes declining over time (c, d). The solid lines are the model estimate, shaded areas are the 95% CI, with the size of the circles being scaled by their precision (i.e., 1/SE).

R^2_{marginal} = 1.0%; Figure 4d). These results were confirmed by the 'all-in' models (see Table S5).

DISCUSSION

Our meta-analysis indicates a lack of strong evidence for the appearance of genetic trade-offs between life-history traits at the within-species level. In contrast, we detected an overall positive genetic correlation between survival and other life-history traits; that is, individuals who live longer tend to also have higher performance at other life-history traits collectively (i.e., grow faster, mature earlier, and have more offspring), although the magnitude of this genetic correlation was rather modest (meta-analytic mean = 0.19 and 95% CI [0.06–0.31]) with large heterogeneity. This result generally suggests a lack of 'paces of life' at the genetic level and is aligned with findings from a previous meta-analysis showing a positive average *phenotypic* correlation between survival and fertility (Haave-Audet et al., 2022). In all, this means that, based on current evidence, the key assumption underpinning the pace-of-life syndrome hypothesis – live fast and die young – is not well supported, or at the very least, not easily observable, calling into question the adequacy of this often well-accepted hypothesis as an explanation for the existence and maintenance of individual differences in behavioral and physiological traits at the within-species level.

Life-history theory was originally developed to explain variation at the among-species level: species differ in how they resolve resource allocation trade-offs generating differences in 'paces of life' (Stearns, 1989). The pace-of-life syndrome hypothesis builds on this theory to predict that behavioral traits, especially those related to risk-taking, and physiological traits are key to resolving this trade-off, thus providing an explanation for the maintenance of phenotypic variation at the within-population level (Réale et al., 2010). In direct contrast to one of the key assumptions of life history theory generally and the pace-of-life syndrome hypothesis specifically, our meta-analysis shows no strong evidence for the expected genetic trade-offs but instead, an overall positive genetic correlation between survival and other life-history traits.

Charnov (1989) showed that for simple two trait models, a negative genetic correlation can be a good indicator of a functional trade-off (i.e., differences in allocation). However, later models that explicitly modeled the relationships between many traits showed that this need not always be the case. First, genetic variation for resource acquisition may produce positive genetic correlations

(van Noordwijk & de Jong, 1986) as some individuals can then allocate more in absolute terms to many traits; the 'big house, big cars' analogy (Reznick et al., 2000). If there are more genetic variants that contribute to variation in resource acquisition than resource allocation, Houle's model showed that mutation-selection balance alone is sufficient to produce positive genetic correlations (1991). These positive correlations may also be expected to be more evident when resources are abundant such as in lab settings where most animals are typically fed *ab libitum*. Indeed, we found a tendency for correlations between survival and other life-history traits collected in lab-based studies to be more positive compared to correlations collected from field studies. Though this comparison between lab and field-based studies should be interpreted very cautiously given that the vast majority of our compiled estimates (492 out of 553) were conducted in lab settings so this could potentially be due to sampling bias. Estimating genetic correlations under limiting resource conditions may better reveal functional trade-offs.

Differences in resource acquisition among individuals have been highlighted in classic life-history theory as potentially obscuring the presence of within-individual, that is, functional allocation trade-offs (de Jong & van Noordwijk, 1992; Reznick et al., 2000; van Noordwijk & de Jong, 1986). Variation in resource acquisition is likely especially relevant when considering the Pace-of-life syndrome hypothesis, which explicitly deals with among-individual variation in behavioral traits. The pace-of-life syndrome hypothesis predicts that behavior helps mediate trade-offs (e.g. risky behaviors can help an animal gather resources to fuel current reproduction but in doing so expose itself to greater mortality risk), but it may be that an individual's behavior is more tightly linked to its acquisition strategies rather than its allocation strategies (Laskowski et al., 2021). This is especially relevant because, while there is good evidence for trade-offs among life-history strategies at the species level (Healy et al., 2019; Promislow & Harvey, 1990), it seems unlikely that a single species would harbor the same level of variation in the key behavioral or physiological traits that moderate allocation trade-offs as is present across a large number of species (Stearns & Rodrigues, 2020; White & Seymour, 2004). Together with results from multiple previous meta-analyses testing for the predictions of the pace-of-life syndrome hypothesis (Haave-Audet et al., 2022; Moiron et al., 2020; Royauté et al., 2018), empirical evidence on individual differences in resource allocation strategy driving individual differences in behavior appears to be weak, at best.

Once resources are acquired, complex genetic relationships between traits and how those resources are allocated can further obscure functional trade-offs. The fitness of an individual will be determined by all traits of an individual; however, most studies, necessarily, often measure just a few. This may be problematic because correlations with unmeasured traits and the relationships between suites of traits can produce positive or negative correlations depending on the relationship (Charlesworth, 1990; de Jong, 1993; de Jong & van Noordwijk, 1992). For instance, a genetic correlation between two life-history traits may not be representative of the underlying functional trade-off if the measured traits interact in a more complex manner than a simple bivariate relationship. The bivariate analyses typically used to estimate genetic correlations do not take into account how the two measured traits might also be related to other (unmeasured or not statistically modelled) life-history traits, ignoring important biological complexity that can ultimately obscure the appearance of genetic correlations (Charlesworth, 1990). Furthermore, De Jong provided a model showing that the order in which resources are allocated between traits can alter the genetic correlation between those traits: initial allocation decisions can generate negative correlations between traits but subsequent sub-allocations can generate positive correlations (1993). Houle (1991) also highlighted how differences in the number of loci underpinning resource acquisition and allocation traits can obscure the appearance of negative genetic correlations as evidence for functional trade-offs, especially when the number of loci underpinning resource acquisition traits is bigger than that in allocation traits and there is little pleiotropy between them. Altogether, this does not necessarily mean that functional trade-offs do not exist, but that just sampling a few traits and fitting them to simple bivariate analyses may not provide the whole picture and make observing the expected trade-offs exceedingly difficult.

In addition to the genetic complexity interlinking traits, it is important to note that these genetic relationships can also be responsive to changes in the environment. Life-history traits are highly responsive to the environment (Acasuso-Rivero et al., 2019), and if individual reaction norms cross, the sign of the genetic correlation can even reverse (Sgrò & Hoffmann, 2004; Stearns et al., 1991). For example, in one environment, genotype A may have higher growth and survival than genotype B (i.e., positive genetic correlation), yet in another environment, genotype A has higher growth but lower survival than genotype B (i.e., negative genetic correlation), thus causing the sign of the overall genetic correlation to reverse. Resource availability can act as an environmental gradient that causes exactly this (Wright et al., 2019). Salzman et al. (2018) modelled how allocation and acquisition decisions can be modified by environmental conditions changing the expected correlations among traits. Indeed, the genetic correlation between longevity and fecundity has been found to switch from positive to negative under low resource availability (Ernande et al., 2003; Messina & Fry, 2003). Altogether, the genetic correlations between life-history traits may be dynamic depending on the environment or genetic background of the animal.

Finally, it is worth mentioning that while we did not find strong evidence for publication bias, there was some indication that the overall positive genetic correlation we found between survival and other life-history traits may be influenced by small sample size effects. While there was no significant effect of the study's standard error (as a proxy for its precision), including this effect in the model reduced the estimate of our overall meta-analytic mean from 0.19 (95% CI: [0.06–0.31]) in the intercept-only model to 0.11 (95% CI: [−0.05 to 0.28]). For non-survival trait pairs, the effect of the standard error was negative, though non-significant, also suggestive of the idea that smaller studies may have been more likely to find (or report) larger effect sizes. Altogether, meta-analyses rely on the quality of the work being analyzed. Coupled together with the high heterogeneity we see in the estimates, we encourage caution in over-generalizing the finding of positive genetic correlations between survival and other life-history traits. It is also worth noting that the vast majority of our correlations between survival and other life-history traits came from studies on invertebrates, and insects (often *Drosophila* fruit flies) in particular. While the genetic tractability of these animal systems makes getting these measures of genetic correlations more feasible, it is possible that this over-representation of a handful of species may limit our ability to generalize these findings to other species with different lifespans, reproductive tactics, or ecologies generally.

CONCLUDING REMARKS

Trade-offs between life-history traits are often invoked as evolutionary mechanisms underlying within-species differences in behavioral and physiological traits, ultimately, with fitness consequences. However, our meta-analysis reveals no strong evidence for the expected overall negative genetic correlation, and instead, it shows evidence for an overall positive genetic correlation. This suggests that genetically based resource allocation trade-offs between life-history traits may not be as common, or at least as commonly observable, as is often assumed. Variation in resource acquisition and/or relationships with unmeasured traits may be obscuring the expected functional trade-offs. Ultimately, our results confirm once again that the jury is still out regarding the validity of the pace-of-life syndrome hypothesis, as it is currently conceived, as an explanation for the ubiquitous existence of individual differences in behavioral and physiological traits at the within-species level. We encourage a renewed focus on investigating the mechanisms underlying such individual differences, manipulative experiments to tease apart such mechanisms, and the development of formal theory to generate quantitative predictions about the relationships we expect to see among relevant traits and the conditions under which we expect them.

AUTHOR CONTRIBUTIONS

M.M., K.L.L., and P.T.N. designed the study. C.C, M.M., and K.L.L performed the literature review. C.C. extracted genetic correlations and performed meta-analysis. A.S-T provided substantial comments and feedback on the meta-analysis as well as revised and confirmed the reproducibility of the code and results. C.C. and K.L.L. wrote the manuscript. All authors revised and approved the final version of the manuscript. The authors declare no conflicts of interest.

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PEER REVIEW

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DATA AVAILABILITY STATEMENT

The data and code recreating these results are publicly available on Zenodo: https://doi.org/10.5281/zenodo.8075879.

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