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Title:

Behavioural mediators of genetic life-history trade-offs: a test of the pace-of-life syndrome hypothesis in field crickets

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ABSTRACT

The pace-of-life syndrome (POLS) hypothesis predicts associations between life-history and 'risky' behaviours. Individuals with 'fast' lifestyles should develop faster, reproduce earlier, exhibit more risk-prone behaviour, and die sooner than those with 'slow' lifestyles. While support for POLS has been equivocal to date, studies have relied on individual-level (phenotypic) patterns in which genetic trade-offs may be masked by environmental effects on phenotypes. We estimated genetic correlations between life-history (development, lifespan, size) and risky behaviours (exploration, aggression) in a pedigreed population of Mediterranean field crickets (*Gryllus bimaculatus*). Path analyses showed that behaviours mediated some genetic relationships between life-history traits, though not those involved in trade-offs. Thus, while specific predictions of POLS-theory were not supported, genetic integration of behaviour and life-history was present. This implies a major role for risky behaviours in life-history evolution.

INTRODUCTION

Within a population, individuals typically differ in behaviour, physiology, metabolism, and morphology [1–6]. Resource allocation trade-offs among costly traits are often invoked to explain the maintenance of this variation in labile traits, with alternative resolutions to trade-offs predicted to have similar fitness outcomes [2,7]. Known life-history trade-offs are those between current versus future reproduction [8], age versus size at maturity [9], and offspring quantity versus quality [10].

Behaviours are often implicated as mediators of life-history trade-offs [11]. For example, aggressive individuals may acquire more resources to invest in current reproduction at the cost of increased risk of mortality [12]. The integration of behaviour and life-history has come to the foreground through research on 'pace-of-life' syndromes (POLS) [5]. The POLS hypothesis predicts integration of behaviour, life-history, and physiology along a 'slow' to 'fast' continuum. Individuals adopting a 'fast' (vs. 'slow') lifestyle should develop faster, reproduce earlier but live shorter [8]. Predicted mediators of POLS are behaviours facilitating resource acquisition at the cost of reduced longevity ('risky' behaviours: aggressiveness, boldness, exploratory tendency, foraging activity [5,13,14]). POLS theory thus proposes that life-history trade-offs maintain variation in individual behaviour. Empirical studies, however, report variation in the direction, causality, and mechanistic bases of behavioural and life-history integration [5,15–17]. A key outstanding question is whether life-histories vary as a function of behavioural type [13], or whether optimal behaviour varies with life-history strategy. Disentangling these scenarios requires fitting models differing in causal pathways, with tools such as path analyses [18–20].

Support for POLS theory might be ambiguous because predicted relationships are often tested at the individual level, while POLS structure is predicted at the genetic level [5,21]. Individual-level tests assume that among-individual correlations reflect underlying genetic correlations [22] (i.e., the 'phenotypic gambit' [23] or 'Cheverud's conjecture' [24]). Empirical data convincingly invalidate this key assumption [25]. For example, individual differences in resource availability often mask negative genetic correlations between life-history traits caused by trade-offs [26–28]. Therefore, forceful tests of POLS theory should involve the estimation of POLS structure at the genetic level. Surprisingly, few studies have attempted to do so [15,29].

We tested whether behaviours mediated genetic life-history trade-offs as predicted by POLS theory. We measured life-history (development, lifespan, size at maturation) and behaviour (exploration, aggression) in adult males of a pedigreed population of Mediterranean field crickets (*Gryllus bimaculatus*) descended from wild-caught grandparents. We first partitioned the phenotypic matrix (**P**) of life-history traits into permanent environmental (**PE**) and genetic (**G**) effects using the 'animal model' [30,31]. We then applied path analysis at each level to test Cheverud's conjecture [24], which we rejected. We subsequently tested for a trade-off between development time and lifespan [8] and asked whether it was mediated by size at maturity [2,9]. As a next step, we estimated genetic covariances among life-history and 'risky' behaviours (exploratory tendency and aggressiveness). Finally, we compared a set of path models (defined *a priori*) to explain the **G** matrix structure. This allowed us to test whether behaviour mediate genetic life-history trade-offs as proposed by POLS theory as an explanation for the maintenance of individual variation in behaviours in wild populations.

METHODS

Cricket collection, breeding, and housing

100 crickets (34 adult males, 33 adult females, 12 near-final instar males, and 21 near-final instar females) were collected from tomato fields in Italy (Capalbio; 42°42'46.7’ N 11°33'99.3’ E) in July 2013. Captured crickets were housed in a climate controlled room at the Ludwig Maximilians University of Munich (Planegg-Martinsried, Germany) that mimicked climatic conditions in the wild (26°C (±0.5), 65% (±0.5) humidity, 14:10 (h) light:dark photoperiod).

Sexually mature wild-caught individuals from this parental generation were randomly paired 4 days after arrival in the laboratory. A total of 35 males and 35 females produced in total 34 hatching clutches. 40 offspring (F1) were raised per parental pair (1360 offspring in total), from which breeders were randomly selected upon adulthood. We adopted a paternal full-sib/half-sib breeding design [32] for the F1 and F2 (each male fertilized clutches of two females). We used 35 males and 70 females from the F1, and 15 males and 30 females from the F2, resulting in 47 (F2) and 21 (F3) viable full-sib families (see Supplementary Material for further details). The number of adult offspring produced was n = 622 for the F2 and n = 281 for the F3 (mean number per female ± SD: (F2) 8.64 ± 2.46, (F3) 5.51 ± 2.44). Of these, 455 males (F2: 335, F3: 120 individuals) were randomly selected and repeatedly screened for behaviour and morphology.

Life-history traits

We recorded three key life-history traits: size at maturity, development time and lifespan. The right hind femur was measured at final moult and at death (correlation: r = 0.90, n = 2036) with vernier calipers (nearest 0.05 mm) as a proxy of adult size [33]; we reduced measurement error by averaging the two measurements. Development time was defined as the difference between final moult and clutch hatching date (days), and adult lifespan as the difference between final moult and death date (days). Lifespan was recorded for F2 only (F3 animals were euthanized prematurely for logistical reasons).

Experimental protocol

Behavioural trials ran between January and June 2014. Each individual was repeatedly assayed for each of 2 behaviours per day, always in the same order (exploration followed by aggression test) following [34]; this ensured that all were treated the same, facilitating comparisons between individuals [35,36]. Each test was repeated 6 times per individual (approximately one week apart; range 7-9 days), except for 15 F2-individuals that were tested twice, and subsequently used for other purposes. Subjects were marked with coloured tape on the pronotum (red or blue, randomly assigned) the day before a focal trial to facilitate individual recognition during the aggression tests [34]. All adult subjects were housed individually throughout the experiments, interacting only in dyads during the aggression test to avoid familiarization or other carry-over effects due e.g. to social interactions.

Each subject was assigned to a "group" according to age (days post-moulting); aggression tests were conducted solely within groups to avoid age effects [34]. Group members

were tested on the same day (in batches of 8 individuals simultaneously), randomized for time of the day and testing location. We formed 7 groups of 40 individuals (F2), one group of 55 individuals (F2), 3 groups of 40 individuals (F3). Individuals were randomly assigned with an algorithm to one unrelated opponent of the same group for each aggression test. All trials were performed on a rack with two shelves, each equipped with a high-resolution (27.81 frames/s; 1600×1200 pixels) camera (Basler GenICam, Germany) fitted 43 cm above the arena [34]. Cameras connected to a computer and recordings managed using MediaRecorder (Noldus, Netherlands). Few trials were excluded: 31 of 1888 (F2; 1.64%) and 3 of 608 (F3; 0.49%) for exploration, and 27 of 944 (F2; 2.86%) and 5 of 304 (F3; 1.64%) for aggression trials due to technical problems. Note that there are approximately twice as many exploration trials, since two individuals are involved in each aggression test. Total sample size was 2462 exploration (5.27, SD 1.23 per individual) and 1195 aggression tests (5.16, SD 1.28 per individual).

Behavioural trials and scoring

Exploration and aggression behaviour were assayed as detailed in [34] (for an illustration of the setup, see Figure 2 there). Briefly, at the onset of the exploration test, the subject was moved (inside its shelter) from its individual container to the exploration arena (14.5 l × 15.5 w × 9 h cm^3). Exploration activity was subsequently recorded for 30 minutes. Shelters were then removed and subjects given 10 minutes to acclimatize, after which a divider separating the two individuals was lifted (creating an arena sized: 29.5 l × 15.5 w × 9 h cm^3). We then recorded the dyad for 10 minutes (aggression test), after which subjects were returned to their individual containers.

Exploration and aggression videos were analyzed using Ethovision version 11.0 (Noldus, the Netherlands) to track and extract spatial coordinates of each individual in each video frame. Distances were summed to calculate total distance moved (cm) in the exploration test as proxy for 'exploration behaviour' [37]. For the aggression test, total time (s) each individual spent moving towards the opponent ('relative movement' for simplicity) was calculated as a proxy of aggressiveness [38]. We have previously shown that relative movement is an appropriate way to assess aggressiveness in crickets as it effectively captures variation in 'initiating contact' during agonistic interactions [38]. This measure was cross-validated by analyses showing tight correlations with other known expressions of aggression, and with manual scores of relative movement (detailed in [34] and the Supplementary Material). An advantage of relative movement is that it can be readily measured for each of the two contestants, while other metrics (e.g., escalation level of the cricket's stereotyped interactions [39]) are better viewed as

characteristics of the dyad instead of single individuals and are therefore not suitable for our study. Relative movement was measured by summing up only the time spent moving towards the opponent in consecutive samples (frames) where the relative distance between contestants decreased (see Ethovision v11.0 User Manual, Noldus Information Technology 2014). Pilot trials defined 8 cm as a distance where directional movement meaningfully measured initiation of aggressive contact.

Statistical analyses

Univariate models

We conducted four sets of analyses. We first estimated sources of variation in each behavioural (aggression, exploration) and life-history (developmental time, lifespan, adult size) trait by fitting each as the focal response variable of a univariate mixed-effect 'animal' model [30] (incorporating the relatedness matrix calculated from the pedigree). We partitioned the total phenotypic variance (V_P) for each trait into residual within-individual (V_R) and among-individual variance (V_I); the latter was further partitioned into additive genetic (V_A), permanent environment (V_{PE}), and common environment (i.e. container) (V_C) effects.

We included the following fixed effects to control for variation caused by the experimental design not of interest here: test sequence (covariate, range 1-6), time of the day (minutes from midnight, covariate), shelf (category: two levels), and within-shelf arena location (category: four levels). Covariates were mean-centered, such that the fixed-effect intercepts were for their average values [40]. Generation (F2/F3) and clutch number ($1^{st}/2^{nd}$) were also fitted as

covariates (both coded as -0.5 and 0.5, respectively, [41]). Significance of fixed effects is detailed in Table S2 (none were directly relevant to the study thus not discussed further).

We also fitted random intercepts for date (64 levels; aggression and exploration only) and opponent identity (455 levels; aggression only). These two sources of variation are not discussed here for brevity (fully detailed elsewhere [38]). For all traits, we fitted individual (455 levels) and group rearing container identity (120 levels) as random effects, while the residual variance was constrained to zero for traits that were not repeatedly measured (i.e., life-history traits) following [40]. Adjusted individual repeatability [42] was defined as the proportion of phenotypic variance not attributable to fixed effects (V_P) explained by among-individual variance ($V_I = V_{PE} + V_C + V_G$). The proportional contributions of V_A (heritability; h^2), V_{PE} (pe^2) and V_C (c^2) were defined as the focal variance component divided by V_P . Traits were mean-centered and variance standardized. Models were fitted using restricted maximum likelihood, assuming a Gaussian error distribution (confirmed for all response variables using visual inspection of residuals).

Multivariate models

As a second step, we used a multivariate extension of this framework to estimate the phenotypic (**P**), among-individual (**I**), additive genetic (**G**), and permanent environmental (**PE**) matrices. Common environment effects were not modelled (as univariate models showed no effects for most traits). We implemented (i) a model fitting life-history traits only, and (ii) a model fitting all behavioural and life-history traits together. Following [41], we only included fixed (sequence) and random effects that explained significant variation in the univariate

analyses. Within-individual (co)variances were modelled (between the two behaviours) or fixed to zero if not identifiable [31]. Note that environmental sources of covariance among all traits were thus modelled in the **PE** matrix.

Significance testing in mixed-effects models

Statistical significance of fixed effects was tested using numerator and denominator degrees of freedom (df) estimated in ASReml 3.0 [43]. We used likelihood ratio tests (LRTs) to evaluate significance of random effects. This χ^2-distributed test statistic is calculated as twice the difference in log-likelihood between a model where a target random effect was fitted versus not fitted [44]. Probability (P) from the LRT of a single variance component was calculated assuming the distribution of the test statistic is an equal mixture of χ^2_0 and χ^2_1 [45–47].

Path analyses

As a third step, we applied path analyses to the estimated **I**, **G**, and **PE** matrices (life-history traits). Natural selection may often act on traits that have sequential or structured causal relationships with one another, and many biological processes have multiple pathways through which they affect fitness [19]. Path analysis [18] allows the estimation of partial correlation coefficients between two variables while simultaneously controlling for effects of all other variables in the model, making this method a powerful tool to disentangle effects on the relationship among two variables produced by other correlated variables [16]. We applied a single type of path model to the point estimates of the standardized correlation matrix estimated for each hierarchical level to quantify paths connecting life-history traits. We fitted the effect of development time on longevity via size (i.e., an indirect pathway) as well as the

226 residual effect of development time on longevity (i.e., due to the mediating effects of any
227 unmeasured, size-unrelated, variable) (Figure 1)[2,9].

228 As a final step, we investigated the relative fit of six alternative causal models (Figure 2) applied
229 to the **G** matrix estimated among all behavioural and life-history traits together. The following
230 scenarios were considered: behaviours are independent from life-history traits (Figure 2, model
231 1), behaviours drive variation in life-history traits (Figure 2, model 2), behaviours mediate
232 specific relationships between specific life-history traits (Figure 2, models 3, 4, 5), all traits are
233 independent (i.e., uncorrelated; "null" model, not illustrated). Input correlation matrices are
234 printed in Tables S4 and S5. Additional analyses carrying forward uncertainty of correlation
235 estimates are detailed in the Supplementary Material.

236 Path analyses were performed using the 'SEM' package in R 3.1.0 (Team R Core 2012).
237 SEM estimates a coefficient and associated standard error (SE) for each specified path and the
238 Akaike information criterion (AIC) value of the model, which we used to compare model fit
239 [48,49]. AIC values were expressed relative to the model with the lowest AIC value (ΔAIC),
240 representing the best fitting model.

241 **RESULTS**

242 **Univariate analyses**

243 Exploration and aggression harboured among-individual variation (adjusted repeatabilities:
244 0.46, SE 0.03 and 0.17 SE 0.02, respectively; Table 1). All traits harboured additive genetic
245 variance; heritability varied between 0.04 and 0.33 (Table 1). Behaviours also harboured

246 significant among-individual variation not attributable to additive genetic effects (pe^2 range:
247 0.11-0.16). Common environment effects (variance attributable to rearing container) explained
248 variation in size and developmental time, but not in behaviour or lifespan (Table 1). Summary
249 statistics, estimates of fixed effects, and test statistics for random effects are printed in Tables
250 S1, S2, and S3.

251 **Path analyses**

252 Path modelling provided evidence for the expected genetic trade-off between development
253 time and lifespan: genotypes associated with slow development were also predisposed to living
254 longer (Figure 1a). This trade-off was not mediated by size: larger genotypes did live longer as
255 expected but development time did not positively affect size. Importantly, the residual (size-
256 independent) relationship indicative of a life-history trade-off between development time and
257 lifespan was opposite at the PE level (Figure 1b); we therefore reject Cheverud's conjecture.
258 Individual-level patterns did not appropriately reflect patterns at the genetic level (compare
259 Figure 1a with 1c).

260 As a next step, we compared the relative fit of the six path structures investigating the
261 role of behaviour in mediating genetic relationships (Figure 2). Our null model (assuming zero
262 genetic correlations among all traits) fitted the data badly, implying the presence of genetic
263 correlation structures. The model where behaviours mediated the relationship between size at
264 maturity and lifespan (model 4), was unequivocally best supported (Figure 2). This model
265 described a structure where size influenced behaviours, which, in turn affected longevity

(Figure 2, Model 4). Specifically, genotypes predisposed for a larger size were also associated with higher levels of aggression, which positively affected lifespan. Interestingly, individuals with such genotypes were simultaneously predisposed towards lower exploratory tendency, which actually negatively affected lifespan (i.e. antagonistic effects of aggression and exploration on lifespan). The two behaviours were genetically linked only because both were affected by size (as they shared no additional covariance). This model also supported the presence of a direct (size-unrelated) genetic influence of development time on lifespan. The trade-off between early reproduction and longevity was, however, not mediated by behaviour. These findings demonstrate relationships between behaviour and life-history traits but fail to support behaviourally mediated trade-offs between life-history traits.

DISCUSSION

Our study combined path analysis with quantitative genetics to assess the role of behaviour in mediating key life-history trade-offs in Mediterranean field crickets. We detected a genetic trade-off between development time and lifespan [8]. *Contra* POLS theory, this trade-off was not mediated by 'risky' behaviours. Aggressiveness and exploration did mediate the size-lifespan relationship, which was not related to the trade-off between development time and longevity: size affected longevity but development time did not affect size. Thus, an individual's 'personality type' did not determine how it resolved this focal life-history trade-off. Our study therefore does not support the existence of a POL syndrome at the genetic level. As expected, we did find differences in how life history traits were associated at the genetic versus individual level (the latter presenting the normal target of behavioural ecology studies of POLS) due to

permanent environmental effects. This finding implies that caution is required in predicting evolutionary consequences of POLS structures without information on its additive genetic architecture.

Heritability and sources of variation

Both behaviours were repeatable; estimates were in line with meta-analyses [50] and similar to those documented previously in this and other cricket species [34,51–56]. Aggression harboured a relatively low repeatability (0.17), as expected for a trait expressed as part of a social interaction [34]. All traits were significantly heritable (range: 0.04 to 0.33); again, heritability of aggression (0.04) was notably low. The relative magnitude of permanent environment (compared to genetic effects) for behaviours confirms that developmental plasticity plays an important role in shaping individual behaviour, which is typical even under standardized laboratory conditions [57–59]. For example, competitive interactions among siblings housed together during development may have led to social niche specialization [60], generating among-individual (co)variation captured statistically as permanent environment effects (PE). Overall, traits varied across multiple hierarchical (environmental and genetic) levels, giving rise to the possibility of level-specific patterns of covariance, i.e. POLS, which we indeed detected.

Life-history trade-offs

Our findings confirm the existence of an allocation trade-off [8] as development time directly and positively affected lifespan at the genetic level when controlling for size. This indicates that individuals seem to pay a cost for fast development, perhaps due to a 'lower

quality' soma, immune function, or repair mechanisms [61]. This trade-off was not mediated by size, as the predicted trade-off between age and size at maturity [2,9,62] was not supported. Interestingly, we did find evidence for a trade-off between age and size at maturity but in the permanent environment rather than genetic matrix. Furthermore, the size-independent effect of development time on lifespan was negative at the **PE**-level. This level-specific relationship was expected as individuals with abundant resources might both develop fast and live long, thereby masking the trade-off between those two life-history traits at the genetic level [26]. This finding is consistent with the famous 'big cars, big houses' scenario [26] predicting that environmental heterogeneity can mask genetic trade-offs.

Do behaviours mediate life-history trade-offs?

Our analyses implied that risky behaviours did not mediate life-history trade-offs (*contra* POLS theory). Rather, the genetic trade-off between development time and lifespan was independent of risky behaviours and caused by other intrinsic factors not measured in our study (detailed below). Behaviours instead mediated the positive relationship between size and lifespan. Genotypes with high residual reproductive value should invest in improving survival and thus be less (instead of more) willing to take risks [5,13,63,64]. We therefore expected that relatively big individuals (having a relatively 'slow' pace-of-life), would be less aggressive and explorative. The effect of size on exploration was negative as predicted, however, size affected aggression positively. This relationship is inconsistent with POLS theory but does align with studies of contest behaviour, where size often represents a key determinant of competitive

328 ability, and larger size often mediates aggressiveness and dominance. Indeed, various cricket
329 studies are consistent with this alternative explanation [33,34,65].

330 Because both aggression and exploration are assumed to represent 'risky' behaviours,
331 we expected both to influence lifespan in a qualitatively similar way. Aggression and
332 exploration instead affected lifespan in opposite directions: individuals genetically predisposed
333 for higher levels of aggression lived longer, but those genetically predisposed for higher
334 exploratory tendencies lived shorter. This finding highlights the strength of our experimental
335 design, which allowed us to disentangle 'intrinsic' from 'extrinsic' causes of mortality (*sensu*
336 [66,67]). The effect of exploration on lifespan in our study represents mostly the intrinsic effect
337 of behaviour on mortality, because no external factors (e.g., predation, food restrictions,
338 adverse weather) were present. Both the antagonistic pleiotropy [67] and the disposable soma
339 [68] theories of aging address intrinsic mortality. These theories lead to similar predictions in
340 terms of life-history trade-offs between reproduction during early life and allocation to
341 mechanisms favouring somatic maintenance, potentially explaining the negative effect of
342 exploration on lifespan. The case of aggression is different, because individuals did meet
343 opponents and therefore more aggressive individuals were involved in more agonistic
344 interactions and may thus have accumulated more damage, which may in turn have shortened
345 their lifespan, causing extrinsic mortality within this laboratory set-up. This reasoning is
346 consistent with patterns observed at the permanent environment level, where the relationship
347 between the two traits tended to be negative (Figure S1). Surprisingly, aggressiveness instead
348 positively affected lifespan at the genetic level, implying that the intrinsic costs associated with
349 aggression stem from a different mechanism than that for exploration. A possible explanation

for this outcome is provided by the 'coping style' literature [69,70], where research on behavioural stress physiology implies that less aggressive individuals are more reactive to their environment and therefore need a more 'expensive' fine-tuned sensory machinery to respond appropriately to external cues. An interesting idea for future research would thus be to test whether aggressive animals have a lower intrinsic mortality but a higher extrinsic mortality compared to less aggressive conspecifics.

Our study focussed solely on males. We thus note that the extent to which our conclusions also apply to females will depend on whether selection pressures are sex-specific, and whether the sexes share the same quantitative genetics architecture. Elsewhere, we show for the same population that cross-sex genetic correlations for exploration and body weight are tight and not different from the value one [71]. This implies that there is no gene-by-sex interaction (GxS) for these traits, and that quantitative genetics patterns were not sex-specific. By contrast, cross-sex genetic correlations for this population are close to zero for both aggression [71] and survival [72] . Our current knowledge of the sex-specific architecture of cricket traits therefore suggests that the evolutionary repercussions of the genetic structure reported here may not necessarily apply to females. We therefore suggest that future quantitative genetics studies should thus focus on incorporate sex-specificity of genetic architectures in the study of POLS.

Conclusions

The POLS framework has been proposed to explain patterns of among-individual correlations [5], however, the implicit underlying assumption is that life-history trade-offs exist at the

371 genetic level. Our study explicitly tested POLS predictions at the genetic level, demonstrating
372 that genetic trade-offs are indeed masked by environmental effects. Our study further
373 highlights the utility of path analyses to uncover causal relationships between traits that may
374 otherwise remain undetected. For example, our path analysis of the **G** matrix showed that
375 aggressiveness and exploratory tendency both depended on size, and both mediated size-
376 dependent effects on lifespan but not the trade-off between developmental time and lifespan.
377 The evolution of these behaviours may thus be linked despite a zero size-unrelated genetic
378 correlation. This illustrates the importance of (multilevel) path analysis in revealing the
379 biological causal pathways explaining genetic correlations [16]. In conclusion, by combining a
380 quantitative genetics approach with path analysis on behaviour and life-history, we were able
381 to draw novel biological inferences concerning POLS research that would otherwise have
382 remained hidden.

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DATA ACCESSIBILITY

Data is available in the dryad repository (www.datadryad.org): doi:10.5061/dryad.q6n64

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554

TABLES

Table 1. Parameter estimates (with SE) of random effects derived from univariate models for exploration, aggression, size, lifespan, and development time. Random effects are expressed as a proportion of total phenotypic variation not attributable to fixed effects. Among-individual and additive genetic variances represent repeatability and heritability, respectively. Values printed in bold face represent significant effects based on likelihood ratio tests (Table S3).

	Exploration	**Aggression**	**Size**	**Lifespan**	**Development time**
Random effects	**σ^2 (SE)**	**σ^2 (SE)**	**σ^2 (SE)**	**σ^2 (SE)**	**σ^2 (SE)**
Individual	**0.459** (0.030)	**0.173** (0.024)	1.0*	1.0*	1.0*
- Additive genetic	**0.281** (0.085)	**0.039 (**0.027)	**0.320** (0.155)	**0.332 (**0.117)	**0.256 (**0.143)
- Permanent environment	**0.162** (0.062)	**0.112 (**0.029)	**0.513 (**0.122)	**0.668** (0.117)	**0.551 (**0.113)
- Common environment	0.016 (0.024)	0.023 **(**0.017)	**0.167** (0.071)	0.000 (0.000)	**0.193 (**0.069)
Within-individual	0.537 (0.018)	0.816 (0.030)	0.0*	0.0*	0.0*
- Opponent	-	**0.111 (**0.022)	-	-	-
- Date	**0.015** (0.006)	0.008 (0.007)	-	-	-
- Residual	0.526 (0.029)	0.708 (0.031)	-	-	-

*Trait did not vary within individuals; all variance is among-individuals.

561 **FIGURE LEGENDS**

562 **Figure 1.** Path models based on estimated correlations between life-history traits (development
563 time, size, and lifespan) at the (a) additive genetic, (b) permanent environment, and (c)
564 phenotypic levels. One-headed arrows indicate the direction of hypothesized causality.
565 Estimated partial regression coefficients are shown with SEs; bolded paths are statistically
566 supported ($p<0.05$).

567 **Figure 2.** Path models investigating the role of behaviour in mediating genetic life-history trade-
568 offs. One-headed arrows indicate the direction of hypothesized causality, double-headed
569 arrows hypothesized correlations lacking a cause-effect relationship. Estimated partial
570 regression coefficients are shown with SEs; bolded paths are statistically supported($p<0.05$).
571 Each model is presented with its associated (Δ)AIC value; the model with the lowest value
572 (Model 4) is best supported. A null model (all traits independent; model 6) was fitted (ΔAIC =
573 168.14) but not depicted.

574 **FIGURES**

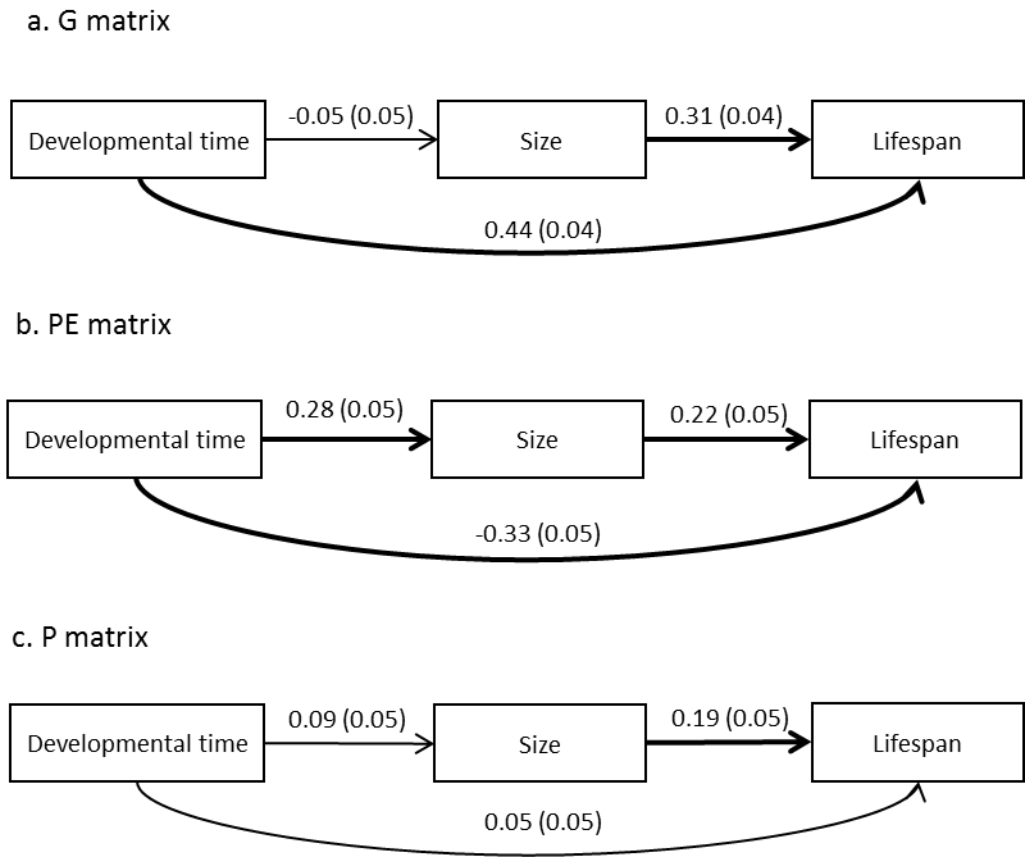

Figure 1.

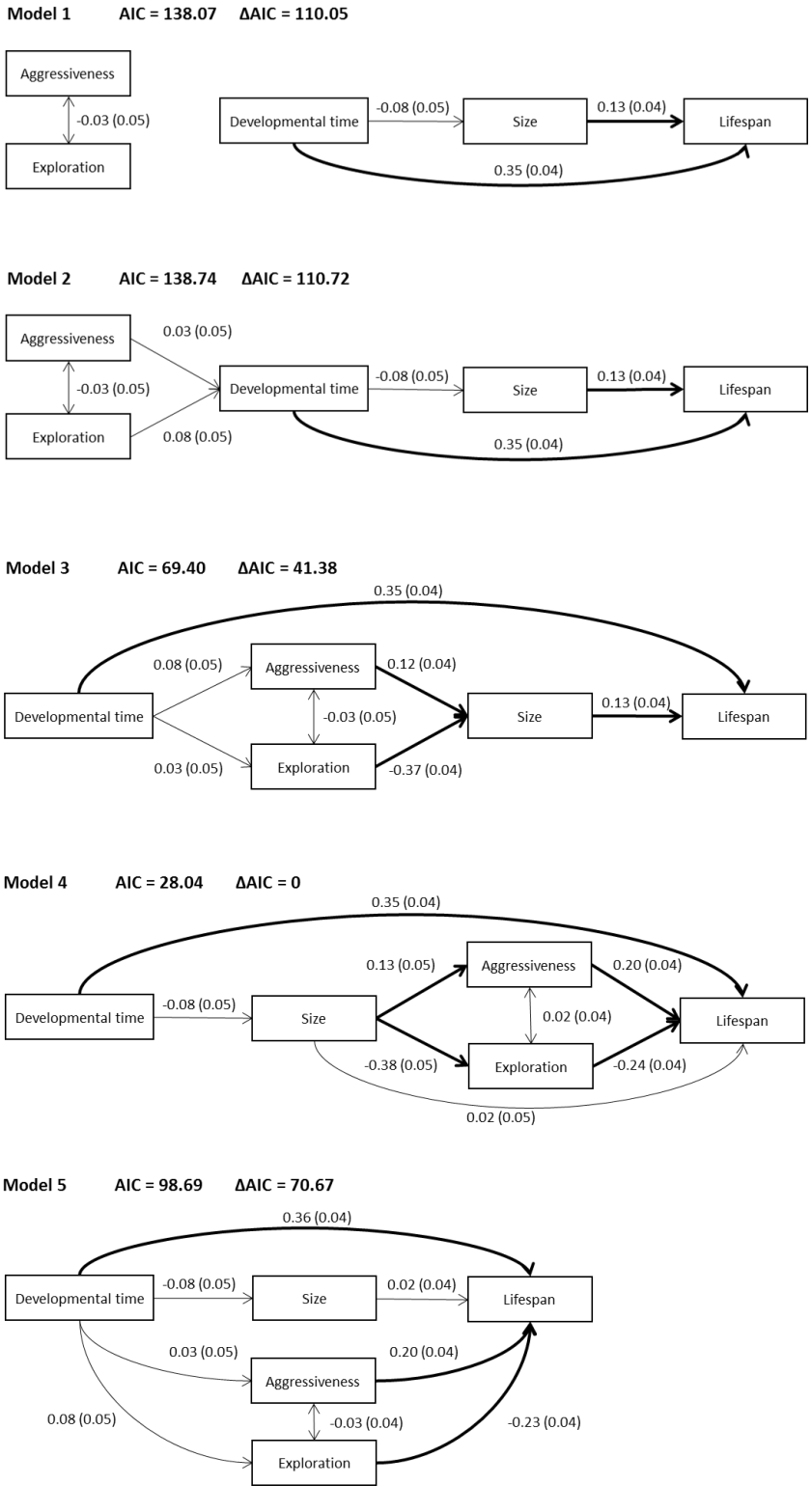
Model 1 AIC = 138.07 ΔAIC = 110.05
Aggressiveness
-0.03 (0.05)
Exploration
Developmental time
-0.08 (0.05)
Size
0.13 (0.04)
Lifespan
0.35 (0.04)
Model 2 AIC = 138.74 ΔAIC = 110.72
Aggressiveness
0.03 (0.05)
-0.03 (0.05)
Exploration
0.08 (0.05)
Developmental time
-0.08 (0.05)
Size
0.13 (0.04)
Lifespan
0.35 (0.04)
Model 3 AIC = 69.40 ΔAIC = 41.38
0.35 (0.04)
0.08 (0.05)
Aggressiveness
0.12 (0.04)
Developmental time
-0.03 (0.05)
Size
0.13 (0.04)
Lifespan
0.03 (0.05)
Exploration
-0.37 (0.04)
Model 4 AIC = 28.04 ΔAIC = 0
0.35 (0.04)
0.13 (0.05)
Aggressiveness
0.20 (0.04)
Developmental time
-0.08 (0.05)
Size
0.02 (0.04)
Lifespan
-0.38 (0.05)
Exploration
-0.24 (0.04)
0.02 (0.05)
Model 5 AIC = 98.69 ΔAIC = 70.67
0.36 (0.04)
Developmental time
-0.08 (0.05)
Size
0.02 (0.04)
Lifespan
0.03 (0.05)
0.20 (0.04)
Aggressiveness
0.08 (0.05)
-0.03 (0.04)
-0.23 (0.04)
Exploration

Figure 2.

SUPPLEMENTARY MATERIAL

Behavioural mediators of genetic life-history trade-offs: a test of the pace-of-life syndrome hypothesis in field crickets

Francesca Santostefano, Alastair J. Wilson, Petri T. Niemelä, and Niels J. Dingemanse

SUPPLEMENTARY TEXT

Breeding and rearing protocol

Each adult male ('sire') was mated twice with each of two unrelated females ('dams') to ensure offspring production with each female in case the first clutch failed. Mating took place inside a plastic box (10×8×14 cm^3) equipped with a cardboard shelter, *ad libitum* food and water, and a plastic cup (diameter × height: 7×4.5 cm^2) filled with moist humus for oviposition. The male was moved after 3 days to the mating box of the second female; at the same time, the oviposition cup of the first female was moved to a plastic box (6×9×9 cm^3), where the eggs hatched on average after 13.04 (SD 2.63) days. Provided that ≥50 offspring hatched from the first clutch, we discarded the second egg batch. If not, we used offspring from the second egg batch for our experiments. 5-6 days following hatching, we counted the nymphs in each box and placed 20 randomly chosen offspring in each of two new plastic rearing boxes (13×15×22 cm^3). In other words, 40 offspring per full-sib family were taken forward. Each rearing box contained a carton shelter, water and food *ad libitum,* and a substrate of fine pebbles and sand. After 5 weeks, containers were checked daily for final-instar nymphs, which were subsequently removed and housed individually awaiting sexual maturation. Adult individuals were housed in isolation in a plastic container (10×10×9 cm^3) with a sand-covered floor and a flow-through plastic netted lid

596 that prevented escape but allowed air circulation. Each container included an artificial, half-
597 cylindrical shelter (6×3.5×2 cm^3), a petri dish (with a diameter of 3.5 cm) with food, and another
598 petri dish with water held within a cotton-plugged vial. Individuals were fed with a mix of dry
599 bird food (Aleckwa Delikat, Germany) and fresh slices of apples *ad libitum*. Food and water
600 were replaced every 3-4 days. Individuals were kept in these same conditions until natural
601 death (F2 generation) or until they were euthanized at the end of the experiment by placing
602 them in a -20°C freezer (F3 generation).

Validation of aggression measurements

The choice of relative movement (defined as the total of time that each individual spent moving towards the opponent within the test, see Main text) as a measure for aggression was taken in steps. First, in another study we scored manually various known metrics of aggression in the closely related species *G. campestris* and asked how they were correlated (detailed in [1]). Briefly, we scored 'approach', 'sing', and 'chase' during an interaction between two males as follows. We scored an individual as 'approaching' during an interaction when it moved towards the other individual from any angle until they came into contact. When only one individual was actively approaching the other (i.e. the other cricket sat still), we assigned the behavior to that individual alone. In cases where both contestants approached each other at the same time, we assigned the behavior to both. Approaching the opponent has been used as a measure of aggression in several studies quantifying aggressiveness in male crickets, sometimes called 'initiating contact' or 'initiating aggression' [2,3]. We also recorded the occurrence of 'singing' during the interaction (following [4]) and 'chasing' the opponent afterwards (e.g., [5–7]). We found that the occurrence of all three behaviours is highly correlated [1]. Viewing aggression as a latent variable, we therefore concluded that approach behaviour represented a reliable observable expression of this behavioural trait. Lastly, our aim was to explore how various candidate metrics (automatically derived from our tracking software) would predict our defined variable for aggression (approach). Amongst the automatically-derived candidate metrics, 'relative movement' provided the highest correlation with this manually scored measure of aggression ($r = 0.85$, 0.03 SE). We therefore selected this metric and validated its correlation with approach, scored manually as defined above, in a randomly chosen subsample of the

current dataset, where the correlation was indeed satisfactory (r= 0.80, 0.06 SE, n = 30 videos). This independent confirmation therefore supported the notion that 'relative movement' represented a reliable measure of aggression, and we relied on this automatically-tracked measure of aggression for the full dataset.

Uncertainty around point estimates of correlations

The path analyses described in the main text were applied to the point estimates of correlations derived from our multivariate animal models. As those estimates come with (substantial) uncertainty, we checked whether taking forward this uncertainty would change the AIC-ranking of the five models, or the point estimates of path coefficients of our best-fitting model. The uncertainty was taken forward by generating 1000 matrices based on the point estimates and associated variances obtained in ASReml. Each path model was subsequently applied to each of these 1000 matrices, and the posterior distribution of the path coefficients, and AIC values associated to each run, extracted. This re-analysis led to the same relative ranking of alternative models and similar point estimates for path coefficients (Results not shown).

SUPPLEMENTARY FIGURES LEGENDS

Figure S1. Path models estimating paths mediating non-genetic relationships between risky behaviors and life-history traits (analysis based on the **PE** correlation matrix; Table S5c). One-headed arrows indicate the direction of hypothesized causal links. Double-headed arrows indicate hypothesized correlations without a hypothesized cause-effect relationship. Estimated partial regression coefficients with correspondent SE are shown with each arrow; bolded arrows represent paths with statistical support ($p<0.05$). Each model is presented with its associated AIC and ΔAIC value. The null model where all traits are independent (model 6) was fitted (ΔAIC = 226.12) but is not depicted graphically here.

SUPPLEMENTARY FIGURES

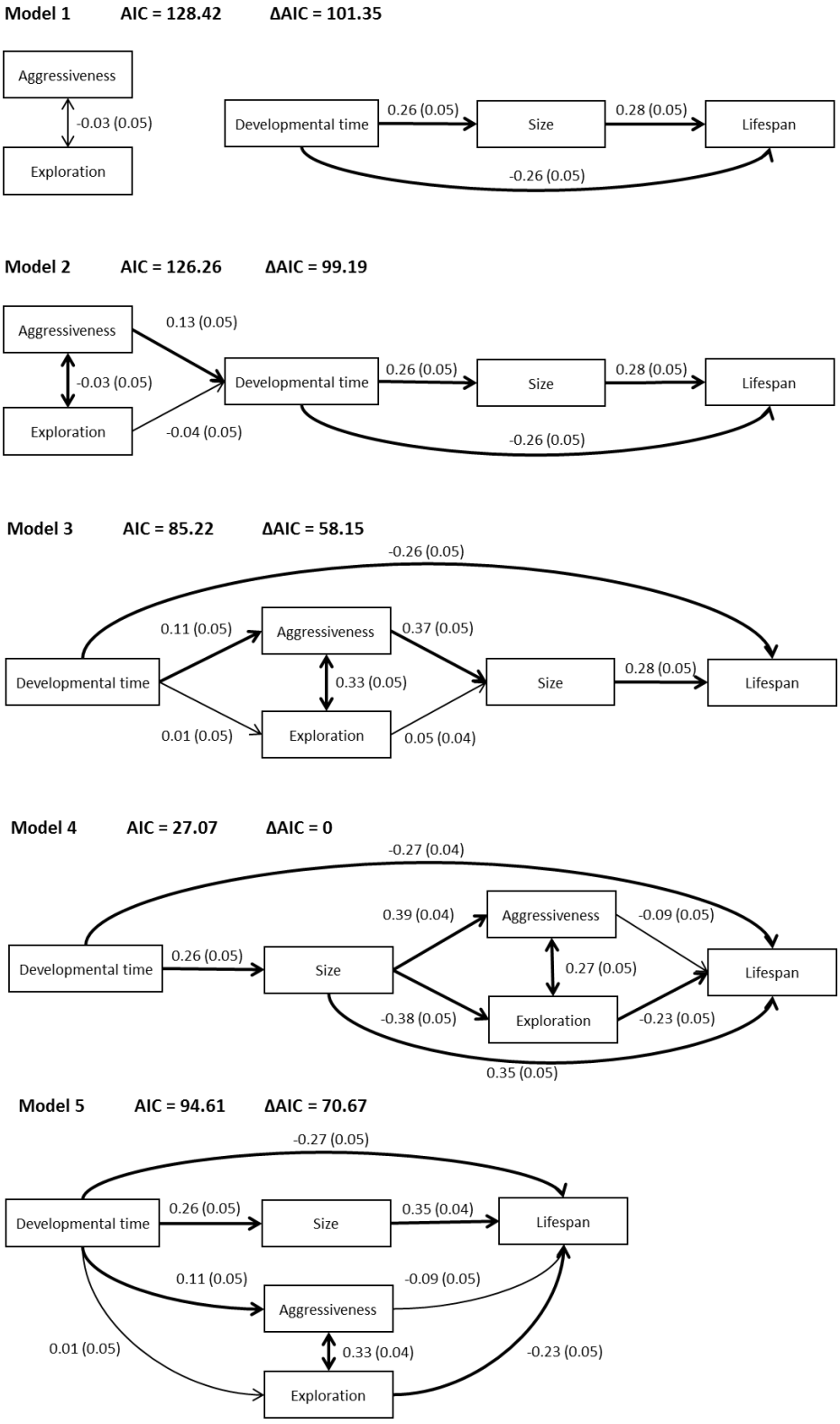

Figure S1.

SUPPLEMENTARY TABLES

Table S1. Summary statistics for the variables used in this study.

Trait	Mean (SD)	Variance	N
Exploration (cm)	923.74 (860.08)	739745.5	2396
Aggression (s)	26.95 (24.69)	609.76	2346
Size (mm)	112.89 (8.26)	68.17	371
Lifespan (days)	85.79 (20.84)	434.42	330
Development time (days)	53.83 (8.26)	68.15	451

Table S2. Parameter estimates (with SE) of fixed effects derived from univariate models detailed in the main text. Values printed in bold face represent significant effects based on Wald F tests.

	Exploration	Aggression	Size	Lifespan	Development time
Fixed effects	**β (SE)**	**β (SE)**	**β (SE)**	**β (SE)**	**β (SE)**
Intercept	0.046 (0.105)	0.053 (0.041)	0.270 (0.114)	0.011 (0.124)	0.312 (0.083)
Sequence	**-0.083** (0.013)	**-0.072** (0.012)	-	-	-
Generation[a]	**0.566** (0.128)	**0.155** (0.078)	**0.599** (0.183)	-	**1.810** (0.132)
Clutch[b]	**-0.337** (0.109)	0.027 (0.069)	**0.397** (0.169)	-0.111 (0.206)	**-0.589** (0.123)
Arena (2)[c]	0.068 (0.045)	0.147 (0.055)	-	-	-
Arena (3)[c]	-0.079 (0.045)	0.103 (0.055)	-	-	-
Arena (4)[c]	-0.007 (0.045)	0.077 (0.055)	-	-	-
Shelf[d]	0.019 (0.032)	**0.190** (0.039)	-	-	-
Time	-0.083 (0.013)	-0.007 (0.020)	-	-	-

[a]Reference is 2nd generation
[b]Reference is 1st clutch
[c]Reference is arena 1
[d]Reference is upper shelf

Table S3. Test statistics associated with random effects estimated in the univariate mixed-effect models detailed in the main text (Table 1). X^2-values, degrees of freedom (df), and values of P are derived from likelihood ratio tests where the full model is compared to one where the random effect of interest was excluded.

	Additive genetic		Permanent Environment		Common Environment		Opponent		Date	
Trait	X^2_{df}	***P***	X^2_{df}	***P***	X^2_{df}	***P***	X^2_{df}	***P***	X^2_{df}	***P***
Exploration	$25.13_{0/1}$	<0.01	$4.54_{0/1}$	<0.05	$0.48_{0/1}$	0.24	-	-	$15.66_{0/1}$	<0.01
Aggression	$2.90_{0/1}$	<0.05	$15.02_{1/2}$	<0.01	$2.26_{0/1}$	0.07	$32.36_{1/2}$	<0.01	$1.50_{0/1}$	0.11
Size	$5.18_{0/1}$	<0.01	$11.17_{0/1}$	<0.01	$8.79_{0/1}$	<0.01	-	-	-	-
Lifespan	$11.42_{0/1}$	<0.01	$17.45_{0/1}$	<0.01	0	1	-	-	-	-
Development Time	$3.17_{0/1}$	<0.05	$11.87_{0/1}$	<0.01	$11.48_{0/1}$	<0.01	-	-	-	-

Table S4. Estimated (a) phenotypic (**P**), (b) permanent environmental (**PE**), and (c) additive genetic (**G**) covariances and correlations (with SEs) between life-history traits (size, development time, and lifespan). Common environment effects were not modelled as univariate models indicated that there was no variation among containers for most of the traits (see Main text). We present covariances (lower-off diagonals) and correlations (upper-off diagonals) for each set of traits.

a. P	**Size**	**Lifespan**	**Developmental time**
Size	-	0.20 (0.06)	0.07 (0.06)
Lifespan	0.18 (0.06)	-	0.09 (0.06)
Developmental time	0.06 (0.04)	0.05 (0.05)	-

b. PE	**Size**	**Lifespan**	**Developmental time**
Size	-	0.13 (0.14)	0.28 (0.17)
Lifespan	0.07 (0.08)	-	-0.27 (0.15)
Developmental time	0.10 (0.06)	-0.12 (0.06)	-

c. G	**Size**	**Lifespan**	**Developmental time**
Size	-	0.29 (0.24)	-0.05 (0.23)
Lifespan	0.13 (0.11)	-	0.43 (0.24)
Developmental time	-0.02 (0.09)	0.14 (0.09)	-

667 **Table S5.** Estimated (a) phenotypic (**P**), (b) among-individual (**I**), (c) permanent environmental (**PE**), and (d) additive genetic (**G**)
668 covariances and correlations (with SE) between two behaviors (aggression and exploration) and life-history traits (development
669 time, lifespan, and size). Common environment effects were not modelled as univariate models indicated that there was no
670 variation among containers for most of the traits (see Main text). We present covariances (lower-off diagonals) and correlations
671 (upper-off diagonals) for each set of traits.

a. P	**Exploration**	**Aggression**	**Size**	**Lifespan**	**Developmental time**
Exploration	-	0.11 (0.02)	-0.15 (0.05)	-0.07 (0.05)	0.04 (0.05)
Aggression	0.11 (0.02)	-	0.16 (0.05)	-0.10 (0.05)	0.02 (0.05)
Size	-0.14 (0.05)	0.15 (0.05)	-	0.19 (0.06)	0.08 (0.06)
Lifespan	-0.07 (0.05)	-0.10 (0.05)	0.02 (0.06)	-	0.08 (0.06)
Development time	0.03 (0.03)	0.02 (0.03)	0.05 (0.04)	0.06 (0.05)	-

b. I	**Exploration**	**Aggression**	**Size**	**Lifespan**	**Developmental time**
Exploration	-	0.14 (0.08)	-0.13 (0.04)	-0.20 (0.04)	0.04 (0.04)
Aggression	0.04 (0.02)	-	0.30 (0.06)	0.03 (0.06)	0.07 (0.06)
Size	-0.08 (0.02)	0.11 (0.02)	-	0.18 (0.06)	0.08 (0.06)
Lifespan	-0.13 (0.03)	0.01 (0.03)	0.16 (0.05)	-	0.07 (0.06)
Development time	0.02 (0.02)	0.02 (0.02)	0.05 (0.04)	0.05(0.05)	-

c. PE	Exploration	Aggression	Size	Lifespan	Developmental time
Exploration	-	0.33 (0.19)	0.17 (0.15)	-0.20 (0.11)	0.01 (0.12)
Aggression	0.04 (0.03)	-	0.39 (0.12)	-0.06 (0.11)	0.11 (0.12)
Size	0.05 (0.03)	0.09 (0.03)	-	0.21 (0.13)	0.26 (0.15)
Lifespan	-0.06 (0.04)	-0.02 (0.03)	0.11 (0.07)	-	-0.19 (0.15)
Development time	0.00 (0.03)	0.02 (0.02)	0.10 (0.05)	-0.09 (0.06)	-

d. G	Exploration	Aggression	Size	Lifespan	Developmental time
Exploration	-	-0.03 (0.23)	-0.38 (0.12)	-0.21 (0.15)	0.08 (0.14)
Aggression	-0.00 (0.03)	-	0.13 (0.19)	0.22 (0.21)	0.03 (0.21)
Size	-0.13 (0.05)	0.02 (0.03)	-	0.11 (0.25)	-0.08 (0.24)
Lifespan	-0.06 (0.05)	0.03 (0.03)	0.04 (0.10)	-	0.34 (0.25)
Development time	0.02 (0.04)	0.00 (0.03)	-0.03 (0.08)	0.10 (0.08)	-

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