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Abstract:

There is increasing evidence that among-individual differences in behavior are, in part, generated by environmental effects. For example, diet quality can have drastically different effects on behavioral variation depending on whether it acts primarily during ontogeny (i.e. as a permanent environmental effect) or has an immediate effect on trait expression as a consequence of energy intake (i.e. temporary source of variation). Moreover, whether diet quality has a stronger effect on a trait's average expression, its variance or its covariance with other traits, remains unclear. We used a 2 × 2 factorial design crossing life-stage (juvenile and adult) and diet quality (low or high-energy content) to disentangle the effects of developmental and adult diets on the expression of behavioral differences. We tested 281 crickets for their activity levels, responses to predator cues, and body-mass. Neither developmental diet nor adulthood diet had any effect on

population means or on the expression of an activity-antipredator response syndrome, suggesting a genetic basis for this syndrome. We did find evidence for increases in the within-individual variance as a result of exposure to a high-quality diet. However, these increases were only found for antipredator response and body-mass. This indicates that diets with higher energy content can increase the potential for behavioral plasticity in antipredator response. In addition to changes in within-individual variation in behavior, diet quality during development also mediated the links between maturation time and exploratory behaviors. More exploratory crickets matured faster when exposed to the low-quality developmental diet, but this relation was absent in the high-quality diet treatment. Our results show that changes in developmental diet quality can mediate the relationship between life-history and behavioral traits later in life.

Keywords: activity, animal personality, antipredator response, behavioral plasticity, behavioral syndromes, developmental environment, environmental matching, phenotypic integration, silver spoon

Introduction

Organisms live in changing environments, experiencing different environmental conditions and selective pressures through their lives. By altering trait expression throughout their lives, e.g. during development versus adulthood, organisms are expected to better match their phenotypes to these environmental changes and thereby increase their fitness. Our understanding of how the environment in which an individual develops affects its fitness is generally based on one of two predictive frameworks: the "silver spoon" model or the "environmental matching" model (Marshall & Uller, 2007; Monaghan, 2008). Under the silver spoon model, offspring born in favorable environments always have higher fitness than individuals born in poor environments, regardless of the adult environment. In contrast, the environmental matching model suggests that experiencing the same environment during development and adulthood should maximize fitness. While the consequences of fluctuating or stable environments on fitness are well understood, how these changes manifest with traits affecting fitness, such as behavior, remains unclear.

While the contrasting predictions of the environmental matching and the silver spoon hypotheses have received some attention in the context of sexually-selected traits (e.g. swordtail ornaments, Basolo, 1998) or mating behaviors (Scharf, Braf, Ifrach, Rosenstein, & Subach, 2015), these studies have typically been restricted to comparing changes in population averages. However, as has been shown in several recent studies, diet manipulations can also have profound effects on trait variances and covariances (DiRienzo & Montiglio, 2016; Han, Jäger, & Dingemanse, 2016; Lichtenstein et al., 2016; Royauté & Dochtermann, 2017). To date the relative contribution of diet quality during developmental *and* adult phases on the generation of among-individual differences in behavior has received little attention (but see Han &

62 Dingemanse, 2017). Moreover, the general framework by which diet quality at different phases
63 of development may affect trait variance and covariance remains, in many cases, unclear.

64 Variation in diet quality during development is particularly interesting because it can lead
65 to long-lasting consequences on an individual's phenotype ("permanent environmental effects",
66 Falconer, Mackay, & Frankham, 1996; Kruuk & Hadfield, 2007; Wilson et al., 2010). Diet
67 quality can also have more immediate effects ("temporary environmental effects", Dingemanse
68 & Dochtermann, 2013, 2014; Kruuk & Hadfield, 2007), particularly during phases of an
69 individual's life-cycle that are energetically costly (e.g. reproduction, exploration of suitable
70 habitats, escape from predators, territorial defense). However, determining how diet quality
71 affects patterns of trait (co)variation among labile traits such as behaviors is challenging because
72 trait (co)variation can be expressed at both among- and within-individual levels (Dingemanse &
73 Dochtermann, 2013; Dingemanse, Dochtermann, & Nakagawa, 2012).

74 Assuming that behavioral flexibility is costly and that the intensity of behavioral
75 expression scales with energy reserves, we should expect individuals to be more constrained in
76 the range of behavioral values they can express when exposed to a low-quality diet (i.e. "cost of
77 plasticity hypothesis", Snell-Rood, 2013). Empirically, this would be detected through lower
78 within-individual variance when exposed to a low-quality diet. By contrast, exposure to a high-
79 quality diet should alleviate the cost of behavioral expression, making individuals more flexible
80 in their behaviors (i.e. within-individual variance should increase). Whether these effects occur
81 during development or adulthood will depend on whether diet acts as a permanent or temporary
82 source of behavioral variation (Figure 1B). Another prediction is that by reducing the amount of
83 energy available to an organism, a poor-quality diet would lead to prioritizing certain functions
84 over others, generating trade-offs. When diet acts primarily as a permanent environment, such

trade-offs are predicted to be manifested at the among-individual level. In contrast, when diet quality has the strongest effect on trait expression as a result of short-term energy intake, trade-offs are predicted to be manifested at the within-individual level (Figure 1C).

Here we investigated how the interaction between developmental and adult diet quality affected variation in and correlations among body mass, activity, and response to cues of predator presence in house crickets (*Acheta domesticus*). Specifically, we aimed to answer the following questions: (1) Does the effect of diet quality on mean behavioral expression support the silver spoon or the environmental matching hypotheses? (2) Does exposure to poor diet quality lead to lower potential for behavioral plasticity? (3) Does diet quality affect the magnitude of trait integration and shape trade-offs at the among- and/or within-individual level? At the population average level, we predicted that if diet acted as a temporary environmental effect, exposure to a high-quality diet during adulthood should result in unconstrained expression of behaviors. In contrast, if diet acted as a permanent environmental effect during development, we should see patterns in line with either the silver spoon or the environment matching hypotheses (Figure 1A). At the level of trait variance, the cost of plasticity hypothesis suggests that a high-quality diet results in higher potential for behavioral plasticity (i.e. higher within-individual variance). If diet quality acts as a temporary environment, we should expect the high-quality adult diets to show the highest within-individual variance, regardless of the developmental diets. Alternatively, diet quality could have a permanent effect on behavioral variance, in which case the within-individual variance is predicted to be highest for individuals experiencing a high-quality diet during their development (Figure 1B). Finally, exposure to a

low-quality diet is expected to generate trade-offs among traits as a result of energy deficits.

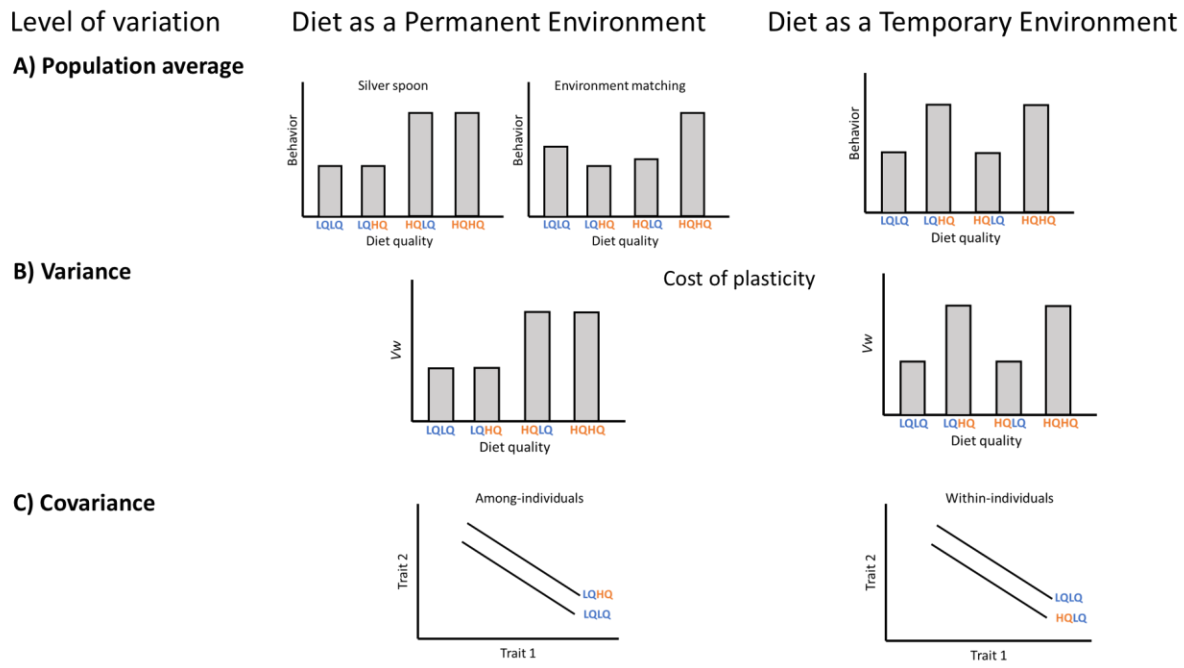

Figure 1. Conceptual framework showing how exposure to low (LQ) or high-quality (HQ) diets may affect behavioral expression at multiple levels depending on whether diet acts on behavior as a permanent environment or as a temporary environment. Diet order represents whether exposure occurs during development (LQ__ and HQ__ diets) or adulthood (__LQ and __HQ diets). At the population average level (A), if diet quality acts as a permanent environment, we should expect results conforming to either the silver spoon or the environmental matching hypotheses. If diet acts as a temporary environment, we should expect high-quality adult diets to have the strongest effect on behavioral expression. At the variance level (B), we expect behavioral plasticity to be costly to express. As a result, most changes are predicted to occur at the within-individual variance level. At the covariance among traits level (C), we expect low-quality diets to generate more pronounced trade-off that can manifest either at the among-individual level if behavioral expression is generated through the permanent environment, or at the within-individual level if diet acts as a temporary environment. If diet acts as both a

permanent and temporary environmental effects, we would expect additive effects across these levels.

These trade-offs should manifest mostly at the among-individual level if diet acts as a permanent environmental effect and should be stronger in individuals from low-quality developmental diets (i.e. negative correlations of greater magnitude). If diet quality acts as a temporary source of behavioral variation, we then expect trade-offs to be generated at the within-individual level for individuals experiencing low-quality adult diets (Figure 1C).

Methods

Cricket housing, rearing, and diet preparation

We obtained 1-week-old *A. domesticus* nymphs from Fluker's Cricket Farm (~1 mm in size) and reared them on one of two different diet treatments: either a high- (HQ, 45 % Protein, 11% Lipid, 23% Carbohydrate and 3% non-nutritive cellulose, with 3.35 cal/g) or low-quality diet (LQ, 15.33% Protein, 3.66% Lipid, 7.66% Carbohydrate and 65% non-nutritive cellulose with 1.12 cal/g). The high percentage of non-nutritive cellulose in the low-quality diet should have imposed a gut limitation on individual crickets such that they could not have overcome the relative energy differences by simply eating more (Royauté & Dochtermann, 2017). Upon reaching sexual maturity, individuals were either switched to the other diet type or maintained on the same diet. This resulted in a 2 × 2 factorial design crossing life-stage (juvenile and adult) and diet type (low or high quality) with 86 individuals in the HQHQ, 78 individuals in HQLQ, 57 individuals in LQHQ, and 60 in LQLQ. Juvenile crickets were reared in plastic containers with each plastic container (34.6 x 21 x 12.4 cm) containing around 10 juvenile crickets with a 12:12

144 hr light cycle and maintained at 32° C. Juvenile crickets were provided with egg carton housing
145 along with food and water ad libitum. Crickets were monitored weekly and, once mature, were
146 moved into individual containers (0.71 L). Adult crickets were kept on their developmental diet
147 for 48 to 72 hours before being switched to their assigned adult diet. Adult crickets were kept at
148 a 12:12 hr light cycle at 25°. Due to logistical constraints, crickets were reared during four
149 periods (hereafter "batches") with 14 rearing containers at a time (7 high-quality and 7 low-
150 quality diets). We did not detect any difference in average survival between developmental diets
151 based on individual counts at the beginning and end of the developmental phase of the
152 experiment (mean % survival, low-quality diet: 47%, high-quality diet: 52%; $F_{1,45.53} = 0.60$ $P = 0.44$, Figure A2).
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154
155 Behavioral tests
156 To test the effects of diet quality on behavioral variation and integration, we repeatedly recorded
157 individuals' activity levels in an open-field arena followed by its response to cues of predator
158 presence (diluted gecko excreta, see details below). Behavioral testing began within 1 week of
159 establishment in individual housing and testing occurred between 2 October 2016 and 13
160 October 2017. Behavioral trials for batches 1–4 began on 4 February 2016, 29 March 2016, 26
161 May 2016 and 26 September 2016, respectively. We used a 60 cm × 60 cm × 15 cm high plastic
162 arena with a Plexiglas lid for both behavioral trials. The arena was split into four 30 cm × 30 cm
163 arenas separated by an opaque Plexiglas divider, allowing us to track the behaviors of up to 4
164 individual crickets at a time. We always conducted open-field trials first followed by antipredator
165 response trials to minimize potential carry-over effects from exposure to cues of predator
166 presence. After each antipredator response assay, we thoroughly cleaned each arena with 70%

ethanol wipes to avoid accumulation of any chemical traces of conspecifics. We recorded mass to the nearest mg immediately after the antipredator response trial. Once we completed behavioral trials for every individual in a given batch, we repeated the procedure two additional times such that every individual was run through behavioral trials for a maximum of three repetitions. With this procedure, we were able to test 281 individuals for a total of 1528 behavioral observations (Table 1).

Open field behavior

Individuals were left to rest for 30s in a 5 cm diameter cup introduced into the lower right section of the arena (Figure A1, left panel). We then allowed the cricket to move freely through the arena for 220 seconds. We measured an individual's activity, calculated as the total distance travelled through the arena (in cm) and its exploratory propensity, calculated as the number of unique zones visited by the cricket with Ethovision X (Noldus Information Technology). Variations of this behavioral protocol have previously been used with *A. domesticus* to evaluate individual differences in activity and exploratory behaviors (Dochtermann & Nelson, 2014; Royauté & Dochtermann, 2017; Royauté, Greenlee, Baldwin, & Dochtermann, 2015).

Antipredator response

To measure responses to cues of potential predator presence here we collected excreta from three adult leopard geckos (*Eublepharis macularius*) that were fed a mixed diet of crickets (*A. domesticus*) and mealworms (*Tenebrio molitor*). Leopard geckos were housed according to North Dakota State University IACUC standards (Protocol number: A14006). Collected excreta was frozen and then finely ground weekly and diluted with deionized water (1 ml H_2O: 5 mg

excreta). This solution was then applied to 15 cm diameter filter paper discs with a 5 cm diameter central cutout that allowed crickets to be left to rest unexposed to the predatory cue (Royauté & Dochtermann, 2017). Each predator cue disc was left to dry for a minimum of 2 h, was stored at 4 °C between trials, allowed to warm to room temperature before the start of a trial, and discarded after a single use. We inserted the predator cue disc at the bottom of a 15cm diameter arena and left the cricket to rest for 30 s under a 5 cm diameter cup in the non-treated central cutout (Figure A1, right panel). We then allowed the cricket to move freely for 220 s and estimated the distance travelled (in cm) through Ethovision. Previous experiments with this protocol showed that crickets had heightened activity levels in presence of diluted gecko excreta compared to a water control (Royauté & Dochtermann, 2017), thus greater activity during antipredator response trials was interpreted as greater responsiveness to predator cues.

Data analysis

All analyses were conducted in R 3.4.4 (R Core Team, 2018).

Effect of diet quality on average trait value

To analyze how diet treatment affected average trait expression, we used univariate linear mixed models for all traits (adult mass, maturation time, open-field activity and unique zones travelled, antipredator activity and adult body-mass during behavioral trials) using the `lme4` package for mixed effect models (Bates, Machler, Bolker, & Walker, 2015). Diet treatment (developmental diet, adult diet and their interaction), temperature at which the trial was conducted (expressed as among and within individual values; van de Pol & Wright, 2009), repetition number, batch, time (expressed as among and within individual values; van de Pol and Wright 2009) and day of recording (centered around the population average) were included as fixed effects. Cricket

identity along with developmental container ID (the container used in the growth chamber) were included as random factors for all traits. Significance was assessed using F-tests based on Kenward-Roger approximations for the degrees of freedom in mixed models using the `lmerTest` package (Kuznetsova, Brockhoff, & Christensen, 2017). We also report the proportion of variance (R^2) explained by fixed and random effects components following Nakagawa & Schielzeth (2013).

Effect of diet quality on variance components

To test whether diet quality influenced trait variation at the among and within–individual level, we compared the fit of four different univariate mixed models on all traits for which repeated measures were obtained (activity and unique zones travelled during open-field trials, antipredator activity and adult body-mass during behavioral trials):

- Model 1: a null model where the among- (*Vi*) and within-individual variances (*Vw*) were kept constant among diet treatments.
- Model 2: a model where only the among-individual variance differed among diet treatments, while the within-individual variance was kept constant ($Vi \neq$ & $Vw =$)
- Model 3: a model where only the within-individual variance differed among diet treatments while the among-individual variance was kept constant ($Vi =$ & $Vw \neq$)
- Model 4: a model where both the among and within-individual variance were allowed to vary among diet treatments ($Vi \neq$ & $Vw \neq$)

These models were specified using the `MCMCglmm` package for Bayesian mixed models (Hadfield, 2010) using Markov chain Monte Carlo (MCMC) with 1.3×10^6 iterations, 3×10^5 burn-in period and a thinning interval of 1000 and an inverse-Wishart prior. Our parameter

estimates were very similar to those obtained by Maximum Likelihood estimation, suggesting that prior type had little influence on our results. We then compared the Deviance Information Criterion (DIC) among each model. The model with the lowest DIC value was considered the best model and models with $\Delta DIC > 5$ were considered a significantly poorer fit. Models with $\Delta DIC < 5$ were considered as having equivalent support compared to the best model (Barnett, Koper, Dobson, Schmiegelow & Manseau 2010; Spiegelhalter, Thomas, Best & Lunn 2007).

All models were specified with the same fixed effect structure as specified above to prevent biased estimates of variance components and repeatability (Nakagawa & Schielzeth, 2010; Westneat, Hatch, Wetzel, & Ensminger, 2011). We included only cricket identity as a random factor since the variance explained by the rearing containers (container ID) did not exceed 7 % for any trait with repeated measurements (Table A1). All response variables were expressed as standard deviation units to facilitate model convergence. We report the posterior modes and 95 % credible intervals for variance components and adjusted repeatability (τ), calculated as the posterior mode of $\tau = Vi / (Vi + Vw)$.

Effect of diet quality on trait integration

We first estimated among- and within-individual correlations among behavioral traits and body-mass by fitting multivariate mixed models fit separately to each diet type. We included all four traits as response variables (i.e. open-field activity and unique zones visited, antipredator activity and body-mass during behavioral trials) and used individual ID as a random effect. All fixed effects and model conditions were otherwise as above. This procedure allowed us to estimate and compare among- (r_i) and within-individual (r_w) correlation matrices between diet types (following Dingemanse & Dochtermann, 2013). The significance of these correlations was

assessed based on the probability that a given correlation excluded 0, calculated as the proportion of posterior estimates excluding 0 (Pmcmc). Correlations with Pmcmc > 0.95 were considered statistically significant and negative correlations were interpreted as representative of trade-offs among traits.

Since we had few *a priori* expectations of how phenotypic integration would vary with diet quality, we conducted an exploratory analysis by comparing different descriptive metrics of integration:

- The density (*d*) of the phenotypic network; defined as the proportion of correlations that reach statistical significance. This metric varies between 0 and 1 with higher values indicating a more strongly integrated trait network (Wilkins, Shizuka, Joseph, Hubbard, & Safran, 2015).
- The average absolute correlation strength |*r*|, calculated as the posterior mode for the mean absolute value of each estimated correlation matrix in the posterior distribution. This metric varies between 0 and 1 and indicates the overall magnitude of trait integration.
- Condition, calculated as the ratio of the variance of the highest eigenvalue over the lowest eigenvalue (Walsh & Blows, 2009). Higher values indicate that more of the variation is represented within the first eigenvalue and therefore is indicative of higher integration levels.
- Modified Mantel tests to test whether a given pair of among- or within-individual correlation matrices differed significantly from 1 (Roff, Prokkola, Krams, & Rantala, 2012). To do so, we first calculated the correlation among off-diagonal elements for each diet treatment pairs in order to obtain the observed Mantel's correlation (r_{Obs}). This was

achieved by calculating the r_{Obs} for each slice of the posterior distribution of correlation estimates between the two treatments considered in order to obtain 95 % credible intervals. We then compared the overlap of the posterior distribution of r_{Obs} values with a randomized r (r_{random}) obtained after 100 permutations of the dataset. We base our inference on the Pmcmc for the overlap between the posterior distribution of r_{Obs} values with r_{random} (i.e. the number of r_{Obs} values that are equal or exceed r_{random}).

Finally, we tested whether integration between behaviors and life-history traits (body-mass at maturation and maturation time) changed with exposure to low- and high-quality developmental diets. To do so we specified a series of 8 bivariate models whereby the among-individual correlation between a behavior and a life-history trait was estimated for a given developmental diet treatment. Because our life-history traits represented unique events, we estimated their correlations with behavioral traits by fixing the within-individual variance of life-history traits to a small value (V = 0.0001) following the recommendations of Houslay and Wilson (2017).

Results

Effect of diet quality on trait averages

Crickets provided with a high-quality diet during development grew 4 % larger and matured 48h faster on average (Table 1). However, these changes were not statistically significant ($P > 0.1$) and we failed to detect any substantial effect of diet quality or its interactions with sex and batch for any of the traits measured. Interestingly, the rearing containers explained up to 19 % of the variation in adult mass and maturation time but had no influence on traits measured during adulthood ($R^2 < 0.07$) (Table A1). Further, maturation time differed between treatments when

305 rearing container was excluded from the analysis ($F_{1,272} = 16.21$, $P = 7 \times 10^{-5}$). This suggests that
306 the social environment during development may play a larger role in how crickets mature and
307 grow than did diet quality. These results also suggest that the social environment interacted with
308 diet quality, but without carryover effects on behaviors into adulthood.

Table 1. Mean, standard errors and sample sizes for behavioral and growth traits compared across diet treatments. *Nobs* indicates the number of observation taken for each diet treatments, *Ni* indicates the number of individuals tested. The number of observations is lower with antipredator activity because a small portion of the individuals managed to escape the test arena or crawl under the predator cue filter paper. These observations were excluded prior to analysis.

Developmental phase	LQ				HQ			
	Mean	SE	*Nobs*	*Ni*	Mean	SE	*Nobs*	*Ni*
Adult mass (mg)	333.69	6.88	325	117	346.67	4.95	446	164
Maturation time (days)	32.92	0.50	325	117	30.7	0.38	446	164

Adult phase	LQLQ				LQHQ				HQLQ				HQHQ			
	Mean	SE	*Nobs*	*Ni*	Mean	SE	*Nobs*	*Ni*	Mean	SE	*Nobs*	*Ni*	Mean	SE	*Nobs*	*Ni*
Activity (OF) (cm)	248.78	12.75	167	60	242.9	12.97	158	57	259.61	11.76	216	78	246.25	11.19	230	86
Unique zones (OF)	20.71	0.65	167	60	20.32	0.71	158	57	21.19	0.62	216	78	20.41	0.58	230	86
Activity (AP) (cm)	294.33	14.49	162	59	322.69	16.6	158	57	322.32	16.02	208	77	330.89	16.47	229	86
Body-mass (mg)	421.24	9.7	162	59	424.02	9.4	158	57	433.47	8.11	208	77	441.37	8.24	229	86

Effect of diet quality on variance components

We found evidence for significant changes in trait variation among diet quality treatments except for antipredator activity and body-mass. For both of these traits, the model allowing differences at the within-individual level as well as the model allowing differences at the among *and* within-individual levels had equivalent support (model 4, $\Delta DIC < 1.5$). Other models were strongly rejected ($\Delta DIC > 10$, Table 2). This indicates that diet manipulation directly affected within-individual variation and possibly affected among-individual variation. For antipredator activity, the strongest differences were detected between the HQHQ and HQLQ diets at the within individual level ($\Delta Vw = 0.28 \pm [0.14; 0.52]$) and the HQLQ and LQLQ diets at the among-individual level ($\Delta Vi = 0.24 \pm [-0.04; 0.56]$). For body-mass, the HQHQ diet had the highest within-individual variance compared to all other diets ($|\Delta Vw| \sim 0.10$, Figure 2; Table 3).

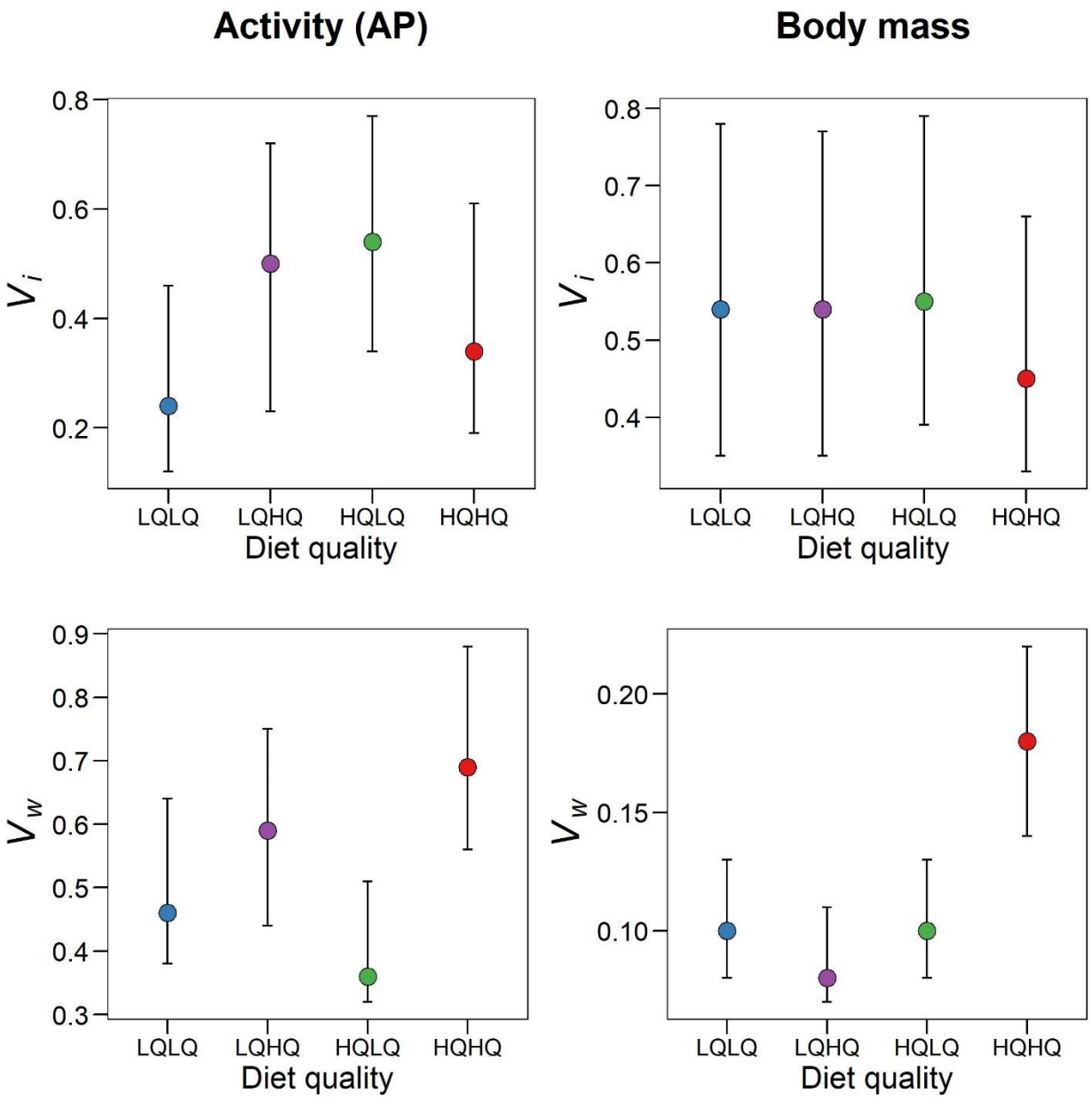


Figure 2. Effect of diet quality on among (*Vi*) and within-individual variance (*Vw*).

Table 2. Model comparison for testing the effects of diet quality on among and within-individual variation. Model 1 represents a null model where both the among (*Vi*) and within individual (*Vw*) variance are equal among diet treatments. Model 2 and 3 are models where either the among or the within-individual variance differs among diet treatments respectively, and model 4 is a model where both the among and within-individual variance differ among diet treatments. Bold indicates the best model (the model with lowest DIC value) and italics indicate models with equivalent support to the best model (ΔDIC < 5).

Model	Variance comparison	Activity (OF)		Unique zones (OF)		Activity (AP)		Mass	
		DIC	ΔDIC	DIC	ΔDIC	DIC	ΔDIC	DIC	ΔDIC
Model 1	*Vi* = & *Vw* =	**2013.17**	**0.00**	**2119.97**	**0.00**	1876.96	11.58	760.54	14.73
Model 2	*Vi* ≠ & *Vw* =	*2014.00*	*0.83*	*2120.05*	*0.08*	1877.08	11.70	761.51	15.70
Model 3	*Vi* = & *Vw* ≠	*2017.16*	*3.99*	*2123.10*	*3.13*	**1865.38**	**0.00**	**745.81**	**0.00**
Model 4	*Vi* ≠ & *Vw* ≠	2017.74	*4.57*	*2122.74*	*2.77*	*1866.32*	*0.94*	*746.82*	*1.01*

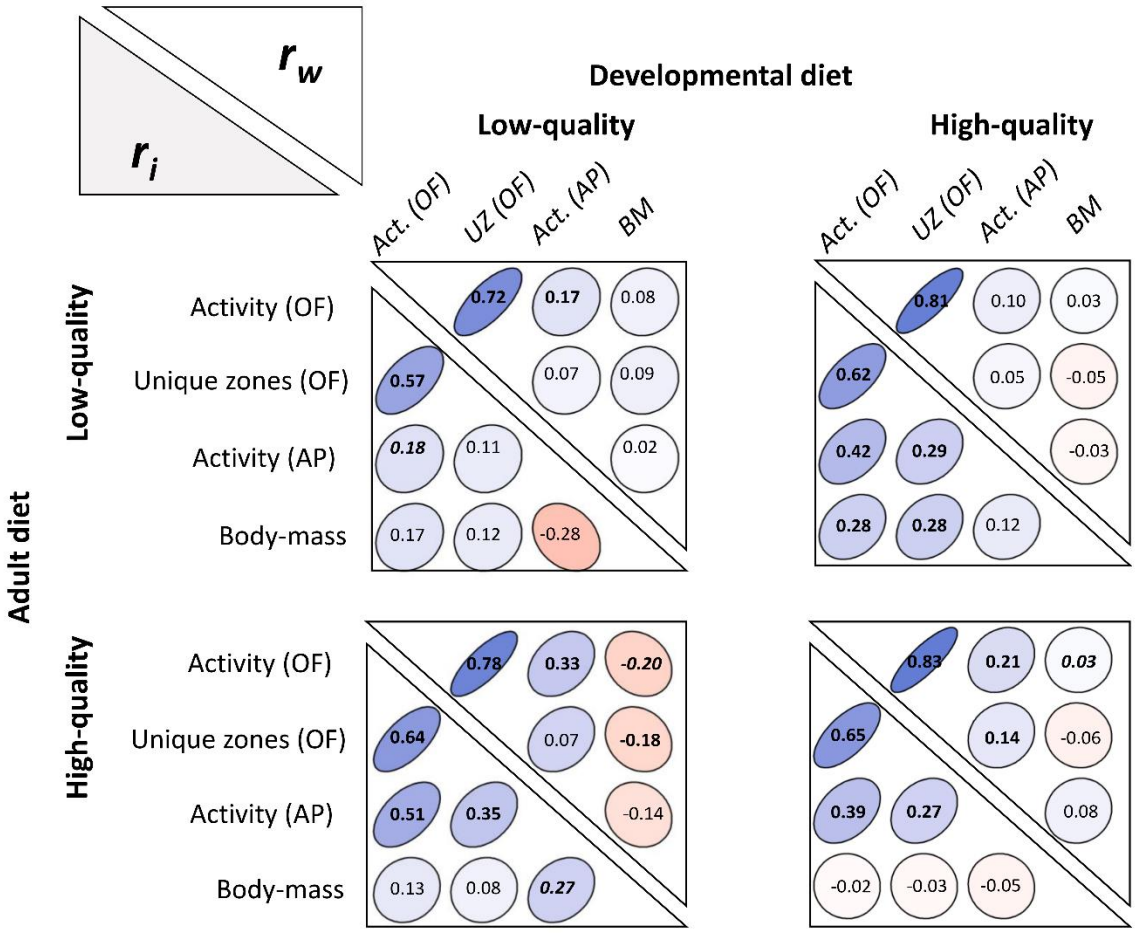
Table 3. Variance components (*Vi*: among-individual, *Vw*: within-individual, *Vp*: phenotypic variance, calculated as $V_i + V_w$) and adjusted repeatability (τ) compared across diet treatments (posterior mode [95% CRI]).

Trait	**Diet quality treatments**							
	LQLQ		LQHQ		HQLQ		HQHQ	
	Variance	[95% CRI]	Variance	[95% CRI]	Variance	[95% CRI]	Variance	[95% CRI]
Activity (OF)								
Vi	0.28	[0.10; 0.41]	0.32	[0.19; 0.62]	0.31	[0.19; 0.55]	0.39	[0.21; 0.63]
Vw	0.58	[0.52; 0.83]	0.56	[0.44; 0.75]	0.66	[0.54; 0.85]	0.65	[0.52; 0.83]
Vp	0.85	[0.69; 1.09]	0.91	[0.72; 1.20]	1.07	[0.84; 1.26]	1.01	[0.83; 1.30]
τ	0.29	[0.13; 0.43]	0.35	[0.24; 0.55]	0.36	[0.22; 0.49]	0.38	[0.26; 0.53]
Unique zones (OF)								
Vi	0.20	[0.11; 0.41]	0.16	[0.08; 0.35]	0.24	[0.11; 0.45]	0.21	[0.11; 0.40]
Vw	0.70	[0.53; 0.88]	0.78	[0.63; 1.03]	0.78	[0.63; 0.98]	0.74	[0.60; 0.93]
Vp	0.89	[0.74; 1.13]	0.94	[0.83; 1.27]	1.05	[0.86; 1.29]	0.99	[0.83; 1.21]
τ	0.21	[0.13; 0.40]	0.17	[0.08; 0.32]	0.28	[0.13; 0.40]	0.24	[0.12; 0.37]
Activity (AP)								
Vi	0.24	[0.12; 0.46]	0.50	[0.23; 0.72]	0.54	[0.34; 0.77]	0.34	[0.19; 0.61]
Vw	0.46	[0.38; 0.64]	0.59	[0.44; 0.75]	0.36	[0.32; 0.51]	0.69	[0.56; 0.88]
Vp	0.72	[0.59; 0.97]	1.04	[0.77; 1.31]	0.85	[0.71; 1.17]	1.11	[0.89; 1.35]
τ	0.33	[0.20; 0.52]	0.40	[0.28; 0.59]	0.60	[0.44; 0.69]	0.36	[0.21; 0.50]
Body-mass								
Vi	0.54	[0.35; 0.78]	0.54	[0.35; 0.77]	0.55	[0.39; 0.79]	0.45	[0.33; 0.66]
Vw	0.10	[0.08; 0.13]	0.08	[0.07; 0.11]	0.10	[0.08; 0.13]	0.18	[0.14; 0.22
Vp	0.56	[0.44; 0.88]	0.62	[0.41; 0.83]	0.69	[0.49; 0.88]	0.62	[0.49; 0.83]
τ	0.84	[0.77; 0.90]	0.87	[0.79; 0.91]	0.85	[0.78; 0.90]	0.73	[0.62; 0.80]

Effect of diet quality on trait integration

We did not find evidence of trade-offs between traits at the among-individual level, i.e. we did not detect negative correlations among behaviors (Figure 3, Table A2). Regardless of diet type, open-field activity and unique zones visited were the most strongly correlated traits at both among- and within-individual levels (r_i > 0.55 and r_w > 0.70). In contrast, we found evidence for diet quality affecting the strength of the activity-antipredator response syndrome. In particular, an activity-antipredator response syndrome was not detectable for the LQLQ diet (correlation ± [95 % CRI], open-field × antipredator activity: r_i = 0.18 ± [-0.08; 0.54], Pmcmc = 0.91; unique zones × antipredator activity: r_i = 0.11 ± [-0.22; 0.47], Pmcmc = 0.78). All other diets showed significant correlations varying between 0.27 (HQHQ unique zones × antipredator activity, [-0.02; 0.56], Pmcmc = 0.96) and 0.51 (LQHQ open-field × antipredator activity, [0.17; 0.72], Pmcmc = 0.99). Body-mass was generally poorly integrated with behavioral traits (average r_i < 0.20) and none of the correlations reached statistical significance (Pmcmc < 0.92).

At the within-individual level, individuals increasing their activity and exploration levels during the open-field trials also tended to increase their antipredator response in these diets (0.14 < r_w < 0.33). We found evidence of trade-offs between body-mass and behaviors in the LQHQ diet (open-field activity x body-mass: r_w = -0.20 ± [-0.33; 0.06], Pmcmc = 0.93; unique zones × body-mass: r_w = -0.18 ± [-0.34; 0.03], Pmcmc = 0.95; antipredator activity × body-mass: r_w = -0.14 [-0.33; 0.06], Pmcmc = 0.89). This means that the individuals that gained mass during the course of behavioral measurements tended to decrease their activity levels. However, these trade-offs run contrary to our predictions that the low-quality adult diet should be the one generating the highest number of trade-offs at the within-individual level.



362

363 **Figure 3.** Among (r_i, lower diagonal) and within-individual (r_w, upper diagonal) correlations for
364 behavioral measurements and body-mass based on multi-response mixed models. Bold values
365 indicate significant correlations based on 95 % of estimates excluding 0 (Pmcmc > 0.95).
366 Italicized and bold values indicate correlations where > 90 % of estimates exclude 0 (Pmcmc >
367 0.90). Shape of ellipses indicates strength and—along with color—the direction of correlations.

368 We did not find evidence for differences in the magnitude of phenotypic integration
369 being mediated by diet quality. Although the HQLQ diet had consistently higher values for all
370 integration metrics at the among-individual level (network density d = 0.83, average correlation
371 strength $|r|$ = 0.31 ± [0.21; 0.51], Condition = 5.42 ± [3.10; 11.63], Table A3), all metrics had

wide 95 % credibility intervals with substantial overlap among diets. Our modified Mantel tests did not reveal any substantial differences among matrices as all pairwise comparisons were statistically undistinguishable from 1 (Table A4). This means that while diets may have affected the correlations between specific pairs of traits, it had little influence on the overall patterns of phenotypic integration at the among- and within-individual levels.

Finally, when investigating the relation between life-history traits and behavior among diets, we found a significant change in the maturation time × unique zones correlation (Δr_i = 0.45 ± [-0.10; 1.06], Pmcmc = 0.95, with Δr_i calculated as r_i HQ – r_i LQ; Table 4). This indicates that faster-maturing crickets have a higher exploratory propensity only when exposed to limits on their energy budgets during development (Figure 4).

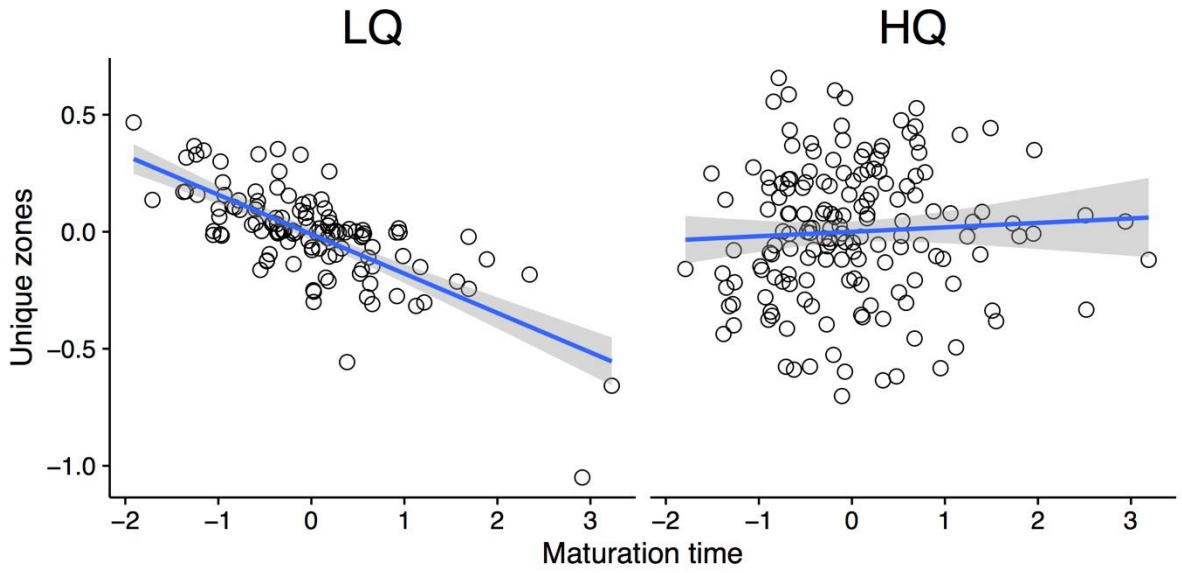


Figure 4. Relation between the number of unique zones explored in open field tests and maturation time compared between developmental diet treatments (all values expressed as standard deviation units). Dots represent Best Linear Unbiased Predicted values (BLUPS) extracted from bivariate mixed models. Blue lines represent least square regressions with 95 % CI.

Table 4. Effect of diet quality during development on the relationships between maturation time, adult body mass and behaviors. Bold values indicate significant correlations based on 95 % of estimates excluding 0 (Pmcmc > 0.95). Italicized and bold values indicate correlations where > 90 % of estimates exclude 0 (Pmcmc > 0.90).

Bivariate correlation	Low Quality			High Quality			Δ*r* (HQ - LQ)		
	r	[95 % CRI]	Pmcmc	*r*	[95 % CRI]	Pmcmc	Δ*r*	[95 % CRI]	Pmcmc
Activity (OF) × Maturation	-0.25	[-0.52; 0.15]	0.83	0.16	[-0.18; 0.39]	0.72	0.29	[-0.17; 0.68]	0.87
Unique Zones × Maturation	**-0.44**	**[-0.83; 0.03]**	**0.95**	0.10	[-0.31; 0.45]	0.68	**0.45**	**[-0.10; 1.06]**	**0.95**
Activity (AP) × Maturation	-0.03	[-0.38; 0.16]	0.78	***-0.11***	***[-0.36; 0.04]***	***0.94***	0.01	[-0.40; 0.27]	0.37
Body-mass × Maturation	-0.14	[-0.30; 0.12]	0.81	**-0.24**	**[-0.38; -0.01]**	**0.98**	-0.19	[-0.39; 0.19]	0.74

Discussion

In the present study, we set out to test whether diet quality during development, diet quality during adulthood, or their interactions had the strongest effect on behavioral variation and covariation (Figure 1). We did not find support for either the environmental matching or silver spoon hypotheses regarding mean behavioral expression since mean behaviors did not vary significantly among diet treatments. Instead, we found evidence of increased variability in antipredator response for individuals provided a high-quality diet while adults. Our hypothesis that a low-quality diet would generate trade-offs among traits (Figure 1C) was poorly supported. The few negative correlations detected between behaviors and body-mass had weak effect sizes and were not consistent with our predictions. Finally, we found that overall integration among behavioral traits and body-mass did not vary by diet treatments, although the presence of an activity-antipredator response syndrome was only detected for specific diet types.

Diet did not affect trait expression at the population average level which leads us to reject both the silver spoon and environment matching hypotheses. Our diet quality manipulation also had surprisingly little influence on body-mass at maturation and maturation time. This is contrary to what was observed in a previous study where crickets tracked during the first 30 days of their development showed higher growth rates when exposed to the high-quality diet (Royauté & Dochtermann 2017). Several compensatory mechanisms may explain these differences. First, these two experiments were conducted at different developmental temperatures (Royauté & Dochtermann 2017: 25°C, current experiment: 32°C). Increased temperatures generally result in faster developmental rates in ectotherms (Angilletta and Dunham 2003, Atkinson, 1994; Carleton, 1960), and this could partially account for the lack of difference in growth rate between our diet treatments. Second, the present experiment required the use of group-housing during the

developmental phase while crickets were reared in isolation in the prior experiment. Container ID during the developmental phase explained between 16-19 % of the variation in body-mass at maturation and maturation time, which suggests that the social environment may play a larger role in cricket growth than diet quality. Finally, we cannot exclude the possibility that cannibalism may have been present during the developmental phase of the experiment and could have affected our results. However, given that mortality rates did not differ significantly between developmental diets, if cannibalism occurred, it likely did not affect individuals in the low-quality diet at a higher rate compare to individuals in the high-quality diet. Interestingly, while the social environment generated by group housing during development affected variation in adult body-mass and maturation time, it had little influence on the expression of behaviors and body-mass measured during the adult phase of the experiment. This suggests that the social environment experienced during the developmental phase did not carry-over to affect adult behaviors.

Instead of affecting average trait expression, most of the effects of our diet manipulations were manifested at the level of variances of traits and covariances among traits. Antipredator response and body mass both showed significant differences in their within-individual variance among treatments, while none of the open-field behaviors showed any difference. For both traits, the HQHQ diet had the highest within-individual variance thus giving support for the cost of plasticity hypothesis. However, we were not able to distinguish whether such patterns were due to diet acting as a permanent source of variation during development or instead represented temporary source of behavioral variation. Our results are in line with recent studies showing that changes in diet quality or composition primarily influence the expression of within-individual variance in behavior in crickets (Han & Dingemanse, 2017; Royauté & Dochtermann, 2017). In

contrast, diet restriction in several spider species resulted in changes at both the among-individual and within-individual levels (Lichtenstein et al., 2016; Pruitt, DiRienzo, Kralj-Fišer, Johnson, & Sih, 2011). Interestingly, these latter studies showed that, contrary to our predictions, individuals experiencing diet restrictions often increased their within-individual variance, although the patterns were species and trait-specific. Thus, there does not seem to be a strong consensus on the direction in which diet quality may affect changes in behavioral variation and taxonomic coverage remains limited to date.

Contrary to our predictions (Figure 1), we found a surprising lack of trade-offs between behavioral traits and body mass. The only instances of negative correlations were observed between open-field behaviors and body-mass at the within-individual level in the LQHQ diet. However, the magnitude of these correlations was low ($-0.20 < r_w < -0.14$), suggesting at best a weak trade-off between changes in activity and exploration levels with changes in body mass. More importantly, this result runs contrary to our expectations that if diet acts as a temporary environmental effect, trade-offs will manifest at the within-individual level for individuals provided with a low-quality diet as adults (Figure 1).

We also found evidence for diet quality acting as a mediator of life-history × behavior correlations. In particular, individuals that matured faster in the low-quality diet had increased exploratory propensity, while this correlation was absent in the high-quality diet. Links between life-history and behavioral traits are often understood within the pace-of-life syndrome hypothesis (POLS) (Réale et al., 2010), where individual with "live fast-die young" strategies adopt more risky behavioral strategies in terms of resource accumulation (i.e. higher exploration propensity and boldness). While support for the POLS hypothesis is mixed at best (Royauté, Berdal, Garrison, & Dochtermann, 2018), there is theoretical evidence that changes in resource

availability can affect the magnitude of integration among some POLS traits (Salzman, McLaughlin, Westneat, & Crowley, 2018). Note that our observed changes to life-history × behavior correlations are hard to put into the context of the predictions of the POLS hypothesis because we did not obtain precise estimates of growth rate and longevity, which would be required to demonstrate the presence of slow-fast life-history strategies. Moreover, many of the predictions of the POLS hypothesis imply the presence of a proactive-reactive behavioral axis, whereby highly active individuals are also superficial explorers and are bolder when exposed to threat. In contrast, the behavioral syndrome we observed did not show the negative correlation between activity levels and thoroughness of exploration expected through the proactive-reactive axis (Koolhaas et al., 1999; Verbeek, Drent, & Wiepkema, 1994). Contrary to our expectations, our results also did not indicate the presence of trade-offs between life-history and behavioral traits being mediated by diet quality. Instead, they suggest that, in a nutritionally depleted environment, only high-quality individuals can mature faster *and* express high exploratory propensity levels. In other words, it seems that diet quality during development can act as an equalizer rather than generate strong trade-offs between life-history and behavior through restrictions on energy budgets (i.e. "reverse trade-off", Careau & Wilson, 2017; Reznick, Nunney, & Tessier, 2000).

By manipulating diet quality another one of our aims was to determine whether correlations among behaviors and body-mass could be environmentally generated. We found little evidence for effects of diet quality on the magnitude of trait integration. The presence of an activity-antipredator response syndrome was detected in all but the LQLQ diet treatment, suggesting that the shape of the covariance among traits is well-conserved even across substantially different nutritional environments. Among-individual correlations are influenced by

485 both genetic level and environmental sources of variation (Dingemanse & Dochtermann, 2014).
486 Our results could therefore indicate that these traits are sustained by strong correlations at the
487 genetic level. Another possibility could be that changes in nutritional state have very little
488 influence on the behaviors we measured as has been shown in a previous meta-analysis of the
489 influence of intrinsic states (i.e. body-mass, body-size, metabolic rate and hormone levels) on
490 behaviors (Niemelä & Dingemanse, 2018). Finally, it is possible that manipulating diet by
491 imposing gut limitation may not be extreme enough to generate strong energetic trade-offs and
492 change the patterns of correlations among traits. As a result, extreme starvation (Lichtenstein et
493 al., 2016; Riechert & Hedrick, 1993) or extreme restriction of specific macro-nutrients (Han &
494 Dingemanse, 2017) may be more efficient as a way to probe the role of diet quality on the
495 generation of trait integration in an omnivorous organism.
496 By examining the consequences of lifelong exposure to diet quality, we were able to
497 show that most of the behavioral changes appeared at the variance and covariance among traits
498 rather than at the population average level. The broadly applicable hypotheses of silver spoon
499 and environmental matching effects were therefore not supported. Instead we found support for
500 the cost of plasticity hypothesis suggesting that high quality diets would increase the potential for
501 behavioral plasticity via increases in within-individual variance. However, many of our
502 predictions failed to be confirmed under our conceptual framework. Whether diet quality acted
503 mainly as a permanent or temporary source of behavioral variation therefore remains unclear.
504 Ultimately, finer scale diet manipulations based on ratio of macro-nutrient intakes seem more
505 well suited to disentangling the role of diet quality on behavioral expression (Harrison,
506 Raubenheimer, Simpson, Godin, & Bertram, 2014; Raubenheimer & Simpson, 2018;
507 Raubenheimer, Simpson, & Mayntz, 2009). Regardless, our results indicate that even if diet

quality had little influence on trait averages, it still generated effects on the expression of individual differences in behavior.

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Appendices for: "**Do developmental environments and current energy state mediate the potential for plasticity and behavioral integration?**"

Figure A1. Arena designs for open-field activity and antipredator response trials.

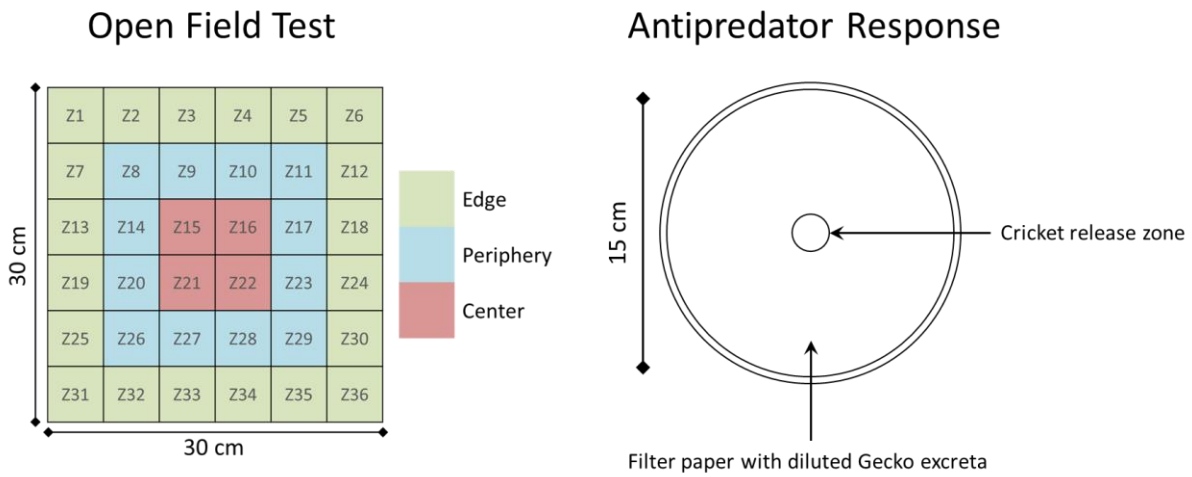
Figure A2. Survival rates did not differ significantly between diet quality treatments during the developmental phase of the experiment.

Table A1. ANOVA table summarizing the significance of fixed effects on trait expression

Table A2. Among (lower diagonal) and within-individual (upper diagonal) correlations for behavioral measurements and body-mass based on multi-response mixed models

Table A3. Integration metrics compared among the different diet treatments for among and within-individual correlations matrices.

Table A4. Modified Mantel's tests for comparing correlation matrices similarity across diet treatments.



672

673 **Figure A1.** Arena designs for open-field activity and antipredator response trials. Left panel:
674 Open-field arena. Individuals were introduced into the bottom-left quadrant (Z31) and
675 allowed 220s to explore the arena. Distance travelled and number of unique zones explored
676 were then extracted for behavioral analysis. Right panel: Antipredator response arena.
677 Individuals were introduced into the center circle which was not exposed to the predator cues
678 and then allowed to move through the arena for 220s. Greater movement during this trial
679 indicates a stronger antipredator response.

680

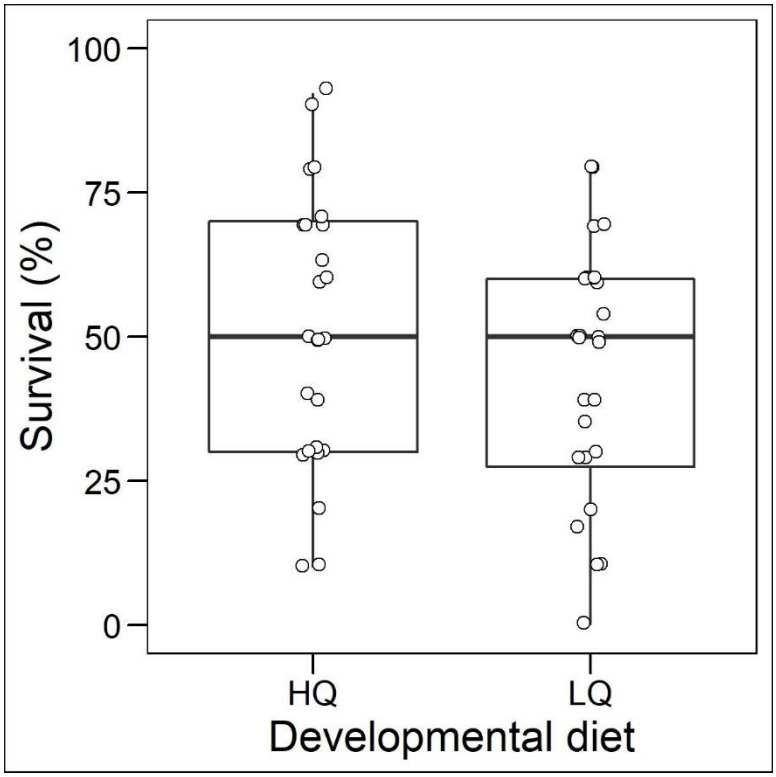


Figure A2. Survival rates did not differ significantly between diet quality treatments during the developmental phase of the experiment (linear mixed model with developmental diet as fixed effect and batch as random effect, $F_{1,45;53} = 0.60$, $P = 0.44$, $N_{containers} = 50$, degrees of freedom calculated using the Kenward-Rodger approximation).

687 **Table A1.** ANOVA table summarizing the significance of fixed effects on trait expression.
688 Open-field and antipredator activities were square-root transformed prior to analysis.
689 Variances are expressed on their original scale and R^2 represents the proportion of variation
690 explained by each variance component. Bold values indicate statistically significant effects.
691

Trait	**Fixed Effects**			**Random Effects**			
	df	F	P	Variance component	Variance	[95% CI]	R^2
Adult mass							
Diet (dev)	1, 43.56	1.14	0.29	V_{Fixed}	630.6	-	0.12
Sex	1, 261.80	18.14	**2.86E-05**	*Vi*	-	-	-
Batch	3, 42.70	2.12	0.11	$V_{container}$	1005	[303; 1483]	0.19
Diet (dev) × Sex	1, 255.20	0.17	0.68	*Vw*	3559	[2928; 4186]	0.69
Diet (dev) × Batch	3, 42.77	1.10	0.36				
Sex × Batch	3, 259.25	0.77	0.51				
Maturation time							
Diet (dev)	1, 43.57	2.60	0.11	V_{Fixed}	8.40	-	0.28
Sex	1, 261.90	0.61	0.43	*Vi*	-	-	-
Batch	3, 42.70	12.89	**3.94E-06**	$V_{container}$	4.81	[1.16; 7.22]	0.16
Diet (dev) × Sex	1, 255.29	0.11	0.74	*Vw*	17.24	[14.23; 20.45]	0.57
Diet (dev) × Batch	3, 42.77	1.39	0.26				
Sex × Batch	3, 259.35	1.09	0.35				
Activity (OF)							
Diet (dev)	1, 41.43	0.10	0.75	V_{Fixed}	2.38	-	0.07
Diet (ad)	1, 263.72	0.93	0.34	*Vi*	10.85	[7.19; 13.52]	0.31
Sex	1, 264.94	9.04	**0.003**	$V_{container}$	0.00	[0.00; 1.06]	0.00
Mass (mean)	1, 258.37	5.07	**0.03**	*Vw*	21.88	[19.24; 24.66]	0.62
Mass (sd)	1, 494.48	0.03	0.87				
Temperature (mean)	1, 245.73	0.21	0.65				
Temperature (sd)	1, 510.38	1.34	0.25				
Day	1, 535.13	2.35	0.13				
Rep	1, 633.31	6.69	**0.01**				
Batch	3, 145.15	1.15	0.33				
Diet (dev) × Diet (ad)	1, 262.50	0.18	0.67				
Diet (dev) × Sex	1, 258.92	0.29	0.59				
Diet (ad) × Sex	1, 264.53	1.04	0.31				

Trait	**Fixed Effects**			**Random Effects**			
	df	F	P	Variance component	Variance	[95% CI]	R^2
Unique zones (OF)							
Diet (dev)	1, 41.21	0.09	0.76	V_{Fixed}	4.72	-	0.06
Diet (ad)	1, 261.22	0.91	0.34	*Vi*	14.18	[6.77; 19.60]	0.18
Sex	1, 263.31	8.80	**0.003**	$V_{container}$	0.00	[0.00; 1.71]	0.00
Mass (mean)	1, 257.41	6.10	**0.01**	*Vw*	61.58	[54.15; 69.39]	0.76
Mass (sd)	1, 497.76	0.17	0.68				
Temperature (mean)	1, 245.16	0.47	0.49				
Temperature (sd)	1, 509.86	5.90	**0.01**				
Day	1, 479.51	6.91	**0.01**				
Rep	1, 611.74	7.44	**0.01**				
Batch	3, 142.30	3.24	**0.006**				
Diet (dev) × Diet (ad)	1, 260.05	0.37	0.54				
Diet (dev) × Sex	1, 256.97	0.33	0.56				
Diet (ad) × Sex	1, 262.22	0.16	0.68				
Activity (AP)							
Diet (dev)	1, 41.69	0.61	0.43	V_{Fixed}	3.38	-	0.09
Diet (ad)	1, 262.87	0.003	0.95	*Vi*	14.42	[10.24; 17.49]	0.39
Sex	1, 264.80	0.04	0.83	$V_{container}$	0.00	[0.00; 1.67]	0.00
Mass (mean)	1, 254.92	1.10	0.29	*Vw*	19.35	[16.96; 21.85]	0.52
Mass (sd)	1, 481.93	0.15	0.69				
Temperature (mean)	1, 261.05	2.36	0.12				
Temperature (sd)	1, 482.48	9.38	**0.002**				
Day	1, 651.26	0.89	0.34				
Rep	1, 713.39	3.10	0.07				
Batch	3, 175.17	3.25	**0.02**				
Diet (dev) × Diet (ad)	1, 262.90	0.03	0.86				
Diet (dev) × Sex	1, 257.46	0.19	0.66				
Diet (ad) × Sex	1, 265.85	1.68	0.19				

Trait	**Fixed Effects**			**Random Effects**			
	df	F	P	Variance component	Variance	[95% CI]	R^2
Adult mass							
Mass							
Diet (dev)	1, 43.41	0.06	0.80	V_{Fixed}	5973	-	0.39
Diet (ad)	1, 261.44	3.00	0.08	*Vi*	6499	[5213.95;7824.37]	0.43
Sex	1, 250.38	188.89	**2.20E-16**	$V_{container}$	1102	[133.65; 2094.03]	0.07
Rep	1, 486.42	74.67	**2.20E-16**	*Vw*	1661	[1642.36; 1884.88]	0.11
Batch	3, 58.20	5.39	**0.002**				
Diet (dev) × Diet (ad)	1, 261.66	0.09	0.77				
Diet (dev) × Sex	1, 250.09	1.32	0.25				
Diet (ad) × Sex	1, 258.72	0.07	0.78				

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700 **Table A2.** Among (lower diagonal) and within-individual (upper diagonal) correlations for
701 behavioral measurements and body-mass based on multi-response mixed models with
702 associated 95 % credible intervals (in bracket). Bold values indicate significant correlations
703 based on 95 % of estimates excluding 0 (Pmcmc > 0.95). Italicized and bold values indicate
704 correlations where > 90 % of estimates exclude 0 (Pmcmc > 0.90).

LQLQ	Activity (OF)	Unique zones (OF)	Activity (AP)	Body-Mass
Activity (OF)	1.00	**0.72**	**0.17**	0.08
Unique zones (OF)	**0.57**	1.00	0.07	0.09
Activity (AP)	***0.18***	0.11	1.00	0.02
Body-mass	0.17	0.12	-0.28	1.00
LQHQ				
Activity (OF)	1.00	**0.78**	**0.33**	***-0.20***
Unique zones (OF)	**0.64**	1.00	**0.26**	**-0.18**
Activity (AP)	**0.51**	***0.35***	1.00	-0.14
Body-mass	0.13	0.08	***0.27***	1.00
HQLQ				
Activity (OF)	1.00	**0.81**	0.10	0.03
Unique zones (OF)	**0.62**	1.00	0.05	-0.05
Activity (AP)	**0.42**	**0.29**	1.00	-0.03
Body-mass	**0.28**	**0.28**	0.12	1.00
HQHQ				
Activity (OF)	1.00	**0.83**	**0.21**	0.03
Unique zones (OF)	**0.65**	1.00	**0.14**	-0.06
Activity (AP)	**0.39**	**0.27**	1.00	**0.08**
Body-mass	-0.02	-0.03	-0.05	1.00

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Table A3. Integration metrics compared among the different diet treatments for among and within-individual correlations matrices with associated 95 % credibility intervals. Network density *d* and the average correlation strength |*r*| indicate the intensity of integration among traits, condition represents the amount of variation along the first eigenvalue relative to the last eigenvalue. Higher values are indicative of higher overall integration. Condition represents the amount of variation along the first eigenvalue relative to the last eigenvalue. The diet(s) with the highest metrics values are indicated in bold.

Level	Treatment	Network density *d*	average |*r*| [95 % CRI]	Condition [95 % CRI]
r_i	LQLQ	0.17	0.25 [0.15; 0.37]	3.93 [2.32; 8.97]
	LQHQ	0.33	0.28 [0.15; 0.44]	4.81 [2.64; 10.20]
	HQLQ	**0.83**	**0.31** [0.21; 0.51]	**5.42** [3.10; 11.63]
	HQHQ	0.50	0.27 [0.17; 0.40]	5.26 [3.02; 11.38]
r_w	LQLQ	0.33	0.19 [0.15; 0.28]	6.57 [4.52; 10.00]
	LQHQ	**0.67**	**0.32** [0.19; 0.39]	9.34 [5.46; 13.72]
	HQLQ	0.17	0.20 [0.16; 0.26]	9.76 [7.02; 14.11]
	HQHQ	**0.67**	0.26 [0.18; 0.30]	**10.34** [8.23; 15.44]

Table A4. Modified Mantel's tests for comparing correlation matrices similarity across diet treatments. The Mantel's *r* represents how closely related the among or within-individual correlation matrices are between two diets and is compared to a randomized *r* obtained through 100 permutations. Pmcmc values indicated the probability that two matrices differ significantly from the randomized *r* and are statistically different from 1. Bold values indicate Pmcmc > 0.95 and bold and italics values indicate Pmcmc > 0.90.

		Observed			Randomized			Pmcmc		
Level	Treatment	Mantel's *r* [95 % CRI]			Mantel's *r* [95 % CRI]					
		LQLQ	LQHQ	HQLQ	LQLQ	LQHQ	HQLQ	LQLQ	LQHQ	HQLQ
r_i		-	-	-	-	-	-			
	LQHQ	0.76	-	-	0.85	-	-	0.66		
		[0.23; 0.98]	-	-	[0.48; 0.93]	-	-			
	HQLQ	0.87	0.65	-	0.85	0.94	-	0.66	0.85	
		[0.26; 0.99]	[-0.08; 0.97]	-	[0.63; 0.95]	[0.74; 0.97]	-			
	HQHQ	0.76	0.87	0.94	0.90	0.89	0.93	0.76	0.64	0.37
		[0.23; 0.99]	[0.24; 0.99]	[0.20; 0.99]	[0.58; 0.94]	[0.63; 0.97]	[0.81; 0.97]			
r_w	LQLQ	-	-	-	-	-	-			
		-	-	-	-	-	-			
	LQHQ	0.95	-	-	0.98	-	-	***0.92***		
		[0.62; 0.99]	-	-	[0.88; 0.97]	-	-			
	HQLQ	0.96	0.94	-	0.97	0.98	-	0.88	0.83	
		[0.81; 1.00]	[0.70; 0.99]	-	[0.91; 0.99]	[0.95; 0.99]	-			
	HQHQ	0.96	0.94	0.97	0.98	0.98	0.98	**0.97**	0.80	0.83
		[0.75; 1.00]	[0.75; 1.00]	[0.83; 1.00]	[0.88; 0.99]	[0.94; 0.99]	[0.96; 0.99]			