

Current energy state interacts with the developmental environment to influence behavioural plasticity

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Current energy state interacts with the 1 developmental environment to influence 2 behavioral plasticity 3 4 Raphaël Royauté^{a,b}, Courtney Garrison^a, Jeremy Dalos^a, Monica A. Berdal^a and 5 Ned A. Dochtermann^a 6 7 8 ^aDepartment of Biological Sciences, North Dakota State University, 1340 Bolley Drive, 201 9 Stevens Hall, Fargo, ND 58102, USA. 10 11 ^bcorresponding author: raphael.royaute@gmail.com 12

13 Abstract:

14 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 15 16 behavioral variation depending on whether it acts primarily during ontogeny (i.e. as a permanent 17 environmental effect) or has an immediate effect on trait expression as a consequence of energy 18 intake (i.e. temporary source of variation). Moreover, whether diet quality has a stronger effect 19 on a trait's average expression, its variance or its covariance with other traits, remains unclear. 20 We used a 2×2 factorial design crossing life-stage (juvenile and adult) and diet quality (low or 21 high-energy content) to disentangle the effects of developmental and adult diets on the 22 expression of behavioral differences. We tested 281 crickets for their activity levels, responses to 23 predator cues, and body-mass. Neither developmental diet nor adulthood diet had any effect on

24 population means or on the expression of an activity-antipredator response syndrome, suggesting 25 a genetic basis for this syndrome. We did find evidence for increases in the within-individual 26 variance as a result of exposure to a high-quality diet. However, these increases were only found 27 for antipredator response and body-mass. This indicates that diets with higher energy content can 28 increase the potential for behavioral plasticity in antipredator response. In addition to changes in 29 within-individual variation in behavior, diet quality during development also mediated the links 30 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-31 32 quality diet treatment. Our results show that changes in developmental diet quality can mediate 33 the relationship between life-history and behavioral traits later in life. 34

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

39 Introduction

40 Organisms live in changing environments, experiencing different environmental 41 conditions and selective pressures through their lives. By altering trait expression throughout 42 their lives, e.g. during development versus adulthood, organisms are expected to better match 43 their phenotypes to these environmental changes and thereby increase their fitness. Our 44 understanding of how the environment in which an individual develops affects its fitness is 45 generally based on one of two predictive frameworks: the "silver spoon" model or the 46 "environmental matching" model (Marshall & Uller, 2007; Monaghan, 2008). Under the silver 47 spoon model, offspring born in favorable environments always have higher fitness than 48 individuals born in poor environments, regardless of the adult environment. In contrast, the 49 environmental matching model suggests that experiencing the same environment during 50 development and adulthood should maximize fitness. While the consequences of fluctuating or 51 stable environments on fitness are well understood, how these changes manifest with traits 52 affecting fitness, such as behavior, remains unclear.

While the contrasting predictions of the environmental matching and the silver spoon 53 hypotheses have received some attention in the context of sexually-selected traits (e.g. swordtail 54 55 ornaments, Basolo, 1998) or mating behaviors (Scharf, Braf, Ifrach, Rosenstein, & Subach, 56 2015), these studies have typically been restricted to comparing changes in population averages. 57 However, as has been shown in several recent studies, diet manipulations can also have profound 58 effects on trait variances and covariances (DiRienzo & Montiglio, 2016; Han, Jäger, & 59 Dingemanse, 2016; Lichtenstein et al., 2016; Royauté & Dochtermann, 2017). To date the 60 relative contribution of diet quality during developmental and adult phases on the generation of 61 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 phases63 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 quality can also have more immediate effects ("temporary environmental effects", Dingemanse 67 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, tradeoffs are predicted to be manifested at the within-individual level (Figure 1C).

88 Here we investigated how the interaction between developmental and adult diet quality 89 affected variation in and correlations among body mass, activity, and response to cues of 90 predator presence in house crickets (Acheta domesticus). Specifically, we aimed to answer the 91 following questions: (1) Does the effect of diet quality on mean behavioral expression support 92 the silver spoon or the environmental matching hypotheses? (2) Does exposure to poor diet 93 quality lead to lower potential for behavioral plasticity? (3) Does diet quality affect the 94 magnitude of trait integration and shape trade-offs at the among- and/or within-individual level? 95 At the population average level, we predicted that if diet acted as a temporary environmental 96 effect, exposure to a high-quality diet during adulthood should result in unconstrained expression 97 of behaviors. In contrast, if diet acted as a permanent environmental effect during development, 98 we should see patterns in line with either the silver spoon or the environment matching 99 hypotheses (Figure 1A). At the level of trait variance, the cost of plasticity hypothesis suggests 100 that a high-quality diet results in higher potential for behavioral plasticity (i.e. higher within-101 individual variance). If diet quality acts as a temporary environment, we should expect the high-102 quality adult diets to show the highest within-individual variance, regardless of the 103 developmental diets. Alternatively, diet quality could have a permanent effect on behavioral 104 variance, in which case the within-individual variance is predicted to be highest for individuals 105 experiencing a high-quality diet during their development (Figure 1B). Finally, exposure to a



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

107

108 Figure 1. Conceptual framework showing how exposure to low (LQ) or high-quality (HQ) diets 109 may affect behavioral expression at multiple levels depending on whether diet acts on behavior 110 as a permanent environment or as a temporary environment. Diet order represents whether 111 exposure occurs during development (LQ_ and HQ_ diets) or adulthood (_LQ and _HQ 112 diets). At the population average level (A), if diet quality acts as a permanent environment, we 113 should expect results conforming to either the silver spoon or the environmental matching 114 hypotheses. If diet acts as a temporary environment, we should expect high-quality adult diets to 115 have the strongest effect on behavioral expression. At the variance level (B), we expect 116 behavioral plasticity to be costly to express. As a result, most changes are predicted to occur at 117 the within-individual variance level. At the covariance among traits level (C), we expect low-118 quality diets to generate more pronounced trade-off that can manifest either at the among-119 individual level if behavioral expression is generated through the permanent environment, or at 120 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 theselevels.

123

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).

129

130 Methods

131 Cricket housing, rearing, and diet preparation

132 We obtained 1-week-old A. domesticus nymphs from Fluker's Cricket Farm (~1 mm in size) and 133 reared them on one of two different diet treatments: either a high- (HQ, 45 % Protein, 11% Lipid, 134 23% Carbohydrate and 3% non-nutritive cellulose, with 3.35 cal/g) or low-quality diet (LQ, 135 15.33% Protein, 3.66% Lipid, 7.66% Carbohydrate and 65% non-nutritive cellulose with 1.12 136 cal/g). The high percentage of non-nutritive cellulose in the low-quality diet should have 137 imposed a gut limitation on individual crickets such that they could not have overcome the 138 relative energy differences by simply eating more (Royauté & Dochtermann, 2017). Upon 139 reaching sexual maturity, individuals were either switched to the other diet type or maintained on 140 the same diet. This resulted in a 2×2 factorial design crossing life-stage (juvenile and adult) and 141 diet type (low or high quality) with 86 individuals in the HQHQ, 78 individuals in HQLQ, 57 142 individuals in LQHQ, and 60 in LQLQ. Juvenile crickets were reared in plastic containers with 143 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%; $F_{1,45,53} = 0.60 P =$ 153 0.44, Figure A2).

154

155 <u>Behavioral tests</u>

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 \times 60 cm \times 15 cm high plastic 162 arena with a Plexiglas lid for both behavioral trials. The arena was split into four 30 cm \times 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%

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

173

174 *Open field behavior*

175 Individuals were left to rest for 30s in a 5 cm diameter cup introduced into the lower right section 176 of the arena (Figure A1, left panel). We then allowed the cricket to move freely through the 177 arena for 220 seconds. We measured an individual's activity, calculated as the total distance 178 travelled through the arena (in cm) and its exploratory propensity, calculated as the number of 179 unique zones visited by the cricket with Ethovision X (Noldus Information Technology). 180 Variations of this behavioral protocol have previously been used with A. domesticus to evaluate 181 individual differences in activity and exploratory behaviors (Dochtermann & Nelson, 2014; 182 Royauté & Dochtermann, 2017; Royauté, Greenlee, Baldwin, & Dochtermann, 2015).

183

184 *Antipredator response*

185 To measure responses to cues of potential predator presence here we collected excreta from three

186 adult leopard geckos (*Eublepharis macularius*) that were fed a mixed diet of crickets (A.

187 *domesticus*) and mealworms (*Tenebrio molitor*). Leopard geckos were housed according to

188 North Dakota State University IACUC standards (Protocol number: A14006). Collected excreta

189 was frozen and then finely ground weekly and diluted with deionized water (1 ml H₂O: 5 mg

190 excreta). This solution was then applied to 15 cm diameter filter paper discs with a 5 cm 191 diameter central cutout that allowed crickets to be left to rest unexposed to the predatory cue 192 (Royauté & Dochtermann, 2017). Each predator cue disc was left to dry for a minimum of 2 h, 193 was stored at 4 °C between trials, allowed to warm to room temperature before the start of a trial, 194 and discarded after a single use. We inserted the predator cue disc at the bottom of a 15cm 195 diameter arena and left the cricket to rest for 30 s under a 5 cm diameter cup in the non-treated 196 central cutout (Figure A1, right panel). We then allowed the cricket to move freely for 220 s and 197 estimated the distance travelled (in cm) through Ethovision. Previous experiments with this 198 protocol showed that crickets had heightened activity levels in presence of diluted gecko excreta 199 compared to a water control (Royauté & Dochtermann, 2017), thus greater activity during 200 antipredator response trials was interpreted as greater responsiveness to predator cues.

201

202 Data analysis

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

204 <u>Effect of diet quality on average trait value</u>

205 To analyze how diet treatment affected average trait expression, we used univariate linear mixed 206 models for all traits (adult mass, maturation time, open-field activity and unique zones travelled, 207 antipredator activity and adult body-mass during behavioral trials) using the lme4 package for 208 mixed effect models (Bates, Machler, Bolker, & Walker, 2015). Diet treatment (developmental 209 diet, adult diet and their interaction), temperature at which the trial was conducted (expressed as 210 among and within individual values; van de Pol & Wright, 2009), repetition number, batch, time 211 (expressed as among and within individual values; van de Pol and Wright 2009) and day of 212 recording (centered around the population average) were included as fixed effects. Cricket

213	identity along with developmental container ID (the container used in the growth chamber) were
214	included as random factors for all traits. Significance was assessed using F-tests based on
215	Kenward-Roger approximations for the degrees of freedom in mixed models using the
216	lmerTest package (Kuznetsova, Brockhoff, & Christensen, 2017). We also report the
217	proportion of variance (R ²) explained by fixed and random effects components following
218	Nakagawa & Schielzeth (2013).
219	
220	Effect of diet quality on variance components
221	To test whether diet quality influenced trait variation at the among and within-individual level,
222	we compared the fit of four different univariate mixed models on all traits for which repeated
223	measures were obtained (activity and unique zones travelled during open-field trials, antipredator
224	activity and adult body-mass during behavioral trials):
225	• Model 1: a null model where the among- (<i>Vi</i>) and within-individual variances (<i>Vw</i>) were
226	kept constant among diet treatments.
227	• Model 2: a model where only the among-individual variance differed among diet
228	treatments, while the within-individual variance was kept constant ($Vi \neq \& Vw =$)
229	• Model 3: a model where only the within-individual variance differed among diet
230	treatments while the among-individual variance was kept constant ($Vi = \& Vw \neq$)
231	• Model 4: a model where both the among and within-individual variance were allowed to
232	vary among diet treatments ($Vi \neq \& Vw \neq$)
233	These models were specified using the MCMCglmm package for Bayesian mixed models
234	(Hadfield, 2010) using Markov chain Monte Carlo (MCMC) with 1.3×10^6 iterations, 3×10^5
235	burn-in period and a thinning interval of 1000 and an inverse-Wishart prior. Our parameter

236 estimates were very similar to those obtained by Maximum Likelihood estimation, suggesting 237 that prior type had little influence on our results. We then compared the Deviance Information 238 Criterion (DIC) among each model. The model with the lowest DIC value was considered the 239 best model and models with $\Delta DIC > 5$ were considered a significantly poorer fit. Models with 240 $\Delta DIC < 5$ were considered as having equivalent support compared to the best model (Barnett, 241 Koper, Dobson, Schmiegelow & Manseau 2010; Spiegelhalter, Thomas, Best & Lunn 2007). 242 All models were specified with the same fixed effect structure as specified above to 243 prevent biased estimates of variance components and repeatability (Nakagawa & Schielzeth, 244 2010; Westneat, Hatch, Wetzel, & Ensminger, 2011). We included only cricket identity as a 245 random factor since the variance explained by the rearing containers (container ID) did not 246 exceed 7 % for any trait with repeated measurements (Table A1). All response variables were 247 expressed as standard deviation units to facilitate model convergence. We report the posterior 248 modes and 95 % credible intervals for variance components and adjusted repeatability (τ), 249 calculated as the posterior mode of $\tau = Vi / (Vi + Vw)$.

250

251 <u>Effect of diet quality on trait integration</u>

We first estimated among- and within-individual correlations among behavioral traits and bodymass 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

282achieved by calculating the r_{Obs} for each slice of the posterior distribution of correlation283estimates between the two treatments considered in order to obtain 95 % credible284intervals. We then compared the overlap of the posterior distribution of r_{Obs} values with a285randomized r (r_{random}) obtained after 100 permutations of the dataset. We base our286inference on the Pmcmc for the overlap between the posterior distribution of r_{Obs} values287with r_{random} (i.e. the number of r_{Obs} values that are equal or exceed r_{random}).

288 Finally, we tested whether integration between behaviors and life-history traits (body-289 mass at maturation and maturation time) changed with exposure to low- and high-quality 290 developmental diets. To do so we specified a series of 8 bivariate models whereby the among-291 individual correlation between a behavior and a life-history trait was estimated for a given 292 developmental diet treatment. Because our life-history traits represented unique events, we 293 estimated their correlations with behavioral traits by fixing the within-individual variance of life-294 history traits to a small value (V = 0.0001) following the recommendations of Houslay and 295 Wilson (2017).

296

297 **Results**

298 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 ($\mathbb{R}^2 < 0.07$) (Table A1). Further, maturation time differed between treatments when

- 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.

309 **Table 1.** Mean, standard errors and sample sizes for behavioral and growth traits compared across diet treatments. *Nobs* indicates the

310 number of observation taken for each diet treatments, *Ni* indicates the number of individuals tested. The number of observations is

311 lower with antipredator activity because a small portion of the individuals managed to escape the test arena or crawl under the

312 predator cue filter paper. These observations were excluded prior to analysis.

313

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	32.92	0.50	325	117					30.7	0.38	446	164				
(days)																
Adult phase	lase LQLQ		LQH	Q			HQL	Q			HQH	Q				
	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

315 Effect of diet quality on variance components

316 We found evidence for significant changes in trait variation among diet quality treatments 317 except for antipredator activity and body-mass. For both of these traits, the model allowing 318 differences at the within-individual level as well as the model allowing differences at the among 319 and within-individual levels had equivalent support (model 4, $\Delta DIC < 1.5$). Other models were 320 strongly rejected ($\Delta DIC > 10$, Table 2). This indicates that diet manipulation directly affected 321 within-individual variation and possibly affected among-individual variation. For antipredator 322 activity, the strongest differences were detected between the HQHQ and HQLQ diets at the 323 within individual level ($\Delta Vw = 0.28 \pm [0.14; 0.52]$) and the HQLQ and LQLQ diets at the 324 among-individual level ($\Delta Vi = 0.24 \pm [-0.04; 0.56]$). For body-mass, the HQHQ diet had the 325 highest within-individual variance compared to all other diets ($|\Delta Vw| \sim 0.10$, Figure 2; Table 3). 326





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 ($\Delta DIC < 5$).

Model	Variance comparison	Activity (OF)		Unique zon	es (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	$Vi \neq \& Vw =$	2014.00	0.83	2120.05	0.08	1877.08	11.70	761.51	15.70	
Model 3	$Vi = \& Vw \neq$	2017.16	3.99	2123.10	3.13	1865.38	0.00	745.81	0.00	
Model 4	$Vi \neq \& Vw \neq$	2017.74	4.57	2122.74	2.77	1866.32	0.94	746.82	1.01	

Trait				Diet quality				
	L	QLQ	LC	QHQ	H	QLQ	HO	QHQ
	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 (O	F)							
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]

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]).

338

339 Effect of diet quality on trait integration

340 We did not find evidence of trade-offs between traits at the among-individual level, i.e. we did 341 not detect negative correlations among behaviors (Figure 3, Table A2). Regardless of diet type, 342 open-field activity and unique zones visited were the most strongly correlated traits at both 343 among- and within-individual levels ($r_i > 0.55$ and $r_w > 0.70$). In contrast, we found evidence for 344 diet quality affecting the strength of the activity-antipredator response syndrome. In particular, 345 an activity-antipredator response syndrome was not detectable for the LQLQ diet (correlation \pm 346 [95 % CRI], open-field × antipredator activity: $r_i = 0.18 \pm [-0.08; 0.54]$, Pmcmc = 0.91; unique 347 zones × antipredator activity: $r_i = 0.11 \pm [-0.22; 0.47]$, Pmcmc = 0.78). All other diets showed 348 significant correlations varying between 0.27 (HQHQ unique zones × antipredator activity, [-349 0.02; 0.56], Pmcmc = 0.96) and 0.51 (LQHQ open-field × antipredator activity, [0.17; 0.72], 350 Pmcmc = 0.99). Body-mass was generally poorly integrated with behavioral traits (average $r_i <$ 351 (0.20) and none of the correlations reached statistical significance (Pmcmc < 0.92). 352 At the within-individual level, individuals increasing their activity and exploration levels 353 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 354 355 diet (open-field activity x body-mass: $r_w = -0.20 \pm [-0.33; 0.06]$, Pmcmc = 0.93; unique zones × 356 body-mass: $r_w = -0.18 \pm [-0.34; 0.03]$, Pmcmc = 0.95; antipredator activity × body-mass: $r_w = -$ 357 0.14 [-0.33; 0.06], Pmcmc = 0.89). This means that the individuals that gained mass during the 358 course of behavioral measurements tended to decrease their activity levels. However, these trade-359 offs run contrary to our predictions that the low-quality adult diet should be the one generating 360 the highest number of trade-offs at the within-individual level.



362

Figure 3. Among (r_i , lower diagonal) and within-individual (r_w , upper diagonal) correlations for behavioral measurements and body-mass based on multi-response mixed models. 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). Shape of ellipses indicates strength and—along with color—the direction of correlations.

We did not find evidence for differences in the magnitude of phenotypic integration being mediated by diet quality. Although the HQLQ diet had consistently higher values for all integration metrics at the among-individual level (network density d = 0.83, average correlation strength $|r| = 0.31 \pm [0.21; 0.51]$, 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 ($\Delta r_i = 0.45$ \pm [-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).



382

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.

388 Table 4. Effect of diet quality during development on the relationships between maturation time, adult body mass and behaviors. Bold

389 values indicate significant correlations based on 95 % of estimates excluding 0 (Pmcmc > 0.95). Italicized and bold values indicate

390 correlations where > 90 % of estimates exclude 0 (Pmcmc > 0.90).

391

Bivariate correlation		Low Quality			High Quality			$\Delta r (\text{HQ} - \text{LQ})$	
	r	[95 % CRI]	Pmcmc	r	[95 % CRI]	Pmcmc	Δr	[95 % CRI]	Pmcmc
Activity (OF) \times 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 $(AP) \times Maturation$	-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

393 **Discussion**

394 In the present study, we set out to test whether diet quality during development, diet quality 395 during adulthood, or their interactions had the strongest effect on behavioral variation and 396 covariation (Figure 1). We did not find support for either the environmental matching or silver 397 spoon hypotheses regarding mean behavioral expression since mean behaviors did not vary 398 significantly among diet treatments. Instead, we found evidence of increased variability in 399 antipredator response for individuals provided a high-quality diet while adults. Our hypothesis 400 that a low-quality diet would generate trade-offs among traits (Figure 1C) was poorly supported. 401 The few negative correlations detected between behaviors and body-mass had weak effect sizes 402 and were not consistent with our predictions. Finally, we found that overall integration among 403 behavioral traits and body-mass did not vary by diet treatments, although the presence of an 404 activity-antipredator response syndrome was only detected for specific diet types.

405 Diet did not affect trait expression at the population average level which leads us to reject 406 both the silver spoon and environment matching hypotheses. Our diet quality manipulation also 407 had surprisingly little influence on body-mass at maturation and maturation time. This is contrary 408 to what was observed in a previous study where crickets tracked during the first 30 days of their 409 development showed higher growth rates when exposed to the high-quality diet (Royauté & 410 Dochtermann 2017). Several compensatory mechanisms may explain these differences. First, 411 these two experiments were conducted at different developmental temperatures (Royauté & 412 Dochtermann 2017: 25°C, current experiment: 32°C). Increased temperatures generally result in 413 faster developmental rates in ectotherms (Angilletta and Dunham 2003, Atkinson, 1994; 414 Carleton, 1960), and this could partially account for the lack of difference in growth rate between 415 our diet treatments. Second, the present experiment required the use of group-housing during the

416 developmental phase while crickets were reared in isolation in the prior experiment. Container 417 ID during the developmental phase explained between 16-19 % of the variation in body-mass at 418 maturation and maturation time, which suggests that the social environment may play a larger 419 role in cricket growth than diet quality. Finally, we cannot exclude the possibility that 420 cannibalism may have been present during the developmental phase of the experiment and could 421 have affected our results. However, given that mortality rates did not differ significantly between 422 developmental diets, if cannibalism occurred, it likely did not affect individuals in the low-423 quality diet at a higher rate compare to individuals in the high-quality diet. Interestingly, while 424 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 425 426 body-mass measured during the adult phase of the experiment. This suggests that the social 427 environment experienced during the developmental phase did not carry-over to affect adult 428 behaviors.

429 Instead of affecting average trait expression, most of the effects of our diet manipulations 430 were manifested at the level of variances of traits and covariances among traits. Antipredator 431 response and body mass both showed significant differences in their within-individual variance 432 among treatments, while none of the open-field behaviors showed any difference. For both traits, 433 the HQHQ diet had the highest within-individual variance thus giving support for the cost of 434 plasticity hypothesis. However, we were not able to distinguish whether such patterns were due 435 to diet acting as a permanent source of variation during development or instead represented 436 temporary source of behavioral variation. Our results are in line with recent studies showing that 437 changes in diet quality or composition primarily influence the expression of within-individual 438 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 amongindividual 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.

446 Contrary to our predictions (Figure 1), we found a surprising lack of trade-offs between 447 behavioral traits and body mass. The only instances of negative correlations were observed 448 between open-field behaviors and body-mass at the within-individual level in the LQHQ diet. 449 However, the magnitude of these correlations was low ($-0.20 < r_w < -0.14$), suggesting at best a 450 weak trade-off between changes in activity and exploration levels with changes in body mass. 451 More importantly, this result runs contrary to our expectations that if diet acts as a temporary 452 environmental effect, trade-offs will manifest at the within-individual level for individuals 453 provided with a low-quality diet as adults (Figure 1).

454 We also found evidence for diet quality acting as a mediator of life-history \times behavior 455 correlations. In particular, individuals that matured faster in the low-quality diet had increased 456 exploratory propensity, while this correlation was absent in the high-quality diet. Links between 457 life-history and behavioral traits are often understood within the pace-of-life syndrome 458 hypothesis (POLS) (Réale et al., 2010), where individual with "live fast-die young" strategies 459 adopt more risky behavioral strategies in terms of resource accumulation (i.e. higher exploration 460 propensity and boldness). While support for the POLS hypothesis is mixed at best (Royauté, 461 Berdal, Garrison, & Dochtermann, 2018), there is theoretical evidence that changes in resource

462 availability can affect the magnitude of integration among some POLS traits (Salzman, 463 McLaughlin, Westneat, & Crowley, 2018). Note that our observed changes to life-history \times 464 behavior correlations are hard to put into the context of the predictions of the POLS hypothesis 465 because we did not obtain precise estimates of growth rate and longevity, which would be 466 required to demonstrate the presence of slow-fast life-history strategies. Moreover, many of the 467 predictions of the POLS hypothesis imply the presence of a proactive-reactive behavioral axis, 468 whereby highly active individuals are also superficial explorers and are bolder when exposed to 469 threat. In contrast, the behavioral syndrome we observed did not show the negative correlation 470 between activity levels and thoroughness of exploration expected through the proactive-reactive 471 axis (Koolhaas et al., 1999; Verbeek, Drent, & Wiepkema, 1994). Contrary to our expectations, 472 our results also did not indicate the presence of trade-offs between life-history and behavioral 473 traits being mediated by diet quality. Instead, they suggest that, in a nutritionally depleted 474 environment, only high-quality individuals can mature faster and express high exploratory 475 propensity levels. In other words, it seems that diet quality during development can act as an 476 equalizer rather than generate strong trade-offs between life-history and behavior through 477 restrictions on energy budgets (i.e. "reverse trade-off", Careau & Wilson, 2017; Reznick, 478 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 ofindividual differences in behavior.

510

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650 651	

652	Appendices for: "Do developmental environments and current energy state mediate the
653	potential for plasticity and behavioral integration?"
654	
655	
656	Figure A1. Arena designs for open-field activity and antipredator response trials.
657	
658	Figure A2. Survival rates did not differ significantly between diet quality treatments during
659	the developmental phase of the experiment.
660	
661	Table A1. ANOVA table summarizing the significance of fixed effects on trait expression
662	
663	Table A2. Among (lower diagonal) and within-individual (upper diagonal) correlations for
664	behavioral measurements and body-mass based on multi-response mixed models
665	
666	Table A3. Integration metrics compared among the different diet treatments for among and
667	within-individual correlations matrices.
668	
669	Table A4. Modified Mantel's tests for comparing correlation matrices similarity across diet
670	treatments.
671	





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



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).

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.

Trait	t Fixed Effects		Random Effects						
	16	F	Л	Variance	Variance	[050/ CI	D ²		
	di	Г	P	component	variance	[95% CI]	K-		
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) \times	1 055 00	0.15	0.60	T 7	2550	50000 (10.6)	0.00		
Sex	1, 255.20	0.17	0.68	Vw	3559	[2928; 4186]	0.69		
Diet (dev) ×	3 12 77	1 10	0.36						
Say y Patab	3, 42.77	0.77	0.50						
Maturation	5, 239.23	0.77	0.31						
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) \times	,								
Sex	1, 255.29	0.11	0.74	Vw	17.24	[14.23; 20.45]	0.57		
Diet (dev) \times									
Batch	3, 42.77	1.39	0.26						
$\text{Sex} \times \text{Batch}$	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) \times									
Diet (ad)	1, 262.50	0.18	0.67						
Diet (aev) ×	1 259 02	0.20	0.50						
Diet (ad) v	1, 230.92	0.29	0.39						
Sex	1, 264.53	1.04	0.31						

Trait	Fixed Effects			Random Effects			
	10			Variance	X 7 ·		D ²
Unique zones	df	F	Р	component	Variance	[95% CI]	R ²
(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) Temperature	1, 497.76	0.17	0.68				
(mean) Temperature	1, 245.16	0.47	0.49				
(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) \times Diet (ad) Diet (dev) \times	1,260.05	0.37	0.54				
Sex Diet (ad) \times	1, 256.97	0.33	0.56				
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) Temperature	1, 481.93	0.15	0.69				
(mean) Temperature	1, 261.05	2.36	0.12				
(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) \times Diet (ad)	1, 262.90	0.03	0.86				
Sex Diet (ad) \times	1, 257.46	0.19	0.66				
Sex	1, 265.85	1.68	0.19				

)94)05

Trait	Fixed Effects			Random Effects			
	df	Variance F P component Varia		Variance	[95% CI]	\mathbb{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) \times							
Diet (ad)	1, 261.66	0.09	0.77				
Diet (dev) \times							
Sex	1,250.09	1.32	0.25				
Diet (ad) \times							
Sex	1, 258.72	0.07	0.78				

Table A2. Among (lower diagonal) and within-individual (upper diagonal) correlations for
behavioral measurements and body-mass based on multi-response mixed models with
associated 95 % credible intervals (in bracket). 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).

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

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

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

715 **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.

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		Observed				Random		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]			