Heredity (2021) 126:684–694
https://doi.org/10.1038/s41437-020-00395-5

the geneticssociety

ARTICLE

The sources of variation for individual prey-to-predator size ratios

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Received: 16 August 2020 / Revised: 3 December 2020 / Accepted: 7 December 2020 / Published online: 15 January 2021

Abstract

The relative body size at which predators are willing to attack prey, a key trait for predator-prey interactions, is usually considered invariant. However, this ratio can vary widely among individuals or populations. Identifying the range and origin of such variation is key to understanding the strength and constraints on selection in both predators and prey. Still, these sources of variation remain largely unknown. We filled this gap by measuring the genetic, maternal and environmental variation of the maximum prey-to-predator size ratio ($PPSR_{max}$) in juveniles of the wolf spider *Lycosa fasciiventris* using a paternal half-sib split-brood design, in which each male was paired with two females and the offspring reared in two food environments: poor and rich. Each juvenile spider was then sequentially offered crickets of decreasing size and the maximum prey size killed was determined. We also measured body size and body condition of spiders upon emergence and just before the trial. We found low, but significant heritability ($h^2 = 0.069$) and dominance and common environmental variance ($d^2 + 4c^2 = 0.056$). $PPSR_{max}$ was also partially explained by body condition (during trial) but there was no effect of the rearing food environment. Finally, a maternal correlation between body size early in life and $PPSR_{max}$ indicated that offspring born larger were less predisposed to feed on larger prey later in life. Therefore, $PPSR_{max}$, a central trait in ecosystems, can vary widely and this variation is due to different sources, with important consequences for changes in this trait in the short and long terms.

Introduction

Different sources of phenotypic variation have different implications for ecology and evolution. Indeed, responses to selection mostly rely on the additive genetic variation, but other sources of variation may affect some of the characteristics of this response. In addition, from an ecological perspective, all sources of trait variation may in principle impact ecosystem functioning. Changes in the latter will in turn set the stage for new selection pressures to operate on individual traits (Bolnick et al. 2003; Violle et al. 2012; Hart et al. 2016; Costa-Pereira et al. 2018). This is particularly important in traits that evolve at fast rates. Indeed, different sources of trait variation may indirectly affect evolutionary responses by inducing environmental changes that subsequently act as new selective pressures. This is the case when phenotypic variation affects ecological interactions, such as predation (e.g., Moya-Laraño 2011; Bolnick et al. 2011; Schreiber et al. 2011). Understanding the potential impact of phenotypic variation on predator–prey interactions and its evolutionary potential thus requires identifying the origin of such variation (Bolnick et al. 2011).

Theory predicts that the effect of intraspecific variation upon the outcome of ecological interactions depends on the relative strength of environmental vs genetic variation (Schreiber et al. 2011; Moya-Laraño et al. 2014; Cortez 2018; Maynard et al. 2019). For example, depending on the type of interaction, systems where the phenotypic variance

These authors contributed equally: Sara Magalhães, Jordi Moya-Laraño

Associate editor: Armando Caballero

Supplementary information The online version of this article (https://doi.org/10.1038/s41437-020-00395-5) contains supplementary material, which is available to authorized users.

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of traits is largely determined by genetic variance tend to be more (e.g., competition—Maynard et al. 2019) or less (e.g., apparent competition—Schreiber et al. 2011) stable than those where trait variation depends on environmental conditions. Also, since genetic variability enhances evolutionary responses, genetic diversity (number of genotypes) in prey can lead to the stabilization of predator–prey dynamics via the evolution of resistance to predation (Yoshida et al. 2003).

Maternal effects can also contribute to stabilizing predator–prey interactions, as shown both theoretically (Benton et al. 2001; Inchausti and Ginzburg 2009) and empirically (Gustafsson et al. 2005; Sheriff et al. 2010). Maternal effects can add up to 50% of the total phenotypic variance of traits (Moore et al. 2019) and these effects can strongly impact the expression of traits involved in predator–prey interactions (LaMontagne and McCauley 2001; Walsh et al. 2016). Maternally driven phenotypic changes may also impact adaptive responses, as they can be a pervasive source of trait variation in the absence of strong additive genetic effects (Wolf and Wade 2016) and can contribute to evolution, especially in variable environments (Dey et al. 2016).

Other non-additive genetic effects, such as dominance and epistasis can potentially affect ecological and evolutionary dynamics as well. Indeed, the contribution of dominance to fitness related traits can be relatively high (Mousseau and Roff 1987; Crnokrak and Roff 1995; Wang et al. 1998; Wolak and Keller 2014; Sztepanacz and Blows 2015; summarized in Caballero 2020; but see Class and Brommer 2020). Dominance can stabilize the dynamics of predator–prey interactions (Stewart 1971). Although the contribution of epistasis should not be ruled out (Hansen 2013), it is difficult to quantify in natural populations (Carlborg and Haley 2004) and laboratory crossing designs are not amenable for species with long generation times (Lynch and Walsh 1998).

Genetic correlations among traits also have the potential to foster or constrain evolutionary (Cheverud 1996; Roff 1997) as well as ecological responses. For instance, simulations show that depending on temperature, genetic correlations can differentially affect predator–prey interactions (Moya-Laraño et al. 2012). Maternal effects may also impact multiple traits simultaneously, acting as a source of covariation among offspring traits, thus generating maternal correlations, through non-genetic factors such as hormones (McGlothlin and Ketterson 2008).

Body size is one of the most fundamental functional traits of an organism (Brown et al. 2004). It determines trophic position, as larger predators may be able to feed on relatively smaller prey (Woodward and Hildrew 2002; Woodward et al. 2010). Therefore, it is a fundamental trait to determine the strength of interactions in food webs, and thus their stability (Jonsson and Ebenman 1998; Emmerson and Raffaelli 2004; Rooney et al. 2006; Otto et al. 2007; Schneider et al. 2016). Variation in body size is determined by several sources, including genetic, maternal, dominance and environmental variation (Gebhardt-Henrich and Van Noordwijk 1991; Mousseau and Fox 1998; De Jong and Imasheva 2000). However, due to the long-standing practice in community ecology of collapsing species to their mean values (Tilman et al. 2014), the relative size of interacting predators and prey, captured by the predator–prey size ratio, is traditionally considered to be invariant for a given predator–prey interaction (Brose et al. 2006, 2008; Laigle et al. 2018; Cuthbert et al. 2020). However, there is ample evidence for within-species variation in size with large consequences for predator–prey interactions and community dynamics (De Roos et al. 2003; Magalhães et al. 2005; Nakazawa et al. 2011). Therefore, ignoring this variability may lead to erroneous estimations of the scaling relationship between predators and prey.

Here, we investigate the sources of intraspecific variation in prey-to-predator size ratio (PPSR) of the soil predator *Lycosa fasciiventris* (Dufour 1835), a non-burrowing wolf spider inhabiting the Iberian Peninsula. Spiders of this genus are generalist predators, feeding on an array of mid-to-large size arthropods including conspecifics (Moya-Laraño et al. 2002; Gavín-Centol et al. 2017). Specifically, we assess the role of additive, maternal and environmental effects in determining the PPSR of spiders feeding on crickets, a common prey of wolf spiders and abundant in the habitat of this species. Identifying the relative contribution of environmental, maternal and genetic components affecting variation in PPSR will shed light into its evolutionary potential and provide a deeper understanding of its potential to modulate community structure and ultimately ecosystem functioning.

Material and methods

Spider collection

Individuals of *L. fasciiventris* were collected from June 23 to July 27, 2015 in four different localities within the Almeria province (South-East Spain), in dry temporal washes ("ramblas"): (1) around Paraje las Palmerillas, Estación Experimental de Cajamar (36.7917° N, 2.6891° O); (2) near Boca de los Frailes village (36.8036° N, 2.1386° O); (3) near Carboneras village (36.9667° N, 2.1019° O) and (4) near Almanzora river (37.3414° N, 2.0078° O). Individuals were then kept separately in the laboratory in a container (22 × 18 × 18 cm) with the bottom filled with 2–3 cm of soil collected from the sampling sites. Two wooden blocks (10 × 8 × 1 cm and 3 × 5 × 1 cm) were

added to each tank to provide shelter. Only sub-adult virgin females were used to form the laboratory population. All individuals (adult and sub-adult males, and sub-adult females) were fed once a week with size-matched crickets (*Gryllus assimilis;* Fabricius 1775) purchased from a pet supply online store Exofauna, Spain (available in: https://exofauna.com). Spiders had access to water ad libitum through a 40 ml vial filled with water and covered with cotton. Tanks were placed in a climate chamber with simulated outdoor climatic conditions (day and night temperature cycles and photoperiod with light fluorescent tubes of 54 W, mimicking natural sunshine, and a relative humidity from 50 to 65%). Climatic conditions were adjusted to the preceding weekly average conditions in the Almeria province, with day–night temperature and light oscillations (temperature: 18.7–34.3 °C; light–dark photoperiod: 17:7–16:8 h).

Breeding design

To assess genetic, maternal and environmental variation in individual PPSR, we performed a paternal half-sib split-brood design (Roff 1997; Lynch and Walsh 1998), in which 52 males (sires) were each mated with two virgin females (dams). Each week, offspring were provided with fruit flies (*Drosophila melanogaster;* Meigen 1830) originated from cultures produced in the laboratory. Flies were fed with a nitrogen rich medium supplemented with high-quality dog food, which highly improves spider survival (Jensen et al. 2011). Maternal families were constituted by 12 offspring, split into two food availability treatments, varying in the number of flies provided. Thus, 3 out of 12 offspring from each maternal family were assigned to the rich environment, being given 3× the amount of food provided in the poor (or standard) environment. Initially, a single fly was offered to the spiders in the poor treatment and three flies in the richer treatment. This quantity was adjusted to three and nine when individuals were approximately 6 months old due to higher food demand at that stage.

After hatching, spiderlings of wolf spiders climb to the female back and, in *L. fasciiventris*, remain with it for a period of a few weeks (Parellada 1998). Due to logistic reasons, all spiderlings were removed from the female back within 1 week, which is approximately 42 ± 8 (mean ± SD) days after they hatched (age at isolation). To estimate and control for post-hatching common environmental effects occurring on the female back, the age at isolation was included in all models. This variable was never significant (data not shown). Spiderlings were carefully collected from the female back with the help of a paintbrush. We took 12 spiderlings from each female and placed them separately in cylindrical containers (5 cm height and 6 cm diameter). Each container had the bottom covered with filter paper, providing a substrate for both locomotion and absorption of excreta, inside the growth chamber. Filter papers were checked weekly and replaced if necessary. A plastic tip was inserted at the bottom of the container, filled with cotton connected to a reservoir, providing water ad libitum to spiders by capillarity (Moskalik and Uetz 2011). The 1248 spiderling containers were then randomly arranged within the growth chamber to ensure that individuals belonging to the same family were spatially interspersed. This allowed mitigating possible common environmental effects after spiderling isolation from their mothers.

Morphometry

Body components were divided between structural body size (carapace width; Hagstrum 1971) and body condition (residuals of abdomen width on carapace width; Jakob et al. 1996). Body condition reflects energy and nutrient storage independently on the size of the spider and thus reflects hunger level (Moya-Laraño et al. 2008). Structural body size may reflect the strength to subdue prey (e.g., Moya-Laraño et al. 2002). Both carapace and abdomen width were measured at their widest point.

Body size and body condition were measured in two instances: after individuals were taken from their mothers and isolated, and immediately before the trials for acceptance. Morphometric measurements were taken to the nearest 0.1 mm with a dissection microscope (Leica MZ125). While structural body size measured at the time of trial was needed to calculate PPSR, body condition at the time of the trial was used to control for the hunger state of each spiderling (i.e., its motivational state). These traits were also measured early in life and used to calculate genetic and maternal correlations, to test how maternal investment in both offspring body size and condition could affect behavioural patterns of the spiders later in life.

Prey acceptance

This experiment aimed to measure the maximum relative size of a prey cricket (*Gryllus assimilis*) that a spider accepted, considering a range of cricket lengths (in mm) decreasing from 5× to 1× (in units of 1) the carapace width of the spider. For that, we placed them in experimental arenas where each spider was offered crickets in a decreasing order of relative size until it subdued and killed a cricket. The response variable, PPSR, is the ratio at which the spider attacks and kills the cricket. This measure corresponds to the maximum PPSR ($PPSR_{max}$) at which predators kill their prey and the larger the relative size of the prey killed, the higher the PPSR. Spiders were measured in blocks of 17 ± 5 (mean ± SD) individuals. Each block was defined as the experimental batch of individuals assessed in each day.

Although this cricket species does not occur in the study site, *L. fasciiventris* is able to effectively prey on it, and a similar species with similar body size, *Gryllus bimaculatus*, is highly abundant in the collection area (Moya-laraño *personal observation*). As it was not feasible to collect *G. bimaculatus* in numbers enough to carry out this study, we used *G. assimilis* individuals from an established laboratory population. Note that this approach allowed testing the response of spiders that were naive to this prey, as all spiders had been fed with *Drosophila* to that point. Thus, this approach minimized environmental variation due to potential effects of previous experiences with cricket prey.

In the trial, we used crickets with a length that differed from the target PPSR (5×, 4×, 3×, 2× or 1× of the width of the spider carapace) by less than 0.2 units. Crickets were weighted, and their length determined from a calibration curve, previously generated with the weight and length of 40 crickets: $L = 3.22 + 0.32 \log(M)$; $R^2 = 0.99$; $p < 0.0001$; where L is cricket body length (in mm) and M is cricket body mass (in mg). Mass was measured to the nearest 0.1 mg using a high precision scale (Mettler Toledo XP26). None of the crickets were used in more than one trial.

To standardize hunger levels across individuals, spiders were left to starve for 7 days before being tested, similarly to other studies (Persons and Rypstra 2000). As it was not possible to standardize age across trials, individuals were randomly assigned to each trial. Spider age at the time of each measurement (331 ± 30 days old, mean ± SD) was recorded and later controlled for in the statistical analysis as a covariate (see below). A single spider and one cricket were placed inside the arena (7.5 cm diameter), in opposite sides, within enclosed inverted plastic vials (3 cm diameter). Then, both vials were gently lifted simultaneously, and crickets and spiders were allowed to interact for 6 min. If the cricket was not captured and subdued, the spider was enclosed in the vial and the cricket was removed. Spiders were then left to recover in the vial for 30 min until a new cricket from the next immediately lower size was presented (lower PPSR). Trials ended as soon as the spider attacked and killed a cricket or if the spider did not catch the smallest (1×) cricket.

Estimation of variance components and statistical analysis

The paternal half-sib breeding design allows partitioning the total phenotypic variance (V_P) into the following sources of variation:

$$V_P = V_s + V_d + V_w, \quad (1)$$

where V_s is the variance among sires, V_d the variance among dams within sires and V_w the variance within full-sib families. The genetic/environmental causal components of the sources contributing to phenotypic variation (V_P) are then (Lynch and Walsh 1998):

$$V_s = \frac{V_A}{4}, \quad (2)$$

$$V_d = \frac{V_A}{4} + \frac{V_D}{4} + V_{Ec}, \quad (3)$$

$$V_w = \frac{V_A}{2} + \frac{3V_D}{4} + V_{Es}, \quad (4)$$

where V_A is the additive genetic variance, V_D is the dominance genetic variance, V_{Ec} is the component of variance attributed to common environmental (maternal) effects, and V_{Es} is the remaining environmental variation. The dam variance component includes, in addition to additive effects, dominance and common environmental (maternal) effects. The potential for post-natal common environmental effects to severely inflate the estimated maternal variance (V_{Ec}) was reduced by isolating offspring from their mothers as soon as possible after hatching, as referred to above (see "Breeding design" section).

Epistatic variance is implicitly included on the residual variance component, i.e., the variance within full-sib families (V_w), as its estimation requires much more complex, cross-classified designs (Pooni et al. 1978; Lynch and Walsh 1998). These designs are unfeasible for sexually cannibalistic spiders such as *L. fasciiventris* (Gavín-Centol et al. 2017), because they require crossing males with several females and vice versa.

The estimation of variance components was performed using univariate and multivariate mixed models in the MCMCglmm package (Hadfield 2010) in R (R 3.4.3 development core team 2018). In all models, we fitted body condition (at the beginning of the trial), food availability (spider in poor (1–3 flies) or in rich (3–9 flies) environment) and age as covariates. We did not include body size at the time of the trial as a fixed factor as it is in the denominator of PPSR. Accounting for it in our models would thus result in assessing the sources of variation for prey size, not those for the relative size differences between predators and prey. Sire (the father identity), dam (the mother identity) and block (trials performed at different times) were included as random effects. All traits were standardized to unit variance and zero centred prior to analyses.

We assessed the significance of variance components of $PPSR_{max}$ by comparing deviance information criterion (DIC) values of a total of four plausible models, which included sire (V_s) and/or dam (V_d) variance components and a null model excluding both random factors. The null model included fixed effects (age, food treatment and body condition), and variance was partitioned only in block (V_B) and

residual (V_R) random effects by fitting these as random terms. We then fitted a model by adding the sire variance component (V_s) to the null model, another adding solely the dam variance component (V_d), and a last model with both random variance components ($V_s + V_d$). Phenotypic variance in the most complete model comprised all the random variance components ($V_P = V_s + V_d + V_B + V_R$). Models that showed a difference between DIC values (ΔDIC) > 2 were considered statistically different (Burnham et al. 2011).

Priors used in this analysis were generated by partitioning the phenotypic variance evenly among each random term (Wilson et al. 2010) and given a low degree of belief ($nu = 0.2$). All models were run for 200,000 interactions, a burn-in of 5000 and a thinning interval of 100.

Narrow sense heritability (h^2) was estimated from the complete model as the proportion of additive genetic variance ($V_A = 4V_s$) to the total phenotypic variance ($h^2 = 4V_s/V_P$). Broad-sense heritability (H^2) was estimated as the proportion of four times the dam variance (Eq. 3) to the total phenotypic variance ($4V_d/V_P$) and thus, includes additive ($h^2 = V_A/V_P$) and dominance effects ($d^2 = V_D/V_P$). As V_d also includes common environmental (maternal) effects ($c^2 = V_{Ec}/V_P$), the estimate of H^2 is an upper limit of its true value.

Multivariate generalized linear mixed models were used to estimate genetic and maternal correlations between $PPSR_{max}$ and body size and body condition at isolation. We considered these morphometric measures at isolation because we aimed to (a) test if there is a relation between early life traits and $PPSR_{max}$ and (b) identify the source of such covariation. We did not test covariance between body size at the time of the trial and $PPSR_{max}$ because the former is included in the denominator of the latter. Also, the covariance between $PPSR_{max}$ and body condition at the time of the trial was not tested. Instead, the latter trait was fit as fixed effect, as its variation is expected to be largely explained by the rearing environment (i.e., the food availability treatment) and is thus a good surrogate trait to control for hunger state.

Genetic correlations (r_A) were calculated using the **G** matrix of covariance (Lynch and Walsh 1998) following the equation:

$$r_A = \frac{COV_{A(xy)}}{\sqrt{\left(var_{A(x)}\right)\left(var_{A(y)}\right)}}, \quad (6)$$

where $COV_{A(xy)}$ is the additive genetic covariance between two characters X and Y, and $var_{A(x)}$ and $var_{A(y)}$ are the additive genetic variance of X and Y, respectively. Maternal correlations (r_M) were calculated similarly but instead of variance and covariances for additive genetic effects, the expression was modified by using maternal variances ($var_{M(x)}$ and $var_{M(y)}$) and covariances ($COV_{M(xy)}$). Priors were 2×2 diagonal matrices where the diagonal corresponded to the variance for each trait and the off-diagonal to zero covariance between traits.

A sensitivity analysis was run for all univariate and multivariate models by testing several *nu* parameters (0.2–2.2) and revealed no substantial difference in the estimates obtained among the models tested. Moreover, we also tested for priors with varying proportion of the raw phenotypic variance attributed to the residual variances (0.025 and 0.95) (Wilson et al. 2010), leaving the remaining to be shared equally between the dam and sire components. Only the most robust results were considered, i.e., the ones which did not change substantially depending on the *nu* parameter or the prior variances. We evaluated model convergence by visual inspection of the time series plots of the model parameters and also ensured that autocorrelation values were less than 0.05 for all parameters included to grant independence of samples in the posterior distribution (Wilson et al. 2010). We also ran the models more than once to test that different chains (replicates) closely replicated our results (not shown).

Posterior credible intervals (CI) for the estimates of narrow and broad-sense heritabilities, and genetic and maternal correlations were calculated from the posterior distributions using the highest-posterior-density function (HPD interval, package MCMCglmm; Hadfield 2010). Covariances were supported when 95% CI excluded zero and when the model with sire and/or dam random effects had lower DIC values than null models. Because variances are bounded above zero, support of variances estimates was assessed by comparing the DIC values between fitted models.

Results

Individual body condition, measured before the trial, had a significant effect on $PPSR_{max}$, as individuals with better condition tended to feed on larger prey (Table 1). Age and food treatment did not significantly affect $PPSR_{max}$ (Table 1). In addition, the food treatment had a significant effect on body size and body condition measured during the

Table 1 Parameter estimates (posterior mean and credible interval) for the fixed effects (Age, body condition and food treatment) from analysis of standardized values from the complete model ($V_s + V_d + V_B + V_R$) for $PPSR_{max}$.

Variables	Post.mean	LCI	UCI	pMCMC
(Intercept)	0.036	−0.142	0.22	0.704
Age	−0.037	−0.151	0.092	0.536
Body condition	0.139	0.052	0.216	<0.001
Food treatment	−0.062	−0.223	0.115	0.475

Post.mean posterior mean, *LCI* lower credible interval, *UCI* upper credible interval, *pMCMC p* value based on MCMC sampling

Table 2 Summary results from models fitting sire and dam variance components.

Model	DIC	Δ DIC	V_s	V_d	V_B	V_R	$h^2 = 4V_s/V_p$	$H^2 \sim 4V_d/V_p$
Null	1578.65	0	–	–	–	–	–	–
V_s	1576.98	−1.668	–	–	–	–	–	–
V_d	1570.48	−8.164	–	0.039 (0.0119–0.098)	0.069 (0.023–0.141)	0.728 (0.664–0.850)	–	0.167 (0.056–0.425)
$V_s + V_d$	1572.82	−5.828	0.0136 (0.006–0.056)	0.034 (0.007–0.080)	0.053 (0.022–0.138)	0.759 (0.660–0.847)	0.069 (0.022–0.230)	0.125 (0.026–0.343)

ΔDIC is the difference between DIC values against the null model (lowest DIC). Estimates (posterior mode and credible interval) are only presented for the two best candidate models.

V_s variance among sire families, V_d variance among dam families, V_B variance among blocks, V_R residual variance, h^2 narrow sense heritability; H^2 broad-sense heritability (possibly inflated by common environmental (maternal) effects c^2, i.e., $H^2 \sim h^2 + d^2 + 4c^2$), d^2 dominance effects.

behavioural trials, where individuals in the richer food treatment had 1.32× larger body sizes (Fig. S1) and 1.14× superior body condition (Fig. S2). Moreover, although accepted prey size covaried positively with spider body size, we found a very wide range of absolute prey sizes accepted for a given spider body size. Also, across spider body sizes, no single optimal (i.e., more frequently hunted) prey size was found (Fig. S3).

Estimates calculated from the complete model ($V_s + V_d$) yielded a narrow sense heritability value for $PPSR_{max}$ of $h^2 = 0.069$ [CI: 0.022–0.230]. This value is low, but the model converged to a bell-shaped posterior distribution from which a global maximum (mode) could be obtained (Fig. S4). Although the best-fitted model, as observed by DIC comparison, included only the dam variance component (V_d), the complete half-sib design model (including $V_s + V_d$) was also different from the null model (Table 2). From the latter model, we found a broad-sense heritability value of $H^2 = 0.125$ [CI: 0.026–0.343], which was nearly twice as large as the h^2 estimate.

In addition, we found a substantial negative maternal correlation between body size at isolation and $PPSR_{max}$ ($r_M = -0.418$; [CI: −0.725; −0.096]; Fig. 1), meaning that individuals provisioned by their mothers with a smaller size are more prone to feed on relatively larger prey in later developmental stages. No maternal correlation between body condition at isolation and $PPSR_{max}$ was found ($r_M = 0.107$; [CI: −0.261, 0.564]; Fig. 1). Also, we did not find any significant genetic correlation between $PPSR_{max}$ and body size or between $PPSR_{max}$ and body condition at isolation ($r_A = -0.129$ [CI: −0.498; 0.413]) and $r_A = 0.089$ [CI: −0.417; 0.462], respectively; Fig. 1).

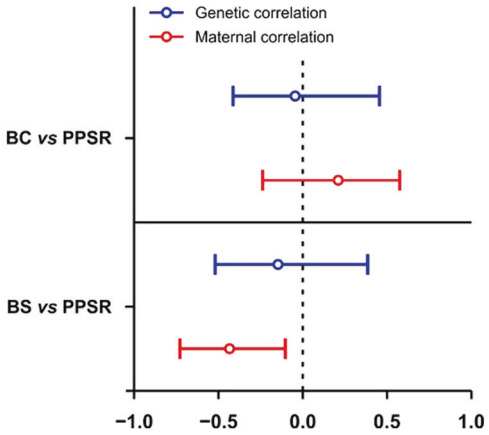

Fig. 1 Genetic (r_A) and maternal correlations (r_M) among the traits measured in this study. White dots represent the posterior mode for the estimates measured and the intervals represent Bayesian credible intervals (95%). Significant estimates are those that do not overlap zero (dashed line). BS body size at isolation, BC body condition at isolation, PPSR prey-to-predator size ratio.

Discussion

In this study, we found that additive and non-additive genetic plus maternal effects contributed to variation in PPSR in the wolf spider *L. fasciiventris*.

We also documented that individuals in better condition before the trial attacked and subdued relatively larger prey (higher $PPSR_{max}$). Moreover, we show that individuals from maternal families giving birth to larger offspring tended to feed on smaller prey ca. 9 months ahead in their ontogeny.

Relative body size differences between predators and prey are often measured through predator–prey body mass ratios. However, several studies also use structural body size differences between predators and prey, particularly in systems similar to ours (García et al. 2018; Grinsted et al. 2020). Indeed, in spiders, body condition accounts for a large proportion of body mass in the form of storage in the abdomen (e.g., Moya-Laraño et al. 2008). Thus, structural body size differences provide better estimates of the probability that spiders subdue the prey. Note, however, that differences among individuals in $PPSR_{max}$ can also be related to differences in risk taking decisions or in costs such as handling time (Woodward and Warren 2007).

Some studies have measured the preference of predators for prey of different sizes (Shultz et al. 2004; Matlock

2005). Preference is clearly an important trait defining dietary breadths (Poore and Hill 2006) and it is therefore ecologically relevant (Singer 1986; Jiang and Morin 2005; Boll and Leal-Zanchet 2016). However, size is a continuous variable, hence choice experiments (which generally use two prey items only) will necessarily leave out much of the variation in prey size. In addition, prey acceptance may be more ecologically realistic than preference, as predators often encounter prey sequentially (Nentwig and Wissel 1986). Therefore, maximum prey size acceptance is probably a relevant trait for this predator, as for many others. For example, a previous study showed that differences in foraging efficiency of two instars of the dragonfly *Aeshna juncea* were more clearly perceived when this trait was measured in trials involving the larger prey size (Hirvonen and Ranta 1996).

The most common measure of PPSR is based on dietary analyses of organisms directly collected from their environment like gut contents (Agashe and Bolnick 2010; Costa-Pereira et al. 2018). These measures correspond to the actual composition of prey eaten, but they can be strongly affected by the relative prevalence of different prey types in the environment (Costa-Pereira et al. 2018). It has been argued that it is this context-dependence that accounts for the discrepancy between model assumptions of a constant PPSR and data, which show variable within-species PPSR (Tsai et al. 2016). Here, we provide a measurement that is independent of the environmental context and show that variation is still present.

One of the compelling advantages of our measure of PPSR is that we were able to estimate the variance components responsible for individual variation in this trait. Indeed, we show that such variation is due to additive and dominance or maternal effects. Therefore, such variation is not simply a by-product of environmental conditions and needs to be accounted for in studies addressing the ecology and evolution of body size in predators (Nakazawa 2017). In our design, we cannot disentangle the relative contribution of dominance and maternal effects to the dam variance. Previous studies exploring the importance of dominance in several traits have concluded that it has a proportionally higher impact on trait variation when additive genetic variance is eroded by natural selection, most commonly in fitness related traits (Crnokrak and Roff 1995; Merilä et al. 2001). Given the low values of narrow sense heritability observed here, dominance (along with maternal effects) may be an important determinant of trait variation (Crnokrak and Roff 1995). Indeed, studies with laboratory populations have shown that dominance can account for as much as 38% of the total phenotypic variation (Wolak and Keller 2014). However, a recent study focusing on morphological and behavioural traits has shown that dominance variance is negligible (or difficult to detect) in wild passerine populations (Class and Brommer 2020). In this same study, based on simulation data, it was observed that neglecting dominance variance can indeed inflate the estimates of additive genetic variance and heritability. However, inflation of the estimates can be kept relatively small if maternal variance is also controlled for. Nonetheless, the data come from a particular case-study and thus one single value of environmental variance, which can greatly differ across species, populations, and traits. Remarkably, the results of these same simulations found that dominance and environmental effects can be strongly confounded in animal models, which suggests that there is still plenty of room for, at least, moderate dominance effects to operate in wild populations. Future work should implement other breeding designs, such as the production of maternal half-sib families to properly estimate dominance in this and other systems. In addition, the traits we are considering are probably polygenic, hence epistasis may significantly contribute to trait variance. However, the complex designs needed to estimate this variance component are beyond the capacity of the current study.

Variation in $PPSR_{max}$, measured ca. 9 months after spiderlings were separated from their mothers, was still affected by dominance or maternal variance. This suggests that either dominance or long-lasting maternal mechanisms, such as hormones and/or other maternally inherited factors (Groothuis and Schwabl 2008), contribute to variation in this trait. Indeed, some studies show that maternal effects can still be found later in life, although they generally wane throughout the ontogeny of organisms (Bernardo 1996; Heath et al. 1999; Lindholm et al. 2006; Wilson and Réale 2006). We found that the relative contribution of maternal plus dominance variance ($d^2 + 4c^2$) was small (0.056) and of similar magnitude than that of the heritability (0.069). Overall, the maximum value of the broad-sense heritability that we estimated was 0.125. This implies that evolutionary responses of this trait may be rather small, suggesting that $PPSR_{max}$ has been under strong selection in the past. A very high environmental variance in $PPSR_{max}$ can still impact predator–prey dynamics, due to predator selection pressure upon prey that differ in size. In addition, part of this environmental variation may be explained by other variables, such as individual state. Indeed, here we found that individuals in better body condition tended to display a higher $PPSR_{max}$, thus subduing relatively larger prey. Previous studies showed that wolf spiders with more energy reserves tend to spend less time and effort hunting (e.g., Moya-Larano et al. 1998; Moya-Larano 2002), suggesting that spiders in better condition are less motivated to hunt. Our results cannot be explained by this motivational state hypothesis. Possibly, in our case, relatively heavier spiders have higher chances of subduing larger crickets, as spiders jump on top of crickets to do so. Alternatively, spiders in

better condition are willing to spend more energy to subdue larger prey.

Surprisingly, the food treatment did not affect $PPSR_{max}$, although spiders in the richer food treatment tended to be of superior body size and body condition (Figs. S1 and S2). Differences in other traits underlying body condition, such as differences in assimilation efficiency, could be responsible for body condition being linked to $PPSR_{max}$, instead of food treatment.

We also found a strong maternal correlation between traits. Indeed, females that provisioned offspring in such a way that these were born with bigger sizes, had also offspring that displayed a lower $PPSR_{max}$ ca. 9 months later in life. Individuals born larger may be less willing to take unnecessary risks later in life, because in the wild they would have enjoyed a relatively milder environment through their ontogeny. These spiderlings, born slightly larger, may be less willing to attack relatively larger prey later in life because while capturing larger prey is more energetically rewarding, it may come with the cost of longer handling time (which includes pursuit and subduing time, ingestion time and digestion) and the possibility of injuries inflicted by the prey (Griffiths 1980), as it is the case for spiders preying on crickets (Gnatzy and Otto 1996).

Alternatively, this maternal correlation may represent a particular case of a "silver spoon effect", defined as an increased fitness throughout the lifetime of an organism due to being better provisioned early in life (Grafen 1988; Cockburn 1991). To disentangle between these hypotheses, we would need to measure the fitness of individuals that were born bigger and exhibit a lower $PPSR_{max}$ and that of smaller individuals with higher $PPSR_{max}$, and observe fitness differences between the two. Finally, there is the possibility that at least part of the variance explained by this correlation is due to pleiotropic dominance effects (Keightley and Kacser 1987), which we cannot distinguish from maternal correlations in our design.

Theory predicts that genetic architecture, including genetic correlations, is a key to understand the impact of trait variation on coexistence (Schreiber et al. 2018; Patel et al. 2018). Moreover, genetic correlations among traits can accelerate or hinder evolutionary responses (Chevin 2013). When evolutionary processes occur within similar timeframes as ecological processes, such correlations can affect eco-evolutionary dynamics and system stability (Patel et al. 2018). Under this rationale, the maternal correlations described in this study could also affect predator–prey dynamics.

The empirical data presented in this work contributes to the understanding of individual dietary specialization, i.e., inter-individual variation in resource use (Bolnick et al. 2002, 2003; Araújo et al. 2011). Specifically, the dam component of $PPSR_{max}$ explains some proportion of the variation in individual niche specialization (Bolnick et al. 2003). Maintenance of inter-individual diet variation allows populations to maintain stability when faced with competition and predation, but it also exerts different forms of selection on prey species (reviewed in Bolnick et al. 2003). Still, there is little evidence for how this specialization affects community dynamics (Araújo et al. 2011) and further studies including the sources of variation on individual specialization are needed.

Our results thus highlight that accounting for individual variation in PPSR may help unravel the evolutionary factors shaping this trait. Such variation can, in turn, impact ecological interactions. In addition, by diversifying prey selection, individual variation in PPSR may allow for the maintenance of variation in prey sizes, as it will spread the predation pressure across prey differing in body size (Ye et al. 2013). Therefore, individual variation in PPSR stands at the intersection between the ecological and evolutionary impacts of predator–prey interactions, playing an important role as a key predictor of food web persistence and its associated ecosystem processes, and less so of evolutionary trajectories, at least as a source of direct responses.

Data availability

Data are archived at https://datadryad.org/stash/share/wXwDhJGCOLagIKnEusPg8Si-z4Lxa3LTGZR4u70q08E.

Acknowledgements We thank D. Mayntz and K. Jensen for providing the recipes for the media used to culturing nitrogen-enriched *Drosophila*. This study was supported by a PhD grant (PD/BD/106059/2015) attributed to JFH by the Portuguese Science and Technology foundation (FCT), by the grant P12-RMN-1521 from the Andalusian government and by the grant CGL2015-66192-R from the Spanish Ministry of Economy and Competitiveness, both partially funded by the European Regional Development Found, both attributed to Jordi Moya-Laraño and to the FPU scholarship (FPU13/04933) from the Spanish Ministerio de Educacion, Cultura y Deporte to Dolores Ruiz-Lupión.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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