1 **Metabolic rate, context-dependent selection, and the colonisation-competition trade-off**

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Abstract

Metabolism sets the pace-of-life, co-varying with survival, growth and reproduction. Metabolic rates should therefore be under strong selection and, if heritable, become less variable over time. Yet intraspecific variation in metabolic rates is ubiquitous, even after accounting for body mass and temperature. Theory predicts variable selection maintains trait variation but field estimates of how selection on metabolism varies are rare. We use a model marine invertebrate to estimate selection on metabolic rates in the wild under different competitive environments. Fitness landscapes varied among environments separated by a few centimetres: interspecific competition selected for higher metabolism, and a faster pace-of-life, relative to competition-free environments. Populations experience a mosaic of competitive regimes; we find metabolism mediates a competition-colonisation trade-off across these regimes. Spatial heterogeneity and the variable selection on metabolic rates that it generates is likely to maintain variation in metabolic rate, despite strong selection in any single environment.

Introduction

Metabolic rate reflects the pace at which organisms use, transform and expend energy to sustain life. Metabolism covaries with components of fitness such as survival, growth, longevity, and reproduction (Glazier 2005; Auer *et al.* 2018; Pettersen *et al.* 2018). Over the last century, metabolic theory has explored the origin and maintenance of metabolic scaling relationships from single-celled organisms to communities (Rubner 1908; Kleiber 1932; Hemmingsen 1960; Kooijman 2000; Gillooly *et al.* 2001; Brown *et al.* 2004; White *et al.* 2008; Glazier 2010). Historically, studies of metabolism have emphasized mechanistic models of physical constraints to explain variation in metabolic rate, but recent evidence suggests that multivariate selection shapes much of the among-species variation in metabolic rate across macroevolutionary scales (White *et al.* 2019). The action of selection across broad, evolutionary timescales may explain macroevolutionary patterns in metabolic scaling, but the nature of selection on metabolism at shorter time scales is much more ambiguous. Within species, considerable variation in metabolism persists - basal and standard metabolic rates vary up to three-fold within species, even after holding mass and temperature constant (Burton *et al.* 2011; Konarzewski & Ksiazek 2013; White & Kearney 2013). Theories focused on mechanism struggle to account for this variation; so we look to evolutionary theory to understand the context-dependent nature of selection acting on metabolic rates (Pettersen *et al.* 2018).

A classic tenant of evolutionary theory is that selection depletes genetic and (or) phenotypic variation, such that heritable traits can become less genetically variable over time (Arnold *et al.* 2001; Merilä *et al.* 2001; Wilson *et al.* 2006). Metabolic rates are both heritable and subject to selection (Garland & Carter 1994; Pettersen *et al.* 2018; White *et al.* 2019), yet vary within populations and among individuals of the same species. Natural environments can fluctuate across spatial and temporal scales, and so too can selection (Bell 2010;

Charmantier *et al.* 2014; Lange *et al.* 2016). Fluctuating selection can maintain trait variation (McDonald & Ayala 1974; Calsbeek *et al.* 2010; Bertram & Masel 2019), and may explain why we observe intraspecific variation in metabolic rates (Sasaki & Ellner 1997). A continually shifting environment, where different phenotypes are favoured under different conditions should maintain phenotypic variation. Accordingly, studies generally show that selection can vary substantially in space and over time, even over very small scales (metres and days (Grant & Grant 2002; Svensson & Sinervo 2004; Siepielski *et al.* 2009; Whitman & Agrawal 2009; Bell 2010; Burton *et al.* 2011)). Variation in selection regimes could maintain intraspecific genetic, and therefore phenotypic variation, in metabolic rate, yet formal estimates of selection on metabolism across natural field conditions are rare.

Competition is a ubiquitous and powerful agent of selection in nature. Both intra- and inter-specific competition are important eco-evolutionary processes, affecting individual access to resources that can ultimately drive evolutionary change (Fussmann *et al.* 2007). Variation in the form and intensity of competition can arise due to differences in densities of individuals or the relative abundance of shared resources, creating competition-dependent selection regimes across time and space. Even within a single population, individuals can experience very different levels of intra- and interspecific competition, with profound consequences for fitness (Wissinger 1989; Stratton 1995; Einum *et al.* 2008; O'Neal & Juliano 2013). Hence, the presence, strength, and form of competition is likely to vary across environments, to produce spatially-explicit selection regimes.

Competition is particularly likely to alter selection on metabolic rates. Competition alters the supply of, and access to, resources to influence metabolic rates and the pace of the life history in unexpected ways (Marshall 2005; DeLong *et al.* 2014; Bassar *et al.* 2016; Ghedini *et al.* 2018). High population densities generally reduce per capita resources, hence higher metabolic rates may be more competitive if they are better able to acquire and

84 assimilate resources so that they may reach a size refuge or outcompete conspecifics (Mueller
85 & Diamond 2001; Burton *et al.* 2011; Nilsson & Nilsson 2016; Auer *et al.* 2018).
86 Alternatively, lower metabolic phenotypes with relatively low resource requirements may be
87 beneficial for preserving energy reserves and resisting starvation (Ghedini *et al.* 2017). The
88 competition environment should thus interact with metabolic rates, and perhaps alter the costs
89 and benefits of a particular metabolic phenotype (Swanson *et al.* 2017). It seems plausible,
90 therefore, that variation in competitive environments may mediate selection on metabolic
91 rates and maintain intraspecific variation more generally.

92 Here we measure how competition alters selection on metabolic rates in the field in
93 the marine bryozoan *Bugula neritina*. *Bugula neritina* produces free-swimming offspring that
94 typically disperse centimetres to hundreds of metres, and can experience a range of
95 competition environments, from newly-disturbed free space to densely packed communities
96 (Marshall & Keough 2009). We leverage the tractability of this system to experimentally
97 manipulate the competitive environment of individuals of known metabolic phenotypes and
98 monitor their survival, fertility, and reproduction in the field. We then formally estimate a
99 series of parameters related to selection: i) the opportunity for selection (I) across competition
100 levels; ii) linear (β) and nonlinear (γ) selection gradients; and iii) the intensity of selection
101 (V). Finally, we iv) measure the covariance between metabolic rates and key life-history traits
102 – growth rate, longevity, and age at onset of reproduction. Combined, measures of form,
103 opportunity, and intensity of selection allow us to quantify selection across environments that
104 are needed to reveal the complex interplay of phenotype, fitness, and the pace-of-life driving
105 natural variation in metabolic rates (Brodie *et al.* 1995). We find that metabolic rates mediate
106 a trade-off between colonisation and competition – high-metabolism individuals were better
107 able to withstand intense competition but low-metabolism individuals lived for longer and are
108 likely to have higher fitness under competition-free conditions.

Materials and Methods

Study species and field deployment

Bugula neritina (named by genus hereafter) is a filter-feeding, arborescent bryozoan that inhabits a range of shallow subtidal surfaces, including boat hulls and pier pylons (Chang & Marshall 2016). *Bugula* colonies brood fertilised eggs in visible reproductive structures (ovicells) for approximately one week (Woollacott & Zimmer 1975). Light at dawn induces spawning of free-swimming, lecithotrophic larvae that spend a short time in the plankton, typically settling within minutes to hours (Marshall & Keough 2003). Settlers then undergo metamorphosis over approximately three days to develop, and form the ancestral zooid (Burgess & Marshall 2011). The ancestrula then begins feeding and grows into a colony via asexual budding of zooids, and reaches sexual maturity approximately 3-8 weeks post-settlement.

All field collections and monitoring were conducted at Royal Brighton Yacht Club, Victoria, Australia (-37.909, 144.986) during March to September 2015. Sexually mature colonies were transported back to the laboratory and induced to spawn following standard procedures (Pettersen *et al.* 2016). After measuring the traits of interest (see below), each individual settler, attached to a small piece of acetate, was glued onto a labelled PVC plate (55 x 55 x 3mm; our unit of replication) assigned to one of three treatments: no competition ("*nocomp*"), intraspecific competition ("*intra*") and interspecific competition ("*inter*"; Figure S1).

Individuals in *nocomp* were glued onto a blank plate, and biofouling kept clear throughout the duration of the study. The *intra* treatment represents an environment of intense intraspecific competition– commonly experienced by *Bugula* in the field (Allen *et al.* 2008). The focal individual was glued onto a plate among eight individual *Bugula* settlers of

the same age. Throughout monitoring, plates were cleared of any exogenous settlers of any species. The *inter* environment mimicked settlement into a pre-established, subtidal community. To obtain these communities, we left the plates in the field for 12 weeks prior to starting our experiment to allow natural fouling onto these plates. Upon return to the laboratory, a small area (15 x 15mm) in one corner of the plate was cleared of any organisms, mimicking a physical disturbance, and the focal individual glued into this section. Once all individuals were introduced into their treatments, plates were maintained overnight in tanks with unfiltered seawater before being deployed the next morning onto backing panels in the subtidal as per Pettersen *et al.* (2016). Overall, our experiment included 360 individuals of known phenotype (120 per competition treatment) that we deployed across 10 backing panels in the field.

Trait and fitness measurements

a. Traits of interest: larval mass and metabolic rates

We measured selection on three traits: larval mass, and metabolic rate at two stages during early ontogeny: two hours post-settlement and 24 hours post-settlement (hereafter referred to as metabolic rate early; MR_E and metabolic rate late; MR_L). MR_E and MR_L occur during crucial stages during the life history and have previously been shown to be under differing selection in this species (Pettersen *et al.* 2016). Larval mass is a key life-history trait and a well-known predictor of performance, however the offspring size-performance relationship is often context-dependent (Marshall *et al.* 2018). We measured the diameter of newly spawned larvae and calculated larval mass using previously developed protocols (Pettersen *et al.* 2015). Metabolic rate was measured for individuals using the common proxy, rate of oxygen consumption or $\dot{V}O_2$, developed from previous methods (Pettersen *et al.* 2015) and the package "LoLinR" to objectively and reproducibly estimate monotonic $\dot{V}O_2$ from our

readings (Olito *et al.* 2017). All analyses were conducted in R version 3.6.1 (R Development Core Team 2016).

b. Fitness measures: viability, fertility, and fecundity

We used survival to reproduction (viability), ability to reproduce (fertility), and cumulative reproductive output during the first five months of the life history (fecundity) as our measures of fitness. Survival, and the presence of reproductive structures (ovicells) indicating ability to reproduce, were recorded once per week – individuals were considered to be alive if they were still attached to their settlement plate and >10% of the colony contained feeding zooids. Viability and fertility were treated as a binary fitness measures - individuals that survived to the average reproductive age (viability), and those that reproduced (fertility) were assigned "1", while individuals that died before reproductive age or the onset of reproduction were assigned "0", respectively. Reproductive output (fecundity) was measured as the cumulative number of ovicells throughout the duration of the study, which were counted using a dissecting microscope (x10) once per week, from the onset of reproduction at approximately six weeks post-outplant. In a previous study of this population, *Bugula* survived up to nine months, and reproductive output during the first five months of the life history reliably predicted lifetime reproductive output (cumulative reproductive output 120 days post-outplant explained 94 % of variance in lifetime reproductive output for this same population; Pettersen *et al*. 2016). In addition, we measured several life-history traits related to fitness over the duration of the study: growth (number of colony bifurcations per week; Keough and Chernoff (1987), longevity (number of days >10% colony remained alive), and age at onset of reproduction (days) up until five months post-outplant.

Estimates of selection on larval mass and metabolic rates

We can estimate parameters derived in evolutionary theory in order to quantify competition-dependent selection on metabolic rates and provide a relative scope for evolutionary change among environments. First, the opportunity for selection (*I*), describes the amount of variation in relative fitness, and determines the maximum potential strength of selection that could occur in a given environment (Schluter 1988). Second, the form of selection acting on any trait, or combination of traits, and whether it changes across environments, can be quantified using formal selection analysis (Lande and Arnold 1983). Finally, the intensity of selection (*V)* provides a measure of the overall strength of selection acting on all combinations of traits in each environment irrespective of the particular form (Schluter 1988).

a. Estimating and testing for differences in the opportunity for selection

For each competition environment, we calculated the opportunity for selection (*I*) across environments, $I = \sigma_w^2/\overline{W}$, where *w* is relative fitness and $\overline{W}$ is the population mean absolute fitness (Jones 2009). We could not assess opportunity for selection in binary (viability and fertility) data, hence only fecundity fitness data was tested. Due to over-dispersion in our reproductive output count data, we calculated nonparametric bootstrap values using BC*a* intervals within the R package "boot" (Davison & Hinkley 1997; Moorad & Wade 2013; Canty & Ripley 2019).

b. Characterising selection within and among competition environments

We used a classic multiple regression approach to formally estimate the form of selection on our three traits of interest (larval mass, metabolic rate early; MR_E, and metabolic rate late; MR_L), for our fitness measures (Lande & Arnold 1983). Using a multiple regression framework allows for standardised, and comparable estimates of linear (β) and nonlinear (γ) selection gradients. To investigate selection further, we split our data into three separate

analyses: viability selection (survival to reproduction), fertility selection (ability to reproduce), and fecundity selection (cumulative reproductive output). For viability and fertility selection, individuals that survived/did not survive to reproduce (viability selection), and reproduced/did not reproduce (reproductive selection) were assigned "1" and "0" respectively, and models were fit using logistic regression in a generalised linear model (Janzen & Stern 1998). Viability and fertility selection coefficients were transformed into linear estimates as per Janzen and Stern (1998). Our reproductive output count data for fecundity selection was over-dispersed and best fit with generalised linear models using a negative binomial distribution (Dobson *et al.* 2008). Since only 14 out of 120 individuals in the interspecific competition environment reproduced, we did not have sufficient power to calculate nonlinear coefficients of fecundity selection, hence only linear estimates were calculated in this environment. We first converted our predictor variables of larval mass, MR_E and MR_L into units of standard deviation (mean of 0 and standard deviation of 1), and mean-centred survival and reproductive output by dividing each absolute measure by the mean absolute fitness (Lande & Arnold 1983). Both predictor and response variables were also standardised by 'experimental panel' – while we found no significant interactions between experimental panel and environment, or with each of our predictor variables, we wanted to account for spatial variation among panels.

Using a series of nested models, we tested whether there were differences in linear selection, non-linear selection, or both, between competition environments via a sequential model fitting approach (Draper and John 1988; Chenoweth and Blows 2005). Linear selection on fertility ($\chi^2 = 70.064$, df = 6, $p < 0.0001$) and linear ($\chi^2 = 188.504$, df = 6, $p < 0.0001$) and nonlinear ($\chi^2 = 27.820$, df = 12, $p < 0.01$) fecundity selection (*nocomp* and *intra* comparison only) differed among environments. For viability selection, all forms of selection (except for correlational viability selection) showed significant interactions with environment (see

Results). When selection x environment interactions were significant, fitness data were standardised within environment, and selection coefficients estimated for each competition treatment separately. Quadratic regression coefficients and their standard errors were doubled before being reported as selection gradients (Stinchcombe *et al.* 2008).

c. Estimating the intensity of selection

Selection intensity (I) is a measure of the overall strength of selection as estimated by the variation in predicted fitness values, and is a function of both selection on, and the distribution of, phenotypes in the population (Schluter 1988). In our study, calculating I allows for direct comparison of differences in overall selection on metabolic rate and larval mass between levels of competition, irrespective of what the form of selection is in each environment. We calculated the expected fitness (survival to reproduction and reproductive output) for each individual using the full regression model, incorporating linear, quadratic and correlational regression coefficients within each environment for viability and fecundity selection separately (Schluter 1988). $I_{\text{viability}}$ and $I_{\text{fecundity}}$ were then calculated as the squared coefficient of variance in the expected fitness values (I = CV[expected fitness]^2). We produced nonparametric bootstrap values for our estimates as described previously (Davison & Hinkley 1997; Moorad & Wade 2013; R Development Core Team 2016).

Competition-dependent covariance between life-history and metabolic traits

Metabolic rates are linked with key life-history traits that together mediate the pace-of-life (Careau *et al.* 2010; Pettersen *et al.* 2016; Auer *et al.* 2018; Niemelä & Dingemanse 2018; Mathot *et al.* 2019). In order to understand how selection on metabolic rates might be mediated through their effects on the pace-of-life, we measured three key life-history traits in individuals of known metabolic phenotype over five months post-outplant.

252 *a. Growth*

253 The relationship between larval mass, MR_E and MR_L on the growth of colonies (number of
254 bifurcations) was estimated using linear-mixed effects regressions in a repeated measures
255 framework, to determine individual growth rate in the field over time ("lme4" package, Bates
256 *et al*. (2015)). Again, we detected significant three-way interactions with environment (χ^2 =
257 15.916, df = 18, p <0.001), hence each competition level was analysed separately. We used a
258 repeated measures ANCOVA to test for significance of the random effect of experimental
259 panel and its interactions with fixed factors of larval mass, MR_E and MR_L across the repeated
260 measure of week. We found a significant main effect of experimental panel for *nocomp* and
261 *inter*, which was retained in the final model, but no support for fitting a random-slopes model
262 (no significant interactions between fixed factors and experimental panel were found).

263 *b. Longevity*

264 Longevity showed an overall bivariate response: while mortality rates were high early in the
265 life history, individuals that survived the first four months post-outplant were often alive by
266 the end of the sampling period. Hence, lifespan data was fit with a logistic regression:
267 individuals that survived less than or more than 140 days were assigned "0" and "1",
268 respectively. The main effects of environment, larval mass, MR_E and MR_L on longevity were
269 tested using a generalised, linear-mixed effects model ("lme4"; Bates *et al*. (2015)). Since
270 environment and its interactions were non-significant, we pooled data across all
271 environments. All interactions with experimental panel were non-significant and were
272 removed from the final model.

273 *c. Age at onset of reproduction*

Age at onset of reproduction was also fit with using logistic regression as per Pettersen *et al.* (2016). Individuals that developed ovicells < 60 days post-outplant were considered to have an early onset of reproduction and were assigned "1", while individuals noted to develop ovicells after this time (>60) were denoted "0". We used generalised, linear-mixed effects logistic regression as described previously. We found no significant effect of environment, or its interactions, thus data was pooled across environments. We also found no main effect of experimental panel or any of its interactions, so it was removed from the final model.

Results

1. *Variation in reproductive output and the opportunity for selection*

Competition imposed increasingly negative fitness consequences along a stress gradient, from benign conditions under *nocomp* to highly stressful conditions under *inter*. Average cumulative reproductive output for all individuals (irrespective of phenotype) was highest under no competition; *nocomp* (mean $n_{ovicells}$ ± SE: 361 ± 44), intermediate under intraspecific competition; *intra* (mean $n_{ovicells}$ ± SE: 136 ± 28), and lowest under interspecific competition; *inter* (mean $n_{ovicells}$ ±SE: 26 ± 10). The opportunity for selection (*I*), also increased with competition stress (*I*(*nocomp*) = 0.645, 95% CI = 0.576 – 0.720; *I*(*intra*) = 0.840, 95% CI = 0.766 – 0.914; *I*(*inter*) = 0.949, 95% CI = 0.909 – 0.976) and was 1.5 times smaller in the absence of competition relative to interspecific competition.

2. *Estimates of competition-dependent selection gradients*

a. *Viability selection*

Our selection analysis revealed significant differences in linear (χ^2 = 20.575, df = 6, p = 0.002) and nonlinear (χ^2 = 44.075, df = 12, $p < 0.0001$) viability selection among environments. Directional selection was strongest under *nocomp*, with fitness highest for smaller individuals with high MR_L (Table 1). Under competition, linear gradients were much weaker and non-significant, although with a reversal in sign for the *intra* environment. We did, however, find evidence for significant correlational selection in all environments. Across all environments, we found negative correlational selection on MR_E and MR_L– individuals with either high MR_E-low MR_L or vice versa were more likely to survive to reproduce (all environments show the same correlational selection coefficients since we found no significant differences in correlational viability selection – see Methods). We also found significant concave selection, but only in the *intra* environment (Figure 1).

305 *b. Fertility selection*

306 Competition affected the probability of reproducing over the first five months of the life
307 history – individuals under *nocomp* and *intra* were more likely to reproduce than individuals
308 under *inter* ($\chi^2 = 72.389$, df = 2, $p < 0.0001$). We found significant differences in linear ($\chi^2 =$
309 70.064, df = 8, $p < 0.0001$) but not nonlinear ($\chi^2 = 12.415$, df = 12, $p = 0.413$) fertility
310 selection across environments. Under *nocomp*, linear fertility selection tended to favour
311 individuals with low metabolic rates (although not significantly), while under *intra* and *inter*,
312 selection favoured higher MR_E and MR_L respectively ($\beta_{MRE} \pm SE = 0.144 \pm 0.062$; $\beta_{MRL} \pm SE$
313 $= 0.120 \pm 0.046$). Across *nocomp* and *intra*, we found evidence for negative quadratic
314 selection on larval mass only ($\gamma_{Mass,Mass} \pm SE = 0.212 \pm 0.058$). All linear and nonlinear fertility
315 selection coefficients are provided in Table S1.

316 *c. Fecundity selection*

317 We found both linear (all environments; $\chi^2 = 188.504$, df = 6, $p < 0.0001$) and nonlinear
318 (*nocomp* and *intra* only; $\chi^2 = 27.820$, df = 12, $p < 0.01$) fecundity selection varied with
319 environment. Under *nocomp*, intermediate MR_E showed highest reproductive output (Table
320 2). Under *intra*, we found evidence for directional selection for high MR_E, and negative
321 quadratic selection on larval mass, MR_E and MR_L, where individuals with high MR_E and
322 intermediate MR_L were favoured. We found negative correlational selection under *intra* –
323 individuals with either high MR_E/low MR_L or vice versa showed highest fecundity. Due to
324 directional selection for high metabolic phenotypes under *inter*, reproductive output was
325 greatest for individuals with both high MR_E and MR_L and lowest for individuals with low
326 metabolic rates (Figure 2).

327 *3. Estimating the intensity of selection*

The intensity of viability selection ($V_{viability}$) was significantly higher in the absence of competition - $V_{viability}$ was over eight orders of magnitude higher under *nocomp* ($V_{viability}$ = 0.261, CI = 0.125 – 0.360) than under either *intra*: $V_{viability}$ = 0.037, CI = 0.017 – 0.090) and *inter* ($V_{viability}$ = 0.031, CI = 0.012 – 0.078). For individuals that survived to reproductive age, we did not detect any significant differences in the intensity of either fertility selection ($V_{fertility}$) or intensity of fecundity selection ($V_{fecundity}$) across environments.

4. *Competition-dependent covariance between larval mass, metabolic rates and life-history traits*

Growth

MR_E and MR_L had significant, but context-dependent effects on growth. Colonies consistently increased in size throughout the first 20 weeks post-outplant, but growth was highest under *nocomp* and lowest under *inter*. Overall, higher metabolic rates were associated with faster growth in both competition environments – and this was most evident under *inter* (Figure 3). Interestingly in *nocomp*, growth was slowest for individuals with both high MR_E and low MR_L, despite a strong positive interaction between the two metabolic rates (Table S2; Figure 3).

Longevity

We found significant main effects of environment, MR_E and MR_L on longevity – overall, colonies under *inter* were shorter lived than those in the *intra* or *nocomp* environments (mean ± SE; *nocomp*: 132 ± 2.89 days, *intra*: 129 ± 3.11 days, *inter*: 101 ± 4.69 days). Across all environments however, individuals with lower MR_E and lower MR_L lived longer (Figure 4). This relationship was consistent among experimental panels and no significant interactions among fixed or random effects were found (Table S3).

351 *Age at onset of reproduction*

352 The onset of reproduction occurred between 28 – 126 days after being deployed into the field
353 (mean ± SE = 65.29 ± 1.73 days). Individuals with higher MR_L began reproducing sooner -
354 we found a significant positive relationship between MR_L and the probability of early onset
355 of reproduction, and this was consistent across all environments (Table S4).

Discussion

Competition changed the strength and form of selection on metabolic rates in the field. Survival, fertility, and fecundity were lowest under interspecific competition and highest in the absence of competition. Fertility and fecundity (but not viability) selection on metabolic rates changed along a stress gradient – when competition was absent, weak quadratic selection favoured intermediate phenotypes, whereas strong directional and quadratic selection favoured higher metabolic rates under competition. The natural environment for *Bugula neritina* is a mosaic of competitor-free, intra- and inter-specific competition, with individuals from the same brood potentially experiencing very different environments (Chang & Marshall 2016). Our competition treatments reflect the scale of this variability – individual settlers were separated by only a few centimetres, yet experienced distinct selection regimes. We find evidence that metabolism mediates a trade-off between competition and colonisation via the pace-of-life – high metabolism individuals withstood competition, but low metabolism individuals are likely to live longer in newly colonised, competitor-free environments. Though selection on metabolic rates was strong, its context-dependent nature will likely hamper its capacity to purge variance in metabolism.

Competition tended to favour higher metabolic rates, perhaps because they also covaried with faster life histories. Under interspecific competition, individuals with higher metabolic rates were more likely to survive, more likely to reproduce, and were more fecund upon the onset of reproduction. Under interspecific competition, higher metabolism covaried with a faster pace-of-life (i.e. faster growth, shorter lifespan (MR_E) and earlier onset of reproduction (MR_L)). Higher metabolic rates are often associated with a faster pace-of-life in stressful environments. For example, individuals with higher metabolic rates grow faster and display more aggression – exerting dominance to secure access to food, mates and territory (Reid *et al.* 2011; Le Galliard *et al.* 2013; Auer *et al.* 2018). In *Bugula*, higher metabolic rates

may increase feeding rates and energy acquisition, allowing individuals to emerge from the canopy of other competing individuals or species sooner, to reach resources such as food and oxygen. Yet, higher metabolic rates also come with the cost of a shorter lifespan, as shown in our study, and in others (Bochdansky *et al.* 2005), possibly due to oxidative stress (Dowling & Simmons 2009). Given the intensity of selection under competition, where life expectancies are lower, the selective advantage of faster growth rates and earlier reproduction is likely to compensate for the reduced longevity associated with higher metabolic rates.

Given that we observe strong directional selection for higher metabolic rates in most environments, why are lower metabolic rates not purged from the population? We find cryptic benefits of low metabolic phenotypes (particularly for MR_E) in the absence of competition – for example, low metabolism individuals had a higher probability of living for longer than high metabolism individuals. Because we ended our experiments before low-metabolism individuals in competition-free environments had perished, we underestimated their fitness – these individuals would have continued to reproduce long after high metabolism individuals had died. As such, low metabolism individuals likely have a fitness advantage over high metabolism individuals when competition is absent. Metabolism therefore appears to mediate a competition-colonisation trade-off via pace-of-life effects in our system. High metabolism individuals grow more and reproduce sooner before dying earlier - a phenotype that confers higher fitness when competition is intense. Meanwhile, low metabolism individuals grow slowly, but live for longer, suffering reduced fitness when competition is strong, but gaining higher fitness when they colonise competition-free environments. Such trade-offs are known to maintain variation (Kisdi & Geritz 2003). Importantly, this is not the only mechanism by which variation in metabolic rate will be maintained – we also found ubiquitous negative correlational selection on different metabolic rates, particularly in high competition environments. Negative correlational selection will act

to increase negative covariance between metabolic traits, hampering the capacity of strong positive directional selection to increase trait values of both simultaneously.

In our system, competition-free habitat is rare and ephemeral in nature – hence, competitive environments should impose strong selection on metabolic rates. However, fitness payoffs for individuals with lower metabolic rates colonising rare, competition-free environments are considerable (they have much higher fecundities). While rare, massive reproductive payoffs in competition-free habitats might therefore be sufficient to maintain low metabolic rate phenotypes, particularly since selection was most intense in competition-free environments. Thus selection may be unable to purge low metabolic rates if these individuals are occasionally able to invade free space (Courbaud *et al.* 2012). Ultimately, countervailing selection pressures acting on survival and reproduction, and the considerable fitness benefits of lower metabolic rates under competition-free environments, however rare, may contribute to maintaining variance in metabolic rates (Wadgymar *et al.* 2017).

We found negative correlational viability selection opposed a positive, albeit weak, phenotypic correlation between MR_E and MR_L. If phenotypic correlations are representative of underlying positive genetic correlations between MR_E and MR_L, then genetic constraints may limit the efficacy of this negative correlational selection (Blows 2007; Pettersen *et al.* 2016). Our findings highlight the importance of measuring multiple metabolic rates – estimates of selection on either MR_E or MR_L in isolation fail to account for any underlying covariance between correlated characters that may override the effects of univariate selection (Hansen *et al.* 2019). We show that metabolic rate is not a single trait, but varies across ontogeny and importantly, selection 'perceives' and distinguishes between these traits. Thus, measures of multivariate selection are necessary to reveal the full picture of selection acting on metabolic rates.

How does competition alter the process and outcome of selection? Competition decreased mean individual fitness, yet increased variation in fecundity. Competition also reduced total viability and fecundity selection intensity. Although competition environments offered greater potential for selection, overall selection on the suite of traits measured (larval mass, MR_E, MR_L), and hence variation in predicted fitness, was reduced under competition. Accordingly, while the opportunity for selection increased, the intensity of selection decreased under competition. Others have argued that higher stress does not always translate into increased strength of selection (Agrawal & Whitlock 2010), our results support this sentiment.

Competition dramatically altered selection on metabolic phenotypes. Many factors act to hamper the purging of any one metabolic phenotype in our system. Because metabolism determines the pace-of-life, it mediates a competition-colonisation trade off - this trade-off in turn generates extremely variable selection within the population. Meanwhile, even within a single environment, ubiquitous negative correlational selection hampers the capacity of strong directional selection to increase trait values. In light of these findings, intra-population variation in metabolic rate, rather than representing a challenge to theory, seems almost inevitable.

447 **Tables**

448 Table 1. Viability selection coefficients (± standard error; SE) for Larval mass (μg),
449 Metabolic rate early (MRE; mJh^{-1}), and Metabolic rate late (MRL; $mJ\ h^{-1}$) with survival to
450 reproduction for *Bugula neritina* colonies across three competition treatments. (β and γ
451 represent linear and nonlinear selection gradients, respectively. Values in bold represent
452 significant results ($p < 0.05$). Shaded boxes show consistent selection gradients among
453 environments.

No competition	β	γ		
		Larval mass	MR_E	MR_L
Larval mass	**-0.295 (0.132)**	-0.038 (0.248)	0.036 (0.073)	0.050 (0.073)
MR_E	0.137 (0.111)		0.152 (0.290)	**-0.080 (0.034)**
MR_L	**0.247 (0.126)**			0.038 (0.326)
Intraspecific competition	β	γ		
		Larval mass	MR_E	MR_L
Larval mass	-0.113 (0.119)	0.858 (0.606)	0.036 (0.073)	0.050 (0.073)
MR_E	-0.149 (0.126)		0.024 (0.218)	**-0.080 (0.034)**
MR_L	-0.012 (0.125)			**-0.288 (0.160)**
Interspecific competition	β	γ		
		Larval mass	MR_E	MR_L
Larval mass	0.054 (0.091)	0.198 (0.174)	0.036 (0.073)	0.050 (0.073)
MR_E	0.102 (0.092)		-0.004 (0.176)	**-0.080 (0.034)**
MR_L	0.013 (0.081)			0.132 (0.136)

454

455

Table 2. Fecundity selection coefficients (± standard error; SE) for Larval mass (μg), Metabolic rate early (MRE; mJh^{-1}), and Metabolic rate late (MRL; $mJ\ h^{-1}$) with reproductive output for *Bugula neritina* colonies across three competition treatments. (β and γ represent linear and nonlinear selection gradients, respectively. Values in bold represent significant results ($p < 0.05$). Note fecundity data was too sparse to estimate γ for interspecific competition.

No competition	β	γ		
		Larval mass	MR_E	MR_L
Larval mass	-0.153 (0.198)	-0.170 (0.340)	-0.213 (0.207)	-0.170 (0.214)
MR_E	-0.001 (0.164)		**-0.404 (0.236)**	0.098 (0.172)
MR_L	0.187 (0.156)			-0.182 (0.254)
Intraspecific competition	β	γ		
		Larval mass	MR_E	MR_L
Larval mass	-0.072 (0.223)	**-0.376 (0.272)**	-0.088 (0.235)	0.058 (0.293)
MR_E	**0.450 (0.226)**		**-0.712 (0.352)**	**-0.521 (0.217)**
MR_L	-0.017 (0.225)			**-0.510 (0.312)**
Interspecific competition	β	γ		
		Larval mass	MR_E	MR_L
Larval mass	**-0.566 (0.349)**			
MR_E	**0.563 (0.349)**			
MR_L	**0.209 (0.315)**			

463 **Figure captions**

464 Figure 1. Viability selection surfaces under three competition environments (no competition,
465 intraspecific competition, interspecific competition) for fitness (survival to reproduction)
466 against metabolic rate early (MR_E; mJh^{-1}) and metabolic rate late (MR_L; mJh^{-1}) of *Bugula*
467 *neritina* settlers. In order to produce standardised estimates of selection, MR_E and MR_L were
468 standardised within experimental panel and converted to units of standard deviation
469 (represented by data points) and fitness was mean centred to provide relative fitness. Note
470 that fitness is estimated based on partial regression coefficients for MR_E and MR_L linear and
471 nonlinear selection gradients.

472

473 Figure 2. Fecundity selection surfaces under three competition environments (no competition,
474 intraspecific competition, interspecific competition) for fitness (total cumulative reproductive
475 output over first five months post-outplant) against metabolic rate early (MR_E; mJh^{-1}) and
476 metabolic rate late (MR_L; mJh^{-1}) of *Bugula neritina* settlers. In order to produce standardised
477 estimates of selection, MR_E and MR_L were standardised within experimental panel and
478 converted to units of standard deviation (represented by data points) and fitness was mean
479 centred to provide relative fitness. Note that fitness is estimated based on partial regression
480 coefficients for MR_E and MR_L linear and nonlinear selection gradients. Due to insufficient
481 fecundity data, interspecific competition selection surface was fit using linear selection
482 coefficients only.

Figure 3. Linear mixed effects models for predicted growth rate (number of bifurcations over the first five months post-output) plotted against metabolic rate early (MR_E; mJh^{-1}) and metabolic rate late (MR_L; mJh^{-1}) in *Bugula neritina* settlers across three competition environments (blue = no competition, red = intraspecific competition, green = interspecific competition). For illustrative purposes, 'week' has been held constant at 5 weeks post-outplant and MR_E and MR_L are standardised by 'experimental panel' as shown by data points (both terms were included in the final mixed effects model; Table S2).

Figure 4: Logistic regression models for predicted longevity (probably of surviving >140 days) plotted against metabolic rate early (MR_E; mJh^{-1}) and metabolic rate late (MR_L; mJh^{-1}) in *Bugula neritina* settlers across three competition environments (blue = no competition,, red = intraspecific competition, green = interspecific competition. Data points represent raw MR_E and MR_L data.

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