Title: Parental stress alters the fitness effects and genetic correlation of offspring performance traits and their gene expression pathways

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Abstract

Transgenerational effects, where parental environments influence offspring phenotype, facilitate acclimation over ecological timescales. Transgenerational effects may also influence evolution by altering the fitness costs of offspring traits and the expression of genetic variation. We tested the evolutionary role of transgenerational effects in the sea urchin *Strongylocentrotus purpuratus*, whose populations are exposed to coastal upwelling (periods of low temperature and pH) and exhibit local adaptation, parental effects, and phenotypic plasticity in response to upwelling. Using a quantitative genetic breeding design, we conditioned parents and larvae to upwelling or control conditions and combined RNA-seq with larval phenotyping (body size, biomineralization, survival). Larval upwelling exposure caused widespread differential expression (DE), reduced biomineralization, and reduced size. Survival was linked to biomineralization, body size, and their plasticity under upwelling, but only in offspring of upwelling-conditioned parents - evidence that parental environment affected selection on offspring traits. DE under upwelling was associated with adaptive plasticity in biomineralization and size, but adaptive gene expression changes shared negative genetic correlations. However, genetic correlations in gene expression associated with body size plasticity were significantly more positive in larvae from upwelling parents. Parental conditioning strengthened selection on offspring performance and reduced genetic tradeoffs between performance-associated expression pathways, potentially accelerating adaptation.

Introduction

The environmental experiences of parents can influence phenotypic outcomes in offspring through a process known as transgenerational plasticity (TGP). During TGP, the environmental cue triggering plasticity is experienced by a parent or parents and the plastic response to that cue is expressed in offspring and subsequent generations (Bell and Hellmann 2019). TGP has the potential to facilitate multigenerational, adaptive responses to environmental change over rapid ecological timescales, potentially aiding the resilience of biodiversity to global change (Donelson et al. 2018; Donelan et al. 2020). Additionally, TGP can influence natural selection, reshaping adaptation over evolutionary timescales (Day and Bonduriansky 2011; Herman and Sultan 2011; Donelson et al. 2018; Che et al. 2024). Theory regarding the evolutionary effects of parental environments has developed over three decades (Kirkpatrick and Lande 1989), but empirical research on the evolutionary consequences of transgenerational effects is lacking (Bogan and Yi 2024; Cao and Chen 2024). It is unclear whether and how strongly transgenerational effects alter the primary components of evolution by natural selection: the fitness effects and heritable genetic variation of traits.

Parental environments are predicted to reshape the fitness costs of offspring phenotypes. This can happen through alteration of parental investment in response to a stressful environment, with consequences for the condition and energy budget of offspring (Bonduriansky and Crean 2018). For example, offspring from parents that performed gametogenesis under oxidative stress may receive less maternal investment of lipids, proteins, and other resources (Schreck et al. 2001; Wong et al. 2019). If an offspring genotype underpins a phenotype that requires these resources, expression of that phenotype may come at a fitness cost (Buchanan et al. 2013). Assuming that there is heritable genetic variation in this phenotype, an effect of parental environment on the

70 fitness effects of phenotypic variation will impact evolution by natural selection. For example,
71 evidence that parental exposure to stress increases the fitness effects and expression of genetic
72 variation in offspring traits would suggest that transgenerational effects can accelerate adaptation.
73 However, an experiment has not been reported that evaluated the joint influence of
74 transgenerational effects on the fitness costs and genetic variation of offspring traits.

75 Transgenerational effects can also alter the expression of genetic variation in offspring
76 phenotype (Snell-Rood et al. 2016). An individual's own environment is widely known to
77 influence the expression of genetic variation. For example, environmental stress generally
78 increases the expression of cryptic genetic variants (Rutherford and Lindquist 1998; Condelli et
79 al. 2019). Therefore, genetic variation and covariation underpinning traits and their expression is
80 dependent on the environment. Parental environments can alter the regulation of gene expression
81 in offspring and phenotypes linked to that gene expression, resulting in the release or masking of
82 genetic variation and changes in total additive genetic variation (Snell-Rood et al. 2016; Strader et
83 al. 2022).

84 Changes in gene regulation that alter the expression of genetic variation have the potential
85 to alter genetic covariance (Innocenti and Chenoweth 2013). Genetic covariance is a measure of
86 how much two traits vary together as a function of genotypic effects. Under positive genetic
87 covariance, genotypes that increase the expression of one trait generally increase the expression of
88 a second trait. Under negative genetic covariance, genotypes exhibit opposing directional effects
89 on two traits (Lande 1980). Genetic covariance is a critical constraint on evolution. Evolutionary
90 rates accelerate when the directions of selection and genetic correlation of two traits align (e.g.
91 positive directional selection on two traits sharing positive genetic correlation), but slow when the
92 direction of these parameters is misaligned - e.g. positive directional selection on two traits sharing

93 negative genetic correlation (Agrawal and Stinchcombe 2008; Blows and McGuigan 2016). If TGP
94 induces differential expression in offspring of a gene with pleiotropic phenotypic effects, genetic
95 covariance between those traits will change according to parental environment (Day and
96 Bonduriansky 2011). If transgenerational effects alter the fitness consequences and genetic
97 (co)variance of offspring phenotypes, then parental environments have potential to act as
98 modulators of evolution by natural selection.

99 Performance traits and their phenotypic plasticity can be highly polygenic, regulated by
100 multiple physiological pathways as identified by multi-environment genome-wide association
101 studies (Li et al. 2018; Liu et al. 2021; Nguyen Ba et al. 2022) and gene expression-phenotype
102 associations (DiLeo et al. 2011; Velotta et al. 2020). Whether a trait's evolution is constrained or
103 facilitated by genetic covariance depends, in part, on the strength and direction of covariance
104 between the regulatory pathways underpinning it (Innocenti and Chenoweth 2013). RNA-seq
105 experiments can be integrated with quantitative genetic breeding designs to (i) measure the
106 additive genetic variation and heritability of regulatory pathways underpinning performance traits
107 and (ii) estimate genetic variance-covariance (*G*) matrices of those pathways, consequently, their
108 genetic correlation (Blows et al. 2015). Progress has been made in determining how developmental
109 environments influence genetic correlation between traits (Sgrò and Hoffmann 2004; Wood and
110 Brodie 2015) and their fitness effects (Kingsolver and Gomulkiewicz 2003). Whether parental
111 environments reshape natural selection on traits through these mechanisms is poorly understood,
112 however (Donelson et al. 2018).

113 Using the purple sea urchin *Strongylocentrotus purpuratus,* an ecologically important
114 model invertebrate (Pearse 2006), we tested the hypotheses that parental exposure to stress
115 reshapes the fitness effects and genetic (co)variance of offspring performance traits. We

116 conditioned adults and larvae to experimental treatments that mimicked variation in temperature
117 and pCO_2 under coastal upwelling frequently experienced by *S. purpuratus*. Upwelling occurs
118 when wind-driven, upward movement of deep seawater lowers the temperature and increases the
119 pCO_2 of surface oceans (Gruber et al. 2012). Coastal upwelling is predicted to increase in intensity
120 as a result of climate change in regions such as the California Current (Snyder et al. 2003).

121 *S. purpuratus* populations inhabiting different *in situ* upwelling regimes exhibit
122 genetically-fixed differences in their degree of phenotypic and transcriptional plasticity induced
123 by experimental ocean acidification and upwelling, demonstrating that plastic, acclimatory
124 response to this stressor are a key performance metric associated with upwelling adaptation (Evans
125 et al. 2013, 2017; Kelly et al. 2013). In response to experimental upwelling, *S. purpuratus* exhibits
126 transgenerational and developmental plasticity of differential gene expression, DNA methylation,
127 and several performance traits including larval growth rate, biomineralization, and lipid content
128 (Wong et al. 2018, 2019; Strader et al. 2019, 2020, 2022; Bogan et al. 2023). Phenotypic data for
129 larval body size and biomineralization from the experiment that we describe here were reported
130 by Strader et al. 2022, who detected significant genetic variation for the plasticity of both
131 phenotypes in response to upwelling and an effect of parental environments on additive genetic
132 variation in plasticity. Therefore, we evaluated our hypotheses by focusing on the plasticity of
133 offspring phenotypes and gene expression as our primary performance traits.

134 We integrated RNA-seq with assays of performance and fitness-correlated traits in a
135 quantitative genetic crossing design of larval families exposed to upwelling or control conditions
136 (Figures 1A & S1), a design that facilitated the measurement of additive genetic variation,
137 heritability, and *G* matrices associated with phenotypic plasticity and associated regulatory

138 pathways (Fig. 1B). We tested whether parental stress modified selection on offspring plasticity
139 and genetic covariance between pathways associated with offspring plasticity.
140
141 **2. Methods**
142 *2.1 Adult conditioning, crossing, and larval culture* – Adult urchins were collected from 2
143 coastal sites in the Santa Barbara Channel in August and September of 2018: Naples Reef
144 (34.4221216, -119.95154) on August 23, 2018, and from Arroyo Quemado Reef (34.46774988, -
145 120.11905). Adults were distributed across 4 90 L tanks per parental treatment at a density of 10
146 urchins per tank. Adults acclimated to parental treatments for approximately 4 months: non-
147 upwelling = 17°C and 596 µatm pCO_2; upwelling = 12.8°C and 1117 µatm pCO_2. Flow rates to
148 adult tanks equaled 20 l h^{-1}. Adults were fed fresh *Macrocystis pyrifera ad libitum* with food
149 changes and tank cleanings conducted once per week. Seawater temperature was controlled using
150 heat pumps regulated by Nema 4X digital temperature controllers. pCO_2 was controlled using a
151 flow-through CO_2 mixing system adapted from Fangue *et al*. (Fangue et al. 2010).
152 Fertilizations were conducted using a staggered cross-classified North Carolina II breeding
153 design. During each phase of the staggered cross, 2 males and 2 females from a common adult
154 condition were reciprocally crossed and their resulting offspring were cultured under non-
155 upwelling and upwelling conditions until the prism stage of larval development. Each cross x larval
156 treatment group was reared using 2 technical replicate culture buckets, resulting in 16 larval
157 cultures per staggered cross. This process was repeated 5x for non-upwelling and upwelling
158 crosses, alternating in order between parental treatments, resulting in 40 breeding adults, 40
159 crosses reared across 160 technical replicates, and 80 biological replicates (Fig. 1A). This sample
160 size of breeding adults and lines exceeds breeding designs used to measure additive genetic

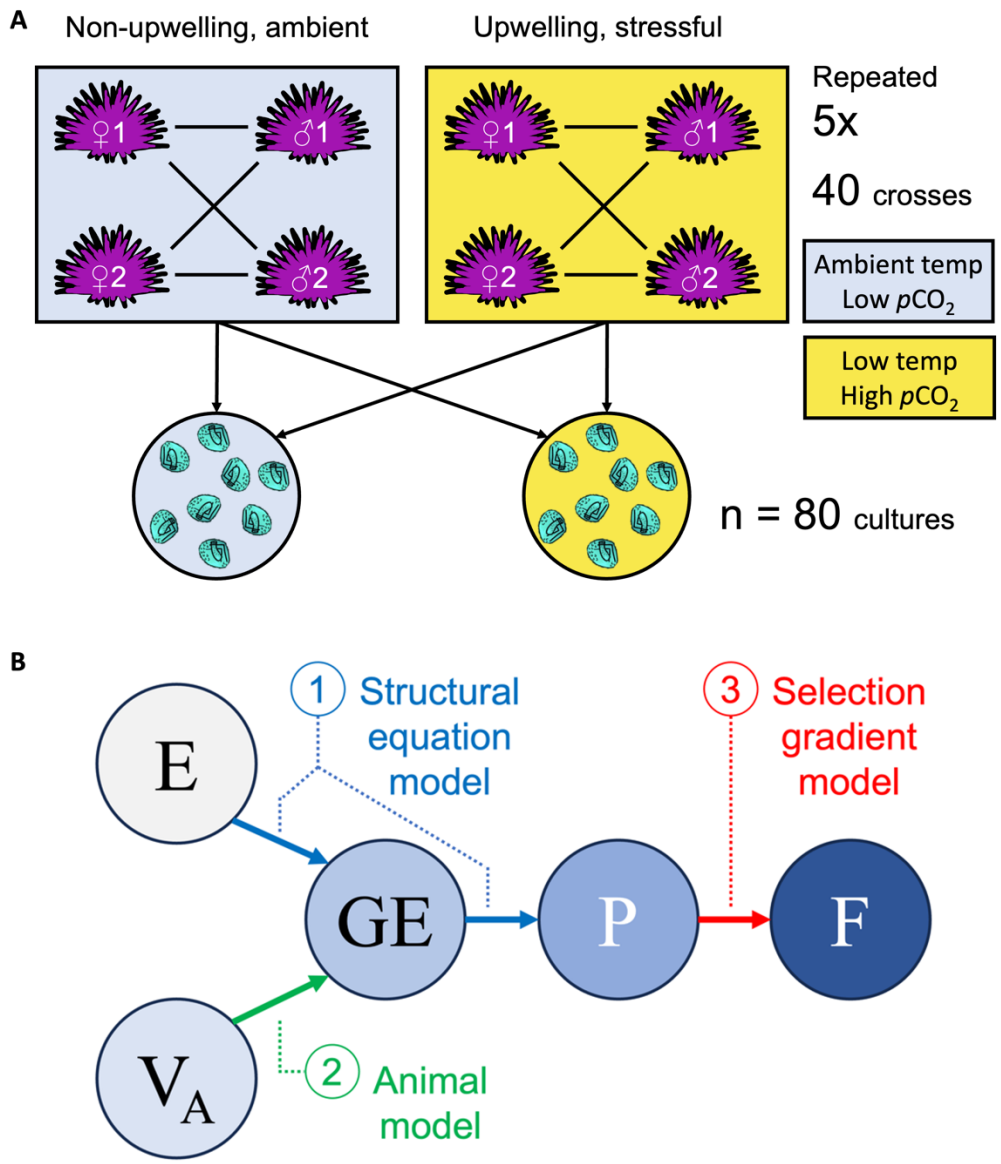

Figure 1 | *Crossing and experimental designs*. **(A)** This graphic depicts adult and larval conditioning to ambient, non-upwelling (blue) and stressful, experimental upwelling conditions (yellow). Crosses between cohorts of conditioned adults are depicted with lines. Reciprocal rearing of offspring resulting from each cross is depicted with arrows directed toward larval non-upwelling and upwelling conditions. **(B)** A visual representation of modeling approaches used to integrate environmental treatments ("E"), additive genetic variance ("V_A"), gene expression ("GE"), phenotypes ("P"), and fitness correlating traits ("F"). Structural equation modeling is visualized in blue which estimated the effect of differential expression on phenotypic plasticity induced by environmental treatments. Animal models predicting additive genetic variance in gene expression are green. Selection gradient models predicting the fitness effects of differential expression are red. Environmental treatments are grey. Parameters predicted by modeling approaches are visualized in shades of blue.

variation and *G* matrices of transcriptome-wide gene expression in other animals (Blows et al. 2015; Tung et al. 2015).

175 Larvae were cultured in replicate flow-through 6 L nested buckets (i.e., an inner bucket
176 with 30 μM mesh openings nested in an exterior bucket) at a flow rate of 3 L h^{-1} and a density of
177 10 larvae ml^{-1} until the early prism stage of larval development signified by the onset of tripartite
178 gut differentiation. Temperature and pH were regulated in larval culture buckets as described
179 above for adult conditioning. Point measures of temperature, salinity, total alkalinity, and pH for
180 adult and larval tanks are described by Strader et al. 2022.

181 *2.2. Phenotyping of performance and fitness-correlated traits* – Three phenotypes were
182 measured in larval cultures: (i) percent developmental abnormality (a corollary of survival), (ii)
183 larval body size, and (iii) spicule length per unit body size (a corollary of biomineralization).
184 Morphometric measurements of body size and spicule length were performed on $n \geq 30$ prism
185 larvae per technical replicated stored in 2% formalin buffered with 100 mM $NaBO_3$ (pH 8.7) in
186 filtered sea water. Body size was defined as the maximum linear distance of a prism body and
187 spicule length defined as length from the tip of the body rod to the branching point of the post-oral
188 rod. Abnormality was scored on $n \geq 30$ larvae during sampling and was measured as the percentage
189 of larvae exhibiting unsuccessful gastrulation. Gastrulation failure is frequently used as a proxy
190 for larval viability and survival, as gastrulation is necessary for embryo viability and development
191 (Duncan et al. 1997; Gambardella et al. 2021). Importantly, body size and spicule length were not
192 measured in non-prism larvae or ungastrulated embryos. Because RNA-seq was performed using
193 pooled RNA samples per culture, performance and fitness-correlating phenotypes were integrated
194 with gene expression data using culture means rather than per-animal values.

195 *2.3. RNA extraction, sequencing, and analysis* – Total RNA was extracted with Trizol from
196 pooled samples of 6,000 larvae per culture replicate. Extractions were performed on 1 technical
197 replicate per cross x developmental treatment resulting in 80 RNA extractions. Total RNA quantity

198 and quality was evaluated via Nanodrop, gel electrophoresis, and Qubit quantification before
199 library preparation. RNA-seq libraries were prepared using polyA enrichment and were quality
200 checked via LabChip GX. Strand specific PE 150 bp reads were sequenced on an Illumina HiSeq
201 4000 platform. Adapter removal, quality filtering, and trimming were performed using CutAdapt
202 v4.4 and Trimmomatic v0.39 before reads were aligned to the 'Spur_5.1' reference genome
203 (Sodergren et al. 2006) using hisat2 v2.2.1 (Kim et al. 2019). Specific parameters for read
204 processing, alignment, and processing of alignment files are reported in Supplemental Methods.
205 Transcript read counts were normalized in edgeR v3.40.2 as counts per million (CPM).
206 Read counts were filtered to keep all transcripts exhibiting CPM > 0.5 in at least 75% of the 80
207 replicates. Differential expression (DE) was modeled using a negative binomial generalized linear
208 model (glm) fitted with edgeR's robust, tagwise dispersion parameter using the robust iteration of
209 the model fitting function 'GLMQLFit' and the DE test function 'GLMQLFTest' (Robinson et al.
210 2010). Expression was predicted as a function of two non-interacting, categorical variables for
211 parental and developmental environment. Models were fit with non-interacting environmental
212 predictors because the study's design only enabled the measurement of V_A for developmental
213 rather than transgenerational plasticity. Fitting an interaction between both effects would confound
214 interpretation of V_A for developmental plasticity. Significant DE was evaluated using FDR
215 adjusted p-values (alpha < 0.05). Functional enrichment was tested using a rank-based Mann
216 Whitney U test of Gene Ontology terms input with logFC coefficients for DE. This test determines
217 whether a given GO term's logFC distribution is significantly skewed from the mean of the
218 background, filtered transcriptome (Wright et al. 2015).
219

220 *2.4 Identifying adaptive differential expression* – The effect of DE on the plasticity of body
221 size and biomineralization (body size-normalized spicule length) was measured using structural
222 equation models (SEMs). SEMs were derived from two linear models: (i) phenotype predicted as
223 a function of transcript abundance, developmental environment, and parental environment and (ii)
224 scaled, signed transcript abundance predicted as a function of developmental and parental
225 environments. Scaled transcript abundance was signed such that samples with low expression
226 resulting in a negative scaled value were multiplied by the direction of the transcript's DE under
227 upwelling. The effect of developmental environment on phenotype mediated by DE was estimated
228 for each transcript using mediation analysis performed with the 'mediate' function of the R
229 package mediation v4.5.0 set to 1000 simulations (Tingley et al. 2014). Positive mediation effects
230 indicated that changes in gene expression in the direction of DE were associated with higher levels
231 of body size or biomineralization under upwelling. Negative mediation effects indicated that DE
232 was associated with reduced phenotypic values under upwelling. To understand how the strength
233 of DE impacted phenotypic outcomes, linear regressions were performed between transcriptome
234 wide, absolute logFC and a second-order, quadratic polynomial for the phenotypic effects output
235 by SEM.

236 The fitness costs and benefits of plasticity in body size and biomineralization were
237 measured using an adaptation of the Lande & Arnold selection gradient model (Lande and Arnold
238 1983), which traditionally assumes uniform relatedness among individuals, for use on non-
239 uniformly related samples. This was achieved by controlling for the effect of genetic covariance
240 on the fitness-correlating outcome using a known pedigree structure. A fitness correlated trait
241 (proportion of normal development, a larval corollary of survival) was modeled as a function of
242 developmental and parental environment, body size or biomineralization per cross in each

243 environment, and the plasticity of body size or biomineralization of a cross between developmental
244 environments. Selection gradient models included a random effect identifying each cross and
245 controlling for genetic covariance using a relatedness matrix generated from custom code (see
246 https://github.com/[REDACTED]) such that offspring-parent relatedness equals 0.5, full-sibling
247 relatedness equals 0.5, half sibling relatedness equals 0.25, and unrelated cohorts share a
248 relatedness of 0. This modification of the Lande and Arnold model has been used to accommodate
249 non-uniform relatedness in other studies (Fontúrbel et al. 2021).

250 Selection gradient models were fitted in brms v2.19.6 (Bürkner 2017), an R interface to
251 the Bayesian programming language Stan (Carpenter et al. 2017). Models assumed uniform priors,
252 employed 40,000 MCMC iterations with a 5,000 iteration warm up, and a beta-distributed
253 generalized linear regression model family. Beta distribution was selected because the proportion
254 of normal development is constrained between 0 and 1. A Bayesian approach was selected for
255 fitting because of the flexibility of packages such as brms for accommodating relatedness matrices
256 within a beta distributed model family. These selection gradient models predicted whether positive
257 versus negative plasticity of larval body size and biomineralization promoted greater fitness under
258 upwelling stress. The significance of fitness effects were tested using probability of direction,
259 which determines whether the ≥ 95% of posterior distribution falls above or below 0 (Makowski
260 et al. 2019). Selection gradient coefficients were then multiplied with the phenotypic effects of DE
261 on plasticity for body size and biomineralization to calculate the associated fitness effect of
262 transcriptional plasticity.

263 *2.5 Measuring the heritability of gene expression and its plasticity* – Additive genetic
264 variation (V_A) and heritability (h_2) of gene expression was measured to determine whether there
265 was sufficient genetic variation for fitness- and phenotype-associated changes in expression to

evolve via natural selection. V_A for gene expression and DE were measured across all transcripts using animal models fit with the 'relmatLmer' function of the R package lme4qtl v0.2.2 (Ziyatdinov et al. 2018). Within animal models, DE was measured as the foldchange of gene expression across developmental environment for a given cross. Mean-standardized CPM (gene expression) was predicted as a function of fixed effects for developmental and parental environment, random effects for dam and sire, and a random effect for cross identity. Genetic covariance between crosses was estimated using the relatedness matrix described above. Mean-standardized DE fold changes were predicted using an identical animal model lacking a fixed effect for developmental environment. h^2 was derived from each model as the heritable proportion of total variance in gene expression or DE. Standard error in h^2 was calculated for each model via bootstrapping using the function bootMer function of lme4set to "parametric" and 100 simulations (Bates et al. 2015). Differences in h^2 of baseline gene expression and gene expression were modeled transcriptome wide using a quasibinomial glm linear model that corrected for standard error of h^2 by weighting observations by $1/SE^2$.

Covariation between DE's heritability and adaptive, phenotypic effects were modelled using three different approaches addressing the questions (i) 'Does the probability of heritability ($h^2 \geq 0.2$) vary as a function of DE's effect on adaptive plasticity in body size or biomineralization?', (ii) 'Does total heritability (h^2 as a continuous variable) vary as a function of DE's phenotypic effects?', and (iii) 'Does total heritability vary according to the fitness costs of DE's combined effect on plasticity in body size and biomineralization?'. Tests of questions i and ii were performed by modelling the probability of $h^2 \geq 0.2$ using a binomial glm. Continuous h^2 was modeled as a quasibinomial linear model weighted by h^2 standard error (weight = $1/SE^2$). Each model type fitted two continuous predictor variables for the phenotypic effect of DE on body size

289 and biomineralization signed toward the adaptive direction of that effect under parental upwelling,
290 which induced yielded fitness effects of plasticity as opposed to neutral effects under parental non-
291 upwelling. Models of continuous h^2 set parameters for DE's phenotypic effects as a second order
292 polynomial to accommodate non-linear variation in h^2 across the parameter space. For question iii,
293 DE's total effect on adaptive plasticity was calculated as the summation of the SEM-predicted
294 coefficient for DE of transcript *i*'s effect (*E*) on body size (*S*) and biomineralization (*B*) multiplied
295 by the selection gradient (β) acting on each plastic trait under upwelling, such that adaptive
296 plasticity associated with transcript $i = (E_{S,i} \text{ x } \beta_{S,i}) + (E_{B,i} \text{ x } \beta_{B,i})$. Covariance between DE h^2 and its
297 total adaptive effect on plasticity was modeled using a quasibinomial linear model weighted by
298 standard error of h^2 (weight = $1/SE^2$). The predictor variable of this model was calculated as the
299 sum of two products that were separately measured for the plasticity of biomineralization and size.
300 For each trait, this product equaled the indirect effect of DE on plasticity multiplied by the selection
301 gradient acting on plasticity. For example, this predictor variable was high when DE had a strong
302 and positive association with plastic decreases in upwelling, which were adaptive.

303 *2.6. Estimating G matrices* – Genetic variance-covariance matrices of absolute logFC
304 values for DE associated with adaptive plasticity in larval body size and biomineralization were
305 estimated by fitting multivariate animal models representing all possible pairs of DEGs in each
306 group. Additive genetic variance in each absolute logFC trait and covariances between pairs were
307 extracted from the multivariate models and assembled into *n*-by-*n* variance-covariance matrices,
308 where *n* equals the number of DEGs associated with adaptive plasticity in body size or
309 biomineralization. Genetic variance-covariance matrices were then converted to correlation
310 matrices using the cov2corr() function of R 'stats' v4.2.2. The number of genetically-correlated
311 modules of adaptive plasticity-associated DEGs was estimated from *G* matrices using hierarchical

312 clustering of genetic distances (1 – genetic correlation). A distance threshold of < 0.5 was used to
313 assign DEGs to their shared modules. Hierarchical clustering was performed using the hclust()
314 function of R stats v4.2.2.

315 Variation in logFC *G* matrices between larvae of upwelling and non-upwelling parents was
316 quantified. First, two multivariate animal models fitted with pedigrees of parental non-upwelling
317 or parental upwelling families were fitted for all pairs of genes with DE associated with adaptive
318 plasticity in larval body size or biomineralization. As described above, genetic (co)variances from
319 each model were used to populate parental upwelling and non-upwelling *G* matrices which were
320 converted to correlation matrices. Significant differences in correlation matrix structure between
321 parental treatments were evaluated using a three-step approach. Two-sided Mantel tests were used
322 to compare parental environment matrices, applying separate tests to logFC associated with
323 adaptive plasticity in body size and adaptive biomineralization plasticity. Mantel tests were run
324 using the mantel.test() function of the R package 'ape' v5.8 using 10,000 iterations (Paradis et al.
325 2004; Paradis and Schliep 2019). Next, a chi-squared test was used to determine if the number of
326 negative-to-positive changes in genetic correlation under upwelling relative to positive-to-negative
327 changes was greater than expected by chance.

328 To determine whether significant enrichment in negative-to-positive changes in genetic
329 correlation was attributed to parental environment or the pedigree structure of populations in
330 parental treatment groups (i.e., differences in genetic (co)variance between animals used in each
331 treatment), random permutations of the experimental pedigree were constructed. These
332 permutations split the pedigree into two halves which evenly represented families of parental
333 treatment groups. Random permutations of split pedigrees were input into animal models and
334 resulting *G* matrices. Chi-squared tests of enrichment for directional changes in genetic correlation

335 between permuted matrices were performed, which determined the effect of pedigree structure on
336 apparent effects of parental environment on genetic correlations. In Results, enrichment of
337 directional changes in genetic correlation between parental environment are reported alongside the
338 percentage of these effects that were attributed to pedigree structure rather than treatment.
339

340 **3. Results**

341 Coastal upwelling is stressful for *S. purpuratus* larvae (Wong et al. 2018, 2019; Strader et
342 al. 2020, 2022). This was similarly observed here, as evidenced by an increased percent
343 abnormality in larvae in cultures conditioned to the treatment transgenerationally and
344 developmentally (Strader et al. 2022). Developmental and parental conditioning to experimental
345 upwelling induced widespread differential expression (DE; Fig. 2). DE induced by developmental
346 upwelling was primarily associated with decreases in larval body size and, to a lesser extent,
347 increases in biomineralization (Fig. 3). The plasticity of body size and biomineralization both
348 yielded significant fitness effects measured as variance in the proportion of normal development
349 among larval families. However, fitness effects were contingent upon the parental environment
350 from which larvae were spawned. Following parental upwelling, reductions in larval body size and
351 plastic increases in biomineralization were both adaptive. Using a heritability threshold of $h^2 \geq 0.2$
352 (Orton 2020), 56.80 – 59.93% of adaptive gene expression plasticity exhibited significant
353 heritability (Fig. 4). Negative genetic correlation was abundant between DE of genes associated
354 with adaptive phenotypic plasticity. However, larvae from upwelling-exposed parents exhibited a
355 significant reduction in negative genetic correlation between DE genes associated with adaptive
356 plasticity in body size, demonstrating that changes in gene regulation following parental stress
357 modified the expression of genetic covariance (Fig. 5). We further describe these results below.

358 *3.1 Differential expression induced by parental and developmental upwelling – RNA-seq*
359 *alignment, quality checking, and filtering* – Following trimming, RNA-seq libraries achieved a
360 mean size of 37.07 ± 5.09 million reads and a mean mapping efficiency of 74.14 ± 1.80%. After
361 read-count filtering for transcripts with > 0.5 CPM in at least 75% of all samples, 12,953 transcripts
362 were retained for downstream analysis. Outlier detection was performed with filtered read count
363 data using arrayQualityMetrics v3.54.0 (Kauffmann et al. 2009), which flagged two half sibling
364 crosses from parental and developmental upwelling treatments as significant outliers (Fig. S2–S3).
365 Removal of these two samples brought the RNA-seq sample size to n = 78. Mean variation in gene
366 expression across samples equaled a biological coefficient of variation of 0.12. Library and
367 alignment metrics are further described in Supplemental File 1.

368 Parental conditioning to upwelling induced upregulation of 1,582 transcripts and
369 downregulation of 1,539 in larval offspring. These differentially expressed genes (DEGs) included
370 50 and 29 upregulated and downregulated transcripts with an absolute $\log_2$FC > 1.0 (Fig. 2). DEGs
371 induced by parental upwelling were enriched for 71 biological process (BP) GO terms, 49
372 molecular function (MF) terms, and 30 cellular component (CC) terms described in Figures S4–
373 S6. Developmental exposure to upwelling induced upregulation of 2,246 transcript and
374 downregulation of 2,205. With a >1.0 $\log_2$FC cutoff, these included 38 upregulated and 184
375 downregulated transcripts (Fig. 2). In comparison of parental and offspring responses to upwelling,
376 39.73% of DEGs induced by transgenerational effects were also differentially expressed in
377 response to developmental conditioning. Developmental upwelling DEGs were enriched for 89
378 BP GO terms, 53 MF terms, and 37 CC terms described in Figures S7–S9.

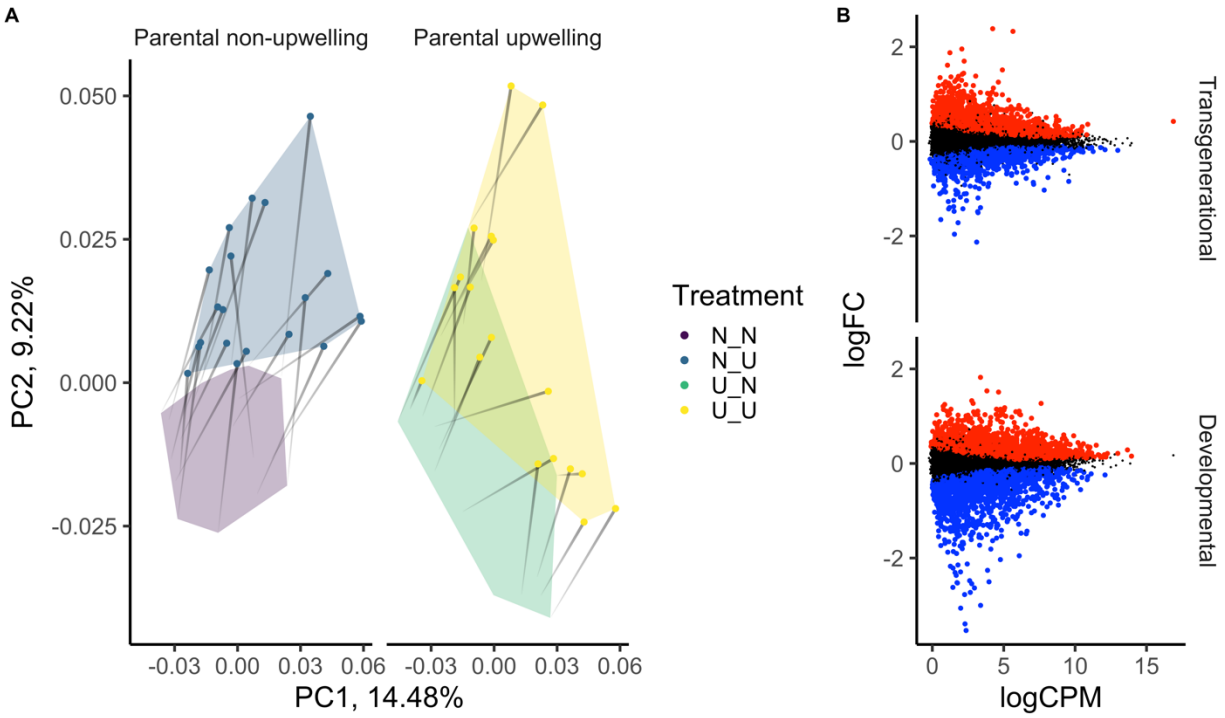

Figure 2 | *Differential expression induced by parental and developmental exposure to upwelling*. **(A)** Loading of samples to principal coordinate axes derived from filtered, normalized read counts. Parental and developmental treatments are represented by color. Paths connect single crosses and their change in loading between non-upwelling ("N"; no point) and upwelling ("U"; point) treatments. **(B)** Mean difference plots of transcript logFC across average CPM per transcript, grouped by parental and developmental effects of upwelling. Significant downregulation is depicted in blue and upregulation in red.

Upregulated transcripts related to ribosomal function included several ribosomal subunits and 16 DEAD-box proteins involved in the initiation of translation. Included in cellular responses to stress was the significant upregulation of 8 heat shock proteins including 3 Hsp70 and 5 Hsp40 chaperones. Interestingly, 8 heat shock proteins were significantly downregulated in response to upwelling, indicating that they were more highly expressed under warmer conditions. These included all 3 Hsp90 isoforms present in the *S. purpuratus* genome as well as 2 Hsp70 and 3 Hsp40 chaperones. These functional enrichment results demonstrate a suite of complex molecular responses to multivariate, abiotic environmental change brought on by experimental upwelling, a third of which were commonly induced by parental and larval conditioning.

396 *3.2. Selection on offspring performance traits and their plasticity* - Selection gradient
397 models predicting the proportion of normal development per replicate culture (a fitness correlated
398 trait) as a function of body size or biomineralization and their plasticity estimated fitness benefits
399 of (i) plastic reductions in size under upwelling and (ii) maintenance of or plastic increases in
400 biomineralization under upwelling. When larvae were spawned from parents exposed to
401 upwelling, larval biomineralization incurred a selection gradient of 0.15 ± 0.06 (Fig. 3A) and, for
402 body size, 0.13 ± 0.04 (Fig. 3B). In larvae from upwelling parents, plastic increases in
403 biomineralization incurred a selection gradient of 0.40 ± 0.09 (Fig. 3A) and plastic reductions in
404 body size incurred a weaker selection gradient of 0.13 ± 0.04 (Fig. 3B). Interestingly, the plasticity
405 of both traits did not exhibit detectable fitness costs or benefits when larvae were spawned from
406 non-upwelling parents. These selection gradient coefficients represent the slopes of logistic
407 regressions between larval survival (abnormality) and phenotypic plasticity visualized in Figures
408 3A and 3B.
409 *3.3. Associations between differential expression and adaptive versus maladaptive*
410 *plasticity* – Structural equation models (SEMs) predicting the effect of differential expression (DE)
411 on plastic changes in body size and biomineralization in response to developmental upwelling
412 identified 231 transcripts associated with increased body size, 564 with reduced body size, 125
413 associated with increased biomineralization, and 113 with decreased biomineralization. Because
414 selection gradients were significantly positive for plasticity of biomineralization, and negative for
415 plasticity in size, DE associated with reductions in body size or increases in biomineralization were
416 associated with adaptive or weakly adaptive phenotypic outcomes following parental exposure to
417 upwelling (Fig. 3C).
418

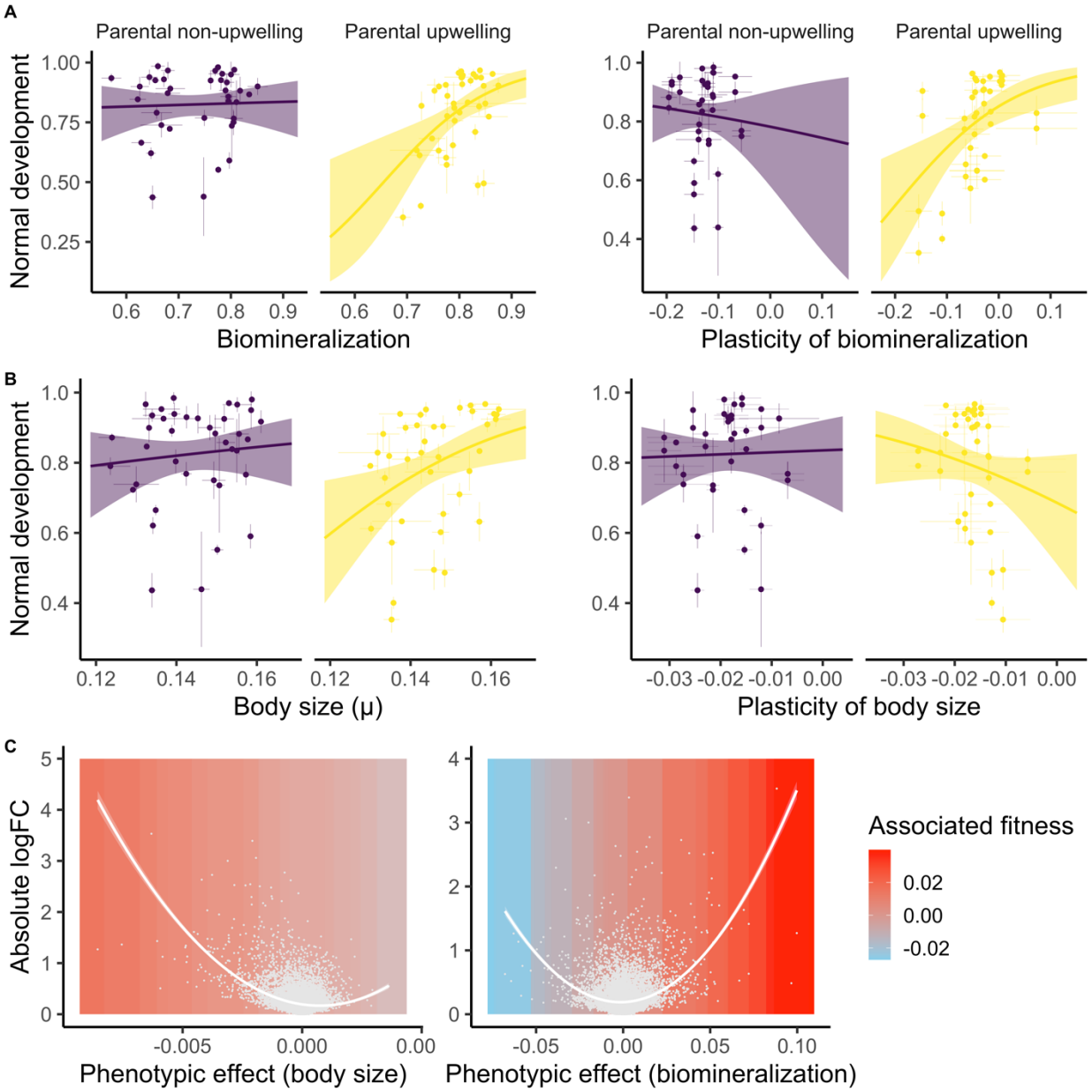

Figure 3 | *Effects of differential expression on adaptive plasticity*. **(A – B)** The effects of phenotypic values and plasticity in biomineralization (spicule length normalized by body length) and body size (maximum body length) on proportion of normal development grouped by parental environment are plotted in A and B, respectively. Points represent phenotypic means (left) or mean reaction norms (right) of crosses reared in each environment. Environments are depicted by color such that non-upwelling is blue and upwelling is yellow. Error bars depict standard deviation in each trait among replicates of each cross. **(C)** Absolute logFC of differential expression induced by developmental upwelling is plotted across differential expression's association with upwelling effects on body size and biomineralization in A and B, respectively. Points represent transcripts. Solid lines depict fitted quadratic curves ± 95% CI. Plot background color corresponds to the product of differential expression's phenotypic effect on the plasticity of body size or biomineralization and the selection gradient for plasticity of each trait under parental upwelling, resulting in an inferred fitness induced by differential expression.

433 As the absolute fold change of a transcript's DE increased, the adaptive effect of DE on
434 reduced body size and/or increased biomineralization significantly increased. From here forward,
435 'adaptive' or 'maladaptive' effects refer to selection on larvae from upwelling-exposed parents
436 unless otherwise specified. Associations between DE and effects on phenotypic plasticity toward
437 maladaptive directions were weaker, demonstrating that DE induced by developmental upwelling
438 was predominantly associated with adaptive plasticity (Fig. 3C). Transcripts associated with
439 reduced body size under upwelling conditions were enriched for BP/MF terms involved in cellular
440 signaling, transmembrane transport, and ribosomal biogenesis localized to cell junctions and the
441 nucleolus. DE driving increases or decreases in biomineralization exhibited no functional
442 enrichment across all GO term categories.

443 *3.4. Genetic (co)variance of adaptive and maladaptive differential expression* – DE was
444 highly heritable across most genes, but levels of heritability varied according to associations
445 between DE and adaptive plasticity. DE was more heritable than baseline gene expression (CPM)
446 by 19.95% h^2 ($p < 2.2e^{-16}$) and the two variables were uncorrelated (Fig. 4A). Most significant DE
447 induced by developmental conditioning (60.46%) exhibited h^2 greater than or equal to 0.20 (i.e.,
448 at least 20% of in DE variance was heritable). The mean h^2 of expression was 0.2229 ± 0.1159.
449 The standard error of expression h^2 was 0.1159 ± 0.0486. The mean h^2 of DE was 0.2674 ± 0.1592.
450 The standard error of DE h^2 was 0.1158 ± 0.0486. 7.59% of DEGs were heritable and associated
451 with adaptive plasticity in body size. However, 59.93% of adaptive DE related to body size was
452 heritable. These transcripts exhibited similar h^2 relative to all DE: 0.2613 ± 0.1516. Less DE was
453 heritable and associated with maladaptive plasticity in body size - only 3.59% of all DEGs.
454 However, maladaptive DE related to body size exhibited greater heritability with a mean h^2 of
455 0.2984 ± 0.1600. Most of the DEGs associated with adaptive increases in biomineralization

456 (56.80%) were heritable, with a mean h^2 of 0.3709 ± 0.1224. 1.55% of DEG's were heritable and
457 associated with adaptive increases in biomineralization. No DEGs that were significantly
458 associated with maladaptive decreases in biomineralization were heritable.

459 DE associated with adaptive reductions in body size under upwelling was less heritable
460 than DE associated with maladaptive increases in size. As the effect of DE on adaptive decreases
461 in body size grew, h^2 significantly decreased (β = -0.96; p = 5.77e^{-8}) as well as the probability of
462 $h^2 \geq 0.2$ (β = -5.47; p = 2.16e^{-8}; Fig. 4B). The phenotypic effect of DE on adaptive
463 biomineralization plasticity had a negative but insignificant effect on its heritability (Fig. 4B).
464 Adaptive DE remained significantly less heritable when considering the cumulative fitness effects
465 of plasticity-associated DE (β = -0.05; p = 0.00376). The cumulative fitness effect of DE was
466 measured by summing DE's phenotypic effects on both traits and multiplying these effects by the
467 selection gradients acting on each trait's plasticity.

468 Genetic variance-covariance matrices (*G* matrices) were estimated for sibships' logFC
469 values for differentially expressed genes associated with adaptive plasticity of body size and
470 biomineralization. DE associated with adaptive plasticity in body size was composed of 28
471 genetically correlated modules of 564 genes. DE associated with adaptive plasticity in
472 biomineralization was composed of 32 genetically correlated modules of 125 genes. Genetic
473 correlations between logFC values were primarily positive and exhibited larger absolute
474 correlation among logFCs sharing positive genetic correlation (Fig. 5). *G* matrices for larvae from
475 upwelling versus non-upwelling parents exhibited differences in genetic correlation for DE
476 associated with adaptive plasticity in body size. Mantel tests were used to evaluate variation
477 between parental treatments in genetic correlation matrices of DE associated with adaptive
478

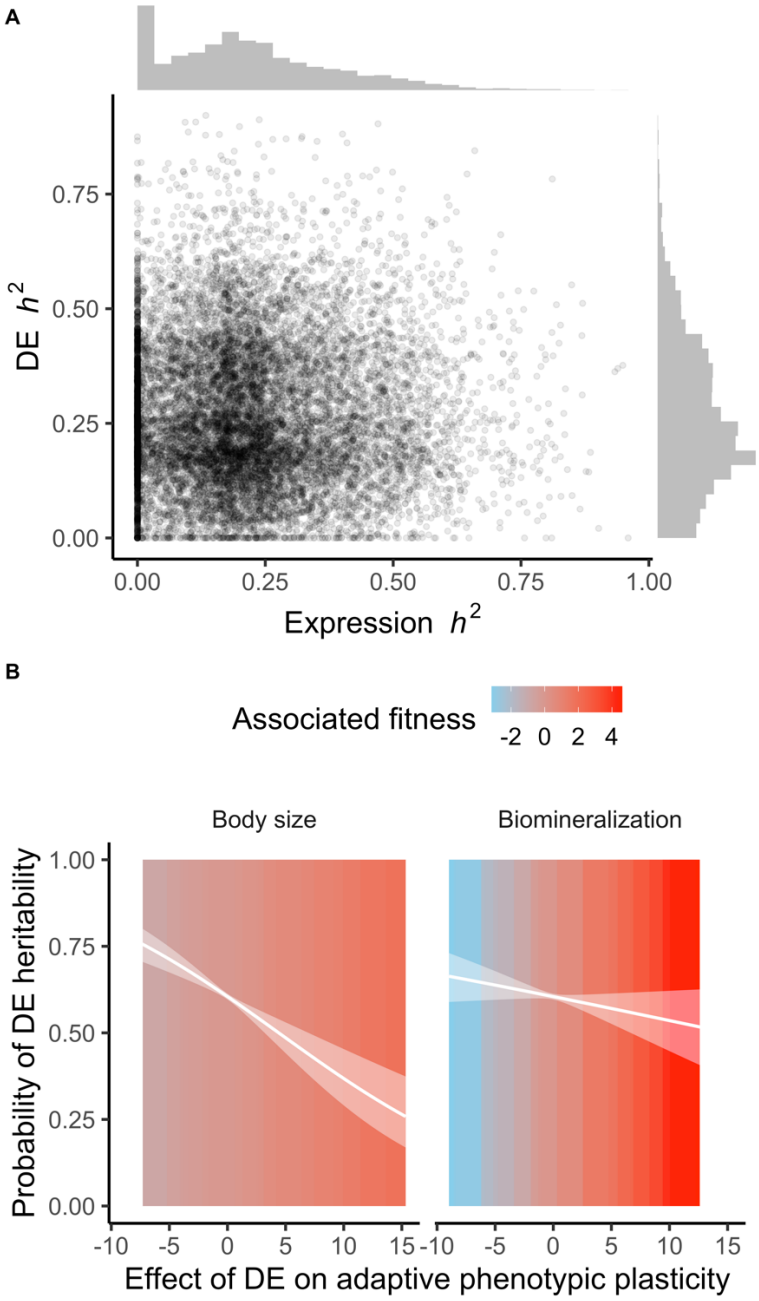

Figure 4 | *Heritability of differential expression and its association with adaptive phenotypic plasticity*. **(A)** h^2 of differential expression (DE) induced by developmental upwelling is plotted across h^2 of baseline gene expression transcriptome wide. The distributions of h^2 for baseline and differential expression are plotted adjacent to the x and y axes, respectively. **(B)** The probability of differential expression $h^2 \geq 0.20$ is plotted as a logistic curve ± 95% CI across differential expression's effect on biomineralization and body size signed for the effect's direction toward adaptive plasticity. Background color represents the coefficient for DE's effect on phenotypic plasticity multiplied by the selection gradient acting on the plasticity of each phenotype under parental upwelling (e.g., the associated fitness outcome of DE's effect on plasticity).

490 plasticity in body size ($Z = 40477.59$; $p < 1e10^{-4}$) and biomineralization ($Z = 1438.176$; $p < 1e10^{-}$
491 ^{4}). For both traits, some correlations shifted from negative-to-positive and positive-to-negative
492 under parental upwelling (Fig. 5). Even in the absence of environmental effects, random chance in
493 changes to the sign of genetic correlation are expected. Thus, a chi-squared test was used to
494 determine if there were greater or fewer positive-to-negative shifts than expected by chance alone.
495 Genetic correlations in body size DE exhibited significant shifts from negative-to-positive under
496 parental upwelling ($\chi^2 = 1767.7$; $p < 2e10^{-16}$).
497 Parental treatment groups contained different genotypes, potentially confounding
498 transgenerational effects on genetic correlation. Permutations of the experimental pedigree were
499 performed, dividing it into two random halves with equal representation of parental treatment
500 groups. Permutation revealed that 7.25% of enrichment for positive changes in genetic correlations
501 in body size DE were attributed to pedigree structure rather than parental environment. It was
502 significantly more likely that directional shifts in genetic correlation under parental upwelling were
503 attributed to environment rather than pedigree ($\chi^2 = 1639.6$; $p < 2e10^{-16}$). Most upwelling-induced
504 directional shifts to genetic correlations in biomineralization DE (94.53%) were attributable to
505 pedigree structure. Thus, there were detectable effects of parental environment on the genetic
506 covariance of pathways associated with adaptive body size plasticity, but not biomineralization.
507

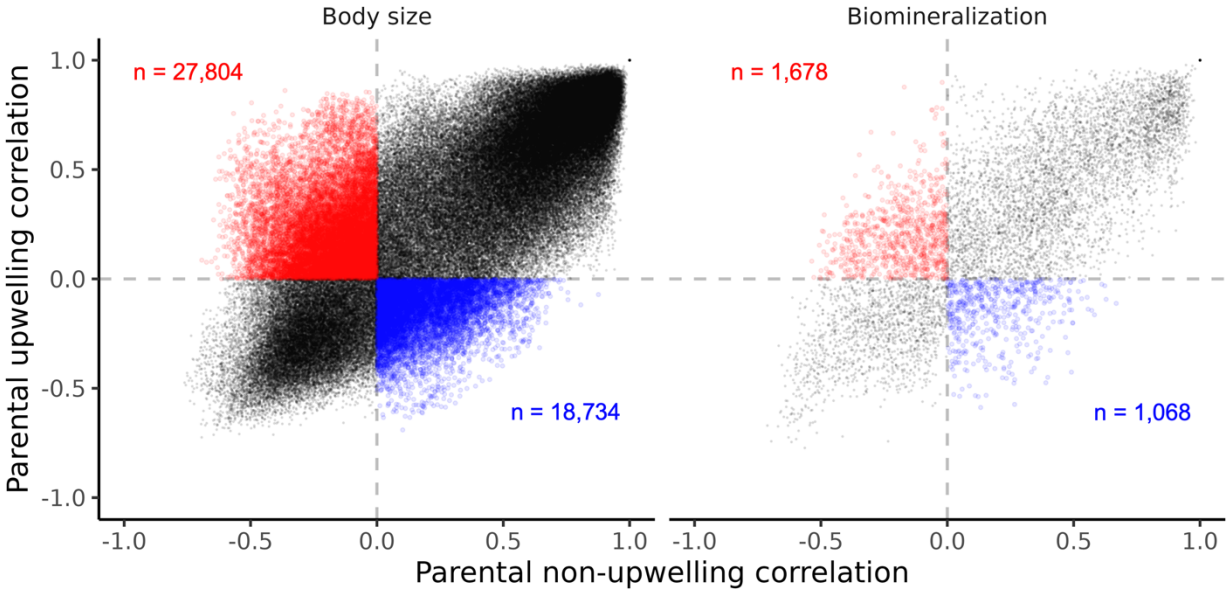

Figure 5 | *Changes in genetic correlation between parental upwelling treatments for log foldchanges (logFC) of differential expression (DE) associated with adaptive plasticity in larval body size and biomineralization*. X and Y axes represent genetic correlations extracted from *G* matrices of logFC values estimated from larvae of non-upwelling and upwelling conditioned parents. Correlations that shifted from negative to positive under parental upwelling are enlarged and colored in red. Correlations that shifted from positive to negative under parental upwelling are enlarged and colored in blue.

Heritable DE associated with plasticity in body size was enriched for functions associated with ribosomal biogenesis and maintenance (Fig. 6). Transcripts with high DE heritability and adaptive reductions in body size under upwelling were enriched for the BP/MF GO terms related to ribosomal biogenesis, RNA processing, and transmembrane transport localized to the nucleolus. Heritable DE associated with maladaptive increases in size was enriched for BP/MF terms related to amide formation (a component of translation) and ribosome structure localized to the cytosolic ribosome and large ribosomal subunit (Fig. 6). Heritable DE with adaptive effects on the plasticity of biomineralization was not enriched for GO terms.

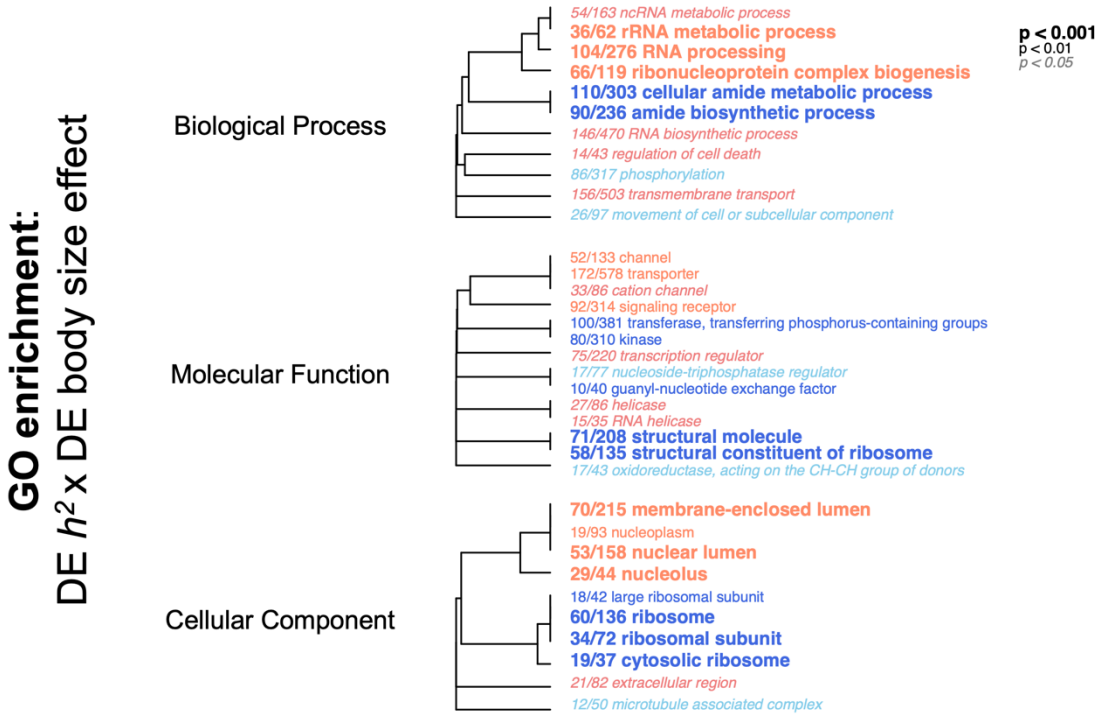

Figure 6 | *Functional enrichment of differentially expressed genes with high heritability and strong absolute effects on the plasticity of body size*. Enriched gene ontology (GO) terms are derived from a Mann Whitney U test of variation in the product of differential expression's heritability (h^2) and DE's effect on adaptive plasticity of body size in response to upwelling. Trees depict clustering of GO terms based on shared transcripts. Red GO terms are enriched within heritable differential expression associated with adaptive reductions in body size in response to developmental upwelling exposure. Blue terms are enriched within heritable differential expression associated with maladaptive increases in body size under upwelling.

4. Discussion

4.1. Transgenerational effects reshaped fitness consequences of offspring performance traits and their plasticity – Conditioning of adult *S. purpuratus* to experimental upwelling during gametogenesis led to an increase in the strength of selection gradients acting on larval size, biomineralization, and the plasticity of both traits in response to larval upwelling exposure (Fig. 3). Transgenerational effects can influence the fitness consequences of offspring phenotypes through changes in provisioning to gametes (Gagliano and McCormick 2007). In these cases, it is expected that expression of a costly phenotype in offspring with fewer resources (e.g. protein,

544 lipid, and amino acids) and lower condition will causes a fitness decline relative to the expression
545 of that same phenotype in higher condition offspring (Roff and Fairbairn 2007). Maternal
546 conditioning of *S. purpuratus* to ocean acidification has been shown to decrease lipid provisioning
547 to eggs while leaving egg size and protein provisions unaffected (Wong et al. 2019). In our
548 experiment, dams conditioned to different treatments did not did not differ in egg size (Strader et
549 al. 2022). Our study showed that abnormal development (a proxy for larval mortality) was
550 significantly higher in larvae from stress-exposed parents, demonstrating a clear decline in
551 offspring condition (Fig. 3).

552 Our findings align with and expand on studies evaluating whether parental effects alter
553 selection on offspring traits. Much of this literature has focused on parental care in nesting
554 vertebrates that have substantial post-zygotic investment, while our study addressed prezygotic
555 parental effects. In collared flycatchers, the strength and direction of selection acting on offspring
556 size changed between low- and high-quality parental rearing conditions (Krist and Munclinger
557 2015). In blue tits, low parental provisioning toward hatched offspring strengthened negative
558 selection on metabolic rate and begging behaviors (Lucass et al. 2016). In mice, maternal care
559 weakened selection gradients acting on offspring traits by buffering against the effects of
560 deleterious phenotypes (Ashbrook et al. 2015). An example of transgenerational effects on
561 selection gradients driven by nutrient provisioning, rather than care, is in damselfish, where low
562 maternal nutrition increased positive selection for increase yolk sac size in larval offspring
563 (Gagliano and McCormick 2007). Our findings connect parental investment to selection on
564 offspring traits through ecological physiology rather than parental care: parental exposure to
565 abiotic stress reduced offspring condition, strengthening the fitness costs of multiple offspring
566 traits that are critical for sustaining performance under stress.

567 Transgenerational plasticity (TGP) has been proposed to buffer against or facilitate natural
568 selection in offspring phenotypes by masking or expressing genetic variation (Vogl 1996;
569 Heckwolf et al. 2018). Our results demonstrate that transgenerational effects also reshape selection
570 on offspring by increasing the fitness effects of expressed genetic variation. Expression of costly
571 phenotypes induced by TGP in offspring that received poor provisioning may accelerate adaptation
572 over multiple generations. While transgenerational effects have received interest in global change
573 biology for their ability to promote rapid adaptive responses to climate stressors on ecological
574 timescales, our findings align with recommendations that additional research should investigate
575 their ability to modulate evolution and climate adaptation (Donelson et al. 2018; Fox et al. 2019).
576 In populations of *S. purpuratus*, for example, local adaptation to ocean acidification is evident
577 across a mosaic of upwelling in the California Current Ecosystem, where upwelling is predicted
578 to become more frequent and severe under climate change (Snyder et al. 2003). Future research in
579 natural populations of *S. purpuratus* assessing the effects of parental upwelling environment on
580 adaptive evolution in larvae may reveal how transgenerational effects alter climate adaptation.

581 *4.2. Transgenerational effects reshaped genetic variance-covariance in pathways*
582 *associated with offspring performance* – Genetic variance-covariance matrices (G matrices) can
583 constrain responses to selection in multivariate trait space when genetic covariance or correlation
584 between traits is strongly negative (Agrawal and Stinchcombe 2008). G matrices of differential
585 expression associated with adaptive offspring plasticity shared a high degree of negative genetic
586 correlation, demonstrating antagonistic pleiotropy or linkage between variants of opposing effects
587 on gene expression traits (Chebib and Guillaume 2021). Pervasive negative genetic correlation
588 indicates that adaptive responses to selection by some gene expression modules should be
589 constrained (Sgrò and Hoffmann 2004). Among DE associated with adaptive plasticity in body

size, genetic correlations between logFC values of thousands of genes significantly shifted from negative to positive in offspring of upwelling-conditioned parents (Fig. 5). Environments can influence genetic correlations between traits by inducing or silencing the expression of genetic variation underpinning those traits (Wood and Brodie 2015; Bogan and Yi 2024). For polygenic traits, this can lead to the expression of variants that share more positive or more negative genetic correlation. Effects of developmental environments on genetic correlation have been reported in pairs of traits (Sgrò and Hoffmann 2004; Wood and Brodie 2015; Fischer et al. 2016). Our results reveal that not only developmental, but parental environment, influences the expression of genetic covariation across thousands of transcriptional traits.

Increases in positive genetic correlation among DE associated with adaptive body size plasticity reduced genetic tradeoffs, mitigating constraints on adaptive evolution (Fig. 5). Shifts toward more positive genetic correlations in trait-associated pathways in larvae from upwelling-exposed parents coincided with increases in the strength of selection acting on those traits. This combination of increased selective pressure and reduced constraints on responses to selection suggest that parental upwelling exposure acted as a facilitator of adaptive evolution. Models of adaptation to environmental change frequently fit an effect of each generation's environment on the expression of genetic (co)variation and selection gradients (Gillberg et al. 2019; Cubry et al. 2022; Gallegos et al. 2025). However, the influence of environmental experience in prior generations on these parameters in descendent generations is rarely considered. Our results motivate consideration of parental environments and their transgenerational effects as processes reshaping genetic evolution and adaptation in later generations, particularly in cases of adaptation to global change.

612 *4.3. Reduced heritability of transcriptional responses associated with adaptive plasticity –*
613 Parental effects on the selection gradients and genetic covariance of offspring performance traits
614 will only influence evolution if there is heritable genetic variation underpinning those traits. We
615 observed that the heritability of DE significantly decreased when it was associated with adaptive,
616 phenotypic responses to larval upwelling conditioning (Fig. 4). This extent of genetic variation for
617 DE, and its decline in DE associated with adaptive plasticity, confirms observations in other
618 studies of plants (He et al. 2021) and animals (Oostra et al. 2018; Campbell-Staton et al. 2021).
619 However, past research has assigned putatively adaptive and maladaptive roles to molecular
620 responses based on prior findings. Here, we directly integrated gene expression plasticity with
621 fitness measures. Quantifying cross-environment genetic correlations (a measure of genetic
622 variance in plasticity) for gene expression in a tropical butterfly, Oostra et al. detected low-to-
623 undetectable genetic variation for DE between two seasonal morphological phenotypes (Oostra et
624 al. 2018). The authors proposed that minimal genetic variation for transcriptional plasticity
625 resulted from a canalized response to a highly reliable seasonal cue (Oostra et al. 2018). Numerous
626 studies have reported moderate-to-high genetic variation in DE measured as genotype x
627 environment (GxE) interactions shaping expression in animals (Grishkevich and Yanai 2013;
628 McCairns et al. 2016; Huang et al. 2020). Oostra et al. provide one of the only measures of additive
629 genetic variance in the reaction norm of gene expression rather than variance attributed to GxE.
630 Thus, it is notable that V_A of DE that they detected is strikingly lower than what we observed here.
631 *S. purpuratus* exhibited reduced genetic variation for transcriptional plasticity associated
632 with adaptive phenotypic effects, but heritability remained significant for a proportion of these
633 genes (Fig. 4). This pattern is potentially a signature of standing genetic variation and selection for
634 DE induced by upwelling in natural populations. *S. purpuratus* larvae are widely dispersed during

635 their planktonic phase resulting in high connectivity across spatial scales and high genetic diversity
636 within populations (Palumbi and Wilson 1990; Edmands et al. 1996; Pespeni and Palumbi 2013).
637 Its dispersal distances can be wide enough that larvae are frequently transported across areas of
638 major and minor upwelling in the California Current and Southern California Bight (Zaytsev et al.
639 2003; Pespeni and Palumbi 2013; Chan et al. 2017). *S. purpuratus* populations exhibit evidence of
640 local adaptation to regional differences in pCO_2 despite high rates of gene flow (Evans et al. 2013;
641 Pespeni et al. 2013). Balancing selection during seasonal changes in upwelling intensity may also
642 sustain genetic variation in plasticity (Lynn and Simpson 1987; Quilfen et al. 2021). Prior research
643 on *S. purpuratus* suggests that an individual population should possess genetic variation in plastic
644 responses associated with coastal upwelling attributed to migration and seasonal variation in
645 selection, but that reductions in genetic variation for adaptive plasticity may occur due to selection.
646 DE that was heritable and associated with adaptive plasticity, and whose genetic
647 correlations were made more positive by parental upwelling, were enriched for specific functions.
648 Heritable and adaptive changes in gene expression were enriched for GO terms associated with
649 ribosomal biogenesis while maladaptive, heritable DEGs retained enriched functions indicative of
650 ribotoxic stress such as downregulated ribosomal subunits (Fig. 6). Induced ribotoxic stress can
651 result in the downregulation of ribosomal subunits and the activation of HSF1, which upregulates
652 the cytosolic, 70 kda heat shock proteins Hsc70 and Hsp40 (Albert et al. 2019). These are the two
653 classes of heat shock proteins that were upregulated in response to larval upwelling conditioning.
654 Abiotic stress can perturbate ribosomal function via denaturation of ribosomal proteins/RNAs or
655 misfolding of nascent proteins that disrupt proximal ribosomes (Iordanov et al. 1998; De and
656 Mühlemann 2022). The overrepresentation of genes involved in ribosomal biogenesis and
657 structure among transcripts with heritable, (mal)adaptive plasticity in larval body size may be

658 explained by essential role of ribosomal homeostasis for growth and development (MacInnes
659 2016). Perturbations in ribosomal function during development bear harmful effects on organismal
660 function and size (Ordas et al. 2008; Freed et al. 2010).
661
662 **5. Conclusion**
663 Understanding how environments reshape the expression of genetic variation is a major
664 frontier in biology (Gibson and Dworkin 2004; Riederer et al. 2022), one that can improve
665 forecasts of adaptation (Martin et al. 2023). Prior research has focused on developmental
666 environments and within-generation processes, while less is understood about transgenerational
667 effects (Goldstein and Ehrenreich 2021). We demonstrated that parental environments not only
668 influence the expression of genetic variation in offspring traits, but the strength of selection on
669 those traits as well. Parental exposure to upwelling in *S. purpuratus* (i) increased the strength of
670 selection on offspring performance traits and their plasticity in response to developmental
671 upwelling exposure and (ii) caused negative-to-positive shifts in genetic covariance between gene
672 expression traits associated with adaptive plasticity. Combined, increases in the strength of
673 selection and reductions in genetic tradeoffs between adaptive traits are expected to accelerate
674 rates of adaptive evolution (Agrawal and Stinchcombe 2008; Blows and McGuigan 2016). Coastal
675 upwelling has shaped local adaptation among *S. purpuratus* populations in the California Current
676 Ecosystem (Kelly et al. 2013; Pespeni and Palumbi 2013; Evans et al. 2017), where upwelling is
677 predicted to become more severe under climate change (Quilfen et al. 2021). Our results suggest
678 that transgenerational effects on selection and genetic (co)variation potentially shaped adaptation
679 to upwelling in *S. purpuratus* and may alter the course of evolution under changing upwelling

680 regimes. More broadly, our experiment suggests that parental environments and transgenerational
681 effects play significant roles in mediating evolution by natural selection.
682
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691
692 **Data availability**
693 Raw RNA-seq data are available on the Short Read Archive under Bioproject
694 PRJNA1169019. All phenotypic data, pedigree data, and code are hosted on the Github repository
695 https://github.com/snbogan/QG_Purp_RNA. An archived copy of this Github repository is
696 available on Zenodo under the DOI [will be produced upon acceptance].
697
698 **Author Contributions**
699 SNB, MS, and GEH conceived of the aims and scope of the study. MS led experimental
700 design and phenotyping. GEH assisted SB assisted with experimental culturing. SB and MS
701 assisted with seawater chemistry and phenotyping. SB performed RNA extractions, oversaw

sequencing, bioinformatics, and all statistical analyses. SB wrote the manuscript. All authors edited and revised the manuscript.

Declarations

The authors declare no competing interests.

References

Agrawal, A. F., and J. R. Stinchcombe. 2008. How much do genetic covariances alter the rate of adaptation? Proceedings of the Royal Society B: Biological Sciences 276:1183–1191. Royal Society.

Albert, B., I. C. Kos-Braun, A. K. Henras, C. Dez, M. P. Rueda, X. Zhang, O. Gadal, M. Kos, and D. Shore. 2019. A ribosome assembly stress response regulates transcription to maintain proteome homeostasis. eLife 8:e45002. eLife Sciences Publications, Ltd.

Ashbrook, D. G., B. Gini, and R. Hager. 2015. Genetic variation in offspring indirectly influences the quality of maternal behaviour in mice. eLife 4:e11814. eLife Sciences Publications, Ltd.

Bates, D., M. Mächler, B. Bolker, and S. Walker. 2015. Fitting Linear Mixed-Effects Models Using lme4. Journal of Statistical Software 67:1–48.

Bell, A. M., and J. K. Hellmann. 2019. An Integrative Framework for Understanding the Mechanisms and Multigenerational Consequences of Transgenerational Plasticity. Annual Review of Ecology, Evolution, and Systematics 50:97–118. Annual Reviews.

Blows, M. W., S. L. Allen, J. M. Collet, S. F. Chenoweth, and K. McGuigan. 2015. The Phenome-Wide Distribution of Genetic Variance. The American Naturalist 186:15–30. The University of Chicago Press.

Blows, M. W., and K. McGuigan. 2016. The Distribution of Genetic Variance Across Phenotypic Space and the Response to Selection. Pp. 187–205 *in* Invasion Genetics. John Wiley & Sons, Ltd.

Bogan, S. N., M. E. Strader, and G. E. Hofmann. 2023. Associations between DNA methylation and gene regulation depend on chromatin accessibility during transgenerational plasticity. BMC Biol, doi: 10.1186/s12915-023-01645-8.

Bogan, S. N., and S. V. Yi. 2024. Potential Role of DNA Methylation as a Driver of Plastic Responses to the Environment Across Cells, Organisms, and Populations. Genome Biology and Evolution 16:evae022.

Bonduriansky, R., and A. J. Crean. 2018. What are parental condition-transfer effects and how can they be detected? Methods in Ecology and Evolution 9:450–456.

Buchanan, K. L., J. L. Grindstaff, and V. V. Pravosudov. 2013. Condition dependence, developmental plasticity, and cognition: implications for ecology and evolution. Trends in Ecology & Evolution 28:290–296. Elsevier.

Bürkner, P.-C. 2017. brms: an R package for Bayesian multilevel models using Stan. J. Stat. Softw. 80:1–28. jstatsoft.org.

Campbell-Staton, S. C., J. P. Velotta, and K. M. Winchell. 2021. Selection on adaptive and maladaptive gene expression plasticity during thermal adaptation to urban heat islands. Nat Commun 12:6195. Nature Publishing Group.

Cao, S., and Z. J. Chen. 2024. Transgenerational epigenetic inheritance during plant evolution and breeding. Trends in Plant Science 29:1203–1223. Elsevier.

Carpenter, B., A. Gelman, M. D. Hoffman, D. Lee, B. Goodrich, M. Betancourt, M. Brubaker, J. Guo, P. Li, and A. Riddell. 2017. Stan: A Probabilistic Programming Language. Journal of Statistical Software 76:1–32.

Chan, F., J. A. Barth, C. A. Blanchette, R. H. Byrne, F. Chavez, O. Cheriton, R. A. Feely, G. Friederich, B. Gaylord, and T. Gouhier. 2017. Persistent spatial structuring of coastal

770 ocean acidification in the California Current System. Scientific Reports 7:2526. Nature
771 Publishing Group UK London.
772 Che, J., Y. Wang, A. Dong, Y. Cao, S. Wu, and R. Wu. 2024. A nested reciprocal experimental
773 design to map the genetic architecture of transgenerational phenotypic plasticity.
774 Horticulture Research 11:uhae172.
775 Chebib, J., and F. Guillaume. 2021. Pleiotropy or linkage? Their relative contributions to the
776 genetic correlation of quantitative traits and detection by multitrait GWA studies.
777 Genetics 219:iyab159.
778 Condelli, V., F. Crispo, M. Pietrafesa, G. Lettini, D. S. Matassa, F. Esposito, M. Landriscina, and
779 F. Maddalena. 2019. HSP90 Molecular Chaperones, Metabolic Rewiring, and
780 Epigenetics: Impact on Tumor Progression and Perspective for Anticancer Therapy.
781 Cubry, P., S. Oddou-Muratorio, I. Scotti, and F. Lefèvre. 2022. Interactions between
782 microenvironment, selection and genetic architecture drive multiscale adaptation in a
783 simulation experiment. Journal of Evolutionary Biology 35:451–466.
784 Day, T., and R. Bonduriansky. 2011. A Unified Approach to the Evolutionary Consequences of
785 Genetic and Nongenetic Inheritance. The American Naturalist 178:E18–E36. The
786 University of Chicago Press.
787 De, S., and O. Mühlemann. 2022. A comprehensive coverage insurance for cells: revealing links
788 between ribosome collisions, stress responses and mRNA surveillance. RNA Biology
789 19:609–621. Taylor & Francis.
790 DiLeo, M. V., G. D. Strahan, M. den Bakker, and O. A. Hoekenga. 2011. Weighted Correlation
791 Network Analysis (WGCNA) Applied to the Tomato Fruit Metabolome. PLOS ONE
792 6:e26683. Public Library of Science.

Donelan, S. C., J. K. Hellmann, A. M. Bell, B. Luttbeg, J. L. Orrock, M. J. Sheriff, and A. Sih. 2020. Transgenerational plasticity in human-altered environments. Trends Ecol. Evol. 35:115–124.

Donelson, J. M., S. Salinas, P. L. Munday, and L. N. S. Shama. 2018. Transgenerational plasticity and climate change experiments: Where do we go from here? Glob. Chang. Biol. 24:13–34.

Duncan, S., A. Nagy, and W. Chan. 1997. Murine gastrulation requires HNF-4 regulated gene expression in the visceral endoderm: tetraploid rescue of Hnf-4(-/-) embryos. Development.

Edmands, S., P. E. Moberg, and R. S. Burton. 1996. Allozyme and mitochondrial DNA evidence of population subdivision in the purple sea urchin Strongylocentrotus purpuratus. Marine Biology 126:443–450.

Evans, T. G., F. Chan, B. A. Menge, and G. E. Hofmann. 2013. Transcriptomic responses to ocean acidification in larval sea urchins from a naturally variable pH environment. Mol. Ecol. 22:1609–1625. Wiley.

Evans, T. G., M. H. Pespeni, G. E. Hofmann, S. R. Palumbi, and E. Sanford. 2017. Transcriptomic responses to seawater acidification among sea urchin populations inhabiting a natural pH mosaic. Mol. Ecol. 26:2257–2275. Wiley Online Library.

Fangue, N. A., M. J. O'Donnell, M. A. Sewell, P. G. Matson, A. C. MacPherson, and G. E. Hofmann. 2010. A laboratory-based, experimental system for the study of ocean acidification effects on marine invertebrate larvae. Limnol. Oceanogr. Methods 8:441–452. Wiley.

Fischer, E. K., C. K. Ghalambor, and K. L. Hoke. 2016. Plasticity and evolution in correlated suites of traits. Journal of Evolutionary Biology 29:991–1002.

Fontúrbel, F. E., P. F. Ferrer, C. Vega-Retter, and R. Medel. 2021. Accounting for relatedness and spatial structure to improve plant phenotypic selection in the wild. Evol Ecol 35:15–26.

Fox, R. J., J. M. Donelson, C. Schunter, T. Ravasi, and J. D. Gaitán-Espitia. 2019. Beyond buying time: the role of plasticity in phenotypic adaptation to rapid environmental change. Philosophical Transactions of the Royal Society B: Biological Sciences 374:20180174. Royal Society.

Freed, E. F., F. Bleichert, L. M. Dutca, and S. J. Baserga. 2010. When ribosomes go bad: diseases of ribosome biogenesis. Mol. BioSyst. 6:481–493. The Royal Society of Chemistry.

Gagliano, M., and M. I. McCormick. 2007. Maternal Condition Influences Phenotypic Selection on Offspring. Journal of Animal Ecology 76:174–182. [Wiley, British Ecological Society].

Gallegos, C., L.-M. Chevin, K. A. Hodgins, and K. Monro. 2025. Predicting adaptation and evolution of plasticity from temporal environmental change. Methods in Ecology and Evolution 16:84–96.

Gambardella, C., F. Marcellini, C. Falugi, S. Varrella, and C. Corinaldesi. 2021. Early-stage anomalies in the sea urchin (*Paracentrotus lividus*) as bioindicators of multiple stressors in the marine environment: Overview and future perspectives. Environmental Pollution 287:117608.

Gibson, G., and I. Dworkin. 2004. Uncovering cryptic genetic variation. Nat Rev Genet 5:681–690. Nature Publishing Group.

837 Gillberg, J., P. Marttinen, H. Mamitsuka, and S. Kaski. 2019. Modelling G×E with historical
838 weather information improves genomic prediction in new environments. Bioinformatics
839 35:4045–4052.
840 Goldstein, I., and I. M. Ehrenreich. 2021. Genetic Variation in Phenotypic Plasticity. P. *in*
841 Phenotypic Plasticity & Evolution. CRC Press.
842 Grishkevich, V., and I. Yanai. 2013. The genomic determinants of genotype × environment
843 interactions in gene expression. Trends in Genetics 29:479–487. Elsevier.
844 Gruber, N., C. Hauri, Z. Lachkar, D. Loher, T. L. Frölicher, and G.-K. Plattner. 2012. Rapid
845 Progression of Ocean Acidification in the California Current System. Science 337:220–
846 223. American Association for the Advancement of Science.
847 He, F., K. A. Steige, V. Kovacova, U. Göbel, M. Bouzid, P. D. Keightley, A. Beyer, and J. de
848 Meaux. 2021. Cis-regulatory evolution spotlights species differences in the adaptive
849 potential of gene expression plasticity. Nat Commun 12:3376. Nature Publishing Group.
850 Heckwolf, M. J., B. S. Meyer, T. Döring, C. Eizaguirre, and T. B. H. Reusch. 2018.
851 Transgenerational plasticity and selection shape the adaptive potential of sticklebacks to
852 salinity change. Evolutionary Applications 11:1873–1885.
853 Herman, J. J., and S. E. Sultan. 2011. Adaptive transgenerational plasticity in plants: case studies,
854 mechanisms, and implications for natural populations. Front. Plant Sci. 2:102.
855 frontiersin.org.
856 Huang, W., M. A. Carbone, R. F. Lyman, R. R. H. Anholt, and T. F. C. Mackay. 2020. Genotype
857 by environment interaction for gene expression in Drosophila melanogaster. Nat
858 Commun 11:5451. Nature Publishing Group.

859 Innocenti, P., and S. F. Chenoweth. 2013. Interspecific Divergence of Transcription Networks
860 along Lines of Genetic Variance in Drosophila: Dimensionality, Evolvability, and
861 Constraint. Molecular Biology and Evolution 30:1358–1367.
862 Iordanov, M. S., D. Pribnow, J. L. Magun, T.-H. Dinh, J. A. Pearson, and B. E. Magun. 1998.
863 Ultraviolet Radiation Triggers the Ribotoxic Stress Response in Mammalian Cells *.
864 Journal of Biological Chemistry 273:15794–15803. Elsevier.
865 Kauffmann, A., R. Gentleman, and W. Huber. 2009. arrayQualityMetrics—a bioconductor
866 package for quality assessment of microarray data. Bioinformatics 25:415–416.
867 Kelly, M. W., J. L. Padilla-Gamiño, and G. E. Hofmann. 2013. Natural variation and the capacity
868 to adapt to ocean acidification in the keystone sea urchin *Strongylocentrotus purpuratus*.
869 Glob. Chang. Biol. 19:2536–2546. Wiley.
870 Kim, D., J. M. Paggi, C. Park, C. Bennett, and S. L. Salzberg. 2019. Graph-based genome
871 alignment and genotyping with HISAT2 and HISAT-genotype. Nat Biotechnol 37:907–
872 915. Nature Publishing Group.
873 Kingsolver, J. G., and R. Gomulkiewicz. 2003. Environmental Variation and Selection on
874 Performance Curves1. Integrative and Comparative Biology 43:470–477.
875 Kirkpatrick, M., and R. Lande. 1989. THE EVOLUTION OF MATERNAL CHARACTERS.
876 Evolution 43:485–503.
877 Krist, M., and P. Munclinger. 2015. Context dependence of maternal effects: testing assumptions
878 of optimal egg size, differential, and sex allocation models. Ecology 96:2726–2736.
879 Lande, R. 1980. THE GENETIC COVARIANCE BETWEEN CHARACTERS MAINTAINED
880 BY PLEIOTROPIC MUTATIONS. Genetics 94:203–215.

Lande, R., and S. J. Arnold. 1983. The measurement of selection on correlated characters. Evolution 37:1210–1226. [Society for the Study of Evolution, Wiley].

Li, X., T. Guo, Q. Mu, X. Li, and J. Yu. 2018. Genomic and environmental determinants and their interplay underlying phenotypic plasticity. Proceedings of the National Academy of Sciences 115:6679–6684. Proceedings of the National Academy of Sciences.

Liu, N., Y. Du, M. L. Warburton, Y. Xiao, and J. Yan. 2021. Phenotypic Plasticity Contributes to Maize Adaptation and Heterosis. Molecular Biology and Evolution 38:1262–1275.

Lucass, C., M. Stöwe, M. Eens, and W. Müller. 2016. Favored parent–offspring trait combinations? On the interplay of parental and offspring traits. Behavioral Ecology 27:134–140.

Lynn, R., and J. Simpson. 1987. The California Current System - the Seasonal Variability of Its Physical Characteristics. J. Geophys. Res.-Oceans 92:12947-. Amer Geophysical Union, Washington.

MacInnes, A. W. 2016. The role of the ribosome in the regulation of longevity and lifespan extension. WIREs RNA 7:198–212.

Makowski, D., M. S. Ben-Shachar, S. H. A. Chen, and D. Lüdecke. 2019. Indices of Effect Existence and Significance in the Bayesian Framework. Frontiers in Psychology 10.

Martin, R. A., C. R. B. da Silva, M. P. Moore, and S. E. Diamond. 2023. When will a changing climate outpace adaptive evolution? WIREs Climate Change 14:e852.

McCairns, R. J. S., S. Smith, M. Sasaki, L. Bernatchez, and L. B. Beheregaray. 2016. The adaptive potential of subtropical rainbowfish in the face of climate change: heritability and heritable plasticity for the expression of candidate genes. Evolutionary Applications 9:531–545.

904 Nguyen Ba, A. N., K. R. Lawrence, A. Rego-Costa, S. Gopalakrishnan, D. Temko, F. Michor,
905 and M. M. Desai. 2022. Barcoded bulk QTL mapping reveals highly polygenic and
906 epistatic architecture of complex traits in yeast. eLife 11:e73983. eLife Sciences
907 Publications, Ltd.
908 Oostra, V., M. Saastamoinen, B. J. Zwaan, and C. W. Wheat. 2018. Strong phenotypic plasticity
909 limits potential for evolutionary responses to climate change. Nat Commun 9:1005.
910 Nature Publishing Group.
911 Ordas, B., R. A. Malvar, and W. G. Hill. 2008. Genetic variation and quantitative trait loci
912 associated with developmental stability and the environmental correlation between traits
913 in maize. Genetics Research 90:385–395. Cambridge University Press.
914 Orton, T. J. 2020. Chapter 3 - Review of Genetics (From The Perspective of A Plant Breeder).
915 Pp. 39–71 *in* T. J. Orton, ed. Horticultural Plant Breeding. Academic Press.
916 Palumbi, S. R., and A. C. Wilson. 1990. Mitochondrial Dna Diversity in the Sea Urchins
917 Strongylocentrotus Purpuratus and S. Droebachiensis. Evolution 44:403–415.
918 Paradis, E., J. Claude, and K. Strimmer. 2004. APE: Analyses of Phylogenetics and Evolution in
919 R language. Bioinformatics 20:289–290.
920 Paradis, E., and K. Schliep. 2019. ape 5.0: an environment for modern phylogenetics and
921 evolutionary analyses in R. Bioinformatics 35:526–528.
922 Pearse, J. S. 2006. Ecological role of purple sea urchins. Science 314:940–941. American
923 Association for the Advancement of Science.
924 Pespeni, M. H., F. Chan, B. A. Menge, and S. R. Palumbi. 2013. Signs of adaptation to local pH
925 conditions across an environmental mosaic in the California Current Ecosystem. Integr.
926 Comp. Biol. 53:857–870. Oxford Academic.

Pespeni, M. H., and S. R. Palumbi. 2013. Signals of selection in outlier loci in a widely dispersing species across an environmental mosaic. Mol. Ecol. 22:3580–3597.

Quilfen, Y., J. Shutler, J.-F. Piolle, and E. Autret. 2021. Recent trends in the wind-driven California current upwelling system. Remote Sensing of Environment 261:112486.

Riederer, J. M., S. Tiso, T. J. B. van Eldijk, and F. J. Weissing. 2022. Capturing the facets of evolvability in a mechanistic framework. Trends in Ecology & Evolution 37:430–439. Elsevier.

Robinson, M. D., D. J. McCarthy, and G. K. Smyth. 2010. edgeR: a Bioconductor package for differential expression analysis of digital gene expression data. Bioinformatics 26:139–140.

Roff, D. A., and D. J. Fairbairn. 2007. The evolution of trade-offs: where are we? Journal of Evolutionary Biology 20:433–447.

Rutherford, S. L., and S. Lindquist. 1998. Hsp90 as a capacitor for morphological evolution.

Schreck, C. B., W. Contreras-Sanchez, and M. S. Fitzpatrick. 2001. Effects of stress on fish reproduction, gamete quality, and progeny. Aquaculture 197:3–24.

Sgrò, C. M., and A. A. Hoffmann. 2004. Genetic correlations, tradeoffs and environmental variation. Heredity 93:241–248. Nature Publishing Group.

Snell-Rood, E. C., M. Burger, Q. Hutton, and A. P. Moczek. 2016. Effects of parental care on the accumulation and release of cryptic genetic variation: review of mechanisms and a case study of dung beetles. Evol Ecol 30:251–265.

Snyder, M. A., L. C. Sloan, N. S. Diffenbaugh, and J. L. Bell. 2003. Future climate change and upwelling in the California Current. Geophysical Research Letters 30.

949 Sodergren, E., G. M. Weinstock, E. H. Davidson, et al., and R. Wright. 2006. The Genome of the
950 Sea Urchin Strongylocentrotus purpuratus. Science 314:941–952. American Association
951 for the Advancement of Science.
952 Strader, M. E., L. C. Kozal, T. S. Leach, J. M. Wong, J. D. Chamorro, M. J. Housh, and G. E.
953 Hofmann. 2020. Examining the role of DNA methylation in transcriptomic plasticity of
954 early stage sea urchins: developmental and maternal effects in a kelp forest herbivore.
955 Front. Mar. Sci. 7:205.
956 Strader, M. E., M. E. Wolak, O. M. Simon, and G. E. Hofmann. 2022. Genetic variation
957 underlies plastic responses to global change drivers in the purple sea urchin,
958 *Strongylocentrotus purpuratus*. Proc Biol Sci 289:20221249.
959 Strader, M. E., J. M. Wong, L. C. Kozal, T. S. Leach, and G. E. Hofmann. 2019. Parental
960 environments alter DNA methylation in offspring of the purple sea urchin,
961 *Strongylocentrotus purpuratus*. J. Exp. Mar. Bio. Ecol. 517:54–64.
962 Tingley, D., T. Yamamoto, K. Hirose, L. Keele, and K. Imai. 2014. mediation: R package for
963 causal mediation analysis. UCLA Statistics/American Statistical Association. UCLA
964 Statistics/American Statistical Association.
965 Tung, J., X. Zhou, S. C. Alberts, M. Stephens, and Y. Gilad. 2015. The genetic architecture of
966 gene expression levels in wild baboons. eLife 4:e04729. eLife Sciences Publications, Ltd.
967 Velotta, J. P., C. E. Robertson, R. M. Schweizer, G. B. McClelland, and Z. A. Cheviron. 2020.
968 Adaptive Shifts in Gene Regulation Underlie a Developmental Delay in Thermogenesis
969 in High-Altitude Deer Mice. Molecular Biology and Evolution 37:2309–2321.
970 Vogl, C. 1996. Developmental Buffering and Selection. Evolution 50:1343–1346. Oxford
971 University Press.

Wong, J. M., K. M. Johnson, M. W. Kelly, and G. E. Hofmann. 2018. Transcriptomics reveal transgenerational effects in purple sea urchin embryos: Adult acclimation to upwelling conditions alters the response of their progeny to differential pCO2 levels. Mol. Ecol. 27:1120–1137. Wiley.

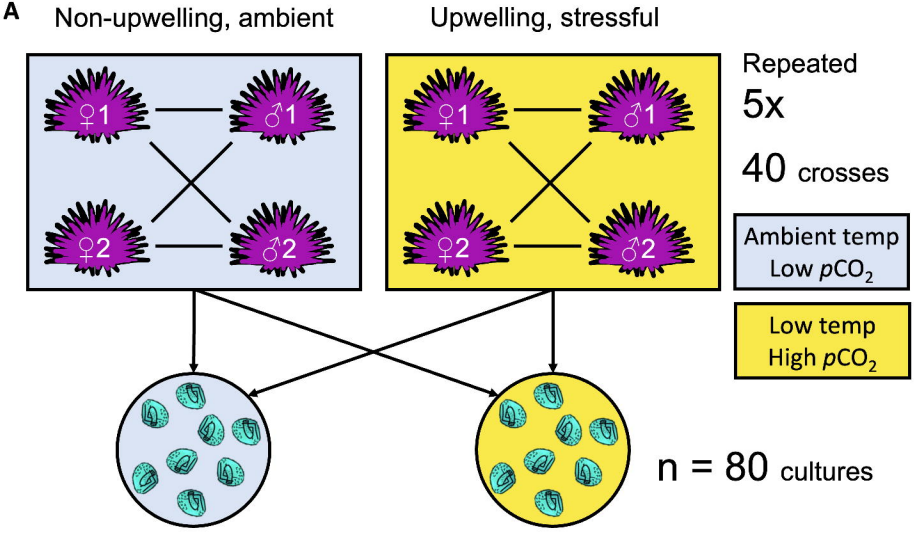
Wong, J. M., L. C. Kozal, T. S. Leach, U. Hoshijima, and G. E. Hofmann. 2019. Transgenerational effects in an ecological context: conditioning of adult sea urchins to upwelling conditions alters maternal provisioning and progeny phenotype. J. Exp. Mar. Bio. Ecol. 517:65–77. Elsevier.

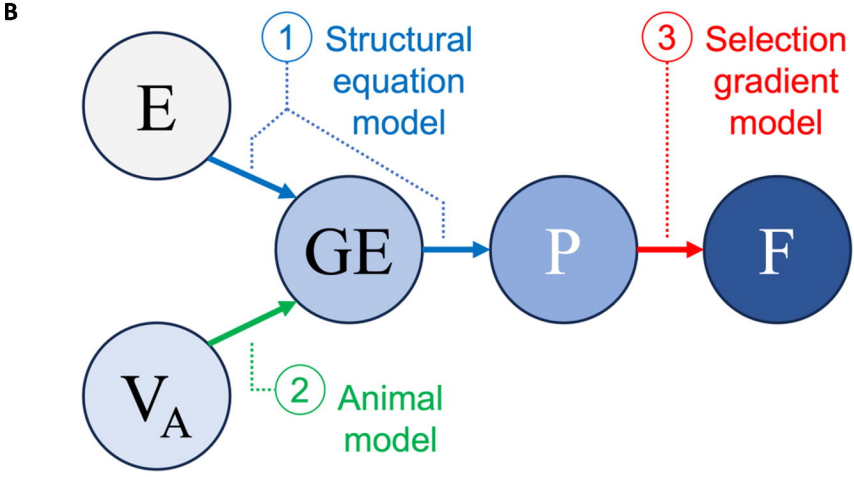
Wood, C. W., and E. D. Brodie III. 2015. Environmental effects on the structure of the G-matrix. Evolution 69:2927–2940.

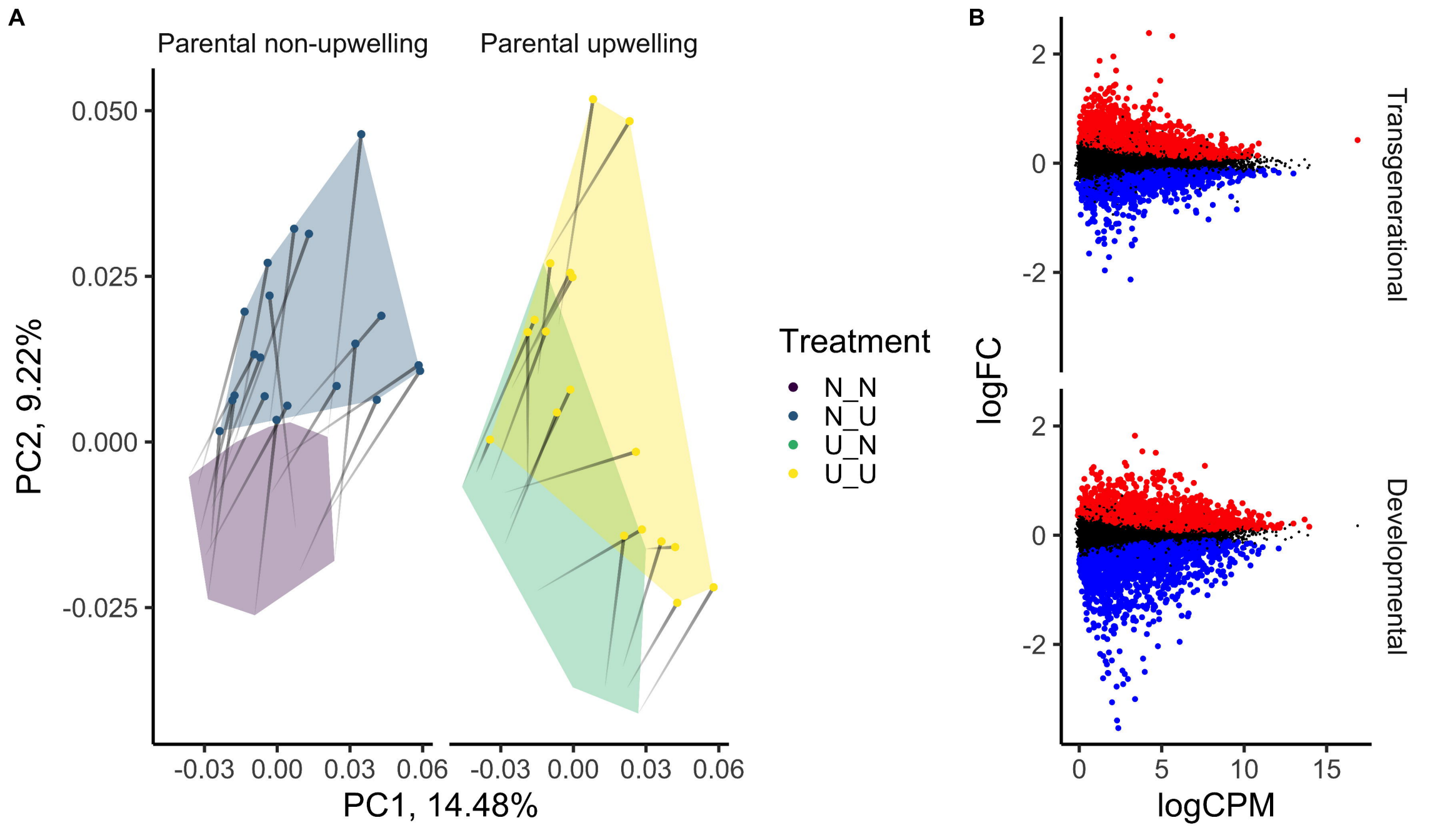
Wright, R. M., G. V. Aglyamova, E. Meyer, and M. V. Matz. 2015. Gene expression associated with white syndromes in a reef building coral, Acropora hyacinthus. BMC Genom. 16:371.

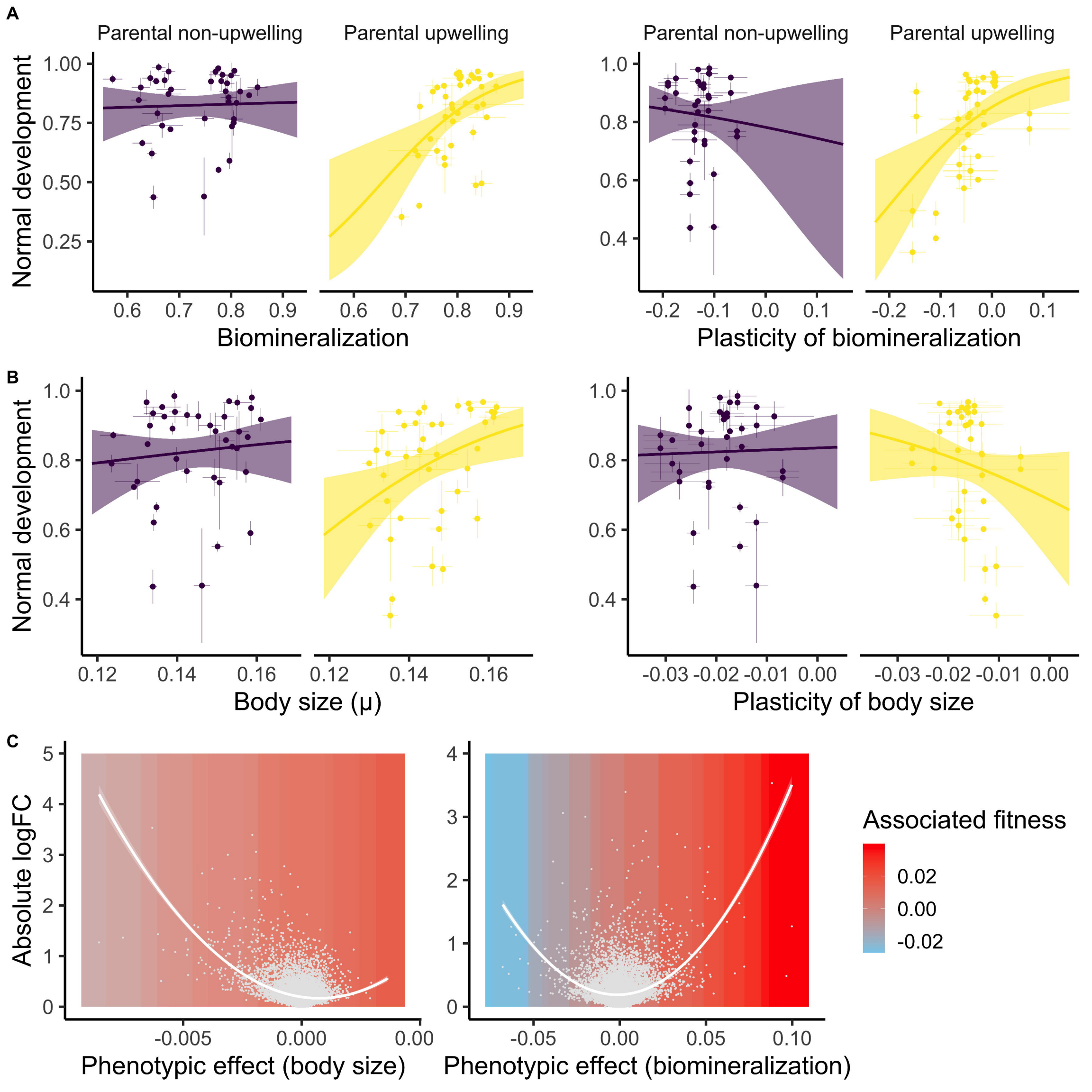
Zaytsev, O., R. Cervantes-Duarte, O. Montante, and A. Gallegos-Garcia. 2003. Coastal Upwelling Activity on the Pacific Shelf of the Baja California Peninsula. Journal of Oceanography 59:489–502.

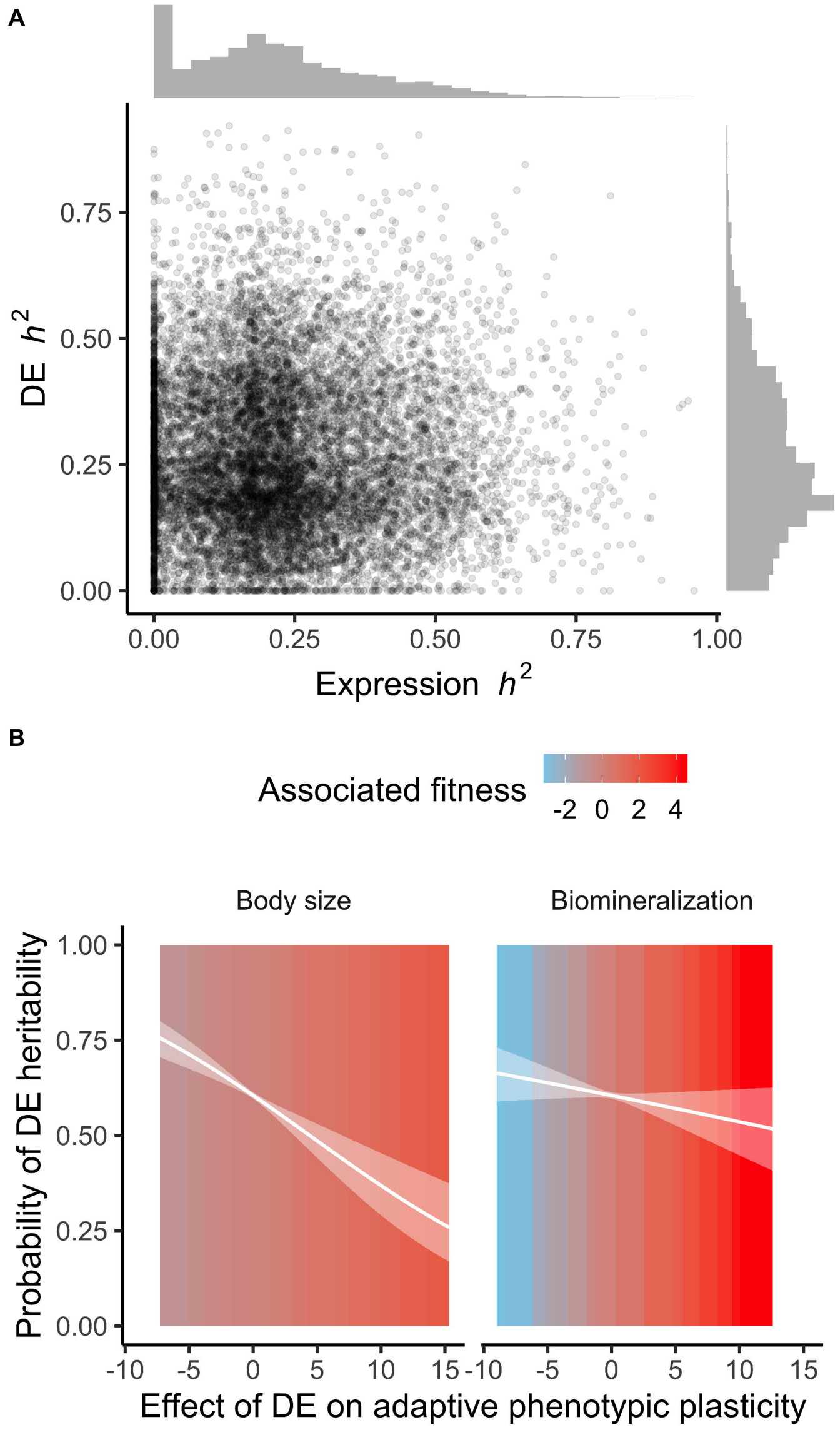
Ziyatdinov, A., M. Vázquez-Santiago, H. Brunel, A. Martinez-Perez, H. Aschard, and J. M. Soria. 2018. lme4qtl: linear mixed models with flexible covariance structure for genetic studies of related individuals. BMC Bioinformatics 19:68.

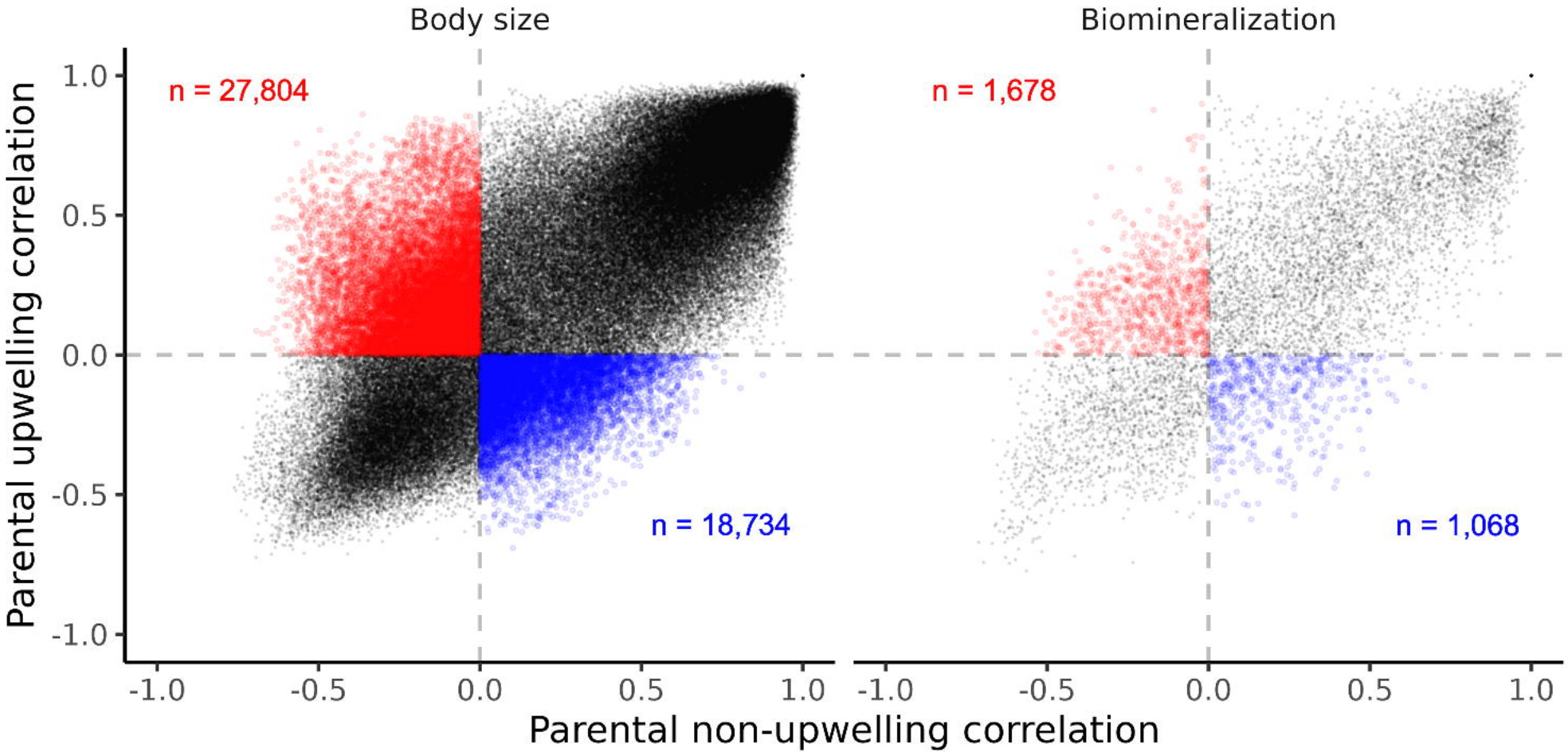

A
Non-upwelling, ambient
Upwelling, stressful
♀1
♂1
♀2
♂2
♀1
♂1
♀2
♂2
Repeated
5x
40 crosses
Ambient temp
Low pCO_2
Low temp
High pCO_2
n = 80 cultures

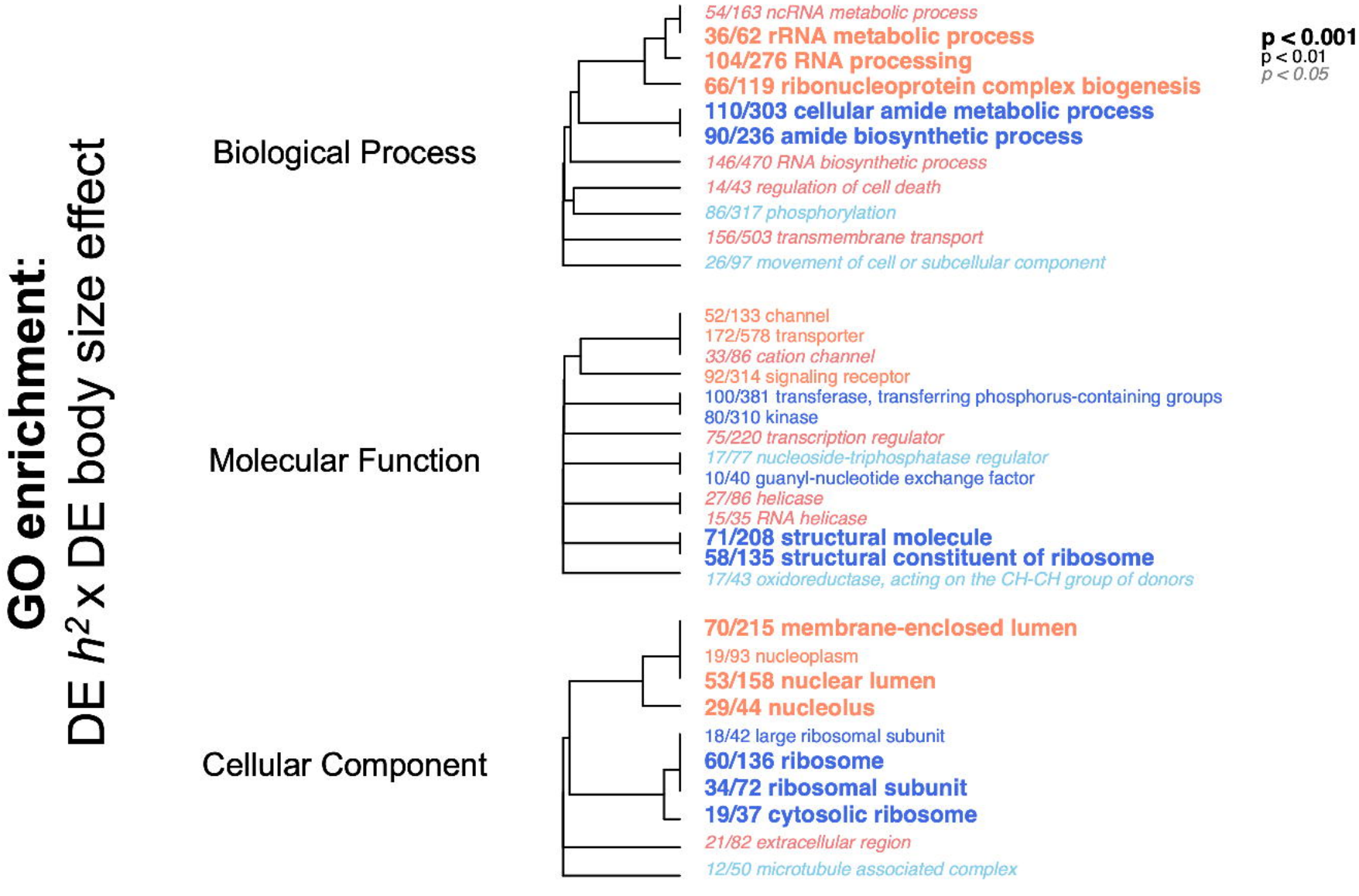

B
E
GE
P
F
V_A
1 Structural equation model
2 Animal model
3 Selection gradient model


A
Parental non-upwelling
Parental upwelling
0.050
0.025
0.000
-0.025
PC2, 9.22%
-0.03
0.00
0.03
0.06
PC1, 14.48%
Treatment
N_N
N_U
U_N
U_U
B
Transgenerational
Developmental
logFC
2
0
-2
0
5
10
15
logCPM


A
Parental non-upwelling
Parental upwelling
Parental non-upwelling
Parental upwelling
Normal development
1.00
0.75
0.50
0.25
0.6
0.7
0.8
0.9
Biomineralization
1.0
0.8
0.6
0.4
-0.2
-0.1
0.0
0.1
Plasticity of biomineralization
B
Normal development
1.0
0.8
0.6
0.4
0.12
0.14
0.16
Body size (μ)
-0.03
-0.02
-0.01
0.00
Plasticity of body size
C
Absolute logFC
5
4
3
2
1
0
-0.005
0.000
0.00
Phenotypic effect (body size)
-0.05
0.00
0.05
0.10
Phenotypic effect (biomineralization)
Associated fitness
0.02
0.00
-0.02


A
DE h^2
0.00
0.25
0.50
0.75
0.00
0.25
0.50
0.75
1.00
Expression h^2
B
Associated fitness
-2
0
2
4
Body size
Biomineralization
Probability of DE heritability
0.00
0.25
0.50
0.75
1.00
-10
-5
0
5
10
15
Effect of DE on adaptive phenotypic plasticity


Body size
Biomineralization
n = 27,804
n = 18,734
n = 1,678
n = 1,068
Parental upwelling correlation
Parental non-upwelling correlation
1.0
0.5
0.0
-0.5
-1.0
-1.0
-0.5
0.0
0.5
1.0


GO enrichment:
DE h^2 x DE body size effect
Biological Process
54/163 ncRNA metabolic process
36/62 rRNA metabolic process
104/276 RNA processing
66/119 ribonucleoprotein complex biogenesis
110/303 cellular amide metabolic process
90/236 amide biosynthetic process
146/470 RNA biosynthetic process
14/43 regulation of cell death
86/317 phosphorylation
156/503 transmembrane transport
26/97 movement of cell or subcellular component
Molecular Function
52/133 channel
172/578 transporter
33/86 cation channel
92/314 signaling receptor
100/381 transferase, transferring phosphorus-containing groups
80/310 kinase
75/220 transcription regulator
17/77 nucleoside-triphosphatase regulator
10/40 guanyl-nucleotide exchange factor
27/86 helicase
15/35 RNA helicase
71/208 structural molecule
58/135 structural constituent of ribosome
17/43 oxidoreductase, acting on the CH-CH group of donors
Cellular Component
70/215 membrane-enclosed lumen
19/93 nucleoplasm
53/158 nuclear lumen
29/44 nucleolus
18/42 large ribosomal subunit
60/136 ribosome
34/72 ribosomal subunit
19/37 cytosolic ribosome
21/82 extracellular region
12/50 microtubule associated complex
p < 0.001
p < 0.01
p < 0.05