Trait Heritability in Major Transitions

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Abstract

A crucial component of major transitions theory is that after the transition, adaptation occurs primarily at the level of the new, higher-level unit. For collective-level adaptations to occur, though, collective-level traits must be heritable. Since collective-level traits are functions of lower-level traits, collective-level heritability is related to particle-level heritability. However, the nature of this relationship has rarely been explored in the context of major transitions. We examine relationships between particle-level heritability and collective-level heritability for several functions that express collective-level traits in terms of particle-level traits. When this relationship is linear, the heritability of a collective-level trait is never less than that of the corresponding particle-level trait and is higher under most conditions. For more complicated functions, collective-level heritability is higher under most conditions, but can be lower when the function relating particle to cell-level traits is sensitive to small fluctuations in the state of the particles within the collective. Rather than being an impediment to major transitions, we show that collective-level heritability superior to that of the lower-level units can often arise 'for free', simply as a byproduct of collective formation.

Introduction

Major transitions, or evolutionary transitions in individuality, are a framework for understanding the origins of life's hierarchy and of biological complexity (Maynard Smith & Szathmáry 1995; Michod & Roze 1997). During such a transition, a new unit of evolution emerges from interactions among previously existing units. Thus the primary level of selection shifts from the particle (lower-level unit) to the collective (higher-level unit), for example from cells to multicellular organisms or from insects to eusocial societies.

Evolution by natural selection requires heritable variation in phenotypes that affect fitness at the level at which selection occurs (Lewontin 1970; Damuth & Heisler 1988). When collective-level traits are exposed to selection, it is collective-level heritability that determines the magnitude of the response. Collective-level heritability of traits is thus necessary for collective-level adaptations, and this has often been assumed to be difficult. For example, Michod claims that collective-level heritability can only arise when particles are specialized in different tasks (Michod & Roze 1997; Michod 2005, 2011). Simpson says that "From the view of some standard theory, these transitions are impossible," in part because particle-level heritability greatly exceeds collective-level heritability.

Major transitions can be conceptualized as a shift from MLS1 to MLS2, in the sense of Damuth and Heisler (1988), as in Okasha (2005) (see also Godfrey-Smith (2009), Shelton & Michod (2009)). In MLS1, properties of the particles are under selection; in MLS2, it is the properties of the collectives. We follow Okasha (2005) in referring to the lower-level units in a transition 'particles' and the higher-level units

48 ‘collectives.’ Although our biological analogies are presented in terms of cells as
49 particles and multicellular organisms as collectives, in principle our results should hold
50 for any pair of adjacent levels.

51 According to Michod (2005 p. 971), “…the challenge of ETI [evolutionary
52 transitions in individuality] theory is to explain how fitness at the group level in the sense
53 of MLS2 emerges out of fitness at the group level in the sense of MLS1.” But fitness, or
54 selection, is only half of the breeder’s equation. Predicting the response to selection
55 requires an estimate of heritability.

56 Whether or not collective-level fitness in MLS2 is a function of particle-level
57 fitness is a matter of some disagreement (for example, Rainey & Kerr 2011 say no).
58 However, collective-level traits must be functions of particle-level traits, unless we
59 accept strong emergence, a philosophical position tantamount to mysticism (Bedau
60 1997). The function may be complex and involve cell-cell communication, feedbacks,
61 environmental influences, etc., but it is still a function that is, in principle, predictable
62 from particle-level traits. Nevertheless, the relationship between heritability of particle-
63 level traits and that of collective-level traits has rarely been considered in the context of
64 major transitions, leading Okasha (2006 p. 47) to wonder, “Does variance at the particle
65 level necessarily give rise to variance at the collective level? Does the heritability of a
66 collective character depend somehow on the heritability of particle characters? The
67 literature on multi-level selection has rarely tackled these questions explicitly, but they
68 are crucial.”

69 We examine relationships between particle-level heritability and collective-level
70 heritability for several functions that express collective-level traits in terms of particle-

level traits. For the simplest (linear) function, we derive an analytical solution for the relationship. For more complex functions, we employ a simulation model to explore the relationship over a range of conditions.

Analytical model

There are several ways to estimate heritability. If the strength of selection is known, heritability can be estimated by back-calculating from the breeder's equation: $R = h^2S$, where R is the response to selection and S the selection differential. This can be rearranged as $h^2 = S/R$. Another method is to compare parent and offspring traits: the slope of the parent-offspring regression is an estimator of heritability (Falconer 1960). We use the latter method in the simulations described in the next section.

Since heritability can be defined as the proportion of phenotypic variance explained by genetic variance, one method of estimation is to partition total variance into its components using an analysis of variance. We employ this approach in an analytical model to derive the relationship between the heritability of a collective-level trait and that of the particle-level trait from which it arises. For the sake of tractability, we begin with the simplest case, assuming that the size (number of particles) of collectives is fixed and that the collective-level trait is a linear function of the particle-level traits. We further assume that reproduction is asexual, so the proper measure of heritability is broad-sense heritability, H^2 (Lynch & Walsh 1998).

We imagine a population in which collectives are made up of particles and genetically distinct clones are made up of collectives. As a concrete example, we can think of a population of undifferentiated volvocine algae, such as *Gonium*, in which case

the particles are cells and the collectives are colonies. Because of asexual reproduction, many genetically identical collectives may comprise a clone. Genetic variation among clones may arise through mutation or because the population is facultatively sexual, in which case these results will only hold for evolution within the asexual phase (in the *Gonium* example, during the summer bloom that precedes autumn mating and winter dormancy).

Broad-sense heritability is the ratio of genetic variance (V_G) to total phenotypic variance (V_P), estimated as the ratio of among-clone variance to total phenotypic variance (Lynch & Walsh 1998). Treating particles and collectives separately, the phenotype of particle k in collective j within clone i can be expressed as

$$\boldsymbol{y_{ijk} = m + A_i + B_{j(i)} + C_{k(ij)}} \quad \textbf{(1)}$$

where m is the mean genotypic value of all clones, A_i is the deviation of clone i from m, $B_{j(i)}$ is the deviation of collective j from the mean of clone i, and $C_{k(ij)}$ is the deviation of particle k from the mean of colony j within clone i. The genetic variance is the sum of squared deviations of A from m:

$$\mathbf{SSA} = \boldsymbol{bc} \sum_{i=1}^{a} \left(\overline{y}_{i..} - \overline{y}_{...}\right)^2, \quad \textbf{(2)}$$

where a, b, and c are the number of clones, collectives within a clone, and particles within a collective, respectively. Phenotypic variance of collectives within clones is

$$\mathbf{SS(B/A)} = \boldsymbol{c} \sum_{i=1}^{a} \sum_{j=1}^{b} \left(\overline{y}_{ij.} - \overline{y}_{i..}\right)^2, \quad \textbf{(3)}$$

that among particles within collectives is

$$\mathbf{SS(C/B)} = \sum_{i=1}^{a} \sum_{j=1}^{b} \sum_{k=1}^{c} \left(y_{ijk} - \overline{y}_{ij.}\right)^2, \quad \textbf{(4)}$$

and total phenotypic variance is

$$\mathbf{SST_y = SSA + SS(B/A) + SS(C/B)}. \quad \textbf{(5)}$$

Broad-sense heritability of a particle-level trait, H_y^2, is the ratio of genetic variance to total phenotypic variance:

$$\boldsymbol{H_y^2 = \frac{V_{G_y}}{V_{P_y}} = \frac{SSA}{SSA+SS(B/A)+SS(C/B)}}. \quad \textbf{(6)}$$

The phenotype of collective *j* within clone *i* can be expressed as

$$\boldsymbol{z_{ij} = \mu + \alpha_i + \beta_{j(i)}}, \quad \textbf{(7)}$$

where μ is the mean genetic value of all clones, α_i is the deviation of clone *i* from μ, and $\beta_{j(i)}$ is the deviation of collective *j* from the mean of clone *i*. The genetic variance is the sum of squared deviations of α from μ:

$$\boldsymbol{SS\alpha = b\sum_{i=1}^{a}(\overline{z}_{i\cdot} - \overline{z}_{\cdot\cdot})^2}. \quad \textbf{(8)}$$

Phenotypic variance among colonies within clones is

$$\boldsymbol{SS(\beta/\alpha) = \sum_{i=1}^{a}\sum_{j=1}^{b}\left(z_{ij} - \overline{z}_{i\cdot}\right)^2}. \quad \textbf{(9)}$$

and total phenotypic variance is

$$\mathbf{SST_z = SS\alpha + SS(\beta/\alpha)}. \quad \textbf{(10)}$$

Broad-sense heritability of a collective-level trait, H_z^2, is the ratio of genetic variance to total phenotypic variance,

$$\boldsymbol{H_z^2 = \frac{V_{G_z}}{V_{P_z}} = \frac{SS\alpha}{SS\alpha+SS(\beta/\alpha)}}. \quad \textbf{(11)}$$

If colony-level trait is the average of cell-level traits, $z_{ij} = y_{ij\cdot}$, $\overline{z}_{i\cdot} = \overline{y}_{i\cdot\cdot}$, and $\overline{z}_{\cdot\cdot} = \overline{y}_{\cdot\cdot\cdot}$. Thus SSα = *c*SSA, and SS(β/α) = *c*SS(B/A). Substituting into (11), we get

$$\boldsymbol{H_z^2 = \frac{SSA}{(SSA+SS(B/A))}}. \quad \textbf{(12)}$$

The ratio of collective-level heritability to particle-level heritability is thus

138 $$\frac{H_z^2}{H_y^2} = \frac{\mathbf{SSA+SS(B/A)+SS(C/B)}}{\mathbf{SSA+SS(B/A)}}. \quad \textbf{(13)}$$

139 Collective-level heritability is therefore never less than particle-level heritability, and is
140 greater unless SS(C/B) = 0, in other words unless particles within each collective have
141 identical phenotype. Although we have derived this relationship assuming that the
142 collective-level trait is the average of particle-level traits, the result holds for any linear
143 function.

144

145 **Simulation model**

146 The correspondence between particle-level and collective-level traits is likely to be more
147 complicated than a linear relationship for many interesting cases. Here we explore more
148 complicated trait mapping functions using a simulation model. As above, particles grow
149 in clonal collectives, which reproduce by forming two new collectives, each with as
150 many particles as its parent. The initial population is founded by ten genetically distinct
151 clones, each of which has a different genetically determined mean particle phenotype
152 (spaced evenly between 1 and 2). These are grown for at least 7 generations, resulting in
153 at least 127 collective-level reproductive events per genotype and 127*n* (where *n* is
154 particle number per collective) particle-level reproductive events per genotype.

155 In this model, we consider two sources of non-genetic effects on particle
156 phenotype (Figure 1), each of which should lower the heritability of both particle- and
157 collective-level traits. The first is intrinsic particle reproductive stochasticity, analogous
158 to developmental instability (Palmer 1994). In the model, we determine the phenotype of
159 daughter cells by sampling from a distribution centered on the parent's genetic mean,

with standard deviation σ. As shown in the analytical model above, by averaging out this variation, collectives can gain a heritability advantage over cells.

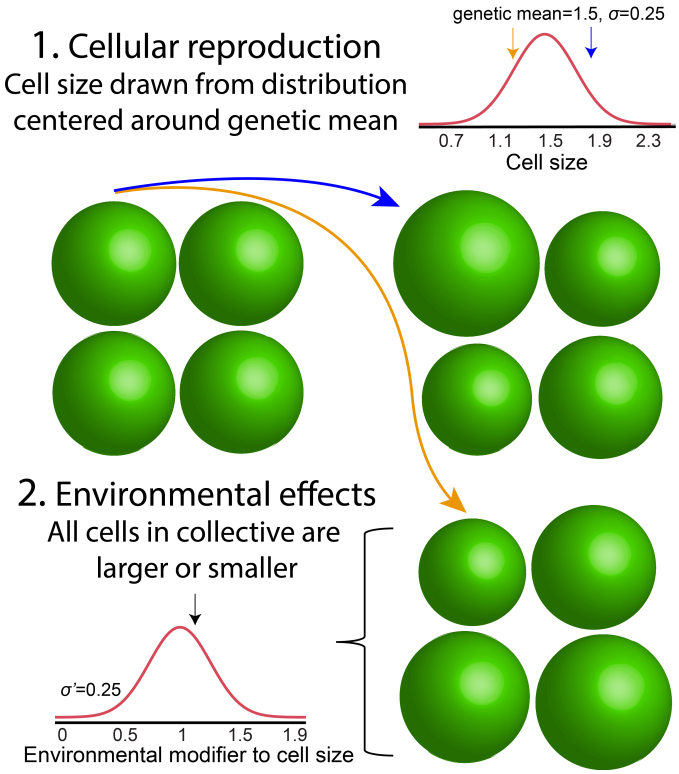

Figure 1. Two non-genetic modifiers to cell size. There are two nongenetic influences on cell size in our model: developmental instability, a stochastic effect that varies a cell's phenotype from its genetic mean size (with standard deviation σ), and environmental effects, which modify the size of all cells in a collective (with standard deviation σ').

Our simulation also considers the phenotypic effects of environmental heterogeneity. Here, we model collectives as independently experiencing different environmental conditions that affect the phenotypes of all cells within them in the same manner. To extend the biological analogy offered above, *Gonium* colonies growing near the surface of a pond (where light and CO_2 are abundant) may form colonies with larger cells than clonemates near the bottom. We implemented this in our model by assigning a size modifier, drawn from a normal distribution centered on 1 with standard deviation σ',

176 to each collective. We then multiplied the phenotype of each particle within the collective
177 by this modifier. This source of phenotypic heterogeneity should reduce the heritability
178 of collectives more than particles, simply because collectives experience a relatively
179 higher frequency of stochastic events than particles do (each collective gets assigned a
180 different size multiplier, but every particle within that collective experiences the same
181 size multiplier).

182 We examine the effect of each of the above sources of phenotypic variation
183 independently for the example of cells (particles) within nascent multicellular organisms
184 (collectives). For a linear relationship, collective size is simply the sum of the sizes of
185 cells within the collective. For both cells and collectives, heritability is assessed by
186 calculating the slope of a linear regression on parent and offspring phenotype (Falconer
187 1960). In this simple case, mean collective-level heritability is always greater than or
188 equal to cell-level heritability. Only when $\sigma = 0$ (*i.e.*, when all cells within a collective
189 have identical phenotype) are cell- and collective-level heritability equal, in agreement
190 with the analytical model. Greater developmental instability for cell size increases the
191 advantage of collective-level heritability over cell-level heritability (Figure 2a). Larger
192 collectives, which average out cellular stochasticity more effectively, experience a greater
193 benefit than smaller collectives (Figure 2a). Note that the simulations run in Figure 2a
194 reflect a very patchy environment in which environmental effects on cell size within
195 collectives are large ($\sigma' = 0.25$). Increasing the magnitude of these environmental effects
196 on cell size diminishes the difference in heritability between collectives and cells, but
197 mean collective-level heritability is still greater than cell-level heritability for all
198 parameter combinations (Figure 2b).

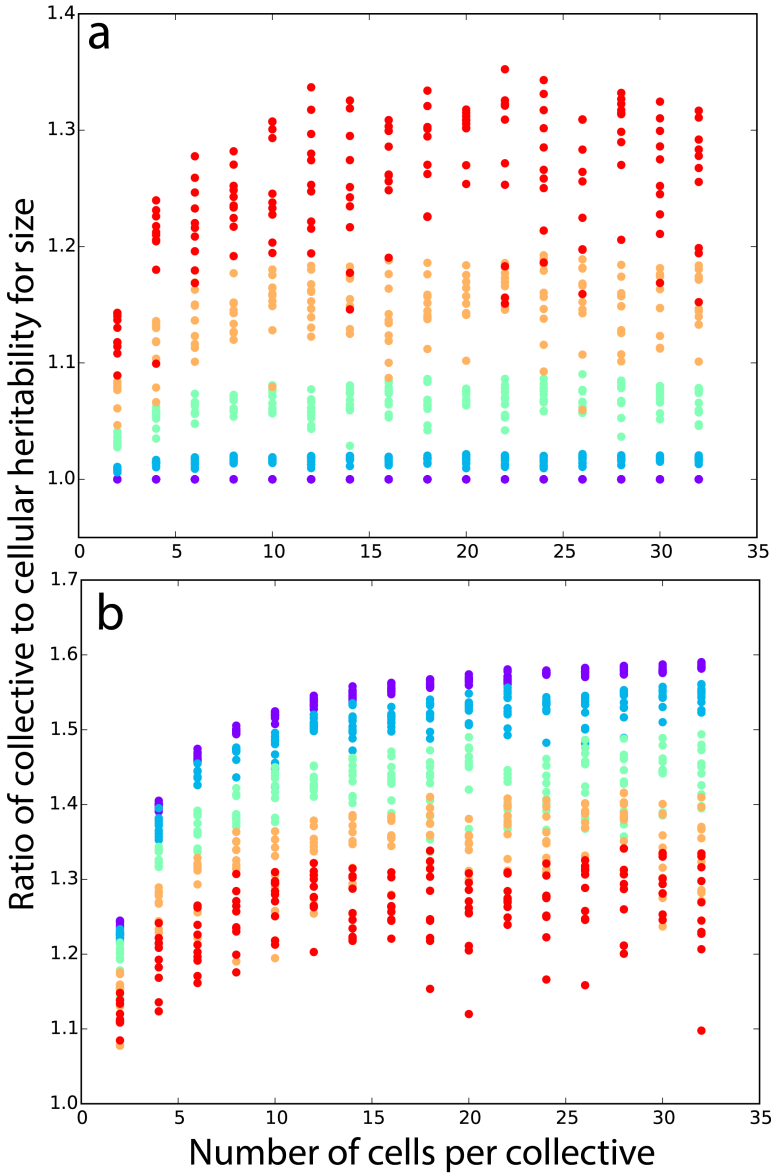

Figure 2. Collective-level heritability of size is greater than cell-level heritability for size. In **a**), we hold the effect of the environment fixed (standard deviation $\sigma' = 0.25$), and vary the degree of developmental instability σ: 10^{-4} (purple), 0.0625 (blue), 0.125 (green), 0.1875 (yellow), 0.25 (red). In the absence of developmental instability, collective and cell-level heritabilites are identical. Greater developmental instability increases relative collective-level heritability. **b**) Here we hold developmental instability fixed at $\sigma = 0.25$, and vary between-collective environmental effects on cell size from $\sigma' = 10^{-4}$ (purple) to 0.25 (red). When developmental instability is nonzero, larger collectives improve collective-level heritability. Ten replicates were run of each parameter combination. Populations were simulated for nine generations of growth.

The volume of the cellular collective (Figure 2, Figure 3a), which is simply the sum of the cell volumes within it, represents the simplest function mapping cellular to

multicellular traits. We now consider more complicated nonlinear functions relating cellular to multicellular traits, some of which have biological relevance to the evolution of multicellularity. For each function, we calculated the relative heritability of collective- to cell-level traits for 32-celled collectives across 1024 combinations of σ and σ' ranging from 0 to 0.25. The first nonlinear collective-level trait we consider is its diameter. Large size is thought to provide a key benefit to nascent multicellular collectives when they become too big to be consumed by gape-limited predators (Stanley 1973; Bell 1985). For a collective that is approximately spherical, the trait that actually determines the likelihood of being eaten is diameter. For geometric simplicity we assume that the cells within the collective are pressed tightly together into a sphere, allowing us to calculate collective radius as $d = 2\left(\frac{3V}{4\pi}\right)^{\frac{1}{3}}$, where V is the sum of the cell volumes within the collective. Collective volume (Figure 3a) and diameter (Figure 3b) exhibit similar dynamics, with collective-level heritability always exceeding cell-level heritability, and being maximized under conditions of strong cell size stochasticity (high σ) and no environmental heterogeneity (low σ').

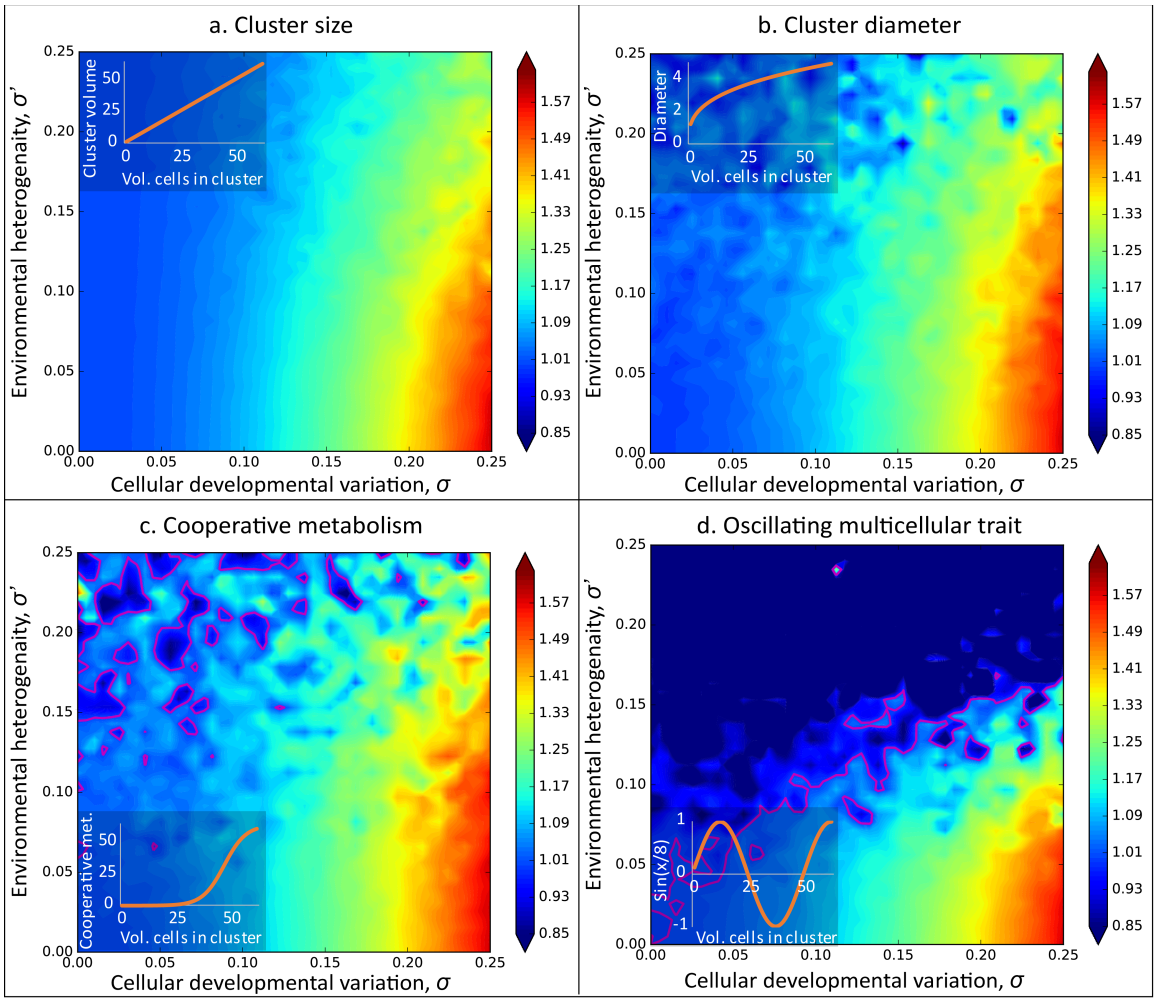

Figure 3. Relative heritability of various collective-level traits to cell-level heritability for size. Here we examine the heritability of four multicellular traits that depend on the size of their constituent cells, relative to cellular heritability for size. The relationship between the size of the cells within collectives and the multicellular trait are shown as insets. We consider three biologically-significant traits with different functions mapping the size of cells within the collective onto collective phenotype. The heritability of collective size (**a**) and diameter (**b**) is always higher than cell-level heritability for size, and is maximized when cellular developmental noise is greatest and among-collective environmental effects are smallest (lower right corner). We modeled cooperative metabolism (**c**) with a logistic function, such that there is a threshold over which collectives are large enough to perform some metabolic task. We also considered a multicellular trait that does not monotonically increase with greater collective size, but instead oscillates with varying collective size (**d**). Like a and b, collective-level heritability is highest relative to cell-level heritability when environmental heterogeneity is minimal. Pink contours denote relative heritability of 1. In these simulations we consider 32 cell collectives grown for 7 generations. The colormap denotes collective-level heritability divided by cell-level heritability for size across 1024 σ, σ' combinations.

249 Next, we consider a logistic function describing among-cell cooperation (*e.g.*,
250 production of costly extracellular metabolites), in which the extent of cooperative
251 metabolism depends nonlinearly on collective volume. Specifically, we calculated the
252 extent of cooperative metabolism as $c = 64/(1 + e^{(-.2(V-48))})$. Here, the center of the
253 collectives' size distribution (*i.e.*, the volume of a collective with all 32 cells having size
254 1.5) lies at the function's inflection point. As with the previous two functions, collective-
255 level heritability is greater than cell-level heritability for much of the trait space, and is
256 maximized under conditions of high cellular stochasticity and low environmental
257 heterogeneity.

258 Finally, we consider a collective-level trait that oscillates between -1 and 1 with
259 increased cell size: $O = \sin(\frac{V}{8})$, where V is the sum of cell volumes. This trait has no
260 obvious biological interpretation, but is distinct from the linear and nonlinear (but
261 monotonically increasing) functions described previously. While the general relationship
262 observed for these other functions still holds (collectives have greater heritability when
263 cellular stochasticity is high and environmental heterogeneity low), we now find that
264 much of the trait space now favors cellular heritability over that of the collective (Figure
265 3d, upper left). This appears to be due to the sensitivity of the function relating collective
266 to particle-level traits. We explore this further in Figure S1, where we consider four
267 versions of the model presented in Figure 3d, varying the sensitivity of the collective's
268 response to its lower-level composition. The heritability of collective-level traits is
269 minimal when small differences in cellular phenotype generate radically different
270 collective-level phenotypes. This makes sense: there is little potential for collective-level

271 heritability when tiny differences in cellular phenotype within collectives generate
272 drastically different collective-level phenotypes.
273
274 **Discussion**
275 Using a quantitative genetics framework, we have derived an analytical solution for the
276 relationship between particle-level and collective-level heritability for a limited case.
277 When the organismal trait is a linear function of the cell level traits, the organismal
278 heritability turns out to be a simple function of the cell-level heritability. In contrast to
279 claims that particle-level heritability is always higher than collective-level heritability
280 (*e.g.*, Simpson 2011), we have shown that collective-level heritability is higher over a
281 wide range of conditions. Furthermore, we have shown that, contrary to Michod's results
282 for fitness heritability (Michod & Roze 1997; Michod 2005, 2011), specialization of
283 particles is not necessary for collective-level trait heritability to arise.
284 This analytical result is a step toward understanding the relationship between
285 heritabilities at two adjacent hierarchical levels, but the assumption that the collective-
286 level trait is a linear function of the particle-level trait is restrictive. The simulation model
287 shows that the results are dependent on the function relating the traits at the two levels.
288 Even under the conditions we model, which are favorable to collective-level heritability
289 (clonal reproduction, negligible within-collective mutation, and fixed cell number per
290 collective), collective-level heritability was not always higher than cell-level heritability
291 (Figure 3d, Figure S1). Specifically, the relative heritability of collectives to cells was
292 below 1 when the collective-level phenotype was extremely sensitive to small changes in
293 the phenotype of cells within the collective (Figure S1). It is important to note, however,

294 that we only saw this high sensitivity in a function with little biological relevance
295 (collective-level phenotype oscillated between -1 and 1 with increased cell volume).
296 Collective-level heritability was higher than cell-level heritability for most of the trait
297 space in the other three biologically significant functions we considered (Figure 3a-c).

298 In our simulation, we examined the effects of two independent sources of
299 phenotypic variation affecting the relative heritability of particle and collective-level
300 traits. Stochastic variation in cell size around the clone's genetic mean (σ) reduces the
301 absolute heritability of cells and collectives by introducing non-heritable phenotypic
302 variation. By averaging across multiple cells, however, collectives reduce the effects of
303 this phenotypic variation, providing them with a relative heritability advantage over cells.
304 We also considered the effect of environmental heterogeneity in which all of the cells
305 within a collective are affected in the same manner (σ'). Collectives are
306 disproportionately affected: each group is assessed a different size modifier, but all of the
307 cells within these groups are affected in the same manner. As a result, collectives
308 experience *n*-fold (where *n* is the number of cells per collective) more stochastic events,
309 which reduces their heritability relative to cells. The influence of these sources of
310 variation is evident in the contour plots of Figures 3 and S1: the relative heritability of
311 collectives to cells is maximized when cellular stochastic variation is high, and
312 environmental heterogeneity low (lower right corner of the plots).

313 Our results differ from previous considerations of heritability in important
314 respects. For example, Queller (1992) presents a useful reformulation of the Price
315 equation for selection at two levels:

316 $$\Delta\overline{G} = S_b h_b^2 + S_w h_w^2,$$

317 in which $\Delta\overline{G}$ is the change in average trait value, S_b and S_w are the selection differentials
318 between groups and within groups, respectively, and h^2_b and h^2_w are the heritabilities of
319 the group-level and individual-level traits, respectively. This formulation partitions the
320 response to selection on a particle-level trait into within- and among-collective change,
321 but the focus is still on particle-level traits. Our focus is on the evolution of collective-
322 level traits. In the terminology of Damuth and Heisler (1988), our focus is on MLS2,
323 while Queller's is on MLS1. In addition, Queller makes no attempt to relate collective-
324 level heritability to particle-level heritability.

325 Michod and Roze (1997) have previously modeled the relationship between
326 particle-level and collective-level heritability of fitness during a major transition.
327 However, as Okasha (2006) points out, heritability of fitness only ensures that mean
328 population fitness will increase over time. For selection to result in directional phenotypic
329 change, it is phenotypes that must be heritable. Futhermore, Michod and Roze focused on
330 within-organism genetic change. Our models assume that such change is negligible, as is
331 likely to be true early in a transition, when collectives (*e.g.*, nascent multicellular
332 organisms) presumably include a small number of clonally-replicating particles (*e.g.*,
333 cells).

334 Okasha (2003) considers heritability in MLS1 (which he refers to as group
335 selection 2) and MLS2 (his group selection 1) but does not attempt to derive a
336 relationship between heritabilities at two levels. We have focused on just this
337 relationship, because knowing the ratio of heritabilities is necessary to predict the
338 outcome of opposing selection at two levels. This has important implications for
339 collective-level traits that arise from cooperation among particles. The presumed higher

heritability of the particle-level traits has been seen as a problem for the evolution of cooperation that benefits the collective (Buss 1986, 1987; Michod 1997; Michod & Roze 1997; Simpson 2011). Our results show that this problem does not always exist.

Several simplifying assumptions underlie our models, but these are not extreme departures from some biological systems. For example, the volvocine algae, an important model system for understanding major transitions, undergo clonal reproduction only occasionally punctuated by sex, are small enough that within-collective mutation is probably negligible, and have cell numbers that are under tight genetic control.

A great deal of work has gone into understanding the selective pressures that may have driven major evolutionary transitions. However, heritability is just as important as the strength of selection in predicting evolutionary outcomes. We have shown that, given some simplifying assumptions, heritability of collective-level traits comes 'for free'; that is, it emerges as an inevitable consequence of group formation. Understanding the emergence of trait heritability at higher levels is necessary to model any process involving multilevel selection, so our results are relevant to a variety of other problems.

Acknowledgments

This work was supported by grants from NASA (Cooperative Agreement Notice 7, Exobiology award #NNX15AR33G), NSF (DEB-1457701), and the John Templeton Foundation (43285). We would like to thank Sam Brown, Peter Conlin, Michael Doebeli, and Rick Michod for helpful discussions.

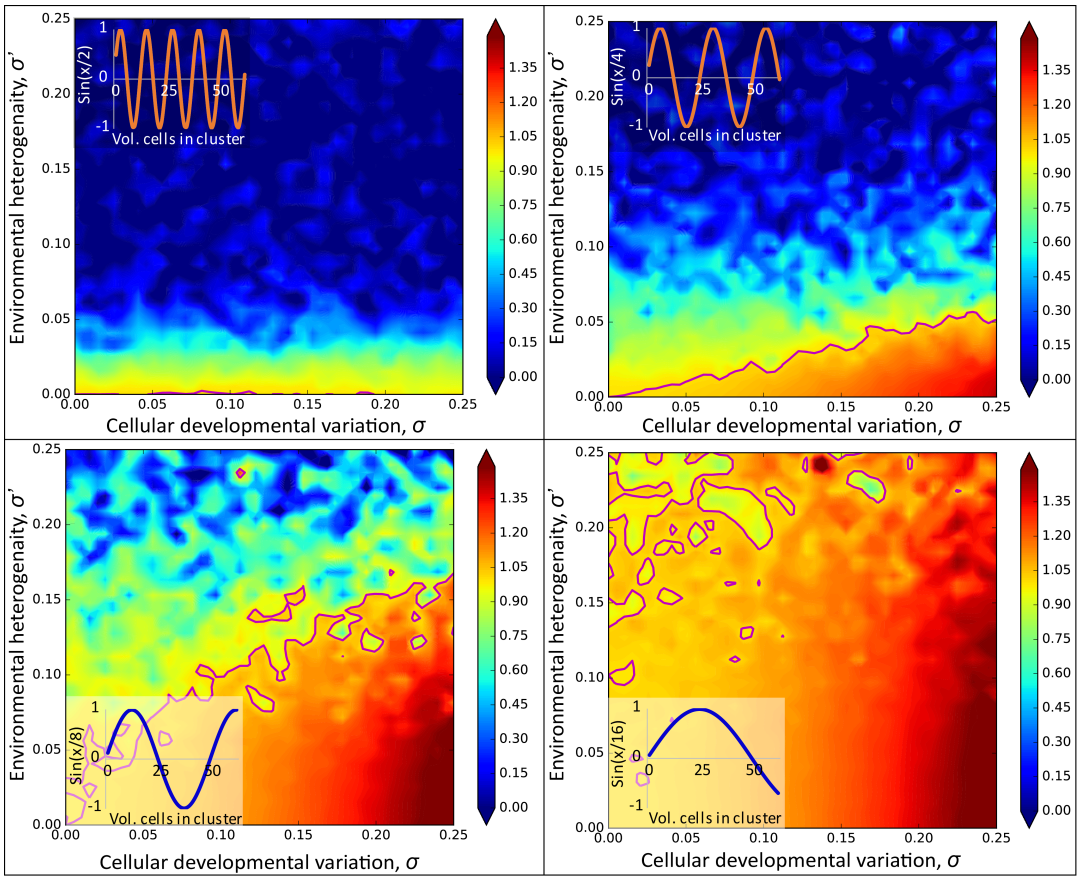

Figure S1. **Multicellular traits that are very sensitive to underlying cell-level traits are less heritable.** Here we vary the sensitivity of the multicellular trait to variation in collective volume (x), ranging from very sensitive (sin(x/2), upper left panel) to relatively insensitive (sin(x/16), lower right panel). Highly sensitive multicellular mapping functions exhibit reduced multicellular heritability relative to cell-level heritability for size. The colormap denotes collective heritability divided by cell-level heritability for size across 1024 x,y combinations. Pink line denotes relative heritability of 1. Populations were simulated for 7 generations.

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