Trait Heritability in Major Transitions

M. D. Herron[1,2] and W. C. Ratcliff[1]

[1] School of Biology, Georgia Institute of Technology. North Avenue, Atlanta, GA 30332.

[2] Correspondence: xprinceps@gmail.com, phone 520-820-6698, fax 406-243-4184

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 trait values are functions of lower-level trait values, 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 trait value in terms of particle-level trait values. When a collective-level trait value is a linear function of particle-level trait values, 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 trait values 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.

Keywords: Evolution; Heritability; Major Transitions; Multicellularity; Quantitative genetics; Simulations

Introduction

Major transitions, or evolutionary transitions in individuality, are a framework for understanding the origins of life's hierarchy and of biological complexity [1,2]. 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 [3,4]. 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 considers the emergence of collective-level heritability through conflict mediation a crucial step in major transitions [2,5,6]. 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 [7].

Major transitions can be conceptualized as a shift from MLS1 to MLS2, in the sense of Damuth and Heisler [4], as in Okasha [8] (see also Godfrey-Smith [9], Shelton & Michod [10]). In MLS1, properties of the particles are under selection; in MLS2, it is the properties of the collectives. We follow Okasha [8] in referring to the lower-level units in a transition 'particles' and the higher-level units 'collectives.' Although our biological analogies are presented in terms of cells as particles and multicellular

organisms as collectives, in principle our results should hold for any pair of adjacent levels.

According to Michod [5], "…the challenge of ETI [evolutionary transitions in individuality] theory is to explain how fitness at the group level in the sense of MLS2 emerges out of fitness at the group level in the sense of MLS1." But fitness, or selection, is only half of the breeder's equation. Predicting the response to selection requires an estimate of heritability.

Whether or not collective-level fitness in MLS2 is a function of particle-level fitness is a matter of some disagreement (for example, Rainey and Kerr say no [11]). However, collective-level phenotypes must be functions of particle-level trait phenotypes, unless we accept strong emergence, a philosophical position tantamount to mysticism [12]. The function may be complex and involve cell-cell communication, feedbacks, environmental influences, etc., but it is still a function that is, in principle, predictable from particle-level trait values. Nevertheless, the relationship between heritability of particle-level traits and that of collective-level traits has rarely been considered in the context of major transitions, leading Okasha [13] to wonder, "Does variance at the particle level necessarily give rise to variance at the collective level? Does the heritability of a collective character depend somehow on the heritability of particle characters? The literature on multi-level selection has rarely tackled these questions explicitly, but they are crucial."

We examine relationships between particle-level heritability and collective-level heritability for several functions that express collective-level trait values in terms of particle-level trait values. 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 trait values: the slope of the parent-offspring regression is an estimator of heritability [14]. 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 value is a linear function of the particle-level trait values. We further assume that reproduction is asexual, so the proper measure of heritability is broad-sense heritability, H^2 [15].

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 [15]. 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 value is the average of cell-level trait values, $z_{ij} = y_{ij\cdot}$, $\overline{z}_{i\cdot} = \overline{y}_{i\cdot\cdot}$, 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

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

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

Simulation model

The correspondence between particle-level and collective-level trait values is likely to be more complicated than a linear relationship for many interesting cases. Here we explore more complicated trait mapping functions using a simulation model. As above, particles grow in clonal collectives, which reproduce by forming two new collectives, each with as many particles as its parent. The initial population is founded by ten genetically distinct clones, each of which has a different genetically determined mean particle phenotype (spaced evenly between 1 and 2). These are grown for at least 7 generations, resulting in at least 127 collective-level reproductive events per genotype and 127*n* (where *n* is particle number per collective) particle-level reproductive events per genotype. Simulation models are provided as Electronic Supplements 1-3.

In this model, we consider two sources of non-genetic effects on particle phenotype (Figure 1), each of which should lower the heritability of both particle- and collective-level traits. The first is intrinsic particle reproductive stochasticity, analogous to developmental instability [16]. In the model, we determine the phenotype of 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.

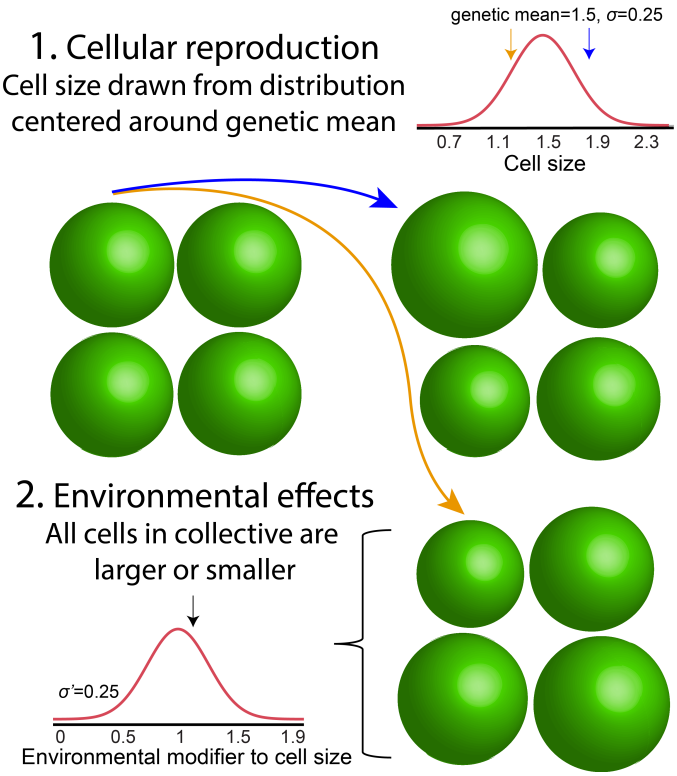

Figure 1. Two non-genetic modifiers to cell size. There are two nongenetic influences on cell size in our model: developmental instability, a stochastic effect that varies a cell's phenotype from its genetic mean size (with standard deviation σ), and environmental effects, which modify the size of all 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', to each collective. We then multiplied the phenotype of each particle within the collective by this modifier. This source of phenotypic heterogeneity should reduce the heritability of collectives more than particles, simply because collectives experience a relatively higher frequency of stochastic events than particles do (each collective gets assigned a different size multiplier, but every particle within that collective experiences the same size multiplier).

We examine the effect of each of the above sources of phenotypic variation independently for the example of cells (particles) within nascent multicellular organisms (collectives). For a linear relationship, collective size is simply the sum of the sizes of cells within the collective. For both cells and collectives, heritability is assessed by calculating the slope of a linear regression on parent and offspring phenotype [14]. In this simple case, mean collective-level heritability is always greater than or equal to cell-level heritability. Only when $\sigma = 0$ (*i.e.*, when all cells within a collective have identical phenotype) are cell- and collective-level heritability equal, in agreement with the analytical model. Greater developmental instability for cell size increases the advantage of collective-level heritability over cell-level heritability (Figure 2a). Larger collectives, which average out cellular stochasticity more effectively, experience a greater benefit than smaller collectives (Figure 2a). Note that the simulations run in Figure 2a reflect a very patchy environment in which environmental effects on cell size within collectives are large ($\sigma' = 0.25$). Increasing the magnitude of these environmental effects on cell size diminishes the difference in heritability between collectives and cells, but mean

collective-level heritability is still greater than cell-level heritability for all parameter combinations (Figure 2b).

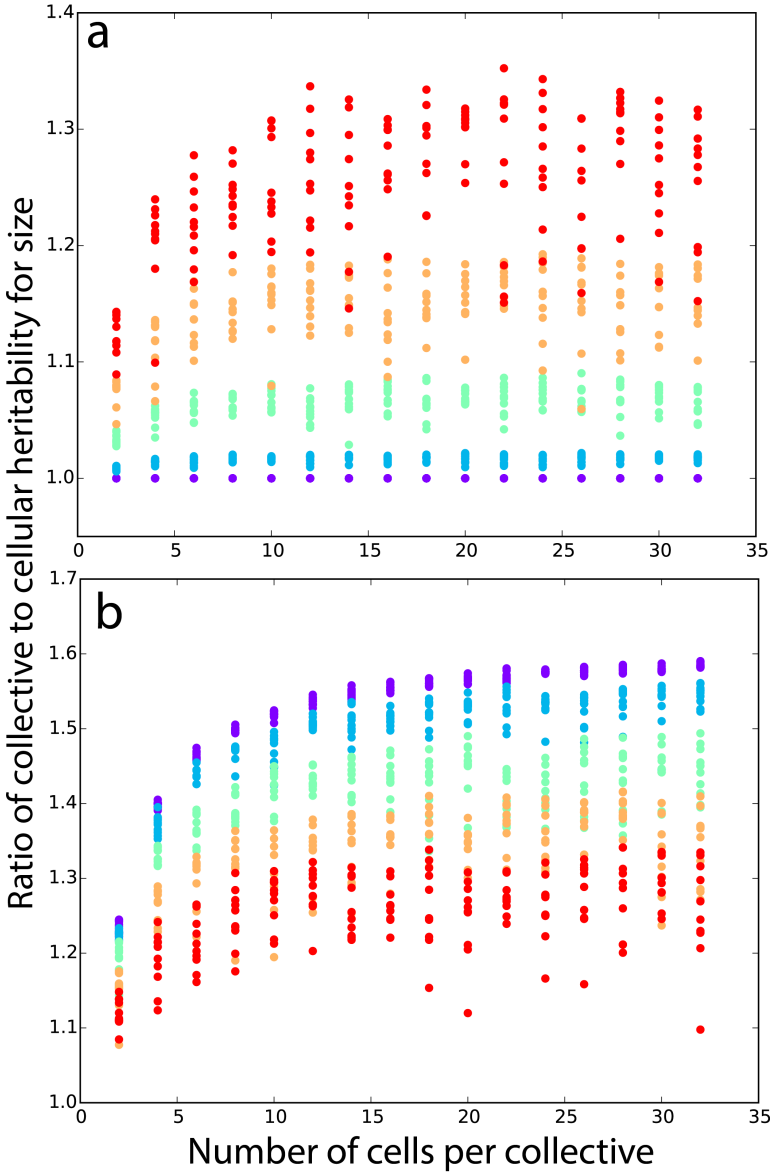

Figure 2. Collective-level heritability of size is greater than cell-level heritability for size. In **a**), we hold the effect of the environment fixed (standard deviation $\sigma' = 0.25$), and vary the degree of developmental instability σ: 10^{-4} (purple), 0.0625 (blue), 0.125 (green), 0.1875 (yellow), 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 trait values. We now consider more complicated nonlinear functions relating cellular to multicellular trait values, 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 [17,18]. 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').

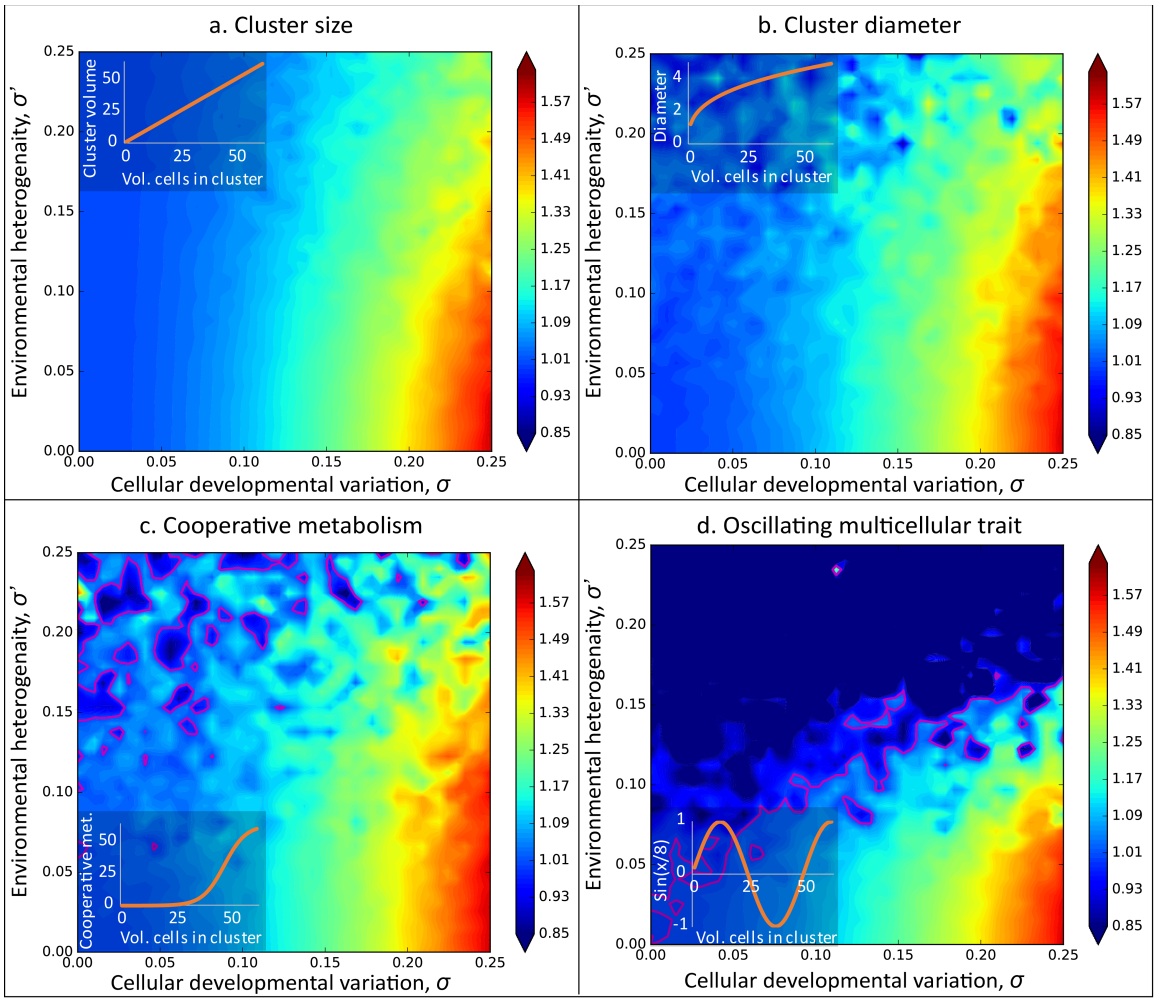

Figure 3. Relative heritability of various collective-level traits to cell-level heritability for size. Here we examine the heritability of four multicellular traits that depend on the size of their constituent cells, relative to cellular heritability for size. The relationship between the size of the 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.

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

Finally, we consider a collective-level trait that oscillates between -1 and 1 with increased cell size: $O = \sin(\frac{V}{8})$, where V is the sum of cell volumes. This trait has no obvious biological interpretation, but is distinct from the linear and nonlinear (but monotonically increasing) functions described previously. While the general relationship observed for these other functions still holds (collectives have greater heritability when cellular stochasticity is high and environmental heterogeneity low), we now find that much of the trait space now favors cellular heritability over that of the collective (Figure 3d, upper left). This appears to be due to the sensitivity of the function relating collective to particle-level trait values. We explore this further in Figure S4, where we consider four versions of the model presented in Figure 3d, varying the sensitivity of the collective's response to its lower-level composition. The heritability of collective-level traits is minimal when small differences in cellular phenotype generate radically different collective-level phenotypes. This makes sense: there is little potential for collective-level

heritability when tiny differences in cellular phenotype within collectives generate drastically different collective-level phenotypes.

Discussion

Using a quantitative genetics framework, we have derived an analytical solution for the relationship between particle-level and collective-level heritability for a limited case. When the organismal trait value is a linear function of the cell level trait values, the organismal heritability turns out to be a simple function of the cell-level heritability. In contrast to claims that particle-level heritability is always higher than collective-level heritability [e.g. ,7], we have shown that collective-level heritability is higher over a wide range of conditions.

This analytical result is a step toward understanding the relationship between heritabilities at two adjacent hierarchical levels, but the assumption that the collective-level trait value is a linear function of the particle-level trait values is restrictive. The simulation model shows that the results are dependent on the function relating the trait values at the two levels. Even under the conditions we model, which are favorable to collective-level heritability (clonal reproduction, negligible within-collective mutation, and fixed cell number per collective), collective-level heritability was not always higher than cell-level heritability (Figure 3d, Figure S4). Specifically, the relative heritability of collectives to cells was below 1 when the collective-level phenotype was extremely sensitive to small changes in the phenotype of cells within the collective (Figure S4). It is important to note, however, that we only saw this high sensitivity in a function with little biological relevance (collective-level phenotype oscillated between -1 and 1 with

increased cell volume). Collective-level heritability was higher than cell-level heritability for most of the trait space in the other three biologically significant functions we considered (Figure 3a-c).

In our simulation, we examined the effects of two independent sources of phenotypic variation affecting the relative heritability of particle and collective-level traits. Stochastic variation in cell size around the clone's genetic mean (σ) reduces the absolute heritability of cells and collectives by introducing non-heritable phenotypic variation. By averaging across multiple cells, however, collectives reduce the effects of this phenotypic variation, providing them with a relative heritability advantage over cells. We also considered the effect of environmental heterogeneity in which all of the cells within a collective are affected in the same manner (σ'). Collectives are disproportionately affected: each group is assessed a different size modifier, but all of the cells within these groups are affected in the same manner. As a result, collectives experience n-fold (where n is the number of cells per collective) more stochastic events, which reduces their heritability relative to cells. The influence of these sources of variation is evident in the contour plots of Figures 3 and S4: the relative heritability of collectives to cells is maximized when cellular stochastic variation is high, and environmental heterogeneity low (lower right corner of the plots).

Our results differ from previous considerations of heritability in important respects. For example, Queller [19] presents a useful reformulation of the Price equation for selection at two levels:

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

in which $\Delta\overline{G}$ is the change in average trait value, S_b and S_w are the selection differentials between groups and within groups, respectively, and h^2_b and h^2_w are the heritabilities of the group-level and individual-level traits, respectively. This formulation partitions the response to selection on a particle-level trait into within- and among-collective change, but the focus is still on particle-level traits. Our focus is on the evolution of collective-level traits. In the terminology of Damuth and Heisler [4], our focus is on MLS2, while Queller's is on MLS1. In addition, Queller makes no attempt to relate collective-level heritability to particle-level heritability.

Michod and Roze [2] have previously modeled the relationship between particle-level and collective-level heritability of fitness during a major transition. However, as Okasha [13] points out, heritability of fitness only ensures that mean population fitness will increase over time. For selection to result in directional phenotypic change, it is phenotypes that must be heritable. Futhermore, Michod and Roze focused on within-organism genetic change. Our models assume that such change is negligible, as is likely to be true early in a transition, when collectives (*e.g.*, nascent multicellular organisms) presumably include a small number of clonally-replicating particles (*e.g.*, cells).

Okasha [20] considers heritability in MLS1 (which he refers to as group selection 2) and MLS2 (his group selection 1) but does not attempt to derive a relationship between heritabilities at two levels. We have focused on just this relationship, because knowing the ratio of heritabilities is necessary to predict the outcome of opposing selection at two levels. This has important implications for 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 [2,7,21–23]. 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.

Conclusion

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, Rick Michod, and Deborah Shelton for helpful discussions.

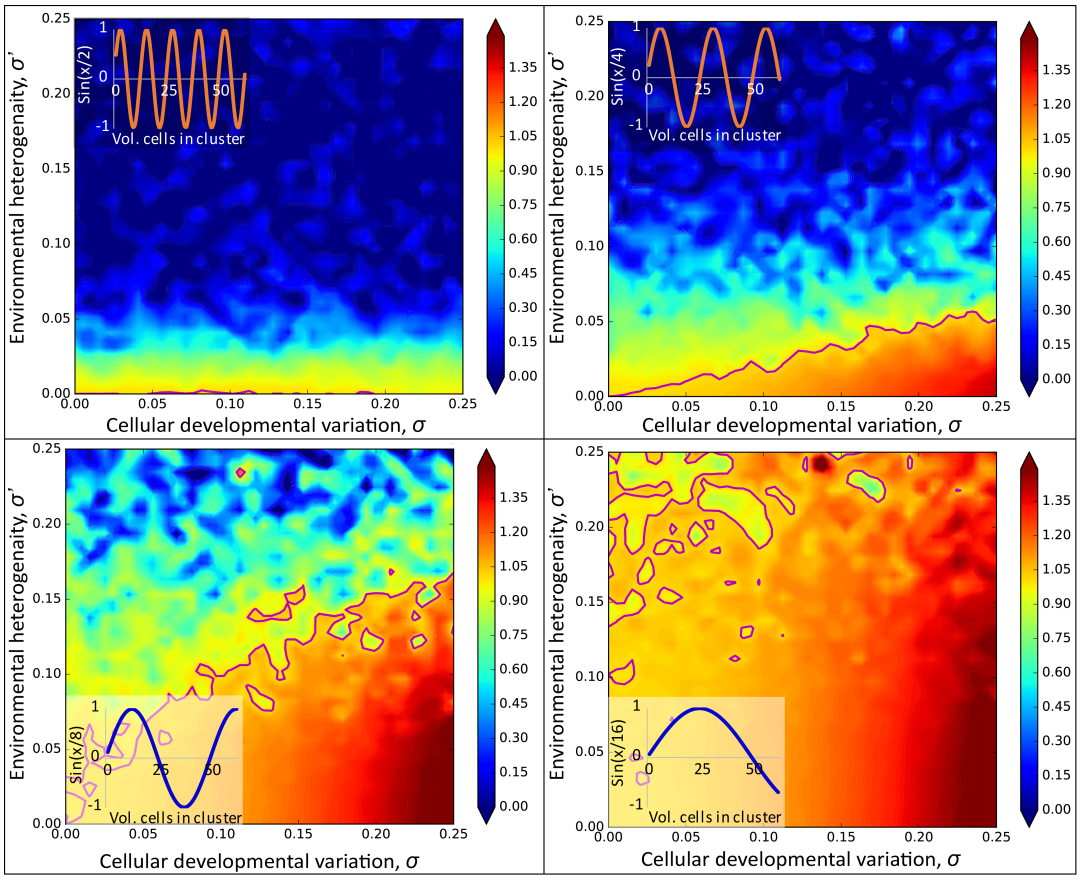

Figure S4. **Multicellular traits that are very sensitive to underlying cell-level traits are less heritable.** Here we vary the sensitivity of the multicellular trait to variation in collective volume (x), ranging from very sensitive (sin(x/2), upper left panel) to relatively insensitive (sin(x/16), 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.

References

[1] Maynard Smith J and Szathmáry E 1995 *The Major Transitions in Evolution* (Oxford: Oxford University Press)

[2] Michod R E and Roze D 1997 Transitions in individuality *Proc. Biol. Sci.* **264** 853–7

[3] Lewontin R C 1970 The units of selection *Annu. Rev. Ecol. Syst.* **1** 1–18

[4] Damuth J and Heisler I L 1988 Alternative formulations of multilevel selection *Biol. Philos.* **3** 407–30

[5] Michod R E 2005 On the transfer of fitness from the cell to the multicellular organism *Biol. Philos.* **20** 967–87

[6] Michod R E 2011 Evolutionary transitions in individuality: multicellularity and sex *The Major Transitions in Evolution Revisited* vol 2348, ed B Calcott and K Sterelny (Cambridge, MA: MIT Press) pp 167–97

[7] Simpson C 2011 How many levels are there? How insights from evolutionary transitions in individuality help measure the hierarchical complexity of life *The Major Transitions in Evolution Revisited* ed B Calcott and K Sterelny (Cambridge: The MIT Press) pp 199–225

[8] Okasha S 2005 Multilevel selection and the major transitions in evolution *Philos. Sci.* **72** 1013–25

[9] Godfrey-Smith P 2009 *Darwinian Populations and Natural Selection* (Oxford: Oxford University Press)

[10] Shelton D E and Michod R E 2009 Philosophical foundations for the hierarchy of life *Biol. Philos.* **25** 391–403

[11] Rainey P B and Kerr B 2011 Conflicts among levels of selection as fuel for the evolution of individuality *The Major Transitions in Evolution Revisited* ed B Calcott and K Sterelny (Cambridge: The MIT Press) pp 141–62

[12] Bedau M A 1997 Weak emergence *Noûs* **31** 375–9

[13] Okasha S 2006 *Evolution and the Levels of Selection* (Oxford: Oxford University Press)

[14] Falconer D S 1960 *Introduction to Quantitative Genetics* (New York: Ronald Press Co.)

[15] Lynch M and Walsh B 1998 *Genetics and Analysis of Quantitative Traits* (Sunderland, MA: Sinauer Associates, Inc.)

[16] Palmer A R 1994 Fluctuating asymmetry analyses: A primer *Developmental Instability: Its Origins and Evolutionary Implications* vol 93, ed T A Markow (Dordrecht, Netherlands: Kluwer) pp 335–64

[17] Stanley S M 1973 An ecological theory for the sudden origin of multicellular life in the Late Precambrian *Proc. Natl. Acad. Sci. USA* **70** 1486–9

[18] Bell G 1985 *The origin and early evolution of germ cells as illustrated by the Volvocales* ed H O Halvorson and A Monroy (New York: Alan R. Liss)

[19] Queller D C 1992 Quantitative genetics, inclusive fitness, and group selection *Am. Nat.* **139** 540–58

[20] Okasha S 2003 The concept of group heritability *Biol. Philos.* **18** 445–61

[21] Michod R E 1997 Evolution of the individual *Am. Nat.* **150 Suppl** S5–21

[22] Buss L W 1986 The uniqueness of the individual revisited *Population Biology and Evolution of Clonal Organisms* pp 467–505

[23] Buss L W 1987 *The Evolution of Individuality* (Princeton: Princeton University Press)