

Fluctuation in the physical environment as a mechanism for reinforcing evolutionary transitions

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Abstract

We hypothesize a mechanism for reinforcing transitions between levels of selection, involving physiological homeostasis and amplification of variation in the physical environment. Groups experience a stronger selection pressure than individuals for homeostasis with respect to reproductively limiting variables, because their greater longevity exposes them more often to suboptimal physical conditions, and greater physical size means they encompass a larger fraction of any resource/nutrient gradient. Groups achieve homeostasis by differentiation into microcosms with specialist functions, e.g. cell types. Such differentiation is more limited in individuals due to their smaller size and shorter lifespan. Hence tolerance of fluctuation in certain physical variables is proposed to be weaker in individuals than in groups. We show that a trait providing increased tolerance (α) to fluctuation ($V - V_{opt}$) in a limiting abiotic variable (V), at relative fitness cost (C), can increase from rarity if the condition $\alpha \cdot |V - V_{opt}| > C$ is met. Groups also sequester larger absolute quantities of resource than individuals, and group death is less frequent, hence the population dynamics of groups cause resource/nutrient availability to fluctuate with greater amplitude than that of individuals. Increasing the amplitude of fluctuation in a reproductively limiting environmental variable is proposed as a mechanism by which a group can limit reproduction of parasitic “cheat” individuals. Enhancing physical fluctuation is frequency dependent, hence only an increase in tolerance to fluctuation can explain the group’s increase from rarity. However, once groups reach intermediate frequencies, a positive feedback process can be initiated in which a differentiated group enhances physical fluctuation beyond the tolerance of any “cheat”, and in so doing enhances the selection pressure it experiences for homeostasis. This may help explain the persistence of transitions in individuality, and the coincidence of some such transitions with periods of change and oscillation in global scale environmental variables.

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1. Introduction

During Earth history there have been several qualitative re-organizations in the way that some lineages transfer genetic information between generations. These “evolutionary transitions” occur when natural selection starts to act on a spatially larger group composed of ancestrally autonomous individuals (Szathmary, 1989; Maynard-Smith and Szathmary, 1995). This principle holds at spatial scales from hypercycles (Maynard-Smith, 1983) through multicellular organisms to kin groups (Hamilton, 1964).

The group units of selection that result are prone to parasitism by “cheat” individuals that evade the reproductive cost of group membership by increasing their own short-term fitness, to the detriment of the group. Social group structures are thought to persist because factors such as an appropriate balance between local competition and global dispersal, as well as internal “policing” mechanisms (Michod, 1997), ensure that kin selection exceeds kin competition. However, the balance between these processes remains sensitive to prevailing physical conditions and ecological context. This work is concerned with explaining the persistence of evolutionary transitions after their initial occurrence. We argue that groups are able to replicate under fluctuation in an essential environmental variable of an amplitude that curtails replication of any single

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autonomous or parasitic individual. Actively increasing the amplitude of fluctuation in such physical variables is suggested as a viable “policing” strategy for the group to limit reproduction of “cheats”. In this regard, we note that fluctuating physical conditions probably coincided with evolutionary transitions—cycles of saturation and desiccation may have been necessary to concentrate reactants during both the origin of a nucleic acid genome, and even for the emergence of homeostatic systems such as the Oklo reactor (Maynard-Smith, 1983; Maynard-Smith and Szathmari, 1995).

The key elements of the hypothesis are as follows. For a given individual, being part of a coordinated group increases the range of its tolerance to deviation of one or a number of essential physical variables from optimum (relative to the tolerance range of an individual with an autonomous life history). This results from the evolution of a group-level physiology that is able to “buffer” the fluctuation that individuals within a group experience relative to autonomous individuals, by functions such as storage, transport or detoxification. Such a physiology requires a degree of homeostasis that a system can only evolve by differentiation into a number of microcosms, each of which perform a separate function. This level of differentiation is, in turn, only possible or adaptive at the greater spatial and temporal scales over which the group reproduces. Of course, individuals are composed of microcosms just as are groups; a cell consists of distinct classes of organelles just as a multicellular organism consists of distinct organs. But the potential for diversification is greater in a group than an individual—because its larger size permits an increased range of microcosms to develop, and because the capacity for autonomous physiological function is greater in the individuals that make up a group than in the component parts (be they organs or organelles) that make up those individuals. Therefore, the potential the group has to protect its component parts from external fluctuation by maintaining internal homeostasis is greater than that of any one individual, making its tolerance within the range over which the variable fluctuates similarly greater. Hence, while extreme directional changes in the abiotic environment may be tolerable to individual parasitic genotypes, or even enhance selection for them, in a fluctuating environment no single “cheat” genotype will be able to maintain a sufficient relative frequency to invade the group, because they lack a homeostatic property (with respect to the given variable) that the group is able to achieve. Prokaryotic species may be extremophilic in specified directions, but no one bacterium encompasses the range of chemical microcosms present in a multicellular eukaryote.

The hypothesis is justified on grounds of size, time-scale, and likely optimal strategies for minimizing kin competition. By definition, groups reproduce more slowly and are larger than the autonomous individuals from which they are descended. The greater longevity of groups means they are likely to encounter greater temporal variation in

limiting resources or other deviation from physiologically optimal conditions, (for example as a consequence of seasonality or fluctuation in weather conditions), than that experienced by more short-lived autonomous individuals. Additionally, because of their larger size, groups will be more likely to experience spatial heterogeneity and will encompass larger gradients in a given resource or nutrient than will individuals.

Such factors will provide a selection pressure, greater than that experienced at the individual level, for the sequestration of resources for periods of sub-optimality and the creation of microcosms for processing toxins, a pressure dealt with by spatial/temporal differentiation. Furthermore, differentiation into distinct niches will be adaptive for competitively inferior individual genotypes and beneficial for the group because it will decrease kin competition. Therefore, regardless of whether early evolution of the group is dictated by kin selection, or (as apparent to a degree in some kin-selected traits, (Griffin and West, 2002)) by adaptation of group-forming individuals that retain a degree of their ancestral autonomy, differentiation into microcosms is likely.

1.1. Increased capacity for physiological homeostasis in groups

The same environmental factors that create the selection pressure for diversification into microcosms will also make it easier for such features to evolve. Separate bacterial microcosms and morphs can arise solely as a consequence of individual selection and spatial heterogeneity (Rainey and Travisano, 1998). Therefore compartmentalization, a prerequisite for evolutionary transitions, may predispose the system to phenotypic divergence, and perhaps, as a result, physiological robustness with respect to limiting variables. Increased cellular compartmentalization in eukaryotes relative to prokaryotes, diversification into cell types after the transition to multicellularity, formation of reproductive/worker castes in social insects, and divergence into sterile and fertile, dominant and subordinate individuals within kin groups, are suggested as examples of this pattern. In each of these cases, the group survives across a greater range of physical conditions than any one of the individuals of which it consists.

To illustrate the hypothesis, consider a tumour growing within a microenvironment inside a multicellular system. The tumourigenic “cheat” cells reproduce within this microenvironment by depriving neighbouring cells of nutrients, respiratory substrate, oxygen, etc., hence impose a cost on the group. Suppose also that an essential resource starts to fluctuate beyond the tolerable range within this microenvironment. In some instances cooperative cells will die as a consequence of this fluctuation just as will tumour cells. But if the fluctuation can be buffered by active transport from or to an organ containing a store of the resource elsewhere in the multicellular system, the impact of the fluctuation will be less dramatic. In some

circumstances the tumour will benefit from this buffering mechanism just as much as the cooperative cells, and one could say that homeostasis in the resource in question is a trait that has been successfully parasitized. However, one way in which tumour cells differ from those of the rest of the body is via cell junction formation, a process likely to play a role in the acquisition of malignancy (e.g. Yamasaki et al., 1999). If the buffering process requires a signalling mechanism that functions through cell junctions absent in the tumour, the whole tumour will die or experience dramatically curtailed growth if the fluctuation continues. Although individual tumourigenic genotypes may have been able to adapt to extreme levels of resource were the change directional, no single cell within the tumour will possess the capacity to be coupled to the organ performing the buffering function, so no single tumour genotype will be able to cope.

The essence of our hypothesis is that evolutionary transitions in individuality result in properties analogous to such cell junctions, properties that cannot be parasitized by “cheat” individuals, regardless of their frequency within the group. The capacity to form cell junctions and achieve physiological co-ordination with neighbouring cells may thus be more than merely a metaphor for such a trait. Similarly, a mechanism for the multicellular system to bias the environment so that fluctuation is enhanced bears a parallel to the administering of chemotherapeutic drugs in discrete doses in order to medically treat such tumours, in that the wild type cells have a greater capacity for recovery.

The ability of social insect colonies and social groups of mammals to sequester a range of resources in quantities that no individual could achieve, or hunt a variety of prey unavailable to any one individual are similar examples. In some instances the benefits from these properties are available to “cheats”. But if a check on group membership or an analogous property exists that restricts the range of group properties from which the “cheat” can benefit, then the group will be able to survive should one resource/prey suddenly fluctuate deleteriously, whereas any one specialist individual will be unable to exploit those properties of the group that derive from individuals with different capabilities to itself.

Finally, whilst symbioses may not be examples of group formation in the strict sense, they do constitute a pooling of genetic resources and a loss of reproductive autonomy by each partner, therefore they are arguably a transition between levels of selection. In a symbiosis in which each partner contributes a resource at levels the other is unable to obtain, a critical stoichiometry between the nutrients provided by each partner is likely to be vital for successful function of the mutualism (Sterner and Elser, 2002). An adaptive nutrient stoichiometry may only be achievable in a fluctuating environment if both partners are sufficiently mutualistic. In a lichen, for example, the C:P ratio must be high enough to meet the needs of the mycobiont (i.e. there must be enough reduced carbon available), but not so high that photobiont growth becomes P limited. Lichens, like all

symbioses, exist on a genetic continuum between mutualism and parasitism (Van Baalen and Jansen, 2001). Imagine a “cheat” fungal genotype that evolves to wholly or partially restrict the supply of P (obtained by the fungus) to its photobiont. Should the steady-state abiotic level of one of the nutrients suddenly drop, the “cheat” mycobiont genotype will be less likely to adequately compensate for the change by elevating supply to its photobionts than will a more mutualistic fungal genotype with a higher initial P flux to its partner; hence more mutualistic genotypes will be selected for. This idea could apply equally under a sudden excessive increase in P (or N), in which cases symbioses with “cheat” photobiont genotypes partially restricting carbohydrate supply to the fungus would be selected against, because the fungus would approach carbon limitation as its growth rate responded to the sudden glut of inorganic nutrient. Dynamics of this sort will affect the continuum of any symbiosis subject to fluctuating resource availability. There exists an adequate “window” of mutualism within which the system is able to compensate for fluctuating resource availability, and should either partner evolve too far toward the “cheat” end of the continuum, the symbiosis will become maladaptive in the presence of such fluctuation.

1.2. Enhancement of fluctuation in physical variables by groups

Evolutionary transitions in individuality are also proposed to correspond to significant changes in the impact that life has on the physical environment, such that the amplitude of fluctuation in certain limiting physical variables is increased as a function of the group’s abundance. Life will have a stronger influence upon its environment when the biota are confined to a microcosm, in which uptake of resources/nutrients and excretion of waste products will be concentrated in a small space. Given that compartmentalization of living material into such microcosms was probably necessary both for successful coevolution of a large, coordinated (RNA) hypercycle, and for concentration of metabolic reactants within protocells (Eigen, 1971; Bresch et al., 1980; Maynard-Smith and Szathmary, 1995), it seems reasonable to assume that a consequence of some transitions in individuality was a significant increase in the impact of the post-transition group on the (micro)environment in which it evolved, when compared with pre-transition autonomous individuals. This idea may be understood in a simple generic sense based on arguments of spatial and temporal scale, and on negative feedback from the physical environment. The group’s larger size means it displaces a greater absolute quantity of any given physical resource or nutrient. Similarly, because the group survives longer than any given individual, the release of such sequestered resources upon group death will be less frequent. Therefore, the population dynamics of groups causes more dramatic fluctuation of such resources/nutrients than that of

individuals, with a more peaked distribution of availability in time and space. If the “cheat” life habit experiences the same physical environment as the group (e.g. if it is a cell growing on the exterior of the multicellular system it is parasitizing), it may be unable to tolerate fluctuations in the availability of essential substances—fluctuations that are non-fatal to the group due to storage/sequestration functions afforded it by its larger size. For example, the origin of swarming and other social behaviour in insects may have resulted in a sudden increase in resource sequestration per individual, relative to an autonomous life history. Upon death of the colony, release of these resources will be more concentrated in time and space than would the release of an equivalent quantity of resource by the same number of individuals living independently, hence will cause resource fluctuation of an increased amplitude. This argument might just as well apply to a comparison between a unicellular and a multicellular system.

A further reason to expect the group to elevate the amplitude of physical change, as opposed to causing a dramatic unidirectional change, involves negative feedback from the physical environment (Lenton, 1998). If the change induced by a group is directional, but triggers a response in the physical environment that counteracts this change and therefore makes the environment more suited to “cheat” reproduction, then the system will oscillate as group numbers rise and fall. This process is thought to have occurred following colonisation of the land by vascular plants. This required a symbiosis with mycorrhizal fungi (e.g. Wilkinson, 2001), a form of transition in individuality. Once vascular plants achieved terrestrial abundance, a dramatic decrease in atmospheric carbon dioxide was induced, due to increased silicate weathering caused by the plants (Berner, 1990; Schwartzman, 1999; Lenton et al., 2004). This caused a decrease in temperature, which in turn decreased biological productivity. It is plausible that repeated cycling through this negative feedback loop would lead to fluctuation in the availability of a variety of nutrients, as a result of the impact of temperature on physical weathering processes and the activity of the hydrological cycle over geologic time (Lenton and Watson, 2000).

Fig. 1 shows a schematic of the hypothesized mechanism. Suppose that any dramatic physical change elicited by the post-transition biological system is countered by a negative feedback process within the physical environment (for example, the sequence: “increased vascular plants → increased physical weathering → decreased CO₂ → decreased temperature → decreased vascular plants” described above). If this occurs, any sufficiently sudden and dramatic biologically triggered physical change will lead to an oscillation, as the environmental effect of that change feeds back on the biological cause (e.g. as above—and within appropriate limits—“decreased vascular plants → decreased weathering → increased CO₂ → increased vascular plant productivity”). All biologically significant

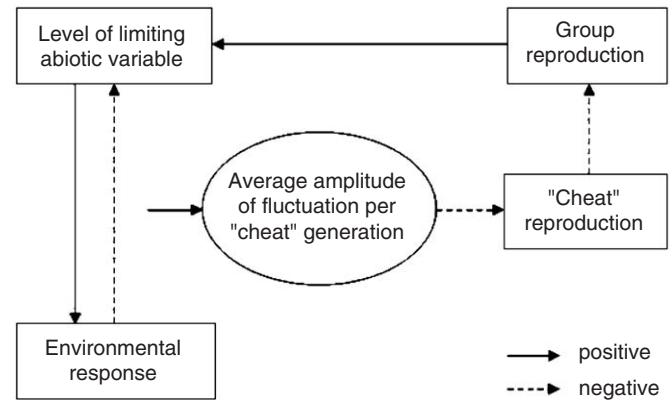


Fig. 1. A directional biologically elicited physical change in a given variable can be converted into an oscillation in conjunction with a negative feedback process in the physical climate that acts on that variable. Boxes represent the magnitude of the processes shown. Arrows represent positive or negative influence according to key.

nutrients and resources are influenced by such negative feedback processes (e.g. Schwartzman, 1999).

2. A simple model

Consider a population of a theoretical haploid, asexual, group-forming species, the reproduction of which is limited by an essential environmental variable, V . V fluctuates over time about the species' optimum, V_{opt} . Let the fitness of a gene within a “cooperative” individual that contributes to the group be F_g . F_g is determined by the replication fidelity R , the (kin-selected) advantage of group membership relative to an autonomous life history β , and the reproductive cost of group membership, C . Within the group there are “cheat” individuals that parasitize any given group trait, hence experience β without incurring C . Replication of both the group as a whole, and of parasitic individuals, is decreased by a factor of the deviation of V from V_{opt} . Positive and negative deviations from this optimum are assumed to be equally deleterious.

The relative fitness of a gene in a cooperative individual is

$$F_g = \beta \cdot R \cdot (\lambda - (C + m + |V - V_{opt}|)), \quad (1)$$

where λ gives the species' birth rate and m the mortality rate when $V = V_{opt}$ and there is no competition or density dependence. F_g thus gives the geometric rate of increase per generation. “cheat” individuals of genotype P lack the cost of group membership but share the same basal fecundity λ and mortality m :

$$F_p = \beta \cdot R \cdot (\lambda - (m + |V - V_{opt}|)). \quad (2)$$

If membership or non-membership of the group has no bearing on the response to fluctuation in V , then parasitic genotypes will increase in frequency by a factor of C each generation, β , the only beneficial consequence of the transition, being fully available to “cheats”. However,

suppose that an additional consequence of the transition is greater tolerance α of deviation of V from optimum, incurring a greater cost C , but dampening the physiological impact of fluctuation in the physical variable. In this case the fitness of a co operative genotype becomes:

$$F_g = \beta \cdot R \cdot (\lambda - (m + C + (1 - \alpha) \cdot |V - V_{opt}|)). \quad (3)$$

Whether or not such a trait is adaptive is a function of the proportion of the time that $V \neq V_{opt}$.

Because the β , R , m and λ terms are the same for both genotypes, the condition for the cooperative genotype to have higher relative fitness is dictated only by the tolerance of fluctuation, magnitude of fluctuation experienced, and relative fitness cost of the extra tolerance. Hence for: $F_g > F_p$

$$\alpha \cdot |V - V_{opt}| > C. \quad (4)$$

Expressing the condition that if α , the increased tolerance to deleterious fluctuation in V that the group has relative to “cheat” individuals, exceeds the relative fitness cost of this decreased sensitivity, then the group will have higher relative fitness if the environment is sufficiently variable. Genotype P might be considered K -selected on variable V , within the window $V = V_{opt}$, whereas genotype G is loosely analogous to an r -selected “generalist” with respect to variable V , but one that possesses the ability to destroy any specialist niches that arise, by making the environment more variable.

It is perhaps improbable that the environment will vary just enough to limit cheat reproduction but not so much as to be outside the habitable window for the group. However, should a further consequence of an evolutionary transition be elevated fluctuation in the physical environment, a positive feedback process may be initiated, by which (within limits) the group makes the environment more variable, and benefits from this variability by the decreased reproduction of “cheat” individuals. In some circumstances, the enhanced tolerance will be parasitized by cheats; this process will apply only in situations where the cheat genotype is incapable of achieving this extra homeostatic ability regardless of its frequency within the group. Once the fluctuation reaches such a level as to cause condition (4) to no longer hold, group reproduction will decline, before the group eventually self-limits through the variability it is able to induce.

We encompass extra deaths (i.e. above m) as a result of deleterious fluctuation in V , within the fitness term by allowing negative fitness values. The absolute frequencies of the group-forming genotype G and parasitic “cheat” genotype P are given by:

$$G_t = G_0 \cdot F_g^t, \quad (5)$$

$$P_t = P_0 \cdot F_p^t, \quad (6)$$

where subscripts denote time t and time zero generations as shown. Differentiating Eqs. (5) and (6), and constraining

total population size by carrying capacity K :

$$\frac{dG}{dt} = G_0 \cdot F_g^t \cdot \ln(F_g) \cdot \frac{dF_g}{dt} \cdot \left(1 - \frac{G + P}{K}\right), \quad (7)$$

$$\frac{dP}{dt} = P_0 \cdot F_p^t \cdot \ln(F_p) \cdot \frac{dF_p}{dt} \cdot \left(1 - \frac{G + P}{K}\right). \quad (8)$$

The only determinant of the relative abundance of each genotype that varies systematically with time is the impact that fluctuation in V has on the fitness terms:

$$\frac{dF_g}{dt} = -\beta \cdot R \cdot (1 - \alpha) \cdot \frac{dV}{dt}, \quad (9)$$

$$\frac{dF_p}{dt} = -\beta \cdot R \cdot \frac{dV}{dt}. \quad (10)$$

Suppose now that V oscillates about V_{opt} with an entirely abiotically determined amplitude τ according to:

$$V = V_{opt} + \tau \cdot \sin\left(\frac{\Pi}{2} + t\right), \quad (11)$$

where t is a time step of length one generation. If the group alters the physical environment such that the amplitude of the oscillation exceeds τ as a function of the group’s abundance, then Eq. (11) becomes:

$$V = V_{opt} + \tau \cdot \sin\left(\frac{\Pi}{2} + t\right) \cdot (1 + \phi \cdot G), \quad (12)$$

where ϕ is the effect on V per individual of genotype G , so that:

$$\frac{\delta V}{\delta G} = \tau \cdot \sin\left(\frac{\Pi}{2} + t\right) \cdot \phi. \quad (13)$$

Differentiating Eq. (12):

$$\begin{aligned} \frac{dV}{dt} &= \tau \cdot \cos\left(\frac{\Pi}{2} + t\right) \cdot (1 + \phi \cdot G) \\ &+ \tau \cdot \sin\left(\frac{\Pi}{2} + t\right) \cdot \phi \cdot \left(\frac{dG}{dt}\right). \end{aligned} \quad (14)$$

But as $G \rightarrow 0$, (i.e. when the “cooperative” genotype is at rarity), the G term in Eq. (12) disappears, hence:

$$\frac{dV}{dt} \rightarrow \tau \cdot \cos\left(\frac{\Pi}{2} + t\right). \quad (15)$$

For the group-forming genotype G to invade a population of potential “cheat” individuals that will parasitize it should it increase from rarity:

$$\frac{dG}{dt} > \frac{dP}{dt}.$$

Because V is the only part of the system varying systematically over time, this is equivalent to:

$$\frac{dG}{dF_g} \cdot \frac{dF_g}{dV} \cdot \frac{dV}{dt} > \frac{dP}{dt} \cdot \frac{dV}{dF_p} \cdot \frac{dF_p}{dV} \cdot \frac{dV}{dt}. \quad (16)$$

Substituting terms, re-arranging and simplifying gives:

$$(1 - \alpha) < \frac{P_0 \cdot F_p' \cdot \ln(F_p)}{G_0 \cdot F_g' \cdot \ln(F_g)} \quad (17)$$

Describing a necessary condition for the frequency of the cooperative genotype to increase from rarity: the detrimental effect of fluctuation in V on genotype G must be less negative than the genotype's demographic inferiority in the population. Therefore, relative physiological robustness and demography alone must be sufficient to cause G to reach a frequency at which it can influence V via the positive feedback process (i.e. $\delta V/\delta G$) described above. After this point, if genotype G is to approach fixation:

$$\frac{\delta G}{\delta|V - V_{opt}|} > \frac{\delta P}{\delta|V - V_{opt}|} \cdot \frac{\delta|V - V_{opt}|}{\delta G} \quad (18)$$

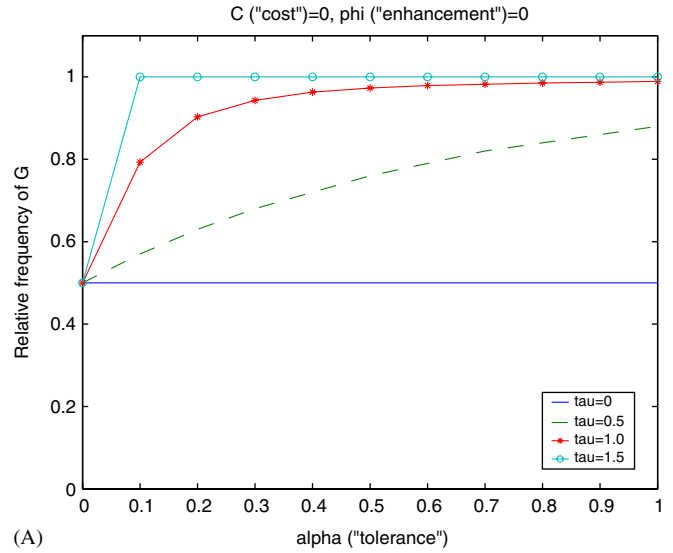
(noting that the partial derivatives of both genotypes with respect to $|V - V_{opt}|$ will be negative).

As absolute growth rate slows whilst the system approaches carrying capacity, genotype G will continue to increase in relative frequency as extra deaths due to deviation in V occur disproportionately in genotype P .

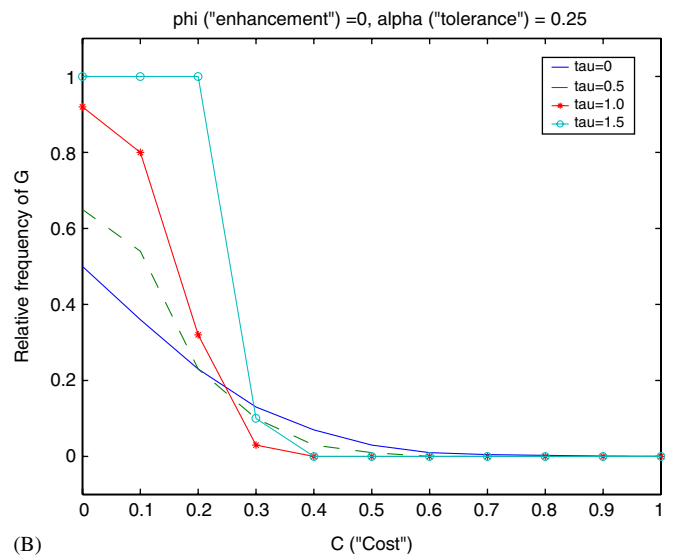
Finally, if suboptimality in V causes extra deaths, then for G to go to fixation (and where $\Sigma = G + P$)

$$\frac{dP/dt}{dG/dt} < \frac{\delta \Sigma}{\delta|V - V_{opt}|} \cdot \frac{\delta|V - V_{opt}|}{\delta G} \quad (19)$$

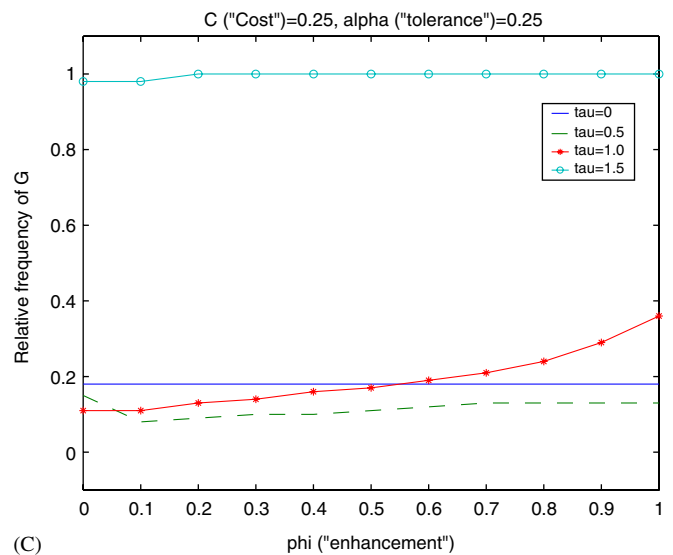
The above inequality implies that genotype G goes to fixation before the entire population goes extinct due to the deleterious fluctuation in V (note that the partial derivative of total population size will be negative, as will the full derivative dP/dt under conditions in which G is amplifying fluctuation). This situation will persist until G self-limits, by reaching an absolute frequency sufficient to drive $|V - V_{opt}|$ high enough that relation (4) no longer holds. In simplifying Eq. (18) all terms cancel except α , meaning that if the group reaches a frequency at which it can influence the environment, the frequency of parasitic P individuals will be driven down until G self-limits, a positive value of tolerance α being the only proviso. If the absolute frequency of G required for self-limitation through V is higher than the carrying capacity K then G will go to fixation and P will go extinct, if not then Eq. (19) will not be realised and an equilibrium will be reached at which growth of both genotypes is limited by the degree of suboptimality in V .



(A)



(B)



(C)

Fig. 2. The impact of each parameter on the relative frequency of the group-forming G genotype after 100 generations in environments of different τ (baseline abiotic variability). Starting relative frequencies of 0.5 and absolute frequencies of 1, for both genotypes. (A) Impact of α (tolerance to deviation of V from optimum), $C = 0$, $\phi = 0$. (B) Impact of C (cost of group membership), $\alpha = 0.25$, $\phi = 0$. (C) Impact of ϕ , (enhancement of fluctuation in V per individual of genotype G). $C = 0.25$, $\alpha = 0.25$.

Table 1

Constant	Value
β	1
R	1
K	1000
m	0.1
λ	1

3. Results

3.1. Sensitivity analysis

Fig. 2 explores the impact of each biotic parameter on the relative viability of the two genotypes, within environments in which V fluctuates at different abiotic amplitudes τ . The values chosen for other constants are given in Table 1. All plots show the results of different simulations (one per datapoint) over 100 generations, from an initial population of one individual of each genotype. Fig. 2A shows how the value of tolerance α (X -axis) impacts upon the relative frequency of the G (group-forming) genotype (Y -axis), with cost C and enhancement of environmental fluctuation ϕ set to zero. The various curves differ in the magnitude of τ , the amplitude of abiotic fluctuation in V . Increasing α causes incremental increases in G 's relative frequency with a gradient dictated by the amplitude of abiotic variability τ . Because C is zero, both fitness functions become identical when τ is zero, and relative frequencies remain at their starting values. Fig. 2B shows how increased cost C decreases the relative frequency of G with tolerance α fixed (at 0.25) with gradient again determined by the value of τ . Fig. 2C shows equivalent results but for $C = 0.25$, $\alpha = 0.25$, and variable magnitudes of ϕ , the increase in fluctuation in V caused per individual of genotype G . The influence of this parameter is highly sensitive to abiotic variability τ . At $\tau = 0.5$ increased enhancement ϕ actually has a slight negative impact on the relative frequency of G . At this tolerance α value, the increase in fitness G incurs as a result of extra variability is insufficient to overcome its inferior growth rate. This results from demographic changes due to the logistic formulation of the model (discussed below). At higher values of abiotic variation τ , enhancement ϕ incrementally increases the relative fitness of G , but as is evident from the plots, G is much more sensitive to α and C than to ϕ . As might be expected intuitively, ϕ is most influential in the dynamics of G at intermediate values of τ , because this is when the relative frequencies of both genotypes are most sensitive to $|V - V_{opt}|$.

3.2. Environmental variability and absolute population size

The mechanism being proposed includes scenarios where genotype G induces a change deleterious both to itself and to genotype P , and increases in relative frequency because

its superior tolerance means its absolute frequency declines more slowly than that of the “cheat” genotype P . If this involves extra deaths it presupposes a decrease in absolute population size. If it involves changes in birth rates then it presupposes the total population taking longer to reach carrying capacity. The process is therefore potentially highly sensitive to founder effects and absolute population size.

Fig. 3A shows the relative frequency of G for different values of the abiotic amplitude of fluctuation τ , again after 100 generations from starting frequencies of one individual per genotype. The curves differ in the value of ϕ , the increase in fluctuation induced per G individual. As fluctuation τ becomes larger, the relative frequency of G increases, with a gradient dictated by ϕ . Fig. 3B shows results from the same simulations, but plots the total population size $\Sigma = G + P$. Comparison of Figs. 3A and B shows how at this combination of α and C values, if G is to approach fixation, $|V - V_{opt}|$ must be so great as to effectively drive the population extinct. When α takes larger values, and/or C lower values, this need not happen (below). This illustrates how amplifying environmental variation in a deleterious direction is a risky strategy, requiring a substantial tolerance advantage in order to be adaptive. This figure thus emphasizes why a strategy of the sort described is only realistically likely to be associated with evolutionary transitions: incremental evolution through these parameter values is not possible. A dramatic and sudden increase in physiological tolerance is a prerequisite for using the environment as a policing strategy in this way. As we have argued above, the origin of multicellularity and of social groups both fulfil this criterion.

Finally, Fig. 3C relates the model's results to the prediction made by relation (4) showing the relation between cost C and $\alpha \cdot |V - V_{opt}|$ over a series of simulations in which $\alpha = 0.6$, $\tau = 1$, $\phi = 0.01$. $\alpha \cdot |V - V_{opt}| = C$ at the point where the two curves meet, and condition (4) is realised as an inequality to the right of this point. Note that the high sensitivity to the magnitude of the cost parameter C manifests as a strong non-linearity; G either rapidly goes to unity at permissive C values, or to zero once C is too high. That is, for fixed values of tolerance α , enhancement ϕ and abiotic variability τ , the relative frequency of genotype G approaches either zero or extinction, depending on whether or not condition (4) is realised (and provided enhancement and abiotic amplitude are both non-zero and within the permissive ranges identified in Figs. 2C and 3A).

3.3. Invasibility

Fig. 4 compares the results of single 100 generation simulations, to test the criteria identified above for group genotype G to invade, within a more realistic context in which a single G individual arises in a population of 99 P individuals, where cost $C = 0.2$, and tolerance $\alpha = 0.7$. In

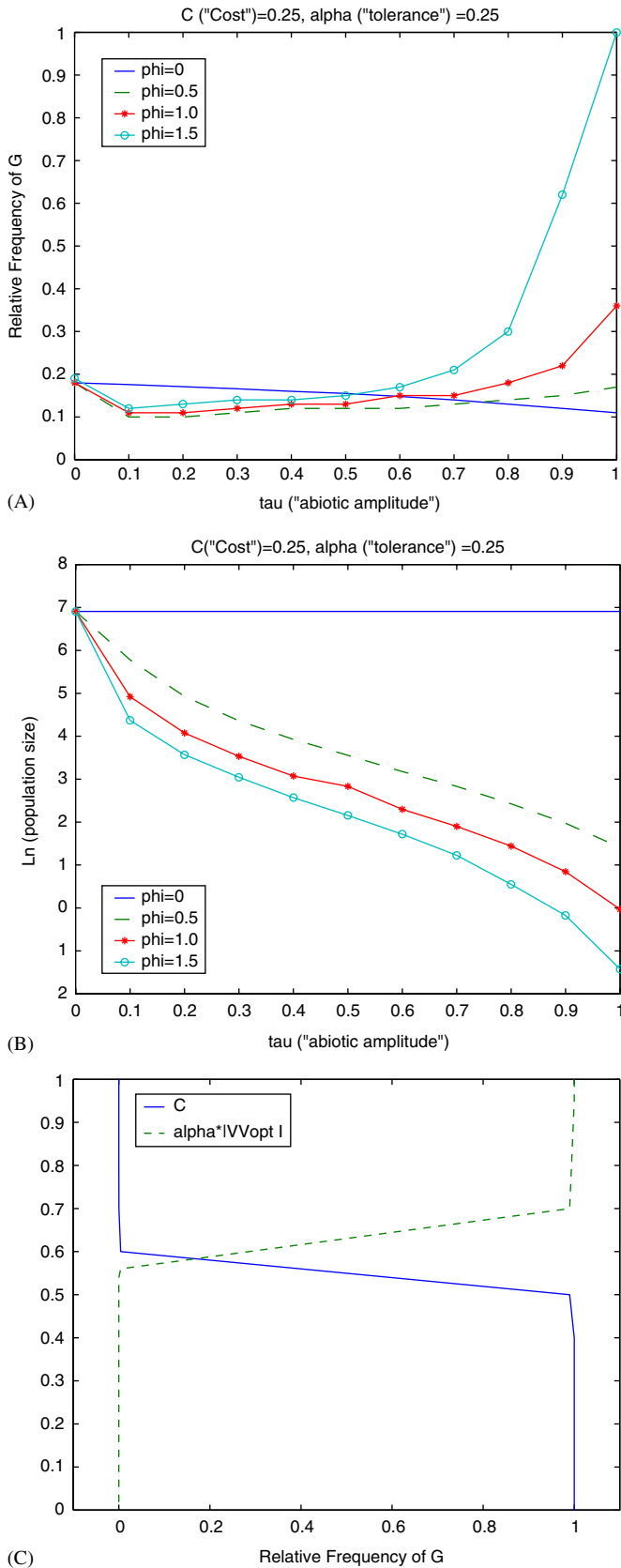


Fig. 3. The interplay between ϕ and relative and absolute genotype frequencies. (A) impact of abiotic variability τ on the relative frequency of G at different ϕ values; (B) Impact of abiotic variability τ on the total population size \sum at different ϕ values; (C) relation between the relative frequency of G (x -axis), and the relative magnitudes of C and $\alpha \cdot |V - V_{opt}|$, where $\alpha = 0.6$, $\tau = 1$, $\phi = 0.01$.

each set of two plots A–C, plot (i) compares the relative frequency of G (circles), P (stars), with the level of V (solid line, relative to the optimum $V_{opt} = 1$), the (constant) threshold C (dashed line), and the value of $\alpha \cdot |V - V_{opt}|$ (dot-dashed line). Note the increased size of the Y-axis through Figs. 4A–C. Fig. 4A illustrates the dramatically lower relative and absolute frequency of G in an environment with low variability where $\tau = 0.1$, in which frequencies of the G genotype do not differ appreciably from their values in a constant environment (G has relative and absolute frequencies of 0.006 and 6 individuals, respectively, with $\tau = 0$ (not shown), versus 0.0069 and 7 individuals in Fig. 4A where $\tau = 0.1$). Note that $\alpha \cdot |V - V_{opt}|$ is much lower than C , hence condition (4) is not realised.

Fig. 4B shows how the G genotype is able to exploit its higher tolerance as a function of the baseline variability in the environment. Note that $\alpha \cdot |V - V_{opt}|$ is driven above the C threshold once abiotic variability $\tau = 1$, and at the same time G starts to persist at tangible (but low) frequencies. Fig. 4C shows that if in addition to its superior tolerance G is able to increase $|V - V_{opt}|$ by a factor 0.01 per individual, then it can drive P extinct by amplifying this variation. Note that the fluctuation in V reaches, and is sustained at, a greater magnitude than that prior to the fixation of G (Fig. 4C(i)). Note also that G self-limits; its absolute frequency remaining below the carrying capacity K (Fig. 4C(ii)), as a result of the deleterious fluctuation that the G genotype is itself causing.

4. Discussion

The effect of a group on the abiotic environment is frequency dependent, therefore it cannot be invoked to explain the increase from rarity of a trait for such an effect. Furthermore, neither high tolerance $\alpha = 1$ or strong enhancement of fluctuation $\phi \geq 1$ is likely to be realistic outside rare microenvironments in which fluctuation is low in the first place, removing the selective advantage of a physiological tolerance trait like α . However, as the environment becomes more variable, even a relatively modest tolerance α value can give the group G a dramatic fitness advantage. Whilst an increase in relative tolerance of a tenth remains a relatively large change, the reader is asked to consider the difference in spatial scale between the pre-transition and post-transition systems when assessing the realism of the values chosen. Any cell within an ancestral multicellular organism is more likely to be shielded from external fluctuations because of the cooperative's greater physical size, even prior to the evolution of the homeostatic physiological systems that will further protect it. If the process described is to be relevant, either the physiological robustness must evolve first, or the group must reach the required intermediate frequencies by genetic drift. The realism of the latter supposition is strengthened by the increased influence of drift in the small populations within microcosms characterizing many evolutionary transitions (Maynard-Smith and Szathmary, 1995).

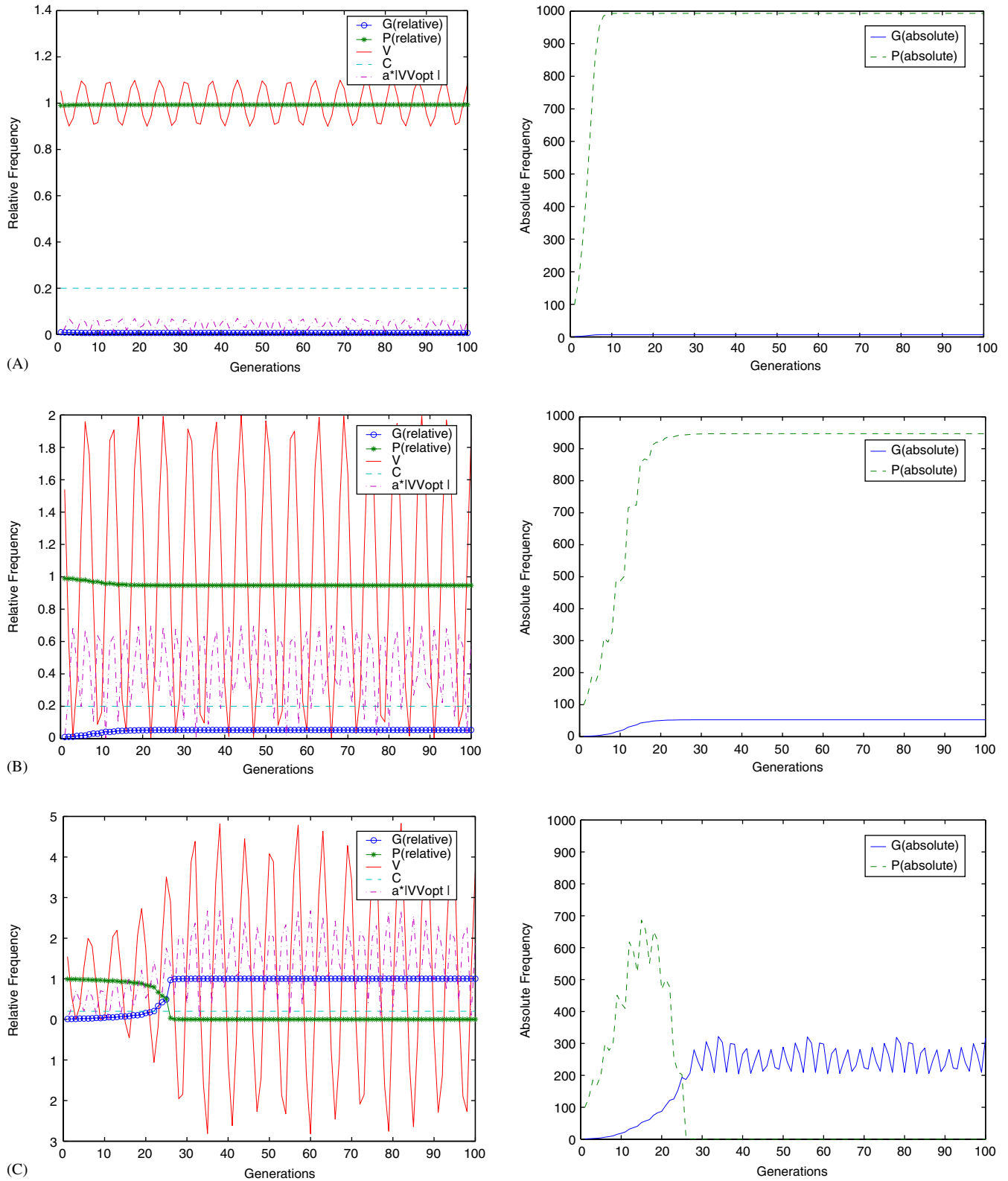


Fig. 4. Single simulation results, cost $C = 0.2$, tolerance $\alpha = 0.7$; (i) relative frequencies, (ii) absolute frequencies. (Note the increasing size through A–C of the y-axis in part (i).). (A) $\phi = 0$, $\tau = 0.1$ (slight biotic enhancement, weak abiotic variation); (B) $\phi = 0$, $\tau = 1.0$ (slight biotic enhancement, strong abiotic variation). (C) $\phi = 0.01$, $\tau = 1.0$ (slight biotic enhancement, strong abiotic variation).

Additionally, the contrast in spatial scale between such microcosms and larger more well mixed environments, is more conducive to the biota achieving localized environ-

mental effects of an order necessary to realise condition (4), because resource/nutrient sequestration and release by an organism has a greater influence on its environment when

contained in a partially closed system (Lenton, 1998). Furthermore, because the group's greater physical size means it displaces a greater absolute quantity of resource within a smaller space than an equivalent number of autonomous individuals, its impact on the dynamics of such a resource will be similarly greater. All of this means that the magnitude of change in resource concentration that the group causes is likely to be dramatically greater than the impact of an autonomous individual. We therefore argue that evolutionary transitions are reasonably described by the parameter values used, whereby the group persists at low frequency, and amplifies variation to drive the "cheat" genotype extinct (enhancement $\phi = 0.01$, tolerance $\alpha = 0.7$). Nonetheless, the greater physical size of the group comes at the cost of a similarly dramatic decrease in short-term fitness, so a high value of tolerance α must also correspond to a high value of cost C .

Hence, a two-stage process is proposed: a transition in individuality occurs and results in enhanced physiological robustness, causing the responsible trait to increase in frequency if the environment is sufficiently variable for this robustness to be adaptive. Once an appropriate frequency is reached, positive feedback with the environmental variable allows the group to approach fixation. In a situation in which the environment is already varying at intermediate amplitude due to physical causes, then population sizes of autonomous individuals are likely to be subject to frequent crashes, further accentuating the influence of genetic drift. The kind of environment in which physiological robustness is advantageous is therefore also one in which a trait for such robustness may be more likely to gain, by chance, a relative frequency sufficient to exploit this robustness. Outside such environments, the physical system is unlikely to permit conditions (4) or (17) to be realised, making the emergence of a trait of this sort rare, and confined to dramatic changes in the structure, function and abiotic impact of life, such as transitions in individuality.

The fact that the group, G , induces a change deleterious to itself in order to purge "cheats", means it also limits its own growth and therefore the magnitude of the change it induces. A transition in individuality should initially cause a dramatic increase in fluctuation through a positive feedback process in which the fluctuation increases the relative fitness of "cooperative" genotypes compared to "cheats". Then, as it becomes too great for the group to tolerate, the abundance of the group should decline to a steady state as the group self-limits through the fluctuation. The resulting magnitude of environmental fluctuation will be greater than prior to the transition. This pattern corresponds well to Fig. 4C. If the hypothesised mechanism occurs, we expect such a pattern to be seen in the geologic record at times of major transitions in individuality. We now discuss two candidate intervals.

Consider the colonization of the land surface by vascular plants after the transition to multicellularity, in symbiosis with mycorrhizal fungi. This resulted in an increase in the

efficiency of weathering of inorganic phosphorous (Lenton, 2001), in addition to an increase in bulk silicate weathering described above. Only the post-transition "group" was able to achieve this—the plant providing reduced carbon and increasing physical weathering via root growth, the mycorrhizal fungus able to selectively weather phosphorous by acidification of the soil and chelation of the cations to which inorganic phosphates are bound (Landeweert, 2001). The consequence of increased phosphorus weathering efficiency is increased export of phosphorus to the oceans, which causes increased marine production and increased burial of marine organic carbon. This burial is the principal source of atmospheric oxygen over geologic time-scales (Lenton and Watson, 2000). The rise in oxygen, in turn, decreases plant growth by increasing photorespiration and fire frequency. This closes a negative feedback loop by limiting phosphorus weathering (Lenton and Watson, 2000; Lenton, 2001). A plant-induced rise in oxygen in the Devonian and Carboniferous has been inferred separately from carbon isotopes (Hayes et al., 1999) and rock-type abundance data (Ronov, 1976). Models driven by such data predict a peak oxygen level of 25–35% around 300 Ma (million years ago) (Lenton, 2001; Berner, 2001). Fossil evidence shows a continuous presence of forests since 350 Ma, placing an upper limit on fire frequency and therefore oxygen, while a continuous charcoal record constrains the lower limit on atmospheric oxygen at around 15% (Lenton & Watson, 2000). Models suggest atmospheric oxygen underwent a damped oscillation within these bounds, reaching a minimum in the late Triassic around 200 Ma before rising again to a secondary peak in the Cretaceous around 100 Ma (Lenton, 2001; Berner, 2001).

The same processes linking phosphorous weathering to atmospheric oxygen may also have been operating as far back as the Neoproterozoic era 1.0–0.55 Ga (billion years ago). During this era, a photobiont-mycobiont symbiosis in the form of lichens evolved (Hedges, 2004; Yuan et al., 2005). This would have had two major environmental consequences. Firstly, a lichen can colonise disparate niches unavailable to fungi or cyanobacteria alone, meaning the productivity and size of the terrestrial biosphere may have suddenly increased after the origin of lichens. This would have enhanced bulk silicate weathering, drawing down atmospheric carbon dioxide and decreasing the temperature. This in turn could have triggered the global scale oscillation between extreme greenhouse and icehouse states that occurred during the Neoproterozoic (Lenton and Watson, 2004; Hedges, 2004). Secondly, in a similar manner to the plant colonisation described above, selective weathering of phosphorous by lichens (Landeweert, 2001), may have caused a net increase in atmospheric oxygen (Lenton and Watson, 2004). During times of extreme glaciation, productivity was decimated and hence oxygen would have declined. However, there was an overall increasing trend in oxygen that provided a necessary condition for the Cambrian explosion (Lenton

and Watson, 2004). Given the polyphyletic nature of the lichen life history (e.g. Nash, 1996), the million year time-scales over which climatic feedbacks of this sort occur is of the same order as that over which one might expect evolution of lichens, following overlap of separate mycobiont and photobiont populations.

Thus the pattern following the evolution of these symbioses is one of overall increases in oxygen and decreases in carbon dioxide and temperature, with signs of oscillation and stabilization at new levels. This is not dissimilar to the consequence of a transition in individuality predicted by our simple model. But do such symbioses correspond directly to a transition in the level of selection? We think so, for two reasons. Firstly, the loss of autonomy at short-term fitness cost involved in a symbiosis bears a strong parallel to formation of group structures between relatives of the same species (Maynard-Smith and Szathmary, 1995). Secondly, it has been noted (Axelrod et al., 2004), that the concept of relatedness can be extended from that of a statistical property of the locus in consideration, to a more dynamic measure of social context in which arbitrary relatedness indicator traits coevolve with kin-selected loci. Perhaps it is plausible that fluctuating abiotic conditions can reinforce, and be reinforced by, the biological traits that give rise to them. The process hypothesized here between physical variables and biological genotypes is loosely analogous to that involved in kin-selected altruism between relatives, in that an otherwise maladaptive trait can increase in frequency as a result of a positive feedback process to which it itself contributes. In this instance the ratio between the contribution made by a genotype to abiotic fluctuation, and the demographic benefit the genotype incurs as a result of this fluctuation, could be considered loosely analogous to kin-relatedness between the two processes—in that it expresses the probability that a suboptimal trait, and a change in the fitness landscape that it is able to induce, will successfully co-evolve. If we now relate this ratio to the ratio between the fitness benefit the post-transition “group” obtains by altering the environment and the cost of the environment-altering trait, (as described by Eq. (4)):

$$\alpha \cdot |V - V_{opt}| : C > \frac{1}{\frac{|V_{biotic} - V_{opt}|}{|V_{abiotic} - V_{opt}| + |V_{biotic} - V_{opt}|} : \frac{G}{G+P}}, \quad (20)$$

where $V_{abiotic}$ denotes V as described in Eq. (11), and V_{biotic} as in Eq. (12). This condition bears qualitative similarities to Hamilton’s rule, $k > 1/r$ (Hamilton, 1964), where k is ratio of gain to the recipient of “altruism” to loss to the “altruist”, and r is the relatedness between the recipient of the “altruism” and the “altruist”. In this instance the post-transition group corresponds to a more energetic system (that is, a higher level of entropy production, e.g. Kleidon and Lorenz, 2004), in which the physical variation increases the relative fitness of the entity able to induce it. A transition between levels of selection is likely to cause the above condition to be realised, because it involves a

dramatic increase in physiological robustness. If this robustness is sufficient for $\alpha \cdot |V - V_{opt}| > C$, the group will increase in frequency. If it reaches a frequency at which it can amplify environmental variation, positive feedback between this variation and the group’s frequency will help purge the group of “cheats”, and as a result further increase group viability. Due to its self-sustaining nature, a positive feedback process of this sort may be a powerful way for the group to persist despite the inherent parasitic tendencies of the individuals of which it is composed.

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