Mutual policing and repression of competition in the evolution of cooperative groups

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EVOLUTIONARY theory has not explained how competition among lower level units is suppressed in the formation of higher-level evolutionary units[1,2]. For example, the key problem of early evolution is how small, individual replicators formed cooperative groups of sufficient complexity to allow accurate copying of the genetic material[3]. The puzzle is why parasites did not subvert the formation of cells by obtaining benefits from the group without contributing to shared traits that enhance reproduction[4]. These parasites would outcompete other replicators within the cell, disrupting reproductive fairness among subunits and destroying the functional coherence of the group. A similar problem arose at a later evolutionary stage with the orderly mendelian segregation of subunits (chromosomes) within cells, and reproductive fairness continued to be a problem in the evolution of insect[5] and human societies[6]. Here I present a simple model to show how reproductive fairness evolves among subunits to create functional coherence and higher-level units. Self-restraint, which evolves according to the kin-selection coefficient of relatedness, is not sufficient: mutual policing and enforcement of reproductive fairness are also required for the evolution of increasing social complexity.

Competition within groups can have both benefits and costs for an individual. If resources are limited within the group, the most competitive individuals will gain a disproportionate share of the local benefits. However, competition often reduces the group's overall efficiency in using local resources, thus lowering the average success of the group members. A simple model describing these costs and benefits of group competition[7] is:

$$w_{ij} = (z_{ij}/z_i)(1 - z_i) \quad (1)$$

where w_{ij} and z_{ij} are the fitness and competitive intensity, respectively, for the jth individual in the ith group, and z_i is the average competitive intensity for members of the ith group. Each individual gains a share z_{ij}/z_i of the local resources, but higher levels of competition reduce the average group productivity, $1 - z_i$.

The model captures the essential tension between individual and group success. For example, the individuals may be parasites and the local resource may be food obtained from the host. Parasites compete within a host by increasing the rate at which host tissues are exploited and consequently damaged. If z_{ij} is the rate of exploitation by an individual parasite, then greater exploitation leads to greater relative success within the host, z_{ij}/z_i. However, rapid exploitation may damage the host, thus reducing the total food available to the parasites by an amount $1 - z_i$.

The equilibrium for the model in equation (1) can be found by maximizing w_{ij} with respect to variants in z_{ij} (see the legend of Fig. 1 for details). The equilibrium is $z^* = 1 - r$, where r is the kin-selection coefficient of relatedness among group members. Self-restraint evolves when relatedness is high, reducing competition among group members and increasing group success. By contrast, low relatedness leads to intense competition and low group productivity. In the parasite example, decreasing relatedness causes greater damage (virulence) to the host[8].

Self-restraint favoured by kin selection promotes improved efficiency of resource utilization. However, there remains a gap between selectively favoured behaviour and complete functional coherence. The gap occurs because within-group competition is increasingly favoured as r declines. In many cases, the genetic relatedness among subunits is low because of mixing among groups and mutation. In addition, many of the important transitions required cooperation between different kinds of units. For example, cooperative symbiosis among different 'quasispecies' of replicators is believed to be essential for the early evolution of genetic systems and the first protocells[3]. The orderly patterns of mendelian segregation are spectacularly rigid controls on the relative success of the different kinds of subunits (chromosomes) within the cell. In many social insects the relatedness among workers is low, and the workers can subvert the cooperative coherence of the colony by laying their own eggs rather than raising those produced by the queen. Thus kin selection alone is unlikely to explain transitions to new evolutionary units or more complex cooperative groups.

If competition within the group can be repressed then the success of each group member would be increased. Reduced

competition would be particularly valuable when relatedness and self-restraint are low. But how can traits that reduce competition evolve when individuals gain by struggling for a larger share of the local resources? This problem is similar to the famous 'tragedy of the commons', apparently first described in formal economic terms in 1833 by William Forster Lloyd[9] (see refs 10, 11). The difficulty is that each individual gains by pursuing interests that increase returns relative to neighbours and decrease the value of common goods. Group-level efficiency is unlikely unless some mechanism exists to repress competition and promote fair distribution of resources.

Kin selection and repression of internal competition can each favour group coherence and the evolution of higher-level units. My intention is to clarify the interaction between these two fundamental processes.

Consider an extension of the previous kin-selection model. A second trait, a, determines each individual's contribution to a mechanism that reduces competition among all members of the local group (mutual policing). The extended model is:

$$w_{ij}=[a_i-ca_{ij}+(1-a_i)z_{ij}/z_i][1-(1-a_i)z_i] \qquad (2)$$

where a_{ij} is an individual's contribution to mutual policing, which has a cost to the individual of ca_{ij}. The average level of policing in the local group is a_i. Each potentially competitive interaction is reduced in both opportunity for gain by the victor, $(1-a_i)z_{ij}/z_i$, and damage to local resources, $(1-a_i)z_i$.

We can obtain an approximate description of evolutionary trends for competitiveness and policing, z and a, by examining the gradient of w_{ij} with respect to variants in z_{ij} and a_{ij}. As explained in the legend of Fig. 1, this gradient is given by

$$\partial w/\partial z=(1-a)[(1-(1-a)z)(1-r)/z-r(1-ca)] \qquad (3)$$

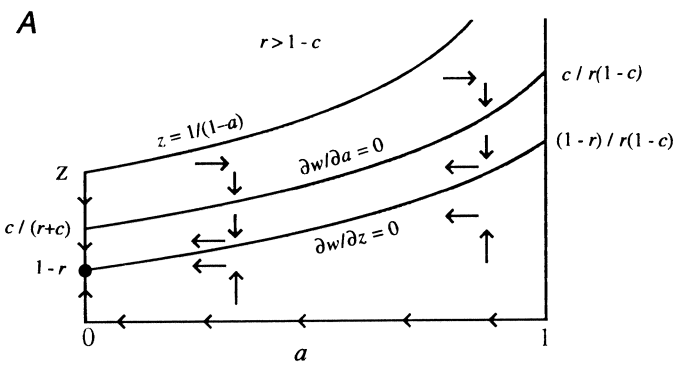
$$\partial w/\partial a=rz(1-ca)-c(1-(1-a)z) \qquad (4)$$

where, as before, r is the coefficient of relatedness among members of the local group. The system moves towards one of two equilibria. When $r>1-c$, the system tends towards $a^*=0$ and $z^*=1-r$, the same equilibrium for the case in which no policing occurs (Fig. 1*a*). When $r<1-c$, the system tends towards complete repression of competition, $a^*=1$ and $z^*=c/[r(1-c)]$ (Fig. 1*b*).

These results can be understood in terms of the self-interest of individuals. When there is no repression of competition, $a=0$, then at the equilibrium $z^*=1-r$ each individual's fitness is r, the degree of self-restraint. An individual benefits by self-restraint because r measures the amount of overlap between individual and group interests. At this equilibrium a rare policing allele, $a_{ij}=\delta>0$, will occur in groups with other rare alleles because relatives interact, thus the group average is $a_i=r\delta$. For small δ, the fitness of a rare policing mutant can be obtained from equation (2) as $r[1-c\delta+\delta(1-r)]$, which is greater than r, the fitness of the resident, non-policing allele, when $r<1-c$. If $r>1-c$, self-restraint yields greater individual success than policing neighbours.

As noted above, relatedness r is often less than one in complex groups. Thus mutual policing and suppression of competition are required for efficient social organization[5,6,12]. The surprising result from a simple model is how strongly natural selection favours individual subunits to contribute resources for suppressing competition. Simultaneously, the subunits are still favoured to strive for their own success against their neighbours. Thus a double standard evolves. Individuals contribute toward universal fairness, but strive for their own reproductive gains within the system of fairness that they helped to create. This duality explains why components of a group, with their own self-interests, have contributed to complex regulatory mechanisms such as mutual policing of workers in insect societies[12] and mendelian segregation of chromosomes during meiosis. However, the dual interests of an individual explain why regulatory mechanisms are rarely sufficient to suppress individual striving within groups, and thus the rarity of the major evolutionary transitions to higher levels of organization.

At first glance, a likely scenario for the formation of higher-level units is the origin of complex regulatory mechanisms and policing when relatedness is high. Subsequent changes in social structure leading to lower relatedness within groups may follow once reproductive fairness is enforced. However, the model clearly shows that situations of high relatedness are the least conducive to large investments in regulatory mechanisms. Thus the origin of complex regulatory mechanisms cannot be explained solely by kin selection.



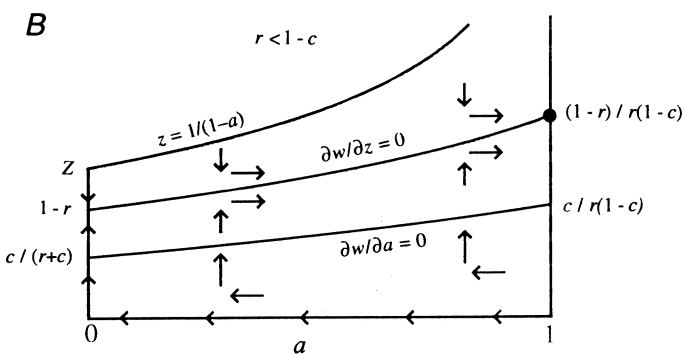


FIG. 1 Evolutionary dynamics of competitiveness, z, and mutual policing, a. In *A*, when relatedness is high relative to the benefits of mutual policing, $r>1-c$, then the outcome tends toward self-restraint, with $z^*=1-r$ and no mutual policing, $a^*=0$. In *B*, when $r<1-c$, the outcome tends towards full investment in mutual policing and complete repression of competition, $a^*=1$. In both *A* and *B*, z is shown on a log scale. In *A*, $c=r=0.7$; in *B*, $c=0.2$ and $r=0.4$. The gradient of fitness with respect to mutual policing, $\partial w/\partial a$, is obtained by taking the partial derivative of w_{ij} with respect to a_{ij}, evaluated at fixed trait values for all individuals, $a_{ij}=a$ and $z_{ij}=z$. A similar approach is used for $\partial w/\partial z$. This is the standard method of evolutionarily stable strategy (ESS) analysis, as outlined by Maynard Smith[19]. An additional method for handling kin selection is used here. The partial derivatives include the derivatives da_i/da_{ij} and dz_i/dz_{ij}. We show elsewhere (S.A.F. and P. D. Taylor, manuscript in preparation) that, by equating these derivatives with the slope of group genotype on individual genotype, these terms are simply the kin-selection coefficient of relatedness, r. This method has a close relationship to the Price equation[20], which has been used extensively for analysis of kin-selection problems[21]. Other assumptions about costs, benefits and genetics complicate the analysis. However, this minimal model demonstrates the powerful selective pressures on self-restraint and mutual policing that favoured increasingly complex evolutionary units.

The model presented here clarifies the simple, logical relation between kin selection and the economics of regulatory mechanisms. The model in its simplest form is a theorem about natural selection rather than a testable prediction. The value of such abstract theory turns on its ability to unify apparently disparate phenomena by highlighting the fundamental processes common to each case. In addition, general understanding of process should lead to new insight for specific systems. A few examples illustrate the range of problems influenced by both kin selection and repression of competition.

Hurst[13] showed that lower relatedness among cytoplasmic genetic elements tends to increase competition within the cell at a cost to individual fitness. This idea is the same as that expressed by equation (1), where lower relatedness leads to higher virulence. Hurst was interested in the forces that influence biparental versus uniparental inheritance of cytoplasmic elements. When there is cytoplasmic competition associated with low relatedness in cells then the nuclear genes suffer reduced fitness because of cytoplasmic 'virulence'. Thus biparental inheritance of organelles, which mixes lineages and lowers relatedness within cells, can reduce nuclear fitness when compared with uniparental inheritance. If relatedness and self-restraint among biparentally inherited cytoplasmic elements are sufficiently low then costly nuclear mechanisms can evolve to enforce uniparental inheritance. Such mechanisms repress competition by preventing the mixing of cytoplasmic lineages. Hurst reviewed evidence suggesting an interaction between costly nuclear control mechanisms and patterns of mixing among cytoplasmic lineages.

Wilson and Sober[1,14] suggested that group competition can be repressed by a randomization mechanism such that all group members have an equal chance of obtaining limited resources or reproductive opportunity. One example is fair segregation of homologues in meiosis. The remarkable life cycle of the slime mould, *Dictyostelium discoideum*, may be another example (D. S. Wilson, personal communication). After a single-celled feeding stage, amoebae aggregate to form a slug[15], which migrates without feeding, and eventually develops into a fruiting body with spores supported by stalk cells. The puzzle is why some cells sacrifice their own reproduction to support that of other cells. One obvious explanation is kin selection. Cells can gain by sacrificing their own reproduction to enhance the reproduction of highly related neighbours. That may indeed be the explanation, but with 10^5 cells per slug there are opportunities for multiple lineages to mix or for mutants to arise that cheat by never developing into stalk cells.

Developmental studies of *Dictyostelium* have shown that cell fate (stalk or spore) is determined early in cell aggregation. The spatial distribution of cell types is apparently random when fate is determined[16–18]. The prestalk cells then migrate to the front and the spore cells aggregate in the rear of the slug. The initial random distribution of cell types certainly fits with the idea that random determination of reproductive opportunity promotes group cooperation beyond the self-restraint favoured by kin selection. However, it is not clear how the group could police a mutant that always developed into a spore. Thus three issues for *Dictyostelium* development highlight the main points of the theory: kin selection and self-restraint among cells within a slug, whether a randomization mechanism that is difficult to cheat is possible, and the cost of such a mechanism.

Kin selection and repression of internal competition have each been discussed widely but, for the most part, separately. The theory and examples presented here show the important interaction between these fundamental processes in the evolution of cooperation and the formation of higher-level units. □

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A gene triggering flower formation in *Arabidopsis*

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In *Arabidopsis*, the apical shoot meristem produces lateral meristems that develop into either shoots or flowers. The decision to form flowers instead of shoots is mediated by the action of floral-meristem-identity genes, such as *APETALA1* (*API*) and *LEAFY* (*LFY*), which specify meristem fate[1–7]. Here we show that transgenic plants which constitutively express the *API* gene show transformations of apical and lateral shoots into flowers, and that these plants flower much earlier than wild-type plants. These results indicate that *API* alone can convert inflorescence shoot meristems into floral meristems, and that ectopic *API* expression can dramatically reduce the time to flowering.

API and *LFY* act redundantly to specify meristem fate and, because no single meristem-identity gene is absolutely necessary for floral meristems to arise, it was unclear if either of them would be sufficient to convert shoots into flowers. The *API* gene, which encodes a putative transcription factor with a MADS domain, is normally expressed in young flower primordia and is not expressed in inflorescence shoot meristems[4]. We generated transgenic plants that constitutively express *API* from the cauliflower mosaic virus 35S (CaMV35S) promoter[8] to determine whether ectopic *API* expression could convert shoots into flowers. The most striking feature of the 35S-AP1 transgenic plants (Fig. 1) is that the normally indeterminate shoot apex (Fig. 1*a*) prematurely terminates as a floral meristem and forms a terminal flower (Fig. 1*e*). In addition, all lateral meristems that would normally produce inflorescence shoots (Fig. 1*b*) are also converted into solitary flowers (Fig. 1*f*). These results demonstrate that *API* alone is sufficient to convert inflorescence shoots into flowers, even though *API* is not normally absolutely required to specify floral meristem identity. The conversion of shoots into flowers in the 35S-AP1 transgenic plants is strikingly similar to the phenotypes caused by mutations in the *Arabidopsis TERMINAL FLOWER* (*TFL*) gene (Fig. 1*c*, *d*)[9–11]. These observations suggest that the conversion of shoots into flowers by ectopic *API* expression may result from an inhibition of *TFL* activity.

To determine whether the 35S-AP1 transgene causes ectopic LFY activity, and whether ectopic LFY activity is required for

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