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Evolutionary dynamics of altruism and cheating among social amoebas

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Dictyostelium discoideum is a eukaryotic amoeba, which, when starvation is imminent, aggregates to form fruiting bodies consisting of a stalk of reproductively dead cells that supports spores. Because different clones may be involved in such aggregations, cheater strategies may emerge that allocate a smaller fraction of cells to stalk formation, thus gaining a reproductive advantage. In this paper, we model the evolutionary dynamics of allocation strategies in *Dictyostelium* under the realistic assumption that the number of clones involved in aggregations follows a random distribution. By determining the full course of evolutionary dynamics, we show that evolutionary branching in allocation strategies may occur, resulting in dimorphic populations that produce stalkless and stalked fruiting bodies. We also demonstrate that such dimorphisms are more likely to emerge when the variation in the number of clones involved in aggregations is large.

Keywords: *Dictyostelium*; slime mould; adaptive dynamics; altruism

1. INTRODUCTION

Altruism is a cooperative behaviour that increases the fitness of others at a cost to the individual. While such behaviour may seem evolutionarily paradoxical at first sight, its existence has been documented in many cases spanning several taxonomic groups (Crespi 1996; Galliard *et al.* 2003). Three types of mechanisms have been suggested to explain the prevalence of altruistic behaviour in nature. First, kin selection (Hamilton 1963, 1964, 1972) has been successful in explaining altruistic behaviour among lower animals, with a wealth of supporting evidence having been accumulated over the past few decades. For kin selection to act efficiently, individuals must be closely related, as is often the case for social insects such as ants or bees. A second mechanism for the evolution of altruistic behaviour is group selection (Wilson & Dugatkin 1997), which acts by differentially affecting the survival of subpopulations in structured populations. Group selection has been used, for example, to demonstrate the existence of an upper bound for the evolution of virulence in spatially structured pathogen–host models (Van Baalen & Rand 1998). Third, direct or indirect reciprocation may also lead to altruistic behaviour (Trivers 1971; Axelrod & Hamilton 1981; Axelrod 1984). Because reciprocation relies on memorizing past behaviour it is thought to apply mainly to cooperation among higher animals.

Dictyostelium discoideum is a eukaryotic amoeba, which, when starvation is imminent, aggregates to form fruiting bodies where a stalk supports a spherical structure containing spores called a 'sorus' (Raper 1984; Fortunato *et al.* 2003*a*). Cells participating in the formation of the stalk exhibit an extreme form of altruism; they die in the process, without any chance of participating in reproduction. Only cells in the sorus form spores, which are subsequently distributed through passive dispersal. *Dictyostelium* is interesting from an evolutionary viewpoint, because it readily forms chimeric aggregations, in which different clones mix in the process of stalk formation and spore dispersal. These chimeric aggregations have been observed in the laboratory, and are assumed to occur under natural conditions as well (Fortunato *et al.* 2003*a*). In chimeric aggregations, there is the potential for the emergence of cheater strategies that allocate less to stalk formation and thus gain a reproductive advantage in spore dispersal. Indeed, several such cheating strains have been documented (Buss 1982; Dao *et al.* 2000; Strassmann *et al.* 2000). The conceptually simplest form of cheating amounts to clones that allocate only a small fixed proportion of all cells to the jointly formed stalk. Most of the documented cheater clones, however, are able to sense whether or not they are participating in a chimeric aggregation and decrease their allocation to stalk formation only in the presence of other clones they can thus exploit. Some clones are even able to parasitize other clones in ways that cannot be explained by stalk-to-spore allocation ratios alone (Kessin 2001; Fortunato *et al.* 2003*a*).

Explaining how altruism can persist in the presence of cheaters is a challenging and important task in evolutionary ecology. It is therefore surprising that, so far, only a handful of theoretical studies have analysed the evolutionary dynamics of *Dictyostelium*. These studies have almost exclusively concentrated on fixed-allocation cheating, and it indeed seems natural to understand this case before considering more complex interactions. In an influential study, Armstrong (1984) investigated a model consisting of altruists, cheaters and loners (amoebas that do not aggregate). In this model, amoebas were situated on a lattice and performed random walks with division until a critical total population size was reached and aggregation occurred. Depending on the parameters used, this led to varying degrees of clustering among clones. Based on these results, Armstrong concluded that altruistic behaviour can indeed persist even in the presence of cheaters. Matapurkar & Watve (1997) later considered an extended model in which cyclic population dynamics could occur. A different path was taken by Matsuda & Harada (1990)

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Table 1. Overview of variables and parameters.

notation	description
α	evolving trait of an amoeba determining its investment into stalk formation
α_g	stalk quality, determined as the weighted arithmetic mean of the trait values of clones participating in a fruiting body
r	resident trait: used instead of α_1 when at most two traits are present
m	mutant trait: used instead of α_2 when two traits are present
n	number of clones present in the total population
D	function describing the dependence of dispersal success on stalk quality
b	product of the number of amoebas in a fruiting body, and the number of spores a single amoeba produces (does not affect any results)
p_k	probability that a fruiting body originates from k founder spores, therefore equals the probability of k founder spores at sites receiving at least one spore
a_i	density of successfully dispersed spores with trait value α_i in the current generation
a_i'	density of successfully dispersed spores with trait value α_i in the next generation
k	total number of founder spores resulting in a fruiting body
k_i	number of founding spores with trait value α_i in a fruiting body
μ, σ	mean and standard deviation of inverse founder spore numbers

who identified evolutionarily stable stalk-to-spore allocation ratios in aggregations formed by a given number of spores with a given distribution of these ratios. In this manner, some far-reaching insights could be derived analytically. Hudson *et al.* (2002) analysed a special case of this latter model, and also presented one of the first models of variable-allocation cheating.

The studies by Armstrong (1984), Matapurkar & Watve (1997), Matsuda & Harada (1990) and Hudson *et al.* (2002) significantly advanced understanding of how altruistic behaviour in *Dictyostelium* could have evolved, despite its apparent vulnerability to cheating. While Armstrong (1984) and Matapurkar & Watve (1997) relied mainly on numerical simulations, Matsuda & Harada (1990) and Hudson *et al.* (2002) only considered evolutionarily stable strategies in chimeric aggregations with fixed sets of founder spores. In this paper, we combine the strengths of these previous approaches by allowing for a probabilistic variable number of founder spores, while retaining analytic tractability to a large extent. Evolutionary dynamics in the extended model are analysed based on adaptive dynamics theory (Dieckmann & Law 1996; Metz *et al.* 1996; Geritz *et al.* 1998).

2. MODEL DESCRIPTION

We study a model in which, in each generation, fruiting bodies are formed at a large (infinite) number of sites. At the beginning of a generation, spores are randomly dispersed among sites, where they turn into amoebas. At each site, these amoebas may then aggregate into a stalk and a sorus, producing new spores. Amoebas differ in their probability α of participating in the formation of stalks. This trait is allowed to evolve (table 1).

(a) *Dispersed spores*

We first determine the number of spores successfully dispersed from a fruiting body when the number and traits of the founder spores, from which that fruiting body is formed, are given. This component of the model closely follows Hudson *et al.* (2002). We consider a fruiting body that is founded by a total of $k = k_1 + \cdots + k_n$ spores, with trait values $\alpha_1,\ldots,\alpha_n$, respectively. The weighted arithmetic mean

$$\alpha_g = \left(k_1\alpha_1 + \cdots + k_n\alpha_n\right)/k,$$

is the expected fraction of amoebas participating in the formation of the stalk. Assuming that the number of amoebas in a fruiting body is constant and large, we take α_g as a measure of the stalk's quality as it may, for example, be quantified in terms of stalk height. The fraction of spores successfully dispersed from a stalk of quality α_g is $D(\alpha_g)$, and this fraction is equal for all clones participating in the corresponding fruiting body. The total number of successfully dispersed spores with trait value α_i is then given by

$$D(\alpha_g)(1-\alpha_i)bk_i/k.$$

Here, $1-\alpha_i$ is the fraction of amoebas with trait value α_i that participate in the fruiting body (rather than in stalk formation), and k_i/k is the fraction of amoebas with trait value α_i at the considered site. Thus, the product $(1-\alpha_i)k_i/k$ is the fraction of amoebas in the fruiting body producing spores with trait value α_i. Finally, we multiply with a constant b to convert this fraction into the number of dispersed spores. This constant determines the product of the number of amoebas in the fruiting body and the number of spores a single amoeba can produce.

(b) *Founded aggregations*

The fraction of all founder spores belonging to a given clone is deterministic, and is given by the clone's proportion among the spores successfully dispersed at the beginning of a generation. By contrast, the number of founder spores arriving at a particular site is random. Sites that do not receive any spores cannot produce fruiting bodies and therefore do not contribute to the generation of new spores. To describe the randomness in the number of founder spores, we can thus restrict attention to the probability distribution $\{p_k\}_{k=1}^{\infty}$ of these numbers at sites that receive at least one spore.

We now consider the densities $a_1,\ldots,a_n$ of spores with trait values $\alpha_1,\ldots,\alpha_n$ successfully dispersed at the beginning of a generation. From these densities, we can derive the densities of spores successfully dispersed at the beginning of the next generation, denoted by $a_1', \ldots, a_n'$. The density a_j' of spores with trait value α_j in the next generation is given by summing over all possible configurations of founder spores that may arise at sites. This sum over founder configurations can be broken up into a first sum over the total number of founder spores

and a second sum over the numbers by which the different clones contribute to this total number of spores,

$$a_j' = \sum_{k=1}^{\infty} p_k \sum_{k_1+\cdots+k_n=k} \mathrm{P}(k_1 \ldots k_n | k_1 + \cdots + k_n = k) \times D(\alpha_g) b(1-\alpha_j) \frac{k_j}{k}. \quad (2.1)$$

Because the number of fruiting bodies is assumed to be large (infinite), p_k describes the fraction of sites at which a total of k spores arrive. Given this total number k of founder spores, $\mathrm{P}(k_1 \ldots k_n | k_1 + \cdots + k_n = k)$ denotes the conditional probability of $k_1,\ldots,k_n$ of these founder spores having trait values $\alpha_1,\ldots,\alpha_n$. Accordingly, the product $p_k \mathrm{P}(k_1 \ldots k_n | k_1 + \cdots + k_n = k)$ describes the joint probability with which the founder configuration $k_1,\ldots,k_n$ occurs. Because we are assuming that the trait values of founder spores are drawn randomly in proportion to the densities that the corresponding clones have among successfully dispersed spores, this joint probability follows a multinomial distribution,

$$\mathrm{P}(k_1 \text{ of trait } \alpha_1, \ldots, k_n \text{ of trait } \alpha_n) = \frac{k!}{k_1! \ldots k_n!} \frac{a_1^{k_1} \ldots a_n^{k_n}}{(a_1 + \cdots + a_n)^k}.$$

When only two clones are present ($a_1 = a_r$, $a_2 = a_m$, $\alpha_1 = r$ and $\alpha_2 = m$), equation 2.1 simplifies to

$$a_m' = \sum_{k=1}^{\infty} p_k \sum_{i=1}^{k} \binom{k}{i} \left(\frac{a_m}{a_m + a_r}\right)^i \left(\frac{a_r}{a_m + a_r}\right)^{k-i} \times D\left(\frac{im + (k-i)r}{k}\right) \frac{ib(1-m)}{k}. \quad (2.2)$$

Equations 2.1 and 2.2 describe how the densities of successfully dispersed spores change over successive generations. The equations show that, to complete the description of our model, two aspects still have to be quantified. First, we have to specify the dispersal benefit of stalk quality (described by the function D), and second, we have to consider the probability distribution of the number of founder spores (described by the probabilities p_k).

(c) *The benefit of stalk quality*

Unlike many spore-producing plants and fungi, spores produced by *Dictyostelium* are not dispersed by wind but by animals, or, in some cases, by rainwater washing through the soil (Bonner 1982). Birds and insects feeding on the fruiting bodies, in particular, provide an effective means of long-range dispersal (Suthers 1985), while passing micro-arthropods or annelids pick up spores and disperse them over shorter distances (Huss 1989). The spores may also be consumed by nematodes, a natural predator of *Dictyostelium*, and thereby be dispersed over short distances up to about 5 cm (Kessin 2001). It is assumed that stalk quality is important in all of these cases, even though it may play less of a role in dispersal through nematodes or water. Yet, no attempts to measure the dependence of spore dispersal on stalk height seem to have been made. Two qualitative observations nevertheless appear to be warranted. First, the spatial autocorrelation of environmental conditions over short distances favour long- and medium-range dispersal. Given that the known mechanisms by which such dispersal is achieved seem to require a stalk, dispersal success must be expected to increase with stalk height, at least at small heights. Second, because dispersal by birds and insects probably requires a certain critical stalk height, a strong increase in dispersal success is expected at certain heights. As no mechanistic derivation is feasible, we will consider flexible functions to describe the dependence of dispersal success $D(\alpha_g)$ on stalk quality α_g. One such function was introduced by Hudson *et al.* (2002), who assumed that dispersal success was given by

$$D(r) = d_0 + (1 - d_0) r^q. \quad (2.3)$$

The parameter d_0 allows for some baseline dispersal success for spores originating from stalkless fruiting bodies, while the parameter q determines the shape of D, which is convex for $q \geq 1$ and concave for $q \leq 1$. This particular choice of function implies that the slope of D at $r = 0$ is either zero (if D is convex) or infinite (if D is concave). By contrast, the two qualitative observations described above suggest functions that combine a positive initial slope (first observation) with a convex shape (second observation). We will therefore consider an alternative, more flexible function,

$$D(r) = \left(\frac{r + \varepsilon}{1 + \varepsilon}\right)^q. \quad (2.4)$$

Examples of the shapes of this function are shown in figure 1 for several values of q and ε. As we can see, this function allows for a positive initial slope combined with a convex shape. As long as $\varepsilon > 0$, spores originating from stalkless fruiting bodies experience a non-vanishing dispersal success. Because the selection pressures acting on the dispersal efficiency of fruiting bodies are probably enormous in small organisms (Bonner 1982), these properties seem reasonable.

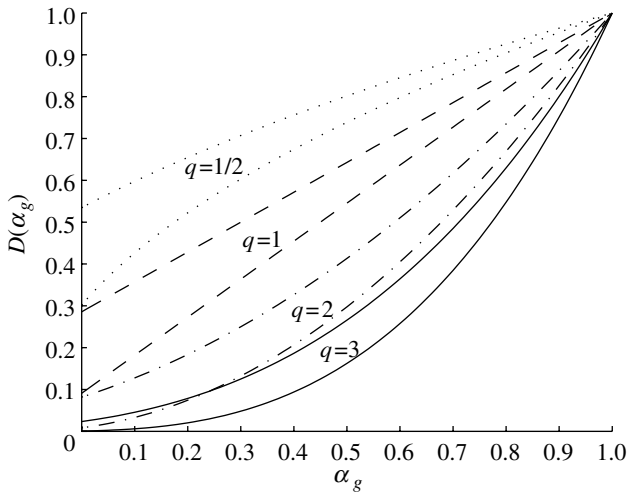

Figure 1. Dispersal success as a function of stalk quality according to equation 2.4, plotted for all combinations of $q = 1/2, 1, 2, 3$ and $\varepsilon = 0.1, 0.4$. Curves with the same line style correspond to identical values of q, with lower curves corresponding to $\varepsilon = 0.1$.

(d) *The distribution of founder spores*

To complete the description of our model, it is necessary to specify the probability distribution for the number of founder spores. Unfortunately, few empirical studies have addressed this question. An exception is the work by

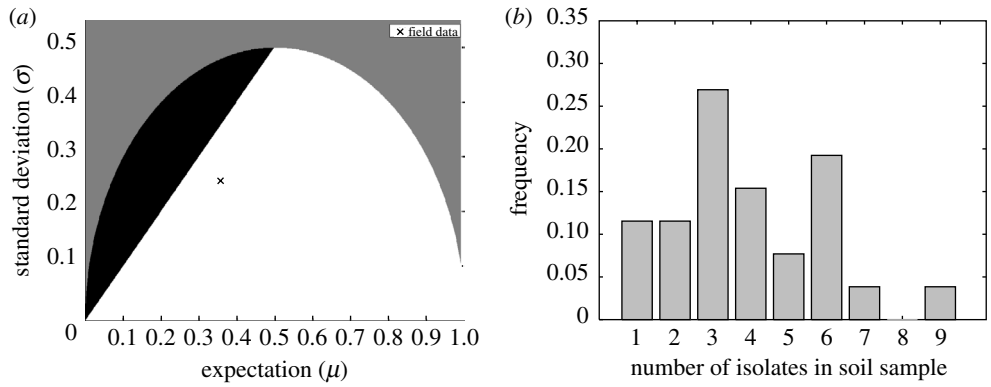

Figure 2. (*a*) Combinations of the mean μ of inverse founder spore numbers and the corresponding standard deviation σ for which evolutionary branching is possible (black region) and impossible (white region). Combinations in the grey region are logically infeasible. The cross in the white region corresponds to the distribution in the right panel. (*b*) Probability distribution of founder spore numbers at sites receiving at least one spore according to field measurements by Fortunato *et al.* (2003*a*).

Fortunato *et al.* (2003*b*), who analysed 50 soil samples collected near the Mountain Lake Biological Station in Virginia, USA, where 26 of these samples contained more than one isolate of *Dictyostelium*. The distribution determined by the number of isolates in these 26 samples is shown in figure 2*b*. Although there are many plausible ways to specify the distribution of founder spores, we will see that the precise form of this distribution is not important. Most of our conclusions below only depend on two statistical features of the considered distribution; the expectation μ and standard deviation σ of the inverse founder spore numbers at sites that receive at least one spore,

$$\mu = \sum_{k=1}^{\infty} \frac{p_k}{k} \quad \text{and} \quad \sigma^2 = \sum_{k=1}^{\infty} p_k \left(\mu - \frac{1}{k}\right)^2.$$

If μ is close to one, then each fruiting body results from only one or a few spores and thus contains only one or a few clones. Under such conditions, we expect kin selection to be a strong force driving the evolutionary dynamics towards altruistic behaviour. Conversely, for low values of μ, such behaviour is not expected to evolve to any large degree. However, if a low value of μ is combined with a relatively large value of σ, which implies a large standard deviation of founder spore number per site, then a significant proportion of sites will still receive only few founder spores and kin selection may be strong. A convenient statistics that roughly captures this joint dependence is the coefficient of variation, σ/μ, obtained by expressing the standard deviation σ in units of the mean μ. Kin selection may be strong when this coefficient of variation is sufficiently large.

3. EVOLUTIONARY INVASIBILITY ANALYSIS

We now proceed with investigating the model's evolutionary dynamics. At the heart of this analysis lies the question of whether or not a mutant clone may successfully invade the population of an established resident clone. This question can be addressed by considering the reproductive ratio of a rare mutant clone with trait value m in the environment determined by a resident clone with trait value r,

$$S_r(m) = \lim_{a_m \to 0+} \frac{a_m'(a_r^*, a_m)}{a_m}.$$

Because the resident clone is assumed to be at equilibrium, $S_r(r) = 1$ always holds. The mutant can invade the resident whenever $S_r(m) > 1$. As long as mutant and resident trait values are similar, such an invasion generically implies a trait substitution, during which the mutant replaces the resident (Geritz *et al.* 2002). In adaptive dynamics theory, the function $S_r(m)$ is known as invasion fitness (Metz *et al.* 1992). We can use the selection gradient $S_r'(r)$ to determine when a mutant with a trait value m similar to r can invade: $m > r$ can invade if $S_r'(m) > 0$, and $m < r$ can invade if $S_r'(m) < 0$. In appendix A, we show that the selection gradient of our model is given by

$$S_r'(r) = \mu \frac{D'(r)}{D(r)} - \frac{1}{1-r}. \tag{3.1}$$

Based on the analysis of invasion fitness and selection gradient, we will show below that, under certain conditions, the evolutionary dynamics of altruism in our model passes through a sequence of four distinct phases. In the course of this evolutionary process, the population of amoebas first changes from stalkless clones to stalked clones, and then to an evolutionarily stable dimorphism of stalkless and stalked clones.

(a) *Incipient altruism*

When can stalked clones, $r > 0$, evolve from stalkless ones, $r = 0$, through a sequence of small evolutionary steps? The selection gradient in equation 3.1 shows that this is possible if $\mu D'(0) > D(0)$. The dispersal success function considered by Hudson *et al.* (2002), equation 2.3, yields $\mu D'(0) < D(0)$ whenever $q > 1$. By contrast, the alternative function in equation 2.4 yields $\mu D'(0) > D(0)$ whenever $\mu q > 0$; under this condition, clones with stalked fruiting bodies can thus evolve from stalkless ones. These conclusions are underscored by the geometric illustrations in figure 3, which, by means of pairwise invasibility plots (Geritz *et al.* 1998) show combinations of mutant and resident trait values for which the mutant can invade the resident. Figure 3*a* shows that, based on equation 2.3, no mutants exist that can invade a resident with trait value $r = 0$. Figure 3*b* shows that this situation is exactly reserved when dispersal success follows equation 2.4. In the following we will investigate the further course of evolution based on equation 2.4.

(b) *Mounting altruism*

Once stalked clones have evolved from stalkless ones, where will the evolutionary trend towards increasing levels of altruism end? To answer this question, we have

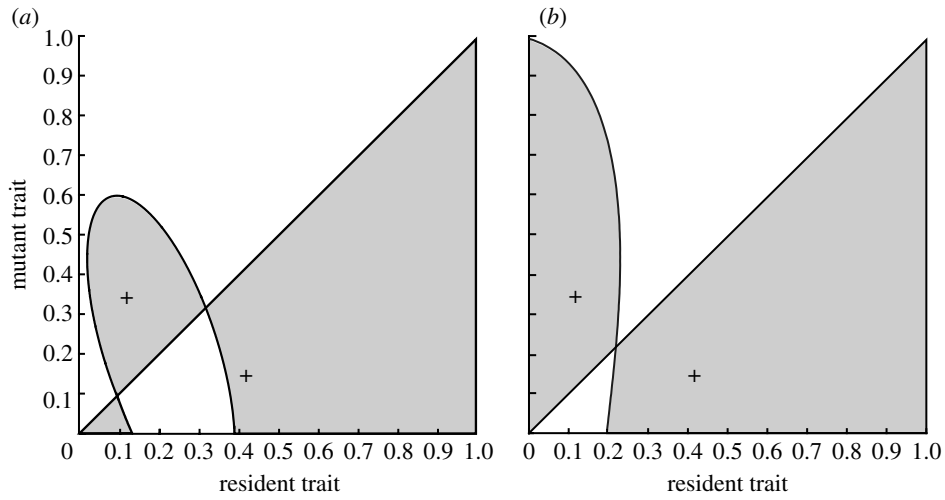

Figure 3. Pairwise invasibility plots for different founder spore distributions and dispersal success functions. (*a*) Founder spore distributions according to figure 2*b*, and dispersal success function according to equation 2.3 with parameters $q=2$ and $d_0=0.05$. Note that the evolution of incipient altruism is precluded. (*b*) Geometric founder spore distribution with parameter 0.04 and dispersal success function according to equation 2.4 with parameters $q=4$ and $\varepsilon=0.2$. Here, the evolution of altruism can take off from $r=0$.

to identify trait values at which directional selection ceases and the selection gradient vanishes. Such points are called 'evolutionarily singular' (Metz *et al.* 1996). When such points serve as attractors of gradual evolutionary change in their neighbourhood, they are called 'convergence stable' (Eshel & Motro 1981; Eshel 1983; Christiansen 1991). With dispersal success given by equation 2.4, a unique convergence stable singular strategy exists at

$$r^* = \frac{\mu q - \varepsilon}{1 + \mu q}. \tag{3.2}$$

As shown in appendix A, this strategy falls in the range $0<r^*<1$ whenever the evolution of incipient altruism is possible, $q>0$ and $0<\varepsilon<\mu q$.

(c) *Evolutionary branching*

Once gradual evolution has reached r^*, what will happen next? The answer depends on whether selection at this point is stabilizing, such that evolution comes to a halt, or disruptive, such that evolution may continue further. In the first case, the evolutionarily singular strategy is situated at a fitness maximum and is said to be locally evolutionarily stable (Maynard-Smith & Price 1973). By contrast, disruptive selection at a convergence stable singular strategy occurs at a fitness minimum, and is expected to initiate a process known as 'evolutionary branching' (Metz *et al.* 1996; Geritz *et al.* 1998). During such a process, a protected dimorphism of clones may evolve. We now show that, under certain conditions, evolutionary branching may occur in our model. When the second derivative of invasion fitness at r^*, given by

$$S_r''(r^*) = \left(\frac{1+\mu q}{1+\varepsilon}\right)^2 \left(\left(\frac{\sigma^2}{\mu^2}+1\right)\frac{q-1}{q} - 2\right), \tag{3.3}$$

is positive, selection at r^* is disruptive and evolutionary branching is possible. This implies that evolutionary branching may occur only if the coefficient of variation of the inverse number of founder spores is sufficiently large,

$$\frac{\sigma}{\mu} > \sqrt{1+\frac{2}{q-1}}.$$

In figure 2*a*, the largest possible region in which this condition applies is highlighted in black. Because the inverse founder spore numbers at sites that receive at least one spore are positive and never exceed one, we have $\sigma<\sqrt{\mu(1-\mu)}$. In figure 2*a*, the region in which this consistency condition does not hold is shown in grey. In summary, evolutionary branching at r^* occurs in the black region of figure 2*a*, whereas evolution comes to a halt at r^* in the white region of figure 2*a*.

Note that combining the two inequalities above implies $\mu>1/2$. This means that for evolutionary branching to occur, on average, there must be more than two founder spores at sites receiving at least one spore. In addition, the coefficient of variation has to be sufficiently high for kin selection to act efficiently. Not many distributions satisfy these criteria. For example, the empirically determined distribution (figure 2*b*) does not. Alternatively, when we assume that the numbers of founder spores are distributed geometrically, $p_k=(1-p)^{k-1}p$ with $0<p<1$, the average number of founder spores needs to exceed 19.3 if evolutionary branching is to occur for $q=3$. This threshold decreases to 13.2 for $q=5$, to 10.2 for $q=10$, and to 7.96 as q tends to infinity.

(d) *Dimorphic evolution*

Once evolutionary branching has been initiated at r^*, what is the further course of dimorphic evolution? To prepare for addressing this question, we first consider all combinations of trait values r_1 and r_2 that result in a protected dimorphism. This is the case if, and only if, the two corresponding clones can mutually invade, that is, a rare mutant with trait value r_1 can invade a resident population with trait value r_2 and *vice versa*. Based on the pairwise invasibility plot in figure 3*b*, we find the region of coexistence as the largest subset of the range of positive invasion fitness that is symmetric around the diagonal. This region is shown in grey in figure 4*a*; dimorphic evolution can only occur in its interior. We now study the evolutionary dynamics of a dimorphic population inside the region of coexistence in much the same way as we did for a monomorphic population, except that the actual

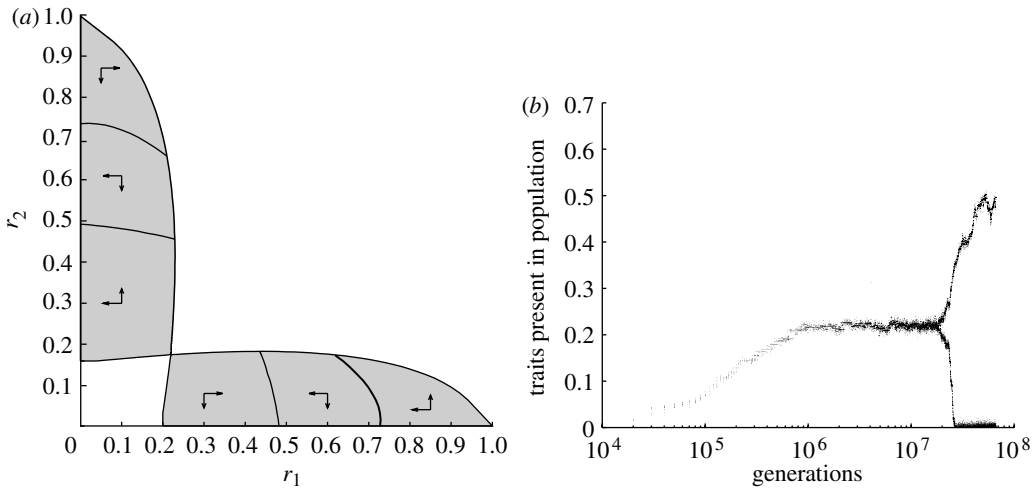

Figure 4. Dimorphic evolutionary dynamics after branching. (*a*) The region of coexistence, in which protected dimorphisms are possible, is shown in grey. Arrows indicate the selection gradient's direction for each of the two resident trait values. The directions of these selective pressures change at the shown evolutionary isoclines, defined by vanishing selection gradients. Thin isoclines are evolutionarily stable, whereas thick isoclines are not. The figure shows that evolutionary change after branching will converge on an evolutionarily stable dimorphism of a cheater and an altruist, resulting in the trait values (0.48,0) at which the evolutionarily stable isocline (thin line) touches the boundary. (*b*) Simulations of the polymorphic evolutionary dynamics based on 1000 fruiting bodies, illustrating all four predicted phases of evolutionary change. In both panels, dispersal success follows equation 2.4 with parameters $q=4$ and $\varepsilon=0.2$, and the number of founder spores is distributed geometrically with parameter 0.04, resulting in an average of 25 founder spores per fruiting body.

calculations have to be done numerically rather than analytically. We denote by $S_{r_1,r_2}(m)$ the reproductive ratio of a rare mutant with trait value m in a dimorphic population with trait values r_1 and r_2 that has attained its population dynamical attractor. In principle, this attractor might imply non-equilibrium dynamics, but—in contrast to the model by Matapurkar & Watve (1997)—the dimorphic population dynamics of our model always settle on stable equilibrium densities a_1^* and a_2^*. The dimorphic invasion fitness

$$S_{r_1,r_2}(m)=\frac{1}{a_1^*+a_2^*}\times\sum_{k=1}^{\infty}\sum_{i=0}^{k-1}\binom{k-1}{i}\left(\frac{a_1^*}{a_1^*+a_2^*}\right)^i\left(\frac{a_2^*}{a_1^*+a_2^*}\right)^{k-1-i}\times D\left(\frac{ir_1+(k-1-i)r_2+m}{k}\right)b(1-m),$$

is obtained analogously to its monomorphic counterpart. If the dimorphic selection gradient $S'_{r_1,r_2}(m)$ is positive (negative) at r_1, then a mutant with a slightly higher (lower) trait value generically invades and replaces the resident with trait value r_1. Figure 4*a* shows—for D given by equation 2.4, with parameters $q=4$ and $\varepsilon=0.2$—the isoclines at which the selection gradient vanishes for one of the two resident traits. Figure 4*a* also highlights the expected directions of gradual dimorphic evolution in the regions between the isoclines, as determined by the dimorphic selection gradient. In addition, lines of different thickness are used to indicate whether, on the isoclines, the fitness S_{r_1,r_2} is at a maximum (thin lines) or minimum (thick lines). Inspection of figure 4*a* shows that after evolutionary branching occurs at $r_1=r_2\approx0.2$, the dimorphic population will evolve towards (0.47,0; or, equivalently, towards 0,0.47), where one of the convergence stable isoclines meets the boundary of trait space. In other words, the dimorphic population will evolve towards a cheater that does not invest in stalk formation and an altruist that invests in stalk formation. The population dynamics in equation 2.1 shows that, for the indicated parameter values, the proportion of cheaters in the dimorphic population will be around one-third. Because the boundary equilibrium thus attained is situated on an isocline that is both convergence stable and locally evolutionarily stable, this dimorphism is recognized as the eventual outcome of gradual evolution in our model.

(e) *Four phases*

To corroborate the conclusions derived above, we simulated the full course of evolutionary dynamics in our model using 1000 fruiting bodies in each generation and considering normally distributed mutations with mean 0 and standard deviation 5×10^{-3}, occurring with a probability of 1% per generation. The resultant evolutionary dynamics are shown in figure 4*b*. In the simulation, the population was typically polymorphic (i.e. several traits where often present at the same time), however, the analysis presented above held up remarkably well. This implies that the two simplifying assumptions underlying this analysis—absence of demographic stochasticity and sufficiently small rates of mutation—had no critical bearing on the derived predictions. In particular, all four phases predicted by the preceding analysis can be observed in the simulation results: incipient altruism (§*a*), mounting altruism (§*b*), evolutionary branching (§*c*), and dimorphic evolution (§*d*), ending up with a population-level mixture of cheaters and altruists.

4. DISCUSSION

We have analysed the expected evolutionary dynamics of fixed-allocation clones of *Dictyostelium*. In doing so, we have shown how, under specific conditions, evolutionary branching may occur, resulting in a mixed population of clones that, when on their own, produce stalkless and

stalked fruiting bodies. When co-occurring on a site, one of these clones exploits the other's investment into stalk formation, without contributing any such investment itself. Our results offer insight as to why altruism persist in *Dictyostelium* despite the (potential or actual) existence of cheating clones; depending on conditions, the population of amoebas evolves either to a monomorphic state of intermediate altruism or to a dimorphic state in which cheaters coexist with clones exhibiting a high degree of altruism. In this way, our findings also contribute to understanding the polymorphism of *Dictyostelium* clones observed in nature. Furthermore, for what is possibly the first time, our analysis has highlighted the importance of variability in the number of founder spores for the evolutionary dynamics of *Dictyostelium*.

We have shown that evolutionary branching occurs under two conditions: first, the coefficient of variation in the number of founder spores must be sufficiently large; and second, the function describing the dependence of dispersal success on stalk quality must be convex. Although these conditions underscore that the scope for evolutionary branching might be limited, it should be borne in mind that our model ignores many extra aspects of variation found in nature, for example, by assuming that sites suitable for *Dictyostelium* colonization are identical in all respects. It must be expected that when other sources of variation are added to the model to enhance its realism, evolutionary branching will occur under more general conditions (as suggested, for instance, by evolution in the site-based model analysed by Geritz *et al.* 1998). Furthermore, most clones of *Dictyostelium* compete more actively among each other than is captured by the passive fixed-allocation strategies studied here. The additional frequency-dependent selective pressures resulting from active competition probably broaden the conditions for evolutionary branching.

We can strengthen the case for a convex function describing the dependence of dispersal success on stalk quality by using equation 3.2 to work backwards. Assuming the empirical distribution reported by Fortunato *et al.* (2003*b*) and a stalk-to-spore ratio of 1:5, commonly observed among naturally occurring *Dictyostelium* clones (Kessin 2001), we can conclude that the parameter q must be expected to exceed about 0.8. If we also assume a positive value of $D(0)$, then this brings us well into the convex region. Nevertheless, any rough and phenomenological description of dispersal success remains artificial, and corresponding quantitative measurements are therefore highly desirable.

Future work may advance the analysis presented here in two directions. First, it will be interesting to incorporate explicit spatial structure into the evolutionary model. This would allow for the differential description of short-range, medium-range, and long-range dispersal. Most likely, however, the analytical tractability preserved throughout most of the investigation presented here will be lost in such spatially explicit evolutionary models. Second, with the dynamics of fixed-investment strategies now being well understood, the time may have come to explore the dynamics of variable-investment strategies in the evolution of *Dictyostelium* clones. A first step in this direction has already been taken by Hudson *et al.* (2002). Equipped with the insights gained from our and many other studies, these two extensions may now be feasible.

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APPENDIX A

Most of the analysis in this study is based on the reproduction ratio of a rare mutant with trait value m

$$S_r(m) = \lim_{a_m \to 0+} \frac{a'_m(a^*_r, a_m)}{a_m}.$$

Because

$$\lim_{a_m \to 0+} \frac{1}{a_m} \mathrm{P}(i \text{ of the } k \text{ founders from } a_m) = \begin{cases} \dfrac{k}{a_r} & \text{if } i = 1, \\ 0 & \text{otherwise,} \end{cases}$$

equation 2.2 implies

$$\begin{aligned} \lim_{a_m \to 0+} \frac{a'_m(a^*_r, a_m)}{a_m} &= \sum_{k=1}^{\infty} p_k \frac{k}{a_r} D\left(\frac{m + (k-1)r}{k}\right) \frac{1-m}{k} b \\ &= \frac{1}{a_r} \sum_{k=1}^{\infty} p_k D\left(\frac{m + (k-1)r}{k}\right) (1-m) b. \end{aligned}$$

From $S_r(r) = 1$, we infer an equilibrium resident population density a^*_r of

$$a^*_r = bD(r)(1-r). \tag{A 1}$$

The model's monomorphic invasion fitness is thus given by

$$S_r(m) = \sum_{k=1}^{\infty} p_k D\left(\frac{m + (k-1)r}{k}\right) \frac{1-m}{1-r} \frac{1}{D(r)}. \tag{A 2}$$

Differentiation of this invasion fitness with respect to m gives

$$\begin{aligned} S'_r(m) = &\sum_{k=1}^{\infty} \frac{p_k}{D(r)(1-r)} \\ &\times \left[D'\left(\frac{m + (k-1)r}{k}\right) \frac{1}{k} (1-m) - D\left(\frac{m + (k-1)r}{k}\right) \right]. \end{aligned}$$

Evaluating this derivative at $m = r$ yields the monomorphic selection gradient

$$S'_r(r) = \mu \frac{D'(r)}{D(r)} - \frac{1}{1-r}.$$

Here, we have used

$$\mu = \sum_{k=1}^{\infty} \frac{p_k}{k}$$

which describes the mean of inverse founder spore numbers at sites receiving at least one spore. The corresponding standard deviation is expressed with the help of

$$\gamma = \sum_{k=1}^{\infty} \frac{p_k}{k^2} \quad \text{as} \quad \sigma = \sqrt{\gamma - \mu^2}.$$

The evolutionarily singular strategies, at which the monomorphic selection gradient vanishes, are found by solving

$$\mu D'(r)(1-r) - D(r) = 0, \quad \text{where} \quad r \neq 1 \quad \text{and} \quad D(r) \neq 0.$$

With $D(r)$ given by equation 2.4, we obtain a unique evolutionarily singular strategy,

$$r^* = \frac{\mu q - \varepsilon}{1 + \mu q}.$$

Because the monomorphic selection gradient is positive for $r<r^*$ and negative for $r>r^*$, r^* is convergence stable. To determine whether r^* is also locally evolutionarily stable (i.e., whether it corresponds to a fitness maximum), we differentiate the monomorphic invasion fitness twice with respect to m,

$$S_r''(m) = \sum_{k=1}^{\infty} \frac{p_k}{D(r)(1-r)} \left[D'' \left(\frac{m+(k-1)r}{k} \right) \times \frac{1}{k^2}(1-m) - 2D' \left(\frac{m+(k-1)r}{k} \right) \frac{1}{k} \right],$$

and evaluate this derivative at $m=r$,

$$S_r''(r) = \gamma \frac{D''(r)}{D(r)} - 2\mu \frac{D'(r)}{D(r)(1-r)}.$$

With $D(r)$ given by equation (2.4), this becomes

$$S_r''(r) = \left(\frac{1+\mu q}{1+\varepsilon} \right)^2 \left(\frac{\gamma}{\mu^2} \frac{q-1}{q} - 2 \right),$$

which is equivalent to equation (3.3). When this second derivative is negative, r^* is a fitness maximum and thus locally evolutionarily stable; otherwise, r^* is a fitness minimum.

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