On kin and group selection, and the haystack model

Vincent A. A. Jansen

Abstract. Kin and group selection are two different ways to describe the evolution of social behaviour. Although these two explanations are compatible in many cases, they lead to a different perspective on the interpretation of the drivers of the evolution of social behaviour. Here, I will illustrate that the haystack model, which is often used in the context of group selection, allows a kin selection as well as a group selection interpretation. To do so I will analyse a variant of the haystack model in which the local dynamics are specified through a continuous time model. From the description of the dynamics the cost and the benefits of the interaction can be calculated, as well as the relatedness. We also revisit the interpretation of Maynard Smith, who originally described the model, and show that this interpretation can be found if one assumes strong selection. This shows how the various interpretations of the evolution of social behaviour all can follow from the same model. It also shows how ecological details of the interaction are crucially important in interpreting and understanding the process of evolution.

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1. Introduction

The evolution of social behaviour poses a puzzle within the Darwinian paradigm. If the process of adaptation results from selection that benefits individuals with favourable traits, how is it then possible that behaviour that

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promotes the reproductive output of others at a cost to oneself evolves? Darwin himself was aware of this issue, and commented on it on various occasions. He wrote: “A tribe including many members who . . . were always ready to aid one another, and to sacrifice themselves for the common good, would be victorious over most other tribes; and this would be natural selection” [1]. In this we recognise what nowadays would be called a group selection argument. In the context of insects with sterile casts he wrote: “. . . with the working ant we have an insect. . . absolutely sterile; so that it could never have transmitted successively acquired modifications of structure or instinct to its progeny. It may well be asked how is it possible to reconcile this case with the theory of natural selection?” and he continues: “. . . selection may be applied to the family, as well as to the individual, and may thus gain the desired end. Thus, a well-flavoured vegetable is cooked, and the individual is destroyed; but the horticulturist sows seed of the same stock, and confidently expects to get nearly the same variety” [2]. This last argument is reminiscent of what could be called a kin selection argument.

Others, notably Fisher [3] and Haldane [4], commented on the evolution of social behaviour, but it was W.D. Hamilton who elaborated and formalised the explanation [5] that later became known as kin selection [6]. Hamilton’s understanding appears to have been based on an intuitive insight, to which he added a formal justification. Hamilton’s basic idea takes a gene centered perspective and the observation that it is not so much the benefit to an individual’s fitness that matters, as the benefit to the fitness associated with the gene that conveys an advantage to the behaviour under study. He observed that populations typically are structured and genes are somehow assorted. This makes it more likely that interactions are with individuals that carry the same gene than when the interactions would be with random members of the population. If this is so, the benefits of a behaviour that are bestowed on another individual benefit the gene that causes the behaviour if this gene is also present in the other individual. Therefore, one need not just consider the fitness consequences of a gene on the individual that carries the gene, but also its effect on the same gene in other individuals that reap the benefit of the interaction.

Hamilton quantified the idea that a gene would benefit directly through its influence of the carrier it finds itself in, as well as indirectly through increasing the fitness of other carriers of the same gene with which its carrier interacts. A crucial part of the argument is that populations have structure, which leads to assortment of genes. This can, for instance, be through limited dispersal (Hamilton used the term ”viscous population” to describe this). The fitness of an altruistic gene is then

$$-C + RB,$$

where C is the cost an individual pays for altruism, B is the benefit that others receive from individuals carrying this gene, and R is a measure of the correlation between the genotype of the acting individual and that of its neighbour. Relatedness is proportional to the probability of a carrier of a gene to encounter this same gene among those that (s)he interacts with, over and above the average frequency of this gene in the population. The coefficient of relatedness is often defined as the regression coefficient that is obtained in a plot of the actors genotype versus the genotype of the receiving individual [7].

Prior to Hamilton's idea being widely accepted, the main explanation for traits from which others benefit was that such traits would be adaptations to living in groups. Groups of similar individuals would benefit from such traits and therefore more of such groups would be formed so that the trait would eventually come to dominate a population. One of the main proponents of this group selection perspective was Wynne-Edwards [8].

It was pointed out by John Maynard Smith [6] that there are two main problems with the idea of group selection. Firstly, the explanation is limited to situations where groups can be identified. To describe situations in which the reproductive success of relatives of the individual carrying certain traits is enhanced, Maynard Smith coined the word kin selection. Secondly, and this was the main point of Maynard Smith' argument, groups which are composed of benevolent individuals are vulnerable to exploitation by individuals who benefit from the advantages of the group without contributing to it. Such exploitation easily arises if there is dispersal between the groups.

To illustrate this idea Maynard Smith used a simple model describing mice living in haystacks. At the beginning of a season a haystack is colonised by a pregnant single female mouse. The female carries an allele which renders her timid or aggressive. Each mouse produces a colony and within a colony aggressive individuals will replace the timid ones through competition. The only colonies that will produce timid mice are those that were founded by females carrying only homozygously timid offspring. Although timid mice are competitively at a disadvantage, they can produce far more offspring in a colony they solely occupy. Maynard Smith showed that if mating is mainly within the colony the timid allele will increase in frequency. Mixing between the colonies will make that the selection is easily favoured towards aggressive mice. This model has become known as the haystack model.

A similar type of argument is used extensively in the literature on group selection. Within groups individuals, often haploid, interact and influence their own and each other's fitness through interactions. At the end of the interaction time individuals disperse and colonise new groups (such groups are sometimes referred to us "trait groups" [12]). The point that is often put forward with these models is that it is possible that a trait evolves that loses out in all groups to competition yet can still evolve because of the increased outputs of groups it produces on its own.

Here, I will scrutinise these arguments by formulating a model for the haystack scenario, based on a detailed, if fictitious, description of the local dynamics of the model. The model is a variation of a mathematical model with a detailed description of the dynamics of the haystack model [9]. We will analyse this model to demonstrate how the details of the local interaction and the details of the biology impact the findings of the model, and in how far it is justified to interpret these models in the light of group and kin selection.

2. Local dynamics including resource

Our model is akin to the haystack model, in that it consist of isolated patches that are colonised at the beginning of a season by a number of haploid individuals. We assume that within the patches there is a total amount of resource U available. The individuals in the patch can sequester this resource with rate c_i. The sequestered resource is converted into new individuals, which can die with rate d and are then converted back into resource.

There are two strains, the numbers in each strain is given by v_i, that compete through the availability of resource. The strains differ in the rate with which they sequester resource, c_i. The strain that sequesters the most resource will also be the most competitive strain.

At the end of the season both type of strains produce dispersers. To produce dispersers, the remaining free resource is converted into dispersers. To optimise the number of dispersers the best strategy would be to be prudent with resources. However, such a strategy backfires if the patch is shared with another strain that sequesters the resource faster and which will be the winner of the competition.

This is a typical scenario used in group selection studies: we have a situation where a strain could lose out in competition, in almost all patches, but where the premium that is gained in patches that are not shared, is sufficient to provide a selective advantage.

3. Model description

We will find out how evolution proceeds in such a system by analysing the dynamics in some detail. The within-patch dynamics are given by

$$\begin{aligned} \dot{u} &= -u(c_1 v_1 + c_2 v_2) + d(v_1 + v_2) \ , \\ \dot{v}_1 &= c_1 u v_1 - d v_1 \ , \\ \dot{v}_2 &= c_2 u v_2 - d v_2 \ , \end{aligned}$$

where u is the amount of free resource, and v_i is the number of mice of type i. Note that the total amount of resource $U = u + v_1 + v_2$ is constant. Using this, we can simplify the local dynamics to

$$\begin{aligned} \dot{v}_1 &= v_1(c_1(U - v_1 - v_2) - d) \ , \\ \dot{v}_2 &= v_2(c_2(U - v_1 - v_2) - d), \end{aligned} \tag{3.1}$$

and thus within a patch the local dynamics of v_1 and v_2 take the form of a Lotka-Volterra type interaction. It is a well-known result from this type of competition model that the local dynamics can result in a process of competitive exclusion in which the type with the highest c_i will be the dominant local competitor. In that case the system has 2 equilibria in which one species is present, and the other absent. The equilibrium in which which can maintain the highest population density, and in which thus the amount of free resource is most reduced, is stable and the other equilibrium is unstable, in the sense that it can be invaded by the other type. This means Fig. 3.1 shows a typical example of the dynamics in the patch, it shows how a more prudent type, that sequesters resources at a low rate, is outcompeted by a type that sequesters resource more readily.

Following the seeding of the patch, system (3.1) will determine the local dynamics. After the patch has incubated for an amount of time, say T, the season ends and the patch has to produce migrants. We assume that this happens through the conversion of all freely available resource that are then availably. These resources will be shared out pro-rata to types in the patch.

The amount of freely available resource at time T is given by $u(T) = U - s(T)$, where $s = v_1 + v_2$. The fraction of type 2 individuals is given by $f = v_2/s$, and consequently, the fraction of type 1 is than $1 - f = v_1/s$. Let the number of type k migrants in a patch that received i fundatrices of type 1, and j of type 2 be given by $m_k(i, j)$. We can then express these quantities as

$$\begin{aligned} m_2\left(v_1(0), v_2(0)\right) &= f(T)(U - s(T)) \ , \\ m_1\left(v_1(0), v_2(0)\right) &= (1 - f(T))(U - s(T)) \ . \end{aligned}$$

The number of migrants is proportional to the amount of free resource, so that more prudent types can produce more migrants. For a patch that is composed of a single type, for sufficiently long T, the number of free resources, and therefore the number of migrants is approximately d/c_i.

To fully specify the model we need to detail how the patches are seeded. We will denote the probability that a patch receives i fundatrices of type 1 and j of type 2 with $Q(i, j; N_1, N_2)$, where N_i is the average number of that type per patch. We will assume that Q, which details the way fundatrices are distributed over the patches, does not depend on the traits c_i. It does, of course, depend on the number of individuals that carry the trait through N_1 and N_2. As a consequence the total number of individuals in a patch depends only on the total number in the population:

$$P(n; N_1 + N_2) = \sum_{i=1}^{n} Q(n - i, i; N_1, N_2).$$

Furthermore, as the allocation of individuals to patches does not depend on their traits, a fundatrix is of type 2 with probability ϕ and of type 1 with probability $1 - \phi$ where $\phi = N_2/N$ and $N = N_1 + N_2$. Therefore, for a patch that receives n fundatrices in total, the distribution over the types is binomial, so that we have:

$$Q(i, j; N_1, N_2) = \binom{i+j}{i} (1 - \phi)^i \phi^j P(i + j; N). \tag{3.2}$$

The average density in the next season is found by harvesting all dispersing individuals from the patches, and exposing them to overwintering

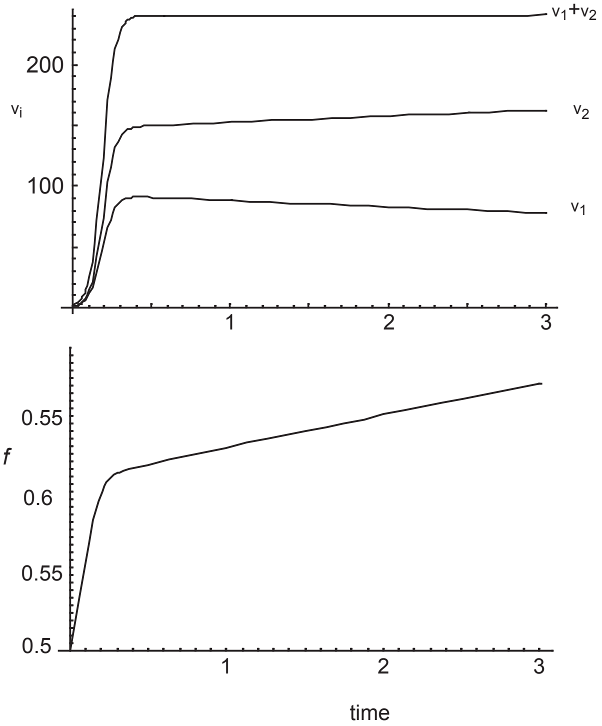

FIGURE 3.1. The local dynamics in a patch. In the top panel the densities of type 1 and type 2 versus time. Type 2 has a higher value of c and therefore this type is competitively superior. In the bottom panel the fraction of type 2 in the patch is depicted. Note how after an initial phase, which lasts as long as the local population has not reached its quasi equilibrium, this fraction changes fast. Following this phase a slow process of replacement takes place. Parameters: $c_1 = 0.1$, $c_2 = 0.11$, $U = 250$, $T = 3$, $d = 1$

mortality:

$$N_1' = \mu \sum_{i=0}^{\infty} \sum_{j=0}^{\infty} Q(i,j;N_1,N_2) m_1(i,j) \ ,$$

$$N_2' = \mu \sum_{i=0}^{\infty} \sum_{j=0}^{\infty} Q(i,j;N_1,N_2) m_2(i,j),$$

where μ is the survival between seasons. This completely defines the dynamics of the haystack model.

This model assumes that the local population growth is a purely deterministic process. This, of course, will rarely be true as in particular the initial phases of the colonisation of the local patches will go through a phase in which stochastic effects can dominate. This will give an advantage to faster growing types (larger c_i). These issues are discussed in more detail in [10].

4. Fitness calculation

The above description is sufficient to simulate the change in frequency of the two types in the population by simulation. However, rather than studying the dynamics, we are interested in how evolution will shape the parameter c_i. We will therefore calculate the rate of invasion of type c_2 in a population dominated by c_1. Once we have established the pattern of invadability we can from that conclude if it is possible to have evolutionarily stable levels of prudence, i.e. parameter c_1 that cannot be invaded by any c_2.

We will apply the concept of invasion dynamics to find out the pattern of evolutionary change. We will therefore assume that there is a resident population of which all individuals are take up resources with rate c_1. We assume that in this population individuals with rate c_2 very infrequently appear, as it would occur through a process of mutation. Because these mutants occur very infrequently, the resident population will converge to its equilibrium value. The dynamics of type 1, in the absence of type 2, is given by:

$$N_1' = \mu \sum_{i=0}^{\infty} Q(i,0;N_1,0) m_1(i,0) = \mu \sum_{i=0}^{\infty} P(i;N_1) m_1(i,0).$$

At equilibrium we have

$$N_1^* = \mu \sum_{i=0}^{\infty} P(i;N_1^*) m_1(i,0).$$

If in this population a new type appears, it will be very rare initially. To find out if such type can invade we will therefore calculate the rate of invasion when it is rare. If it is rare, it will have very little influence on the equilibrium density, which we therefore assume to be, to a good approximation, at N_1^*. The dynamics of type 2 when it is rare is then approximately given by:

$$
\begin{aligned}
N_2' &= \mu \sum_{i=0}^{\infty} \sum_{j=0}^{\infty} \left(Q(i, j; N_1^*, 0) + N_2 \left. \frac{\mathrm{d}Q(i, j; N_1, N_2)}{\mathrm{d}N_2} \right|_{N_2=0, N_1=N_1^*} \right) m_2(i, j) \\
&= \frac{\mu N_2}{N_1^*} \sum_{i=0}^{\infty} (i+1) P(i+1, N_1^*) m_2(i, 1),
\end{aligned}
$$

where we used $m_2(i, 0) = 0$. This shows that if type 2 is rare, one would not expect to find more than one individual of this type in a haystack. Following [11] we define the fitness of type c_2 in an environment dominated by c_1 as:

$$
S_{c_1}(c_2) = \frac{N_2'}{N_2} = \frac{\mu}{N_1^*} \sum_{i=0}^{\infty} (i+1) P(i+1, N_1^*) m_2(i, 1).
$$

Note that the dependence on the resident trait c_1 comes through both N_1^* and $m_2(i, j)$ both are potentially dependent on c_1. If $c_1 = c_2$ there is no selection. It is easy to show, using that if $c_1 = c_2$ then $m_2(i, 1) = m_1(i+1, 0)/(i+1)$, that $S_{c_1}(c_1) = 1$

This allows us to find out if a type carrying c_2 can invade a population dominated by c_1, and from this, deduce some properties of the evolutionary process. In figs. 4.1 and 4.2 we have shown such plots for 2 different choices of P. In fig. 4.1 the patches are seeded by a constant number of individuals. We see that in this case any trait can always be invaded by types that sequester marginally more resources. We can deduce that in this case the evolutionary process leads to an ever increasing value of c_i, which only stops at the point where the c_i is so large that the population can not persist. In fig. 4.2 the number of fundatrices is Poisson distributed. In this case for low values of c_1 types with a (somewhat) larger value than c_1 can invade, but for very high values of c_1 types with a lower value of c_1 can invade. If evolution would involve a sequence of invasions with small steps, this will eventually lead to the value found at the crossing of the two invasion boundaries: this is a type that cannot be invaded by other types which have a marginally different value of. This can be seen in the figure because below the crossing point the marginal fitness is negative (but only just for these parameter values).

5. Weak selection and marginal fitness calculation

We can work out how the process of evolution works under small mutational steps through knowing the marginal fitness. To derive the marginal fitness we will revisit the local dynamics. Much of the above argument is based on a process where the differences between c_1 and c_2 are small. In this case the pressure of selection is weak. We will derive an approximation of the selection coefficient under weak selection. To do so, we make use of the fact that if selection is weak the replacement of one strain by another through competition is a slow process. We will use throughout the shorthands $\epsilon = c_2 - c_1$ and $c = c_1$

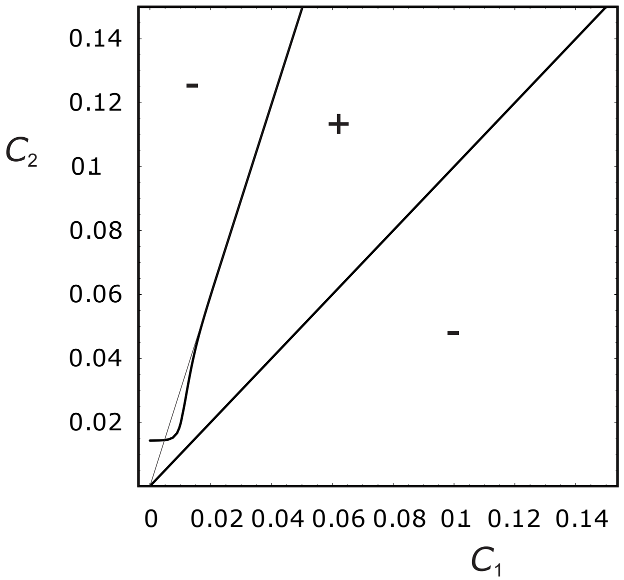

FIGURE 4.1. A pairwise invasibility diagram for the case where all occupied patches have received 3 fundatrices. Regions in which type 2 can invade a population of type 1, and for which combination of trait values the marginal fitness is positive (labelled with +) or negative (labelled −). If $c_2 > c_1$ there is a large range of values for which invasion if possible. The area just above the diagonal is labelled +: types with a marginally larger value of c than the resident population can always invade. This will lead to a process of replacement leading to increasing values of c.

The dynamics in the variables s, and f are given by:

$$\dot{s} = s\left[u(c + \epsilon f) - d\right], \tag{5.1}$$

$$\dot{f} = \epsilon f(1 - f)u, \tag{5.2}$$

and remember that $u = U - s$. If ϵ is small clearly the change in f is slow compared to the changes in s. For sufficiently large T the dynamics of u will settle on a quasi steady state, given by

$$\tilde{u}(\epsilon) = \frac{d}{c + \epsilon f}.$$

For small ϵ this is approximately $\tilde{u}(0)\left(1 - \epsilon\frac{f(0)}{c}\right)$. The dynamics of s will also settle at a quasi steady state $\tilde{s} = U - \frac{d}{c+\epsilon f}$. Based on this argument it is possible to find a approximation for the number of dispersers produced

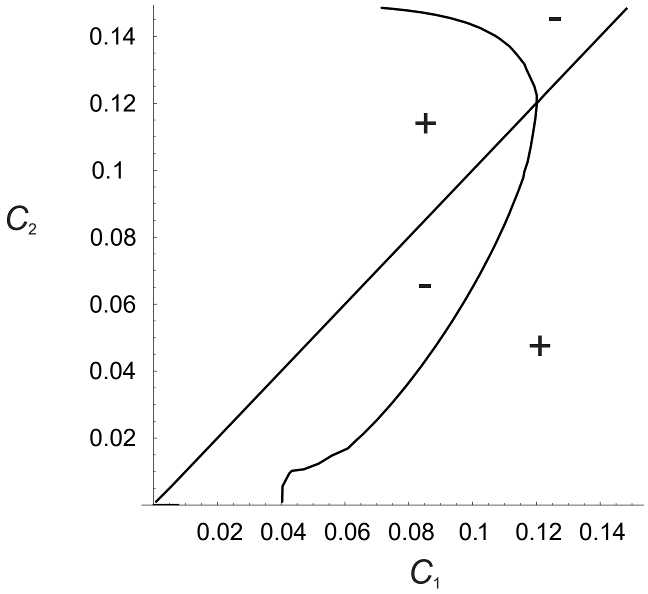

FIGURE 4.2. A pairwise invasibility diagram for the case where patches have received a number of fundatrices that is Poisson distributed. A process of repeated invasion of marginally different types will make that the evolutionary dynamics proceed to a value of c_1 for which the 2 curves cross. Here, marginally different types cannot invade (although the region of stability is small). Parameters: $U = 250$, $T = 3$, $d = 1$, $\mu = 0.15$

under weak selection (see appendix)

$$m_2(i,j) \approx \tilde{u}(0)f(0) - \epsilon\frac{\tilde{u}(0)}{c}f(0)^2 + \epsilon\tilde{u}(0)f(0)(1-f(0))g(0,T),$$

where $g(0,T) = \int_0^T u(t)\mathrm{d}t\Big|_{\epsilon=0}$ which for sufficiently large T is approximately

$$g(0,T) \approx \tilde{u}(0)T + \frac{1}{c}\ln\frac{\tilde{s}}{s(0)}.$$

The fitness is now given by

$$\frac{\mu}{N_1^*}\sum_{i=0}^{\infty}(i+1)P(i+1;N_1^*)\tilde{u}(0)\frac{1}{i+1}\left[1+\epsilon\left(-\frac{1}{c}\frac{1}{i+1}+\left(1-\frac{1}{i+1}\right)g(0,T)\right)\right]$$
$$= 1+\epsilon\frac{\sum_{i=1}^{\infty}P(i;N_1^*)\left[-\frac{1}{ci}+(1-\frac{1}{i})\left(\tilde{u}(0)T+\frac{1}{c}\ln\frac{\tilde{s}}{i}\right)\right]}{1-P(0;N_1^*)},$$

and the marginal increase in fitness is

$$\epsilon\sum_{i=1}^{\infty}\frac{P(i;N_1^*)}{1-P(0;N_1^*)}\left[-\frac{1}{ci}+\left(1-\frac{1}{i}\right)\left(\frac{dT}{c}+\frac{1}{c}\ln\frac{U-d/c}{i}\right)\right].$$

The marginal fitness can be used to find candidate end points of the evolutionary process. These are values of c for which the marginal fitness is zero. If these points in the phenotype space are stable against invasion, and if the evolutionary process leads towards them, they are called evolutionary stable states. Here, we are not so much interested in the value of the ESS, as in the interpretation of the marginal fitness equation. We will discuss two ways to interpret the marginal fitness

5.1. Inclusive fitness representation

To interpret the marginal fitness in terms of inclusive fitness, we will introduce the concept of relatedness. This is the normalised probability to pick two individuals of the same type from the same patch over and above the probability of picking two of the same type from the overall population. We show in the appendix that this is

$$R=\frac{\sum_{i=1}^{\infty}P(i;N_1^*)\frac{1}{i}}{1-P(0;N_1^*)}. \tag{5.3}$$

Using this, we can rewrite the marginal fitness as:

$$\epsilon\left[-\frac{R}{c}+(1-R)\frac{dT}{c}-\frac{1}{c}\sum_{i=1}^{\infty}\frac{P(i;N_1^*)}{1-P(0;N_1^*)}\left(1-\frac{1}{i}\right)\ln\frac{U-d/c}{i}\right].$$

The last term in the marginal fitness results from the difference in growth rate between individuals. In the initial stages of the exploitation of the patch the growth is approximately exponential, and a faster growing type will come to occupy a larger proportion of the patch. How long the period of approximate exponential growth lasts, depends on the logarithm of the number of fundatrices a patch receives.

One can interpret the terms in the sum as relatedness measures by defining

$$R_i'=\frac{1}{i},$$

that is, as the normalised probability to pick two identical individuals from a patch that was seeded by i individuals, relative to the probability in the population as a whole (see appendix). With this the last term in the marginal fitness can be written as

$$\sum_{i=1}^{\infty}\frac{P(i;N_1^*)}{1-P(0;N_1^*)}(1-R_i')\ln R_i'(U-d/c).$$

Strictly speaking we can thus interpret the marginal fitness in terms of a relatedness measures. Although this is formally correct, this is practically of little value. The usefulness of the relatedness measure comes, in part, through the fact that one can assess the relatedness through sampling of neutral genes.

By introducing the measures R_i' which depend on the number of fundatrices a patch receives, one can only assess such measures if one knows how many fundatrices a patch has received; information which would normally not be available after the incubation period.

It is, in fact, possible to link the sum $\sum_{i=1}^{\infty} \frac{P(i;N_1^*)}{1-P(0;N_1^*)}\left(1-\frac{1}{i}\right)\ln\frac{1}{i}$ to the average rate of finding a rare mutant in a patch (see appendix). This shows that it is, in principle, possible to uncover this information from a population, without having to know how many fundatrices funded a patch. However, where the relatedness is independent of the frequency of a particular gene in the population, this measure does depend on the frequency (hence the requirement of the gene to be rare) which makes the applicability limited.

Therefore, it is helpful to approximate the sum in the last term by Taylor expanding the logarithmic term around the average for $i = \frac{N_1^*}{1-P(0,N_1^*)}$, which if all patches that have at least one fundatrix go to quasi equilibrium is approximately $\frac{N_1^*}{1-P(0,N_1^*)} \approx \frac{\mu d}{c}$ so that we find for the last term in the fitness equation:

$$\sum_{i=1}^{\infty} \frac{P(i;N_1^*)}{1-P(0;N_1^*)}\left(1-\frac{1}{i}\right)\ln\frac{U-d/c}{i} \approx (1-R)\ln\left(\frac{cU-d}{\mu d}\right)$$

(see [13] for a similar argument and an application to the evolution of social behaviour in aphids). With this we can now write the marginal fitness as:

$$-R\frac{\epsilon}{c} + (1-R)\frac{\epsilon}{c}\left(dT + \ln\left(\frac{cU-d}{\mu d}\right)\right),$$

in which we recognise the effects of the change in the free resource which a change in c will cause (note $\frac{\mathrm{d}\tilde{u}(0)}{\mathrm{d}c} = -\frac{1}{c}$). This effect will contribute to the fitness through all related individuals. The term preceded by $(1-R)$ represents the effects of competition. A increase in c will increase the competitive ability.

Alternatively, we can partition the marginal fitness as

$$\underbrace{\frac{\epsilon}{c}\left(dT + \ln\left(\frac{cU-d}{\mu d}\right)\right)}_{-C} + \underbrace{R\,\frac{\epsilon}{c}\left(-1 - dT - \ln\left(\frac{cU-d}{\mu d}\right)\right)}_{B}.$$

A decrease the amount of resource sequestered, i.e. a negative ϵ, is costly in that the first term is negative and will constitute a negative direct effect, yet will result in a positive indirect effect and is therefore an act of altruism.

5.2. Group selection

For a group selection interpretation of the same model we will partition the fitness differently. A group, or multi-level, selection perspective considers the variation within and between patches. One can then interpret the advantage of an altruistic act as a consequence of the higher yield of groups which largely consist of altruists.

Now that we have an expression in terms of costs and benefits we can relate this to personal costs and benefits. We will denote the phenotype of individual j in patch i with x_{ij} which takes the value 0 if the individual has phenotype c_1, and 1 if it has phenotype c_2. We can assign the approximate personal fitness to individual j in patch i

$$W_{ij} = W_0 - Cx_{ij} + B\bar{x}_i,$$

where W_0 is the fitness if all individual carry trait c_1 (which will be 1, but for generality we keep this open) and $\bar{x}_i = \frac{1}{n_i}\sum_{j=1}^{n} x_{ij}$ is the mean phenotype among the individuals that i interacts with, which here is the same as the mean phenotype of the individuals in the patch. The number of fundatrices in patch i is n_i. It is straightforward, if tedious, to calculate the variation in fitness between groups $\mathrm{E}(\mathrm{var}_g(W)) = -(1-R)C\mathrm{var}(x)$, and the average variation within groups $\mathrm{var}(\overline{W}_i) = R(-C+B)\mathrm{var}(x)$, where $\mathrm{var}(x) = \phi(1-\phi)$ is the variation in the variable x.

We can now rewrite the marginal fitness as:

$$-C + RB = \underbrace{\frac{\mathrm{E}(\mathrm{var}_g(W))}{\mathrm{var}(x)}}_{\text{variation within patch}} + \underbrace{\frac{\mathrm{var}(\overline{W}_i)}{\mathrm{var}(x)}}_{\text{variation between patches}}.$$

6. Strong selection and fitness calculation

The above shows how the marginal fitness can be partitioned in the difference in variances within and between group, as it is frequently done in the context of multilevel (group) selection. However, to explain group selection verbally an argument along the lines of that of Maynard Smith [6] is sometimes given, stating even if altruists are at a competitive disadvantage to the point that they disappear in all patch, but the ones where they are the sole occupants. If the output in the solely occupied patches is sufficiently high, it is possible for altruists to invade a population of selfish individuals. This implies that selection is strong and is therefore not covered by the arguments above. To find out if this is the case in our model we consider the situation in which selection is strong (large ϵ) or the interaction is of sufficiently long duration so that local competition would always oust the inferior competitor.

If $\epsilon > 0$ than the type with c_2 will outcompete all c_1 in mixed patches. The fitness then is

$$\frac{\mu}{N_1^*}\sum_{i=0}^{\infty}(i+1)P(i+1, N_1^*)m_2(i,1).$$

If c_2 the local dynamics are sufficiently fast for type 2 to outcompete type 1, and for the dynamics of type 2 to settle at the equilibrium $u(\epsilon, T) \approx \frac{d}{c_2}$ we find that the fitness is

$$\frac{\mu}{N_1^*}\sum_{i=0}^{\infty}(i+1)P(i+1, N_1^*)\frac{d}{c_2} = \frac{d}{c_2}\frac{\mu}{N_1^*}\sum_{i=0}^{\infty} iP(i, N_1^*) = \frac{d\mu}{c_2}.$$

If $\epsilon < 0$ than the type with c_2 will be outcompeted all c_1 in mixed patches. Type 2 will be able to produce dispersers only in patches which had a type 2 fundatrix and no type 1 fundratices will. The fitness then is

$$\frac{\mu}{N_1^*} P(1, N_1^*) m_2(0, 1).$$

If we assume the local dynamics approximately go to equilibrium this is

$$\frac{d\mu}{c_2} \frac{P(1, N_1^*)}{N_1^*}.$$

Note that because the equilibrium level N_1^* depends on c_1, the fitness of type 2, does depend on the trait that type 1 carries because the number of dispersers that type 1 produces depends on how it exploits its local resources.

This argument formalises the group selection argument: a trait that is competitively inferior will be ousted from any group in which it would not have sole occupancy. However, if groups which exclusively consist of such a trait produce a sufficient excess of migrants so that it compensates for the loss due to competition, such a trait can invade in a population. This analysis also shows that this argument is based on a number of implicit assumptions. If the interaction within the groups is time limited, which is a realistic requirement for this mechanism to work, the difference between the traits needs to be large. Therefore the group selection argument assumes implicitly that strong selection is at work.

If selection is strong, this assumption gives a reasonably good indication of when an altruist can invade in a selfish population. In figure 6.1 we have plotted the invasion boundaries by numerically calculating our earlier derived fitness function. It can be seen that if the difference between c_1 and c_2 is sufficiently large that the agreement can be good (provided c_1 is not too small). This shows that if the difference between the two types is sufficiently large the replacement argument can be valid. If the difference is small, the argument is clearly not valid. Extrapolating the immediate replacement argument to marginal differences between two types leads to qualitatively incorrect approximation (the crossing point between the curves moves away to a point where no population can be sustained.)

7. Discussion

The analysis given above demonstrates that the inclusive fitness argument and the group selection argument can both be valid, albeit under different conditions. Inclusive fitness arguments, which rely on the fact that the fitness can be partitioned in additive components, normally requires weak selection. Moreover, if the distribution of genes depends on the trait, one can only validly infer the statistical association of two traits from the association of neutral genes if one assumes weak selection.

The partitioning of fitness in between and within group variance generally works if one actually can identify the groups to which it applies. It is

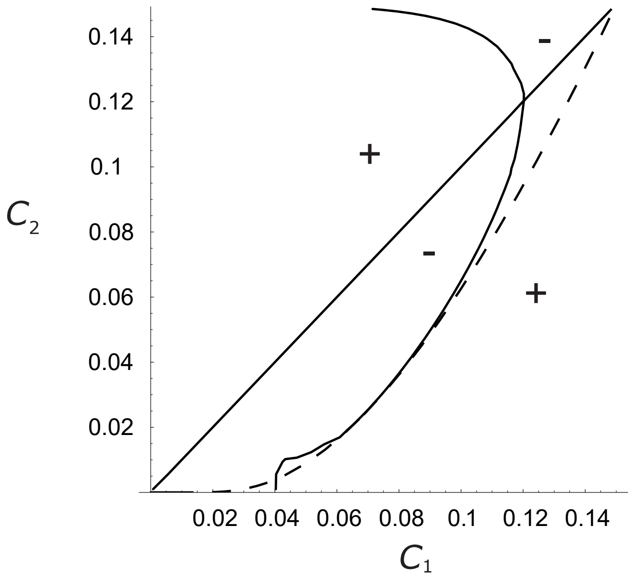

FIGURE 6.1. A pairwise invasibility diagram for the case where patches have received a number of fundatrices that is Poisson distributed to which the approximate invasion boundary is added for which it is assumed that replacement is immediate and complete and that the local dynamics go to equilibrium within the lifetime of a local patch (dashed line). Note that there is good agreement if $c_1 \gg c_2$ and c_1 not too small. Parameters: $U = 250$, $T = 3$, $d = 1$, $\mu = 0.15$

generally not possible to independently measure or assess these fitness components in a real world population.

For the group selection argument based on local replacement to work, the traits under study need to be sufficiently different. This requires strong selection, as it would result from a finite, and sufficiently different set of different phenotypes, or from sufficiently large mutation steps. What the group selection argument can predict is whether two types can replace each other or whether they can coexist, but the argument is ill-suited to predict the long term course of evolution under small mutations steps.

Whilst this analysis confirms that most of the views in the debate on kin and group selection can be shown to operate in the haystack model, it also demonstrates why this debate is so persistent. In most of the mathematical arguments that are presented in the literature, arbitrarily chosen fitness functions are used. Mostly these define what the fitness of different types is, without being specific how the fitness will vary under a change of trait values. Therefore, it is ambiguous whether these statements refer to a weak or a strong selection scenario. I hope that the above analysis demonstrates how these ecological details are of importance in interpreting the results from mathematical models as used in evolutionary ecology.

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References

[1] Darwin, C. (1871) The Descent of Man, and Selection in Relation to Sex, John Murray, London

[2] Darwin, C. (1859) On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life, John Murray, London

[3] Fisher, R.A. (1930). The Genetical Theory of Natural Selection. Oxford: Clarendon Press.

[4] Haldane, J.B.S. (1955) "Population Genetics". New Biology 18, 34-51.

[5] Hamilton W.D. (1964) The genetic evolution of social behaviour, II. *J. theor. Biol.* 7, 17-52.

[6] Maynard Smith, J.S. (1964) Group selection and kin selection. *Nature* 201, 1145-1147

[7] Grafen, A. (1984) A geometric view of relatedness. Oxford Surveys in Evolutionary Biology 2: 28-89.

[8] Wynne-Edwards, V.C. (1962) Animal Dispersion in relation to social behaviour. Oliver and Boyd, Edinburgh.

[9] Jansen, V.A.A. and Mulder, G.S.E.E. (1999) Evolving biodiversity. *Ecol. Lett.* 2: 379–386.

[10] Jansen, V. A. A. and Vitalis, R. (2007) The evolution of dispersal in a Levins' type metapopulation model. *Evolution* 61, 2386-2397.

[11] Metz, J.A.J, Nisbet, R.M, and Geritz, S.A.H. (1992) How should we define fitness for general ecological scenarios. Trends. Ecol. Evol. 7, 198-202.

[12] Wilson, D.S. A theory of group selection (1975) Proc. Natl. Acad. Sci. USA 72, 143-146.

[13] Bryden, J. and Jansen,V. A. A. (2010) The impact of clonal mixing on the evolution of social behaviour in aphids. *Proc. Roy. Soc. B: Biological Sciences* 277 (2010), 1651-1657

[14] Queller D.C., Goodnight K.F. (1989) Estimating Relatedness using Genetic Markers. *Evolution* 43:258-275. doi: 10.1111/j.1558-5646.1989

Appendix A. Relatedness calculation

We first observe that the probability to pick an single individual from a patch is simply ϕ. This follows directly from the assumption that the types are distributed according to a binomial distribution in patches of equal size.

Let us call the probability to pick, with replacement, two individuals of type 1, from the same patch

$$p_1(\phi) = \frac{\sum_{i=1}^{\infty}\sum_{j=0}^{\infty} Q(i,j;N(1-\phi),\phi)\left(\frac{i}{i+j}\right)^2}{1-Q(0,0;N(1-\phi),\phi)},$$

and likewise

$$p_2(\phi) = \frac{\sum_{i=0}^{\infty}\sum_{j=1}^{\infty} Q(i,j;N(1-\phi),\phi)\left(\frac{j}{i+j}\right)^2}{1-Q(0,0;N(1-\phi),\phi)},$$

where ϕ is the fraction of individuals of type 2, and N is the total population size. Note that $p_1(\phi) = 1-2(1-\phi)+p_2(\phi)$

The relatedness is measure of how likely it is that two genotypically individuals are found in the same haystack, relative to the probability of picking the same individuals in the population at large (this is $(1-\phi)^2+\phi^2$). This measure is normalised so that if haystacks only contain a single type the relatedness is unity. The relatedness is defined as [14]

$$R = \frac{p_1(\phi)-(1-\phi)^2+p_2(\phi)-\phi^2}{1-(1-\phi)^2-\phi^2} = \frac{p_2(\phi)-\phi^2}{\phi(1-\phi)}.$$

If we now evaluate this $p_2(\phi)$ to find

$$\begin{aligned}\frac{\sum_{i=0}^{\infty}\sum_{j=1}^{\infty} P(i,j;N(1-\phi),\phi)\left(\frac{j}{i+j}\right)^2}{1-P(0;N)} &= \frac{\sum_{n=1}^{\infty}\sum_{j=0}^{n} P(n;N)\frac{n!}{j!(n-j)!}\left(\frac{j}{n}\right)^2}{1-P(0;N)}\\ &= \frac{\sum_{n=1}^{\infty} P(n;N)\left(\frac{\phi(1-\phi)}{n}+\phi^2\right)}{1-P(0;N)}\\ &= \phi(1-\phi)\frac{\sum_{n=1}^{\infty} P(n;N)\frac{1}{n}}{1-P(0;N)}+\phi^2,\end{aligned}$$

from which (5.3) follows.

Similarly, we have for R'_n :

$$\begin{aligned}R'_n &= \frac{\frac{\sum_{i=0}^{n} P(i,n-i,N(1-\phi),N\phi)((\frac{i}{n})^2+(\frac{i-n}{n})^2)}{P(n,N)}-\phi^2-(1-\phi)^2}{1-\phi^2-(1-\phi)^2}\\ &= \frac{\left(\sum_{i=0}^{n}\phi^i(1-\phi)^{n-i}\frac{n!}{i!(n-i)!}(2\left(\frac{i}{n}\right)^2-2\frac{i}{n}+1)\right)-\phi^2-(1-\phi)^2}{2\phi(1-\phi)}\\ &= \frac{\left(\frac{\phi(1-\phi)}{n}+\phi^2\right)-\phi^2}{\phi(1-\phi)} = \frac{1}{n}.\end{aligned}$$

Appendix B. Interpretation of logarithmic term

Here we will demonstrate that the term $\sum_{i=1}^{\infty} \frac{P(i;N_1^*)}{1-P(0;N_1^*)} \left(1-\frac{1}{i}\right) \ln \frac{1}{i}$ can be interpreted in terms of a sampling procedure.

The sampling procedure we apply is as follows: identify a rare mutant, which is not selected for in the process. Within a patch, keep drawing individuals, with replacement, until this mutant is encountered. Subtract 1 from the number of individuals, and, if the result is larger than 0, take the average of the reciprocal of this number by sampling over different patches.

In a patch containing i mutants among n individuals, the probability of encountering the mutant for the first time after k draws is given by the hypergeometric distribution $\frac{i}{n}\left(1-\frac{i}{n}\right)^k$. The expectation of reciprocal of positive values of $k-1$, provided $i>0$, is

$$\sum_{k=2}^{\infty} \frac{i}{n}(1-\frac{i}{n})^k \frac{1}{k-1} = -\frac{i}{n}\frac{n-i}{n} \log\left(\frac{i}{n}\right).$$

If $i=0$ then

$$\lim_{i/n\to 0} \sum_{k=2}^{\infty} \frac{i}{n}\left(1-\frac{i}{n}\right)^k \frac{1}{k-1} = 0.$$

When sampled over occupied patches, and if ϕ is the mutant's fraction of the population the expectation is

$$-\sum_{n=1}^{\infty} \frac{P(n,N)}{1-P(0,N)} \sum_{i=0}^{n} \frac{n!}{i!(n-i)!}\phi^i(1-\phi)^{n-i}\frac{i}{n}\frac{n-i}{n} \ln\left(\frac{i}{n}\right).$$

For small ϕ this is approximately

$$-\sum_{n=1}^{\infty} P(n,N)\left(1-\frac{1}{n}\right)\ln\left(\frac{1}{n}\right).$$

Appendix C. Derivation of marginal fitness under weak selection

The find the change over the incubation time of the fraction of type 2, f, we will solve (5.2). To do so we first rearrange:

$$\left(\frac{1}{f}+\frac{1}{1-f}\right)\mathrm{d}f = \epsilon u(\epsilon,t)\mathrm{d}t,$$

which we can solve by integration from $t=0$ to $t=T$:

$$\ln\frac{f(T)}{f(0)} - \ln\frac{1-f(T)}{1-f(0)} = \epsilon\int_0^T u(\epsilon,t)\mathrm{d}t$$

and thus

$$f(T) = \frac{f(0)}{f(0)+(1-f(0))e^{-\epsilon\int_0^T u(\epsilon,t)\mathrm{d}t}}.$$

To make the dependence of u on ϵ explicit we have written $u(\epsilon, T)$. If ϵ is small the we can now find the approximate value of $f(T)$ as:

$$f(T) \approx f(0) + \epsilon f(0)(1 - f(0)) \int_0^T u(0,t)\mathrm{d}t.$$

To solve the integral we will turn our attention to s. The dynamics of s for $\epsilon = 0$ are given by the logistic growth model: from a positive initial condition the total number of individuals will increase and saturate at $\tilde{s}(0)$. The solution to the logistic equation is

$$s(t) = \frac{s(0)\tilde{s}}{s(0) + (\tilde{s} - s(0))e^{-c\tilde{s}t}}. \tag{C.1}$$

This also prescribes the dynamics of $u(t) = U - s(t)$.

For $g(0,T) = \int_0^T u(0,t)\mathrm{d}t$ we have

$$g(0,T) = \tilde{u}(0)T + \frac{1}{c}\ln\frac{s(T)}{s(0)}.$$

For sufficiently large T this is approximately

$$g(0,T) \approx \tilde{u}(0)T + \frac{1}{c}\ln\frac{\tilde{s}}{s(0)}.$$

If we assume that the resource levels settle on the quasi equilibrium $u(0,T) = \tilde{u}$ we find that if there is a marginal difference in the traits number of type 2 dispersers that will be produced for large T is equal to

$$\begin{aligned} m_2(i,j) &= u(\epsilon, T)f(T) \\ &\approx \tilde{u}(0)f(0) - \epsilon\frac{\tilde{u}(0)}{c}f(0)^2 + \epsilon\tilde{u}(0)f(0)(1 - f(0))g(0,T). \end{aligned}$$

Appendix D. Calculation of within and between group variance in fitness

Assume a local group is seeded by n_i individuals with k with trait value c_2, and $n - k$ with trait value c_1. Such a group has as mean value $\bar{x}_i = \frac{k}{n_i}$, and the variance in x in the local group is

$$\mathrm{var}_g(x) = \frac{1}{n_i}\sum_{j=1}^{n_i}\left(x_{ij} - \frac{k}{n_i}\right)^2 = \frac{k}{n_i}\left(1 - \frac{k}{n_i}\right).$$

The expected value of this variance is

$$\mathrm{E}[\mathrm{var}_g(x)] = \sum_{n_i=1}^{\infty}\sum_{k=0}^{n_i} P(n_i, N)\binom{n_i}{k}\phi^k(1-\phi)^{n_i-k}\frac{i}{n}\left(1 - \frac{k}{n_i}\right)\phi(1-\phi)(1-R).$$

The expected value of the group mean is

$$\mathrm{E}(\bar{x}_i) = \sum_{n_i=1}^{\infty}\frac{P(n_i, N)}{1 - P(0, N)}\sum_{k=0}^{n}\binom{n_i}{k}\phi^k(1 - \phi)^{n_i-k}\frac{k}{n_i} = \phi,$$

and the variance

$$\begin{aligned}\mathrm{var}(\bar{x}_i) &= \sum_{n_i=1}^{\infty}\sum_{k=0}^{n_i}\frac{P(n_i,N)}{1-P(0,N)}\binom{n_i}{k}\phi^k(1-\phi)^{n_i-k}(\frac{k}{n_i}-\phi)^2\\ &=\sum_{n=1}^{\infty}\frac{P(n,N)}{1-P(0,N)}\phi(1-\phi)\frac{1}{n}=\phi(1-\phi)R.\end{aligned}$$

The overall variance in x is given by $\mathrm{var}(x)=\phi(1-\phi)$.

A group selection interpretation partitions the fitness in the variation in fitness within and between patches. Now that we have identified the individual fitness we can calculate the average mean fitness within a patch j as

$$\overline{W}_i=\frac{1}{n_i}\sum_{j=1}^{n}W_{ij}=W_0+(-C+B)\bar{x}_i.$$

The average within patch variance is given by

$$\begin{aligned}\mathrm{E}(\mathrm{var}_g(W)) &= \mathrm{E}(\frac{1}{n_i}\sum_{j=1}^{n_i}(W_{ij}-\overline{W}_i)^2)\\ &= -C\mathrm{E}(\frac{1}{n_i}\sum_{j=1}^{n_i}(x_{ij}-\bar{x}_i)^2)\\ &= -C\mathrm{E}[\mathrm{var}_g(x)]=-C\phi(1-\phi)(1-R).\end{aligned}$$

The variance between the mean fitness of patches is

$$\begin{aligned}\mathrm{var}(\overline{W}_i) &= \mathrm{E}[(\overline{W}_i-W_0-(-C+B)\phi)^2]\\ &= (-C+B)\mathrm{E}[(\bar{x}_i-\phi)^2]\\ &= (-C+B)\mathrm{var}(\bar{x}_i)\\ &= (-C+B)\phi(1-\phi)R.\end{aligned}$$

Vincent A. A. Jansen
School of Biological Sciences
Royal Holloway, University of London,
Egham, Surrey TW20 0EX, United-Kingdom
e-mail: `vincent.jansen@rhul.ac.uk`