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Detecting kin selection at work using inclusive fitness

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A recent model shows that altruism can evolve with limited migration and variable group sizes, and the authors claim that kin selection cannot provide a sufficient explanation of their results. It is demonstrated, using a recent reformulation of Hamilton's original arguments, that the model falls squarely within the scope of inclusive fitness theory, which furthermore shows how to calculate inclusive fitness and the relevant relatedness. A distinction is drawn between inclusive fitness, which is a method of analysing social behaviour; and kin selection, a process that operates through genetic similarity brought about by common ancestry, but not by assortation by genotype or by direct assessment of genetic similarity. The recent model is analysed, and it turns out that kin selection provides a sufficient explanation to considerable quantitative accuracy, contrary to the authors' claims. A parallel analysis is possible and would be illuminating for all models of social behaviour in which individuals' effects on each other's offspring numbers combine additively.

Keywords: inclusive fitness; altruism; relatedness; structured populations; varying group size; public goods game

1. INTRODUCTION

The evolution of altruism remains an active topic in biological research, but the power and the scope of inclusive fitness theory (Hamilton 1964, 1970) remain largely unappreciated. Killingback *et al.* (2006) recently claim to have demonstrated a new mechanism for evolving altruism that cannot be fully accounted for by kin selection. Here, we put this claim to the test using inclusive fitness theory, developing an approach that could equally be taken for many other papers.

The test is conducted using a new formulation of inclusive fitness theory recently proposed by Grafen (2006), which fulfils Hamilton's original intentions by combining the best features of his 1964 and 1970 derivations, eliminating minor flaws, incorporating uncertainty, permitting arbitrary ploidies and arbitrary genetic architecture, and being fully explicit about optimization. Furthermore, Hamilton's original generality of interactional structure is retained; individuals may engage in different numbers of social interactions, and the interactions may all be different in nature. Most subsequent rederivations of inclusive fitness have insisted on just one kind of interaction that individuals all engage in the same number of times.

All of the inclusive fitness results used in the present paper are from Grafen (2006): the proof that the model falls within the inclusive fitness framework, the expression for inclusive fitness, the formula for relatedness and the result that inclusive fitness is maximized by selection. Other important bodies of work on inclusive fitness are by Frank (1998; see also Taylor & Frank 1996), Taylor and co-workers (e.g. Taylor 1990, 1996; Taylor *et al.* 2000) and Rousset and co-workers (Rousset 2004). The generality, directness and the immediate applicability of Grafen (2006) are bought at the cost of saying nothing about the link between relatedness and common ancestry, which is a central element of many other papers. The assumption of additivity is also relaxed in some of them.

An important distinction made by Hamilton (1975) is emphasized here. Inclusive fitness theory is very general and applies to genetic similarity, however caused, whether by common ancestry, assortation of genotypes or kin recognition. As Grafen (2006) shows, provided social interactions combine additively in determining offspring numbers, there is (almost always) a relatedness that can be calculated such that the direction of gene frequency change is determined by inclusive fitness. However, it is useful to reserve the term 'kin selection' for situations in which the relatedness arises through common ancestry. (The situation in which the relatedness cannot be defined occurs when a quantity called the 'Hamilton residual' cannot be rendered zero by choice of relatedness, and is discussed in detail in section 3.3 of Grafen (2006). It arises when the actors are genetically representative of the population, so that the gene frequency being studied has an exactly zero correlation with the altruistic phenotype.)

Section 2 describes the model of Killingback *et al.* (2006), and shows how it can be represented within the framework of Grafen (2006), and thus derives a formula for the relatedness with which inclusive fitness is maximized. The analysis of that section could be repeated with few changes for many other models. Section 3 moves on to the particularities of the current model. It defines the model more precisely, gives details of the demographic properties of the model and calculates the relatedness that would arise within a group through common ancestry alone. This relatedness allows predictions to be made

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about selection, and a computer calculation of the model (detailed in Appendix A) allows Section 4 to put those predictions to the test.

2. THE VARYING GROUP SIZE MODEL OF KILLINGBACK *ET AL.* (2006)

Killingback *et al.* (2006) study a 'public goods' game in a grouped asexual population. Each individual j plays a value x_j, which is directly a cost. x_j is constrained to lie between 0 and V. Each individual receives, as a benefit, the average value of x_i in the group, multiplied by an 'interest rate' k. Let Γ_j be the set of individuals in the group to which j belongs, and n_j be the number in that group. We can write the net pay-off in two equivalent ways:

$$\begin{aligned} w_j &= V - x_j + \frac{k}{n_j}\sum_{i\in\Gamma_j} x_i, \\ w_j &= V - x_j\left(1-\frac{k}{n_j}\right) + \frac{k}{n_j}\sum_{i\in\Gamma_j, i\neq j} x_i. \end{aligned} \tag{2.1}$$

V is the baseline fitness in the absence of social interactions. The two forms suggest two equivalent ways forward. The upper version includes j in the recipients of her own action, as part of the group, and counts the 'cost' as the full x_j; the lower version nets off the benefit to herself from her cost. We follow the first course for simplicity; it will affect the details of the analysis, but the outcome is obviously the same.

Killingback *et al.* (2006) further assume that individuals are haploid, asexual reproduction occurs proportionally to fitness, each individual has an independent chance of dispersal with probability d so that dispersers join another group at random and then population-wide mortality reduces the mean group size to the prescribed value m. Groups will thus vary in size. The total population size was fixed at 500.

The authors found that with a mean group size of 5, altruism was broadly selected when d was less than approximately 0.125 for $k=2$ and when d was less than approximately 0.27 for $k=3$. The obvious explanation is that limited dispersal (low d) brings about intra-group relatedness, which favours altruism to group members. The authors reject this explanation as insufficient. They state that in another unpublished study, they found that a different game did not evolve altruism in the same demographic circumstances. However, this is a weak argument as there may well be important differences between the two games (it would be premature to investigate these before both models have undergone peer scrutiny as part of the process of publication). They also propose that there might be a new mechanism for evolving altruism, and suggest it could be based on a combination of the fact that at very small group sizes higher x is individually advantageous, that small group sizes arise in the model, and Simpson's paradox (Blyth 1972). This proposal is rather vague: as we shall see, it is also unnecessary.

Grafen's (2006) new formulation of inclusive fitness assumes additivity of fitness interactions by representing the number of successful gametes of an individual j as

$$w_j = b_{jje} + \sum_{i,t\neq e} b_{ijt},$$

b_{ijt} is the effect of individual i on the number of successful gametes of individual j, when i is acting in 'role' t in relation to j. Role is a crucial concept employed by Hamilton (1964) and revived more formally by Grafen (2006). A role will typically delineate a situation in which a decision needs to be made, for example, 'parent towards offspring it is feeding' or 'first of two strangers to meet at a food resource towards the second'. The significance of roles is that the relatedness is defined not for individuals, but for each role. There is a special role e for ego, in which an individual affects her own number of successful gametes through non-social action.

We now show that the model of Killingback *et al.* (2006) falls within the scope of inclusive fitness theory by showing how to define the b_{ijt} to establish an equivalent formula for offspring number. This is simply done using equation (2.1). Let g be the role in which an individual plays the public goods game with her group members. Here are the two different ways to represent the model in inclusive fitness terms

$$b_{ijt} = \begin{cases} V - x_i & t = e, \quad i = j \\ \dfrac{k}{n_j}x_i & t = g, \quad j \in \Gamma_i \\ 0 & \text{otherwise} \end{cases}$$

$$b_{ijt} = \begin{cases} V - x_i(1-k/n_j) & t = e, \quad i = j \\ \dfrac{k}{n_j}x_i & t = g, \quad j \neq i, \quad j \in \Gamma_i \\ 0 & \text{otherwise} \end{cases} \tag{2.2}$$

We will pursue the analysis with the upper version. The key assumption here is just the additivity of fitness interactions. The definitions of the b_{ijt} are needed to implement the formulae of Grafen (2006) for inclusive fitness and relatedness. Non-additive situations can be handled by, for example, Taylor & Frank (1996).

This expression in terms of b_{ijt} is the crucial step: it follows from section 3.1 of Grafen (2006) that gene frequency change will favour types of individual with higher inclusive fitness. The formula for the inclusive fitness of individual i is

$$b_{iie} + \sum_{t\neq e} r^t \sum_j b_{ijt} = V - x_i + rn_i k\frac{x_i}{n_i} = V + x_i(rk-1), \tag{2.3}$$

where the relatedness r is to all group members (including self), and we drop the superscript t because there is only one role in this simple example. The mean value of x will therefore increase if k is high enough, and specifically if $rk-1>0$. The critical value of k, at which the mean of x remains unaltered, is

$$k_{\text{crit}} = \frac{1}{r}. \tag{2.4}$$

The group size n_i cancels out because each individual gives the same amount to the group as a whole, whatever its size.

But what is the value of r? The notation r_{IF} is introduced to mean the relatedness that makes inclusive fitness work. Equation (2.3) of Grafen (2006) shows that in general

$$r_{\text{IF}} = \frac{\sum_i\sum_j (p_j - p)b_{ijt}}{\sum_i\sum_j (p_i - p)b_{ijt}}. \tag{2.5}$$

(The formula fails when the denominator equals 0—see Grafen (2006), section 3.3.) Note this depends on a particular p-score, which is either a gene frequency or a weighted linear sum of gene frequencies. The evolution of a p-score depends on inclusive fitness calculated with its value of r_{IF}. r_{IF} also depends on all the demographic details and the current state of the population. Thus, in line with the very general conditions for inclusive fitness, there is a great deal of complexity here, reflecting the complexity in the original model. It may be worth pointing out that if we inserted an extra dispersal stage so that the groups had the same size distribution but individuals were allocated to groups at random, it is clear that $r_{\mathrm{IF}}=0$ and so in that case inclusive fitness would equal $V-x_i$ and selection would reduce x as far as it could.

In an asexual one-locus model, the useful p-score to study is x itself. Thus, we let $p_j=x_j$, and substitute using equation (2.2), and define $\tilde{x}_j$ as the average value of x in the group to which j belongs. It is easy to show that $r_{\mathrm{IF}}=\mathrm{Cov}[x_i,\tilde{x}_i]/\mathrm{Var}[x_i]$, which could be expected from the kin selection approach of Taylor & Frank (1996), but note that the weighting by b_{ijt} has automatically ensured the appropriate weighting of different-sized groups. We further consider the situation in which there are only two genotypes with population frequencies $1-\gamma$ and γ, playing distinct values x^1 and x^2, and let a^1 be the expected frequency of type 1 in the group of a randomly chosen individual of type 1, and *mutatis mutandis* for a^2. It is easy to show that

$$r_{\mathrm{IF}}=\frac{x^1\frac{a^1-(1-\gamma)}{1-\gamma}-x^2\frac{a^2-\gamma}{\gamma}}{x^1-x^2}. \tag{2.6}$$

This form shows that all the necessary demographic details for the polymorphic model (as defined in Appendix A) are encapsulated in a^1 and a^2. In particular, the effects of both the mean group size and the variability in group size are included in them.

The values of a^1 and a^2 will be affected by all factors influencing genetic similarity of group members. If only common ancestry is important, then we would say that kin selection explained the results. Section 3 derives an expression for the value of r that measures the genetic similarity among group members that would be expected from common ancestry alone, r_{anc}, which is called the *ancestral relatedness*.

Whether $r_{\mathrm{IF}}=r_{\mathrm{anc}}$ will be answered here by direct calculations for this particular model. The same question is considered analytically in general contexts by, for example, Wild & Taylor (2004) and Rousset (2004), though it is usually phrased as whether identity-by-descent suffices to calculate relatedness. So far as I am aware, none of the existing general results apply directly and unequivocally when there are groups of different sizes. There is a general sense that in the absence of genetic discrimination, indeed $r_{\mathrm{IF}}=r_{\mathrm{anc}}$. Future work will surely provide an analytic result that encompasses the present model.

It is worth noting that the analysis so far could be replicated without difficulty for any model that can be brought within the scope of inclusive fitness theory by establishing additivity of fitness interactions. From now on, the analysis becomes more particular.

3. THE EXTENT OF COMMON ANCESTRY

In order to investigate the extent of common ancestry in groups, we need to define the model more precisely. Killingback *et al.* (2006) fixed their population size at 500, and obtained the number of offspring of the individuals in the parental generation from a multinomial distribution with probabilities proportional to fitnesses. Here an infinite population is assumed to ease analysis, and the appropriate limiting case of the multinomial distribution is assumed: each individual has a Poisson number of offspring with a mean proportional to its fitness, scaled so that the mean of the individual Poisson means equals 1. Parents do not survive to the next generation. Contrariwise, the original model can be understood as making the Poisson assumption, but then conditioning on the total number of offspring being 500. The size of the population is not implicated in the authors' arguments about the selective pressures at work, so it is fair to study the infinite population model to investigate their conclusions.

Two models are defined in Appendix A, which implement the assumptions above in a conceptually straightforward way. A monomorphic model with just one value of x present is used to calculate ancestral relatednesses, and a polymorphic model with two genotypes each with its own value of x is used to calculate the direction of selection at different values of the 'interest rate' k.

The individuals in a group whose ancestry within the group goes back to the same immigrant are called a clone, and the distribution of clone size depends on the number of generations since the immigrant's arrival. Calculations in Appendix B reveal that after G generations, the distribution of clone size in the monomorphic model has

$$\text{mean}=(1-d)^G \qquad \text{variance}=\frac{(1-(1-d)^G)(1-d)^G}{d},$$

while the distribution of the size of a whole group has

$$\text{mean}=m, \qquad \text{variance}=\frac{m}{(2-d)d}.$$

If we pick an individual at random, she is likely to be in a larger than average clone and larger than average group. The 'experienced clone size' has

$$\text{mean}=\frac{1}{d(2-d)}, \qquad \text{variance}=\frac{(1-d)^2(3-2d)}{(2-d)^2d^2(3-3d+d^2)},$$

and the 'experienced group size' has

$$\text{mean}=m+\frac{1}{d(2-d)},$$

$$\text{variance}=\frac{m}{(2-d)d}+\frac{(1-d)^2(3-2d)}{(2-d)^2d^2(3-3d+d^2)}.$$

Now we turn to calculating the ancestral relatedness. We use the definition of relatedness in equation (2.5), and apply it to a p-score that indicates belonging to a very rare clone as distributed across the population. Hence, we assume that there has only ever been one arrival of that clone in any one group, and that the clone makes only a zero fraction of the population as a whole, so that $p=0$. We set $p_i=1$ for clone members and zero for others, and use equation (2.2) to obtain

$$r_{\mathrm{anc}}=\frac{\sum_i\sum_{j\in\Gamma_i}p_j\frac{k}{n_i}x}{\sum_i\sum_{j\in\Gamma_i}\frac{k}{n_i}x}.$$

We can cancel kx and note that $\sum_{j\in\Gamma_i} 1/n_i = 1$. If we let c_i be the number of individuals in the group that are a member of the clone, then $\sum_{j\in\Gamma_i} p_j = c_i$. This yields

$$r_{\mathrm{anc}} = \frac{\sum_i \sum_{j\in\Gamma_i} \frac{p_j}{n_i}}{\sum_i \sum_{j\in\Gamma_i} \frac{1}{n_i}} = \frac{\sum_i \frac{1}{n_i} \sum_{j\in\Gamma_i} p_j}{\sum_i \sum_{j\in\Gamma_i} \frac{1}{n_i}} = \frac{\sum_i \frac{c_i}{n_i}}{\sum_i 1}. \tag{3.1}$$

Appendix C finds an analytic expression for this quantity in the monomorphic model, which allows it to be calculated. r_{anc} will be the same for all loci, but may differ from r_{IF} owing to assortation or genetic discrimination at the x locus. If selection follows inclusive fitness with this relatedness, that is, if $r_{\mathrm{IF}} = r_{\mathrm{anc}}$, then only kin selection is at work in the model.

4. RESULTS

Selection was measured in the polymorphic model of appendix A at $k_{\mathrm{anc}} = 1/r_{\mathrm{anc}}$, and at 1% above and 1% below k_{anc}. The theory tells us that selection proceeds according to $k_{\mathrm{crit}} = 1/r_{\mathrm{IF}}$. If $r_{\mathrm{IF}} = r_{\mathrm{anc}}$ then $k_{\mathrm{crit}} = k_{\mathrm{anc}}$, and selection should be neutral at k_{anc}, favour higher x at 1% above, and lower x at 1% below.

The analytic values of r_{anc} from equation (3.1) were derived in Appendix C and are shown in figure 1 for the parameter values used by Killingback *et al.* (2006), along with the relatednesses r_{IF} from equation (2.5) measured in the polymorphic model. Clearly, the relatedness is very high at low dispersal rates, and comes down to approximately 0.2, which is the inverse of the mean group size and reflects the fraction of the group the individual itself comprises. The virtual equality of r_{anc} and r_{IF} shows that common ancestry does explain the selection in the model to considerable numerical accuracy.

Figure 2 has further results from the polymorphic model. It shows how closely the ancestral relatedness predicts the direction of selection. To pursue the discrepancies would involve numerical analysis and questions of machine accuracy. There is no clear biological issue causing the minute discrepancies from the effects of common ancestry.

It is of interest to consider fig. 3 of Killingback *et al.* (2006) in relation to our figure 1. With $k = 2$, the current model would predict selection to increase contributions when d is less than approximately 0.125, and to decrease it for higher d. This is fully consistent with their fig. 3(a). For $k = 3$, theory predicts the break point to lie between 0.25 and 0.3. This is again fully consistent. The most significant discrepancy is that many of the points in their fig. 3 are intermediate between $x = 0$ and 5, and this is probably due to their mutational scheme which will tend to push away from boundaries, relatively more strongly where selection is weak. The theory matches their findings.

The anomalous behaviour of $d = 0$ in both parts of fig. 3 of Killingback *et al.* (2006) is simply explained. In the absence of any migration, their population of 500 individuals would find itself eventually all in one group for the rest of time; within any one closed group, contributions must be selected downwards.

The unavoidable conclusion is that no force other than common ancestry is required to explain the detailed quantitative pattern of selection when the public goods game is played in varying-sized groups, as modelled by Killingback *et al.* (2006). It is wholly unsurprising that kin selection plays some role, as it is inevitable that kinship ties build up in groups in which siblings are likely to be present together. Such ties and genetic similarity have been one of the primary objects of study in population genetics from its early days, as embodied in the F-statistics of Wright (1969). The power of the current analysis is to be able to show decisively that kin selection is the only quantitatively significant force at work. This confirms by calculation in the present case what is clearly suggested in general by the

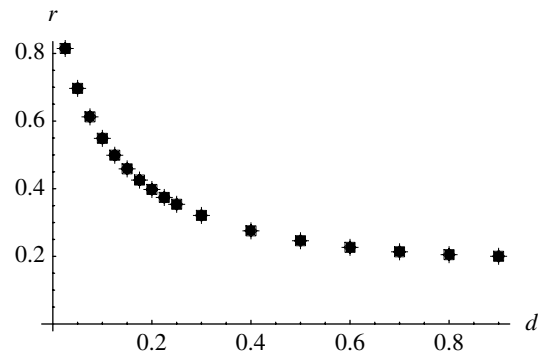

Figure 1. The probability of migration (d) on the x-axis affects the relatedness (r) of an individual to her whole group (including herself). The theoretical value in the monomorphic model given in equation (A 2), r_{anc}, is shown with an open rectangle. The measured values in the polymorphic model are shown for k_{anc} (filled rectangle), $0.99\times k_{\mathrm{anc}}$ (triangle) and $1.01\times k_{\mathrm{anc}}$ (cross). The values are too close together to be graphically distinguished. The data are provided in the electronic supplementary material.

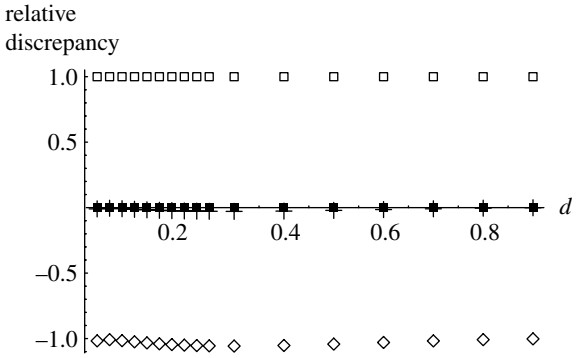

Figure 2. The probability of migration (d) on the x-axis is related to the fractions of emigrants that are type 2 on the y-axis. The immigrants have a fraction of 0.4, so values above (below) 0.4 indicate selection for higher (lower) x. The cases are $0.99\times k_{\mathrm{anc}}$ (diamond), k_{anc} (cross), $1.01\times k_{\mathrm{anc}}$ (open square) and the null case with $x^1 = x^2 = 0$ (filled square). For clarity across the range of d, the deviations from 0.4 are shown as relative to the discrepancy of the $1.01\times k_{\mathrm{anc}}$ case, whose values are therefore all $+1$. The deviations of the k_{anc} case from zero are caused by (i) slight deviations of r_{anc} in the selection model compared with the monomorphic model arising from the difference between x^1 and x^2, (ii) numerical inaccuracies in the calculation of r_{anc}; and deviations in both the k_{anc} and null cases, (iii) the restriction of the calculation to a finite grid when in theory groups are of unlimited size as well as, (iv) general numerical accuracy issues in computer programs. The figure shows that within high numerical accuracy, the critical value of k at which selection is neutral is k_{anc}. The relative and absolute discrepancies are provided in the electronic supplementary material.

analytical work of Rousset (2004) and Wild & Taylor (2004): common ancestry is the only cause of genetic similarity in the absence of assortation or genetic discrimination.

5. DISCUSSION

On the particular case studied here, the previous section shows that altruism in the model of Killingback *et al.* (2006) is precisely explained by the building up of ties of common ancestry between group members. The authors themselves specifically assert that their mechanism is 'clearly quite distinct from kin selection', but the results of the previous section show the contrary. If variable group size has an effect, then it does so through affecting the extent of common ancestry among the members of a group. Whether that effect exists would naturally be investigated using models with different degrees of variability of group size. The paper's title is 'Evolution in group-structured populations can resolve the tragedy of the commons': this turns out to be true to the extent that limited migration increases the strength of common ancestry within groups, a point already made by Hamilton (1964, 1970, 1975).

Lehmann & Keller (2006) have appealed for the results from repeated games to be interpreted in terms of Hamilton's inclusive fitness, and present a kind of meta-model to make that task easier. The work of Grafen (2006) allows any model of social behaviour with discrete non-overlapping generations and additive fitness effects to be interpreted in terms of inclusive fitness. One advantage is conceptual clarity of biological interpretation, and another is the value for a field of having a single central theory to which everything can be referred. Developments following Wild & Taylor (2004) and Rousset (2004) are increasing the range of conditions under which we can be sure that the only cause of genetic similarity is common ancestry. It is therefore good practice to place models of social behaviour in the context of Hamilton's inclusive fitness theory, and recent theoretical advances make that increasingly straightforward in a widening set of circumstances.

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APPENDIX A. DEFINING AND IMPLEMENTING THE MODELS

It is satisfactory to measure common ancestry in a population in which only one strategy is being played, as those values of relatedness will hold when the competing values of x are close enough to each other. Hence, we now define a monomorphic model. The infinite population consists of groups of mean size m. It is more accurate to think of these groups as 'locations' that can support a group, as they continue to exist with a 'group size' of zero and in particular continue to receive immigrants. There are discrete, non-overlapping generations. At the end of each generation, just before dying, each individual produces a Poisson number of offspring with a mean that is proportional to her fitness. Each individual offspring independently disperses with probability d and remains in the natal group with probability $1-d$. Each disperser independently joins a random group. The population numbers are then reduced to make the mean group size m by giving each individual in the population an equal independent chance of dying. This model is studied analytically in Appendices B and C.

Selection is studied in a model with two genotypes $x^1=0$, $x^2=0.05$ and with $m=5$, $V=5$. The frequency of type 2 in the migrant cloud is taken to be $\gamma=0.4$. The value of d is varied according to the values used by Killingback *et al.* (2006). The aim is to find whether x is selectively neutral at $k=k_{\mathrm{anc}}$, so selection was studied at $0.99\times k_{\mathrm{anc}}$, k_{anc} and $1.01\times k_{\mathrm{anc}}$. The values of x were chosen close together so that the ancestral relatedness would be close to the monomorphic case. Selection brought about by large discrepancies in x will certainly exist, but is unlikely to be of biologically interest: in any event, the weak selection result is significant in itself. An additional 'null' case was studied with $x^1=x^2=0$.

An array π^z of size $n+1\times n+1$ is used to represent the probability distribution of groups in iteration z, where $\pi^z_{I\mathcal{J}}$ is the fraction of groups that have exactly I type 1 individuals and exactly $\mathcal{J}$ type 2 individuals. When individuals are produced above n of one type in a group, that number is reduced to n. Each group plays the public goods game within itself, and individuals have Poisson distributed offspring with mean given by $(1-d)$ times their fitness divided by the population mean fitness. The factor of $1-d$ represents selecting only the offspring that do not emigrate, and making it relative to population mean fitness accounts for the population-wide mortality that maintains mean population size. The mean population fitness from the previous iteration is used, as this iteration's value is not known in advance. In all cases studied, the mean population fitness converged. Independent Poisson numbers of type 1 immigrants with mean $(1-\gamma)md$ and Poisson numbers of type 2 immigrants with mean γmd arrive in each group in each generation. These assumptions allow the calculation of π^{z+1} from π^z. The process is started by setting π^0 as the product of two single-variable probability distributions derived from the monomorphic model assuming the mean number of types 1 and 2 individuals is $(1-\gamma)m$ and γm, respectively. The process continued until the squared stepsize (defined as $\sum_{I\mathcal{J}}(\pi^z_{I\mathcal{J}}-\pi^{z-1}_{I\mathcal{J}})^2$) was less than 4×10^{-30} for two consecutive generations. Let the final iteration be at $z=Z$. If the fraction of type 2 among the emigrants in iteration Z is greater than γ then selection is favouring type 2, and if it is less then it is favouring type 1.

n was made as large as practicable and necessary, and in every case the fraction of the population in the outer margin of π^Z was less than 10^{-6}. Checks were made and the mean and variance of total numbers in the groups were always within a proportion 6×10^{-5} and 8×10^{-4} of the monomorphic theoretical value as derived in Appendix A. Relatedness as defined by equation (2.5) was calculated and was always within 1.5×10^{-4} of the monomorphic value. The $d=0.025$ case was always the least well-conditioned, and with $n=196$ took over 48 h to converge on a moderate desktop computer.

All calculations of the models, and indeed much of the mathematical analysis, were performed using MATHEMATICA v. 5.2 (Wolfram Research, Inc. 2005).

APPENDIX B. GROUP AND CLONE SIZES

A discrete random variable X taking values in the non-negative integers with probabilities $q_0,q_1,q_2,\ldots$ has a probability generating function $f(s)$ defined by

$$f(s) \equiv \mathbb{E}(s^X) = \sum_{I=0}^{\infty} s^I q_I.$$

Probability generating functions (pgfs) have many useful properties (see Feller (1968), particularly pp. 267–288). We study the pgf of the numbers of a clone G years after its arrival at a location, denoted as $f_G(s)$. In the initial year, $f_0(s)=s$, as there is exactly one member. If a random number of independently and identically distributed random variables are summed, then the pgf of the sum is $g(f(s))$ where $f(s)$ is the pgf of each summed variable, and $g(s)$ is the pgf of the number of terms in the sum. In this case, the number of individuals in generation $G+1$ of a clone is the sum of a Poisson distribution with mean $1-d$ (pgf is $\mathrm{e}^{-(1-d)(1-s)}$) for each of the individuals in generation G (pgf is $f_G(s)$). Summarizing,

$$f_0(s) = s \qquad f_{G+1}(s) = f_G(\mathrm{e}^{-(1-d)(1-s)}).$$

The total number of individuals at a location with an immigrant ancestor who arrived G generations ago is the sum of a Poisson number (with mean md) of such Poisson branching processes. The composition rule stated earlier shows that the random sum has a pgf of $\mathrm{e}^{-md(1-f_G(s))}$ and the total number of individuals is the sum of those individuals for all values of G. Summing independent random variables corresponds to multiplying pgfs, yielding the pgf for the total number of individuals at a site as

$$h(s) = \mathrm{e}^{-md\sum_{G=0}^{\infty}(1-f_G(s))}. \tag{B 1}$$

It is important that this formula allows us to compute h numerically, as the sum converges provided $d>0$, and can be taken to enough terms for practical purposes.

To find the mean number in the group of a randomly chosen individual, we need to obtain the pgf of 'experienced group size', by weighting the probabilities of each group size by the group size itself, to reflect the proportional chance of its selection, and to normalize so they sum to one. The new pgf is $sh'(s)/h'(1)$. The same exercise for clone size requires adding over all the generations for which a clone might have persisted, to obtain the pgf of 'experienced clone size' as $s\sum_{G=0}^{\infty} f'_G(s)/\sum_{G=0}^{\infty} f'_G(1)$.

The mean and variance of a variable can be found from its pgf as $f'(1)$ and $f''(1)+f'(1)-f'(1)^2$, and these provide the values in the main text.

APPENDIX C. RELATEDNESS THROUGH ANALYSIS

In this appendix, we calculate the quantity in equation (3.1) using pgfs. An individual in a group is the descendant of an immigrant G generations ago. Agree that we can select a random individual in the population. Then we define two random variables (C,R) as the total number of individuals descended from the same initial migrant by continuous ancestry within the group, and the total number of individuals in the rest of the group. C includes the individual herself, and $C+R$ is the total number in the group.

Let the joint pgf of (C, R) be $u(s,t) \equiv \mathbb{E}(s^C t^R)$. We first derive a formula for u, and then use it to find relatednesses. The distribution of C is the distribution of experienced clone sizes, whose pgf was obtained in Appendix B. In a pragmatically crucial move, we note that $h'(s) = mdh(s)\sum_{G=0}^{\infty} f'_G(s)$, leading to the second of these forms for C's pgf

$$\frac{s\sum_{G=0}^{\infty} f'_G(s)}{\sum_{G=0}^{\infty} f'_G(1)} = \frac{sh'(s)}{h(s)h'(1)}.$$

To obtain the pgf of R, we will show that R and C are independent, and that R has the same pgf as the total number in a group, namely h. For any given G, the number of clones starting G generations ago has a Poisson distribution, and so the fact that the selected individual shows a clone was started then does not change the distribution of the remaining number of clones starting at the same time. The number starting at any other time is unaffected by knowledge that a clone was started G generations ago. Thus, R has the same distribution as the total number of individuals in a group, conditional on each possible G, and therefore unconditionally too. The joint pgf of two independent variables is simply the product of the individual pgfs, so putting them together gives

$$u(s,t) = \frac{sh'(s)h(t)}{h(s)h'(1)}$$

Now the pgf $v(s,t)$ of $(C, R+C)$ is $\mathbb{E}(s^C t^{R+C}) = \mathbb{E}((st)^C t^R)$ and so equal to $u(st,t)$. To study the distribution of the relatedness, which is $C/(R+C)$, note that

$$\int_{\tau=0}^{t}\left(\frac{\frac{s}{\tau}\partial\sum_{I,\mathcal{J}} s^I\tau^{\mathcal{J}}q_{I\mathcal{J}}}{\partial s}\right)\mathrm{d}\tau = \sum_{I,\mathcal{J}} s^I t^{\mathcal{J}}\frac{I}{\mathcal{J}}q_{I\mathcal{J}},$$

and that $I/\mathcal{J}$ is the relatedness for an individual in a clone of size I in a group of size $\mathcal{J}$. (Indeed $I/\mathcal{J}$ here equals c_i/n_i in the notation of equation (3.1)). Evaluating the expression at $s=t=1$ gives the mean relatedness over the population. Formally, we use this to express the mean relatedness in terms of v, and substitute back with u and h in turn to obtain

$$r_{\mathrm{anc}} = \int_{t=0}^{1}\frac{s}{t}\frac{\partial v(s,t)}{\partial s}\mathrm{d}t\bigg|_{s=1} = \int_{t=0}^{1}\frac{-th'(t)^2+h(t)(h'(t)+th''(t))}{h(t)h'(1)}\mathrm{d}t \tag{C 1}$$

The final integral is readily evaluated numerically using equation (B 1), and provides an analytic value for the relatedness of equation (3.1) for the given parameter values m and d.

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