Group Theory in Homogeneous Populations (Rescuing Darwin from the mud)

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Abstract. Considerable recent work on the evolution of behaviour has been set in structured populations. An interesting "cancellation" result is known for structures, such as lattices, cycles and island models, which are homogeneous in the sense that the population "looks the same" from every site. In such populations all proximate or immediate fitness effects on others (for example, payoffs in a game or contest) play no role in the evolution of the behaviour. The altered competitive effects of such behaviour exactly cancel the proximate fitness effects. In mathematics, the internal symmetry which drives this result is powerfully described by the theory of mathematical groups and recent work has used this theory to clarify and extend a number of existing results. I review this body of work here.

Mathematics Subject Classification (2000). 05C57, 91A43, 92D50.

Keywords. Population structure, graph, group theory, homogeneous, selection, relatedness, allele frequency.

1. Introduction

It is a pleasure and a privilege to have been asked to talk about the Mathematics of Charles Darwin on this very day, the 150$^{\text{th}}$ birthday of the publication of the "Origin," particularly among such distinguished colleagues. The invitation prompted me to think about my early encounters with Darwin and I realized that one of these occurred 50 years ago, no doubt very close to the 100$^{\text{th}}$ birthday. I was in late high school and was interested in science and had discovered *The Origin of Species* [1] in my father's collection of the Harvard Classics. I read bits of it, but not very much. My father, on noticing the book in my possession, told me I must talk to a biologist he knew at the museum, where he was working, and in spite of my mild resistance, he set up a meeting a few days hence. When the time came he

This work was partially supported by a grant from the Natural Sciences and Engineering Research Council of Canada. I am grateful to the organizers of conference on The Mathematics of Darwin's Legacy for hosting me at this marvelous meeting.

F.A.C.C. Chalub and J.F. Rodrigues (eds.), *The Mathematics of Darwin's Legacy*, Mathematics and Biosciences in Interaction, DOI 10.1007/978-3-0348-0122-5_6,

drove me into town, took me firmly up the curling flight of stone steps and knocked at his colleague's door. And there he left me saying he would meet me in the lobby.

Well there we were, both of us not quite sure what I was doing there. He asked me uneasily what I wanted and I didn't dare reply with the truth – that there was nothing I wanted but my father had made me come. I told him I had been reading *The Origin of Species* and had found it interesting. "That's perhaps not the best place to start," he replied gently, and for the rest of the mercifully short interview he gave me a list of books I might read "next" and told me a bit about them in words that meant little to me. And here I am 50 years later. It would be interesting now for me to have a copy of that list, but I am certain I never consulted it again.

A couple of years later it was physics I chose at university and then, after a year or two, I settled on mathematics, I believe, for its structure, its beauty, its simplicity, and its independence from the world. Another 15 years would pass before I encountered biology again, and that was in a remarkable series of lectures delivered by John Maynard Smith at a special symposium organized by the Canadian Mathematics Society. John talked about something called "evolutionary game theory" and I suddenly realized that the structure and beauty I had sought in turning, many years ago, to mathematics, was what Charles Darwin had given us in biology, that the theory of evolution opened up to us a whole new way of asking questions about why organisms behave this way rather than that, and thereby gave us powerful new tools for understanding this behaviour. It was a striking revelation.

The impact on me was even greater as I had recently turned to a study of the applications of game theory to economics and had been troubled by its unsettling assumptions of rationality and purpose. But now I could see where game theory might really belong, in biology where the actors were not us crazy humans, but genes, where rationality was not needed, and where purpose was not a precondition but appeared miraculously as a consequence of the unfolding of the evolutionary game.

The 40 years since the work of Maynard Smith and Price have given us an enormous spectrum of rich interactions between mathematics and biology. A wonderful account of many of these is found in Joel Cohen's remarkable 2004 essay called *Mathematics Is Biology's Next Microscope, Only Better; Biology Is Mathematics' Next Physics, Only Better* [2]. Cohen discusses an impressive range of areas in which mathematics and biology interact and in particular presents five biological challenges that could stimulate and benefit from major innovations in mathematics, and then five mathematical challenges that would contribute to the progress of biology. When I heard about this symposium, I went back to this essay looking for ideas and one sentence I read was this: 'Charles Darwin was right when he wrote that people with an understanding "of the great leading principles of mathematics ... seem to have an extra sense" ' [2, page 2017].

Reading that, I started to wonder what else Darwin might have thought of mathematics, so I looked on the web and I found this: "A mathematician is a blind

man in a dark room looking for a black cat which isn't there". I had heard this famous remark before but had never known that it was credited to Darwin. And another: "I suppose you are two fathoms deep in mathematics, and if you are, then God help you, for so am I, only with this difference, I stick fast in the mud at the bottom and there I shall remain". This second quote quite challenged me and prompted me to choose for this talk a slice of mathematical biology that I found simple and beautiful, and that might just have had the power to rescue Darwin from the mud, if only he could have seen it.

2. Homogeneous population structures

For the past twenty years I have been interested in the effects of population structure on the direction of evolution, more specifically on allele frequency change. To illustrate the impact of structure, imagine an interaction between two individuals i and j which changes the fitness of each of them. Does the size of these fitness changes tell us all we need to know to work out the evolutionary effects? In general, no. The point is that each of the two fitness effects will in general have an impact on the fitness of other individuals k. For example if the offspring of i have some tendency to compete with the offspring of an individual k, then a change in i's fitness might affect the fitness of k. Thus, a full accounting of evolutionary change requires knowledge of the fitness effects on all individuals in the population.

To have some terminology, let's call the first set of effects proximate, and classify the second set under the general label of ecological feedback. The proximate effects are typically those that are observed in the field or specified in a behavioural model. For example in a two-party altruistic interaction, these are the cost incurred by the actor and the benefit gained by the recipient. The feedback effects are those that derive from the resulting altered competitive pressures and depend on the population structure and the nature of population regulation. For example if the proximate effects are on fecundity, the feedback effects might be a change in mortality among the offspring; if the proximate effects are on adult survival, the feedback effects might be altered offspring recruitment.

To have a specific scenario to work with, I will suppose here that the proximate effects are on breeder fecundity and the feedback effects are on offspring survival deriving from altered competitive pressures.

The proximate effects are typically known (measured or assumed) but the feedback effects are much harder to get hold of and typically require detailed knowledge of offspring dispersal and recruitment patterns. That's the story told by the population structure.

One of my interests has been to identify simple classes of population structures for which the feedback effects can be very simply described. Here I will look at what would appear to be the very simplest such structures, those for which the population "looks the same" to every individual. The term "homogeneous" has been used in the literature with many different meanings, but I will use it here

to capture this notion and I will define it more precisely below. This notion of homogeneity might seem rather special or restricted, but some approximate version of it is often assumed. Theoretical models which are "open" in the sense that no population structure is actually given, typically work with a randomly chosen "focal" individual, and for this to make sense, the environment of this individual must be, in some sense, generic. This is effectively an assumption of homogeneity.

To keep things simple, I suppose that the population consists of a fixed collection of breeding sites, each occupied by a single asexual haploid individual. Instead of using the index i to keep track of individuals (who are being born and dying), I will use it to index the sites. Indeed I will assume that the set of sites is permanent and there is no change in population size.

One can depict these breeding sites as a set of nodes with arcs or edges between nodes to represent the relationship among them. For us, these relationships will be of two types, the rate at which offspring disperse from one node to another and the effect of the behaviour of the breeder at one node on the fitness of the breeder at another. I define the *dispersal probability* $d(i,j)$ to be the probability that an offspring born at site i competes to breed at site j and, counting only those offspring who attain a breeding site, I assume that $\sum_j d(i,j) = 1$. I let F_i denote the fecundity of the breeder at site i. In general, F_i will depend on the behaviour z_j (defined as the level of a behavioural trait) at many different sites j and I define the *fecundity effect* of j on i to be the partial derivative $\partial F_i/\partial z_j$.

The notion of homogeneity can be specified in terms of these relationships. To say that the population looks the same from node i as from node j is to say that an individual who could perceive only the dispersal probabilities and the fitness effects could not tell whether it was situated on node i or on node j. In [2] the notion of isomorphism is used to describe this. An isomorphism T (iso $\sim$ same; morph $\sim$ structure) is a bijection of the node set which preserves the dispersal probabilities and the fecundity effects, i.e., for any i and j,

$$\begin{cases} d\left(T(i),T(j)\right) = d(i,j) \ , \\ \dfrac{\partial F_{T(i)}}{\partial z_{T(j)}} = \dfrac{\partial F_i}{\partial z_j} \ . \end{cases} \tag{2.1}$$

The population structure is called *transitive* if, for every pair of nodes i and j, there is an isomorphism T for which $T(i) = j$. The transitive structures are precisely those that I am calling homogeneous.

3. The main result

There is a surprising, even extraordinary result which obtains in this homogeneous case. Suppose an actor at site j carries an allele A which causes her to give a fecundity benefit b to the breeder at site i different from j (that's the proximate effect). We want to measure the selective effect of this behaviour and by this we mean the effect on the population frequency of the allele A. Now as we have

discussed, the altered fecundity of i will change the competitive environment in a neighbourhood of i and as a result the fitness of a number of other individuals will also be affected. Some of these others may carry the allele A, so to calculate the change in frequency of A, we will need to work out the effects of the action on all those whose fitness is affected as well as the probability that they will carry A. This can be a big job but it can, after some work, be obtained from knowledge of the population structure.

Now here's the remarkable result – if the structure is homogeneous, this substantial calculation doesn't have to be done [3, 4, 5, 6, 7, 8]. In this case, all these effects on the frequency of A, the proximate effect, b, on i and the resulting competitive effects on any number of others, will all cancel out *so that the net effect on the frequency of A will be zero.* Some who carry A will have increased fitness and others who carry A will have decreased fitness, and the net result is that on average, the decreases will exactly balance the increases. If you like, the ecological feedback effects of the fecundity change of i will exactly neutralize the proximate effect.

Assumptions. Right away I have to declare that there are a number of significant assumptions that are needed for this result to hold. A first class of assumptions is standard for inclusive fitness methods to be valid [9, 10] – fitness effects have to be additive and small. Thus, if the behaviour of several of my neighbours affects my fitness, the net effect must be the sum of the individual effects. Also the analysis is done to first order in the fitness deviations and this will give accurate results only if these deviations are small compared to baseline fitness. A second class of assumptions are more technical and have to do with the way in which generations succeed one another (overlapping or not), whether proximate effects are on fecundity, as we have assumed here, or survival, and whether offspring dispersal is symmetric, i.e., $d(i, j) = d(j, i)$ [8]. Finally, the population needs to be large or a small correction is required. In a finite population, the average effect on the non-focal individuals must be subtracted (equation (7.2)).

I emphasize that this "cancellation result" holds for the effects of the interaction on all other breeders, but not for the actor herself. As a result of this, the direction of change in frequency of the allele A is determined by the sign of the proximate effect of the actor on her own fecundity. If the act is costly to the actor (say it incurs a survival cost c) the allele frequency decreases, and if beneficial, the allele frequency increases. This simple result is indeed surprising, almost unbelievable, and I now discuss its interesting history.

4. History of the result

In 1992 I had a call from David Wilson. He had constructed a simulation to test Hamilton's Rule [9] on a two-dimensional lattice (Fig. 5.2c) and what he was finding quite surprised (and intrigued) him. No matter how large he made the benefit b bestowed by an altruistic allele, he could not get altruism to be

selected. In his simulations, it always eventually died out and that appeared to flatly contract Hamilton's Rule [3].

Let's go back to Hamilton. Consider an allele A which causes the bearer (the actor) to bestow a fitness benefit b on a "neighbour" at a personal fitness cost c. Then if we want to calculate the resultant direction of frequency change of A, we will need to know the probability that the neighbour is an A-individual and (in this simple haploid case) that is a measure of what is called the relatedness of the actor to the neighbour [10, 11]. This idea, that some notion of relatedness ought to have something to do with allele-frequency change was recognized long ago, most famously by Haldane [12] who while walking beside a flooded river, considered the question of whether to jump in to save a drowning child and decided that the closer was his relatedness to the child, the more likely he ought to jump in. Haldane, of course, was able to think in genetic terms, but even Darwin [1], before he knew of Mendel's work, knew that what counted was the "blood" [13] rather than the individual. But it was Hamilton's genius that put forward the simple quantification of this idea. Hamilton's Rule states that the allele will be selectively favoured when $bR > c$ where R is the relatedness of the actor to the recipient.

Now back to Wilson's population. It had the structure of a lattice and each generation offspring dispersed to neighbouring sites, so there was certainly a significant probability that a neighbour would be an offspring or grandchild or niece etc. and we ought to have a positive R. Thus if b is big enough, we should have $bR > c$ and Hamilton's Rule [9] ought to give a selective advantage to the altruistic trait. Hence Wilson's (and my!) dismay.

The discussion above clarifies the dilemma. Wilson's b and c were in fact changes in *fecundity* – they represented a small increase (to the recipient) and decrease (to the actor) in offspring number. As we have seen, that's only the first part of the story. To translate these changes in fecundity into changes in fitness, we need to know the effect of these extra offspring on the fitness of others who might live nearby and who might also share genes with the actor. And when this is done all the effects of the b-gift cancel, leaving only the effects of the actor's fecundity decrement c. This certainly reduces the fitness of the actor and any positive effects on others (from reduced competition) cannot be strong enough to turn that negative effect around. Wilson's lattice population is an example of a homogeneous population structure and the main result applies.

What becomes of Hamilton's Rule $bR > c$? It has a number of versions which are valid. It holds when b and c represent the total fitness effect on recipient and actor and the fitness of no other individual is affected. There is one special case in which it holds with b and c representing only the proximate effects on fecundity, and that is when the resulting competitive effects (of the fecundity changes) are randomly distributed in the population as a whole. This might typically be the case in a randomly mixed population.

But in the usual situation the fitness effects of a proximate fitness transaction will be felt by a number of individuals, in this case a generalization of Hamilton's

Rule would have the form $\sum_i w_i R_i > 0$ where w_i is the overall fitness effect on individual i and R_i is the relatedness of i to the actor.

5. Mathematical groups

Different treatments of the main result, at various levels of generality, have appeared [3, 4, 5, 6, 7, 8, 14, 15] but the most recent of these [8] presents the result in a particularly elegant mathematical framework, and to celebrate Darwin's anniversary, I discuss that here.

It turns out that a powerful description of structural homogeneity can be obtained using the language and notation of the theory of mathematical groups. That's hardly surprising – group theory arose as a need for a formal structure to study the geometry of objects, such as regular polyhedra, that have a significant amount of internal symmetry.

First of all, instead of working with the individuals in the population (who are ephemeral – they die and are replaced by others) we work with the collection of breeding sites, each occupied by a single adult breeder. To have a picture we represent them as the nodes of a graph and use the arcs between nodes to represent the relationship between them, capturing both the dispersal probabilities and the fitness interactions (Fig. 5.1).

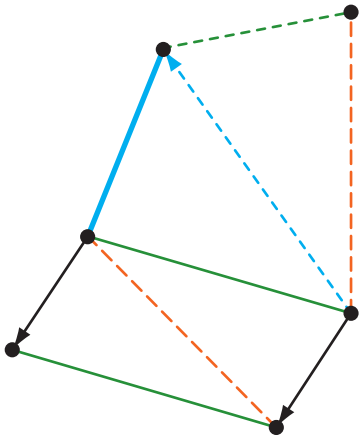

FIGURE 5.1. A directed graph. The different arcs represent different dispersal probabilities and/or different fitness interactions between the nodes.

So far we have described what is simply called a directed graph. What we don't yet have is the condition that the population "look the same" from every node. An elegant way to obtain that is to suppose that the set of nodes can be given the structure of a group.

We begin with a formal definition. A group G is a set of elements i with a closed binary operation (which we represent multiplicatively) which satisfies the following three axioms:

1. There is an identity element e with the property $ei = ie = i$ for all i.
2. Every element i has an inverse (denoted i^{-1}) such that $ii^{-1} = i^{-1}i = e$.
3. The operation is associative: $i(jk) = (ij)k$ for all i, j and k.

Now the wonderful thing about groups is that we have a natural transitive set of bijections which can serve as our isomorphisms, and these are the group multiplications. Indeed, given two elements j and k of the group, multiplication on the left by $i = kj^{-1}$ is a bijection T of the group which maps j into k. Indeed, $T(j) = ij = (kj^{-1})j = k(j^{-1}j) = ke = k$. Thus if our breeding sites are the elements of a group, we can use these left multiplications as a natural transitive set of maps preserving our two critical relationships – offspring dispersal and fecundity effects. That is, for any i, j and k, we specify:

$$\begin{cases} d(j,k) = d(ij,ik)\ , \\ \dfrac{\partial F_j}{\partial z_k} = \dfrac{\partial F_{ij}}{\partial z_{ik}}\ . \end{cases} \tag{5.1}$$

It turns out that the homogeneous population structures that have appeared in the theoretical literature can all be given a group structure in a natural way so that the group multiplication provides the isomorphisms satisfying equation (2.1).

For example how do we put a group structure on the examples depicted in Fig. 5.2? The answer is that in every case this comes from the geometry of our representation. In each case we choose an arbitrary node to be the identity element e. Then for (a) and (b) we can simply use the rotations about the centre identifying each node with the angle required to rotate the identity to that node. Group multiplication, of course, is composition. For (c) we use the horizontal and vertical translations if the population is infinite, but in the case of a finite lattice, we fold the array into a torus by identifying boundaries (right with left and top with bottom) and use a 2-parameter family of rotations. In (d) we again have a 2-parameter family of rotations, first the four rotations through 90° that rotate each island within itself, and secondly the three rotations through 120° which cycle the islands. All possible products of these give us the twelve elements of the group.

6. Can every homogeneous population be given a group structure?

That's all fine for these standard examples which we can easily draw, but the question arises as to whether any homogeneous population structure can be represented as a group. Note first that the converse of this holds and was mentioned above – any group-structured population for which the invariance equations (5.1) hold is homogeneous and the left multiplications give us a transitive set of isomorphisms (equation (2.1)). But suppose that we have a population structure with a

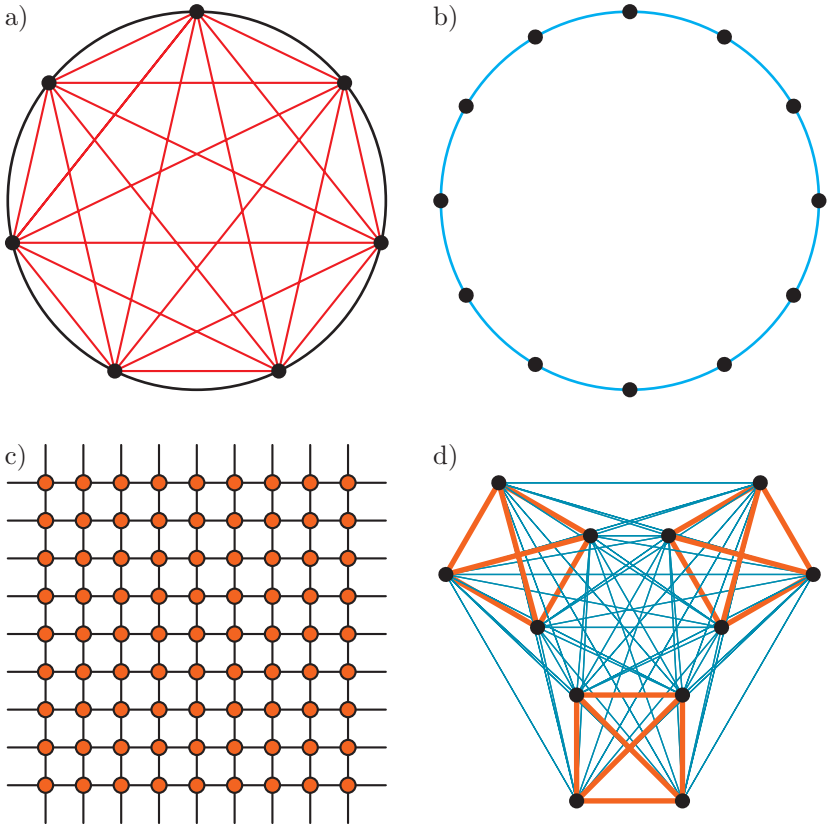

FIGURE 5.2. Some examples of standard homogeneous populations structures. (a) A random-mixing population with 7 breeding sites. (b) A cycle. Interactions with neighbours. Dispersal to neighbours or at random. (c) A lattice. Interactions with neighbours. Dispersal to neighbours or at random. (d) An island structure with 3 demes of size 4. Interactions at random within deme. Dispersal at random within deme or at random in population.

transitive set of isomorphisms (equation (2.1)). Can we put a group structure on the set of nodes so that the left multiplications are isomorphisms (equation (5.1))?

Well here is an idea. Take a random node and label it e. Now take any other node i. The homogeneity property tells us that the population should "look the same" from i as it does from e. Thus, for any node j, there should be a node which "looks the same" from i as j looks from e. *We could call that node* ij and this in fact would define the group multiplication operation on the node set. That seems at first to work nicely, at least the three group axioms (above) seem to hold.

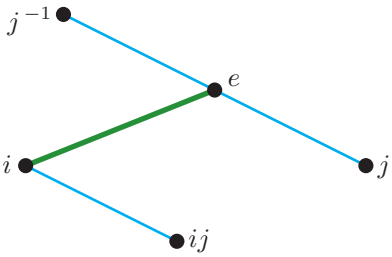

FIGURE 6.1. ij is the node that looks the same from i as j looks from e.

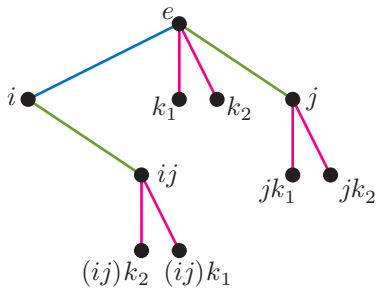

FIGURE 6.2. Possible difficulties with associativity. The nodes k_1 and k_2 bear the same relationship to e and there is no consistent way to distinguish them. Of course any diagram gives us a temporary right-left distinction. In the realization here we have used one ordering attached to j and another attached to ij. In this case, the verification that $(ij)k = i(jk)$ for either of the k's, will fail.

Except they don't. If you argue carefully, you run across a problem with the associative axiom 3.

The problem arises when there are several candidates bearing the same relation to a fixed node. This would not be the case in the structures of Figures 5.2 (b) and (c), but it could be an issue in (a) and (d) (Figure 6.2).

Typically, of course, we can use other aspects of the population structure (such as geometry) to pick out a consistent set of nodes playing the role of k, but the question we started with is whether this can always be done. Are there homogeneous populations which cannot be given a group structure?

It turns out that this is unknown, indeed it is closely related to an open problem in the theory of mathematical groups [8]. Certainly any homogeneous population I have ever seen (or imagined) can be given a group structure, but that is the best I can do. For sure this question is of much more mathematical than biological interest, but it is intriguing none the less.

7. Finite and infinite populations

Our main result takes a slightly different mathematical form in a finite and an infinite population and I look at that now. Recall that the result says that the effects of allele action on the fecundity of others do not "at the end of the day" cause a change in the average frequency of the allele. We conclude that the selective change in the average frequency will only be determined by, and must have the same sign as, the effect of the behaviour on the actor's own fecundity – if the actor suffers a cost, the overall frequency of the allele must decrease; if she gives herself a net benefit, the overall frequency of the allele will increase.

Actually there is one clarification or adjustment needed. The factor to pay attention to is not the effect of the behaviour on the actor's own fecundity, but rather the effect relative to the average population-wide effect. In an infinite population (or even a very large one), this correction is negligible, as a single actor could have only a negligible average effect. But in a small finite population, this normalization factor has to be included. Formally, the conditions for the sign of the selective change in the average allele frequency are written [8]:

$$\text{Infinite population:} \qquad \Delta\bar{x} \equiv \frac{\partial F_e}{\partial z_e} \ , \tag{7.1}$$

$$\text{Finite population:} \qquad \Delta\bar{x} \equiv \frac{\partial F_e}{\partial z_e} - \mathbb{E}_{i\neq e}\left(\frac{\partial F_i}{\partial z_e}\right) \ , \tag{7.2}$$

where "$\equiv$" means "has the same sign as". In a finite population we normalize by subtracting the average effect of the behaviour on the fecundity of all *other* breeders in the population.

8. Examples

To illustrate the result, I present three brief examples of altruistic behaviour.

Example. Suppose we have a finite population with a deviant trait which provides a cost-free public good which increases the fecundity of everyone by the same amount. Such a trait should have no effect on the frequency of the allele causing it. Since focal behaviour has the same effect on everyone, $\partial F_i/\partial z_e = \partial F_e/\partial z_e$ for all i, and equation (7.2) gives us $\Delta\bar{x} = 0$ as expected.

Example. Suppose we have an infinite island population with demes of size n, and a cost-free deviant trait which provides a fecundity benefit to all deme-mates, but not to self. Then there is no fecundity effect on the actor and equation (7.1) gives us $\Delta\bar{x} = 0$. Do we in fact expect no change in allele frequency? In this population, an individual's fitness depends on the number of its deme-mates which are deviant. If we suppose the demes are randomly formed, then the average number of deviant deme-mates should be the same for a deviant and a normal individual and there should indeed be no change in allele frequency, confirming the result. Now suppose that the demes are not randomly formed, but some offspring stay at home. Then

we expect that a deviant individual will have more deviant deme-mates on average than a normal individual and will have higher average fitness. But this will also be the case for the deviant individual's deme-mates so that the competitive pressures at home will be higher than average. Equation (7.1) implies that these two opposing factors must exactly cancel.

Example. Altruism. Consider an altruistic trait in which individuals give fecundity benefits to various other individuals at total cost c. Then focal fecundity might have the form $F_e = -cz_e + \sum_i b_i z_i$ where b_i is the focal benefit received from site i when there is an altruist at that site. To move from there to a calculation of $\Delta\bar{x}$ we need the offspring dispersal patterns, both to calculate the competitive effects (which are needed for focal fitness w_e) and to get the focal relatedness coefficients. However, in a homogeneous population, equations (7.1) and (7.2) tell us that none of that is needed:

$$\text{Infinite population:} \qquad \Delta\bar{x} \equiv \frac{\partial F_e}{\partial z_e} = -c \ , \tag{8.1}$$

$$\text{Finite population:} \qquad \Delta\bar{x} \equiv \frac{\partial F_e}{\partial z_e} - \mathbb{E}_{i\neq e}\left(\frac{\partial F_e}{\partial z_i}\right) = -c - \bar{b} \ , \tag{8.2}$$

where $\bar{b}$ is the average value of the b_i over all non-focal individuals. The finite population equation was obtained in [6] under the assumption that the actor gives b to a single other individual so that $\bar{b} = b/(N-1)$ where N is population size. These equations make it clear that altruism can never be selected. In an infinite population, spite [16, 17] can also never be selected, but it can be selected in a finite population if the average harm done by a focal actor to other individuals in the population exceeds the focal cost.

What does the group-formalism do for us that the old notion of transitivity did not? First of all it does allow us to strengthen a number of the results previously obtained working with the notion of transitivity. But secondly, and of more mathematical significance, it provides simpler more elegant proofs of a number of previous results. In many ways it provides the right "natural" setting for the type of homogeneity we are looking for.

9. Limitations

I end with a warning that these results apply only to a restricted class of behaviours. They do not generally apply in a class- or age-structured population, nor to ploidies other than 1, though the results do extend to a sexual diploid population if males and females are treated the same. In particular, they do not apply to sex ratio traits, or to sex-specific behaviour. Secondly, while the trait is supposed to affect the fecundities F_i, it cannot affect the offspring dispersal probabilities $d(i,j)$. In particular, it does not apply in models of optimal dispersal. It seems to apply most readily in models of cooperation and competition.

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