Selection in group-structured populations

Well-mixed populations allow us to ignore mutant–mutant interactions

So far we have been looking at selection in well-mixed populations. This meant we could ignore interactions among rare mutants because they are considered marginal compared to interactions with residents. This simplification made the mathematics more tractable, and it allowed a relatively direct move from a lineage-based to an individual-based formulation of invasion fitness.

Spatial structure is common

In nature, dispersal is often limited. Constraints on movement, social interactions, or geographical barriers prevent populations from being well-mixed. Examples include microbial colonies expanding on a surface, plants dispersing seeds in the vicinity of the parent, or animals living in fragmented habitats. Such limitations generate **spatial structure**, in which individuals mostly interact with neighbours rather than with the entire population.

This leads to mutant–mutant interactions and kin selection

When dispersal is limited, offspring tend to remain close to their ancestors. As a result, a mutant that is globally rare can become locally common. This fundamentally changes invasion dynamics: the fate of the mutant depends not only on its direct effect on the bearer's fitness, but also on its effect on the fitness of other mutants nearby. This is what gives rise to **kin selection**: whenever a trait expressed by a focal individual affects the fitness of others who are genetically related to it at the locus determining the trait [1, 2, 3, 4].

Aim

In this last section, we will study directional and disruptive selection under limited dispersal through the lens of kin selection.

Kin selection represents a major theoretical advance in evolutionary ecology. It provides the key to understanding the evolution of competition, cooperation, conflict, and a wide range of social behaviours.

Yet the concept is often misunderstood and sometimes criticised (e.g. "kin selection applies only within families"; "kin selection is valid only when traits have weak effects on fitness").

We will therefore spend time developing the theory carefully, to dispel such misconceptions and avoid future confusion.

The island model: uniform dispersal among demes

We consider the case where the population is subdivided into a large (effectively infinite) number of groups, each of which can be arbitrarily small.

The key assumption is that these groups are equally connected to one another: there is no isolation-by-distance. If an individual disperses and leaves its natal group, it is equally likely to immigrate into any other group.

This is the classical **island model of dispersal** [5]. We focus on the case where there are no exogenous differences among groups (e.g. no variation in environmental conditions).

A general life cycle

The simplest setting is where all groups are of the same fixed size n, and where individuals within groups are otherwise homogeneous. We'll consider a haploid population with the following life cycle events:

1. **Group extinction:** with some probability, entire groups go extinct and all their members die.
2. **Reproduction and survival:** each adult in surviving groups produces a large number of offspring and then either survives or dies.
3. **Dispersal and competition:** offspring may disperse with nonzero probability, and density-dependent competition fills any vacated breeding spots.

This accommodates a wide range of scenarios: overlapping generations, metapopulation dynamics, propagule dispersal, and soft/hard selection.

Mutant groups and their dynamics

To describe the dynamics of the mutant then, we track not individuals directly but the number of groups that contain mutants, i.e. "mutant groups". There are n possible types of mutant groups: those with one mutant, two mutants, ... up to n mutants. The state of the mutant population is then given by the $n \times 1$ vector

$$\mathbf{g}_t = (g_{1,t}, g_{2,t}, \ldots, g_{n,t}), \tag{1}$$

where $g_{k,t}$ is the number of groups with k mutants at time t.

The dynamics of this vector follow

$$\mathbf{g}_{t+1} = \mathbf{A}(y, x) \cdot \mathbf{g}_t, \tag{2}$$

with $\mathbf{A}(y, x)$ an $n \times n$ mean matrix, where $a_{ij}(y, x)$ is the expected number of groups with i mutants at $t + 1$ produced by a group with j mutants at t (including the possibility that the group itself changes mutant number).

Group-level fitness

This $a_{ij}(y, x)$ can be seen as a group-level description of fitness, valid because:

- the mutant is rare,
- the number of groups is large,
- dispersal among groups is random.

These assumptions imply that: (i) a mutant group (at least one mutant) cannot receive additional mutant immigrants; and (ii)a resident group (no mutants) cannot receive immigrants from multiple mutant groups.

Hence parentage between groups is well defined: each new mutant group can be traced back to a unique parent group that sent a successful immigrant.

Invasion fitness in the island model

Whether or not the mutant can invade is determined by the leading eigenvalue of the mean matrix,

$$\rho(y,x)\,\mathbf{u}(y,x) = \mathbf{A}(y,x)\cdot\mathbf{u}(y,x), \tag{3}$$

which gives the **invasion fitness** $\rho(y,x)$ (since the mutant goes extinct with probability one if and only if the number of mutant groups with probability one).

Here $\mathbf{u}(y,x)$ is the right eigenvector of $\mathbf{A}(y,x)$, scaled such that its entries sum to one:

$$\sum_{i=1}^{n} u_i(y,x) = 1.$$

With this normalisation, $u_i(y,x)$ is the asymptotic frequency of groups with i mutants among all mutant groups.

The goal task is then to move from this **group-level description of fitness** to an **individual-level representation** of invasion fitness.

From a group to an individual perspective of fitness

To do so, we first left multiply both sides of eq. (3) by the vector $(1, 2, \ldots, n)$ and rearrange to get

$$\rho(y, x) = \frac{(1, \ldots, n) \cdot \mathbf{A}(y, x) \cdot \mathbf{u}(y, x)}{(1, \ldots, n) \cdot \mathbf{u}(y, x)} = \frac{\sum_{i=1}^{n} \sum_{j=1}^{n} i a_{ij}(y, x) u_j(y, x)}{\sum_{i=1}^{n} i u_i(y, x)}, \tag{4}$$

where the denominator, $\sum_{i=1}^{n} i u_i(y, x)$, is the expected number of mutants in a mutant group. Second, observe that the total expected number of mutant individuals produced by a mutant group with j mutants can be written as

$$\sum_{i=1}^{n} i a_{ij}(y, x) = j w_j(y, x), \tag{5}$$

where $w_j(y, x)$ is the expected number of mutants produced by a mutant individual in a group with $1 \leq j \leq n$ mutants, i.e. an individual level measure of fitness.

An individual perspective of fitness

By substituting eq. (5) into eq. (4), invasion fitness can be written directly in terms of individual fitness:

$$\rho(y,x) = \sum_{k=1}^{n} w_k(y,x)\, q_k(y,x). \tag{6}$$

Here $w_k(y,x)$ is the expected number of mutant offspring produced by a mutant individual in a group with k mutants, and

$$q_k(y,x) = \frac{k\, u_k(y,x)}{\sum_{i=1}^{n} i\, u_i(y,x)} \tag{7}$$

is the asymptotic probability that a mutant individual belongs to a group with $k \geq 1$ mutants.

Thus invasion fitness is simply **the expected reproductive success of a randomly sampled mutant from its lineage**.

Individual fitness in group-structured populations

To move further, we define the function

$$w(z_\bullet, \mathbf{z}_n), \quad \text{with } \mathbf{z}_n = (z_1, z_2, \ldots, z_{n-1}), \tag{8}$$

as the expected number of descendants of a focal individual with trait $z_\bullet \in \{y, x\}$ when its $n-1$ group neighbours have traits $z_1, \ldots, z_{n-1}$ (each $z_i \in \{y, x\}$). Because groups are homogeneous and interactions within groups are random, $w(z_\bullet, \mathbf{z}_n)$ is insensitive to the order of the neighbour traits in $\mathbf{z}_n$. It depends only on how many of them are mutants versus residents. In particular, the fitness of a mutant in a group with k mutants (the term $w_k(y, x)$ in eq. (6)) is

$$w_k(y, x) = w\left(y, (\underbrace{y, \ldots, y}_{k-1}, \underbrace{x, \ldots, x}_{n-k})\right). \tag{9}$$

That is, the focal mutant has $k-1$ mutant neighbours and $n-k$ resident neighbours.

Directional selection with mutant–mutant interactions

Plugging eq. (9) into invasion fitness eq. (6) and differentiating, we obtain the selection gradient

$$S(x) = \left.\frac{\partial w(z_\bullet, \mathbf{z}_n)}{\partial z_\bullet}\right|_{z_i=x} + (n-1)\, r_2^\circ \left.\frac{\partial w(z_\bullet, \mathbf{z}_n)}{\partial z_1}\right|_{z_i=x}, \tag{10}$$

where

$$r_2^\circ = \sum_{k=1}^{n} \frac{k-1}{n-1}\, q_k(x,x) \tag{11}$$

is the probability that a randomly sampled neighbour of a mutant is also mutant, under neutrality. This is the **pairwise relatedness**: it measures how likely a neighbour is to transmit the same allele as a focal individual at a neutral locus.

Hamilton's (marginal) rule : the inclusive fitness effect

Eq. (17) can be recognised as the well-known inclusive fitness effect, or Hamilton's rule in gradient form:

- the **direct effect**: how a trait change in a focal affects its own fitness (the "cost" $-C$ in Hamilton's rule);
- the **indirect effect**: how a trait change in neighbours affects the focal's fitness (the "benefit" B), weighted by relatedness r_2°.

Relatedness here quantifies mutant–mutant interactions. Hamilton's rule highlights their evolutionary significance: interactions among relatives tend to favour prosocial traits (traits with positive indirect fitness). Note that, by a change of perspective, the indirect term can equivalently be read as the effect of the *focal* on its neighbour; hence the term "inclusive fitness effect."

Hamilton's (marginal) rule : advantages

A key advantage of Hamilton's rule over working directly with invasion fitness is its simplicity: invasion fitness depends on the entire distribution of group compositions $q_k(y, x)$, Hamilton's rule depends only on neutral relatedness, a single moment of that distribution. Further advantages:

1. Relatedness coefficients can be computed using standard coalescent methods (we will work through an example on the board).
2. Relatedness can be systematically estimated in natural populations, since r_2° is directly connected to F_{ST}.

Because of this simplicity, empirical accessibility, and the biological insights it affords, Hamilton's rule has become one of the most widely used tools for understanding directional selection on social traits such as cooperation, sex ratio, and dispersal.

Disruptive selection: the basis of social polymorphism

Directional selection will lead the population to express a singular strategy that balances direct and indirect fitness effects. Will disruptive selection then favour polymorphism ? In group-structured populations, disruptive selection can also be decomposed into two components:

$$H(x^*) \;=\; H_{\mathrm{w}}(x^*) \;+\; 2 \times H_{\mathrm{r}}(x^*). \tag{12}$$

- H_{w}: curvature due to non-linear effects of traits on fitness.
- H_{r}: interaction between variation in social context and fitness synergy.

Together these two pathways capture how disruptive selection — and hence polymorphism — can emerge in structured populations.

Non-linear fitness effects

The first pathway is

$$H_{\mathrm{w}}(x^*) = \left.\frac{\partial^2 w(z_\bullet, \mathbf{z}_n)}{\partial {z_\bullet}^2}\right|_{z_i=x^*} + 2(n-1)\, r_2^\circ \left.\frac{\partial^2 w(z_\bullet, \mathbf{z}_n)}{\partial z_\bullet \partial z_1}\right|_{z_i=x^*} + (n-1)\, r_2^\circ \left.\frac{\partial^2 w(z_\bullet, \mathbf{z}_n)}{\partial {z_1}^2}\right|_{z_i=x^*} + (n-1)(n-2)\, r_3^\circ \left.\frac{\partial^2 w(z_\bullet, \mathbf{z}_n)}{\partial z_1 \partial z_2}\right|_{z_i=x^*}. \tag{13}$$

where

$$r_3^\circ = \sum_{k=1}^{n} \frac{k-1}{n-1}\frac{k-2}{n-2}\, q_k(x,x) \tag{14}$$

is the **three-way relatedness**: the probability that two neighbours of a mutant are also mutant under neutrality. This reflects the tendency of interacting with more than one relative within a group under neutrality.

Relatedness inhibits polymorphism under negative trait-dependent selection

- Non-linear fitness effects depend on **synergistic effects of neighbours** on fitness: – between focal and neighbour, and – among neighbours.
- Example: cooperation with antagonistic payoffs (snowdrift game), i.e. when

$$\partial^2 w(z_\bullet, \mathbf{z}_n)/(\partial z_\bullet \partial z_1) < 0.$$

- In this case, increasing relatedness tends to reduce disruptive selection ($H(x^*)$ decreases with r_2°).
- Put differently: kin selection favours equal contribution among partners and thereby inhibits polymorphism.

Social context $\times$ fitness synergy

While relatedness can inhibit one form of polymorphism, it can open another. The second pathway contributing to disruptive selection is

$$H_{\mathrm{r}}(x^*) \;=\; (n-1)\,\frac{\partial w(z_\bullet, \mathbf{z}_n)}{\partial z_1}\bigg|_{z_i=x^*} \frac{\partial r_2(y,x)}{\partial y}\bigg|_{y=x=x^*}, \tag{15}$$

where

$$r_2(y,x) = \sum_{k=1}^{n} \frac{k-1}{n-1}\, q_k(y,x) \tag{16}$$

This term is the **product of two factors**:

1. how a trait change in neighbours affects focal fitness,
2. how a trait change affects the probability of interacting with relatives (change in relatedness).

Example: cooperation–dispersal trade-off

Hence disruptive selection favours mutants that either:

- increase neighbour fitness and chance those neighbours are mutants, or
- decrease neighbour fitness and chance those neighbours are mutants.

Example: Consider a trait that increases investment in a common good (helping neighbours) but reduces dispersal propensity (due to a trade-off).

Consequences:

- Mutants that invest more cooperate more, disperse less, and thus interact more with relatives.
- Defectors invest less, disperse more, and thus interact mainly with non-relatives.

Social evolution : relatedness, kin competition and reciprocity

The ubiquity of social traits

Broadly speaking, a **social trait** (or behaviour) is any trait that has indirect effects on the fitness of others. Formally:

$$B \;=\; \partial w(z_\bullet, \mathbf{z}_n)/(\partial z_1) \;\neq\; 0.$$

Social traits are ubiquitous in nature. Some biologists even argue that *all* evolution is social, since virtually all traits influence not only their bearer but also others — either directly (e.g. through ccoperative breeding) or indirectly (e.g. through altering the local environment).

The four types of social traits

We can classify social traits according to their direct ($-C$) and indirect (B) fitness effects:

Harming traits ($B < 0$):

- **Selfishness**: direct benefit ($-C > 0$), e.g. stealing food from conspecifics.
- **Spite**: direct cost ($-C < 0$), e.g. bacteriocin production in bacteria that kills competitors at a cost to the producer.

Helping traits ($B > 0$):

- **Cooperation**: direct benefit ($-C > 0$), e.g. group hunting in carnivores.
- **Altruism**: direct cost ($-C < 0$), e.g. sterile worker ants helping the colony.

Altruism requires positive relatedness

The evolution of altruism has fascinated biologists since Darwin, because it seems puzzling that an individual would reduce its own fitness to increase that of others.

Hamilton's key insight was that altruism can evolve if it preferentially benefits relatives, thereby increasing the transmission of the underlying allele.

Much confusion has surrounded the topic, partly due to terminology and the tendency to conflate **cooperation** (mutual benefit) with **altruism** (net cost to the actor).

Limited dispersal: a driver of altruism?

Helping behaviours are favoured by high relatedness, which naturally arises under limited dispersal as extended families interact more often.

But limited dispersal also introduces **kin competition**: relatives compete locally for resources, so that improving the fitness of neighbours may reduce the success of one's own offspring or other relatives.

This tension between the benefits of relatedness and the costs of kin competition is central to understanding the evolution of altruism. It explains why high relatedness is a *necessary but not sufficient* condition for altruism to evolve.

Selection via payoff effects: kin competition

Assume fitness depends on an intermediate quantity "payoff" (e.g. fecundity)

$$w(z_\bullet, \mathbf{z}_n) = \tilde{w}(\pi_\bullet, \pi_1, \ldots, \pi_{n-1}, \pi_x) = \tilde{w}_\bullet$$

where π_j is the payoff of individual $j \in \{\bullet, 1, \ldots, n-1, x\}$ which depends on the trait according to

$$\pi_j = \pi(z_j, \mathbf{z}_{-j}, x)$$

where $\mathbf{z}_{-j}$ denotes the vector of trait values in the patch with actor j removed. Then, the selection gradient reduces to

$$S(x) \propto \left.\frac{\partial \pi_\bullet}{\partial z_\bullet}\right|_{z_i = x} + (n-1)\,\kappa^\circ \left.\frac{\partial \pi_\bullet}{\partial z_1}\right|_{z_i = x}, \tag{17}$$

where

$$\kappa^\circ = \frac{1}{1 - \alpha r_2^\circ}\left(r_2^\circ - \frac{\alpha}{n-1}\left[1 + (n-2) r_2^\circ\right]\right) \tag{18}$$

is **scaled relatedness**.

Balancing the benefits and costs of interacting with relatives

Let's take a closer look at scaled relatedness

$$\kappa^{\circ} = \frac{1}{1-\alpha r_2^{\circ}} \left(r_2^{\circ} - \frac{\alpha}{n-1} \left[1 + (n-2) r_2^{\circ} \right] \right) \tag{19}$$

where

$$0 \leq \alpha = -(n-1) \frac{\partial \tilde{w}}{\partial \pi_1} \Big/ \left. \frac{\partial \tilde{w}}{\partial \pi_{\bullet}} \right|_{z_i = x} \leq 1 \tag{20}$$

measures the scale of competition: $\alpha = 0$ entirely global, $\alpha = 1$ entirely local. Therefore global competition favours helping because it reduces kin competition.

Two avenues for kin competition ($[1 + (n-2) r_2^{\circ}]$) (i) "1" : direct competition for the focal. (ii) "$(n-2) r_2^{\circ}$" : indirect competition for the focal's relatives.

Scaled relatedness κ° thus discounts the effect of relatedness by the intensity of local competition.

Kin competition can cancel the benefits of relatedness

A famous result in social evolution is **Taylor's cancellation**: in an island model with fixed group size, non-overlapping generations, and when the trait affects fecundity,

$$\kappa^{\circ} = 0,$$

so that kin competition exactly cancels the benefits of interacting with relatives.

However, there are many ways to overteurn this result. We saw for instance that to if generations overlap, surviving adults reduce the intensity of kin competition. This boosts effective relatedness, making $\kappa^{\circ} > 0$ and allowing cooperation to evolve. Elatsic local population size can also do this.

Cooperation can evolve without relatedness under repeated interactions

There have been 1000s of papers on the evolution of cooperation. In spite of this, the fundamental mechanisms can be reduced to just two:

1. kin selection, as we saw,
2. reciprocity, whereby individuals help now in order to reap benefits later through repeated interactions.

Reciprocity allows cooperation to evolve even in well-mixed populations. It is driven by direct fitness effects (reciprocity is not altruism!).

An example for the evolution of cooperation via reciprocity

Consider a well-mixed population of fixed size N with the following life cycle :

1. Individuals randomly pair up and each chooses an action $a_\bullet = a(z_\bullet, z_1)$ (investment into cooperation) depending on its trait $z_\bullet$ and that of its partner z_1.
2. Each individual obtains a payoff $\pi(a_\bullet, a_1) = 1 + b\, a_1 - c\, a_\bullet$, determined by its own investment and that of its partner. Payoff maps to fecundity.
3. Adults produce offspring in proportion to their fecundity and then die.
4. Offspring compete randomly to become adults of the next generation.

The selection gradient under reciprocity

For this model,

$$S(x) \propto -c + \lambda^{\circ} b \tag{21}$$

where

$$\lambda^{\circ} = \frac{\partial a(z_1, z_\bullet)}{\partial z_\bullet} \Bigg/ \left. \frac{\partial a(z_\bullet, z_1)}{\partial z_\bullet} \right|_{z_i = x} \tag{22}$$

is a **response coefficient**, measuring how much the partner changes its action in response to a trait-driven change in the focal's action :

- $\lambda^{\circ} > 0$ – partners change action in the same way, i.e. reciprocity.
- $\lambda^{\circ} < 0$ – partners change action in opposite ways.

The response coefficient enters the selection gradient as relatedness does in the island model. It shows that cooperation can evolve when there is a mechanism of reciprocity in place such that $\lambda^{\circ} > 0$. If there's time, we'll model such an evolution on the white board.

Summary

- Limited dispersal leads to mutant-mutant interactions, which inevitably generate kin selection
- Selection then depends on relatedness-weighted indirect effects on individual fitness, where relatedness captures the frequency of mutant-mutant interactions
- Directional selection then tends to favour helping though this can be offset by kin competition
- Limited dispersal also generates a form of disruptive selection that favours social polymorphism with different morphs showing different preferential interactions
- An alternative mechanism for the evolution of helping is reciprocity, which emerges from repeated interactions with behavioural responses

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