

Supplementary material for “The evolution of trans-generational altruism: kin selection meets niche construction”

The selective pressure on a trait that results in both intra and inter-temporal effects on fitness will be evaluated in this supplementary material. This is done by extending the probability of fixation method developed in Rousset (2003) and Rousset (2004, chapter 6), whose analysis is followed here very closely. The results essentially demonstrate that individuals affecting the fitness of other individuals living in different time periods can be treated as different “classes” of actors in the same manner as is usually done in inclusive fitness theory for sex, age, geography or other kind of kin classes (Taylor, 1990, 1996; Taylor and Frank, 1996; Frank, 1998; Rousset and Billiard, 2000; Rousset, 2004). The selective pressure for the infinite island model of dispersal used in the main text is then obtained as a particular case of the approach.

A convergence measure of stability of a trait under selection can be obtained by analyzing the probability of fixation of a single mutant allele (say A) whose phenotypic effect deviates (with small magnitude δ) from the phenotype expressed by a resident allele (say a) fixed in the population. The direction of evolutionary change on the mutant is then determined by asking whether it has a larger or smaller probability of fixation than a neutral mutant. That is, does the phenotypic deviation δ results in a positive or negative effect on the probability of fixation of the mutant. This increment or decrement in the probability of fixation can be expressed as a function of the effect of the mutant on the between-generation change in expected average allele frequency $\Delta\bar{p}(t)$ in the population according to the equation

$$\phi = \text{E} \left[\sum_{t=0}^{\infty} \frac{d\Delta\bar{p}(t)}{d\delta} \right], \quad (1)$$

where the sum adds up until fixation of either the mutant or the resident allele (Rousset, 2003, p. 666).

Let us consider that evolution occurs in a population following the haploid island or isolation by distance model of dispersal. There are n_d demes, each with N adults so that the total population size is of constant size $N_T = n_d N$. For simplicity, we assume that dispersal is isotropic and homogeneous and that environmental conditions are the same in each deme. Accordingly, the fitness of each individual is independent of the geographical position of the deme in which it resides and its dispersal distribution is symmetric and the

same in each deme. Let a random sequence of mutant allele frequencies in the population since its appearance and up to time t be denoted by

$$\omega(t) \equiv \{\mathbf{p}(t), \mathbf{p}(t-1), \dots, \mathbf{p}(1), \mathbf{p}(0)\}, \quad (2)$$

where

$$\mathbf{p}(t) \equiv (p_1(t), \dots, p_{n_d}(t)) \quad (3)$$

is the vector of the frequencies of the mutant allele in the different demes at time t and $p_i(t)$ is the frequency of the mutant in deme i . In the presence of intra and inter-temporal effects on fitness, the change in allele frequency $\Delta\bar{p}(t)$ at generation t may depend on the whole history of allele frequencies $\omega(t)$ in the population up to time t . The change over one generation is thus conditional on the various realizations of deme gene frequencies and can be written as

$$\Delta\bar{p}(t) = \sum_{\omega(t)} (\mathbb{E}[\bar{p}(t+1)|\omega(t)] - \bar{p}(t)) \Pr(\omega(t)), \quad (4)$$

where $\Pr(\omega(t))$ is the probability of the occurrence of the sequence $\omega(t)$.

Following the argument given in Rousset (2003, eq. 4), the effect of the mutant on its probability of fixation is then given by

$$\phi = \mathbb{E}^\circ \left[\sum_{t=0}^{\infty} \frac{d}{d\delta} (\mathbb{E}[\bar{p}(t+1)|\omega(t)] - \bar{p}(t)) \right], \quad (5)$$

which is equivalent to eq. 5 of Rousset (2003) except that the expectation \mathbb{E}° over realization of deme gene frequencies in the neutral process is running here over all sequences $\omega(t)$ of present and past mutant gene distributions.

The sensitivity of average gene frequency change to mutant deviation in eq. 5 can be computed from the direct fitness of a focal individual $w \equiv w(z_\bullet, \dots, z_{k,h}, \dots)$, which is expressed here as a function the phenotype of the focal individual $z_\bullet = z_a + \delta$ and as a function of the average phenotypes $z_{k,h} = z_a + \delta p_{k,h}$ of individuals located in different demes (labeled k) at different points in time (labeled h) prior to the focal generation, where $p_{k,h}$ is the frequency of the mutant allele in such demes and z_a is the phenotype of the resident. Then, following the same development as Rousset (2003, eq. 9 to eq. 11), the first order effect of the mutant on its probability of fixation is given by

$$\phi = \mathbb{E}^\circ \left[\sum_{t=0}^{\infty} \left(\frac{\partial w}{\partial z_\bullet} + \sum_{h=0}^T \sum_{k=1}^{n_d} \frac{\partial w}{\partial z_{k,h}} p_k(t-h) \right) p_0(t) \right], \quad (6)$$

where $p_0(t)$ is the frequency of the mutant allele in a focal deme at time t , $p_k(t-h)$ is the frequency of the mutant in a deme at distance k from the focal deme at h generations prior to t (if $h > t$, $p_k(t-h) = 0$) and T is the time horizon of inter-temporal effects on fitness. Since we assumed a homogeneous environment and isotropic dispersal, only the spatial and time separation of gene frequencies relative to a focal deme in a focal generation matter to evaluate the selective pressure on the mutant. This is the reason why we do not have the sum outside the parentheses given in eq. 11 of Rousset (2003).

By the property that the partial derivatives of the fitness function sum up to zero (Rousset, 2004, eq. 6.9), eq. 6 can be written as

$$\phi = \sum_{k=1}^{n_d} \sum_{h=0}^T \frac{\partial w}{\partial z_{k,h}} \mathbb{E}^\circ \left[\sum_{t=0}^{\infty} (p_0(t)p_k(t-h) - p_0(t)) \right]. \quad (7)$$

The mutant appears as single copy in the population and its initial frequency in the deme where it appears is $1/N$. The probability that the mutant appears in a given focal deme is $1/n_d$, hence $\mathbb{E}^\circ [p_0] = 1/N_T$. The probability that a gene taken in a focal deme at time t and another gene taken in a deme at distance k from the focal deme at h generations prior to t are both mutants is given by

$$\begin{aligned} \mathbb{E}^\circ [p_0(t)p_k(t-h)] &= \frac{1}{N_T} \sum_{g=0}^t C_{k,h}(g) \\ &= \frac{1}{N_T} \sum_{g=h}^t C_{k,h}(g), \end{aligned} \quad (8)$$

where $C_{k,h}(g)$ is the probability that a gene taken in a focal deme and another gene taken in a deme at distance k from the focal deme at h generations prior to the focal generation coalescence in a common ancestor living in generation g prior to h ($g \geq h$). We can now

write

$$\begin{aligned}
\mathbb{E}^\circ \left[\sum_{t=0}^{\infty} (p_0(t) - p_0(t)p_k(t-h)) \right] &= \frac{1}{N_T} \sum_{t=0}^{\infty} \left(1 - \sum_{g=h}^t C_{k,h}(g) \right) \\
&= \frac{1}{N_T} \sum_{t=0}^{\infty} \left(\sum_{g=h}^{\infty} C_{k,h}(g) - \sum_{g=h}^t C_{k,h}(g) \right) \\
&= \frac{1}{N_T} \sum_{t=0}^{\infty} \sum_{g=t+1}^{\infty} C_{k,h}(g) \\
&= \frac{1}{N_T} \sum_{t=0}^{\infty} t C_{k,h}(t) \\
&= \frac{T_{k,h}}{N_T}, \tag{9}
\end{aligned}$$

where the second equality is obtained by using the identity $\sum_{g=h}^{\infty} C_{k,h}(g) = 1$, which amounts to say that two genes sampled at k generations of interval must eventually coalesce in a common ancestor if we look sufficiently far enough into the past. The last two steps are in fact strictly equivalent to those given in eq. 14 of Rousset (2003) and $T_{k,h}$ designates the average coalescence time between two genes, one sampled in the focal deme and the other sampled in a deme at distance k from the focal deme at h generations prior to the focal generation.

Then, following again the line of arguments spelt out in Rousset (2003), the effect of the mutant on its probability of fixation is given by

$$\phi = \lim_{\mu \rightarrow 0} \frac{S}{1 - r_0}, \tag{10}$$

where r_0 is probability of identity of a pair of genes within a deme and S is the inclusive fitness effect measuring the direction of selection on the mutant allele. The inclusive fitness effect takes here the form

$$S = \frac{\partial w}{\partial z_{\bullet}} + \sum_{h=0}^T \sum_{k=1}^{n_d} \frac{\partial w}{\partial z_{k,h}} r_{k,h}, \tag{11}$$

where $r_{k,h}$ is the probability that a gene sampled in a focal individual is identical to a homologous gene sampled in an individual of class k living h generations prior to the focal generation. In the infinite island model of dispersal (that is when $n_d \rightarrow \infty$), we have $r_{k,h} = 0$ for all k except $r_{0,h} \geq 0$ and the inclusive fitness effect gives the first order effect of selection on gene frequency change (Rousset, 2004, pp. 206–207). The resulting change in average

mutant allele frequency p in the population can then be written as

$$\Delta p = \delta S p(1 - p) + O(\delta^2), \quad (12)$$

where $O(\delta^2)$ is a remainder of phenotypic effects on fitness of second order in δ .

References

- Frank, S. A. 1998. Foundations of social evolution. Princeton University Press, Princeton, NJ.
- Rousset, F. 2003. A minimal derivation of convergence stability measures. *Journal of Theoretical Biology* 221:665–668.
- Rousset, F. 2004. Genetic structure and selection in subdivided populations. Princeton University Press, Princeton, NJ.
- Rousset, F. and S. Billiard. 2000. A theoretical basis for measures of kin selection in subdivided populations: finite populations and localized dispersal. *Journal of Evolutionary Biology* 13:814–825.
- Taylor, P. 1990. Allele-frequency change in a class-structured population. *American Naturalist* 135:95–106.
- Taylor, P. D. 1996. Inclusive fitness arguments in genetic models of behaviour. *Journal of Mathematical Biology* 34:654–674.
- Taylor, P. D. and S. A. Frank. 1996. How to make a kin selection model. *Journal of Theoretical Biology* 180:27–37.