



Crozier's paradox and kin recognition: Insights from simplified models

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ABSTRACT

Crozier's paradox suggests that genetic kin recognition will not be evolutionarily stable. The problem is that more common tags (markers) are more likely to be recognised and helped. This causes common tags to increase in frequency, eliminating the genetic variability that is required for genetic kin recognition. In recent years, theoretical models have resolved Crozier's paradox in different ways, but they are based on very complicated multi-locus population genetics. Consequently, it is hard to see exactly what is going on, and whether different theoretical resolutions of Crozier's paradox lead to different types of kin discrimination. I address this by making unrealistic simplifying assumptions to produce a more tractable and understandable model of Crozier's paradox. I use this to interpret a more complex multi-locus population genetic model where I have not made the same simplifying assumptions. I explain how Crozier's paradox can be resolved, and show that only one known theoretical resolution of Crozier's paradox – multiple social encounters – leads without restrictive assumptions to the type of highly cooperative and reliable form of kin discrimination that we observe in nature. More generally, I show how adopting a methodological approach where complex models are compared with simplified ones can lead to greater understanding and accessibility.

1. Introduction

Kin selection theory predicts that individuals should preferentially help their closer relatives (Hamilton, 1964). The conditional helping of closer relatives, termed *kin discrimination*, is favoured because relatives share genes, and so by helping a relative reproduce, an individual is still passing its genes to the next generation, just indirectly. This process requires *kin recognition*, which is the identification of relatives through either environmental or genetic cues (Bourke, 2011; Leedale et al., 2020b).

Kin recognition via genetic cues is not necessarily evolutionarily stable (Bourke, 2011; Crozier, 1986; Field et al., 2018; Gardner and West, 2007; Gilbert, 2015; Holman et al., 2013; Leedale et al., 2020b; Madgwick et al., 2019; Rousset and Roze, 2007; Strassmann et al., 2011). The problem, known as Crozier's paradox, is that more common tags (markers) at the recognition locus are more likely to be recognised (Crozier, 1986). Consequently, individuals with more common tags are more likely to be helped, increasing their fitness (Fig. 1). In contrast, individuals with rare tags are less likely to be recognised and helped, reducing their relative fitness. This means that common tags will increase in frequency, and rare tags will decrease in frequency and be lost (positive frequency dependence). Therefore, genetic kin recognition drives its own ruin, by eliminating the genetic variability that is required

for genetic kin recognition. Despite this potential for instability, genetic kin recognition has been observed in animals, microorganisms and plants, suggesting that Crozier's paradox has been solved by nature (Benabentos et al., 2009; Charpentier et al., 2007; Dudley and File, 2007; Green et al., 2015; Lihoreau and Rivault, 2009; McDonald and Wright, 2011; Mehdiabadi et al., 2006; Rosengarten and Nicotra, 2011).

Crozier's paradox has been theoretically resolved in three ways. First, Scott et al. (2022) showed that Crozier's paradox can be resolved if individuals have multiple social encounters, because this allows individuals with rare tags to find others with the same tag, preventing common tags from being more likely to be helped. In terms of Fig. 1a, this means that the orange birds find other orange birds to pair up with. Second, it has been shown theoretically that genetic kin recognition can be stable even without multiple social encounters, if there is: extreme spatial structure (low migration); tight linkage between the tag locus and the helping (trait) locus; strong selection (Axelrod et al., 2004; Jansen and van Baalen, 2006; Rousset and Roze, 2007; Scott et al., 2022). This hypothesis has been called 'beard chromodynamics' because of its reliance on physical tag-trait linkage, which is a characteristic of greenbeard genes (Biernaskie et al., 2013; Gardner and West, 2010; Jansen and van Baalen, 2006; Madgwick et al., 2019). Third, Crozier (1986) argued that genetic kin recognition could be stabilised if the recognition tags have an additional (pleiotropic) role unrelated to social

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behaviour that maintains tag diversity, like a role in parasite resistance (Crozier, 1986; Gardner and West, 2007; Holman et al., 2013; Rousset and Roze, 2007). Scott et al. (2023) clarified that host-parasite coevolution can only stabilise genetic kin recognition if parasites evolve rapidly and have intermediate or high virulence.

Previous theory is based on multi-locus population genetics (Gardner et al., 2007; Kirkpatrick et al., 2002; Roze and Rousset, 2008). This is the gold standard for examining whether hypotheses in evolutionary biology stand up to theoretical scrutiny, but its significant complexity leads to two downsides. First, it is hard to interpret the models, to see exactly what is going on, and how results arise. Second, it is hard to compare between different models, to see whether different types of kin discrimination evolve in different scenarios. It is possible that some of the theoretical resolutions of Crozier's paradox do not actually facilitate the type of kin discrimination that we see in nature, for instance the highly cooperative and reliable form of kin recognition that we observe in cooperatively breeding animals.

I resolve these issues using a mixture of modelling techniques that have different advantages and disadvantages. I use two approaches: (1) deliberately simplifying and unrealistic assumptions to produce a more tractable and understandable model of genetic kin recognition; (2) a more complex multi-locus population genetic model where I have not made the same simplifying assumptions. I obtain the simpler model by assuming that selection on recognition tags proceeds very slowly compared to selection on social traits (separation of timescales). I examine how genetic kin recognition evolves in the simple model, and use this to explain how: (i) genetic kin recognition evolves in the complex multi-locus model; (ii) different resolutions of Crozier's paradox lead to the evolution of different types of kin discrimination (varying in reliability, cooperativeness, stability, polymorphism, etc.). I focus on the 'multiple social encounters' (Scott et al., 2022) and 'beard chromodynamics' (Jansen and van Baalen, 2006) resolutions of Crozier's paradox. I have analysed another possibility, the 'host-parasite coevolution' resolution, elsewhere (Scott et al., 2023). Finally, I examine which of the different known resolutions of Crozier's paradox is likely to provide the most robust explanation for genetic kin recognition in nature.

2. Models

2.1. Roadmap

I construct and analyse two models – a simple model, and a complex model. I do so in the following five sections:

- Constructing the complex model. In this section, I construct a multi-locus population genetic model of genetic kin recognition. To do so, first I describe my model assumptions. Then, I write equations to describe how genotype frequencies change across a generation (recursions). After constructing the model, I do not analyse it straight away. Instead, I ask when inclusive fitness theory predicts kin discrimination should evolve.
- Inclusive-fitness predictions. I examine when kin discrimination (help relatives) confers greater inclusive fitness returns to the actor than indiscriminate defection (never help) and indiscriminate helping (help everyone), meaning it is favoured by kin selection. This predicts when we should expect to find genetic kin recognition. These predictions are high-level, in that they do not examine how genetic details come into play, but they provide useful framing for subsequent genetic analyses.
- Constructing the simple model. I assume that evolution at the trait locus (whether to provide help) proceeds much more quickly than evolution at the tag locus. The separation of timescales assumption is unrealistic, and I take it only because it simplifies the multi-locus model, and allows us to see things that would have otherwise been buried in the model's complexity.
- Analysing the simple model. I examine: when genetic kin recognition evolves; whether it can be understood in terms of fitness optimisation; how the amount of tag diversity, divergence, oscillation and cheat load vary across different areas of parameter space, leading to different types of kin discrimination.
- Analysing the complex model. I relax the separation of timescales assumption, so that evolution at the tag and trait loci proceed at a comparable rate. This is the complex model described in the initial 'Constructing the complex model' section. I use this model to verify

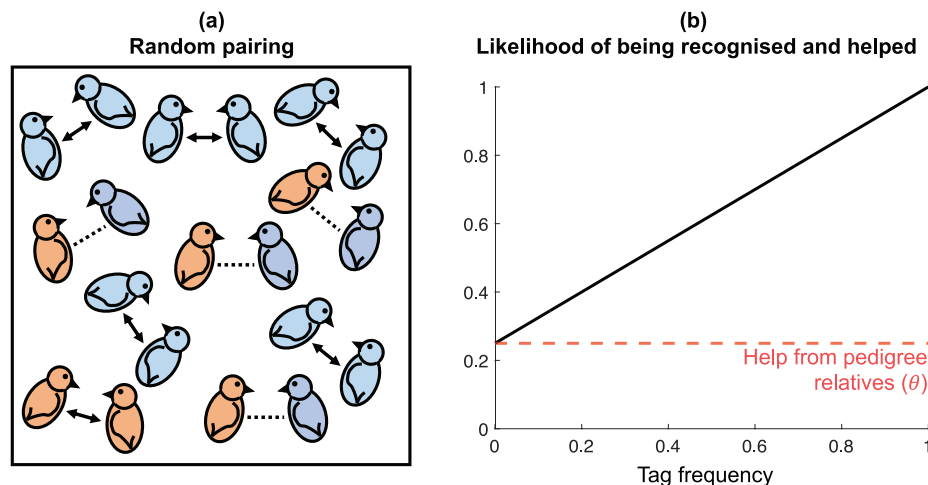


Fig. 1. Crozier's paradox. (A) Birds with a more common genetic tag (blue) are more likely to encounter birds with the same tag, compared to birds with a less common tag (orange). (B) Consequently, in the absence of multiple social encounters ($\alpha = 0$), individuals with more common tags are more likely to be recognised and helped (black solid line). Rare tags are only recognised and helped by pedigree (genealogical) relatives (red dotted line), but common tags are additionally recognised and helped by non-relatives. I assumed parameter values that lead to Crozier's paradox: $\alpha = 0$ (encounter parameter), $b = 0.4$ (helping benefit), $c = 0.1$ (helping cost), $\mu_{\text{Trait}} = 0$ (trait mutation rate); individuals have a 25 % chance of encountering a clone and a 75 % chance of encountering a non-relative ($\theta = 0.25$; θ is population viscosity), meaning individuals evolve to help whoever they recognise ($\theta b > c$). Figure previously published in Scott et al. (2023), Creative Commons (<https://creativecommons.org/licenses/by/3.0/>). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

the results of the simple model, to examine when and why they hold, and I use the simple model to interpret the complex model.

2.2. Constructing the complex model

2.2.1. Tag & Trait loci

I assume a very large (effectively infinite) population of individuals and that, at the start of each generation, individuals are haploid. Each individual encodes a given phenotype (tag). Tags are recognised by other individuals, and distinguishable. There are L_{max} possible tags, and each tag is encoded by a specific allele at the 'tag locus' (L_{max} possible alleles at the tag locus). L_{max} therefore gives the upper bound on the number of tags that can be distinguished between ('tag availability'). This upper bound (L_{max}) is set by the efficacy of the sensory system responsible for recognising the tags (evolutionary constraint). More sophisticated sensory systems will be capable of reliably distinguishing between greater numbers of tags (higher L_{max}). Each allele at the tag locus is denoted by a number, i , within the set $i \in \{1, 2, \dots, L_{max}\}$. At a given point in time, the number of segregating tags (i.e., number of tags present at nonzero population frequency) is denoted by L ($1 \leq L \leq L_{max}$). At equilibrium, the number of segregating tags is denoted by L^* (L tends to L^* in the evolutionary long term).

Each individual also adopts a given 'trait', which dictates how it behaves in social interactions. There are two possible traits, and each trait is encoded by a specific allele at the 'trait locus' (2 possible alleles at the trait locus). Trait allele '1' encodes (conditional) helping and trait allele '0' encodes defection. In addition to conditional helping and defection, there is a third possible trait phenotype – indiscriminate helping (help everyone). However, for simplicity, I do not explicitly track an indiscriminate helping allele at the trait locus. Instead, I note that the 'indiscriminate helping' phenotype can still evolve in my models, if tag diversity is lost (one tag goes to fixation) and the conditional helping allele goes to fixation.

2.2.2. Genotype frequency notation

The population frequency of conditional helpers bearing a given tag i is denoted by x_{i1} . The population frequency of defectors bearing a given tag i is denoted by x_{i0} . The overall population frequency of a given tag i is given by $x_{i1} + x_{i0}$, and denoted by x_i . The proportion of individuals bearing a given tag (i) that are helpers ('helper proportion') is given by x_{i1}/x_i , and denoted by p_i . The proportion of individuals bearing a given tag (i) that are cheaters ('cheat load') is therefore given by $1 - p_i$.

2.2.3. Social encounters & interactions

For each individual, in each generation, I define an 'interaction group'. This is the group of neighbours with whom the individual is close enough to socially interact with. Social interactions are pairwise and asymmetrical, comprising one actor (who may give help) and one recipient (who may receive help).

Each individual has one 'social search' per generation. In a given social search, a focal individual encounters a random member of its interaction group (partner). If the focal individual and its partner share the same tag, they interact, with the focal individual potentially giving help (actor) and its partner potentially receiving help (recipient) – the social encounter becomes a social interaction (successful social search).

In contrast, if the focal individual and its partner do not share the same tag, what happens depends upon the encounter parameter, α . With a probability α , the focal individual abandons its tag-mismatched partner and re-associates for a new social encounter, with a new partner drawn at random from its interaction group (Fig. 2). With a probability $1 - \alpha$, the focal individual remains with its tag-mismatched partner, but they do not interact – the opportunity to socially interact is wasted (failed social search).

I reiterate that a given individual has one social search per generation, meaning it socially interacts as an actor either once (successful social search) or zero (failed social search) times per generation.

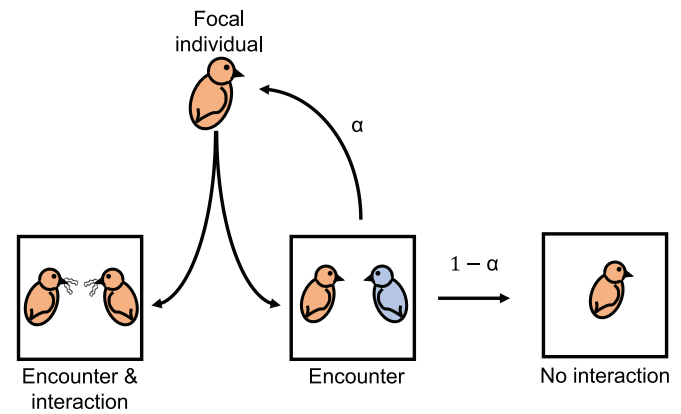


Fig. 2. Social encounters and social interactions. If the focal individual encounters a tag-matched individual (both orange), it socially interacts. Conversely, if the focal individual encounters a tag-mismatched individual (one orange; one blue), the focal individual may encounter a new partner (α), or forgo the social search ($1 - \alpha$). Higher values of the encounter parameter (α) correspond to individuals having more encounters to find a matching partner (partner search). During an interaction with a (tag-matched) partner, the focal individual may help or not (defect), depending upon its allele at the trait locus. Figure previously published in Scott et al. (2022), Creative Commons (<https://creativecommons.org/licenses/by/3.0/>). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

However, a given individual may be chosen once, zero or multiple times per generation, by other individuals on their social searches. A given individual may therefore socially interact as a recipient once, zero or multiple times per generation.

When $\alpha = 1$, individuals are free to have encounters with all the other individuals in their interaction group, if need-be, to find a tag-matched individual to interact with. In this case, even individuals with a rare tag are still likely to interact with another individual with the same tag. At the other extreme, when $\alpha = 0$, any individual who doesn't encounter a tag-matched individual on its first try does not get to engage in a social interaction, and so population tag frequency will determine the rate of interaction. These assumptions allow natural forms of social behaviour to be captured, such as the behaviour of cooperatively breeding vertebrates, where individuals can encounter several individuals before providing help (Scott et al., 2022).

2.2.4. Cost of abandoning social partners

I assume that searching for partners is costly. Specifically, each time an individual abandons a social partner for a new social encounter, it pays a fecundity cost of c_{search} . The search cost (c_{search}) is incurred every time an individual abandons a partner and re-associates for a new social encounter. Consequently, the total search cost can be much higher than c_{search} . For limiting rare tags under conditions of limiting low relatedness, the total search cost will be $\frac{\alpha c_{search}}{1 - \alpha}$. The search cost can capture many different things empirically, such as an increased predation risk whilst out searching for a social partner, or a loss of time that could be spent doing other things like foraging for food.

2.2.5. Cooperative Game

For each social interaction, which comprises pairs of individuals sharing the same tag, there is one actor and one recipient. The actor and recipient play a (nonreciprocal) helping game. Actors provide help (cooperate) if they have the conditional helping allele, suffering a fecundity cost of c to give a benefit of b to the recipient. Actors don't provide help if they have the defection allele. Recipients do not provide help. There is a net benefit to helping ($b > c$).

2.2.6. Mutation

I assume that trait mutation occurs with the generational probability μ_{Trait} . I assume that there is no mutation at the tag locus, because tag mutation can maintain tag diversity even when it is disfavoured by selection, leading to spurious (non-adaptive) kin recognition. I am interested in when *selection* maintains tag diversity.

2.2.7. Assigning interaction groups

I assume that, within an individual's interaction group, a proportion of individuals, θ , are genealogical clones (identical by descent at both the tag and the trait locus). The remaining proportion, $1-\theta$, are drawn randomly from the population (identical by descent at neither the tag or trait locus). The parameter θ is a measure of population viscosity: higher values of θ mean that individuals are situated closer to their relatives.

2.2.8. Lifecycle

I assume a very large (effectively infinite) population of haploids. First, haploid individuals have the opportunity to socially interact, as detailed in section 2.2.3 above. Next, haploid individuals produce a very large number of gametes, before dying, where an individual's fecundity is given by how well it fared across the social encounters. Next, gametes fuse randomly (panmictic) to produce diploid zygotes, and this is followed immediately by meiosis, with recombination between tag and trait loci occurring with probability r . The haploid juveniles then undergo mutation, as detailed in section 2.2.6 above. Finally, haploid adults are sampled (randomly, without replacement) from the haploid juvenile population, such that the resulting adult haploid population is the same size as the adult haploid population at the start of the lifecycle (population regulation occurs at the global level). This completes the lifecycle. I describe the relation between this lifecycle and previous theory in Appendix A in the supplemental material.

2.2.9. Mathematical representation of the lifecycle assumptions

Based on my lifecycle assumptions, I write equations to describe how the population frequency of a given genotype changes across a generation. Specifically, I write three equations, partitioning the respective effects of selection, recombination and mutation, on genotype frequency change. I assume that: (1) Selection takes the frequency of a genotype (ij) from x_{ij} to x'_{ij} , where i gives tag identity, and j gives trait identity, with $j = 1$ for a conditional helper and $j = 0$ for a defector. (2) Recombination takes genotype frequency from x'_{ij} to x''_{ij} . (3) Mutation takes genotype frequency from x''_{ij} to x'''_{ij} . I partition my model in this way – as three successive equations – because the logic behind my model is clearer when the effects of selection, recombination and mutation are presented in isolation from each other. Taken together, I obtain a recursion describing the change in frequency of a genotype (ij) from one generation (x_{ij}) to the next (x'''_{ij}).

Readers uninterested in the mathematical details of the full multi-locus model may skip the rest of section 2.2, as it is simply turning the lifecycle assumptions listed above into algebra.

2.2.10. Selection

Following standard population genetic methodology, I take an individual's (absolute) fitness to be its number of adult offspring (i.e., its number of descendants after one full iteration of the life cycle) (Hamilton, 1964; Rousset, 2004). In lifecycles, such as the one considered here, where population size remains constant over time (the population is not growing or shrinking), the population average absolute fitness is necessarily 1, and absolute fitness is equivalent to relative fitness (where relative fitness is given by dividing absolute fitness by the population average absolute fitness). I use w_{ij} to denote the absolute fitness of an individual bearing a given genotype ij ($\sum_{j=0}^1 \sum_{i=1}^L x_{ij} w_{ij} = 1$).

One round of selection causes the population frequency of genotype ij to change according to $x'_{ij} = x_{ij} w_{ij}$. The absolute fitness (w_{ij}) of conditional helpers (w_{i1}) and defectors (w_{i0}) is given by:

$$w_{i1} = 1 + \frac{-c_{\text{search}}\alpha(1-x_i)(1-\theta) + b(\theta + (1-\theta)x_{i1}) - c(\theta + (1-\theta)x_i)}{1 - \alpha(1-x_i)(1-\theta)} - A, \quad (1)$$

$$w_{i0} = 1 + \frac{-c_{\text{search}}\alpha(1-x_i)(1-\theta) + b(1-\theta)x_{i1}}{1 - \alpha(1-x_i)(1-\theta)} - A. \quad (2)$$

The first terms on the right-hand-side, 1, give the baseline absolute fitness – i.e., the absolute fitness that would arise in the absence of any selective effects.

The second terms give the generational cost of searching for partners. I derived these terms as follows. The probability of abandoning *exactly* one partner in a given social search (generation) is given by $\alpha(1-x_i)(1-\theta)(1-\alpha(1-x_i)(1-\theta))$, which is the per-encounter probability of abandoning a partner ($\alpha(1-x_i)(1-\theta)$), multiplied by the per-encounter probability of not abandoning a partner, bringing the social search to a close ($1-\alpha(1-x_i)(1-\theta)$). Abandoning exactly one partner results in a generational partner search cost of c_{search} . Generalising this argument, the probability of abandoning *exactly* η partners in a given social search (generation) is given by $(\alpha(1-x_i)(1-\theta))^\eta (1-\alpha(1-x_i)(1-\theta))$, which is the per-encounter probability of abandoning a partner, raised to the power η , and multiplied by the probability of not abandoning a partner, bringing the social search to a close. Abandoning exactly η partners results in a generational partner search cost of ηc_{search} . The *expected* generational partner search cost is then given by summing the generational partner search costs of abandoning 1, 2, 3, ..., ∞ partners, where each of these costs are weighted by the respective probabilities of abandoning exactly that many partners. Formally, the expected generational partner search cost is given by the infinite sum: $c_{\text{search}}\alpha(1-x_i)(1-\theta)(1-\alpha(1-x_i)(1-\theta)) \sum_{\eta=1}^{\infty} \eta (\alpha(1-x_i)(1-\theta))^{\eta-1}$, which is a (converging) geometric series, and can be written as $c_{\text{search}}\alpha(1-x_i)(1-\theta)(1-\alpha(1-x_i)(1-\theta)) \left(\frac{1}{(1-\alpha(1-x_i)(1-\theta))^2} \right)$. Simplification gives the cost $\frac{c_{\text{search}}\alpha(1-x_i)(1-\theta)}{1-\alpha(1-x_i)(1-\theta)}$, which corresponds to a net fitness effect of $\frac{-c_{\text{search}}\alpha(1-x_i)(1-\theta)}{1-\alpha(1-x_i)(1-\theta)}$, which features in Equations (1) & (2). I note that the expected generational partner search cost increases monotonically as the searching individual: becomes less related to its social group (lower θ); uses a rarer tag (lower x_i); has a higher probability of abandoning tag-mismatched partners (higher α).

The third terms give the generational benefit of receiving help. For a defector, help can only be received by interacting (as recipient) with a non-clone (genealogical nonrelative) who happens to be a tag-matched helper (i.e., not *identical by descent*, *IBD*). This happens $\frac{(1-\theta)x_{i1}}{1-\alpha(1-x_i)(1-\theta)}$ times per generation (in expectation), which results in an expected generational benefit of $b \frac{(1-\theta)x_{i1}}{1-\alpha(1-x_i)(1-\theta)}$. For a conditional helper, help can be received in this way, but it can alternatively be received by interacting (as recipient) with a clone (genealogical relative), who will (necessarily) be a tag-matched helper (i.e. *IBD*). This happens $\frac{\theta}{1-\alpha(1-x_i)(1-\theta)}$ times per generation (in expectation), which results in an overall expected generational benefit of $b \frac{\theta + (1-\theta)x_{i1}}{1-\alpha(1-x_i)(1-\theta)}$.

The fourth term gives the generational cost of giving help. This is zero for defectors. For conditional helpers, help can be given by: interacting (as actor) with a non-clone (genealogical nonrelative) who happens to be tag-matched (i.e. not *IBD*); or, interacting (as actor) with a clone (genealogical relative), who is *IBD*, so definitely tag-matched. The respective probabilities of these things happening, per generation, are: $\frac{\theta}{1-\alpha(1-x_i)(1-\theta)}$, and $\frac{(1-\theta)x_i}{1-\alpha(1-x_i)(1-\theta)}$, which results in an overall expected generational cost of $c \frac{\theta + (1-\theta)x_i}{1-\alpha(1-x_i)(1-\theta)}$.

The fifth term (A) arises due to competition. Given that population size is constant, any overall (net) fecundity increase in the population due to helping will be exactly offset by an overall (net) loss in fecundity due to competition. In this lifecycle, competition occurs at the level of

the population (i.e., no local, or 'kin', competition), and affects all individuals equally, regardless of their genotype. I can write A explicitly in terms of model parameters as follows: $A = (b - c) \sum_{l=1}^{L_{\max}} \left(x_l p_l \frac{\theta + (1-\theta)x_l}{1 - \alpha(1-x_l)(1-\theta)} \right) - c_{\text{search}} \alpha (1-\theta) \sum_{l=1}^{L_{\max}} \left(\frac{x_l(1-x_l)}{1 - \alpha(1-x_l)(1-\theta)} \right)$.

Using my fitness functions (Equations (1) & (2)), I can also derive 'tag fitness', which I take to be the average fitness of all individuals bearing a given tag i . I denote tag fitness by w_i , and calculate it by taking $w_i = w_{i1}p_i + w_{i0}(1-p_i)$. Tag fitness (w_i) does not feature explicitly in my equation for genotype frequency change due to selection ($x_{ij}' = x_{ij} w_{ij}$), but I derive it here nevertheless, as I will make use of it later:

$$w_i = 1 + \frac{(b - c)p_i(\theta + (1-\theta)x_i) - c_{\text{search}}\alpha(1-x_i)(1-\theta)}{1 - \alpha(1-x_i)(1-\theta)} - A. \quad (3)$$

2.2.11. Recombination

Recombination takes genotype frequencies from x_{ij}' to x_{ij}'' , where x_{ij}'' is written explicitly as follows, and where k denotes the alternative allele to j at the trait locus (i.e. $k = 1$ if $j = 0$; $k = 0$ if $j = 1$):

$$x_{ij}'' = x_{ij}' \left(x_{ij}' + x_{ik}' + (1-r) \sum_{l \neq i}^{L_{\max}} x_{lk}' + \sum_{l \neq i}^{L_{\max}} x_{lj}' \right) + r x_{ik}' \sum_{l \neq i}^{L_{\max}} x_{lj}'. \quad (4)$$

Under random association, the proportion of diploid associations comprised of two ij haploids is given by x_{ij}^2 , and these diploid associations exclusively give rise to ij haploid offspring, meaning the first term in Eq. (4) is x_{ij}^2 .

The proportion of diploid associations comprised of an ij haploid and an ik haploid (same tag; different trait allele) is $2x_{ij}'x_{ik}'$, and half of the haploid progeny from these associations have the ij genotype, regardless of recombination, meaning the second term is $x_{ij}'x_{ik}'$.

The proportion of diploid associations comprised of an ij haploid, and a haploid with a different tag and trait allele, is $2x_{ij}' \sum_{l \neq i}^{L_{\max}} x_{lk}'$. From these associations, the proportion $(1-r)/2$ of haploid progeny have the ij genotype, meaning the third term is $(1-r)x_{ij}' \sum_{l \neq i}^{L_{\max}} x_{lk}'$.

The proportion of diploid associations comprised of an ij haploid, and a haploid with a different trait allele but the same tag, is $2x_{ij}' \sum_{l \neq i}^{L_{\max}} x_{lj}'$. From these associations, half of the haploid progeny have the ij genotype, regardless of recombination, meaning the fourth term is $x_{ij}' \sum_{l \neq i}^{L_{\max}} x_{lj}'$.

Finally, the proportion of diploid associations comprised of an ik haploid, and a haploid with a different tag and different trait allele, is $2x_{ik}' \sum_{l \neq i}^{L_{\max}} x_{lj}'$. From these associations, the proportion $r/2$ of haploid progeny have the ij genotype, meaning the fifth term is $r x_{ik}' \sum_{l \neq i}^{L_{\max}} x_{lj}'$.

2.2.12. Mutation

Mutation takes genotype frequencies from x_{ij}'' to x_{ij}''' , where x_{ij}''' is written explicitly as follows

$$x_{ij}''' = x_{ij}'' (1 - \mu_{\text{Trait}}) + x_{ik}'' \mu_{\text{Trait}}. \quad (5)$$

2.2.13. Dynamically sufficient recursions

Taken together, my equations for selection, recombination and mutation comprise recursions describing how genotype frequencies change over a single generation (from x_{ij} to x_{ij}'''). By iterating these recursions over many generations, I can work out the amount of helping and tag diversity that evolves at evolutionary equilibrium (as I do below in section 2.6).

2.3. Inclusive-fitness predictions

2.3.1. Approach

Before presenting the numerical results of the population genetic

model (section 2.6), I take a step back to ask, when does inclusive fitness theory predict that genetic kin recognition will evolve? Inclusive fitness theory predicts that conditional (tag-based) altruism may only evolve if it leads to a higher inclusive fitness payoff, to the actor, per social interaction, than both indiscriminate (not tag-based) altruism and indiscriminate defection. In this section, I show when this is the case.

I note at the outset, however, that this is a necessary but not a sufficient condition for genetic kin recognition to evolve. If recognising kin is too costly, either because it reduces social interaction rate too much, or because searching for partners is too costly, then kin recognition will not evolve, even if it results in a higher (inclusive fitness) payoff per social interaction than indiscriminate strategies.

This analysis closely follows the inclusive-fitness analysis undertaken in Scott et al. (2023) (e.g., Equations 6–12 also appear there). The analyses are similar because social partners are determined in the same way in both models (see Appendix A in the supplemental material).

2.3.2. $IF_{\text{conditional}}$

First, I calculate the inclusive fitness payoff of conditional (tag-based) altruism, which I denote by $IF_{\text{conditional}}$. The inclusive fitness payoff of an action is calculated by: (1) identifying all individuals (including the actor) affected by the action; (2) weighting each of these individuals according to their genetic relatedness to the actor (similarity at the trait locus); (3) summing the (relatedness-weighted) fitness consequences of the action across all of the affected individuals. The fitness consequences are measured here relative to the non-social case (i.e., the fitness consequences of defecting), following Hamilton (1964).

An act of conditional (tag-based) altruism has consequences for: (1) the altruist (actor); (2) its recipient (i.e. the altruist's ultimate social partner after the possibility of recycling social encounters); (3) competitors (those who suffer fecundity losses as a result of altruism exhibited by the actor). I note here that the altruist (actor) and its recipient are tag-matched – if they weren't tag-matched, there would be no social interaction (no altruism exhibited), and therefore no fitness effect! However, the altruist (actor) and its competitors may be tag-mismatched.

The relatedness between the altruist (actor) and another individual ('affected individual') is given by:

$$R = \frac{\lambda - \bar{p}}{1 - \bar{p}}, \quad (6)$$

where \bar{p} gives the population frequency of the conditional helping allele, and λ gives the probability that the affected individual also has the conditional altruism allele (Frank, 1998; Grafen, 1985; Hamilton, 1970; Queller, 1994, 1992; Scott and Wild, 2023). Eq. (6) is a simplified version of Eq. (7) in Grafen (1985); see Grafen (1985) for its derivation.

I see by plugging in $\lambda = 1$ that the actor is related to clones of itself by 1 (complete genetic similarity). I see by plugging in $\lambda = \bar{p}$ that, given that competition occurs at the population level (i.e., not locally, or between kin), meaning competitors are randomly drawn from the population with respect to the actor, an individual is related to its competitors by zero (unrelated).

To work out the relatedness between the actor (altruist bearing a given tag i) and its recipient, I need to plug in $\lambda = \frac{\theta + (1-\theta)x_i p_i}{\theta + (1-\theta)x_i}$, which is the (per-interaction) probability that the actor's recipient is also an altruist (recall that θ denotes population viscosity; x_i denotes the population frequency of the actor's tag i ; p_i denotes the proportion of individuals bearing the actor's tag i who are helpers). To interpret this expression for λ , note that the denominator gives the per-encounter probability of encountering a tag-matched individual, and the numerator gives the per-encounter probability of encountering a tag-matched altruist. Plugging this into Eq. (6), I obtain the following explicit expression for relatedness between actors and their recipients:

$$R_{tag} = \frac{\theta + (1 - \theta)x_i p_i - \bar{p}}{\theta + (1 - \theta)x_i - \bar{p}} \quad (7)$$

My explicit relatedness expression clarifies that, when an individual is using an exceedingly rare tag to recognise kin ($x_i \rightarrow 0$), tag-matching perfectly identifies kin (who, in this model, are clones), leading to maximal relatedness ($R_{tag} = 1$). When an individual is using a more common tag ($x_i > 0$), tag-matching does not always imply kinship, meaning the tag brings about a reduced relatedness ($R_{tag} < 1$). When an individual is using a maximally common tag (i.e., a tag at fixation; $x_i = 1$), meaning that everyone in the population is using the actor's tag ($\bar{p} = p_i$), tag-matching brings about a relatedness of θ , which is the relatedness that would arise by choosing group members at random (the tag provides no extra information about relatedness). The relationship between relatedness (R_{tag}) and tag frequency (x_i) is plotted in Fig. 3b.

A notable feature of the relatedness expression (Eq. (7)) is that, if helpers are evenly distributed across tags (no linkage disequilibrium), such that $p_i = \bar{p}$, helper frequency (p_i, \bar{p}) 'drops out' of the expression for relatedness. This leads to a relatedness of

$$R_{tag}|_{p_i=\bar{p}} = \frac{\theta}{\theta + (1 - \theta)x_i}, \quad (8)$$

which is invariant (unchanging) with respect to the frequency of the conditional helping allele (p_i, \bar{p}).

Let us assume that each of the L_{max} available tags are held at equal frequency in the population (I relax this assumption later, when solving my population genetic models), and that there is no linkage disequilibrium (i.e. $p_i = \bar{p}$). This means that each tag is at the population frequency $1/L_{max}$. Furthermore, it means that, if there are more available tags (increased L_{max}), each given tag is rarer in the population, resulting in a higher relatedness between altruists and their recipients (R_{tag}). Specifically, if each of the L_{max} tags are maintained at equal frequency, relatedness at equilibrium is given by:

$$R_{tag}|_{p_i=\bar{p}, x_i=1/L_{max}} = \frac{\theta}{\theta + \frac{1 - \theta}{L_{max}}} \quad (9)$$

This would result in an inclusive fitness payoff of conditional (tag-based) altruism, on the assumption that tags have evolved to obtain equal frequencies ($p_i = \bar{p}$) and helper proportions ($x_i = 1/L_{max}$), of $IF_{conditional} = R_{tag}|_{p_i=\bar{p}, x_i=1/L_{max}} b - c$. Using Eq. (9), this can be written explicitly as:

$$IF_{conditional} = \left(\frac{\theta}{\theta + \frac{1 - \theta}{L_{max}}} \right) b - c. \quad (10)$$

2.3.3. $IF_{defection}$

Having derived, under the assumption that tags have obtained equal frequencies and helper proportions, the inclusive fitness payoff of conditional altruism (genetic kin recognition), I can now ask when genetic kin recognition will be favoured over defection. The inclusive fitness payoff of defection is zero, by definition, as it is a non-social trait (Hamilton, 1964). This means that conditional (tag-based) helping will be favoured over defection whenever the following condition is satisfied (obtained by evaluating $IF_{conditional} > IF_{defection}$). Failure to satisfy this condition implies that defection will persist at evolutionary equilibrium:

$$\left(\frac{\theta}{\theta + \frac{1 - \theta}{L_{max}}} \right) b > c. \quad (11)$$

2.3.4. $IF_{indiscriminate}$

To derive the inclusive fitness payoff for indiscriminate altruism ($IF_{indiscriminate}$), I assume that there is one single tag at fixation (no tag diversity; $x_i = 1, \bar{p} = p_i$). I evaluate my relatedness coefficient (Eq. (7)) under this assumption of no tag diversity, which gives $R_{tag}|_{x_i=1} = \theta$. This leads to an inclusive fitness payoff of indiscriminate altruism of:

$$IF_{indiscriminate} = \theta b - c. \quad (12)$$

Conditional (tag-based) helping confers a greater inclusive fitness return than indiscriminate helping whenever $IF_{conditional} > IF_{indiscriminate}$. Using Eqs. (10) & (12), I can evaluate this condition, and I see that it holds whenever there is diversity at the tag locus ($L_{max} > 1$). This is intuitive – by discriminating who it interacts with, an individual is more likely to interact with kin, meaning there is a greater inclusive fitness return from helping. Therefore, as long as individuals are capable of differentiating individuals based on their tag ($L_{max} > 1$), genetic kin recognition will confer a greater inclusive fitness payoff, per social interaction, than indiscriminate helping.

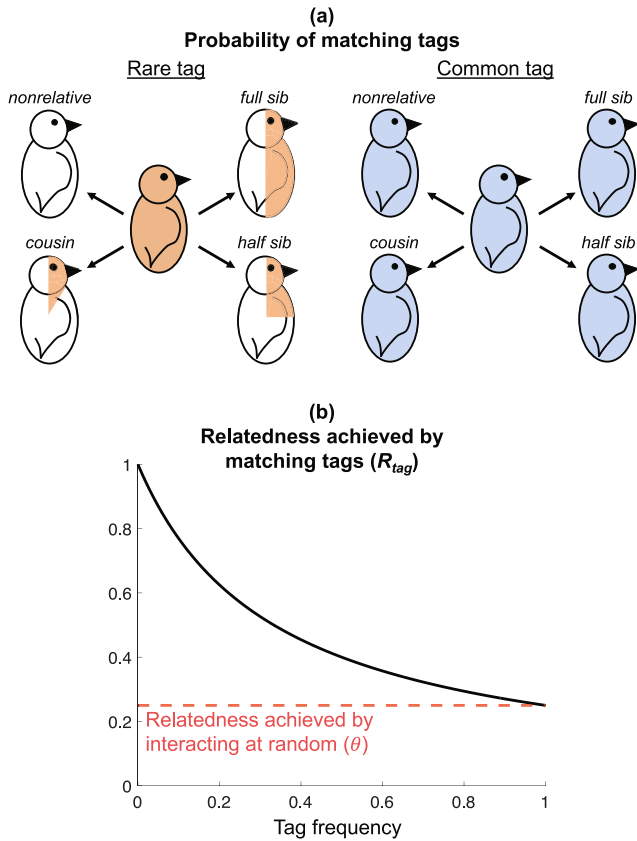


Fig. 3. Relatedness decreases with tag frequency. (A) For birds with rare tags (orange), the probability of matching tags with someone (proportion shaded) is given by pedigree relatedness. For birds with common tags (blue), the probability of matching tags with someone (proportion shaded) is always high, regardless of pedigree relatedness. More common tags are therefore worse indicators of both relatedness at the trait locus and pedigree relatedness. (B) Therefore, the relatedness achieved by matching tags (black solid line) decreases with tag frequency. A tag at fixation achieves the same relatedness as choosing social partners at random (no tag used; red dashed line). I assumed: $\bar{p} = p_i$ (no tag-trait linkage disequilibrium); $\theta = 0.25$ (population viscosity). The full R_{tag} expression is given in Eq. (7). Figure previously published in Scott et al. (2023), Creative Commons (<https://creativecommons.org/licenses/by/3.0/>). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

2.3.5. Predictions based on inclusive fitness theory

This analysis reveals that, if tags have equalised in frequency and helper proportion, genetic kin recognition confers a greater inclusive fitness return, per social interaction, than: (i) indiscriminate defection, whenever the condition in Eq. (11) is satisfied; (ii) indiscriminate helping, whenever there is tag diversity ($L_{\max} > 1$).

Therefore, putting these results together, we can say that genetic kin recognition *could* evolve in regions of parameter space where the condition in Eq. (11) is satisfied and there is tag diversity ($L_{\max} > 1$). However, I emphasise that kin discrimination *could* evolve here; not that it *will* evolve here (it is a necessary rather than sufficient condition).

Specifically, if the costs of recognising kin (partner search cost/reduced social interaction rate) exceed the benefits (increased inclusive fitness payoff from a given social interaction), then tags will not equalise in frequency. Instead, tag diversity will be lost at equilibrium ($L^* = 1$), and an indiscriminate strategy will evolve. If $\theta b < c$ (where θ is the relatedness that would arise by choosing group members at random), this strategy will be indiscriminate defection; if $\theta b > c$, this strategy will be indiscriminate helping.

I have expressed these predictions, based on inclusive fitness theory, with minimal reference to genetic details. We will see that my prediction – that genetic kin recognition evolves whenever Eq. (11) holds, tags are available ($L_{\max} > 1$), and there is a low cost of kin recognition – is borne out by my explicit population genetic analyses (provided there is some trait mutation, $\mu_{\text{Trait}} > 0$). However, this prediction is high-level and somewhat vague, in that I haven't examined the causal effects of model parameters such as partner search (α), recombination (r), trait mutation (μ_{Trait}), and so on, and I haven't examined how evolution proceeds at the level of loci (tag & trait). For this, we need explicit genetic analyses, and I come onto this next.

2.4. Constructing the simple model

2.4.1. Crozier's paradox and Grafen's linkage disequilibrium

Having undertaken a high-level inclusive fitness analysis, I now explicitly consider the genetic details of my model. Before presenting the numerical results of my full complex model (section 2.2), here I make some additional assumptions, which will allow me to construct and analyse a simplified version of the model. This will be illuminating in itself, and helpful when finally turning to the complex model analysis in section 2.6.

Crozier argued that genetic kin recognition will often be evolutionarily unstable (Crozier, 1986). His argument was that more common tags are more likely to be recognised and helped than rare tags, generating positive frequency dependence at the tag locus, and resulting in a loss of tag diversity, destabilising kin recognition.

Grafen disputed Crozier's claim, and argued that coevolution between helping and kin recognition could allow genetic kin recognition to evolve (Grafen, 1990). Specifically, Grafen's (1990) argument was that, as tags become more common, they will become less useful cues of relatedness, and so can be invaded by non-cooperative cheats. Rare tags will be good indicators of relatedness, preventing invasion by cheats (Fig. 3). Technically, a statistical association between genes for helping and rare tags will build up (linkage disequilibrium). This leads to individuals with rare tags having greater payoffs from social interactions. Grafen (1990) argued that this advantage for rare tags (reduced risk of being cheated) could exceed Crozier's advantage for common tags (increased chance of being recognised and helped), meaning rare tags gain an overall advantage, maintaining tag diversity.

However, as shown by Rousset and Roze (2007), Grafen's linkage disequilibrium can be broken down by direct selection at the tag locus (for increased social interaction rate) and recombination. Here, I examine a hypothetical world where linkage disequilibrium is not broken down, meaning Grafen's linkage disequilibrium builds up rapidly. I ask when genetic kin recognition evolves in this scenario. This analysis will give a hypothetical best-case-scenario for genetic kin

recognition. I analyse this idealised 'simple model' first, as it will help us when trying to understand the full 'complex model' (described in section 2.2 above), where linkage disequilibrium can be broken down.

Grafen's linkage disequilibrium will be maximal (not broken down) if: (i) evolution at the tag locus proceeds very slowly relative to evolution at the trait locus (separation of timescales), and (ii) recombination is absent ($r = 0$). My separation of timescales assumption breaks an assumption of my full complex model – a strict adherence to my stated lifecycle assumptions (section 2.2) would imply that evolution at the tag and trait loci proceed at a comparable rate. I therefore note that the purpose of the simple model analysis is not to deduce, in any realistic biological scenario, when genetic kin recognition will evolve (I will come to that in section 2.6). It is simply to deduce what would happen in a hypothetical best-case scenario favouring genetic kin recognition.

In this simple model, I also assume for simplicity, except where stated, that the population is monomorphic. By this, I mean that each tag segregating in the population has equal frequency and equal helper proportion. I denote the monomorphic tag frequency by x (i.e., I drop the i/l subscript), and note that $x = \frac{1}{L}$, where L gives the number of tags segregating in the monomorphic population.

2.4.2. Roadmap for simple model construction

In this simple model, a tag's helper proportion (proportion of individuals bearing a tag who are helpers as opposed to cheaters, p) evolves much more rapidly than its population frequency, which means that, at (effectively) any point in time, a tag's helper proportion will be unchanging, having reached a steady state. Given this, I will consider a tag at a given population frequency x , and take the following methodological steps:

- i. Write a recursion to describe how the tag's helper proportion (p) changes over time (as it evolves rapidly towards its steady-state value).
- ii. Solve this recursion, to find the tag's rapidly-obtained helper proportion, which I denote by p_{SoT} (the *SoT* is short for 'Separation of Timescales').
- iii. Use this expression for helper proportion (p_{SoT}) to calculate the fitness of a tag (w_{SoT}) whose helper proportion has evolved to p_{SoT} .
- iv. Use this tag fitness function (w_{SoT}) to model tag evolution, to find the equilibrium tag frequency, which I denote by x_{Target} , and the corresponding equilibrium helper proportion, which I denote by p_{Target} ($p_{\text{Target}} = p_{\text{SoT}}|_{x=x_{\text{Target}}}$).

This approach allows me to see how many tags are maintained at equilibrium ($L^* \approx \frac{1}{x_{\text{Target}}}$), and how much helping there is at equilibrium (p_{Target}), in a broadly analytical framework.

2.4.3. Terminology

As mentioned, I refer to the (long term) equilibrium helper proportion & tag frequency as the 'target' helper proportion & tag frequency (p_{Target} & x_{Target}). I use this terminology to emphasise that, when linkage disequilibrium is not broken down, as in the present simple model, helper proportion & tag frequency evolve to this end-point, but when linkage disequilibrium *can* be broken down (as in the complex model analysis; section 2.6), this helper proportion & tag frequency may be transiently approached, but not necessarily obtained.

2.4.4. Helper proportion recursion

Technically, here I am assuming that, each generation, each tag changes in population frequency by a negligible amount (it is held approximately constant). Conversely, a tag's helper proportion (p_i) changes as normal (appreciably each generation), according to the following recursion, where w_{i1} and w_{i0} denote the respective expected fitness of: helpers bearing a given tag i ; defectors bearing a given tag i .

These fitness functions are given in Eqs. (1) and (2) respectively (derived in section 2.2). A tag's helper proportion changes according to:

$$p'_i = (1 - \mu_{\text{Trait}})p_i w_{i1} + \mu_{\text{Trait}}(1 - p_i)w_{i0}. \quad (13)$$

To see the logic of this recursion, note that the first term of the right-hand-side is comprised of: the helper proportion in the first time-step (p_i); weighted by the likelihood that the helpers do not mutate into defectors ($1 - \mu_{\text{Trait}}$); weighted by the fitness of helpers using tag i (number of adult offspring). The second term of the right-hand-side is comprised of: the cheat load in the first time-step ($1 - p_i$); weighted by the likelihood that the cheaters mutate into helpers (μ_{Trait}); weighted by the fitness of cheaters using tag i (number of adult offspring). The sum of these two terms gives the helper proportion in the next time-step (p'_i ; left-hand-side). Cheat load in the next time step is given by $1 - p'_i$.

2.4.5. Target helper proportion

As stated above, I assume a monomorphic population, such that tag frequencies, and helper proportions, are equal across all tags. This allows me to evaluate w_{i1} and w_{i0} (Eqs. (1) & (2)) for the case where all tag frequencies are $x = 1/L$ and helper proportions are all p_{SoT} . I can then solve Eq. (13) by substituting in my evaluated fitness functions, setting $p'_i = p_i = p_{\text{SoT}}$, and simplifying. This gives me an exact expression for the helper proportion that tags converge quickly on in my simple model:

$$p_{\text{SoT}} = \left(\frac{-b\theta + c\theta + 2\mu_{\text{Trait}}(1 - \alpha + (\alpha + b - c)\theta) + (c + 2\alpha\mu_{\text{Trait}} - 2c\mu_{\text{Trait}})(1 - \theta)x - ((b\theta - c\theta - c(1 - \theta)x)^2 - 4\mu_{\text{Trait}}(b\theta - c\theta - c(1 - \theta)x)^2 + 4\mu_{\text{Trait}}^2(1 + \alpha^2(1 - \theta)^2(1 - x)^2 + ((b - c)\theta - c(1 - \theta)x)^2 - 2\alpha(1 - \theta - x + \theta x))^{1/2}}{(2(1 - 2\mu_{\text{Trait}})(-b\theta + c\theta + c(1 - \theta)x))} \right)^{\frac{1}{2}} \quad (14)$$

Eq. (14) is too long to be easily understood by inspecting the algebra – I therefore plot the expression in Fig. 4, and explain the behaviour of this expression in the next paragraphs. The cheat load that tags converge quickly on is given by $1 - p_{\text{SoT}}$.

Rarer tags have greater helper proportions (Fig. 4). This trend arises because rarer tags lead to greater relatedness in social interactions (Fig. 3a). Specifically, helper proportion (p_{SoT}) decreases as a sigmoidal (S-shaped) function of tag frequency (x). To understand the specific form of this function, note that, when $R_{\text{tag}} = c/b$ (which, in terms of model parameters, is equivalent to: $x = \frac{(b-c)\theta}{c(1-\theta)}$), cheaters and helpers using a given tag will have equal fitness (no selection at the trait locus). At this point, trait mutation (μ_{Trait}) will cause cheaters and helpers to equalise in frequency, leading to a helper proportion of $p_{\text{SoT}} = 0.5$. This is the *inflection point* of the curve (x satisfies $R_{\text{tag}} = c/b$; $p_{\text{SoT}} = 0.5$).

For tag frequencies (x) below this inflection point, helpers are positively selected, and therefore over-represented ($p_{\text{SoT}} > 0.5$). For tag frequencies (x) above this inflection point, helpers are negatively selected, and therefore under-represented ($p_{\text{SoT}} < 0.5$). For more extreme tag frequencies (x approaching 0 or 1), and for reduced trait mutation (μ_{Trait}) relative to the magnitude of social payoffs (b, c), the strength of selection is increased relative to mutation, and as a result, deviations from the neutral helper proportion are exaggerated (p_{SoT} approaches 0 or 1).

Increased population viscosity (θ) leads to increased relatedness (R_{tag} increased over all x), and therefore to increased helper proportion (p_{SoT}). Specifically, for increased population viscosity, helpers are positively selected over a greater range of tag frequencies, meaning tag frequency need not be as low for helpers to be selected. Another way of putting this is to say that an increase in population viscosity (θ) pushes the inflection

point of the curve to the right (Fig. 4). For very high population viscosity, such that $\theta > c/b$ is satisfied (meaning helping is favoured amongst individuals drawn randomly from a social group, irrespective of tag identity), cheaters are *never* positively selected. This means that the inflection point is undefined (it veers off to the right of the x-axis).

Partner search (α) does not affect relatedness (R_{tag} ; Eq. (7)), and as a result, it does not qualitatively affect the shape of the helper proportion (p_{SoT}) curve (e.g. the inflection point is unaffected). Partner search (α) may affect the level of (global) competition, which may in turn affect the strength of trait selection relative to trait mutation, which may lead to a slight quantitative change in the helper proportion (p_{SoT}) curve (not shown), but this is negligible.

2.4.6. Tag fitness

Having calculated how helper proportion (p_{SoT}) varies with tag frequency (x), I can now calculate tag fitness (w_{SoT}) as a function of tag frequency (x). The intention here would then be to use the tag fitness function (w_{SoT}) to deduce the evolutionary end point of tag evolution. Specifically, the evolved tag frequency (x_{target}) would correspond to the x value that optimises the tag fitness function (i.e., the x value that leads to the highest w_{SoT}).

Our first task is deciding on an appropriate fitness measure. My intention is to choose a fitness measure such that, at equilibrium, fitness

is maximised. We quickly run into difficulties if we try to use *number of adult offspring* (absolute fitness) as the fitness measure. The reason is that, to calculate absolute fitness, we need to know the distribution of genotypes in the population. This is because the A term in my absolute fitness formula (Eq. (3)), capturing the effect of competition, depends on the distribution of genotypes in the population. In populations that produce a lot of juveniles, the A term (capturing competition) is larger. The upshot of this is that we cannot easily use absolute fitness to infer the end point of evolution at the tag locus, because the fitness function is in effect moving as evolution is occurring (Rousset, 2004). It is difficult to calculate what tag frequency (x) maximises a given function if the function itself is changing!

Fortunately, we can simplify things, by choosing a fitness function that is invariant with respect to the population genotype distribution, but still accurately predicts the end-point of tag evolution. I use *number of juvenile offspring* as my measure of fitness (henceforth: $\text{fitness}_{\text{juv}}$), which omits the effect of competition (the A term of Eq. (3) is dropped), meaning it is unaffected by the population genotype distribution. I am justified in choosing a fitness measure ($\text{fitness}_{\text{juv}}$) that ignores competition because, owing to my assumption of global population regulation (bland density dependence), competition, despite affecting the *strength* of tag selection, does not influence the *direction* of selection (doesn't affect whether an individual, with a given genotype, is more or less fit than another individual, with a different genotype), and therefore doesn't change the tag frequency for which fitness is maximised.

The tag $\text{fitness}_{\text{juv}}$ function, for my simple model, is then given by:

$$w_{\text{SoT}} = 1 + \frac{(b - c)p_{\text{SoT}}(\theta + (1 - \theta)x) - c_{\text{search}}\alpha(1 - x)(1 - \theta)}{\psi(1 - \alpha(1 - x)(1 - \theta))}, \quad (15)$$

where p_{SoT} is given in Eq. (14), and where ψ is a very large, arbitrary constant. ψ captures my separation of timescales assumption that tag

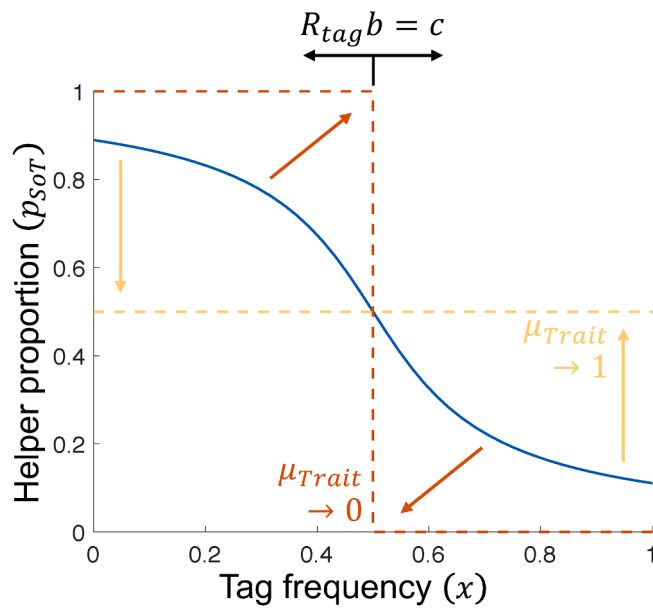


Fig. 4. Helper proportion in the simple model. The proportion of individuals bearing a tag who are helpers ('helper proportion', p_{SoT}) is plotted against the frequency of the tag (x). Helper proportion decreases with tag frequency, with an inflection point occurring when x satisfies $R_{tag} = c/b$ (in terms of model parameters: $x = \frac{(b-c)\theta}{c(1-\theta)}$) and $p_{SoT} = 0.5$. When trait mutation is absent ($\mu_{Trait} = 0$), helper proportion is given by a step function (red dashed line); when trait mutation is maximal ($\mu_{Trait} = 1$), helper proportion is 0.5 for all x ; (yellow dashed line); when trait mutation is intermediate ($0 < \mu_{Trait} < 1$), helper proportion is intermediate between these two extremes, and given by a smooth curve (blue line). The blue line in this figure was generated by substituting the following parameter values into Eq. (14): $b = 0.03, c = 0.01, \theta = 0.2, \mu_{Trait} = 0.005, \alpha = 0$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

evolution proceeds very slowly compared to trait evolution. Importantly, ψ has no effect on the outcome of tag evolution (i.e., it doesn't change the tag frequency for which fitness is maximised). I plot this function (w_{SoT}) in Fig. 5.

The equilibrium tag frequency, which I denote by x_{Target} , is then given by the value of x associated with the highest value of w_{SoT} . I cannot obtain an exact (algebraic) value for x_{Target} ; conversely, I can only obtain x_{Target} using numerical methods (which assume specific parameter values). Having obtained x_{Target} , I can calculate the equilibrium amount of helping (frequency of the conditional helping allele, p_{Target}) by substituting x_{Target} into my expression for helper proportion (Eq. (14)). Some example x_{Target} values are given in Fig. 5 – they are the x values for which the fitness functions (curves) are maximised (peaks of the curves). I interpret these results in the next section (section 2.5).

Before I move on, I emphasise that this analysis rests on the assumption that the population evolves to the 'fitness peak', at which each individual is using a tag at the fitness-maximising population frequency. Similar 'optimisation' assumptions are often made in evolutionary theory (Eshel, 1996; Parker and Smith, 1990; Scott and Wild, 2023; Taylor and Frank, 1996). However, in many models, including multi-locus population genetic ones, this optimisation assumption has been broken, with populations failing to climb the fitness peak (Charlesworth, 1994; Feldman et al., 1996; Kawecki, 1993; Rousset, 2004). It is therefore important to note that I have verified my optimisation assumption with dynamical simulations of my simple model. Specifically, I found that, when one tag starts at near-fixation, and the other available tags start at rarity (corresponding to negligible initial tag diversity), and when trait evolution occurs rapidly compared to tag evolution, the population generally evolves so that tag frequencies cluster around the $fitness_{juv}$ -maximising tag frequency (x_{Target}).

There are a few exceptions to this. Specifically, in some cases, dynamical simulations reveal that the population does not evolve to maximise my fitness function (Eq. (15)). That is, in some cases, tags don't evolve to the population frequency at the 'peak' of the fitness function (Eq. (15)). In sections 2.5.3 and 2.5.4, I discuss when and why this happens, and what the outcome of evolution is in these cases, if not $fitness_{juv}$ maximisation.

2.5. Analysing the simple model

2.5.1. Introduction

Having derived the number of tags and the amount of helping at equilibrium ($L^* \approx \frac{1}{x_{Target}}, p_{Target}$), I will address the following specific questions

- When there are no genetic constraints on tag availability ($L_{max} \rightarrow \infty$), when does genetic kin recognition evolve?
- When there are genetic constraints (finite L_{max}), when does genetic kin recognition evolve?

Key results are labelled sequentially (*Simple Model Result 1, 2, etc.*). Results derived for this simple model should not be taken as fact – they need to be verified by the full complex model analysis, and may only hold in certain areas of the complex model's parameter space. Later, I will clarify when the results of the simple model hold, and why they sometimes do not hold (section 2.6). In fact – to reveal a spoiler – we will see that the results of the simple model are recovered in the complex model when partner search is cheap and abundant (high α ; low c_{search}).

2.5.2. Optimisation of tag fitness

The first key result is that, if tag availability is unconstrained, meaning tags are freely available (specifically, if $L_{max} > 1/x_{Target}$), approximately $1/x_{Target}$ tags are maintained at equilibrium (I explain the meaning of 'approximately' in section 2.5.3). Therefore, if the target tag frequency (x_{Target}) is sufficiently low, more than one tag will be maintained at equilibrium (tag diversity), meaning kin recognition evolves.

Simple Model Result 1

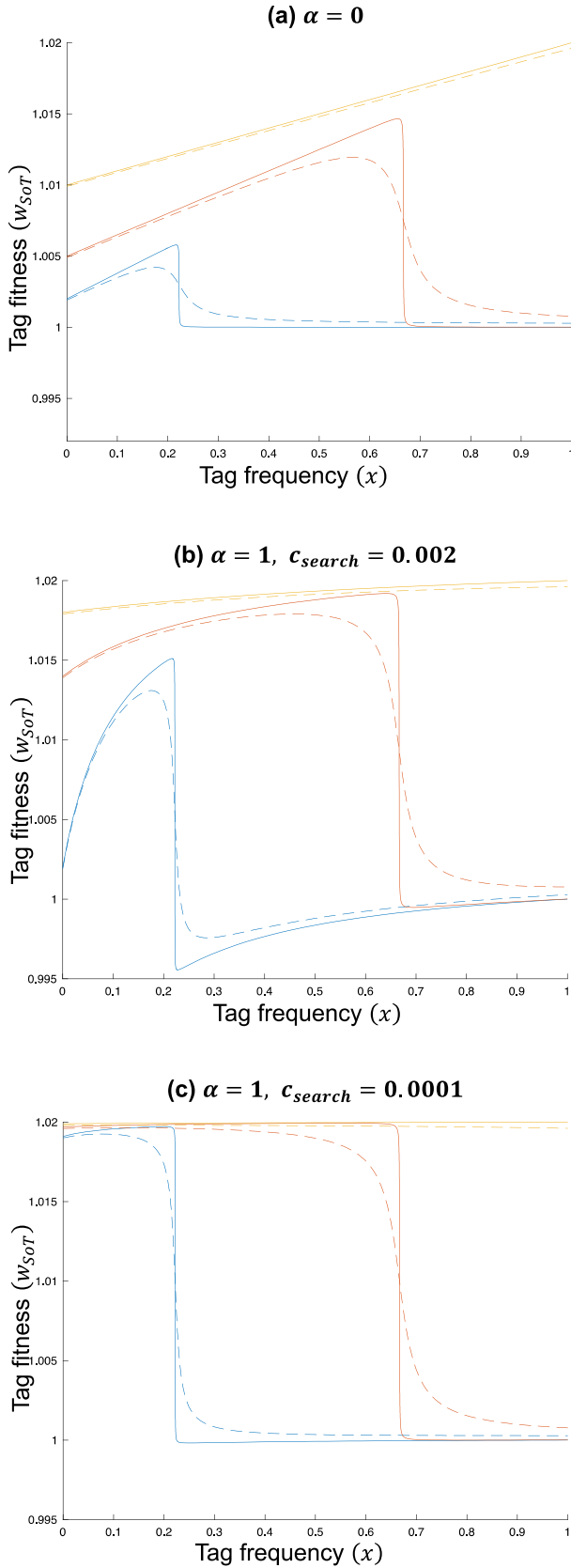
If tag availability is unconstrained ($L_{max} > 1/x_{Target}$), approximately $1/x_{Target}$ tags are maintained at equilibrium ($L^* \approx 1/x_{Target}$), each at a population frequency of approximately x_{Target} .

The inflection point of my helper proportion function (p_{SoT} ; Fig. 4) occurs at the tag frequency at which $R_{tag} = c/b$ (in terms of model parameters: $x = \frac{(b-c)\theta}{c(1-\theta)}$). This is the threshold tag frequency, below which, helpers are positively selected, and above which, cheaters are positively selected. I find that my tag fitness function (w_{SoT} ; Fig. 5) has an inflection point in the same position (the x for which $R_{tag} = c/b$). For reasonable parameterisations of my model (sufficiently low c_{search}), the optimum tag frequency (x_{Target}) occurs at ($\mu_{Trait} = 0$) or just below ($\mu_{Trait} > 0$) this inflection point. This makes sense – the optimum tag frequency is the highest frequency that can be obtained (maximising interaction rate/minimising partner search cost) without triggering cheater selection and spread.

An exception to this general result is if partner-search is widespread and extremely costly (high α & c_{search}). In this case, kin recognition is too costly to be favoured, meaning the optimum tag frequency (x_{Target}) occurs at $x_{Target} = 1$ (i.e., above the inflection point at $R_{tag} = c/b$). This means tag diversity is lost at equilibrium, and cheat load is high (because the inflection point at $R_{tag} = c/b$ has been passed) – this is *indiscriminate defection*.

Simple Model Result 2

Tag fitness is maximised at the population frequency at ($\mu_{Trait} = 0$) or just



(caption on next column)

Fig. 5. Tag fitness in the simple model. The fitness of a tag (w_{SoT} ; Eq. (15)), whose helper proportion has converged on p_{SoT} , is plotted against the frequency of the tag (x), for 18 different parameter regimes (18 curves). The peak of each curve gives the tag frequency that evolves at equilibrium in the simple model (x_{Target}).

Blue, red and yellow curves correspond, respectively, to low ($\theta = 0.1$), intermediate ($\theta = 0.25$) and high ($\theta = 0.5$) population viscosity. Solid and dashed curves correspond, respectively, to negligible ($\mu_{Trait} = 0.000001$) and higher ($\mu_{Trait} = 0.0001$) trait mutation. Panels a, b and c correspond, respectively, to: no partner search ($\alpha = 0$); costly partner search ($\alpha = 1, c_{search} = 0.002$) and cheap partner search ($\alpha = 1, c_{search} = 0.0001$). Each curve additionally assumes that $b = 0.03, c = 0.01$.

The inflection point of each curve occurs when x satisfies $R_{tag} = c/b$ (in terms of model parameters: $x = \frac{(b-c)\theta}{c(1-\theta)}$). Increased population viscosity (θ) pushes the inflection point to the right (the inflection point is undefined if $R_{tag} > c/b$ for all x , as is the case for the yellow lines).

Tag fitness (w_{SoT} ; Eq. (15)) is generally maximised (x_{Target}) either at the inflection point ($\mu_{Trait} = 0$; solid lines) or slightly to the left of it ($\mu_{Trait} > 0$; dashed lines). Tag fitness may be maximised at $x_{Target} = 0$ (e.g., red dashed line in panel c), in which case, for arbitrarily high tag availability (L_{max}), all available tags evolve to be as rare as possible, meaning all of them are maintained at equilibrium ($L^* = L_{max}$).

below ($\mu_{Trait} > 0$) the point at which cheaters spread (i.e. x_{Target} is positioned at or just below the inflection point of p_{SoT} , at which $R_{tag} = c/b$). Exception: very high α & c_{search} means kin recognition is exceedingly costly, resulting in $x_{Target} = 1$ & defection.

An increased population viscosity (θ) means that cheaters are less likely to spread (inflection point of p_{SoT} is pushed to the right). This increases the optimum tag frequency (x_{Target}), reducing the number of tags that are maintained at equilibrium ($L^* \approx 1/x_{Target}$), and therefore reducing the discriminating-power (precision) of kin recognition. This result makes intuitive sense, given that, for increased population viscosity, individuals are more likely to encounter their kin by chance (proximity), meaning kin recognition is increasingly redundant, and less worth paying costs for. As a result, the individual uses a higher-frequency tag, which means that kin recognition is less precise, but the individual pays fewer of the associated costs of kin recognition (reduced interaction rate; increased costly partner-search).

Simple Model Result 3

If population viscosity is increased (θ), tag fitness is maximised at a greater population frequency (higher x_{Target}), meaning kin recognition evolves to be less precise (fewer tags maintained).

If trait mutation (μ_{Trait}) is increased relative to the magnitude of social payoffs (b, c), cheaters are less effectively purged, meaning the helper proportions (p_{SoT}) of low-frequency tags are increased. This means there is more chance of being cheated, which increases the incentive for recognising kin, meaning the optimum tag frequency is reduced (reduced x_{Target}). Therefore, increased trait mutation leads to more tags being maintained at equilibrium, facilitating more precise kin recognition.

Simple Model Result 4

If trait mutation (μ_{Trait}) is increased relative to the magnitude of social payoffs (b, c), tag fitness is maximised at lower population frequency (x_{Target} is pushed further to the left of the inflection point of p_{SoT} , at which $R_{tag} = c/b$).

An implication of Simple Model Result 3 & 4 is that, if population viscosity (θ) is low enough, and trait mutation (μ_{Trait}) is high enough relative to trait selection (b, c), then tag fitness will be maximised at $x_{Target} = 0$. This means that each tag will evolve to become rarer and rarer, allowing all available tags to be maintained ($L^* = L_{max}$), regardless

of how many tags are available (i.e., for arbitrarily high L_{\max}). Biologically, this result means that open-ended tag diversification (persistent negative frequency dependence at the tag locus), and highly precise kin recognition, may be favoured in very mixed populations (low θ).

Simple Model Result 5

When population viscosity is low enough ($\theta \rightarrow 0$), and there is sufficient trait mutation (μ_{Trait}) relative to trait selection (b, c), tag fitness is maximised at $x_{\text{Target}} = 0$, leading to open-ended tag diversification & highly precise kin recognition.

If population viscosity (θ) is very high, such that $\theta > c/b$ is satisfied (helping favoured amongst individuals drawn randomly from a group), cheaters are *never*, for any tag frequency (x), positively selected. This means the inflection point of p_{SoT} is pushed to the right of the x axis (undefined). In this case, if there is sufficient trait mutation (μ_{Trait}) relative to trait selection (c, b), x_{Target} may still be below 1 (Simple Model Result 4), meaning multiple tags may persist at equilibrium (kin discrimination). Alternatively, if there is insufficient trait mutation (μ_{Trait}) relative to trait selection (c, b), meaning cheaters are purged more effectively, the incentive for recognising kin is reduced, and we see that $x_{\text{Target}} = 1$, meaning indiscriminate helping evolves (e.g. yellow lines in Fig. 5).

Simple Model Result 6

When population viscosity is high enough that $\theta > c/b$ (such that helping is favoured amongst individuals drawn randomly from a group), tag fitness is generally maximised at $x_{\text{Target}} = 1$, leading to indiscriminate helping. Exception: high μ_{Trait} relative to c & b may lead to $x_{\text{Target}} < 1$ (kin discrimination).

2.5.3. Constraints on optimisation

Tags may fail to reach the optimum population frequency (x_{Target}), for three reasons (three sources of deviation from fitness-maximisation, or strictly, three sources of deviation from maximisation of the w_{SoT} function with respect to x). Two out of these three reasons can be thought of as *constraints on optimisation*, in which all tags in the population approach the fitness peak, but fail to reach it exactly. They are discussed here (the third source of deviation from fitness-maximisation is discussed below in section 2.5.4).

The first reason is that tags are discrete, rather than continuous, in nature. This means that, for a given optimum tag frequency (x_{Target}), there can't literally be $1/x_{\text{Target}}$ segregating tags at equilibrium (unless $1/x_{\text{Target}}$ happens to be an integer). Rather, there could either be $\lceil 1/x_{\text{Target}} \rceil$ ($1/x_{\text{Target}}$ rounded up) tags, each at a lower-than-optimal frequency ($x < x_{\text{Target}}$), or $\lfloor 1/x_{\text{Target}} \rfloor$ ($1/x_{\text{Target}}$ rounded down) tags, each at a greater-than-optimal frequency ($x > x_{\text{Target}}$). Given that tag fitness drops sharply for frequencies above the optimum (cheaters selected), tag fitness is greater just below, rather than just above, the optimum frequency (x_{Target}). As a result, we see that $\lceil 1/x_{\text{Target}} \rceil$ ($1/x_{\text{Target}}$ rounded up) tags are maintained at equilibrium, each at a lower-than-optimal frequency ($x < x_{\text{Target}}$).

Therefore, owing to the discrete nature of tags, the evolved phenotype may be slightly suboptimal. However, the deviation from optimality will be in the direction of more precise kin recognition (more tags being maintained than are favoured). This constraint on optimality therefore (slightly) facilitates the evolution of genetic kin recognition. I note, however, that when precise kin recognition is favoured (low x_{Target}), this deviation from optimality will be negligible (the deviation, given by $x_{\text{Target}} - 1/\lceil 1/x_{\text{Target}} \rceil$, decreases to zero as x_{Target} decreases to zero).

The second reason why tags may fail to reach the optimum tag frequency (x_{Target}) is that tag availability may be limited. If more tags are favoured than are available ($L_{\max} < \lceil 1/x_{\text{Target}} \rceil$), the evolved tag frequency will lie to the right of the optimum ($x^* > x_{\text{Target}}$). This means that,

relative to when tag availability is unconstrained, fewer tags will be maintained at equilibrium ($L^* < \lceil 1/x_{\text{Target}} \rceil$), and, by nature of being more common, the tags that are maintained will have higher cheat loads.

Simple Model Result 7

If tag availability is sufficiently low that $L_{\max} < \lceil 1/x_{\text{Target}} \rceil$, the amount of helping (p_{SoT}) and the precision of kin discrimination (L^*) are reduced at equilibrium.

2.5.4. Disruptive selection

I find, with dynamical simulations of my simple model, that when partner-search is abundant and very costly (high α ; very high c_{search}), tags may diverge in frequency at equilibrium. The result is that that some individuals exhibit kin discrimination (conditional helping) at equilibrium, and others exhibit indiscriminate defection. I will explain why tag divergence occurs. Before doing so, I will have to make a further assumption about tag fitness.

If tags are divergent in frequency, the fitness of any given tag (Eq. (15)) is technically undefined, because Eq. (15) was derived under the assumption that the population is monomorphic (i.e., that each tag has the same frequency). Therefore, for the purpose of explaining why tag frequency divergence occurs, I will assume that the relationship between tag fitness and tag frequency (shape of the fitness function) is qualitatively the same, in a population where tag frequencies have diverged, as when the population is monomorphic (Eq. (15)). This assumption should be reasonable, given that the distribution of genotypes in the population, including whether tag frequencies are divergent or not, only matters for tag fitness in that it *slightly* affects competition (the A term in Eq. (3)). Nevertheless, if the reader is unsatisfied with this justification, they may wish to disregard the following paragraphs, in which an evolutionary explanation for disruptive selection on tag frequency is given.

Whether tag diversification occurs depends on what happens to tag fitness (w_{SoT}) as tag frequency (x) increases past the optimum (x_{Target}). One possibility is that, after the sharp drop in tag fitness just after x_{Target} due to cheater selection, tag fitness continues to decrease with tag frequency ($\frac{dw_{\text{SoT}}}{dx} < 0$ for all x such that $x > x_{\text{Target}}$). In this case, for tag frequencies above the optimum ($x > x_{\text{Target}}$), rarer tags will always be fitter than more-common tags, leading to selection for tag rarity, and therefore to tag equalisation. In this scenario, each of the L tags will evolve to the same frequency ($1/L$), which is above the optimum ($1/L > x_{\text{Target}}$) – no tag diversification occurs.

A second possibility is that, after the sharp drop in tag fitness just after x_{Target} due to cheater selection, tag fitness starts to increase slightly as tag frequency increases. This can occur if the cost of partner search (c_{search}) is very large, meaning common tags (who engage in less partner search) gain an advantage despite having increased cheat load (e.g., the blue dashed line in Fig. 5b). In this case, for tag frequencies above the optimum ($x > x_{\text{Target}}$), common tags may be fitter than rarer tags, causing the common tags to increase further in frequency, pushing the rare tags to lower frequency. This may push the rarer tags back to the top of the fitness peak (x_{Target}), leading to an evolutionary end-point comprising rarer tags, engaging in discriminating helping (kin discrimination), and more common tags, suffering greater cheat loads, and therefore engaging in less discriminating and less cooperative social behaviours (i.e. defection).

However, this second scenario, leading to disruptive tag selection, arises under more restrictive conditions – it requires very high α and c_{search} . In general, tags do not diverge in frequency.

Simple Model Result 8

Tags in general do not diverge in frequency (monomorphism). Exception: in restrictive scenarios, involving high α & c_{search} , tags may diverge in frequency, with some supporting helping and others supporting defection.

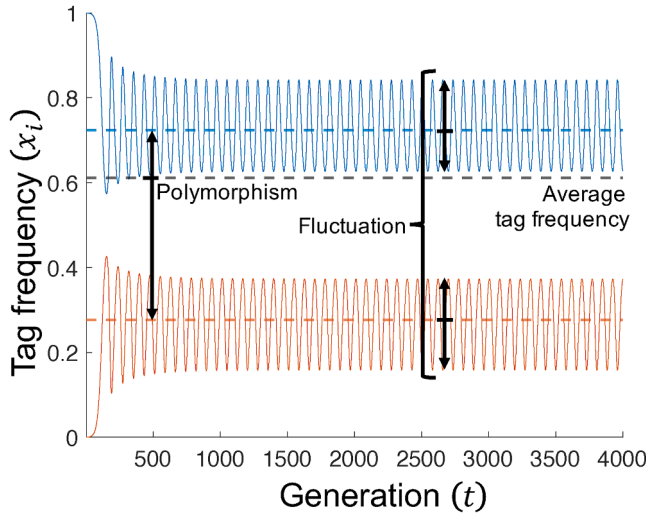


Fig. 6. Summary statistics. An example run of the full population genetic model is plotted, in which two tags persist at equilibrium. The average-over-time frequencies of the two tags are plotted as blue and red dashed lines (Equation B.1 in Appendix B). The average-over-time-and-over-tags tag frequency is plotted as the grey dashed line (Equation B.2), and the inverse of this value gives the equilibrium number of segregating tags (L^*). The root-mean-square deviation of tag frequencies from their average-over-time frequency (fluctuation; $fluc^*$; Equation B.3), and the root-mean-square deviation of tag frequencies from the average-over-time-and-over-tags frequency (polymorphism; $poly^*$; Equation B.4), are also illustrated. Exact numerical values for the equilibrium summary statistics are: $fluc^* = 0.0762$, $poly^* = 0.2001$, $L^* = 1.6358$. This plot was generated using the following parameter values: $r = 0.26$, $\theta = 0.22$, $L_{max} = 2$, $\alpha = 0$, $\mu_{trait} = 0.005$, $b = 3$, $c = 1$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

2.6. Analysing the complex model

2.6.1. Roadmap for complex model analysis

In this section, I present the results of my full complex model (outlined in section 2.2), in which tag and trait evolution proceed at comparable rates. I obtained these results by, for each run, numerically iterating my genotype frequency recursions (Eqs. (1), (2), (4), (5)), from a start point where kin discrimination is negligible, until an equilibrium is reached. I recorded the following four summary statistics for each run: (i) number of segregating tags (L^*); (ii) the extent to which tags fluctuate (/oscillate) in frequency ($fluc^*$); (iii) the extent to which different tags are polymorphic (divergent in frequency) ($poly^*$); (iv) frequency of the conditional helping allele ($coop^*$) (Fig. 6).

I generated results for a range of parameter combinations and plotted this in Supplementary Fig. 1 (r is varied across y-axes; θ is varied across x-axes; α , c_{search} , μ_{trait} , c , b , L_{max} are varied across panels a–e). The reader may wish to consult the legend of Supplementary Fig. 1 for a guide on how to navigate the wealth of numerical data. A heavily simplified summary figure of the main features of Supplementary Fig. 1 is given in Fig. 7. See Appendix B in the supplemental material for more details about how the results were generated, and for mathematical definitions of the summary statistics.

My broad aims in this section are to use this numerical data to examine: (i) how genotype frequencies change as they approach equilibrium; (ii) whether, and when, kin discrimination is found at equilibrium (multiple tags + conditional helping). This section builds on my previous (simple model) analysis, by: (i) examining whether, and when, the simple model results hold; (ii) deriving new results, which were not observable from my simple model, which abstracted-away certain aspects of tag-trait coevolution. More specifically, I will examine the following questions, labelling key results sequentially as I go (Complex

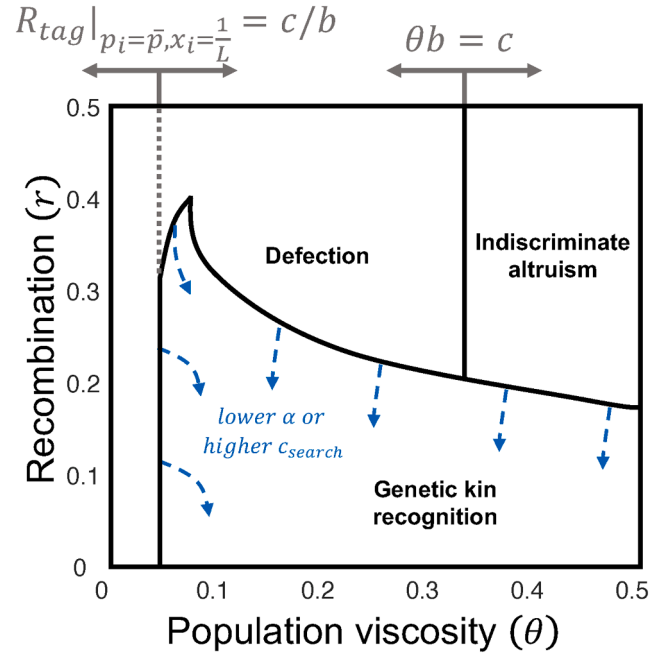


Fig. 7. Summary of complex model results. When population viscosity low, such that $R_{tag|p_i=\bar{p}, x_i=\frac{1}{L}}b < c$, defection evolves. An increase in the number of available tags (L_{max}) reduces the parameter space where $R_{tag|p_i=\bar{p}, x_i=\frac{1}{L}} < c/b$ (the grey dotted line moves to the left), disfavouring defection. When population viscosity is intermediate, such that $R_{tag|p_i=\bar{p}, x_i=\frac{1}{L}} > \frac{c}{b} > \theta$: defection evolves if kin recognition is too costly (due to reduced social interaction rate, α , or costly partner-search, c_{search}), or recombination is too high (breaking down Grafen's linkage disequilibrium); genetic kin recognition evolves otherwise. When population viscosity is high, such that $\theta b > c$: indiscriminate altruism evolves if kin recognition is too costly, or recombination is too high; kin discrimination evolves otherwise. The blue dashed arrows and the blue text ('lower α or higher c_{search} ') illustrate that, for increased costs of kin recognition, the upper border of the region where genetic kin recognition is favoured moves down, meaning the area diminishes. In this figure, the 'genetic kin recognition' label corresponds to cases where all 10 available tags are maintained at equal frequency and helping is approximately at fixation (mutation-selection balance) ($L^* = L_{max}$; $coop^* \approx 1$). This plot was generated from the full population genetic (complex) model using the parameter values: $\alpha = 1$, $b = 0.3$, $c = 0.1$, $c_{search} = 0.0001$, $\mu_{trait} = 0.0005$, $L_{max} = 10$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Model Result 1, 2, etc.):

- i. How does tag and trait evolution proceed? (Section 2.6.2.)
- ii. When are the simple model results recovered? (Section 2.6.3.)
- iii. When there is widespread partner search (high α), when does genetic kin recognition evolve? (Section 2.6.4.)
- iv. When there is limited partner search (low α), when does genetic kin recognition evolve? (Section 2.6.5.)
- v. What happens in the evolutionary short-term when the 'genetic kin recognition equilibrium' is perturbed? (Section 2.6.6.)
- vi. What happens in the evolutionary long-term when the 'genetic kin recognition equilibrium' is perturbed? Specifically, do genotype frequencies cycle stably around the equilibrium, or collapse back into the equilibrium? (Appendix C.)
- vii. When do tags diverge in frequency at equilibrium, with some supporting higher levels of helping, and others supporting lower levels of helping (polymorphism)? (Section 2.6.7.)

Before proceeding to answer these questions, I note that the high-level results of the inclusive-fitness analysis (section 2.3) are

recovered in the full complex model (provided there is some trait mutation, $\mu_{\text{Trait}} > 0$). To re-cap the results of the inclusive-fitness analysis, I found that $R_{\text{tag}}|_{p_i=\bar{p}, x_i=\frac{1}{L}} > c/b$ is a necessary (but not sufficient) condition for genetic kin recognition to evolve. Satisfaction of this condition implies that kin discrimination returns a higher payoff per social interaction than defection. Failure to satisfy this condition means that defection evolves. The condition is not a sufficient condition for genetic kin recognition to evolve, though, because even if kin discrimination returns a higher payoff per social interaction than indiscriminate strategies, kin discrimination may still be disfavoured overall if it incurs other costs, such as a reduced social interaction rate or an increased cost of searching for social partners. If $R_{\text{tag}}|_{p_i=\bar{p}, x_i=\frac{1}{L}} > c/b$ is satisfied, but the costs of kin recognition are too high for it to be favoured overall, an indiscriminate strategy will evolve instead. This strategy will be (indiscriminate) defection if $\theta b < c$, and indiscriminate helping if $\theta b > c$. These results, first obtained in the inclusive-fitness analysis (section 2.3), are illustrated for the full model in Fig. 7.

2.6.2. Direct and indirect tag selection

First, I examine how tag and trait evolution proceed. I find that, whenever tags are at unequal population frequencies, any tags that are above the population-average frequency ($x_i > 1/L$) gain an immediate direct fitness advantage due to: reduced partner search ($\alpha > 0$), or increased social interaction rate ($\alpha < 1$), or both ($0 < \alpha < 1$). These common tags ($x > 1/L$) also begin to accumulate cheaters (build-up of linkage disequilibrium), leading to an increasing indirect fitness cost, which may eventually surpass the direct fitness advantage, causing the common tags to fall in frequency.

Analogously, any tags that are below the population-average frequency ($x < 1/L$) incur an immediate direct fitness disadvantage due to: increased partner search ($\alpha > 0$), or reduced social interaction rate ($\alpha < 1$), or both ($0 < \alpha < 1$). These rare tags also begin to accumulate helpers (build-up of linkage disequilibrium), leading to an increasing indirect fitness benefit, which may eventually surpass the direct fitness disadvantage, causing the rare tags to increase in frequency.

Direct selection at the tag locus responds *instantaneously* to any given change (perturbation) in tag frequency (*first order effect*). Indirect selection at the tag locus does not respond instantaneously to a given change (perturbation) in tag frequency, because trait evolution, leading to the build-up of linkage disequilibrium, takes time (*second order effect*).

2.6.3. When are simple model results recovered?

As generations pass, tags change in both their population frequencies (x_i) and their helper proportions (p_i), owing to direct and indirect tag selection. A given tag's helper proportion (p_i) is constantly being pulled towards p_{SoT} , derived in my simple model analysis (Fig. 4), which is the end-point of trait evolution in a hypothetical scenario where the tag's frequency (x_i) is constant ($p_i \rightarrow p_{\text{SoT}}|_{x_i}$). However, a given tag's frequency (x_i) is constantly changing, which means the 'end-point' of tag evolution (p_{SoT}) is constantly moving. This means, given that trait evolution is not instantaneous, a given tag will rarely obtain a helper proportion of $p_{\text{SoT}}|_{x_i}$ exactly. Furthermore, recombination (r) serves to redistribute helpers and defectors evenly across different tags. Therefore, the actual helper proportion obtained by a tag at a given point in time (p_i) is likely to be intermediate between the average helper proportion, \bar{p} (no linkage disequilibrium), and the simple model helper proportion, $p_{\text{SoT}}|_{x_i}$ (maximal linkage disequilibrium).

To infer how strong selection for linkage disequilibrium is, I can consult my tag fitness function, w_{SoT} (Eq. (15)). The more stretched this function (w_{SoT}) is in the y-axis, the greater the fitness consequences of linkage disequilibrium, and therefore, the stronger the selective pull of tags towards their simple model helper proportions (p_{SoT}) and target population frequencies (x_{Target}). This selection for linkage disequilibrium is counteracted by the breakdown of linkage disequilibrium by

recombination (r).

I find that, if the fitness consequences of linkage disequilibrium are great enough (w_{SoT} is y-stretched enough) relative to recombination (r), then tags acquire helper proportions that are close enough to p_{SoT} that tag selection proceeds in qualitatively the same way as it did in the previous simple model analysis. Specifically, tag frequencies approach x_{Target} and helper proportions approach p_{Target} (given by $p_{\text{SoT}}|_{x_{\text{Target}}}$). As a result, the equilibrium derived in my simple model ($x_i = x_{\text{Target}}, p_i = p_{\text{Target}}$) is obtained approximately, with the caveat that, sometimes, genotype frequencies cycle stably around the equilibrium instead of resting at it stably (see Appendix C in the supplemental material for a population genetic explanation of cycling).

Conversely, if the fitness consequences of linkage disequilibrium are insufficient (w_{SoT} is insufficiently y-stretched) relative to recombination (r), then direct selection (for common tags) is increased relative to indirect selection (for rare tags), meaning tag diversity is reduced or lost at equilibrium ($x_i > x_{\text{Target}}$).

2.6.4. Widespread partner search

I now ask, for what areas of parameter space do strong fitness consequences of linkage disequilibrium (y-stretched w_{SoT}) arise, leading to appreciable linkage disequilibrium build-up and recovery of the simple model equilibrium ($x_i = x_{\text{Target}}, p_i = p_{\text{Target}}$)? One such area of parameter space corresponds to: prevalent partner search (high α), where partner search cost (c_{search}) and population viscosity (θ) are not excessively high. The blue and red curves in panels b and c of Fig. 5 are examples of this. We see that these curves are spread over a large range of fitness values (y-stretched), meaning there is strong selection for linkage disequilibrium. We see that a reduction in the prevalence of partner search (α ; Fig. 5 panel a), or an excessively high population viscosity (θ ; Fig. 5 yellow lines), result in the function becoming less y-stretched, meaning linkage disequilibrium is less strongly selected.

For sufficiently high α , & low c_{search} & low θ , linkage disequilibrium is built up sufficiently strongly that the simple model equilibrium is approached, regardless of the recombination rate (i.e., even under free recombination, $r = 0.5$). I also find that, for high partner search (α), genotypes either settle exactly on the equilibrium ($\alpha = 1$), or only cycle around it slightly (α near to 1), meaning the simple model equilibrium is closely approximated.

Complex Model Result 1

When partner search is sufficiently prevalent and cheap (high α ; low c_{search}), and the population is sufficiently mixed (low θ), genotype frequencies approximately obtain the simple model equilibrium, with little or no oscillation ($x_i = x_{\text{Target}}, p_i = p_{\text{Target}}$).

As a result, when partner search is sufficiently prevalent and cheap (high α and low c_{search}), and the population is sufficiently mixed (low θ), genetic kin recognition can easily evolve (i.e., whenever $x_{\text{Target}} < 1$), and support high (approximately maximal) levels of discriminating helping (p_{Target} is very high when $x_{\text{Target}} < 1$; Simple Model Result 2). This is subject to the caveats and qualifications detailed in Simple Model Results 1–8.

Before moving on, I re-iterate some important results regarding trait mutation (μ_{Trait}), which were obtained in the simple model analysis and are recovered here. Trait mutation (μ_{Trait}) reduces x_{Target} , and therefore facilitates genetic kin recognition (Simple Model Result 4) when partner search is prevalent and cheap (high α and low c_{search}). For instance, compare Supplementary Fig. 1b & d, which are identically parameterised except for having different values of μ_{Trait} . The reason, put simply, is that, for increased trait mutation (μ_{Trait}), cheaters are less effectively purged (selection is weaker relative to mutation), even when in linkage disequilibrium with common tags. This increases the risk of

being cheated, which increases the incentive for restricting social interactions to kin (kin recognition).

A further implication of this effect of trait mutation (μ_{Trait}) is that, when trait mutation is absent ($\mu_{\text{Trait}} = 0$), cheaters are completely purged at equilibrium whenever $\theta b > c$ (no equilibrium cheater risk). This means that, when $\mu_{\text{Trait}} = 0$, kin discrimination never evolves in the region of parameter space given by $\theta b > c$; instead, when $\mu_{\text{Trait}} = 0$, indiscriminate helping always evolves in this region ($\theta b > c$). For kin discrimination to evolve in this region ($\theta b > c$), there needs to be some trait mutation ($\mu_{\text{Trait}} > 0$) (Simple Model Result 6).

2.6.5. Limited partner search

The (indirect) fitness advantage for rare tags, relative to common tags, arises because rare tags are less likely to be cheated in social interactions, meaning they have a higher expected payoff per social interaction. This advantage for rare tags is ‘cached in’ every social interaction. However, when partner search is limited (i.e., when $\alpha < 1$), individuals bearing rare tags have reduced rates of social interaction relative to individuals bearing common tags, meaning they have fewer opportunities to ‘cash in’ on this indirect fitness advantage. As a result, for reduced partner search (α), there is an increased ‘interaction’ between indirect and direct tag fitness, which disproportionately hurts rarer tags. This has three notable consequences.

Firstly, for reduced partner search (α), the disproportionate fitness cost for rarer tags means that any overall fitness advantage for rare tags (occurring when $x_{\text{Target}} < 1$) is likely to be reduced (w_{SoT} is likely to become less y-stretched; Fig. 5), meaning linkage disequilibrium builds up more slowly, and tag diversity is more likely to be lost (simple model equilibrium is less likely to be obtained). A rare exception to this is when there is a very high cost of partner search (c_{search}), which means that, for reduced partner search (α), rare tags may conversely gain an increased overall fitness advantage (w_{SoT} may become more y-stretched).

Secondly, for reduced partner search (α), the fitness of rarer tags is reduced more appreciably than less rare tags. Therefore, when partner search (α) is low: (i) if very rare tags are favoured (x_{Target} is very low), their selective advantage over other tags is likely to be small; (ii) if less rare tags are favoured (x_{Target} is not as low), their selective advantage over other tags is likely to be larger (not as small). Therefore, when partner search (α) is low: if x_{Target} is decreased (i.e., if more tags, $[1/x_{\text{Target}}]$, are favoured), linkage disequilibrium builds up more slowly, meaning tag diversity is more likely to be lost (simple model equilibrium is less likely to be reached). The result is that, when partner search (α) is low, the more tags that are favoured by selection ($[1/x_{\text{Target}}]$), the weaker selection will be, reducing the likelihood that (any) tag diversity is maintained. This means that, for reduced partner search (α), the number of tags that can be sustained by selection (precision of kin discrimination) is reduced.

Thirdly, when partner search (α) is low, whenever the simple model equilibrium is approached ($x_i \rightarrow x_{\text{Target}}$, $p_i \rightarrow p_{\text{Target}}$), genotype frequencies do not rest at it stably; instead, they fluctuate/cycle around it appreciably (see Appendix C in the supplemental material for a population genetic explanation of cycling; see also that $\text{Fluc}^* > 0$ in Supplementary Fig. 1ciii). This means that tag frequencies are constantly (albeit transiently) pushed above the threshold at which cheaters are selected ($x_i > x_{\text{Target}}$). This increases the average cheat load relative to when there is no cycling – cheaters are generally at negligible frequency (mutation-selection balance) when there is no cycling (Simple Model Result 2). Genotype frequency cycling also introduces temporal and spatial heterogeneity in helper proportion (p_i) – that is, tags have helper proportions that are different from each other, and which change (go up and down) over time.

Putting these three consequences together, a reduced partner search (α) means that genetic kin recognition: (i) is less likely to evolve; (ii) is less likely to be precise (i.e., it is likely to be underpinned by fewer tags); (iii) supports a lower amount of helping. In the extreme, when partner

search is absent ($\alpha = 0$), I find that the simple model equilibrium is only approached when recombination (r) is very low and the optimum tag frequency (x_{Target}) is reasonably high, and this only results in a small number of tags being maintained and relatively low helping (Supplementary Fig. 1c). This recovers the results of previous theoretical studies of genetic kin recognition that didn’t allow partner search (Jansen and van Baalen, 2006; Rousset and Roze, 2007).

Complex Model Result 2

When partner search is limited (low α), tag diversity is generally lost, with the following exception. Exception: when the optimum tag frequency (x_{Target}) is relatively high, and recombination is very low ($r \approx 0$), genotype frequencies approach the simple model equilibrium, but oscillate appreciably around it, meaning only a limited amount of discriminating helping is supported.

2.6.6. Short-term dynamics near equilibrium

In this section, I make further points about the nature of the ‘genetic kin recognition equilibrium’, where multiple tags are segregating alongside appreciable frequencies of the conditional helping allele ($x_i \rightarrow x_{\text{Target}}$, $p_i \rightarrow p_{\text{Target}}$). Specifically, I examine how perturbing tag frequency (x_i) away from this equilibrium (x_{Target}) affects fitness, and what affect this has on short-term genotype frequency changes near the equilibrium.

Here, I work with number of juvenile offspring as my measure of fitness ($\text{fitness}_{\text{juv}}$), rather than absolute fitness (number of adult offspring). This simplified fitness measure ($\text{fitness}_{\text{juv}}$) ignores competition, which is acceptable because my assumption of global population regulation means that competition has no effect on the direction of selection (section 2.4.6).

Tag $\text{fitness}_{\text{juv}}$ is given by $w_i + A$. This is obtained by: taking the right-hand-side of Eq. (3), and adding A (see section 2.2 above). The instantaneous change in $\text{fitness}_{\text{juv}}$ ($w_i + A$) for a given change in tag frequency (x_i) is given by $\frac{d(w_i + A)}{dx_i}$, which can be expressed using the chain rule as $\frac{d(w_i + A)}{dx_i} = \frac{\partial(w_i + A)}{\partial p_i} \frac{dp_i}{dx_i} + \frac{\partial(w_i + A)}{\partial x_i} \frac{dx_i}{dx_i}$. For a given change in tag frequency, helper proportion does not change instantaneously (it will only change after a lag), meaning $\frac{dp_i}{dx_i} = 0$. Substituting this, and the trivial result $\frac{dx_i}{dx_i} = 1$, into my expression for $\frac{d(w_i + A)}{dx_i}$, I obtain $\frac{d(w_i + A)}{dx_i} = \frac{\partial(w_i + A)}{\partial x_i}$. This shows that the instantaneous change in $\text{fitness}_{\text{juv}}$, with a given change in tag frequency (x_i), is given exclusively by direct selection at the tag locus ($\frac{\partial(w_i + A)}{\partial x_i}$), which is a first-order effect.

Evaluating $\frac{\partial(w_i + A)}{\partial x_i}$, I obtain:

$$\frac{d(w_i + A)}{dx_i} = \frac{(1 - \theta)((1 - \alpha)(b - c)p + \alpha c_{\text{search}})}{(\alpha(1 - \theta)(1 - x_i) - 1)^2}. \quad (16)$$

We see by inspection that this is always positive (for $\theta < 1$, i.e., non-clonal interaction groups). This means that: (i) any increase in tag frequency (positive perturbations from equilibrium) will result in increased tag $\text{fitness}_{\text{juv}}$ ($w_i + A$), meaning tag frequency increases further in the short term; (ii) any decrease in tag frequency (negative perturbations from equilibrium) will result in reduced tag $\text{fitness}_{\text{juv}}$ ($w_i + A$), meaning tag frequency decreases further in the short term. In other words, perturbations are transiently reinforced.

Complex Model Result 3

Even when multiple tags persist over time at equilibrium, they will not necessarily be at the population frequency $1/L$, because perturbations in tag frequency will be reinforced in the short-term, and only overturned in the longer-term.

In Appendix C in the supplemental material, I track the long-term fate of perturbations from the ‘genetic kin recognition equilibrium’ (the short-term fate of perturbations was described in this section).

Specifically, in Appendix C, I focus on when perturbations: (i) diminish over time, with genotype frequencies settling back on the equilibrium; (ii) persist over time, in the form of stable genotype frequency fluctuations/oscillations. I note that Appendix C is only likely to be of technical interest, hence its relegation to the supplemental material. Specifically, it explains why tag frequency fluctuations/oscillations arise. The biological implication of tag frequency oscillations was given previously (section 2.6.5).

2.6.7. Disruptive selection

I found in the simple model that, when partner-search is abundant and very costly (high α & c_{search}), tags may diverge in frequency at equilibrium (*Simple Model Result 8*). The reason was that very high partner search costs are disproportionately paid by rarer tags, which can: (i) generate a 'local fitness peak' at high tag frequency, in addition to the 'global fitness peak' that arises at low tag frequency due to low cheat load; (ii) reduce the fitness of limitingly rare tags below common tags, preventing them from invading, meaning some common tags cannot be 'reigned in' from the local to the global fitness peak, leading to tag frequency polymorphism (section 2.5.4). I recover this result in my full analysis. For instance, $\text{poly}^* = 0$ in [Supplementary Fig. 1a](#) but $\text{poly}^* > 0$ in [Supplementary Fig. 1b](#), where the figures are identically parameterised except that the former has no cost of partner search, but the latter does.

I find that tag divergence can evolve for less costly partner search (lower α or c_{search}) in the complex model relative to the simple model, as long as there is high recombination (though not so high that tag diversity is lost completely). For instance, $\text{poly}^* > 0$ in [Supplementary Fig. 1d](#), e and b for sufficiently high recombination, even though partner search is not extremely costly. The reason why recombination (in combination with partner search cost) facilitates tag divergence is that recombination distributes helpers more evenly across tags, increasing the fitness of common tags relative to rarer tags, making it less likely that common tags are 'reigned in' from the local to the global fitness peak, leading to tag frequency polymorphism.

Additionally, I find that tag divergence can evolve in the complex model simply as a consequence of tags fluctuating in frequency (see Appendix C in the supplemental material for an explanation of tag frequency fluctuation). The reason for this is that, when tags fluctuate around the 'global fitness peak' (x_{Target}), this generally causes the fluctuating tags to differ in their average-over-time population frequencies, leading to tag frequency polymorphism, except in the special case where each tag happens to fall into the same 'orbit' around the equilibrium. For instance, $\text{poly}^* > 0$ in [Supplementary Fig. 1c](#) for relatively low recombination, owing to $\text{fluc}^* > 0$.

An implication of these results on disruptive selection is that there may exist, in natural populations, a range of co-existing social strategies. In other words, populations may sometimes comprise mixtures of: 'risk-averse discriminators', who interact less often or engage in more partner-search, but are rarely cheated; and 'risk-tolerant non-discriminators', who interact more often or engage in less partner-search, but are often cheated.

Complex Model Result 4

Appreciable recombination (r), alongside appreciable kin recognition costs (low α or high c_{search}), can result in disruptive tag selection, with some individuals engaging in high levels of discriminating helping, and others engaging in low levels.

3. Discussion

I found, in a full ('complex') multi-locus model of genetic kin recognition, that when individuals can search for multiple social encounters at low cost (high α , low c_{search}), this can lead to strong selection for helping amongst individuals with rare tags, and strong selection for defecting amongst individuals with common tags (i.e., strong selection

for *linkage disequilibrium*). This causes tags to evolve analogously to a 'simple model' where linkage disequilibrium is assumed to be maximal (not broken down). Specifically, tags tend to evolve towards the highest frequency that they can attain (increasing the social interaction rate) without triggering the spread of non-cooperative cheats, maximising their 'tag fitness' (Eq. (15)), and justifying an interpretation of tags as adaptive optimisers of their own population frequency (Ågren and Patten, 2022; Gardner and Welch, 2011). This can support a type of kin discrimination that is highly precise (good at picking out genealogical relatives) and cooperative. Conversely, I found that, if individuals cannot search for multiple social encounters at a low cost, but there is low recombination (r) and population viscosity (θ), kin discrimination can still evolve, but it is relatively imprecise (bad at picking out genealogical relatives) and less cooperative (characterised by relatively more non-cooperative cheating).

Recent theory on Crozier's paradox has found that genetic kin recognition can be stabilised in a variety of ways: (i) multiple social encounters; (ii) tag-trait physical linkage (beard chromodynamics); (iii) host-parasite coevolution. However, multiple social encounters may provide the most robust resolution of Crozier's paradox, for two reasons. Firstly, it is the only resolution that works without restrictive assumptions about genetic architecture and parasite evolution (Scott et al., 2023, 2022). Secondly, I found that the type of kin discrimination may be different, depending on how Crozier's paradox is resolved. Specifically, multiple social encounters can lead to kin discrimination that is underpinned by a large number of non-oscillating tags and a high helper frequency. The large number of tags means that individuals that share tags are highly likely to be genealogical relatives, leading to highly precise kin recognition. The high helper frequency means that kin discrimination is highly cooperative. The lack of tag oscillation means that kin discrimination is prevalent across space and time (homogeneously distributed). This is the type of kin discrimination that we observe in animals such as cooperative breeders (Charpentier et al., 2007; Green et al., 2015; Leedale et al., 2020b; Lihoreau and Rivault, 2009; McDonald and Wright, 2011).

In contrast, tag-trait physical linkage tends to lead to a type of kin discrimination that is underpinned by a relatively small number of oscillating tags and a low helper frequency, meaning it is relatively imprecise, non-cooperative and heterogeneously distributed. Additionally, I showed elsewhere that host-parasite coevolution may lead to similarly imprecise, non-cooperative and heterogeneously distributed kin recognition, as a consequence of parasite-induced selection causing tags to fluctuate wildly in frequency ('Red Queen dynamics') (Scott et al., 2023). This is not the kind of behaviour that we typically think of as 'true' kin discrimination, nor do we commonly observe it in nature (Penn and Frommen, 2010; Strassmann et al., 2011). A direction for future work is examining when individuals evolve to use environmental rather than genetic cues for recognising kin (e.g., sharing a nest), and whether this is affected by how Crozier's paradox was resolved (Bourke, 2014; Cornwallis et al., 2009; Leedale et al., 2020a; Sharp et al., 2005; Scott et al., 2022).

More generally, my methodological approach was to compare two different types of model: unrealistically simple, and complex. In one model I made unrealistic simplifying assumptions to produce a more tractable and understandable model. I then compared this to a more complex multi-locus population genetic model where I had not made the same simplifying assumptions. The benefits of this combined methodological approach are that it: (i) renders the topic of genetic kin recognition more accessible to non-mathematicians; (ii) explains genetic kin recognition in multiple complementary ways, in terms of evolutionary forces acting on populations (population genetics), as well as the fitness optimisation of tags and individuals (adaptation); (iii) shows that previous theoretical results still hold under different assumptions, implying that they reflect biological principles rather than mathematical artefacts (Axelrod et al., 2004; Jansen and van Baalen, 2006; Rousset and Roze, 2007; Scott et al., 2022).

CRediT authorship contribution statement

Thomas W. Scott: Conceptualization, Formal analysis, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jtbi.2024.111735>.

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