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Evolutionary biology

Evolution of behavioural resistance in host–pathogen systems

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Behavioural resistance to parasites is widespread in animals, yet little is known about the evolutionary dynamics that have shaped these strategies. We show that theory developed for the evolution of physiological parasite resistance can only be applied to behavioural resistance under limited circumstances. We find that accounting explicitly for the behavioural processes, including the detectability of infected individuals, leads to novel dynamics that are strongly dependent on the nature of the costs and benefits of social interactions. As with physiological resistance, evolutionary dynamics of behavioural resistance can also lead to mixed strategies that balance these costs and benefits.

1. Introduction

Hosts resist parasites using diverse mechanisms, with broad implications for host–parasite coevolution [1–4]. Previous theoretical models of resistance evolution have largely focused on physiological or biochemical resistance [1,5–7]. Yet resistance against parasites can also take the form of behavioural traits [3,8,9] such as direct avoidance or 'disgust' in response to diseased individuals [10,11] or general avoidance of interactions with other individuals, which in a human context is now termed 'social distancing' [12]. Ecologists have long recognized that social behaviours can both facilitate and prevent transmission [13–15]. Here, we ask whether the ecological and evolutionary dynamics of behavioural defenses against parasites operate according to similar principles as physiological defenses.

Host behaviour is implicit in classical models of microparasite transmission as a component of the parameter β, the transmission coefficient [16,17]. β is a composite of multiple factors [18], including the contact rate between hosts, which is a function of host behaviour, and the per-contact transmission probability, which is a function of pathogen infectiousness and host physiology [19]. Models of resistance evolution typically vary the physiological resistance of the susceptible host [5,20]. Nevertheless, variation in avoidance of infected conspecifics exists across and within species: e.g. crustaceans [21], birds [22,23] and primates [24], including humans [10]. Despite the broad diversity of these behaviours [8,25,26], behavioural resistance has rarely been examined explicitly in theoretical contexts [27,28]. It remains unknown whether evolutionary dynamics of behavioural resistance follow the same patterns as physiological resistance. Previous theoretical research on physiological resistance has shown that susceptible and resistant individuals can coexist in the presence of a disease when resistance carries a direct physiological cost [5,7,29], but in social species, lost interactions with others as a function of avoiding disease could constitute a social cost. Models have not yet considered how such costs might influence resistance evolution.

Here, we develop a theoretical model of a disease transmitted in a social context, through direct contact or aerosol, and investigate the evolution of behavioural resistance under several assumptions about behavioural processes and cost–benefit trade-offs. We show with this heuristic model that behavioural

resistance can result in evolutionary dynamics that differ from physiological resistance, depending on the specificity of behavioural responses to diseased conspecifics and the nature of the costs and benefits of sociality.

2. The model

We model social behaviour in a population of individuals that enter into groups or remain singletons. Let S be the number of singletons and G the number of groups of size T. Thus, the total population size in a given time-step is the sum of singletons and individuals in groups, $N = S + TG$. (We provide full derivations of subsequent equations in electronic supplementary material, information S1.) We assume group formation occurs rapidly, reaching equilibrium within each time-step, prior to transmission, birth, and death. Once groups are formed, disease transmission is only possible within groups. We assume a large population and deterministic dynamics.

(a) Model structure

(i) Group formation

The frequency of groups depends on the group encounter rate, ρ, and group dissociation rate, υ. We model the simplest case: pair formation ($T = 2$). Pair formation has been studied in the context of mating and marriage and represents a complex problem of sampling without replacement [30,31]. Following previous work [32], we considered two forms of encounter. First, singletons could encounter one another at a constant frequency, independent of their density, as would occur when individuals seek others out to form associations. Second, singletons could encounter others randomly, such that encounters occur at a higher rate at greater densities. These two types of group formation have parallels with frequency-dependent and density-dependent disease transmission processes [32]. Given that the two types of encounter gave qualitatively similar results, in the main text, we present only the frequency-dependent case (density-dependent results are in electronic supplementary material, information S1). The differential equations for the number of groups and singletons are

$$\frac{dG}{dt} = \rho S - \upsilon G \tag{2.1}$$

and

$$\frac{dS}{dt} = T(\upsilon G - \rho S). \tag{2.2}$$

Within a time-step, when pairs form, the total population size (N) is fixed. At equilibrium, the ratio of groups to singletons, $G/S = \rho/\upsilon$. Converting to a frequency, the equilibrium number of groups is

$$G = \left(\frac{\rho/\upsilon}{1 + \rho/\upsilon}\right)\left(\frac{N}{T}\right). \tag{2.3}$$

(ii) Behavioural resistance

We compare two types of behavioural resistance: specific avoidance of diseased individuals and general avoidance of all associations. For specific avoidance, a healthy individual can detect and avoid pairing only with infected individuals by a factor ϕ. For general avoidance, a healthy individual encounters all others at a reduced rate ($\rho - a$).

(iii) Resistance costs

Physiological resistance is usually assumed to carry some cost that results in reduced fitness in the absence of the parasite [5,29]. We assume behavioural resistance can have two types of cost. Costs of avoidance may be fixed, in that they are incurred regardless of whether avoidance is carried out; for example, a less active genotype could have fewer social encounters, but also reduced feeding. The cost reduces births by c relative to the birth rate of non-avoiding individuals, b. Alternatively, sociality could be beneficial, such that costs of avoidance may only be instantiated when the individual avoids being in a group. We examine the case in which reproduction increases additively with the frequency at which each type pairs (see electronic supplementary material, information S1: SE19–SE20).

(b) Model implementation

(i) Dynamics with no evolution

We first examine how the equilibrium frequency of individuals in pairs and disease dynamics vary across a range of general and specific avoidance parameters (ϕ and a) when all individuals avoid disease. We derive how R_0 depends on the equilibrium frequency of pairs.

(ii) Evolution of behavioural resistance

To understand the evolution of behavioural resistance, we use the one-locus, two allele dynamical framework developed for physiological resistance evolution [5]. In this system, X_1 and X_2 represent two haploid genotypes that differ in their resistance, with X_2 avoiding disease. X_1 and X_2 are equivalent in their transmission once infected and are pooled into one class of diseased individuals, Y. We assume that X_1 and X_2 are the only genetic variants for behavioural resistance. We also assume that once an individual is diseased, it no longer avoids others. If we assume instead that individuals retain their avoidance once infected, it can be shown that the results are identical for frequency-dependent pair formation, whereas for general avoidance, the boundaries of the polymorphism region are slightly different under this assumption, though the results are qualitatively equivalent (electronic supplementary material, information S1: figure S3).

Transmission occurs at rate δ from infected (Y) individuals to X_1 or X_2 when they are in a pair. We assume the disease is sterilizing but does not influence mortality, i.e. diseased individuals do not reproduce. We impose density dependence on the birth rate of healthy individuals because without a numerical (i.e. ecological) feedback, the system does not reach stable equilibrium [29]. We represent background mortality as μ. These processes are represented by

$$\frac{dX_1}{dt} = X_1(b - kN - \mu) - \delta\left(\frac{2GX_1Y}{N^2}\right), \tag{2.4}$$

$$\frac{dX_2}{dt} = X_2((b - c) - kN - \mu) - \delta\left(\frac{2GX_2Y}{N^2}\right) \tag{2.5}$$

and

$$\frac{dY}{dt} = \delta\left(\frac{2GY}{N^2}\right)(X_1 + X_2) - \mu Y. \tag{2.6}$$

The process of pair formation is nested within each time-step, such that N does not change during pair formation

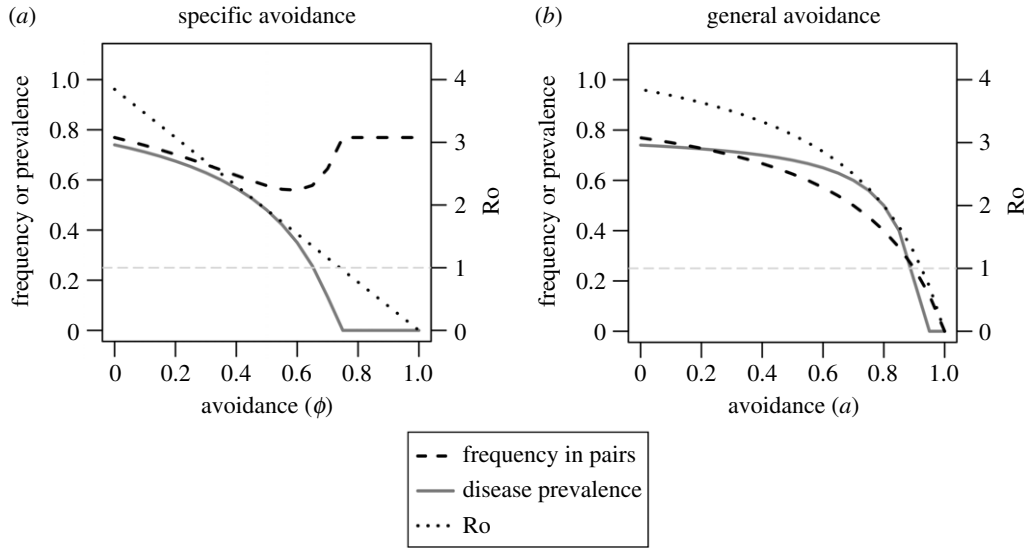

Figure 1. Pairing and disease dynamics at equilibrium when only the avoiding genotype X_2 is present in the population under different avoidance strategies. The light grey horizontal dotted line represents the basic reproductive number $R_0 = 1$, below which the disease cannot persist in the population, and above which sustained transmission is possible. Note the different *y*-axis scales for frequency/prevalence and R_0. $b = 1$, $\mu = 0.2$, $\delta = 1$, $\rho = 1$, $\upsilon = 0.3$, $k = 0.01$.

($N = S + 2G$), but changes at each time-step due to changes in numbers of X_1, X_2 and Y individuals.

We obtained equilibria using the differential equation solver (function 'ode' Runge–Kutta 'rk4' method) from the R package *deSolve* [33,34] and confirmed the stability of the equilibria by perturbation of initial values above and below equilibria.

3. Results

(a) Dynamics with no evolution

It can be shown that if all individuals are in pairs, i.e. in contact, then the dynamics of disease with pair formation are identical to the physiological resistance model (electronic supplementary material, information S1: SE22). We first examined the effect of the different avoidance strategies on the equilibrium frequency of individuals in pairs, the prevalence of the disease, and R_0 when only X_2, the avoiding genotype, was present (figure 1).

The basic reproductive number of the parasite, $R_0 = 2\delta G/N\mu$, is equivalent to canonical formulations for R_0 for frequency-dependent transmission, taking into account the frequency of groups within which transmission occurs. Increased specific avoidance of infected individuals is highly effective at reducing the prevalence of the disease, and also results in a decrease in the frequency of individuals in pairs (figure 1*a*). However, at high levels of specific avoidance, the frequency of individuals in pairs increases again, because few infected Y individuals remain for the X_2 individuals to avoid. With further avoidance, R_0 falls below 1, prevalence drops to 0, and pair formation is only among healthy individuals. Thus, at high levels of specific avoidance, hosts can successfully extirpate the disease from the population while maintaining their social structure.

General avoidance also reduces R_0 and prevalence, but if per-contact transmission rate (δ) is high, avoidance of pairing must be nearly complete to reduce R_0 below the threshold of 1 (figure 1*b*). Therefore, if hosts cannot detect infection in conspecifics but avoid pairing generally, behavioural avoidance effectively reduces disease risk, but at levels that concomitantly compromise host social structure.

(b) Evolution of behavioural resistance

We next examined the evolutionary dynamics in a population with genetic variants that do (X_2) and do not (X_1) avoid disease. When behavioural resistance was through specific avoidance of infected individuals and costs were fixed, X_1 and X_2 could stably coexist over an increasing range of costs to the avoider as avoidance levels increased (including greater than 50% reduction in birth rate at high levels of avoidance; figure 2*a*). When behavioural resistance was through general avoidance and costs were fixed, the same overall pattern emerged, but the spread of resistance required much higher levels of avoidance, and the coexistence of X_1 and X_2 was only possible under extreme levels of avoidance, although still over a wide range of costs (figure 2*b*).

When we modelled costs that were a consequence of not being in a group, costs were a function of the model dynamics. We thus investigated a range of birth rates of X_2 (Y-axis of figure 2*c*,*d*), which generated variation in the relative costs of X_2 (see electronic supplementary material, information S1, figure S4). In the case of specific avoidance, the benefits of reduced disease risk balanced the costs of lost social interactions, such that X_2 went to fixation only when its birth rate was higher than X_1 (figure 2*c*). When avoidance was general, X_1 could even sometimes reach fixation when X_2 had a higher birth rate, because at high rates of general avoidance, loss of social contacts carried costs that could not be compensated by inaccurate avoidance of disease (figure 2*d*). In both cases, when costs were linearly dependent on the frequency of pairs, stable polymorphism between X_1 and X_2 was not possible (see electronic supplementary material, information S1, figure S4 for details).

4. Discussion

Our results show that the dynamics of behavioural resistance can differ from physiological or biochemical resistance evolution depending on the nature of social behaviour and whether the costs are fixed or depend on sociality. As expected, the avoidance of social interactions with diseased individuals results in reductions of disease prevalence.

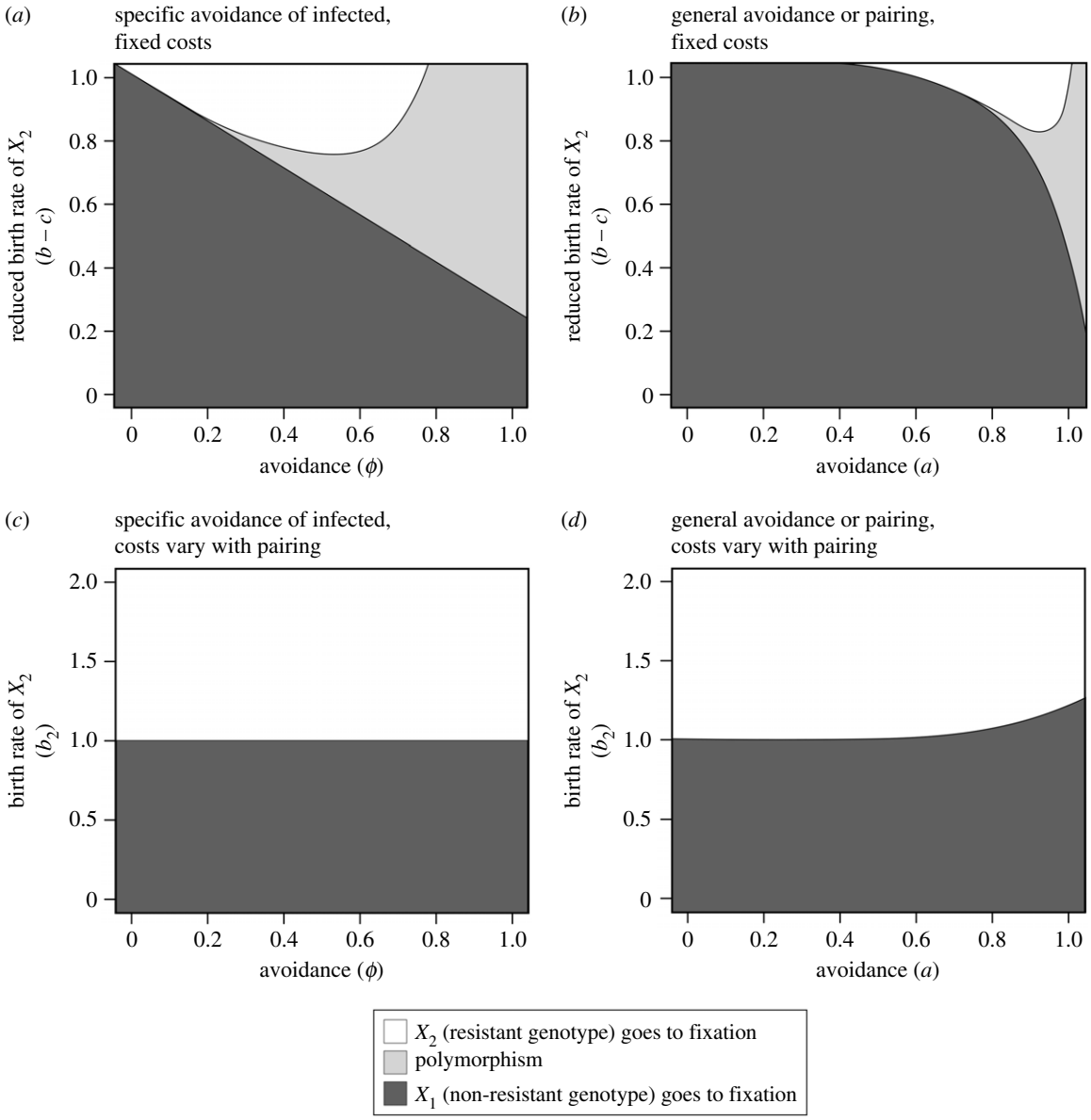

Figure 2. Shaded areas represent equilibrium gene frequency states for the models when the cost and the avoidance strategy of X_2 are varied. $b = 1$, $\mu = 0.2$, $\delta = 1$, $\rho = 1$, $\upsilon = 0.3$, $k = 0.01$. Note the different y-axis scales between (*a*)/(*b*) and (*c*)/(*d*).

Avoidance is more effective when it is specifically of diseased individuals, as opposed to general avoidance of all social interactions. High levels of specific avoidance result in full preservation of social structure because hosts can extirpate the disease through behavioural mechanisms, whereas at levels of general avoidance that prevent disease spread, social structure is harder to maintain. The spread of genotypes that avoid group formation depends on the type, level and nature of the costs of avoidance. When avoidance is specific and costs are fixed, the outcomes are identical to those for physiological resistance evolution, including the counterintuitive outcome that stable genetic polymorphism is more likely when resistance is extreme and costs are large rather than small [5,29]. However, when the costs represent the loss of benefits of group living itself, genetic variation in resistance is much harder to maintain, although the shape of the trade-off curve is likely to influence this result [20]. The possibility of stable genetic variation in behavioural resistance suggests not only that mixed avoidance strategies may represent stable states, but also that genetic differences may be at least partially responsible for individual differences in parasite avoidance in many species, including humans [35,36].

To dissect basic differences between behavioural and physiological resistance, we have deliberately kept the models simple. Future application to specific host–pathogen contexts would require more complexity in the temporal and social structure of the interactions. For example, in larger groups, transmission within and movement between groups would be possible, and behavioural resistance strategies could be more diverse. Additional models could also examine the effect of different disease costs, including mortality, or reproduction costs less severe than sterility. Previous research on physiological resistance suggests a similar extension of this study using adaptive dynamics [20,29]. Consistent with previous research, this simple model highlights trade-offs between the benefits of reducing disease risk and the costs of foregoing other opportunities, whether nutritional [27], reproductive [28] or in the case of our model, social.

Behavioural and physiological resistance are not separate phenomena but likely interact, with behavioural effects being

antecedent to physiological resistance, similar to a two-step infection process [37]. In such situations, genetic associations can arise between genes determining resistance, even without any direct physiological interaction. Physiological and behavioural defenses against parasites might also trade-off with one another. For example, house finches that avoid sick conspecifics invest less in immune defenses [23].

A genetic basis for parasite avoidance behaviours has support from knockout experiments in laboratory mice [38] and selective breeding in livestock [39]. There is also direct evidence of genetic polymorphism in social behaviour in halictid bees [40]. Behavioural resistance can thus be innate, as we model it, or learned through prior exposure [41–43]. How dynamics of learned resistance differ from innate is a rich direction for future research. Together these responses represent a suite of psychological and cognitive mechanisms that psychologists have termed the 'behavioural immune system' [44]. Our study shows that how this metaphor translates to dynamics of behavioural resistance merits further examination.

Data accessibility. This article has no additional data.

Authors' contributions. C.R.A. and J.A. conceived the project and derived the equations together. C.R.A. carried out the simulations and drafted the manuscript. J.A. provided critical input on the simulations and contributed to writing and revising the manuscript. All authors gave final approval for publication and agree to be held accountable for the work performed therein.

Competing interests. We declare we have no competing interests.

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