THE ROYAL SOCIETY

Cooperation and conflict in host-manipulating parasites

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The existence of adaptive host manipulation by parasites has received increasing empirical support in recent years. Here I develop an optimality model of the extent of host manipulation, incorporating within-host group size, relatedness and a range of realistic cost–benefit functions. The model highlights the cooperative nature of host manipulation, and the potential for cheating this entails. When relatedness in parasite groups is minimal, manipulation is suppressed, but not eradicated, reflecting the importance of interhost selection. A distinctive threshold phenomenon is predicted for a wide range of parameter values. Below the group size threshold, manipulation is zero. Above the threshold, the predicted behaviour depends critically on the biological details of the individual–group interaction.

The host-manipulation model is discussed in the light of two potential applications. If parasite group size is assumed to be a static characteristic of a parasite species or strain, the model generates a set of comparative predictions best suited to macroparasite systems. Additionally, the model can be used to predict density-dependent behavioural changes in expanding groups of parasites, as seen in quorum-sensing bacteria.

Keywords: host–parasite; host manipulation; cooperation; quorum-sensing bacteria

1. INTRODUCTION

The term 'host manipulation' is commonly used in the restricted context of macroparasites within intermediate hosts (e.g. Poulin 1998; table 1). Here I wish to widen the scope of the manipulation concept to include all host–parasite systems where the parasite adaptively brings about physiological, morphological or behavioural changes in the host to promote parasite fitness, either through an increase in transmission rate and/or persistence time. Table 1 summarizes, with examples, the scope of the manipulation concept used in this paper.

Contributing to host manipulation will in many cases bear a cost to the individual, while the benefits will be felt by every conspecific parasite within the host. If manipulation is costly, a key question emerges: how are cost-cutting cheats, or 'free riders', selected against?

Parasites have highly clustered spatial distributions, delimited by the bodies of their hosts. As a result, the fitness of an individual parasite is frequently related to the behaviour of its neighbours. The interrelated nature of fitness is a common theme in models of social evolution, and can be described by expressing fitness as the product of an individual and a group component (Frank 1998).

Analytical studies of social evolution in parasites have until now focused on the evolution of virulence (Frank (1996) provides a thorough review). Virulence (parasite-induced damage to the host) is a good biological example of a 'tragedy of the commons' (Hardin 1968), where individual gains lead to group losses. The gain to an individual parasite from higher virulence might be greater transmission to new hosts, while the group loss might be increased host mortality. The details of the models differ, but inevitably they describe virulence as a compromise between individual- and group-mediated effects on parasite fitness.

In this paper I present, to my knowledge, the first mathematical model of the evolution of host manipulation using a related approach to the virulence models outlined above. The key difference is that in the manipulation models the individual fitness component becomes a declining function of individual effort (reflecting the costs to the individual of host manipulation), while the group fitness component typically becomes an increasing function of total manipulation, reflecting the benefit of manipulation to the parasite group. Rather than a tragedy of the commons, the logic parallels the economic puzzle of collective action (Olson 1965). Human societies tend to ensure cooperative contributions to society through the use of social enforcement (e.g. taxation, peer pressure). For groups of parasites within hosts, the model suggests that 'collective action' is favoured by selection between groups, with groups containing more altruists outcompeting groups of free riders in the battle to infect new hosts. Furthermore, the model makes a novel prediction concerning the existence of a group size threshold, below which manipulative effort is constrained to zero.

In the next section I construct a fitness function for host-manipulating parasites, illustrating the basic trade-off, modulated by relatedness, between individual and group interest in locally interacting populations. Then I investigate the evolutionarily stable strategy (ESS) of host manipulation using a standard game-theoretic procedure, given differing functions relating individual action to group success. Finally, in § 3, I relate the model findings to potential empirical test-cases, focusing on comparative

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Table 1. *A broad classification of host manipulation*

modifications increasing transmission rate
(i) increased susceptibility to predation (e.g. behavioural alterations caused by macroparasites in intermediate hosts: Poulin 1994*a*,*b*, 1998; Kuris 1997)
(ii) increased propagule release (e.g. via tissue lesions caused by microparasite secretions: Robson *et al*. 1998)
modifications increasing persistence time
(iii) Resource diversion (e.g. by host castration; references in Kuris 1997)
(iv) Resource defence (e.g. by immune suppression, concomitant immunity: Grenfell *et al*. 1995*a*,*b*; S. P. Brown and B. T. Grenfell, unpublished results)

predictions of host manipulation by macroparasites, and experimental predictions of cooperative behaviour by quorum-sensing bacteria.

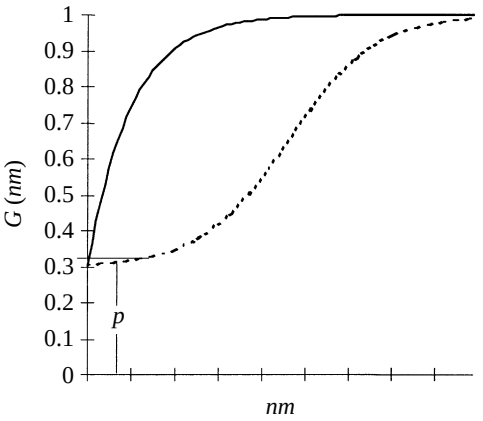

Figure 1. Sketch of the group fitness functions, $G(n\bar{m})$, where $n\bar{m}$ is the total manipulative effort per infrapopulation. The 'diminishing returns' function (equation (5)) is represented by the solid line. The sigmoidal function (equation (7)) is represented by the dotted line. Passive fitness, p, sets the level of fitness received for zero group manipulation. Saturation rate, s, sets the level of $n\bar{m}$ required for group fitness to reach its maximum.

2. THE MODEL

(a) *Cost to individuals, benefit to the group*

Fitness functions in interacting groups can be usefully separated into two elements: individual fitness and group fitness (e.g. virulence, Frank 1996; vigilance, McNamara & Houston 1992; sibling competition, Godfray & Parker 1992). In the case of parasitic host manipulation—or any other cooperative venture—cooperation has a negative fitness impact on the individual, and a positive fitness impact on the group (and hence, indirectly, on every individual within the group). The approach taken below is to construct a fitness function, W, consisting of an individual (I) and a group (G) component in a multiplicative form.

$$W(m_1,m_2) = I(m_1)G(n\bar{m}). \tag{1}$$

Here m is the individual contribution to host manipulation (scaled between 0 and 1), and $W(m_1,m_2)$ is the fitness of an m_1 strategist in a group of m_2 strategists. $I(m_1)$ equals the individual fitness function, a declining function of m_1, and $G(n\bar{m})$ equals the group fitness function, typically a rising function of summed host manipulation, $n\,\bar{m}$. Starting from a simple case of linear G and I functions, we have

$$W(m_1,m_2) = (1 - cm_1)(p + n\bar{m}). \tag{2}$$

Here c represents the cost to manipulation, and p represents passive fitness, the fitness of a non-manipulating parasite in a group of non-manipulators. An ESS m (m^*) can be found by maximizing $W(m_1,m_2)$ with respect to m_1, then setting $m_1 = m_2 = m^*$, and solving for m^* (Maynard Smith 1982). This technique finds a value of m^* such that any slightly deviating m has lower fitness. Applying this approach to equation (2) yields

$$m^* = \frac{n(\mathrm{d}\bar{m}/\mathrm{d}m_1) - cp}{cn(1 + (\mathrm{d}\bar{m}/\mathrm{d}m_1))}. \tag{3}$$

Here $\mathrm{d}\bar{m}/\mathrm{d}m$ is the rate of change of mean genotype contribution to host manipulation within the parasite infrapopulation relative to the individual genotype. The slope of group genotype on individual genotype is equivalent to the coefficient of relatedness between an individual and its group; thus we can express the equilibrium level of manipulation for differing levels of relatedness by replacing $\mathrm{d}\bar{m}/\mathrm{d}m_1$ with R, the coefficient of relatedness to the group (Taylor & Frank 1996), yielding $m^* = (nR - cp)/[cn(1 + R)]$. Note that R can be expressed as a function of group size n, and the between-pair coefficient of relatedness, r, as follows:

$$R = \frac{1 + (n-1)r}{n}. \tag{4}$$

(b) *Nonlinear group fitness*

Under the fitness equation (2), it is assumed that as total group manipulative effort ($n\bar{m}$) increases, group fitness increases at the same rate. The simple linear function does not allow group fitness to saturate with increasing group cooperation, leading to an impossible infinite group-optimum manipulation level. In reality, the relationship between group effort and group fitness is unlikely to be linear: upper and lower thresholds are likely to exist. The exact nature of the function $G(n\bar{m})$ will depend on the biology of the group interaction under study. First I consider a decelerating function of $n\bar{m}$, based on Poulin's (1994*a*) sketch of a realistic group fitness function,

$$G(n\bar{m}) = 1 - (1 - p)\mathrm{e}^{-sn\bar{m}}. \tag{5}$$

The parameters p and s control the behaviour of $G(n\bar{m})$ between 0 and 1. Passive fitness, p, describes the lower group fitness threshold, received when summed manipulative effort is zero; s controls the sensitivity to increasing summed manipulation. The function is sketched in figure 1.

Substituting equation (5) into equation (1), we have $W(m_1,m_2) = (1 - cm_1)[1 - (1 - p)\mathrm{e}^{-sn\bar{m}}]$. Using the technique outlined above, we find an expression capturing the ESS m^*:

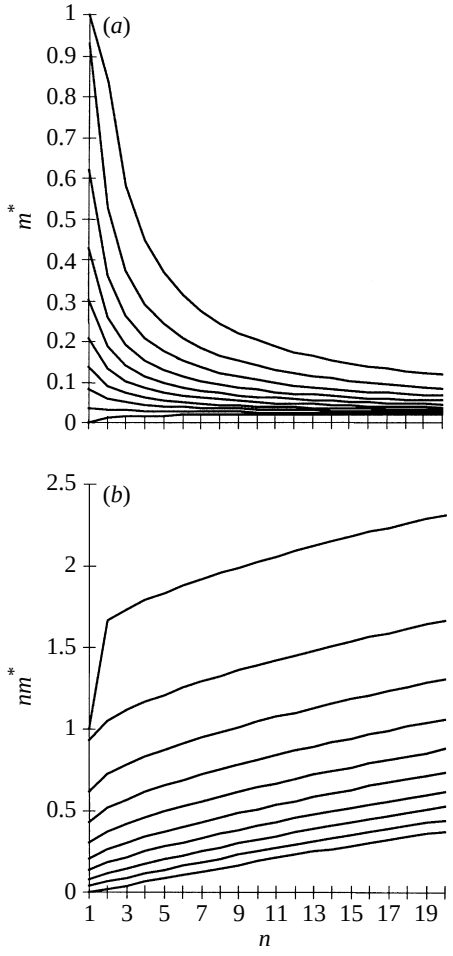

Figure 2. The effects (*a*) of group size (*n*) and cost of manipulation on ESS manipulation, m^*, and (*b*) resulting group behaviour at equilibrium, nm^*, plotted from numerical solutions to equation (6). The cost of manipulation ranges in steps of 0.1 from $c=0.1$ (uppermost lines) to $c=1$ (lowermost lines). Mean pairwise relatedness, r, is 0.05. The 'diminishing returns' group fitness function (see equation (5)) is defined by a passive fitness, p, of 0.5 and a saturation rate, s, of 1.

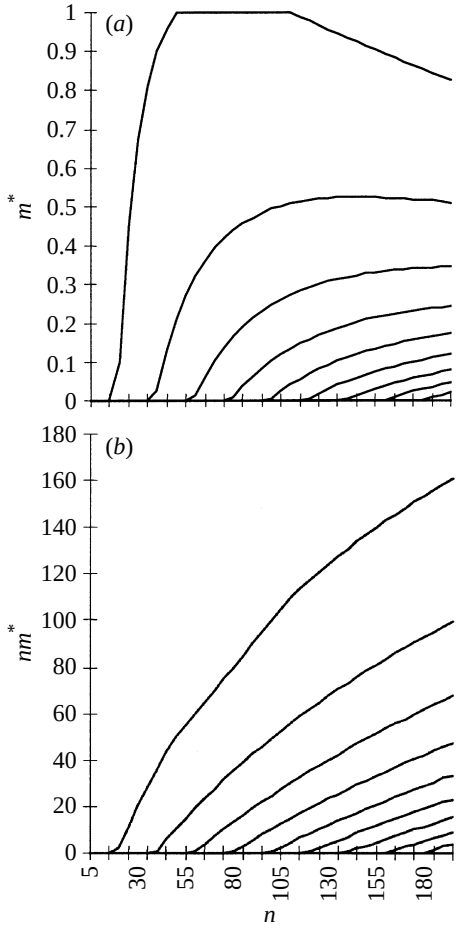

Figure 3. The effects of (*a*) group size (*n*) and cost of manipulation on ESS manipulation, m^*, and (*b*) resulting group behaviour at equilibrium, nm^*, plotted from numerical solutions to equation (6). The cost of manipulation ranges in steps of 0.1 from $c=0.1$ (uppermost lines) to $c=1$ (lowermost lines). Mean pairwise relatedness, r, is 0.5. The 'diminishing returns' group fitness function (see equation (5)) is defined by a passive fitness, p, of 0.5 and a saturation rate, s, of 0.01.

$$m^* = \frac{1}{c} - \frac{1-(1-p)e^{-snm^*}}{snR(1-p)e^{-snm^*}}. \qquad (6)$$

A solution for m^* was found numerically from equation (6), and is sketched in figures 2 and 3. When group fitness rapidly saturates (high s, figure 2), m^* appears highly constrained by n, showing relatively little variation with either relatedness, passive fitness or the cost of manipulation. As group size increases, m^* decreases at a rate sufficient to cause the summed degree of manipulative effort (nm^*) to show a slight increase. Replace manipulation with vigilance, and figure 2 reflects the key empirical trends found in the vigilance behaviour of a range of social birds and mammals (see Roberts (1996) and references therein), highlighting a shared underlying logic of collective action.

Introducing a broader period of responsiveness to the $G(n\bar{m})$ function (low s, figure 3) leads to greater model diversity, depending on c, p and R. In figure 3, s is defined so that group fitness saturates for n of around 500, as opposed to $n \approx 5$ in figure 2. A high saturation point reduces the return to manipulation in small group sizes. When passive fitness is zero, manipulation in small groups is still stable, as it is the only way to secure any fitness. The resulting pattern of m^* against group size is qualitatively similar to the pattern in figure 2, with m^* decreasing near inversely to group size. When passive fitness is non-zero, a trade-off between individual fitness costs and group fitness benefits is introduced. This trade-off explains the zero m^* region in figure 3 at low group sizes. As group size increases, the potential for cooperatively achieving a significant manipulative benefit increases, until a threshold is reached (whose position is sensitive to p, R and c). Above the threshold, manipulation becomes a stable strategy, as group size is sufficiently large to cooperatively generate sufficient group fitness to outweigh a reduction in passive fitness.

For a given parameter set, the effect of increasing R is, unsurprisingly, to increase m^*. However, it is important to note that manipulation is stable under a range of parameter values in unrelated groups. The fitness pay-offs for $R=1$ illustrate pure group selection, i.e. they maximize $W(m,m)$. When relatedness is imperfect, intragroup selection becomes increasingly important as n increases

(relatedness within the group, and genetic diversity between groups decreases with increasing n; see equation (4)). Pure intragroup selection would require every group to consist of the same proportion of competing strains, an unlikely situation. What we see in the models of imperfect relatedness is a tension between inter- and intragroup level selection. Take, for example, a case where $n=20$, $c=0.1$, $p=0.5$ and $s=1$ (illustrated for minimally related groupings in figure 2). When $R=1$, m^* equilibrates to the best group strategy, $m^*=0.23$. However, if different genotypes were to exist within the same host, any with a lower m would have a higher fitness. The fitness of $m^*=0.23$ in a population of m^* is $W(0.23,0.23)=0.972$, while the fitness of $m=0$ in a population of m^* is $W(0,0.23)=0.994$. However, groups of $m=0$ are limited to receiving passive fitness alone, $W(0,0)=0.5$. The tension between group selection working towards $m^*=0.23$ and individual selection working towards $m^*=0$ leads to an intermediate value. So for outbred sexual sibling groups (pairwise $r=0.5$), $m^*=0.20$, and for randomly assorted groups (pairwise $r=0$), $m^*=0.09$, much less than the group optimum.

(c) *Sigmoidal group fitness*

The 'diminishing returns' group fitness function illustrated in figure 1 and equation (5) is appealing when one considers the net outcome of host manipulation. Poulin (1994*a*) argued that investment in manipulation would show diminishing returns, using the following imaginary example. Consider a parasite that increases the proportion of time spent by its intermediate host away from a shelter, enhancing the probability of consumption by the definitive host. The parasite would tend to benefit more if it increased time in the open from 0 to 10%, than if it increased it from 50 to 60%, as the benefit of a fixed extra exposure period would be lower if exposure was already high. This is plausible, but the perspective on manipulative outcome may be misleading, as the relationship between manipulative effort and outcome is unlikely to be linear. Nonlinearities between manipulative effort and outcome are likely to be particularly evident in chemically mediated manipulation, where lower response thresholds will make small manipulative efforts relatively unrewarding. An alternative group fitness function might therefore be sigmoidal. The following group fitness function gives a sigmoidal response to increasing summed manipulation, scaled between p and 1

$$G(n\bar{m}) = p + \frac{1-p}{1+100\mathrm{e}^{-sn\bar{m}}}. \qquad (7)$$

Equation (6) is sketched in figure 1. Substituting equation (6) into equation (1) we have

$$W(m_1,m_2) = (1-cm_1)\,[p + (1-p)/(1+100\mathrm{e}^{sn\bar{m}})],$$

which by the method outlined in § 2(a) yields an expression capturing the ESS, m^*:

$$m^* = \frac{1}{c} - \frac{(100pc\mathrm{e}^{-snm^*}+c)(1+100\mathrm{e}^{-snm^*})}{(1-p)100Rcsn\mathrm{e}^{-snm^*}}. \qquad (8)$$

A solution for m^* was found numerically from equation (8), and is sketched in figure 4. The distinctive pattern of the response is very conserved; parameter manipulation simply alters the position and spacing of the m^* threshold points. Given a cost to manipulation of c, the threshold group size for cooperative manipulation rises with decreasing R and s, and with increasing p.

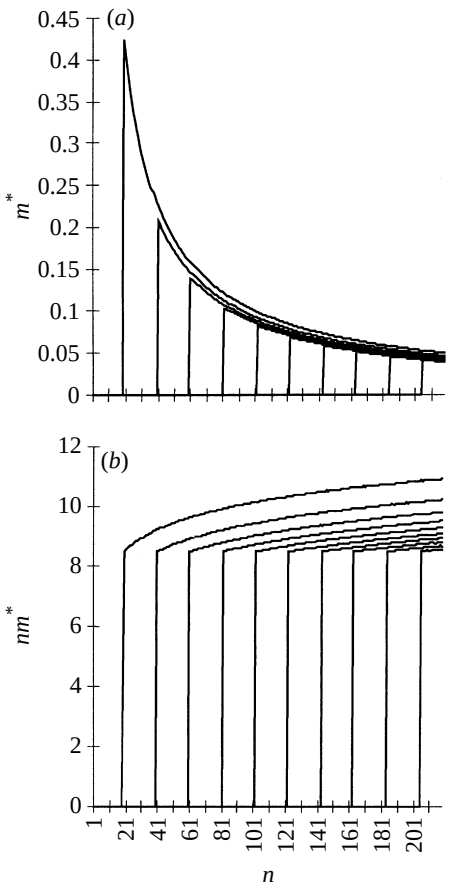

Figure 4. The effects of (*a*) group size (n) and cost of manipulation on ESS manipulation, m^*, and (*b*) resulting group behaviour at equilibrium, nm^*, plotted from numerical solutions to equation (8). The cost of manipulation ranges in steps of 0.1 from $c=0.1$ (uppermost lines) to $c=1$ (lowermost lines) Mean pairwise relatedness, r, is 0.5. The sigmoidal group fitness function (see equation (7)) is defined by a passive fitness, p, of 0.5 and a saturation rate, s, of 1.

3. DISCUSSION

The most striking model prediction is the existence of a group size threshold, below which manipulation is zero. The predicted behaviour above the threshold (which may be zero; see figure 2) depends critically on the biological details of the individual–group interaction (compare figure 3 with figure 4). The 'diminishing returns' model predicts a gradual fade-in of group manipulative behaviour above the threshold point, whereas the 'sigmoidal' model predicts an abrupt switch in group behaviour. The position of the threshold is the result of a compromise between relatedness (R) and group sensitivity (s) promoting a low threshold, and individual cost (c) and passive fitness (p) promoting a high threshold.

Despite an underlying generality, it remains crucial to tailor the model to specific biological systems. The results of this study demonstrate the significant impact on

predicted group behaviour of altering the form of the group fitness function. Many other significant variants of the group fitness function exist; for example, $G(n\bar{m})$ may decline as n becomes large, due to parasite-induced host mortality and crowding effects. Furthermore, an individual's fitness function may vary depending on location, both spatially and temporally in the hierarchy of parasite recruitment. To illustrate the importance of tailoring the models to specific biological systems, I shall review a number of model assumptions and data limitations in the light of potential biological test-cases (unpublished results).

(a) *Macroparasites and static group size*

To make between-lineage predictions on the level of host manipulation, it is necessary to treat parasite infrapopulation size, n, as a characteristic of a particular parasite species or strain. This assumption underpins the idea that an optimal level of manipulation (m^*) characterizing a particular lineage evolves in response to a particular group size, n. Describing the infrapopulation size of a parasite species with a single static figure n is a simplification best suited to the relatively stable infrapopulation dynamics of macroparasites. Dynamic changes in cooperative behaviour within expanding infrapopulations are discussed in § 3(b).

The results of this study lead to two related epidemiological predictions (see figures 2*b*–4*b*): (i) that summed host manipulation (nm^*) will be more extensive in parasite species characterized by greater mean burdens, and (ii) that the frequency of manipulation will be greater in higher-burden species. Predictions based on the other parameters (p, c, s and R) are easy to generate using the model, but would be extremely difficult to test due to a lack of data on p, c, s and to a lesser extent, R.

Despite a multitude of qualitative reports of host manipulation, quantitative estimates of the extent of host manipulation are available for no more than 30 species of macroparasite, scattered across two phyla (Poulin 1994*b*; Thomas *et al*. 1998). Given the scarce and phylogenetically scattered data, the most productive approach would be to focus on a particular species of manipulative macroparasite, and compare manipulative behaviour between differing strains or infection foci characterized by differing infrapopulation sizes. A between-strain analysis would be far more robust to confounding variables (parasite size, life cycle, mechanism of manipulation, phylogeny, etc.), and so require a smaller sample size.

Further considerations must be taken into account in selecting suitable test systems, as a number of well-documented cases of host manipulation occupy extremes of the model parameter space, lessening the power of the model to account for variation in manipulative behaviour. The parameter n is perhaps most likely to be observed at an extreme, as in several well-documented helminth–invertebrate systems, infrapopulation sizes are commonly one (Poulin & Thomas (1999) and references therein), thus losing the cooperative element of manipulation central to the model. Unfortunately, little or no information exists on the costs of manipulation (Poulin 1994*a*), yet it is conceivable that adaptive manipulation may in certain circumstances be costless, being, for example, a consequence of a costless shift in parasite niche within the host body. Were manipulation to be cost free, then the basic trade-off between individual cost and group benefit would be lost.

The most productive test-cases for the model would involve groups of parasites contributing in a dose-dependent (i.e. potentially costly) manner to a manipulative goal. Examples abound of chemically mediated manipulation, particularly in microparasite systems, as I shall discuss below. Among macroparasites, perhaps the most promising study cases involve parasites of humans, due to a relative abundance of data. S. P. Brown and B. T. Grenfell are currently working on a stochastic simulation model of manipulation of the human immune system by schistosome worms. Briefly, adult worms within the definitive (e.g. human) host are thought to secrete an antigen that is cross-reactive with larval surface antigen (Smithers & Terry 1967, 1969), thus effectively vaccinating the host against larval competitors. By using a tailored version of the basic model outlined in this paper, it is possible to generate epidemiological predictions linking schistosome prevalence at a particular infection focus with the intensity of immune system manipulation.

Comparative predictions about the manipulative behaviour of individual parasites (see figures 2*a*–4*a*) offer a more direct test of the model, as adaptive variation in manipulative effort between parasite strains would be tested by direct observation, rather than by inference from epidemiological trends. Before these predictions can be assessed properly, a quantitative understanding of the mechanisms of host manipulation is required.

(b) *Microparasites and variable group size*

In addition to differences in manipulative effort between lineages, the model can be applied to changes in manipulative behaviour within single, expanding, infrapopulations of parasites. Indeed, the most compelling qualitative support for the threshold phenomenon can be found in the pattern of host manipulation within expanding populations of bacteria.

Cooperative behaviour in bacterial populations is commonplace. Colonies modify their environment in many ways, for example by the release of enzymes, fibrous building materials or antibacterial agents (Fuqua *et al*. 1996). *Erwinia carotovora*, a plant tuber pathogen, provides a well-studied example of bacterial cooperation. Established colonies of *E. carotovora* produce a range of digestive enzymes and antibacterial agents ensuring efficient digestion of the host in the absence of bacterial competitors (Bainton *et al*. 1992; Jones *et al*. 1993). Intriguingly, individual bacterial cells only begin to secrete 'cooperative' enzymes once a distinct density threshold has been passed, in line with the main qualitative prediction of the model presented above.

In order to tailor phenotypic expression to changing group size, bacteria face the problem of assessing group size. Flocks of birds face the same problem when adjusting vigilance levels. Crested terns respond to flock arrivals by decreasing vigilance, and to departures by increasing vigilance, illustrating a dynamic adjustment of strategy (Roberts 1995). Quorum-sensing bacteria use chemical rather than visual cues to adjust their levels of cooperation. Through the release of low molecular weight signalling molecules, they are able to monitor and respond to changes in bacterial density (Fuqua *et al*. 1996;

Robson *et al*. 1997). Once a particular density threshold is crossed, cooperative behaviour (e.g. the production of tissue-degrading enzymes in *E. carotovora*) is switched on.

The additional strategic complexity of producing and responding to a signalling molecule adds an extra dimension to the system captured in the current models. At present, equations (8) and (6) describe an ESS of manipulative effort for a quorum-sensing bacterium, under the assumptions of honest, costless, group size information. More realistically, signalling will have a cost, and it may not pay to signal honestly. S. P. Brown and R. A. Johnstone (unpublished results) explore the conditions for signal stability in a two-trait game-theoretic model of quorum-sensing bacteria.

In conclusion, the models suggest that the extent of host manipulation is the result of a tension between intrahost selection favouring lower manipulative effort and interhost selection favouring higher manipulative effort. By linking the effects of infrapopulation size, relatedness and cost–benefit functions, plausible quantitative predictions are generated. Two contrasting applications are highlighted for further investigation, each requiring theoretical care in tailoring the model to specific biological systems.

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