A model of insect–pathogen dynamics in which a pathogenic bacterium can also reproduce saprophytically

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We developed a model of the population dynamic interaction between an insect and a pathogenic bacterium motivated by study of *Serratia entomophila*, a commercially exploited pathogen of the New Zealand grass grub (*Costelytra zealandica*). The bacterium is able to reproduce saprophytically, though it competes for saprophytic substrates with non-pathogenic strains, which appear to be superior competitors, probably because they lack a plasmid that carries genes required for pathogenicity. The effect of saprophytism and competition on the invasion criterion (R_0), short-term dynamics and long-term dynamics are described. Saprophytism can reduce (possibly to zero) the host threshold at which the pathogen can invade, though this reduction is less when there is competition with non-pathogenic strains. In a model of short-term population dynamics designed to mimic the application of bacteria to a host epizootic, saprophytism enhances the reduction in host density, though again this is tempered by competition with non-pathogens. In the long term, a pathogen that can develop saprophytically can drive its host to extinction in the absence of competition with non-pathogens. When the latter are present, host extinction is prevented. The addition of saprophytic reproduction can stabilize an otherwise unstable host–pathogen model, but we were unable to find a stable equilibrium given the further addition of a wholly saprophytic bacterial strain. The model suggests that enhancing or selecting for saprophytic ability could be a way of improving biological control.

Keywords: population dynamics; insects; bacteria; disease models; pastures

1. INTRODUCTION

Pathogenic bacteria provide an important resource for the control of insect pests (Tanada & Kaya 1993). Most commercial success in insect microbial control has been obtained with *Bacillus thuringiensis* (Bt). Strains of this bacterium possess plasmids that carry a variety of genes encoding toxins that are selectively pathogenic to a range of insect species (Höfte & Whiteley 1989). Bt is usually applied as a spray and insects die after consuming contaminated food; bacterial reproduction in the field and secondary infections are normally unimportant. Other entomopathogenic bacteria used in pest management infect and multiply in the host, releasing bacteria into the insect’s environment and causing epizootics of disease. This group includes *Bacillus popilliae*, which has been produced commercially for control of the Japanese beetle (*Popillia japonica*, Scarabaeidae) in lawns and other grasslands (Klein 1992), and *Serratia entomophila* which is marketed for control of the New Zealand grass grub (*Costelytra zealandica*, Scarabaeidae), an important pasture pest (Jackson 1994).

S. entomophila is found throughout New Zealand grasslands in both pathogenic and non-pathogenic forms (Grimont *et al.* 1988). Pathogenicity is conferred by plasmid-borne genes (Glare *et al.* 1993; Grkovic *et al.* 1995), and is specific to larvae of *C. zealandica*. Both pathogenic and non-pathogenic forms can reproduce saprophytically (i.e. on decaying organic material) in the absence of hosts, but plasmid-bearing strains appear to be at a competitive disadvantage in competition with non-pathogenic strains (M. O’Callaghan, T. R. Glare and T. A. Jackson, unpublished data; in other bacteria, plasmid costs have been identified on a number of occasions, for example: Bouma & Lenski 1988; Nguyen *et al.* 1989; Modi & Adams 1991; Turner *et al.* 1998). Saprophytic reproduction and susceptibility to competition from non-pathogenic strains will have major effects on the population dynamics of a pathogenic bacterium. The aim of this paper is to explore these interactions and determine their effect in helping or hindering biological pest management.

A full model of the *Serratia*–grass grub system would need to take into account seasonal variation in saprophytic substrates and the subsequent bacterial response. Unfortunately, insufficient data are currently

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available to construct such a model, although aspects of the within-season dynamics of the *Serratia*–grass grub interaction have been modelled without the additional complexity of saprophytism and bacterial competition (Barlow & Jackson 1992; Drummond *et al.* 1996). Our intention here is to explore the population dynamics of the insect–pathogen–saprophyte interaction using the type of detail-independent model pioneered in insect–pathogen studies by Anderson & May (1980, 1981; see also, for example, Begon *et al.* 1992; Begon & Bowers 1994; Bowers & Begon 1991; Dwyer 1992; Dwyer & Elkinton 1993; Hochberg 1989, 1991; Thomas *et al.* 1995; White *et al.* 1996a). While such models are unable to make precise quantitative predictions about the population dynamics of specific systems, they are often highly successful at predicting qualitative dynamic patterns (e.g. Briggs & Godfray 1996a).

The paper is organized as follows. In the next section we describe a continuous-time model of an insect, a pathogenic bacterium and a non-pathogenic bacterium. In the following sections we explore the effects of saprophytism and strain competition on the invasion criterion (R_0); on the rate at which a non-reproducing host population can be controlled by the bacterium; and on long-term dynamics.

2. THE SERRATIA–GRASS GRUB MODEL

We develop a model of *Serratia*–grass grub interactions based on model G of Anderson & May (1981) which describes insects that are infected by a disease possessing a free-living stage in the environment. Like that model, it includes time-dependent state variables (where t is time) that represent the densities of susceptible hosts $S(t)$; of infected hosts $I(t)$; and of the infectious disease organism in the environment $P(t)$. It differs in allowing the disease organism to be capable of reproduction in the absence of its insect host, and in including a fourth state variable $B(t)$ to represent non-pathogenic bacteria with which the pathogenic bacteria compete when reproducing saprophytically.

The model is

$$\begin{aligned} S'(t) &= (a - b)S(t) - \nu S(t)P(t), \\ I'(t) &= \nu S(t)P(t) - (b + \alpha)I(t), \\ P'(t) &= \Lambda\alpha I(t) + \lambda_p f_p[P(t), B(t)]P(t) - \mu_p P(t), \\ B'(t) &= \lambda_B f_B[B(t), P(t)]B(t) - \mu_B B(t). \end{aligned} \quad (1)$$

Throughout, primes are used to denote derivatives. Susceptible hosts reproduce at a rate a and die at a rate b , both of which are assumed to be density independent and seasonally invariant. Infection is modelled as a random encounter process with rate determined by the transmission parameter ν . Deaths of infected hosts result from the same density-independent causes as those that afflict susceptibles (at rate b), but also through the action of the bacterium (rate α). Those hosts killed by the disease release Λ bacteria into the soil. Bacteria in the soil reproduce at a per capita rate $\lambda_p f_p[P(t), B(t)]$ where λ_p is the growth rate of the pathogenic strain in the absence of any competition, and the function $f_p[\cdot]$ represents the reduction in growth rate due to intra- and inter-strain

competition. Pathogenic bacteria die at a rate μ_p . The dynamics of the non-pathogenic bacteria are structurally identical to the pathogenic bacteria, except that there is no term representing bacteria released on the death of an infected host.

In analysing the model, we shall have to specify the competition functions $f_p[\cdot]$ and $f_B[\cdot]$. We use

$$f_P = f_B = \exp[-c_P P(t) - c_B B(t)],$$

the exponential form ensuring that saprophytic reproduction asymptotes at zero as bacterial density goes to infinity. The two parameters c_P and c_B describe the competitive effects of the two bacterial strains. We assume $c_P < c_B$ and hence that non-pathogenic strains exert greater competitive effects on all other bacteria, perhaps by faster use of limiting resources, while both strains suffer the same proportional reduction in saprophytic growth rates for the same distribution of competitors. Parameter values used in simulations are guided where possible by studies of the *Serratia*–grass grub system (O'Callaghan *et al.* 1989; Jackson 1994; O'Callaghan & Jackson 1996; M. O'Callaghan, T. R. Glare and T. A. Jackson, unpublished data).

3. R_0

When will a pathogenic strain of bacterium be able to invade an environment with constant densities of susceptible hosts (S) and non-pathogenic bacteria (B)? In other words, when does the pathogen more than replace itself ($R_0 > 1$)? If we define the vector $\mathbf{P}(t) = (I(t), P(t))$ and write $\mathbf{P}'(t) = \mathbf{A}[\mathbf{P}(t)] \mathbf{P}(t)$ then $R_0 > 1$ is equivalent to the dominant eigenvalue of $\mathbf{A}|_{\mathbf{P}(t) \rightarrow 0}$ being greater than zero. While this is straightforward to calculate from equation (1), we proceed by a more biologically intuitive route.

Suppose, first, that the pathogen is incapable of other than pathogenic reproduction, as in Anderson & May's model G. Our model differs from their basic model in that we assume that (i) infecteds have zero fecundity (bacterial infection kills insects prior to reproduction); (ii) pathogens are produced only when infected hosts die of the disease; and (iii) the numbers of pathogens consumed by insects is minute compared with the numbers in the environment. In fact, Anderson & May consider all these extensions, although not together. Then

$$R_0 > 1 \implies \left[\frac{\alpha}{\alpha + b} \right] [\Lambda] [\nu S] \left[\frac{1}{\mu_p} \right] > 1. \quad (2)$$

$R_0 > 1$ means that every infection must lead to more than one further infection. R_0 is the product of the four square-bracketed terms in equation (2). The probability that an infected insect gives rise to infectious bacteria (as opposed to dying for other reasons) is $\alpha/(\alpha + b)$, in which case Λ bacteria are released into the environment where they infect susceptible hosts at a certain rate, $\nu S(t)$, throughout their average lifetime of $1/\mu_p$ time units. Hence invasion is more likely when most mortality of the infecteds results in the production of a large number of highly infectious bacteria that survive for a long period of time in an environment with many susceptible hosts. R_0 is a function of host density and equation (2) can be rearranged to obtain

Figure 1. The effect of saprophytism on disease persistence: host threshold for disease persistence as a function of the number of competitors for different values of the saprophytic growth rate. All three quantities have been made dimensionless for increased generality: the host threshold is expressed as a fraction of the host threshold in the absence of any saprophytic growth (i.e. S_T/S_T^* where S_T^* is the no-saprophytism threshold), the number of competitors is expressed as $1 - f_P[0, B]$, and the saprophytic growth rate is expressed as a fraction of the bacterial death rate (λ_P/μ_P).

the threshold host density (S_T) that allows invasion: $S_T = \mu_P(\alpha + b)/(\nu\Lambda\alpha)$.

Consider now the invasion criterion when the pathogen is able to reproduce as a saprophyte as well as a pathogen,

$$R_0 > 1 \Rightarrow \left[\frac{\alpha\Lambda\nu S}{(\alpha + b)} \right] \left[\frac{1}{(\mu_P - \lambda_P f_P[0, B^*])} \right] > 1, \quad (3)$$

where B^* is the equilibrium density of non-pathogenic bacteria. The first three terms of equation (2) appear identically in equation (3) where they are combined together within the first square brackets. Saprophytic reproduction influences the last term of equation (2), the average lifespan of a pathogenic bacterium in the soil, which becomes the expression in the second square brackets of equation (3). This can be interpreted as the average lifespan of a clone of pathogenic bacteria in the soil, taking into account their potential for saprophytic reproduction. If saprophytic reproduction exceeds mortality, i.e. $\mu_P < \lambda_P f_P[0, B^*]$, then the clone is potentially immortal and the pathogen will always persist in the soil, even when the host is absent. Although we shall not pursue it here, an invasion criterion can also be calculated if $B(t)$ varies rather than being at equilibrium (briefly, the logarithm of the geometric mean of the expression in equation (3) must exceed zero).

Thus the invasion criterion is influenced by (i) the number of susceptible hosts (S); (ii) the saprophytic growth rate (λ_P which is determined implicitly by resource availability); and (iii) the density of competitors (B). How they combine is illustrated in figure 1 where it can be seen that as λ_P increases from zero, the threshold host density falls, the reduction being greatest when competitors are rare.

Figure 2. The epizootic that results from applying pathogenic bacteria at a density of 10^7 m^{-2} to a susceptible host population of density 200 m^{-2} . S , I and P are the densities of susceptibles, infecteds and pathogenic bacteria ($\times 10^{-12}$) respectively, and time is measured in days. Parameter values used in simulations are $b = 5 \times 10^{-4}$; $\alpha = 0.015$; $\nu = 5 \times 10^{-11}$; $\Lambda = 10^8$; $\lambda_P = 0.01$; $\lambda_B = 0.02$; $c_P = 0.001$; $c_B = 0.002$; $\mu_P = 0.1$ and $\mu_B = 0.03$.

4. SHORT-TERM POPULATION DYNAMICS

Consider now the short-term population dynamics of the insect and its pathogen. We are motivated by situations where pathogenic bacteria are applied to a field to control larval insects and we seek to find out how bacterial saprophytic growth and competition influence the efficacy of biological control. Because larval insects do not reproduce, and we are interested here in short-term dynamics only, we set the insect reproductive rate equal to zero ($a = 0$).

Suppose that the density of the pest insect is 200 m^{-2} and that pathogenic bacteria are applied to the field so that their density is 10^7 m^{-2} . In the absence of saprophytic reproduction, an epizootic occurs resulting in a crash in host density (figure 2; the parameter values used are given in the figure legend). An indication of the efficiency of biological control is the time taken to reduce the host density by a half, which in the case of our basic parameter set is ca. 80 days.

Figure 3 shows how faster control is obtained when the pathogenic bacteria are also able to increase in density saprophytically (ignoring for the moment the influence of non-pathogenic bacteria). As λ_P increases from 0 to 0.15 the time taken to halve host density is itself reduced by nearly one-half, as long as pathogenic bacteria do not compete for resources. If competition occurs, saprophytic reproduction still leads to faster control, though the advantages are reduced.

Now consider the presence of non-pathogenic bacterial strains, which can potentially compete for saprophytic resources. We assume that prior to the application of the pathogenic strain, the non-pathogens are at the equilibrium density set purely by intra-strain competition, which is $(1/c_B) \log(\lambda_B/\mu_B)$. Note the obvious fact that $\lambda_B > \mu_B$ is required for an equilibrium to exist. In figure 4 we show how the reproduction and death rates of the non-pathogenic strains of bacteria influence the speed of biological control. In the absence of competitors, pathogenic bacteria with the parameters used in figure 4 can

Figure 3. The number of days required for the pathogen to halve the density of the host for different levels of saprophytic reproduction and intraspecific competition. In the absence of saprophytic reproduction ($\lambda_P = 0$), host densities are halved in just under 80 days. Increasing levels of saprophytic growth speed the reduction in host density, though the reduction is less when intraspecific competition (measured by c_P) is important. Other parameter values are given in figure 2.

halve the density of their host in a little under 60 days with saprophytic growth (and *ca.* 80 days acting purely as a pathogen). The presence of the competitor reduces the advantage of saprophytic reproduction, the more so when the competitor has a high growth rate and, especially, when it has high longevity in the soil. The third parameter characterizing the non-pathogenic strain is its competitive effect on other bacteria, c_B , which can be interpreted as a surrogate parameter for the extent to which it depletes resources. For a fixed density of non-pathogenic bacteria, the higher the value of c_B , the greater the reduction in pathogenic bacterial growth rates. However, if we assume that the non-pathogenic bacterium is at equilibrium prior to the application of the pathogen, then the value of c_B has no effect on the time taken to reduce host density by 50%. The reason for this is that a higher value of c_B leads to (i) a lower non-pathogen bacterial equilibrium; and (ii) the pathogen suffering greater reductions in saprophytic growth rates per individual non-pathogenic competitor; the two effects exactly cancel each other out.

5. LONG-TERM POPULATION DYNAMICS

How do saprophytism and competition with non-pathogenic bacteria influence equilibrium population densities and population stability? We derived equilibrium population densities for the different scenarios discussed above and analysed the local stability of the equilibria. Stability analysis involves linearizing the dynamics around the equilibrium and calculating the Routh–Hurwitz criteria (for further details see May (1974) and Anderson & May (1981)).

For our version of Anderson & May's model G (without saprophytism), the equilibrium densities (denoted by asterisks) of the host and pathogenic bacterium are $P^* = (a - b)/\nu$; $S^* = S_T$ and $I^* = (a - b/\alpha + b)S_T$, where

Figure 4. The effect of non-pathogenic bacteria reproductive and death rates on the time taken to halve host densities (see text). Pathogenic bacteria have $\lambda_P = 0.15$ and $\mu_P = 0.1$; other parameters as in figure 2. The non-pathogen is unable to persist when $\mu_B < \lambda_B$ and in this region, the 'white floor' in the figure, the time taken to halve the host population is determined purely by the pathogenic bacteria.

S_T is the threshold host density defined in the section on R_0 . At equilibrium, more virulent (high ν) pathogens are rare, while those attacking hosts with high net intrinsic growth rates ($a - b$) are common. The susceptible host population is reduced to S_T , the level at which one infection causes on average one daughter infection (i.e. the level at which $R_0 = 1$). The number of infecteds increases with the host's intrinsic growth rate ($a - b$) but decreases as the total mortality of infecteds rises ($a + b$). The Routh–Hurwitz criteria are never satisfied: the model has no stable equilibrium but shows diverging oscillations. Anderson & May's basic version of model G was stable over a wide range of parameter values. What makes our version of the model unstable is the assumption that infected hosts do not reproduce.

Adding saprophytic reproduction and bacterial competition has no effect on the equilibrium density of pathogenic bacteria ($P^* = (a - b)/\nu$) but does affect equilibrium densities of susceptibles and infecteds. These are $S^* = \hat{S}$ and $I^* = (a - b/\alpha + b)\hat{S}$ where $\hat{S}$ is a quantity closely related to the host threshold, S_T ,

$$\hat{S} = \frac{(\alpha + b)(\mu_P - f_P(P^*, B^*))}{\alpha \Lambda \nu}. \quad (4)$$

$\hat{S}$ is the host density at which an infection causes on average one daughter infection when both pathogenic and non-pathogenic bacteria are at equilibrium density. S_T is simply $\hat{S}$ but with the pathogen very rare, i.e. P^* replaced by zero. $\hat{S}$ is larger than S_T because competition between pathogenic bacteria reduces their populations' growth rate. The equilibrium population density of non-pathogenic bacteria is

$$B^* = \frac{1}{c_B} \left(\ln \frac{\lambda_B}{\mu_B} - c_P P^* \right). \quad (5)$$

Non-pathogenic bacteria are more abundant when they have high fecundity (λ_B), low death rates (μ_B) and low intra-strain competition (c_B), and their densities are reduced when they compete strongly (c_P) with abundant pathogenic bacteria (P^*).

Inspection of equation (4) shows that for the host to have a positive equilibrium $\lambda_P f_P[P^*B^*] < \mu_P$. In the absence of saprophytism ($\lambda_P = 0$) this condition is always met, but when saprophytism occurs the pathogenic bacterium may be able to maintain itself at a high enough density to drive the host extinct. Both intra-strain and inter-strain competition, which act to reduce $f_P[\cdot]$, make host extinction less likely. In the case of saprophytism with no competition from non-pathogens, another way of expressing the criterion for host persistence is that the equilibrium density of the pathogen when the host is present must be greater than the pathogen's equilibrium density were the host to be absent and the bacterium to reproduce purely saprophytically. When inter-strain competition occurs, host persistence requires that the non-pathogenic bacteria are superior competitors ($\lambda_B/\mu_B > \lambda_P/\mu_P$) and hence in the absence of hosts would drive the pathogen extinct (stable coexistence is not possible between the two bacterial strains in the absence of hosts).

Equation (5) shows that the non-pathogenic bacterium can be driven to extinction by the pathogenic bacterium if $P^*(a-b)/\nu > (1/c_P) \ln(\lambda_B/\mu_B)$. The right-hand side of this inequality is determined by bacterial competition but the left-hand side is governed solely by the parameters of the host–pathogen interaction. Hosts with high net rates of increase ($a-b$) and pathogens with relatively low rates of transmissibility (ν) are more likely to maintain standing crops of pathogenic bacteria that reduce the densities of non-pathogens, perhaps driving them extinct.

Turning to the stability of the equilibrium, consider first the case of saprophytic reproduction without competition from non-pathogenic bacteria. A stable equilibrium is now possible, though the Routh–Hurwitz criteria are complex and not open to easy interpretation, and so have been explored numerically. Stability depends on five parameter combinations: $(a-b)$; μ_P ; c_P/ν ; λ_P and $a+b$. Where an equilibrium is possible (i.e. the hosts do not go extinct), stability is favoured by low host net intrinsic rates of increase ($a-b$) and long-lived pathogens (low μ_P). High host growth rates makes regulation more difficult, while a long pathogen lifespan enables them to survive periods when hosts are absent. Increasing intra-strain competition relative to transmissibility (c_P/ν) reduces stability while saprophytic reproduction (λ_P) makes stability more likely. Stability is enhanced by those factors that promote saprophytic reproduction because this allows the pathogenic bacteria to survive periods of low host densities. Finally, large increases in the rate of deaths of infecteds ($a+b$) increase slightly the likelihood of stability, though the effect is small. The number of bacteria released by a dying host (A) has no effect on stability. Near to stability boundaries, populations show limit cycle behaviour, while further away they display diverging oscillations that lead to the extinction of the host or pathogen.

The stability analysis of the four-state variable model including non-pathogenic bacteria is technically difficult

and the Routh–Hurwitz criteria are again very difficult to interpret. However, in a numerical search of parameter space, we failed to find regions where the three-species equilibrium was stable. We cannot exclude the possibility that a stable equilibrium occurs under some parameter combinations, but if it does occur it is under very restrictive conditions. The reason that competition with non-pathogenic bacteria lessens stability appears to be that it reduces the ability of the pathogen to survive at reasonable densities during periods of low host abundance.

6. DISCUSSION

The models presented here represent an initial attempt to study the dynamic consequences of saprophytism and competition with non-pathogenic strains on an insect–pathogen interaction. We begin by criticizing the models and highlighting the simplifications we have made, before discussing the consequences of saprophytism and bacterial competition.

(a) Model criticism

The models have been deliberately phrased in as simple a manner as possible, partly so that they can be compared with the classical models of insect–pathogen interactions, and partly because the available experimental information does not yet justify a more elaborate model framework. The results must thus be interpreted cautiously as providing broad predictions about likely population dynamic consequences, rather than specific hypotheses about the *Serratia*–grass grub system or other systems.

Age structure and seasonality are probably the two most important structural simplifications we have made. Our model divides the host into two classes, susceptible and infected, while in reality most insect pathogens (including *Serratia*) are chiefly parasites of the larval stage. We also treated demographic processes as continuous (except host reproduction in §4) which may approximate to what occurs in relatively non-seasonal tropical environments, but is a poorer description of temperate systems. In the *Serratia*–grass grub system, both host reproduction and the availability of substrates for saprophytism are markedly seasonal. The effects of introducing age structure and seasonality into the classical models of insect–pathogen interactions (Anderson & May 1980, 1981) have been examined by Briggs & Godfray (1995, 1996b) and largely involve increased instability as a consequence of the time-lags inherent in age structure, and the possibility of cycles caused by dynamical interference between the different time-lags in the system. Seasonality was included in the specific *Serratia*–grass grub models of Barlow & Jackson (1992) and Drummond *et al.* (1996), but these models did not address saprophytism or bacterial competition.

Within the context of our modelling framework, we made several simplifying assumptions about the interaction. First, we assumed that the transmission process was simple linear mass action. At the moment, our data do not justify a more sophisticated function, but we suspect that for most soil-borne insect pathogens the risks of infection probably increase at a less than linear rate as bacterial densities rise, the effect being due to heterogeneities in the

distributions of the host and its pathogen (Reeve *et al.* 1994). Nonlinear transmission may be important in stabilizing host–pathogen interactions (Hochberg 1991; Briggs & Godfray 1995, 1996b) and has been demonstrated experimentally by d'Amico *et al.* (1996), Knell *et al.* (1996a,b) and Dwyer *et al.* (1997). A second simplification is to assume that all bacteria in the soil have the same demographic and transmission parameters. After a pathogenic bacterium such as *Serratia* is applied to soils, its densities initially drop very rapidly, and then decline more slowly. This probably occurs both from lack of fitness of the cultured pathogen and through initial leaching from macropores in the soil, the residue persisting in micropores (O'Callaghan *et al.* 1989; O'Callaghan & Jackson 1996; unpublished data). We have not modelled the initial rapid decline in bacterial density in the short-term dynamics section as we suspect that the leached bacteria are lost before they can cause infections. Yet we are conscious that our understanding of the structure of the soil as it affects bacterial dynamics is still rudimentary. One possibility is that locations within the soil provide reservoirs for bacteria where they do not cause infection but have enhanced persistence. Hochberg (1989) has shown that such reservoirs may have an important effect in stabilizing insect–pathogen interactions. A third simplification is to ignore plasmid transfer via conjugation. The disease-causing plasmid can be transferred naturally between strains of *Serratia* (Glare *et al.* 1996). While transfer is very important as an evolutionary process, the rate at which we believe it occurs is so low compared with the demographic processes that we have ignored it in our population modelling. Fourth, we have treated the non-pathogenic bacterial population as a single unit. In reality, non-pathogens (and possibly pathogens) will consist of a variety of strains or species. For example, in the *Serratia* system, a second species (*S. proteamaculans*) is always present in New Zealand pastures and competes with pathogenic and non-pathogenic *S. entomophila* for saprophytic substrates. Fifth, we have assumed no density dependence in the host, as in Anderson & May's (1981) model G. In severe outbreaks of grass grub in New Zealand, the below-ground biomass of beetle can exceed the above-ground biomass of sheep, and at least in these conditions host density dependence is significant (Barlow & Jackson 1992). Finally, this model deals only with local dynamics; it is possible that the interactions between the insect and the pathogen, or between the pathogen and the saprophyte occur within a metapopulation with local extinctions and colonizations (White *et al.* 1996b; Wood & Thomas 1996). For soil bacteria, such metapopulation dynamics might operate on quite small physical scales.

(b) *Saprophytism and bacterial competition*

Pathogenic bacteria (and other micro-organisms) that attack insects may be used in much the same way as conventional insecticides, or they may be used as biological control agents that are able to reproduce and cause secondary infections (Thomas *et al.* 1995). The extent to which a bacterium will cause secondary infections depends on the magnitude of the invasion parameter R_0 . We quantified the extent to which saprophytic reproduction enhances R_0 and increases the

likelihood of secondary infections. In the equivalent Anderson & May model without saprophytism, R_0 is proportional to the expected lifespan of a bacterium in the soil. With saprophytism, R_0 is proportional to the expected lifespan of a bacterial lineage in the soil and if saprophytic reproduction is high enough this will guarantee persistence, irrespective of host density. Saprophytism thus seems a desirable property for a biocontrol agent as it may allow persistence in the absence of the pest. Also, as shown by the short-term population model, it can increase the speed with which a pest outbreak is controlled. Although we have no data on this point, it is possible that saprophytism has some negative implications for pest management. We know that there is a trade-off between possession of the plasmid that carries genes required for pathogenicity, and the efficiency of the bacterium in using saprophytic substrates; conceivably the possession of the biochemical machinery that allows saprophytism reduces the bacterium's efficiency as a pathogen.

The benefits of saprophytic reproduction are reduced by competition for resources with non-pathogenic bacteria which, without the plasmid, enjoy a competitive advantage. The invasion criterion, R_0 , now becomes both a function of host density and competitor density (and implicitly the density of the saprophytic substrate). The presence of competitors can also reduce or eliminate the benefits of saprophytism on the speed with which a pest outbreak is reduced. One assumption that we have made is that bacterial competition influences the rate of reproduction and not the rate of mortality, were the latter to happen, perhaps through an allelopathic process, non-pathogenic bacterial strains would have a far stronger negative impact on biological control.

The equilibrium density of pathogenic bacteria is not influenced by saprophytism or competition (though its stability may be). Saprophytism decreases the equilibrium host density because a standing crop of pathogenic bacteria can be maintained on the saprophytic substrate. The extent of this decrease is reduced by intra- and inter-strain competition. If there is sufficient reproduction on saprophytic substrates, the pathogen can drive the host to extinction. This suggests that a useful long-term microbial control strategy would be to increase the bacterial saprophytic growth rate by cultural practices, or perhaps by supplying suitable substrate. But if there are non-pathogenic bacteria present—as there almost certainly will be—and if these strains or species are competitively superior—as is expected if there is a cost of pathogenicity—then our models suggest that host extinction is not feasible. The benefits of extra saprophytic growth are outweighed by increased competition. The model also suggests that new *Serratia* strains for commercial exploitation might be engineered or selected for their competitive ability. This is similar to the conclusions reached by Jackson & O'Callaghan (1997) on other grounds in their identification of environmental competence as a key factor in the success of microbes applied for control of soil dwelling pests.

Our basic model without saprophytism never has a stable equilibrium but shows diverging oscillations. Including saprophytism allows for a stable equilibrium at least for some parameter values. This occurs because bacterial populations are less likely to decline dramatically in

periods when hosts are scarce. However, we were unable to find parameter values for which the full three-species equilibrium was stable. Pathogenic bacteria increase in frequency during host epizootics but are outcompeted by non-pathogens when the host is rare or absent, the typical outcome being diverging oscillations and extinction of one of the species.

(c) *Future work*

Several other aspects of the insect–bacterium interaction may influence population dynamics, but these are not at present sufficiently well understood to incorporate into a model. First, to infect a host, the bacterium must be eaten by the insect, adhere to the wall of the digestive tract, and then gain entry to the insect's body cavity (Jackson *et al.* 1993). Plasmid-borne genes are required to enter the body cavity, but not for adhesion. If non-pathogenic bacteria are fed to a grass grub, higher subsequent doses of pathogenic bacteria are required for infection (Glare *et al.* 1992). The reason for this appears to be that non-pathogenic bacteria occupy a high proportion of the possible adhesion sites for the pathogen. If this were to occur in the field, the host could be partially 'immunized' against the pathogen by high densities of non-pathogens. Such a phenomenon would almost certainly reduce the effectiveness of biological control, and tend to destabilize the population dynamics.

Second, different enzyme systems are probably involved in pathogenic and saprophytic nutrition. As in other bacteria, the state of the *Serratia*'s enzyme systems is probably inherited epigenetically. Hence, a dying host will release bacteria adapted for feeding as a pathogen rather than as a saprophyte. If this adaptation enhances the probability of transmission and successful infection, the resulting density-dependent increase in pathogen efficiency will be a stabilizing factor in the host–pathogen interaction.

Non-pathogenic modes of reproduction are found in other insect pathogens, especially fungal diseases, and are the rule for many human and animal disease-causing bacteria (*Salmonella*, *Listeria*, pathogenic strains of *E. coli*), but have been relatively little studied in insect pathology. Our models show the potential significance of saprophytism in population dynamics and pest management, but also underline the many gaps in our knowledge of these organisms. In particular, it would be helpful to know more about the structure of bacterial communities, how the physical structure of the soil affects bacterial dynamics, and—as usual—more about transmission. Finally, modern molecular techniques offer the opportunity of manipulating bacterial and plasmid genomes to enhance pathogenicity or other bacterial properties. But an understanding of the dynamics of insect-pathogenic bacteria, and their interaction with the non-pathogenic microbial flora, is essential in predicting the value and safety of a genetically manipulated organism after its release into the environment.

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