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THE IMPACT OF PARASITE MANIPULATION AND PREDATOR FORAGING BEHAVIOR ON PREDATOR–PREY COMMUNITIES

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Abstract. Parasites are known to directly affect their hosts at both the individual and population level. However, little is known about their more subtle, indirect effects and how these may affect population and community dynamics. In particular, trophically transmitted parasites may manipulate the behavior of intermediate hosts, fundamentally altering the pattern of contact between these individuals and their predators. Here, we develop a suite of population dynamic models to explore the impact of such behavioral modifications on the dynamics and structure of the predator–prey community. We show that, although such manipulations do not directly affect the persistence of the predator and prey populations, they can greatly alter the quantitative dynamics of the community, potentially resulting in high amplitude oscillations in abundance. We show that the precise impact of host manipulation depends greatly on the predator's functional response, which describes the predator's foraging efficiency under changing prey availabilities. Even if the parasite is rarely observed within the prey population, such manipulations extend beyond the direct impact on the intermediate host to affect the foraging success of the predator, with profound implications for the structure and stability of the predator–prey community.

Key words: *community structure; functional response; Lotka-Volterra; trophic transmission.*

Introduction

There is currently considerable interest in the phenomenon of host manipulation by parasites (Moore 2002, Thomas et al. 2005). Such manipulations are often interpreted as adaptive acts by the parasite that facilitate transmission and increase fitness. The most striking examples involve trophically transmitted parasites which manipulate their intermediate hosts to make them more vulnerable to predation by the parasites' definitive hosts. These manipulations can occur through increasing conspicuousness, or by altering the behavior of the intermediate hosts to make them easier to be captured. Such manipulations of host behavior are fascinating examples of the extended phenotype of the parasite. Considerable theoretical and empirical work has been carried out to explore the circumstances under which manipulation evolves, and to determine the factors affecting the optimal degree of manipulation (Poulin 1994, Thomas et al. 1998, Brown 1999, Brown et al. 2001, Tompkins et al. 2004).

While host manipulation has led to much research from an evolutionary perspective, the role it plays in driving host–parasite population dynamics and structuring predator–prey communities is much less well known (Dobson 1988, Morand and Gonzalez 1997). Considerable theoretical work over the last few decades has demonstrated that parasites may have a significant impact on host population dynamics (Anderson and May 1978, 1981, Bowers and Begon 1991, Dobson and Hudson 1992). These models show that one of the most important components driving the dynamics of host–parasite systems is the rate at which susceptible individuals contact infected hosts or parasite infective stages in the environment (Getz and Pickering 1983, Swinton et al. 1998, Keeling 1999, McCallum et al. 2001, Turner et al. 2003, Dobson 2004). For trophically transmitted parasites, these contacts are determined by the collective behavior of both the predator and prey species in the community and, in particular, by the predator's foraging behavior.

The relationship between a predator's feeding rate and prey density is known as the predator's functional response (Holling 1959, Holt 1983) and the form of this functional response will be vital for determining transmission rates of trophically transmitted parasites. If parasite-induced manipulation is occurring, this will affect the rate at which predators contact infected prey response and hence the parasite's transmission rates. For example, the acanthocephalan *Polymorphus paradoxus* uses amphipods as its intermediate host (Bethel and Holmes 1973, Moore 1984). Amphipods infected with *P. paradoxus* cystacanths show a markedly different response to light, becoming photophilic rather than the typical photophobic behavior of an uninfected amphipod, making them much more vulnerable to predation by visually hunting ducks, the definitive host of the

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parasite. This example demonstrates the effects that manipulation can have upon the likelihood of contact between intermediate and definitive hosts. As the inventory of host-manipulating parasites increases to include medically important species such as *Plasmodium falciparum*, which has been shown to increase the attractiveness of its human host to its mosquito vector (Lacroix et al. 2005), the development of population models that include these behavioral features becomes much more urgent if we are to realistically model host–parasite dynamics.

Here, we develop a suite of models to assess the impact that different forms of host manipulation may have on the population dynamics of predator–prey communities, and determine the host and parasite characteristics that favor the occurrence of host manipulation. We consider the community dynamics in detail for a model using a Type I functional response, and then extend these findings to consider Type II and Type III functional responses. Finally, we distinguish between microparasites and macroparasites, and outline a model that considers the degree of manipulation to be dependent upon the parasite load of the host. Overall, we show that such manipulations can alter the stability and dynamics of predator–prey interactions, and that the outcome of manipulation is greatly affected by the nature of the predator's functional response and the trait of the intermediate host undergoing manipulation.

Basic Model Structure

We begin by modeling the simplest trophically transmitted parasite system, similar to that used by Lafferty (1992). We assume a community of two host species, one a predator on the other. This predator–prey community harbors a parasite, which uses the prey species as an intermediate host to transmit to the definitive host predator. Initially, we assume the parasite is a microparasite (e.g., virus, bacteria, protozoa, and so forth) and so we only need consider whether an individual of each species is infected or not. The parasite can manipulate the intermediate host prey species, for instance by increasing its conspicuousness, making infected individuals more likely to be preyed upon than uninfected individuals. We assume manipulation is instantaneous and "all or nothing," so that, as soon as an individual becomes infected, it is manipulated to a fixed degree and subsequent infections of the same host do not further affect its level of manipulation. Note that, by "manipulation," we simply assume that the presence of the parasite causes a behavioral or morphological alteration in the conspicuousness of its intermediate host, but do not consider whether this change is directly caused by an adaptive manipulation from the parasite, or is simply a by-product of the parasite's presence (Brown et al. 2001, Damsgaard et al. 2005). A schematic diagram of the basic model is given in Fig. 1, and is described by the following equations:

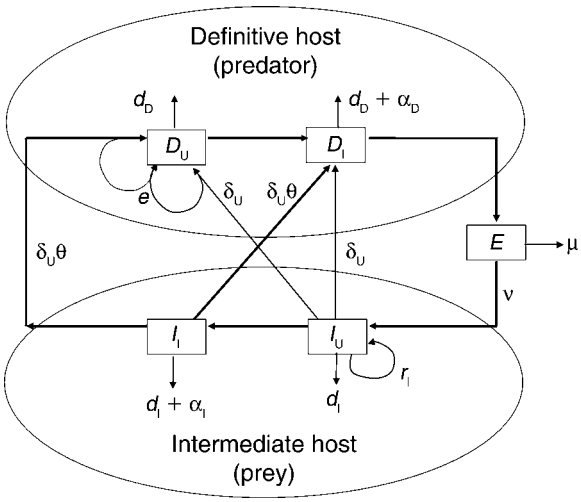

FIG. 1. Schematic diagram of the basic (microparasite) trophic transmission model, involving predation by a definitive host of an intermediate host. Parasite manipulation increases the conspicuousness of infected prey by a factor θ. The thick black lines show the passage of the parasite through the host community. I_U is the density of uninfected prey species, I_I is the density of infected prey, D_U is the density of the uninfected predator species, D_I is the density of infected predators, and E is the density of parasite infective stages in the environment; r_I is the prey's reproductive rate, d_I is the prey's baseline mortality rate, and α_I is the parasite-induced prey mortality rate; μ is the mortality of parasite infective stages, and ν is the transmission rate of the parasite to the prey; δ_U is the baseline predation rate, e is the predator's conversion efficiency of consumed prey, d_D is the predator's baseline mortality rate, and α_D is the parasite-induced predator mortality rate; θ is the proportional increase in predation rate due to manipulation of the prey by the parasite.

$$\begin{aligned}
\frac{dI_U}{dt} &= I_U r_I(1 - qI_U) - (D_U + D_I)\delta_U f_U(I_U, I_I) - \frac{\nu\lambda D_I I_U}{\mu} \\
\frac{dI_I}{dt} &= \frac{\nu\lambda D_I I_U}{\mu} - I_I(d_I + \alpha_I) - (D_U + D_I)\delta_U\theta f_I(I_U, I_I) \\
\frac{dD_U}{dt} &= eD_U\delta_U f_U(I_U, I_I) - D_U\delta_U\theta f_I(I_U, I_I) - D_U d_D \\
\frac{dD_I}{dt} &= D_U\delta_U\theta f_I(I_U, I_I) - D_I(d_D + \alpha_D)
\end{aligned} \tag{1}$$

where I_U is the density of the uninfected prey species (the intermediate host), I_I the density of infected prey, D_U the density of the uninfected predator species (the definitive host), and D_I the density of infected predators. In the absence of the predator or parasite the prey population reproduces at per capita rate r_I and is regulated in a density-dependent manner to a carrying capacity K (=$1/q$, where q is the strength of intraspecific density dependence acting on the intermediate host population). In the presence of the predator (but in the absence of the parasite), the prey species are consumed at rate $\delta_U f_i(I_U, I_I)$ where δ_U is the baseline predation rate and $f_i(I_U, I_I)$ is the predator's functional response on species i which, in the presence of the parasite, may

vary according to the relative densities of both uninfected and infected prey. The predator reproduces at a rate determined by the number of prey consumed and a "conversion rate" e, which represents the efficiency of the predator species at converting consumed prey into offspring. Predators die at a background per capita rate d_D. Hence, in the absence of the parasite, the predator–prey dynamics are described by the classic Lotka-Volterra model with density-dependent regulation of the prey.

The parasite affects the prey species (its intermediate host) in three ways. First, it increases the mortality rate of infected prey from the baseline rate d_I to $d_I + \alpha_I$ (α_I is the additional parasite-induced host mortality, or virulence of the parasite). Second, we assume that infection leads to castration of the prey species, although analysis of models allowing infected prey to reproduce shows the dynamics are not qualitatively different from those presented here. Finally, the parasite manipulates the host prey, increasing its likelihood of being captured by a predator by a factor θ (the per capita predation rate of infected prey, δ_I, is $\delta_U\theta$). Hence, when $\theta = 1$, infected prey are as likely to be preyed upon as uninfected prey; when $\theta < 1$, predators are deterred from capturing infected prey; and, when $\theta > 1$, infected prey are more likely to be preyed upon than uninfected prey. Once a predator consumes an infected prey item, it immediately becomes an infected definitive host (D_I), which again is assumed to be parasitically castrated, and which dies at background rate d_D and due to parasite-induced mortality at rate α_D. Parasites in the predator definitive host produce infective stages at rate λ, which are passed into the environment where they either die at rate μ, or are consumed by intermediate hosts at rate ν. For simplicity, we assume the rate of loss of infective stages in the environment due to consumption by the prey is negligible compared to the background mortality rate, and that the dynamics of this external parasite stage are relatively fast, so we do not model them explicitly.

Building on this basic model structure, we conduct stability analyses to assess the factors affecting the persistence of the predator and prey species and the parasite in the community, and consider the impact host manipulation has on the fitness of the parasite and the dynamics of the two host species. First, we illustrate the behavior of the model under the simplest scenario, assuming a Type I functional response (Holling 1959) by the predator feeding on the prey.

The community dynamics of host manipulation and a Type I functional response

The predator's functional response, captured by the term $f_i(I_U, I_I)$, determines how the rate of predation on prey type i varies according to the relative densities of the two prey types. In the simplest case (type I), this is a linear response, such that $f_i(I_U, I_I) = I_i$ so, for instance, the net predation rate on the uninfected prey species I_U is $(D_U + D_I)\delta_U I_U$.

First, we can derive an expression for the basic reproductive ratio (R_0) of the parasite (Dobson and Keymer 1985):

$$R_0 = \left[\frac{D_U\delta_U\theta}{d_I + \delta_U\theta(D_U + D_I)}\right]\left(\frac{\lambda}{d_D}\right)\left[\frac{\nu D_I}{\mu + \nu(I_U + I_I)}\right]. \quad (2)$$

This is a measure of the fitness of the parasite, and provides a threshold level of parasite persistence (given by $R_0 = 1$), showing that persistence is determined by the balance of those factors that increase parasite numbers and those that deplete them. The first term in Eq. 2 describes the flow of parasites from infected prey to the predatory definitive host (i.e., the net infection rate of predators divided by the rate of loss of infected prey). The second term describes the net rate of production of parasites from infected predators and the final term describes the infection process of the prey population. Therefore, the two infection processes of the parasite's life cycle (infection of the prey and infection of the predator) act independently of each other (see also Dobson and Keymer 1985), suggesting that the parasite may use one feature of the lifecycle (e.g., fast transmission to the intermediate host, ν) to compensate for shortfalls in another aspect (e.g., low susceptible definitive predator host densities, D_U). This expression for R_0 also allows us to determine how the abundance of predators and prey affect the net reproductive rate of the parasites. Formally, by setting the above equation equal to 1 and solving for D_U and I_U, it is possible to obtain expressions for the threshold host densities of intermediate host prey and definitive host predators (T_{D_U} and T_{I_U}, respectively), below which pathogen persistence cannot occur (Dobson 1988).

Stability analysis (detailed in Appendix A) reveals that there are four regions of parameter space with qualitatively different dynamics (Fig. 2). First, there is a region where the prey persists alone (and reaches its carrying capacity, K) in the absence of either predator or parasite (region i, Fig 2). This occurs when

$$\frac{d_D}{\delta_U e} > K.$$

If the predator has a low birth rate (low e), captures prey rarely (low δ_U) or has a high death rate (high d_D), it is unlikely to persist. Since the parasite requires the definitive predator host to complete its life cycle, it cannot persist if the predator does not. If the above inequality is reversed the predator will persist with the prey and reduce its abundance to a level below its carrying capacity (region ii, Fig. 2). An important result is that predator persistence is unaffected by the presence or absence of the parasite. Therefore in qualitative terms, a trophically transmitted parasite should be unable to force the predator population to extinction. However, as we show later, the parasite is able to affect the quantitative abundance of the predator, potentially driving it to very low densities.

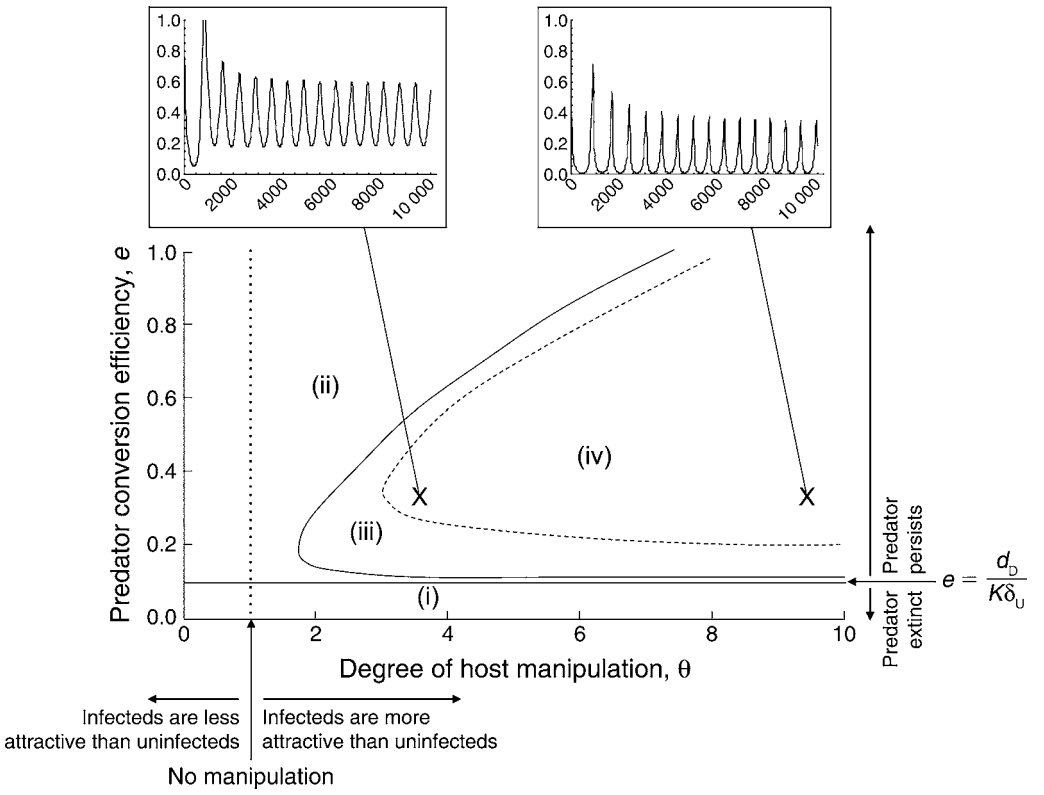

FIG. 2. Parameter space for a Type I functional response, showing regions where (i) intermediate host persists alone, (ii) intermediate host and predator persist alone (disease absent), (iii) disease persists within the two-host community, and (iv) disease persists, but with cyclical dynamics. The dotted vertical line shows where infecteds have the same attractiveness as uninfecteds (i.e., no manipulation). The horizontal line shows where net predator reproduction (the prey capture rate, δ_U, multiplied by the number of prey at their carrying capacity, K, multiplied by the predator conversion rate, e) exactly equals predator mortality, d_D in the absence of the parasite. Values of e above this line allow predators to persist, whereas values of e below this line mean predators cannot sustain themselves. The inset graphs show numerical simulations of the number of uninfected predators (D_U) over time, from the locations in parameter space shown. The degree of host manipulation, θ, is the proportional increase in predation rate due to manipulation of the prey by the parasite.

The third region of interest is one where the parasite can persist and become endemic in the community (region iii, Fig. 2).This is facilitated if the parasite manipulates its intermediate prey host making it more likely to be captured by the predator (i.e., large θ). Indeed, for the parameter values used in Fig. 2, the parasite has to manipulate its host in order to survive. Parasite persistence can only occur for intermediate predator birth rates (Fig. 2). When predator birth rates are very low [$e \approx b_D/(K\delta_U)$], the turnover rate of predator definitive hosts is too low for the parasite to be able to persist ($D_U < T_{D_U}$). However, when predator birth rates are high, the high density of predators results in rapid prey removal, reducing the density of susceptible intermediate hosts below the threshold for disease persistence ($I_U < T_{I_U}$). Under these conditions, it is only through high degrees of host manipulation that the parasite can ensure sufficient numbers of infected prey are consumed to favor parasite persistence.

In the final region of interest (region iv, Fig. 2), the parasite is again endemic within the host community, but the dynamics are cyclical. This region is more likely with increasing degrees of host manipulation. Hence, manipulation per se can alter the dynamics of an otherwise stable predator–prey community. An empirical test of this prediction would be to assess whether parasites with high degrees of manipulation (those where infected prey are much more likely to be preyed upon than uninfected prey) tend to display epidemic dynamics, or are found to occur within cyclical host communities. An additional point to note is that minimum population densities decrease with increasing degrees of host manipulation (see inset graphs in Fig. 2), so that in the stochastic reality of the natural world, either the predator or prey population may go extinct in one of the dynamic troughs, purely as a result of high levels of host manipulation.

Host manipulation also affects the quantitative levels of abundance of the host community. Increasing the degree of manipulation reduces the density of uninfected predators (Fig. 3a). However, host manipulation increases the number of uninfected prey (Fig. 3b), because manipulated infected individuals are more likely to be removed as the degree of manipulation increases. As

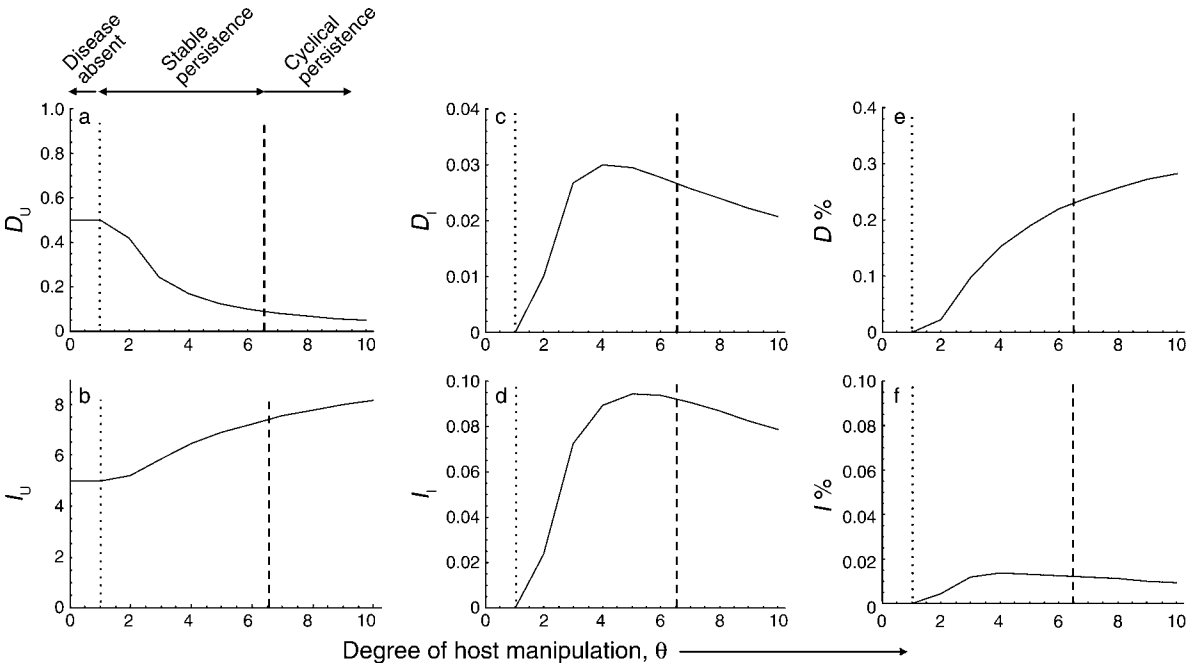

FIG. 3. Equilibrium densities for (a) uninfected definitive predator hosts (D_U), (b) uninfected intermediate prey hosts (I_U), (c) infected definitive predator hosts (D_I), (d) infected intermediate prey hosts (I_I), and prevalence of infection in (e) the definitive predator hosts ($D\% = D_I/[D_U + D_I]$) and (f) the intermediate prey hosts ($I\% = I_I/[I_U + I_I]$) as a function of the degree of host manipulation (θ). The vertical dotted line divides the regions of disease absence and persistence, and the dashed line divides the regions of stable dynamics from cyclical.

may be expected, manipulation initially increases the number of both infected predators and prey (Fig. 3c, d). However, high levels of manipulation reduce the numbers of infected individuals, due to considerable parasite-induced host mortality of both hosts. These changes in host density are reflected in changes in disease prevalence in the two host populations (see also Lafferty 1992). Increasing host manipulation increase disease prevalence in the predator (Fig. 3e). However, in the prey there is an initial increase in prevalence for low levels of host manipulation, but large manipulations lead to a decrease in disease prevalence (Fig. 3f). While prevalences may be quite high in the predator, prevalences in the prey population may be very low (note the different scales on the axes in Fig. 3e and f). Clearly, high levels of manipulation mean that infected prey survive for a very short time before they are consumed, and so they make up a very small proportion of the total prey population. Therefore, just because disease is very rare in the prey population, this does not mean it is not an important factor driving host dynamics or that host manipulation is not an important force in structuring the host community.

Finally, many of the host and parasite life-history parameters have important impacts on the outcome of manipulation (Fig. 4). In general, those terms that increase the parasite's basic reproductive ratio (Eq. 2) tend to favor parasite persistence so that, for instance, parasites with high fecundity (as is typical for many parasites; Fig. 4b), or prey species with rapid reproductive rates (which may apply in particular to invertebrate species used as intermediate hosts; Fig. 4c), may be able to persist with little or no host manipulation. Conversely, high degrees of host manipulation may allow the persistence of highly virulent parasites that would otherwise be driven to extinction (Fig. 4d).

Alternative Predator Functional Responses

Type II functional response

The previous section considered the dynamics of the parasite–host community assuming a linear, Type I functional response for the predator. Possibly a more likely scenario is that predation saturates, due to "handling time" constraints on the predator, resulting in a limit to the number of prey a predator can consume in a given time period. This may be modeled as a Type II functional response:

$$f_i(I_U, I_I) = \frac{I_i}{1 + I_U\delta_U h_U + I_I\delta_I\theta h_I}$$

where the h_i are the handling times per prey item (Holling 1959, Holt 1983). Note that when $h_U = h_I = 0$, we get a Type I response and, as the h_i increase, predation becomes increasingly constrained. Here we initially assume the handling times of the infected and uninfected prey are the same ($h_U = h_I = h$), but we explore the scenario where they may differ in a later section.

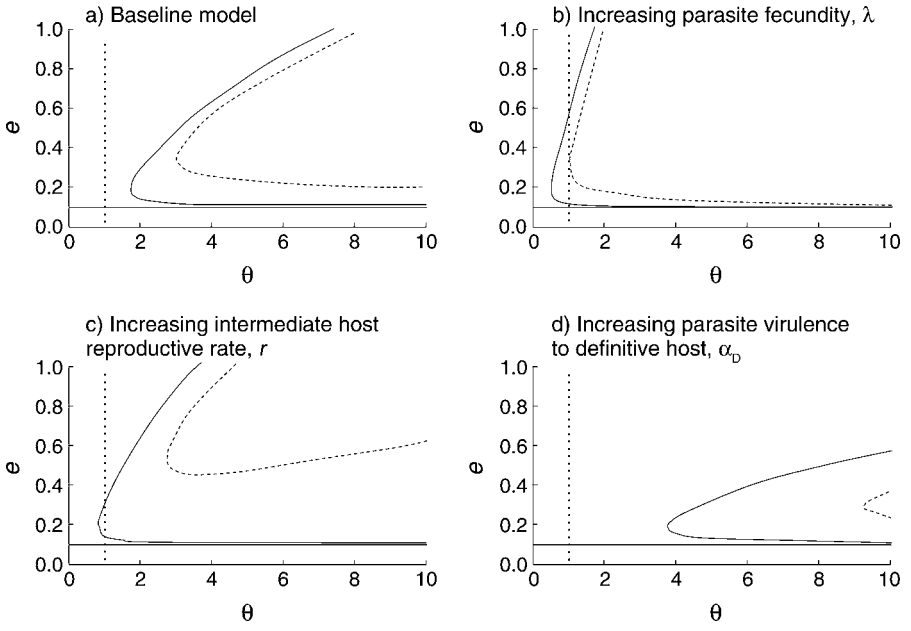

FIG. 4. Parameter space for a Type I functional response, showing the effect of varying different parameters. The dotted vertical line shows where infecteds have the same attractiveness as uninfecteds (i.e., no manipulation). Dynamical regions and parameters correspond to those in Fig. 2.

Stability analysis reveals similar qualitative dynamics to those observed for a Type I functional response (Fig. 5), so many of the conclusions described above still apply. However, the degree of constraint imposed by the predator's handling time can have important implications. Increased handling times make it more difficult for the predator to persist, even in the absence of the parasite; clearly, increasing handling time reduces the number of prey a predator can consume in a given time period, so the predator has to be more efficient at converting those prey into its own offspring (i.e., a higher e is needed) in order to facilitate predator survival. However, perhaps counterintuitively, providing that the predator can survive, increasing handling time reduces the degree of host manipulation needed to ensure parasite persistence (Fig. 5) because it leads to a buildup in the number of prey, as they are not removed from the population so quickly. This in turn allows the parasite to build up in the intermediate host population, favoring parasite persistence. Finally, it should be noted that parasite manipulation has important consequences for the stability of the host community and the likelihood of cyclical dynamics (Fig. 5). In particular, when prey handling times are large (e.g., Fig. 5b, c), as the degree of host manipulation is increased, the system may initially undergo population cycling, but increasing manipulation further can lead to re-stabilization of the system.

Type III functional response

A third form of predator functional response occurs when there is some degree of habituation on more common prey types, resulting in reduced predation rates on rare prey types. Such functional responses may arise through predators developing a search image for the more common prey, leading to switching between prey types as one becomes more common. Such a Type III functional response may be modeled as

$$f_i(I_U, I_I) = \frac{I_i^2}{1 + I_U^2 \delta_U h_U + I_U^2 \delta_I \theta h_I}.$$

Under this scenario, the predator is able to survive at a much lower reproductive rate (lower e) in the absence of the parasite than for a Type II response (compare the horizontal line in Fig. 6 with Fig. 2). Second, considerable levels of parasite manipulation are required in order for the parasite to persist; the very low prevalence of infected intermediate hosts means that without high degrees of manipulation they are preyed upon very infrequently, due to the predator's aversion to rare prey. However, if a parasite is able to manipulate its host sufficiently, parasite persistence may occur even for very high degrees of host reproduction (high e values), which would otherwise not allow parasite persistence assuming a Type II functional response.

ALTERNATIVE FORMS OF MANIPULATION

Parasites may not just increase the conspicuousness of infected prey, but may also make infected prey easier to catch by the predator, thereby reducing the handling time of infected prey (i.e., $h_I < h_U$). For example, parasites which inhibit the intermediate host's escape response, such as trematode metacercariae that encyst in the eyes of fish causing blindness (Seppala et al. 2005) or debilitating *Echinococcus* infections of ungulates which

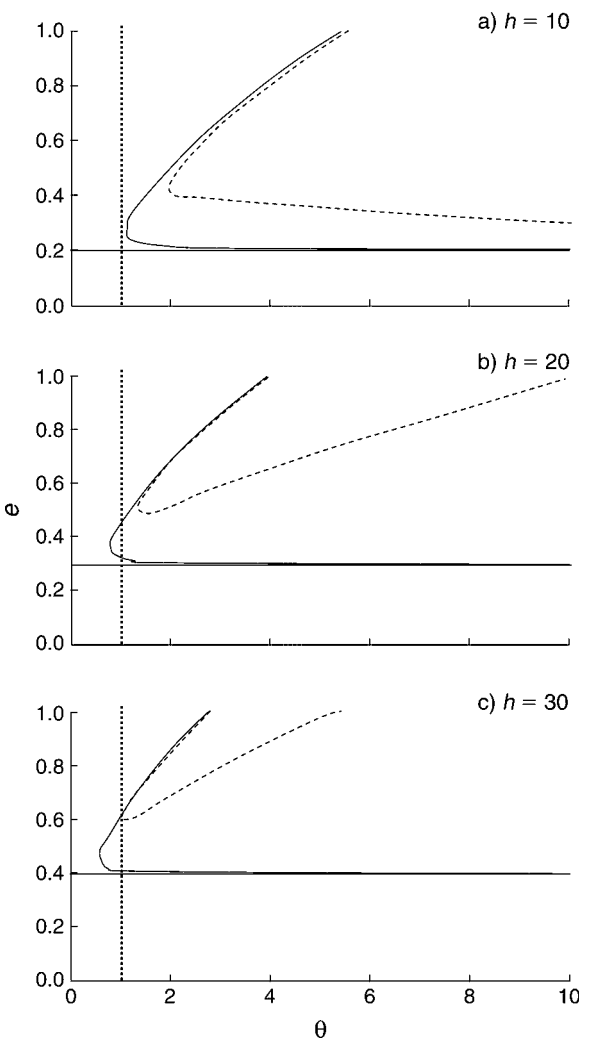

FIG. 5. Parameter space for a Type II functional response, showing the effect of varying the predator's "handling time" (h). When $h = 0$, the Type I functional response is recovered. The dotted vertical line shows where infecteds have the same attractiveness as uninfecteds (i.e., no manipulation). Dynamical regions and parameters correspond to those in FIG. 2.

prevent escape from predators (Moore 2002), are likely to reduce the time taken for the predator to capture the prey. In Appendix B, we show that such manipulations facilitate parasite persistence at high predator reproductive rates. Hence, parasites are more likely to persist with highly fecund predatory definitive hosts if they decrease the time taken for those predators to capture their prey than when they increase the exposure rate of those prey to the predators.

Burden-dependent host manipulation

The models described above all assume a microparasite, such that hosts are either infected or uninfected, and the relative conspicuousness of the two prey types is determined by a single, fixed parameter (θ). However, many of the parasites that are believed to manipulate their hosts are macroparasitic helminths (Moore 2002), which requires a different modeling framework that keeps track of how many parasites hosts are infected with. This becomes important when we consider how host manipulation occurs and, specifically, if it increases with increasing worm burdens (i.e., the degree of manipulation is burden dependent and all worms contribute equally to the overall conspicuousness of the host). In this case, the corresponding model (similar to Dobson and Keymer 1985, Dobson 1988) is

$$\begin{aligned}
\frac{dI}{dt} &= Ir(1 - qI) - \alpha_{\mathrm{I}} P_{\mathrm{I}} - \delta D(1 + \theta P_{\mathrm{I}}) \\
\frac{dD}{dt} &= e\delta D(I + \theta P_{\mathrm{I}}) - \alpha_{\mathrm{D}} P_{\mathrm{D}} - d_{\mathrm{D}} D \\
\frac{dP_{\mathrm{I}}}{dt} &= \frac{\nu\lambda DI}{\mu} - P_{\mathrm{I}}[\Gamma_{\mathrm{I}} + \delta D(1 + \theta\ )] - \frac{P_{\mathrm{I}}^2(k_{\mathrm{I}} + 1)(\alpha_{\mathrm{I}} + \beta D\theta)}{k_{\mathrm{I}} I} \\
\frac{dP_{\mathrm{D}}}{dt} &= D\delta P_{\mathrm{I}}\left[1 + \theta + \frac{\theta P_{\mathrm{I}}(k_{\mathrm{I}} + 1)}{k_{\mathrm{I}} I}\right] - P_{\mathrm{D}}\Gamma_{\mathrm{D}} - \frac{P_{\mathrm{D}}^2(k_{\mathrm{D}} + 1)\alpha_{\mathrm{D}}}{k_{\mathrm{D}} D}
\end{aligned} \tag{3}$$

where P_{I} is the density of parasites in intermediate hosts (I) and P_{D} is the density of parasites in the predatory definitive hosts (D). The Γ_{I} and Γ_{D} terms represent the net loss of parasites within infected prey and predators respectively due to density-independent processes ($\Gamma_i = \mu_i + \alpha_i + \mathrm{b}_i$ where μ_i is the mortality rate of parasites within host i, α_i is the rate of parasite induced host mortality of host i, and b_i is the background mortality rate of host i). The k_{I} and k_{D} terms are parameters for the negative binomial distribution describing the degree of aggregation of parasites in the prey and predator populations, respectively. In this model, prey suffer a baseline predation rate δ, which is increased by θ times the number of parasites they have (predation occurs at per capita rate $\delta D(1 + \theta M_{\mathrm{I}})$, where M_{I} is the mean parasite burden per prey individual, P_{I}/I). Clearly, when $\theta = 0$, no manipulation occurs and all prey individuals suffer the same predation risk regardless of their parasite burden.

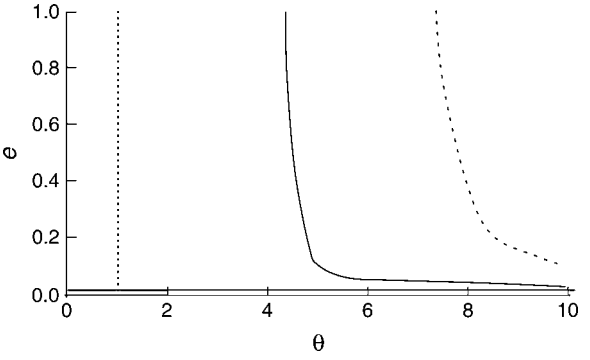

FIG. 6. Parameter space for a Type III functional response. Dynamical regions and parameters correspond to those in Fig. 2.

Stability analyses reveal the dynamics to be qualitatively similar to those seen for the simpler microparasite model described in Eq. 1, with parasite persistence only being possible for intermediate levels of predator reproduction, and being facilitated by increasing degrees of host manipulation (Fig. 7). However, what is particularly striking is the large region of parameter space where the predator–prey–parasite community persists, but with cyclical dynamics; only very low predator reproductive rates allow stable persistence of the parasite within the host community. Such oscillatory tendencies have previously been observed for similar models, which show that additional density-dependent constraints, for instance acting on adult parasite mortality within the definitive host, are needed in order to stabilize the dynamics (Dobson and Keymer 1985, Dobson 1988). As before, both parasite and host life history parameters are important in determining the dynamics of the community, with high levels of host manipulation allowing the persistence of short-lived, or highly virulent parasites that would otherwise be driven to extinction (Fig. 7b–d).

Discussion

Parasites can play an important, but often overlooked role in shaping population and community dynamics and maintaining biodiversity (Holt and Pickering 1985, Hudson et al. 1998, 2002, Dobson 2004). However, we can only truly assess the impact parasites have on ecosystems and predict their spread through ecological communities if we understand the processes underlying parasite transmission. For trophically transmitted parasites, which are transmitted up the food chain from intermediate host species preyed upon by their definitive host predators, this means we need to consider the behavior of both the predator and the prey, the latter of which may be affected by parasite-induced host manipulation. In this paper, we show that the degree of this manipulation can greatly alter the dynamics and stability of the predator–prey community, but the precise outcome of the interaction depends on both the form of the manipulation and the nature of the predator's functional response.

In qualitative terms, the presence of a trophically transmitted parasite and the degree of manipulation do not affect whether a predator is able to persist with its prey species, but it can have an impact on the quantitative abundance of the predator and prey and also on their dynamics. Manipulation has a tendency to induce oscillations in population densities, which may be of sufficient amplitude that either the predator or the prey densities become perilously close to zero; in the stochastic reality of the real world, one of these species may be driven to extinction as a result of the level of parasite-induced host manipulation. This tendency to induce cyclical dynamics is particularly apparent for macroparasites, where the degree of manipulation increases according to the burden of parasites per host

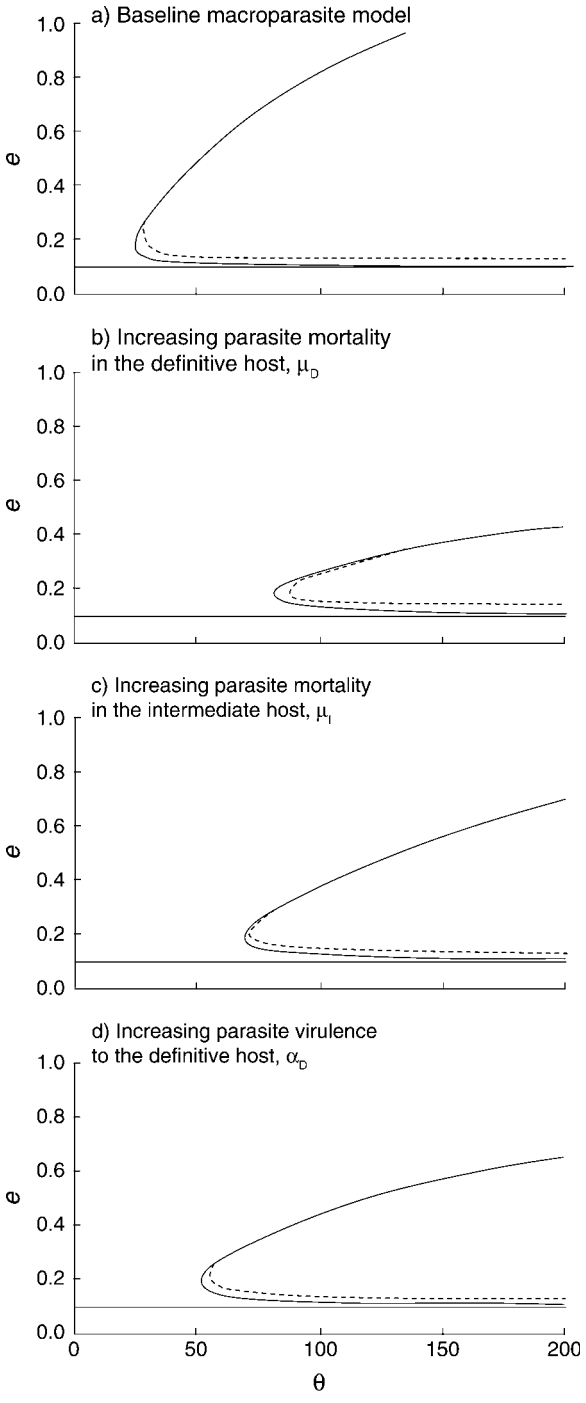

Fig. 7. Parameter space for the macroparasite model, where the degree of host manipulation increases linearly (at rate θ) with the mean burden of parasites per host, showing the effect of varying different parameters in the model. Dynamical regions and parameters correspond to those in Fig. 2.

(Dobson and Keymer 1985, Dobson 1988). For example, in roaches (*Rutilus rutilus*) infected by the cestode *Ligula intestinalis*, both the number and size of the parasite burden affect the host's proximity to the shore and consequently its likelihood of being predated by the

parasite's definitive bird host (Brown et al. 2001). The majority of examples of host manipulation involve macroparasites, such as acanthocephalans, cestodes, and digeneans (Moore 2002). Therefore the tendency for these parasites to induce cycles in their intermediate and definitive host populations should not be overlooked as a major factor driving the population dynamics of such predator–prey communities.

Although host manipulation may be a powerful force underlying predator–prey dynamics, the detection of manipulation in the field may be very difficult, as is proving that manipulations are directly induced by the parasite, rather than being a side effect of parasitism (Brown et al. 2001, 2002, Franz and Kurtz 2002, Tompkins et al. 2004). One of the major problems with identifying manipulation in the field is that typically the parasite can only be found at very low levels amongst the intermediate host population. For instance, Moore (1983) found the prevalence of acathocephalan cystacanth infection among isopods in the field was less than 0.4%. However, subsequent estimates of infection rates of starlings which preyed upon the isopods were significantly lower than those observed (Moore 1983). As the model presented here showed, prevalences of infection in prey may be vanishingly small, while prevalences in the predator population may be very high. Furthermore, the resulting impact of the manipulating parasite causes the system to exhibit high-amplitude oscillations. Therefore, just because parasites are barely detectable in the prey species, this does not mean that manipulation is not important for parasite transmission, or that the parasite does not greatly affect community dynamics.

Clearly the life histories of the predators and their prey will be important in determining the persistence of the parasite and the optimal level of host manipulation. High levels of manipulation are needed to enable parasite persistence when the prey have low fecundity or when the predators have short handling times of captured prey. However, if the degree of manipulation is too high, the parasite population begins to decline. Furthermore, extreme oscillations may occur, during which extinction of either predator or prey may occur, resulting in elimination of the parasite. Hence, there may be ecological costs constraining the optimal level of manipulation, resulting in a trade-off between increasing transmission rates between hosts and driving host population sizes down to levels where environmental stochasticity could render the host (and therefore the parasite) extinct.

Following on from this, the ecological models presented here could provide an ideal framework for exploring the evolution of life history strategies within predator–prey–parasite communities. Using a similar framework, Lafferty (1992) adopted an economic approach to determine the conditions under which predators should consume infected prey. This may be extended to explore, for instance, the evolution of prey processing, where the handling time component occurs mainly for the predator to remove parasites from the prey (Stephens and Krebs 1986, Ydenberg 1998, Rands et al. 2000). Such foraging decisions by the predator will then affect the evolution of parasite and intermediate host strategies, requiring a game-theoretic approach. Similarly, these models could be extended to consider how changes in both parasite and host energy reserves could shape behavior (Thomas et al. 2002, Fenton and Rands 2004). Given that manipulation may well occur in medically important parasites (e.g., Lacroix et al. 2005), gaining a realistic understanding of how parasite manipulation can affect such population processes is vital if we are to understand the effects of these parasites from both ecological and medical perspectives.

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Literature Cited

Anderson, R. M., and R. M. May. 1978. Regulation and stability of host–parasite population interactions. I. Regulatory processes. Journal of Animal Ecology **47**:219–247.

Anderson, R. M., and R. M. May. 1981. The population dynamics of microparasites and their invertebrate hosts. Philosophical Transactions of the Royal Society of London, Series B **291**:451–524.

Bethel, W. M., and A. J. Holmes. 1973. Altered evasive behavior and responses to light in amphipods harboring acanthocephalan cystacanths. Journal of Parasitology **59**: 945–956.

Bowers, R. G., and M. Begon. 1991. A host–host–pathogen model with free-living infective stages, applicable to microbial pest control. Journal of Theoretical Biology **148**:305–329.

Brown, S. P. 1999. Cooperation and conflict in host-manipulating parasites. Proceedings of the Royal Society of London, Series B **266**:1899–1901.

Brown, S. P., G. Loot, B. T. Grenfell, and J. F. Guégan. 2001. Host manipulation by *Ligula intestinalis*: accident or adaptation? Parasitology **123**:519–529.

Brown, S. P., G. Loot, A. Teriokhin, A. Brunel, C. Brunel, and J. F. Guegan. 2002. Host manipulation by *Ligula intestinalis*: a cause or consequence of parasite aggregation? International Journal for Parasitology **32**:817–824.

Damsgaard, J. T., K. N. Mouritsen, and K. T. Jensen. 2005. Surface activity of *Corophium volutator*: a role for parasites? Journal of Sea Research **54**:176–184.

Dobson, A. P. 1988. The population biology of parasite-induced changes in host behavior. Quarterly Review of Biology **63**:139–165.

Dobson, A. 2004. Population dynamics of pathogens with multiple host species. American Naturalist **164**:S64–S78.

Dobson, A. P., and P. J. Hudson. 1992. Regulation and stability of a free-living host–parasite system: *Trichostrongylus Tenuis* in Red Grouse. 2. Population models. Journal of Animal Ecology **61**:487–498.

Dobson, A. P., and A. E. Keymer. 1985. Life history models. Pages 347–384 *in* D. W. T. Crompton and B. B. Nickol, editors. Biology of the Acanthocephala. Cambridge University Press, Cambridge, UK.

Fenton, A., and S. A. Rands. 2004. Optimal parasite infection strategies: a state-dependent approach. International Journal for Parasitology **34**:813–821.

Franz, K., and J. Kurtz. 2002. Altered host behaviour: manipulation or energy depletion in tapeworm-infected copepods? Parasitology **125**:187–196.

Getz, W. M., and J. Pickering. 1983. Epidemic models: thresholds and population regulation. American Naturalist **121**:892–898.

Holling, C. S. 1959. Some characteristics of simple types of predation and parasitism. Canadian Entomologist **91**:385–398.

Holt, R. D. 1983. Optimal foraging and the form of the predator isocline. American Naturalist **122**:521–541.

Holt, R. D., and J. Pickering. 1985. Infectious disease and species coexistence: a model of Lotka-Volterra form. American Naturalist **126**:196–211.

Hudson, P. J., A. P. Dobson, and D. Newborn. 1998. Prevention of population cycles by parasite removal. Science **282**:2256–2258.

Hudson, P. J., A. Rizzoli, B. T. Grenfell, H. Heesterbeek, and A. P. Dobson. 2002. The ecology of wildlife diseases. Oxford University Press, Oxford, UK.

Keeling, M. J. 1999. The effects of local spatial structure on epidemiological invasions. Proceedings of the Royal Society of London Series B **266**:859–867.

Lacroix, R., W. R. Mukabana, L. C. Gouagna, and J. C. Koella. 2005. Malaria infection increases attractiveness of humans to mosquitoes. Plos Biology **3**:e298.

Lafferty, K. D. 1992. Foraging on prey that are modified by parasites. American Naturalist **140**:854–867.

McCallum, H., N. Barlow, and J. Hone. 2001. How should pathogen transmission be modelled? Trends in Ecology and Evolution **16**:295–300.

Moore, J. 1983. Responses of an avian predator and its isopod prey to an acanthocephalan parasite. Ecology **64**:1000–1015.

Moore, J. 1984. Altered behavioral responses in intermediate hosts–an acanthocephalan parasite strategy. American Naturalist **123**:572–577.

Moore, J. 2002. Parasites and the behavior of animals. Oxford University Press, New York, New York, USA.

Morand, S., and E. A. Gonzalez. 1997. Is parasitism a missing ingredient in model ecosystems? Ecological Modelling **95**:61–74.

Poulin, R. 1994. The evolution of parasite manipulation of host behaviour: a theoretical analysis. Parasitology **109**:S109–S118.

Rands, S. A., A. I. Houston, and C. E. Gasson. 2000. Prey processing in central place foragers. Journal of Theoretical Biology **202**:161–174.

Seppala, O., A. Karvonen, and E. T. Valtonen. 2005. Manipulation of fish host by eye flukes in relation to cataract formation and parasite infectivity. Animal Behaviour **70**:889–894.

Stephens, D. W., and J. R. Krebs. 1986. Foraging theory. Princeton University Press, Princeton, New Jersey, USA.

Swinton, J., J. Harwood, B. T. Grenfell, and C. A. Gilligan. 1998. Persistence thresholds for phocine distemper virus infection in harbour seal *Phoca vitulina* metapopulations. Journal of Animal Ecology **67**:54–68.

Thomas, F., S. Adamo, and J. Moore. 2005. Parasitic manipulation: where are we and where should we go? Behavioural Processes **68**:185–199.

Thomas, F., S. P. Brown, M. Sukhdeo, and F. Renaud. 2002. Understanding parasite strategies: a state-dependent approach? Trends in Parasitology **18**:387–390.

Thomas, F., F. Renaud, T. Demee, and R. Poulin. 1998. Manipulation of host behaviour by parasites: ecosystem engineering in the intertidal zone? Proceedings of the Royal Society of London, Series B **265**:1091–1096.

Tompkins, D. M., K. N. Mouritsen, and R. Poulin. 2004. Parasite-induced surfacing in the cockle *Austrovenus stuchburyi*: adaptation or not? Journal of Evolutionary Biology **17**:247–256.

Turner, J., M. Begon, and R. G. Bowers. 2003. Modelling pathogen transmission: the interrelationship between local and global approaches. Proceedings of the Royal Society of London Series B **270**:105–112.

Ydenberg, R. C. 1998. Behavioral decisions about foraging and predator avoidance. Pages 343–378 *in* R. Dukas, editor. Cognitive ecology: the evolutionary ecology of information processing and decision making. University of Chicago Press, Chicago, Illinois, USA.

APPENDIX A

Stability analysis of the Type I microparasite manipulation model (*Ecological Archives* E087-172-A1).

APPENDIX B

Model in which manipulation reduces the predator's handling time (*Ecological Archives* E087-172-A2).