J. theor. Biol. (1998) **193,** 485–495
Article No. jt980717

The Coexistence of Competing Parasites. Part II—Hyperparasitism and Food Chain Dynamics

ROBERT D. HOLT* AND MICHAEL E. HOCHBERG†

**Natural History Museum, Department of Systematics and Ecology, University of Kansas, Lawrence, KS* 66045, *U.S.A. and †Institut d'Ecologie, Université Pierre et Marie Curie, Ecole Normale Superieure, 7 quai St. Bernard, Bât. A, 7ème étage, CC*237, 75252 *Paris Cedex* 05, *France*

(*Received on* 29 *April* 1997, *Accepted in revised form on* 20 *March* 1998)

Hyperparasitism is a widespread interaction in natural communities, but has to date received little attention in the theoretical literature. In this paper, we compare canonical models for food chains (resource–prey–predator systems) and host–parasite–hyperparasite interactions. We focus on microparasites, so the dynamical variables are the abundances of host individuals in different classes (e.g. with or without a particular parasite), and assume that the parasite is the only factor regulating a host population. Analysis of a "donor-controlled" model in which the primary parasite regulates host population growth, but with no additional demographic impact of the hyperparasite, suggests that intrinsic growth rate r of the host population is a fundamental parameter governing persistence of the hyperparasite. We then examine a model in which the hyperparasite can affect host births, deaths, and rate of recovery from the primary parasite. A wide range of outcomes are possible. For instance, hyperparasites can stabilize inherently unstable host–parasite systems, or destabilize stable systems. Persistence at a stable equilibrium often requires that the host intrinsic growth rate r lie within defined bounds; at low r, the hyperparasite may not be able to persist (in stable systems), whereas at high r the system is unstable and the host population grows in an unbounded fashion. We conclude by sketching directions for future work, and suggesting some possible practical implications of our results.

Introduction

Obligate hyperparasitism is an interaction wherein one species of parasite is parasitized by a second species of parasite, which cannot on its own parasitize an uninfected host. We here use the term "parasitism" in an expansive sense, for instance to include the "kleptoparasitism" which may arise when one parasite differentially attacks and in effect "steals" hosts already infected by another parasite (the primary parasite), as well as systems where one parasite lives intimately within the body of the primary parasite. Though pervasive in nature (e.g. Cook & Baker, 1983; Levin & Bull, 1996), this interaction has received rather scant attention in the theoretical literature of host–parasite interactions (though for early studies involving insect parasitoids, see Beddington & Hammond, 1977; Hassell, 1978; May & Hassell, 1981), at least compared with the enormous theoretical literature on single parasite–host dynamics (see, e.g. Grenfell & Dobson, 1995; Anderson & May, 1991). There is an increasing appreciation of the need to consider multispecies dynamics when examining host–parasite interactions (Hochberg & Holt, 1990; Begon & Bowers, 1995), and more broadly the factors organizing ecological communities (Holt, 1996a). The dynamical interactions between any pair of species, including host and parasite, can be strongly modulated by effects of other species (Begon *et al.*, 1996).

In this paper, we examine some aspects of the dynamics of host–parasite–hyperparasite systems. Our aim is to compare the dynamics of these systems to the more familiar dynamics of food chains in ecological communities, highlighting both similarities

0022–5193/98/150485 + 11 $30.00/0

and differences. An obligatory hyperparasite is in some ways akin to a top predator in a food chain. For instance, energy and nutrients may flow from host, to parasite, to hyperparasite, much as energy and matter flow up food chains from plants, to herbivores, to carnivores (Hutchinson, 1959). With specialized parasitism, as with trophic specialization in a food web, there is a kind of sequential dependency among species, which can lead to regular patterns in community assembly even in the absence of more complex population dynamical effects (Holt, 1996b). Moreover, if parasites negatively influence demographic parameters in their hosts, hyperparasitism can alter the impact of a primary parasite upon the host, much as predators can reduce the impact of prey upon a basal resource population (the "top-down" effects of food web ecology).

But there are also obvious differences between the two systems, which may have important dynamical consequences. For instance, if an individual host mounts a defensive reaction against a primary parasite and recovers, that reaction can incidentally remove any hyperparasites within the primary parasite, as well. This response does not correspond closely to any obvious feature in standard food chains. Moreover, parasitized hosts may still give birth, or recover from parasitism, thus providing a source of recruitment of healthy individuals into host populations that does not have any ready parallel in prey recruitment in predator–prey systems. Hyperparasitism may, at times, increase the impact of such reproduction, by allowing hosts to recover from deleterious effects of the primary parasite. Depending on the natural history of the within-host dynamics, it is also possible for a hyperparasite to impair more severely host recruitment or recovery, compared with just the effects of the primary parasite. As we will see below, the direction of the impact of hyperparasitism on host demography has a major effect on the stability of systems with hyperparasitism. One crucial difference we will observe between a host–parasite–hyperparasite system and a classic food chain is that the existence of a persistent equilibrium in standard food chain models requires direct density-dependence in births or deaths (usually assumed to be present in the basal species), whereas host–parasite–hyperparasite systems can stably persist without any such direct density-dependence in the birth or death rates of either healthy or infected hosts.

This paper constitutes the second part of a study on the coexistence of parasites. The first part focused on the role of interference between two microparasite species competing for the same host (Hochberg & Holt, 1990). The model we examined there permitted one pathogen strain to infect either hosts previously infected with another strain (which was then supplanted) or healthy, susceptible hosts. We found that parasite coexistence is governed by an interplay between exploitative competition (driven by direct transmission to healthy hosts) and interference competition (arising from mixed infections). Depending upon the relative strengths of these two effects, it was shown that a wide range of outcomes could occur; two parasite species may coexist at a stable or unstable equilibrium point; or, one parasite species may always competitively exclude the other; or, alternative states exist in which either parasite can dominate (with the winner dependent on initial population densities of the host and both parasites). In a broad sense, this system exhibits all the dynamical features of omnivory or intraguild predation (Holt & Polis, 1997), a class of interspecific interactions in which a top predator feeds on multiple trophic levels.

We begin by briefly reviewing well-known results of the dynamics of a simple Lotka–Volterra food chain. We than examine a "donor-controlled" system, in which the hyperparasite is in effect a commensal of the host–primary parasite system. We next consider a limiting case of the model explored in Hochberg & Holt (1990), corresponding to a host–parasite–hyperparasite chain where the hyperparasite has substantial demographic impacts, and describe in detail how hyperparasitism modulates the stability of the system. We conclude by summarizing key similarities and differences between standard food chain models and models of hyperparasitism, and briefly discuss some potential implications of hyperparasitism for biological control.

A Simple Food Chain Model

The canonical food chain model involves a chain of predator–prey interactions, where a basal resource population sustains an intermediate predator population, which in turn is consumed by a top predator. If we assume that the basal species experiences logistic growth, and linear functional responses linking each successive predator–prey interaction, the food chain dynamics can be described by the following Lotka–Volterra model (e.g. Hallam, 1986):

$$\begin{aligned} \frac{dR}{dt} &= R[r(1 - R/K) - aN] \\ \frac{dN}{dt} &= N[baR - m - a'P] \\ \frac{dP}{dt} &= P[b'a'N - m'] \end{aligned} \tag{1}$$

where R, N, and P are, respectively, the densities of the basal prey, intermediate predator, and top predator; r and K are the intrinsic growth rate and carrying capacity of the basal prey; a and a' are attack rates, respectively, of the intermediate predator on the basal prey, and the top predator on the intermediate predator; b and b' convert prey consumption into predator births; and, m and m' are density-independent mortality rates. The basal prey, when alone, equilibrates at $R = K$. The equilibrium of the basal prey and just the intermediate predator is $R' = m/ab$, $N' = (r/a)(1 - m/abK)$. The intermediate predator only persists if $K > m/ab$. The full chain has an equilibrium of $R^* = K(1 - aN^*/r)$, $N^* = m'/a'b'$, and $P^* = (baR^* - m)/a'$. This equilibrium exists if two conditions hold: $K > (m/ab)(1 - aN^*/r)^{-1}$, and $m'/a'b' < r/a$. If these two- and three-species equilibria exist, they are asymptotically stable (Hallam, 1986).

This simple model illustrates key properties of more general food chain models (e.g. Oksanen *et al*., 1981; Logofet, 1993). Comparing persistence conditions for the two- and three-link systems, it is clear that adding a top predator requires the basal prey to have a larger K than required for just the intermediate predator to persist. Hence, along a gradient in K (i.e. a gradient in basal prey density-dependence) one should find longer food chains only at higher K, where density-dependence is weak in the basal species (Pimm, 1982; Oksanen *et al*., 1981). However, large K is not sufficient for the full chain to persist; r must also be sufficiently large (ensuring enough intermediate predators sustained by the basal resource to support the top predator).

A final observation about the above food chain model is that direct density-dependence in recruitment of the basal species is necessary for a stable equilibrium to exist. Assuming that the top predator is absent, if one lets $K \to \infty$ then the two-link model reduces to the classical, neutrally stable Lotka–Volterra model:

$$\frac{dR}{dt} = R[r - aN]$$
$$\frac{dN}{dt} = N[baR - m] \qquad (2)$$

If the parameters are all positive, then the equilibrium $N' = r/a$, $R' = m/ab$ always exists, and the predator limits prey population growth. However, solutions of the Lotka–Volterra predator–prey model are neutrally stable, and small changes in assumptions can have radical effects on system behavior (May, 1973). Nonetheless, the equilibrial abundance expressions do capture aspects of more complex, realistic models. For instance, increased r translates into increased predator abundance, whereas increases in predator efficiency (a) depress both predator and prey numbers.

If $m'/a'b' > r/a$, the top predator cannot invade a system comprised of the basal prey and intermediate predator. If, instead, $m'/a'b' < r/a$, then the top predator can invade. In this case, however, there is no finite equilibrium for the system defined by eqns (1) in the limit $K \to \infty$—abundances of the basal species and top predator are unbounded, and the system explodes exponentially (Hallam, 1986). Similar effects arise in any food chain model without direct density-dependence (Logofet, 1993).

Thus, in the above food chain model, persistence at an equilibrium with finite abundances requires direct density-dependence in the basal species. Moreover, the food chain persists only if the intrinsic growth rate (r) of the basal resource population is sufficiently high. We now compare these results with comparable host–parasite and host–parasite–hyperparasite systems.

A Canonical Host–parasite Model

We suggest that the canonical host–microparasite model, appropriate to systems where hosts do not have an immune response, is the basic differential equation model of Anderson & May (1986). This model embodies several basic assumptions:

(1) the host is regulated (if at all) by parasitism. Hence, in the absence of the primary parasite, the population of susceptible hosts of density S grows exponentially. These hosts have per capita rates of birth and death of a and b, respectively, so their net intrinsic rate of increase is $r = a - b$;

(2) the rates of parasitism are described by mass action terms βSI, where β is the coefficient of direct transmission of the primary parasite (of density I) from infected to susceptible hosts;

(3) once infected, a host may either recover to the susceptible state at a per capita rate of v, give birth to susceptible offspring at a per capita rate of a, or die from natural or disease-induced causes, at per capita rates of b or m, respectively. The parameter d denotes the net intrinsic loss rate of infected hosts (i.e. $d = m + b + v$). The parameter e represents the net contribution of infected hosts to the growth of the susceptible fraction of the host population (i.e. $e = v + a$).

With these assumptions, the model takes the following form:

$$\frac{dS}{dt} = rS + eI - \beta IS$$
$$\frac{dI}{dt} = \beta IS - sI \quad (3)$$

The equilibrium $S^* = d/\beta$, $I^* = rd/[\beta(d-e)]$ exists and is stable, provided $1 > e/d$ (see Holt & Pickering, 1985).

The above model is a close analogue for host–parasite models of the Lotka–Volterra predator–prey model [eqns (2) above]. Some parallels are apparent from inspection of the expressions for equilibrial abundances. For instance, in model (2), an increase in the predator attack parameter a depresses the abundance of both predator and prey (due to overexploitation). Likewise, in the host–parasite model, an increase in the transmission coefficient β reduces both host and parasite. Similarly, an increase in r indirectly enhances abundance of the natural enemy (predator or parasitized hosts), with no effect on prey or host abundance. In both models, there is assumed no direct density-dependence in the basal population (prey or healthy hosts).

The key difference between the models is the eI term, describing how infected hosts can give rise to uninfected hosts (via birth or recovery); when e approaches 0, the host–pathogen model converges on the neutrally stable Lotka–Volterra model. This difference has crucial effects on dynamics. First, it can weaken the ability of the parasite to regulate the host population. If $e/d > 1$, then the host population grows in an unbounded fashion (presumably until limited by some factor other than parasitism, not included in the above model). Second, as long as $0 < e/d < 1$, the dynamics converge to a stable equilibrium. In effect, given some births or recovery, parasitized hosts provide a kind of time-delayed recruitment into the subpopulation of susceptible hosts, buffering changes in host numbers. A general message of this model is that in some circumstances, parasitism on its own can regulate host numbers to a stable equilibrium, independent of any other regulatory factors (e.g. food limitation).

The demographic ratio e/d thus plays a defining role in host–parasite dynamics. We will see that this ratio also characterizes the behavior of systems with hyperparasites. If parasitism affects death rates, but not birth rates, the condition for population regulation may be written as $r < m$, where m is additional mortality from parasitism (Anderson & May, 1986). This simple inequality suggests that pathogens imposing a given level of mortality are likely to stably regulate only hosts with a sufficiently low intrinsic growth rate. However, expressing the condition for persistence in this way to a degree obscures the fact that what matters in determining stability in this canonical model is the intrinsic growth rate of the parasitized portion of the host population, and not the growth rate of unparasitized hosts. If $e/d < 1$, the birth rate of parasitized individuals is less than their own death rate, and the population is regulated by parasitism, irrespective of the value for r. As will be seen shortly, hyperparasitism changes this conclusion; host r plays a vital role in determining the persistence and stability of host–parasite–hyperparasite systems.

A Donor-controlled System

One limiting case of hyperparasitism involves "donor-controlled" (DeAngelis, 1992) systems, in which the hyperparasite is an obligate commensal of the primary parasite, with no effect on host reproduction or mortality beyond those imposed by the primary parasite alone, and no effect on persistence or transmission of the primary parasite. In the above canonical host–parasite model, we divide the infected portion of the host population into two parts, such that $I = I_1 + I_2$, where I_1 and I_2, respectively, denote hosts parasitized with only the primary parasite, and hosts parasitized with both primary and hyperparasites. To complete the model, we need to include an equation for the dynamics of hosts with hyperparasites. We assume that a mass action law describes the rate at which hosts with primary parasites acquire hyperparasites, with parameter δ scaling the transmission rate of hyperparasites. The following equations describe the dynamics of a tritrophic, donor-controlled, host–parasite–hyperparasite system:

$$\frac{dS}{dt} = rS + e(I_1 + I_2) - \beta S(I_1 + I_2)$$
$$\frac{dI_1}{dt} = \beta S(I_1 + I_2) - dI_1 - \delta I_1 I_2$$
$$\frac{dI_2}{dt} = \delta I_1 I_2 - dI_2$$

In this model, the subscript 1 is for hosts which have just the primary parasite, where subscript 2 denotes hosts which have both the primary parasite and hyperparasite. Note that adding the equations for the two infected components leads to the canonical host–parasite model (3), because the hyperparasite has no influence on the demographic consequences of

parasitism imposed upon the host by the primary parasite. An equilibrium clearly only exists if $d/e > 1$. Assuming this is the case, the equilibrial density of hosts infected with just the primary parasite is $I_1^* = d/\delta$, and of hosts with the hyperparasite is $I_2^* = (rd\delta - d\beta(d-e))/\delta\beta(d-e)$. The hyperparasite persists if

$$r\delta > \beta(d-e)$$

This inequality is also the condition for invasion by the hyperparasite, given that the primary parasite is present at its equilibrial prevalence.

This donor-controlled model predicts that the presence of the hyperparasite depends on a combination of host properties, parameters describing the host–primary parasite interaction, and the rate of transmission of the hyperparasite itself. Hyperparasites can only persist in donor-controlled systems if host growth rates are sufficiently large. The more pathogenic (higher d, lower e) is the primary parasite, the more difficult it is for a hyperparasite to persist. Moreover, increases in transmission of the primary parasite, paradoxically, make it harder for the hyperparasite to persist. This reflects a kind of overexploitation of the host by the primary parasite, parallel to the effect of the attack parameter a in the food chain model noted above.

Many of these features also arise in models in which hyperparasitism influences host demographic parameters.

Two Parasites Competing for a Single Host

More generally, one might expect that hyperparasites could influence the demographic parameters of infected hosts, and the transmission success of the primary parasite in multiply infected hosts. Hyperparasitism involves two species of parasite, both sustained (directly or indirectly) by the same host individuals, and so in a broad sense competing for the same packet of host resources. For reasons that will become apparent shortly, it is useful to review briefly the basic features of a model for multi-parasite competitive interactions explored by Hochberg & Holt (1990) before going on to consider the special case of obligate hyperparasitism. This model permits either parasite species to infect uninfected, susceptible hosts. The model is a system of ordinary differential equations describing changes in the abundance of susceptible host (of density S), hosts infected with parasite species 1 (of density I_1), and hosts infected with parasite species 2 (of density I_2), as follows:

$$\begin{aligned}
\frac{dS}{dt} &= rS + e_1 I_1 + e_2 I_2 - \beta_1 I_1 S - \beta_2 I_2 S \\
\frac{dI_1}{dt} &= \beta_1 I_1 S - d_1 I_1 - \delta I_1 I_2 \qquad (4) \\
\frac{dI_2}{dt} &= \beta_2 I_2 S - d_2 I_2 + \delta I_1 I_2
\end{aligned}$$

The assumptions leading to this model include those for the canonical Anderson–May model presented above. In addition, we assume that when a given host individual is jointly infected with parasite 1 and 2, either parasite 1 is rapidly displaced by parasite 2, or, if it persists within the host, it is dynamically irrelevant, in that it is unable to produce propagules to infect healthy hosts. The rate of infection by parasite 2 of hosts already infected by the primary parasite 1 is given by a mass action term, scaled by the transmission parameter δ. The parameter δ can be thought of as a form of predation between the parasites, since cross-transmission entails the effective elimination of reproductive output from the primary parasites in multiply infected hosts.

Hochberg & Holt (1990) discuss in some detail the conditions for coexistence between parasites in this model. One general necessary condition is that two parasite species can only coexist if one is the superior exploitative competitor (i.e. at transmission), whilst the other parasite is the superior interference competitor (i.e. at cross-transmission). A further necessary condition for parasite coexistence is that $e_2/d_2 < e_1/d_1$, so that the parasite species which can successfully infect hosts already infected with the other parasite, must also inflict greater demographic damage on the host.

The quantity e_i/d_i will also prove to be crucial in the following analysis of the dynamics of hyperparasitism. When $e_i/d_i < 1$, we say that parasite i is *potentially regulatory*, which is to say that in model (4), where each $\beta_i > 0$, parasite i could by itself regulate the host population, without any other regulatory factors being involved. By contrast, when $e_i/d_i > 1$, we say that a parasite is *non-regulatory*, in that some other factor is required to regulate the host population (which is not to say that the parasite does not play a contributory role, only that on its own it is insufficient to regulate host growth to an equilibrium).

Obligate Hyperparasitism

The special case of the multiparasite model (4) comparable to a simple food chain arises when one of the parasites is an obligate hyperparasite. Formally, we say that parasite 2 is an obligate hyperparasite if $\beta_2 = 0$ and $\delta > 0$, so the hyperparasite can only parasitize individuals already infected with the primary parasite. This dynamical definition of hyperparasitism encompasses several distinct biological scenarios. For instance, the hyperparasite may actually be a superior competitor within individual hosts, supplanting the primary parasite. Or, the hyperparasite may not supplant the primary parasite, but instead subsist within it and suppress transmission by the primary parasite to fresh, uninfected hosts. Because the model assumes complete suppression of transmission of primary parasites from hosts with the hyperparasite, it represents an opposite limiting case of hyperparasitism to the assumptions of the donor-controlled model considered above, where the hyperparasite had no effect upon transmission dynamics of the primary parasite.

It is clear that the hyperparasite (species 2) cannot regulate the host in the absence of the primary parasite (regardless of the value of e_i/d_i), and indeed it faces inevitable extinction if the primary parasite, species 1, is absent. There are two basic questions we can ask about obligate hyperparasitism. In what circumstances can the hyperparasite persist? Given that it persists, what is the effect of the hyperparasite on the stability of the original primary parasite–host association?

The system is at equilibrium when

$$\begin{aligned} I_1^* &= d_2/\delta, \\ I_2^* &= \frac{\frac{\beta_1 d_2}{\delta}(d_1 - e_1) - r d_1}{r\delta + \beta_1(e_2 - d_2)}, \\ S^* &= [d_1 + \delta I_2]/\beta_1. \end{aligned} \tag{5}$$

For this equilibrium to exist with each species present in positive numbers, either

$$\frac{d_2}{d_1}(d_1 - e_1) > \frac{r\delta}{\beta_1} > (d_2 - e_2) \tag{6}$$

or

$$\frac{d_2}{d_1}(d_1 - e_1) < \frac{r\delta}{\beta_1} < (d_2 - e_2). \tag{7}$$

must hold. The Appendix shows that the system is locally stable at the above equilibrium, if

$$r\delta < \beta_1(d_2 - e_2). \tag{8}$$

This implies that only inequality conditions (7) define a stable equilibrium for the host–parasite–hyperparasite chain; conditions (6) lead to a locally unstable joint equilibrium. A fact that is useful below is that (6) implies $d_1/e_1 > d_2/e_2$, whereas (7) requires $d_1/e_1 < d_2/e_2$.

The various possible outcomes can be categorized in terms of the potential regulatory ability of each parasite, as summarized in Table 1, and discussed as follows:

(I) $d_1/e_1 < 1$ and $d_2/e_2 < 1$: there is no equilibrium. The quantity $e_i - d_i$ gauges the net per capita

TABLE 1

Potential outcomes of obligate hyperparasitism in model (4)

A: Invasion of hyperparasite if $r\delta > \beta_1 d_2(1 - e_1/d_1)$. *B*: Local stability criterion: $r\delta < \beta_1(d_2 - o_2) < \beta_1(d_2 - e_2)$ $\beta_1(d_2 - e_2)$

I. Neither parasite is potentially regulatory ($d_1/e_1 < 1$, $d_2/e_2 < 1$)
Full host–parasite–hyperparasite chain persists, but with unbounded host growth

II. Only the hyperparasite is potentially regulatory ($d_1/e_1 < 1$, $d_2/e_2 > 1$)
Full chain persists, but stable only if *B* holds.

III. Only the primary parasite is potentially regulatory ($d_1/e_1 > 1$, $d_2/e_2 < 1$).
Alternative states arise if *B* holds:
Hyperparasite absent, and system stable, or
Full chain persists, but with unbounded growth
If *B* does not hold, full chain persists, but with unbounded growth

IV. Both primary and hyperparasites are potentially regulatory ($d_1/e_1 > 1$, $d_2/e_2 > 1$)
If *A* holds, full chain persists
at a stable equilibrium if *B* also holds, otherwise with unbounded growth
If *A* does not hold, but *B* holds.
Alternate states exist:
Hyperparasite absent, and system stable, or
Full chain persists, but with unbounded growth
If neither *A* nor *B* hold, the hyperparasite cannot persist

contribution of a host parasitized with parasite i to host population growth—births of new hosts to parasitized hosts, minus parasitized host deaths. The primary parasite cannot regulate the host in the absence of the obligate hyperparasite ($e_1 - d_1 > 0$), and the latter is not even potentially regulatory ($e_2 - d_2 > 0$). Because $dN/dt = rS + (e_1 - d_1)I + (e_2 - d_2)I_2 > 0$, (where $N = S + I_1 + I_2$), the total host population has unbounded growth, with the hyperparasite increasing once $I_1 > d_1/\delta$. The magnitude of host r is in this case clearly irrelevant to hyperparasite persistence.

(II) $d_1/e_1 < 1$ and $d_2/e_2 > 1$: the hyperparasite increases deaths or decreases fecundity of parasitized hosts, relative to hosts with just the primary parasite. Without the hyperparasite, the primary parasite cannot regulate the host, and both S and I_1 increase without limit; the hyperparasite can invade when $I_1 > d_2/\delta$. If $r\delta < \beta_1(d_2 - e_2)$, a stable equilibrium exists; otherwise, no point equilibrium exists, and the host population grows in an unbounded fashion. Thus, if the primary parasite is non-regulatory, the hyperparasite can persist and moreover can stabilise an unstable host–primary parasite association if (1) the hyperparasite is sufficiently pathogenic or depresses host birth or recovery (relative to the primary parasite), (2) the transmission rate of the primary parasite is high, (3) the transmission rate of the hyperparasite is relatively low, and (4) host r is low.

These conditions make intuitive sense. For parasitism to be able to regulate the host, some parasitized hosts must have a birth rate less than their own death rate. Given our assumptions about parameter values, the only hosts with such demographic characteristics are hyperparasitised hosts. Each such host must first pass through a state with just the primary parasite. In effect, the non-regulatory primary parasite is a "bottleneck" diluting the regulatory impact of parasitism on the host. The hyperparasite tends to limit the abundance of hosts with just primary parasites. High transmission rates for the hyperparasite lowers this density (a kind of over-exploitation), thereby freeing uninfected hosts to grow. By contrast, higher transmission rates for the primary parasite more quickly move healthy hosts into a state where they can be then attacked by the potentially regulatory hyperparasite. The higher the intrinsic growth rate of the host, the more likely it an escape regulation by parasitism. Hyperparasitism is thus most likely to foster stable host dynamics for low-r hosts.

(III) $d_1/e_1 > 1$ and $d_2/e_2 < 1$: now, the primary parasite is regulatory, but the hyperparasite is non-regulatory (e.g. it may free the host from deleterious effects of the primary parasite). Without the hyperparasite, the host and primary parasite settle into a stable point equilibrium. The hyperparasite can invade if $r\delta > \beta_1 d_2(1 - e_1/d_1)$. Given such an invasion (e.g. high host r) there is no equilibrium, and the host population grows in an unbounded manner. If instead $r\delta < \beta_1 d_2(1 - e_1/d_1)$, the hyperparasite cannot invade. An equilibrium exists with all three populations present, but this equilibrium is locally unstable. Numerical studies suggest that when perturbed, the system may either converge toward the equilibrium with the hyperparasite absent, or grow exponentially, depending upon the magnitude and direction of the perturbation.

Thus, if the primary parasite is regulatory but the hyperparasite is not potentially regulatory, either the hyperparasite will not invade (e.g. low host r) and the host will be regulated by the primary parasite, or the hyperparasite will invade, but the host population will not then be regulated to a constant abundance by parasitism at all (e.g. high host r). A non-regulatory hyperparasite creates a potential for a host population with high r to escape regulation, because such a hyperparasite automatically weakens regulation by the primary parasite.

(IV) $d_1/e_1 > 1$ and $d_2/e_2 > 1$: this is the most complicated case. In the absence of the hyperparasite, the host and primary parasite persist at a stable equilibrium, and the hyperparasite is potentially regulatory. There are two sub-cases to consider:

(1) if $r\delta > \beta_1 d_2(1 - e_1/d_1)$, the hyperparasite can invade the equilibrium consisting of the primary parasite and host. If, in addition, (i) $r\delta < \beta_1(d_2 - e_2)$, a locally stable 3-species equilibrium exists. These inequalities together imply as a necessary condition for stability that $d_1/e_1 < d_2/e_2$. Note that if both the e_i approach 0, these two inequalities cannot be jointly satisfied. In this limit, the host–parasite–hyperparasite model converges on the Lotka–Volterra food chain model without basal density-dependence, which as noted above is unstable. By contrast, if (ii) $r\delta > \beta_1(d_2 - e_2)$, the system is destabilized by an invading hyperparasite.

We can conclude that for the full chain of host–parasite–hyperparasite to stably persist at an equilibrium, hosts with the hyperparasite must have either higher death rates, or lower birth rates, or lower recovery rates, than hosts with the primary parasite. Moreover, persistence of such a chain requires that the compound quantity $r\delta/\beta_1$ lie within particular bounds. If this quantity is too low, the hyperparasite cannot invade or persist; if too high, the hyperparasite

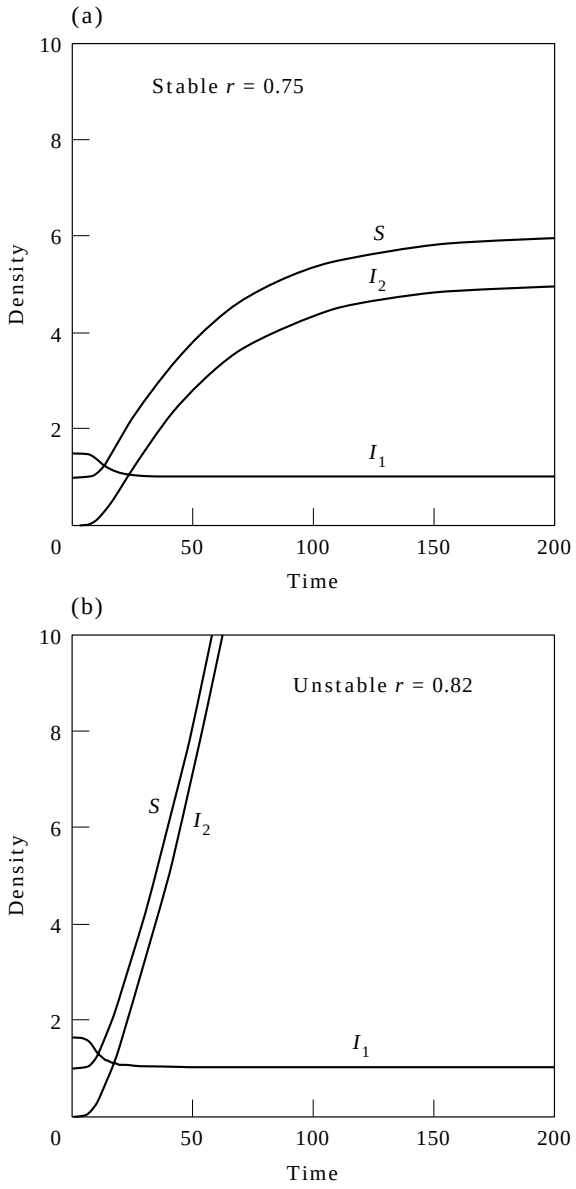

FIG. 1. A numerical example of how increasing host growth rates can destabilize a host–parasite–hyperparasite system. Model (4) was numerically integrated using a Runge–Kutta, variable step size algorithm. Initial conditions were equilibrial densities for the host and primary parasite, calculated in the absence of the hyperparasite, and an invasive propagule of the hyperparasite equal to 0.001. Parameter values were $\delta = 1.0$, $\beta_1 = 1.0$, $e_1 = 0.5$, $e_2 = 0.2$, $d_1 = 1.0$, $d_2 = 1.0$. S = uninfected host density; I_1 = density of hosts infected with primary parasite, I_2 = density of hosts infected with hyperparasite. (a) Host intrinsic growth rate $r = 0.75$. The three species system settles into a stable equilibrium; (b) at higher host production, $r = 0.82$, the three species system is unstable.

destabilizes an intrinsically stable host–parasite interaction. Figure 1 provides a numerical example of how an increase in host intrinsic growth rate can destabilize a host–parasite–hyperparasite interaction. Although a host–parasite–hyperparasite chain can stably persist without the operation of other limiting factors, the parameter combinations permitting stable persistence may be rather restricted. For instance, if the transmission coefficients and depletion coefficients (the d_is) are approximately unity, the condition for stable persistence is $1 - e_1 < r < 1 - e_2$. Stable persistence occurs only for a narrow band of values for the host rate of increase.

(2) if $r\delta < \beta_1 d_2(1 - e_1/d_1)$, the hyperparasite cannot invade when the primary parasite and host are at equilibrium. With $r\delta > \beta_1(d_2 - e_2)$ as well, an unstable 3-species point equilibrium exists. (These inequalities together imply $e_1/d_1 < e_2/d_2$.) Following a perturbation, numerical studies suggest that the system can either lose the hyperparasite, or explode exponentially, depending upon the magnitude and direction of the perturbation. Finally, when $r\delta < \beta_1(d_2 - e_2)$, no 3-species equilibrium exists at all, and the hyperparasite cannot persist.

In the canonical host–parasite model (3), parasitism is regulatory provided only that infected hosts have higher death than birth rates. In a multispecies context with obligate hyperparasites, this simple demographic "rule-of-thumb" for potential regulation of hosts by parasitism may not hold. Hyperparasites tend to limit the response of primary parasites to increases in host numbers, thereby weakening or even precluding the regulatory potential of parasitism, even if all parasitized hosts are observed to have higher death than birth rates.

Discussion

We have considered here the persistence of hyperparasites, in systems where parasitism is the sole factor regulating host populations. To recapitulate our main results, we first examined donor-controlled parasite–hyperparasite interactions, and showed that hyperparasites are more likely to persist if hosts have high intrinsic growth rates. Moreover, hyperparasites are less likely to occur if the primary parasite is highly pathogenic, or has a high transmission rate. We then turned to systems where hyperparasites either suppress or supplant primary parasites, and showed that a hyperparasite may either facilitate host regulation (mediated by the primary parasite), or preclude such regulation. There are many parallels between food chain and host–parasite–hyperparasite dynamics, and some key differences as well. A hyperparasitoid functions much as a top-level predator in conventional food-chain models. However, the greater complexity of the parasitic life-style means a much wider array of demographic effects, and thus dynamic outcomes, are possible than would

be the case for predators (Hochberg & Holt, 1990). Structurally speaking, predation in the Lotka–Volterra model (2) is isomorphic to a special limiting case of the canonical host–parasite model (3) where births and recoveries by the parasitised hosts are precluded. We expect that the implications of hyperparasitism, and more generally parasitism, for population and community ecology will be more variable and complex than the effects created by predation.

We have seen that persistence of the full system in each case can depend upon the intrinsic growth rate of the basal resource or host population. A common result of food chain models is that an increase in the productivity of the lowest level makes it easier for the secondary consumer to persist. In like manner, when the primary parasite is able to regulate the host population (i.e. $d_1/e_1 > 1$), the hyperparasite persists only if $r\delta > \beta_1 d_2(1 - e_1/d_1)$. So one should tend to observe hyperparasitism only in host populations with high intrinsic growth rates. However, this condition becomes irrelevant when the primary parasite is non-regulatory.

The Lotka–Volterra model (1) for a food chain requires direct density-dependence in birth or death rates (e.g. in the basal species) to provide a reasonable description of systems with bounded dynamics. The canonical host–parasite model introduces density-dependence through the back door (via recruitment or recovery from the infected fraction of the population). An intriguing difference between host–parasite and predator–prey dynamics is thus that the former can exhibit population stability even in the absence of direct density-dependence on births or deaths of healthy or infected hosts, so long as infected hosts can provide recruits (via birth or recovery) to the infected population. An important potential effect of hyperparasitism is that it may modulate the likelihood of population stability arising solely from parasitism.

Hyperparasitism can have diverse effects upon population stability. If the primary parasite is unable to regulate the host, addition of a hyperparasite may stabilize the system. This requires that the host does not have too great an intrinsic rate of increase, which seems intuitively reasonable. More surprisingly, stability also requires that the hyperparasite's rate of infection not be too great, relative to the primary parasite. The reason is that as δ increases, so does the equilibrial density of healthy hosts, without compensatory increases in the densities of infected hosts; this allows the destabilizing effect of the host's positive intrinsic growth rate to overwhelm the stabilizing effect of the parasites.

Conversely, a hyperparasite can destabilize an otherwise stable primary parasite–host interaction. If $d_2/e_2 < 1$ and the hyperparasite can invade, it is always destabilizing. However, unlike the general model (4) examined in Hochberg & Holt (1990), where we assumed the hyperparasite could also directly infect healthy hosts, it does not appear that obligatory hyperparasitism leads to sustained, bounded fluctuations around an unstable equilibrium point.

Scant data exists to assess these theoretical suggestions, and to know which (if any) of the above cases might fit natural systems. It might seem plausible to assume that in many systems, hyperparasitism would reduce the impact of the primary parasite upon the host. If this were the case, then case II might be less likely than case III. However, it should be noted that the model form also describes systems in which a secondary parasite attacks hosts harboring the primary parasite, which it then replaces (a form of parasitism referred to above as "kleptoparasitism"). The realized level of virulence is expected to reflect in part a within-host competitive struggle between parasites (Frank, 1996). Competition among parasites within host individuals can lead to an increase over evolutionary time scales in virulence. A primary parasite in a host freshly infected by a hyperparasite, which will supplant or suppress it, has nothing to gain by restraining reproduction, and so hosts with hyperparasites might well show higher mortality rates than hosts with just the primary parasite. Thus, we predict that rich parasite communities containing both obligate and facultative hyperparasites should have the most virulent primary parasites, all else being equal.

Recognizing the multifarious possible effects of obligate hyperparasitism on stability may at times have important practical consequences. In biological control the establishment of hyperparasites is routinely discouraged (Bennett, 1981). We have shown (Case II above) that hyperparasites can sometimes be responsible for the success of biological control (defined here as a population regulated at densities too low to experience direct density-dependence) in some cases where the primary parasite (i.e. a biological control agent) might on its own be unable to regulate the population of the target pest. In effect, biological control may emerge from a multispecies "team effort" of the primary parasite and hyperparasite. We were not able to uncover any field studies which might match this theoretical possibility. Furthermore, we certainly would not claim that the conditions leading to this effect are likely to be realized with high frequency in biological control systems, and in any case, control which rests on a

delicate fabric of multispecies interactions is not likely to be robust.

An important direction for future work will be to examine the issue of oligatory hyperparasite persistence in a broader range of systems, relaxing the epidemiological assumptions of the above models (e.g. as in Gubbins & Gilligan, 1996), and incorporating the effects of other regulatory factors. For instance, permitting hosts to lose hyperparasites, without also losing the primary parasite, leads to a model resembling (5) above, but with an additional term in the middle equation (reflecting recovery of primary parasites from infection by hyperparasites). Analysis of this model leads to a set of results differing in detail, but qualitatively resembling, those presented above; these results include the requirement that host r lie within a range of values for stable equilibrium, and the potential of hyperparasitism to stabilize certain host–parasite systems, and to destabilize others (Hochberg & Holt, unpublished results).

For all the above cases in which parasitism was non-regulatory, in natural populations one would expect host numbers to increase to a point at which direct density-dependence becomes relevant, even if it is insignificant at low densities. As in food chain models, adding direct-dependence to the host population can lead to an additional constraint on hyperparasite persistence, beyond that provided by host intrinsic growth rates and transmission coefficients (resembling the effect of K in the food chain model above). Taylor *et al*. (1998) have recently examined from an evolutionary perspective a model of this form, differing in its details from the obligate hyperparasitism model discussed here [they include transmission of the hyperparasite to healthy hosts [$\beta_2 > 0$ in model (4)], direct density-dependence in host births, and functionally coupled transmission and mortality rates]; their results suggest that hyperparasites may not persist at very low host K. They make the very interesting suggestion that in field studies hyperparasite invasion might be mistaken for an evolved reduction in virulence in the primary parasite.

The results presented here provide additional examples to a growing list of how indirect interactions in a multispecies context can be pivotal in modifying species interactions and determining community structure (for further discussion see e.g. Begon & Bowers, 1995; Holt, 1996b; Price *et al*., 1986; Wootton, 1994). Hyperparasitism is likely to be an important modulator of many aspects of community structure, such as the impact of shared pathogens on species coexistence (Holt & Pickering, 1985) and the likelihood of complex dynamics (Begon *et al*., 1996). This class of parasitic interactions warrants much more theoretical attention.

R. D. H. and M. E. H. gratefully acknowledge the support of the National Science Foundation, NERC and CNRS, and the hospitality of staff and scientists of the CNRS Laboratoire d'Ecologie in Paris. We thank M. Barfield for assistance reviewers for their helpful comments.

REFERENCES

Anderson, R. M. & May, R. M. (1986). The invasion, persistence, and spread of infectious diseases within animal and plant communities. *Phil. Trans. R. Soc. B* **314,** 533–570.

Anderson, R. M. & May, R. M. (1991). *Infectious Diseases of Humans: Dynamics and Control*. Oxford: Oxford University Press.

Beddington, J. R. & Hammond, P. S. (1977). On the dynamics of host–parasite–hyperparasite interactions. *J. Anim. Ecol.* **46,** 811–821.

Begon, M. & Bowers, R. G. (1995). Beyond host–pathogen dynamics. In: *Ecology of Infectious Diseases in Natural Populations* (Grenfell, B. T. & Dobson, A. P., eds) pp. 478–509. Cambridge: Cambridge University Press.

Begon, M., Sait, S. M. & Thompson, D. J. (1996). Two's company, three's a crowd: host–pathogen–parasitoid dynamics. In: *Multitrophic Interactions in Terrestrial Systerms* (Gange, A. C. & Brown, V. K., eds) pp. 307–332. Oxford: Blackwell.

Bennett, A. W. (1981). Hyperparasitism in the practice of biological control. In: *The Role of Hyperparasitism in Biological Control: A Symposium* (Rosen, D., ed.) pp. 43–49. Berkeley: Div. Agric. Sci., Univ. of California.

Cook, R. J. & Baker, K. F. (1983). *The Nature and Practice of Biological Control of Plant Pathogens*. St. Paul, MN: American Phytopathological Society.

DeAngelis, D. L. (1992). *Dynamics of Nutrient Cycling and Food Webs*. London: Chapman & Hall.

Frank, S. A. (1996). Models of parasite virulence. *Quart. Rev. Biol.* **71,** 37–78.

Grenfell, B. T. & Dobson, A. P. (eds) (1995). *Ecology of Infectious Diseases in Natural Populations*. Cambridge: Cambridge University Press.

Gubbins, S. & Gilligan, G. A. (1996). Population dynamics of a parasite and hyperparasite in a closed system: model analysis and parameter estimation. *Proc. R. Soc. Lond. B* **263,** 1071–1078.

Hallam, T. G. (1986). Community dynamics in a homogeneous environment. In: *Mathematical Ecology: An Introduction* (Levin, S., ed.) pp. 241–285 Berlin: Springer-Verlag.

Hassell, M. P. (1978). *The Dynamics of Arthropod Predator–Prey Systems*. Princeton, NJ: Princeton University Press.

Hochberg, M. E. & Holt, R. D. (1990). The coexistence of competing parasites. 1—The role of cross-species infection. *Am. Nat.* **136,** 517–541.

Holt, R. D. (1996a). Community modules. In: *Multitrophic Interactions in Terrestrial Systerms* (Gange, A. C. & Brown, V. K., eds) pp. 333–350. Oxford: Blackwell.

Holt, R. D. (1996). Food webs in space: an island biogeographic perspective. In: *Food Webs: Contemporary Perspectives* (Polis, G. & Winemiller, K. eds) pp. 313–323 London: Chapman & Hall.

Holt, R. D. & Pickering, J. (1985). Infectious disease and species coexistence: a model of Lotka–Volterra form. *Am. Nat.* **126,** 196–211.

Holt, R. D. & Polis, G. A. (1997). A theoretical framework for intraguild predation. *Am. Nat.* **149,** 745–764.

Hutchinson, G. E. (1959). Homage to Santa Rosalia, or why are there so many kinds of animals. *Am. Nat.* **93,** 145–159.

LEVIN, B. R. & BULL, J. J. (1996). Phage therapy revisited: the population biology of a bacterial infection and its treatment with bacteriophage and antibiotics. *Am. Nat.* **147,** 881–898.
LOGOFET, D. O. (1993). *Matrices and Graphs*: *Stability Problems in Mathematical Ecology*. Boca Raton: CRC Press.
MAY, R. M. (1973). *Stability and Complexity in Model Ecosystems*. Princeton, NJ: Princeton University Press.
MAY, R. M. & HASSELL, M. P. (1981). The dynamics of multi-parasitoid–host interactions. *Am. Nat.* **117,** 234–261.
OKSANEN, L., FRETWELL, S. D., ARRUDA, J. & NIEMALA, P. (1981). Exploitation ecosystems in gradients of primary productivity. *Am. Nat.* **118,** 240–261.
PIMM, S. L. (1982). *Food Webs*. London: Chapman & Hall.
PRICE, P. W., WESTOBY, M., RICE, B., ATSATT, P. R., FRITZ, R. S., THOMPSON, J. N. & MOBLEY, K. (1986). Parasite mediation in ecological interactions. *Ann. Rev. Ecol. Syst.* **17,** 487–505.
TAYLOR, D. R., JAROSZ, A., LENSKI, R. E. & FULBRIGHT, D. (1998). Acquisition of hypovirulence in host–pathogen systems with three trophic levels. *Am. Nat.* **151,** 343–355.
WOOTTON, J. T. (1994). The nature and consequences of indirect effects in ecological communities. *Ann. Rev. Ecol. Syst.* **25,** 443–466.

APPENDIX

The Jacobian of the model in the main text has the general form

$$J = \begin{pmatrix} a & b & c \\ 0 & 0 & d \\ f & g & 0 \end{pmatrix}$$

where $a = r - \beta_1 I_1^*$, $b = e_2$, $c = e_1 - \beta_1 S^*$, $d = \delta I_2^*$, $f = \beta_1 I_1^*$, and $g = -\delta I_1^*$. The characteristic equation is

$$\lambda^3 + \alpha_1 \lambda^2 + \alpha_2 \lambda + \alpha_3 = 0$$

where $\alpha_1 = -a$, $\alpha_2 = -(dg + cf)$, and $\alpha_3 = adg - bdg$. The Routh–Hurwitz criteria that the dominant root of the characteristic equation has a negative real part are that each $\alpha_i > 0$, and $\alpha_1 \alpha_2 > a_3$.

At equilibrium, $\mathrm{d}S/\mathrm{d}t = 0$ implies that $\beta_1 I_1^* - r = (e_2 I_2^* + e_1 I_1^*)/S^* > 0$. The left side is just α_1, so the first Routh–Hurwitz criterion is satisfied. Similarly, from the same equation, $\beta_1 S^* - e_1 = (rS^* + e_2 I_2^*) I_1^* > 0$; substitution shows that $\alpha_2 > 0$.

The coefficient $\alpha_3 = \delta I_2^* I_1^* [\beta_1 \delta I_1^* - r\delta - e_2 \beta_1] > 0$. Using the fact that $\delta I_1^* = d_2$, we find that this criterion is satisfied if

$$\beta_1 (d_2 - e_2) > r\delta \qquad \text{(A.1)}$$

The final Routh–Hurwitz criterion $\alpha_1 \alpha_2 > \alpha_3$ can be written as $(\beta_1 I_1^* - r)(\beta_1 S^* - e_1)\beta_1 I_1^* > -e_1 \beta_1 \delta I_2^* I_1^*$. We showed above that the two parenthetical terms on the left hand side are positive at an equilibrium, hence this inequality is always satisfied. Thus, the sole condition for local stability of the host–parasite–hyperparasite model is (A.1) above.