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PREDATORS, PARASITOIDS, AND PATHOGENS: A CROSS-CUTTING EXAMINATION OF INTRAGUILD PREDATION THEORY

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Abstract. Although the canonical concept of intraguild predation evokes images of predators and prey, several subdisciplines within ecology have developed theory not specifically framed in terms of predation and competition and often using system-specific terminology, yet functionally quite similar. Here, we formulate models combining exploitation and competition in predator–prey, host–parasitoid, and host–pathogen communities to compare dynamics, food web structure, and coexistence criteria for these disparate communities. Although dynamic stability in the coexistence region varies strongly among systems, in all cases coexistence of two consumers on a single resource occurs only if the intraguild prey species is more efficient than the intraguild predator at suppressing the abundance of the basal resource, and if the intraguild predator accrues a sufficient gain from attacking the intraguild prey. In addition, equilibrial abundances of all species in all three formulations respond similarly to increases in productivity of the basal resource. Our understanding of predator–prey and parasitoid–host communities has benefited from explicit examination of intraguild predation (IGP) theory, and we suggest that future research examining pathogen communities, in particular, will benefit substantially from explicit recognition of predictions from IGP theory.

Key words: coexistence; community module; competition; disease transmission; exploitation; food web; heuristic model; trophic interactions.

Introduction

Intraguild predation (IGP), predation on a consumer species by its guild member, is common in natural communities (Polis et al. 1989) and has received substantial attention in the theoretical literature over the past decade, following the publication of a general theory of IGP by Holt and Polis (1997). Theory has shown that the presence of intraguild predation in a community has implications for important ecological issues including the relationship between food web stability and diversity, coexistence of resource competitors, suppression of the shared resource, and effects of productivity on trophic interactions. Thus, intraguild predation is relevant to both ecological theory and important applied problems such as biological control of pest species and conservation of threatened species.

Theory has demonstrated that intraguild predation counterbalanced by superior resource exploitation by the intraguild prey can lead to coexistence of guild members on a single, shared resource (Holt and Polis 1997). This type of feeding relationship among guild members is commonly observed within complex food webs (Polis et al. 1989); however complexities such as resource subsidies, host resistance, defensive behavior, or immigration may provide alternative avenues for coexistence (Heithaus 2001, Mylius et al. 2001, Finke and Denno 2002, MacNeil et al. 2003, Briggs and Borer 2005). Here we examine the theoretical similarities among diverse systems with only three interacting species and explore the dynamics of these simple communities.

Although the original IGP concept focused on a combination of exploitation and competition that blurred the distinctions between predators and prey, several subdisciplines within ecology have developed theory not specifically framed in terms of predation and competition, yet that is functionally quite similar. Model formulations combining exploitative and interference competition have been developed for predator–prey (e.g., Holt and Polis 1997), host–parasitoid (Briggs 1993), and host–parasite (e.g., Hochberg and Holt 1990) systems. In predator–prey models, predation provides a unique gain for the intraguild predator that is independent of the shared resource. In host–parasitoid models, there is less of a distinction between gains from a host containing a parasitoid and gains from a healthy host. In host–parasite (or pathogen) models, one parasite outcompetes another within a co-infected host.

Many models of IGP have been published spanning the continuum from simple, heuristic models intended to

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build intuition (e.g., Hochberg et al. 1990, Holt and Polis 1997, Borer et al. 2003) to complex models tailored to specific systems that are more amenable to direct, quantitative tests with real data (e.g., Gismervik and Andersen 1997, Borer 2006, van de Wolfshaar et al. 2006). Here, we examine heuristic models with parallel structures designed to reflect existing published and influential simple models, with the goal of showing that they are different manifestations of the same, fundamental underlying module of interactions (e.g., Hochberg and Holt 1990, Holt and Polis 1997). We compare these simple models to demonstrate that although predator–prey, host–parasitoid, and host–pathogen interactions appear to differ substantially in terms of taxonomic identity and natural history, the underlying interaction structure and general dynamical predictions generated among systems are, in fact, quite similar. We use this approach to unify predator–prey, host–parasitoid, and host–parasite IGP formulations to clarify the meaning of parameters, examine model dynamics, food web structure, and regions of coexistence for these diverse types of IGP communities, and suggest directions for future research.

Model Structure, Formulation, and Parameter Values

Predator–prey

The basal prey (Fig. 1a; H) is usually considered to be an animal that is killed and consumed by both predator species. The intraguild prey (IG prey, P) is a specialist that kills and consumes only the prey resource, H. The intraguild predator (IG predator, Q) is a generalist predator that kills and consumes both H and P. In this formulation, when H dies, it is converted into Q (via pathway 1) or P (via pathway 3), usually with low conversion efficiency ($\ll$1 predator created per prey consumed). When Q kills and consumes P (pathway 2), the conversion is often substantially less than 1, as well.

In this type of formulation (see Holt and Polis 1997), all three species are generally assumed to interact only via consumption. When predation occurs, it always ends in death of the prey (H or P). For simplicity, we assume that in the absence of predators, the basal resource grows logistically, with maximum intrinsic growth rate, r, and carrying capacity, K. The IG prey feeds on the basal resource with attack rate a_{HP}, converting consumed resource into new predators with conversion efficiency, c_{HP}. The IG predator can feed on both the basal resource (with attack rate a_{HQ} and conversion efficiency c_{HQ}) and the IG prey (with attack rate a_{PQ} and conversion efficiency c_{PQ}). The IG predator may have a preference for one of the prey types (a_{HQ} and a_{PQ} may differ) which does not change with density (i.e., no switching). In each case, for simplicity, we assume that predators have linear (type I) functional responses. Predators P and Q have density-independent per capita death rates, d_P and d_Q, respectively. With these assumptions, IGP in a predator–prey system can be described as follows:

Basal resource:

$$\frac{dH(t)}{dt} = rH(t)\left[1 - \frac{H(t)}{K}\right] - a_{HP}H(t)P(t) - a_{HQ}H(t)Q(t)$$

Intraguild prey:

$$\frac{dP(t)}{dt} = c_{HP}a_{HP}H(t)P(t) - a_{PQ}P(t)Q(t) - d_PP(t)$$

Intraguild predator:

$$\frac{dQ(t)}{dt} = c_{HQ}a_{HQ}H(t)Q(t) + c_{PQ}a_{PQ}P(t)Q(t) - d_QQ(t)$$

where H, P, and Q are densities of basal prey, intraguild prey, and intraguild predator, respectively.

Host–parasitoid (or pathogen–host system with free-living pathogen stage)

The shared host (Fig. 1b; H) is usually considered to be an insect that is parasitized, killed, and consumed by two parasitoid species. The IG predator (Q) is a facultative hyperparasitoid that can successfully parasitize, consume, and produce progeny from both healthy hosts and those containing the IG prey parasitoid (P). The IG prey (P) is a primary parasitoid that can only successfully parasitize previously unparasitized hosts (H), but succumbs in within-host competition to the IG predator. In this formulation, when the IG prey or IG predator attack a host, the host is converted immediately into an immature attacker species (I_Q, I_P) which develops within a single host before emerging to become capable of further attacks; the IG predator (Q) does not necessarily receive an independent gain from consumption of a host containing the IG prey, compared to consumption of a healthy host (i.e., Fig. 1b, pathways 1 vs. 2).

The population dynamics of all three species involved in host–parasitoid IGP systems are tightly linked for two primary reasons. First, for every host attacked, a parasitoid is produced. Second, interactions among hosts and parasitoids are assumed to take place within a single host, thereby spatially constraining all of the interacting species. Interaction among species occurs through searching and oviposition by adult parasitoids, and parasitism ends in death of the host or both the host and an embedded IG prey.

In the parasitoid–host system, we assume that the host population grows logistically in the absence of the parasitoid. For this system, a_{HP} and a_{HQ} are the attack rates on the host by adult parasitoids P and Q, respectively, and a_{PQ} is the attack rate on juvenile P by adult Q. Hosts attacked by P or Q enter the juvenile stage of their respective parasitoid species, I_P and I_Q. The conversion efficiencies which determine the number of juvenile parasitoids produced per host attacked are c_{HP} and c_{HQ} for attacks on the host by parasitoid P and Q, respectively, and c_{PQ} is the number of juveniles of

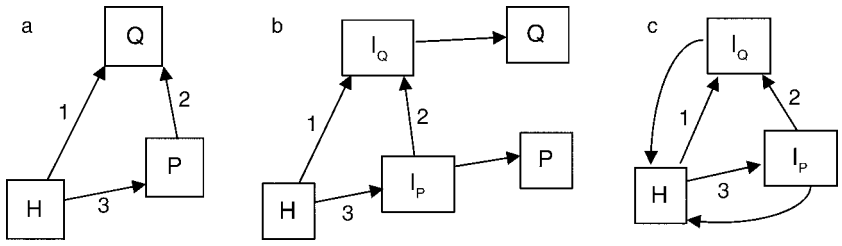

FIG. 1. Generalized diagrams of intraguild predation (IGP) for (a) predator–prey, (b) host–parasitoid, and (c) host–pathogen interactions. Arrows represent conversion of biomass from one stage or species to the next; H, P, Q, I_P, and I_Q are defined in *Model structure, formulation, and parameter values*.

parasitoid Q produced per attack by Q on juveniles of parasitoid P. Here we assume that juveniles of parasitoid P and Q mature out of the immature stage at rates m_P and m_Q, respectively, and enter into their adult stages. This assumes an exponential distribution of the time spent in the juvenile stage, which is a convenient, but perhaps not the most realistic, form of stage-structure (e.g., Murdoch et al. 2003). The parasitoid death rates are assumed to be density independent, at rates d_{IP} and d_{IQ} for immature parasitoids P and Q, respectively, and d_P and d_Q for adult parasitoids P and Q, respectively. These equations are:

Host:

$$\frac{dH(t)}{dt} = rH(t)\left[1 - \frac{H(t)}{K}\right] - a_{HP}H(t)P(t) - a_{HQ}H(t)Q(t)$$

Immature primary parasitoid:

$$\frac{dI_P(t)}{dt} = c_{HP}a_{HP}H(t)P(t) - a_{PQ}I_P(t)Q(t) - (m_P + d_{IP})I_P(t)$$

Adult primary parasitoid:

$$\frac{dP(t)}{dt} = m_P I_P(t) - d_P P(t)$$

Immature facultative hyperparasitoid:

$$\frac{dI_Q(t)}{dt} = c_{HQ}a_{HQ}H(t)Q(t) + c_{PQ}a_{PQ}I_P(t)Q(t) - (m_Q + d_Q)I_Q(t)$$

Adult facultative hyperparasitoid:

$$\frac{dQ(t)}{dt} = m_Q I_Q(t) - d_Q Q(t)$$

where I_P and I_Q are densities of immature primary parasitoids and facultative hyperparasitoids, respectively.

Alternatively, this system of equations could represent a single host species attacked by two pathogens that are spread by free-living infectious stages (e.g., insect baculoviruses). In many insect baculovirus systems, infected hosts stop feeding, do not reproduce, and do not recover from infection. In this case, P and Q represent the densities of free-living infectious particles of the two pathogen species, and I_P and I_Q represent hosts that have been infected with pathogens P and Q, respectively. In this interpretation, hosts infected by P and Q shed pathogen particles at rates m_P and m_Q, respectively (under this interpretation, the $-m_P I_P(t)$ or $-m_Q I_Q(t)$ terms are removed from the $dI_P(t)/dt$ and $dI_Q(t)/dt$ equations). Within a host, pathogen Q outcompetes P. Therefore, hosts infected by P can become reinfected by Q, and will release infectious particles only of pathogen Q.

Pathogen–host (for a directly transmitted infectious disease)

In this rendition, the host (Fig. 1c; H) is usually considered to be an animal or plant that is susceptible to multiple pathogens. Fig. 1c shows the interactions between two directly-transmitted pathogens that do not induce an immune response in the host (in the notation of the disease modeling literature, the interaction between each single disease and the host would be represented by a susceptible-infected-susceptible (SIS) model). Each pathogen can potentially increase host mortality, but individual hosts that clear either infection return directly to the susceptible host stage. The IG predator (Q) is a pathogen that can infect hosts regardless of their infection status; this species clears a host of infection by P. The IG prey (P) is a pathogen that can infect only healthy hosts. In this formulation, hosts are directly infected by either pathogen (P or Q) via intra-host contact, according to a standard mass-action (density-dependent) transmission function (McCallum et al. 2001). As discussed above, pathogens transmitted via free-living infectious particles (e.g., insect baculovirus) are better represented by Fig. 1b, in which there are unique parameters for both infected hosts (I_Q, I_P) and free-living pathogen particles (P and Q).

As in the host–parasitoid formulation, population dynamics are tightly linked and occur within individual hosts. Pathogens depend on hosts for additional infections; contact among hosts allows pathogen transmission. Unlike the other two formulations, as a rule, infected hosts can reproduce. Recovery from infection or resistance/immunity following recovery (not included

in Fig. 1c), can weaken the link between host mortality and IG predator and IG prey reproduction compared to the other two formulations.

Here, a single host species can become infected by two different pathogen species or strains. Infected hosts can reproduce and contribute to density dependence. Density dependence occurs only in host reproduction, and so we separate the maximum intrinsic rate of increase (r) of the host into birth (b) and death (d_H) components, with $r = b - d_H$. Disease transmission is assumed to take on a mass-action (density-dependent) form, with a_{HP} and a_{HQ} representing the transmission rates between susceptible hosts and hosts infected by P and Q, respectively. Pathogen Q is the superior competitor within a host; hosts infected by P can become infected by Q following successful contact with Q (with transmission rate a_{PQ}). We have assumed a very simplified form of the interaction between the pathogens here, with the IG predator completely displacing the IG prey from a host; in principle, many other outcomes are possible (e.g., Hudson et al. 2002). Infection by each pathogen can lead to increased mortality, with d_P and d_Q representing the increased mortality of hosts due to infection by P and Q, respectively. Unlike the cases involving predators or parasitoids, infected hosts can potentially recover from infection, and γ_P and γ_Q are the recovery rates. For simplicity, we assume no acquired immunity. In most cases, each susceptible host leads to one infected host following disease transmission; thus the terms that are equivalent to conversion efficiencies in the above models (c_{HP} and c_{HQ}) are generally equal to 1. However, we allow c_{PQ} to take on values less than 1 if some hosts do not survive co-infection by the second pathogen. The equations describing IGP in a host–pathogen system are:

Susceptible hosts:

$$\frac{dH(t)}{dt} = bN(t)\left[1 - \frac{N(t)}{K}\right] - a_{HP}H(t)P(t) - a_{HQ}H(t)Q(t) + \gamma_P P(t) + \gamma_Q Q(t) - d_H H(t)$$

Hosts infected by pathogen P:

$$\frac{dP(t)}{dt} = c_{HP}a_{RP}H(t)P(t) - a_{PQ}P(t)Q(t) - (\gamma_P + d_H + d_P)P(t)$$

Hosts infected by pathogen Q:

$$\frac{dQ(t)}{dt} = c_{HQ}a_{HQ}H(t)Q(t) + c_{PQ}a_{PQ}P(t)Q(t) - (\gamma_Q + d_H + d_Q)Q(t)$$

where total host density, $N = H + P + Q$.

Parameter value ranges

Realistic ranges for parameter values specific to each biological system described here are discussed in the Appendix.

Results

For all three system-specific formulations of the IGP model, predator–prey, parasitoid–host, and pathogen–host, coexistence of the two consumers on the single resource is possible only if the IG prey is more efficient than the IG predator at suppressing the abundance of the basal resource, and if the IG predator accrues a sufficient gain from attacking the IG prey. Fig. 2 shows the equilibrium invasion criteria for each of the three model formulations. In each case, the criteria are shown in terms of the relative attack rates of the two consumer species on the basal resource (a_{HQ}/a_{HP}) and the relative conversion efficiency of the IG predator on the IG prey vs. the basal resource (c_{PQ}/c_{HQ}). Coexistence of the two consumers is possible (but not ensured) if invasion by each species into an equilibrium of its competitor is possible. Mutual invasibility may not lead to sustained coexistence because the full system has unstable dynamics (Holt and Polis 1997). In each case, the IG prey can invade if its attack rate is not too low relative to the IG predator (i.e., to the left of the thick vertical line), and the IG predator can invade as long as it has a high attack rate and a relatively high conversion efficiency from attacks on the IG prey (i.e., to the right and above the thin curved line).

Details of the model formulations affect the dynamics of the three-species interaction. The three-species equilibrium is stable throughout the coexistence region for the parameters chosen as typical for the predator–prey system (Fig. 2a). In contrast, the parasitoid–host equilibrium (Fig. 2b) is an unstable equilibrium throughout the coexistence region. In the directly transmitted disease system (Fig. 2c), recovery of infected hosts reduces the demographic impact of exploitation on local prey dynamics and has a strongly stabilizing effect, so the three-species equilibrium is always stable. Stability in these models depends on both structure and parameter values. In particular, instability in the parasitoid–host model and stability in the disease model are due to model structure: respectively, the time lag in the parasitoid case, and recovery and reproduction by infected hosts in the disease case. The stability observed in the predator model depends more on the specific parameter values used in this example (see Appendix) than the intrinsic structure of the system.

Food web structure varies predictably with productivity; equilibrial abundances of the consumers and resource in all three formulations respond similarly to increases in productivity of the basal resource (Fig. 3). At low productivities, only the IG prey persists on the host. At intermediate productivity, coexistence is possible. At high productivity, the IG predator displaces the IG prey. In regions of parameter space where the IG prey and IG predator can coexist on the resource, the IG prey is the superior resource exploiter; thus, the IG predator interferes with the IG prey, leading to increases in abundance of the basal resource.

Discussion

In spite of differences in both terminology and formulation, these models have broadly similar results: coexistence requires intermediate resource productivity and occurs only when the IG predator is a poorer exploiter of the shared resource. Thus, in all model formulations, the resulting basal resource equilibrium is predicted to be higher with both guild members present than with just the superior resource exploiter (the IG prey). Given this qualitative similarity in dynamics among apparently very different exploitative relationships, we are left with two critical questions: To what extent do we expect these simple models to predict the dynamics of real biological systems? And, can we use these similarities to suggest new directions for inquiry?

Biological details of real systems can relax the tight coupling described in the simple models we examined here. For example, refuges that increase the unexploited host equilibrium can take many forms including recovery from infection, immigration of healthy hosts, spatial segregation or aggregation of attacks by the IG predator and IG prey that maintain unexploited host patches, or resistance to attacks (e.g., Mylius et al. 2001, Briggs and Borer 2005, Snyder et al. 2005). As noted above for the case of a directly transmitted disease, host recovery can be stabilizing. More broadly, all these disparate refuge types relax the requirements for coexistence via IGP by providing an alternative coexistence mechanism via reduced coupling among the IG predator, IG prey, and shared resource. Together, multiple mechanisms can jointly maintain coexistence, even if none alone is sufficient (e.g., Snyder et al. 2005). IGP interactions embedded within a larger food web can also relax or alter the requirements for coexistence by generating complex feedbacks and diffuse species interactions (McCann et al. 1998). Thus, in many cases, biological details will provide additional mechanisms for coexistence in real systems.

Although we explicitly examined only three basic community types, IGP theory makes similar predictions for other community combinations. For example, systems with a mixture of predation and parasitism will often resemble the basic intraguild predation module (Hochberg et al. 1990). Hochberg et al. (1990) explored the dynamics of interactions in which hosts were exploited jointly by parasitoids and pathogens, and particularly emphasized the potential for emergent complex dynamics, and alternative states where dominance is determined by initial conditions. Several authors (e.g., Packer et al. 2003, Ostfeld and Holt 2004, Hall et al. 2005) have recently examined the consequences of generalist predators attacking a prey that is also a host for a specialist pathogen. Polis et al. (1989) noted that "predators eat pathogens, parasites, and parasitoids when they eat the host," and referred to this as "coincidental intraguild predation." When predators consume infected hosts, they often directly kill pathogens. When predators consume uninfected

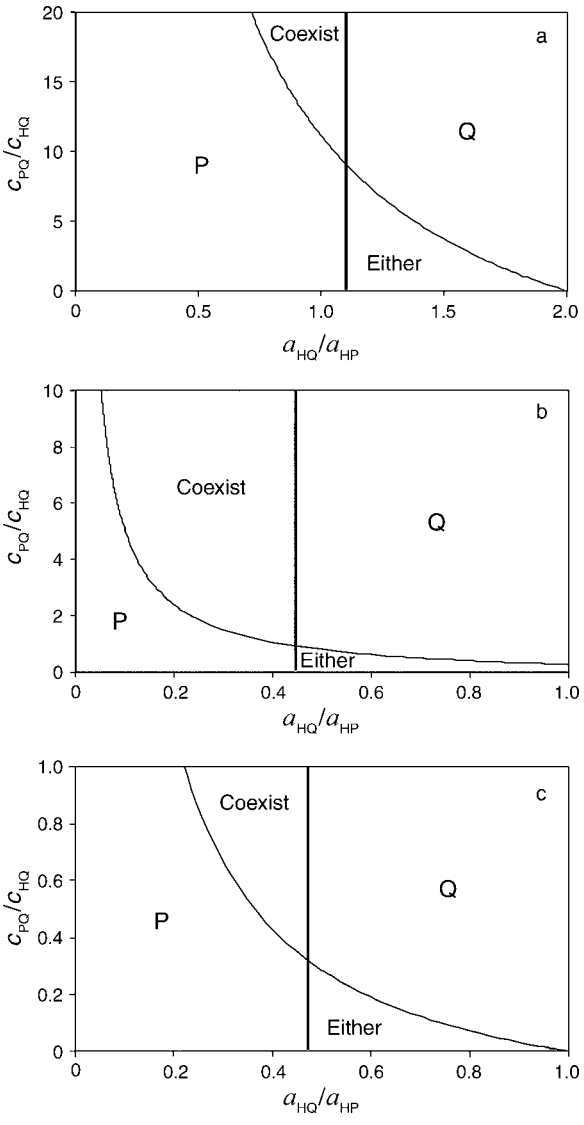

Fig. 2. Regions of potential coexistence for (a) predator–prey, (b) host–parasitoid, and (c) host–pathogen IGP formulations as functions of the relative attack rates of the two consumers on the basal resource (a_{HQ}/a_{HP}) and the ratio of the conversion efficiencies of the IG predator on the IG prey and basal resource (c_{PQ}/c_{HQ}). Lines delineate the regions in which each consumer species can invade a system with its competitor and the resource at equilibrium. In the region labeled "P," the IG prey extirpates the IG predator, whereas in the region labeled "Q," the IG predator extirpates the IG prey. Regions labeled "either" are those in which coexistence is not possible, but the species that persists depends on initial conditions. Parameter values were chosen to be system appropriate. In (a), $r = 1$, $K = 1000$, $a_{HP} = 0.1$, $a_{PQ} = a_{HQ}$, $c_{HP} = 0.1$, $c_{HQ} = 0.05$, and $d_P = d_Q = 1$. In (b), $r = 0.1$, $K = 1000$, $a_{HP} = 0.005$, $a_{PQ} = a_{HQ}$, $c_{HP} = 1$, $c_{HQ} = 0.5$, $d_{IP} = d_{IQ} = 0.001$, $m_P = m_Q = 0.025$, and $d_P = d_Q = 0.1$. In (c), $b = 0.001$, $d_H = 0.0001$, $K = 50\,000$, $a_{HP} = 0.0001$, $a_{PQ} = a_{HQ}$, $c_{HP} = c_{HQ} = 1$, $\gamma_P = \gamma_Q = 1$, and $d_P = d_Q = 0$. See the Appendix for information about parameter values.

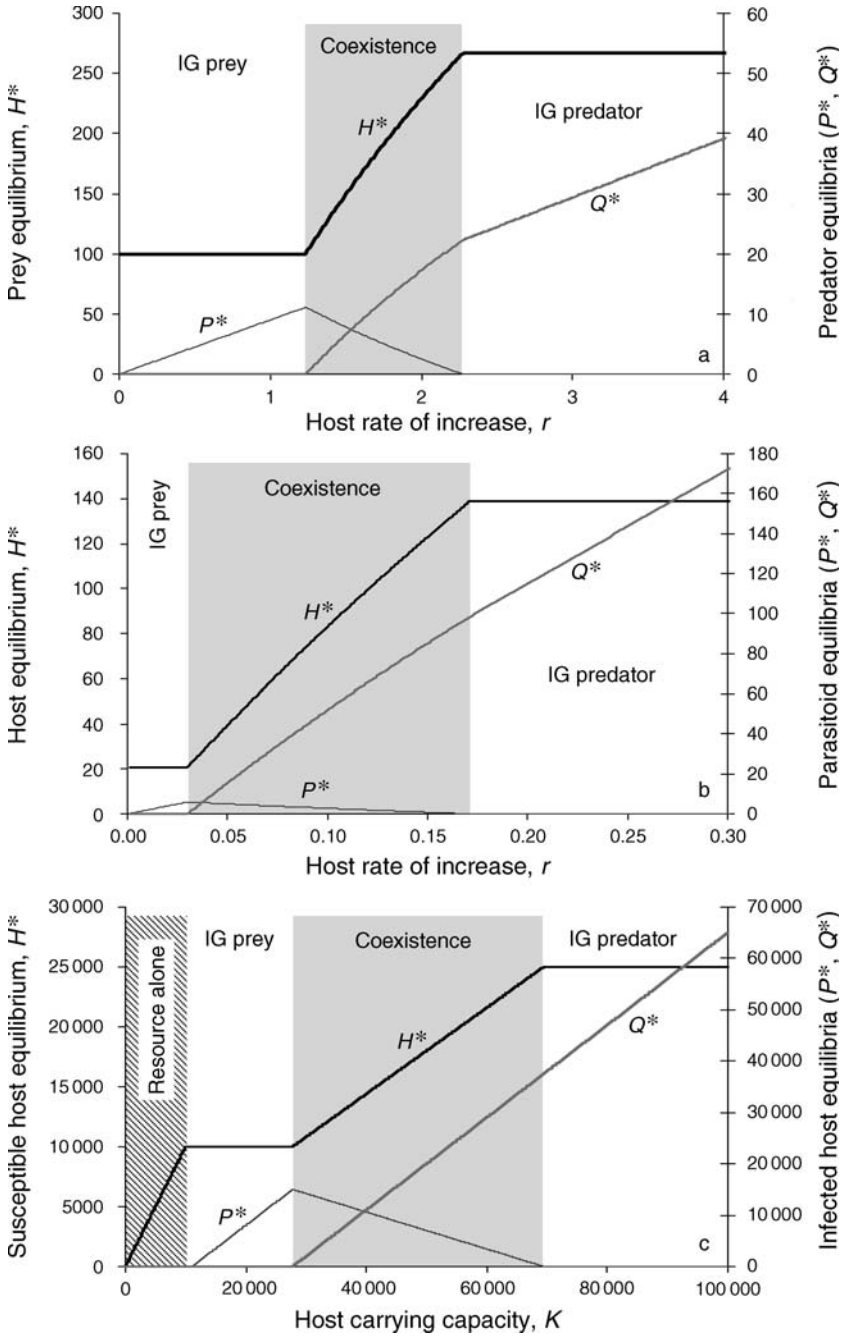

FIG. 3. Effects of increasing the productivity (either r or K) for the basal resource on the equilibrium abundances of the resource (H^*), IG prey (P^*), and IG predator (Q^*). At low productivities, only the IG prey can persist on the resource. At intermediate productivities, coexistence of the IG prey and IG predator is possible. At high productivities, the IG predator displaces the IG prey. At low values of K, neither consumer can persist on the basal resource (shown only for the directly transmitted disease case in Fig. 3c). All parameters are as in Fig. 2, with (a) $a_{HQ}/a_{HP} = 0.75$, $c_{PQ}/c_{HQ} = 15$, and varying r; (b) $a_{HQ}/a_{HP} = 0.3$, $c_{PQ}/c_{HQ} = 5$, and varying r; (c) $a_{HQ}/a_{HP} = 0.4$, $c_{PQ}/c_{HQ} = 1$, and varying K.

hosts, in effect there is indirect, exploitative competition between the predator and the pathogen. Packer et al. (2003) suggest that predation often will reduce infection prevalence in a host population (though this is not always the case when there is acquired immunity [Holt and Roy 2007]), and Hall et al. (2005) demonstrate that including a saturating functional response can generate unstable dynamics.

In these models (Packer et al. 2003, Hall et al. 2005, Holt and Roy 2007) the predator is assumed to be a

generalist, whose own numerical dynamics are decoupled from the host. In some cases, by contrast, predators themselves can be relatively specialized (e.g., the Canadian lynx is a near-specialist on the snowshoe hare), so the dynamics of the system could be much closer to that of the canonical IGP model discussed above. To our knowledge, the first paper to deal formally with this system was Anderson and May (1986), who spliced together the conventional Lotka-Volterra model with density-dependent disease transmission. Chattapodhyay et al. (2003) have recently examined a model combining exploitation by a predator and pathogen in some detail, with a model structure nearly identical to the IGP cases we have considered here, and they show that if the equilibrium with all species exists, then both the predator and pathogen can invade when rare. If the equilibrium is locally stable, then it is also globally stable. However, as in the canonical IGP model (Holt and Polis 1997) mutual invasibility does not imply stability. When the predator does not experience direct density dependence, the model is identical to that considered by Holt and Polis (1997). Moreover, all the qualitative conclusions emphasized by Holt and Polis (1997) should pertain to a specialist predator attacking a host with a specialist pathogen, when infected hosts neither reproduce nor participate in density dependent regulation.

While IGP models of predator–prey and parasitoid–host systems have received a great deal of attention from both empiricists and theoreticians, relatively little work has explicitly considered pathogen–host communities in the context of IGP theory. Yet, perhaps more than either of the other two community types, pathogen–host models correspond well to the tightly coupled and specialized exploitative linkages among species found in many real pathogen–host communities. Relatively few natural predator–prey systems exhibit the rigid specialization assumed in the predator–prey IGP model. By contrast, many pathogens are specialized to one or a few host species. Importantly, the relevance of IGP theory to host–pathogen communities suggests several possibilities for new avenues of inquiry. First, an explicit investigation of variable host productivity may produce interesting and relevant insights into pathogen-pathogen interactions in real communities (see Hochberg and Holt 1990). For example, our results demonstrate that the relative prevalence of pathogens exploiting a shared host should change across a host-productivity gradient. Second, while competition between pathogens sharing a host has received some attention (e.g., Dobson 1985, Hochberg and Holt 1990, Rohani et al. 2003, Holt and Dobson 2006), IGP theory suggests that quantifying the trade-off between the transmission rate and competitive replacement ability of two competing pathogens leads to direct predictions about pathogen prevalence rates in host populations. Finally, recovery of infected individuals can reduce the tight coupling between pathogens and hosts, and may lead to stable multi-pathogen–host interactions.

Key applications for IGP theory in pathogen–host communities are predicting dynamics of emerging diseases, conservation of rare species influenced by pathogens, and efficacy of introduced pathogens for biological control. Recent work has shown that exploitative abilities of competing pathogens (or predator–pathogen pairs) can determine the abundance of healthy (susceptible) hosts with a single pathogen vs. a more complex community (Dwyer et al. 2004). Explicit incorporation of IGP theory into inoculation programs may improve the implementation and outcome of this type of program.

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APPENDIX

Empirical basis of parameter ranges for focal parameters (*Ecological Archives* E088-163-A1).