How parasites affect interactions between competitors and predators

Abstract
We present a synthesis of empirical and theoretical work investigating how parasites influence competitive and predatory interactions between other species. We examine the direct and indirect effects of parasitism and discuss examples of density and parasite-induced trait-mediated effects. Recent work reveals previously unrecognized complexity in parasite-mediated interactions. In addition to parasite-modified and apparent competition leading to species exclusion or enabling coexistence, parasites and predators interact in different ways to regulate or destabilize the population dynamics of their joint prey. An emerging area is the impact of parasites on intraguild predation (IGP). Parasites can increase vulnerability of infected individuals to cannibalism or predation resulting in reversed species dominance in IGP hierarchies. We discuss the potential significance of parasites for community structure and biodiversity, in particular their role in promoting species exclusion or coexistence and the impact of emerging diseases. Ongoing invasions provide examples where parasites mediate native/invader interactions and play a key role in determining the outcome of invasions. We highlight the need for more quantitative data to assess the impact of parasites on communities, and the combination of theoretical and empirical studies to examine how the effects of parasitism scale up to community-level processes.

Keywords
Parasite, coexistence, indirect effect, competitor, predator, intraguild predation, community module, parasite-modified interactions, parasite-mediated interactions, reservoir.

INTRODUCTION
One of the biggest challenges facing ecologists today is to understand how, and to what extent, interspecific interactions influence community structure, species coexistence and biodiversity. Coupled with this is an increasing awareness of the potential importance of parasites and pathogens in determining the outcomes of trophic interactions (MacNeil et al. 2003a; Tompkins et al. 2003; Malmstrom et al. 2006) and community processes (de Castro & Bolker 2005; Mouritsen & Poulin 2005a; Collinge & Ray 2006; Wilmers et al. 2006). One way we can attempt to characterize a community is to decompose it into subsets of strongly interacting species (a community module: Holt 1997). Given that any realistic community is composed of many (hundreds) of modules (see, for example, Bascompte & Melian 2005), the resulting dynamics are potentially very complex and, indeed, a fully analytical understanding of how interactions between species translate into community structure may never be possible. Despite this potential intractability, a first step is to understand interactions within modules, and then attempt to examine the consequence of these module-level processes as they propagate through a community (Holt 1997). Although some modules have been examined in depth both empirically and theoretically, there are still many areas of uncertainty, particularly where modules involving parasitism are concerned (Holt & Dobson 2006). Here, we present a synthesis of current knowledge and indicate future directions for research into how parasites influence competitive and predatory interactions between the species they infect.

Parasites can potentially be important players in many different community modules, and can enter into a given module in different positions. For example, two species that
share a parasite may be competitors, intraguild predators, one may be prey for the other, or they may interact only via the effects of the parasite (Fig. 1). Furthermore, parasites can influence interactions between species even if they do not share the parasite. For instance, a parasite can, in principle, reduce the competitive strength of infected hosts against a non-host species. Alternatively, it can infect the prey but not the predator, or vice versa, altering prey capture rates as a result of effects on activity levels or behaviour of the infected host. In this review we synthesize theoretical and empirical studies that examine how parasites influence interactions in different community modules, namely, the modules of apparent competition via a shared parasite (Fig. 1a); resource competition between the hosts of a shared parasite (Fig. 1b); a specialist parasite of one of a competing species pair (Fig. 1c); predator–prey modules where the parasite infects the prey (Fig. 1d) or the predator (Fig. 1c) or both (Fig. 1f); and intraguild predation (IGP) with a shared parasite (Fig. 1g).

The modern approach to predicting the population dynamics of the two-species interaction between host and parasite was developed by Anderson & May (1981). One host–one parasite models such as these demonstrate that the population dynamics of hosts and parasites depend crucially on many different factors, including parasite transmission (rates or functions), stages in the parasite life-cycle, host mortality (parasite-induced or otherwise), and host immune or defence responses to infection. When additional interactions with other species or the environment are added, the scope for complexity of outcomes is enhanced (see, for example, Altizer et al. 2006; Keesing et al. 2006). In particular, the combination of two interactions involving three species can lead to dynamics that cannot be predicted from an analysis of the pairwise interactions alone (see, for example, Hochberg et al. 1990).

One reason why we cannot extrapolate multispecies interactions from pairwise components is that indirect interactions are also involved. Indirect effects occur when

![Figure 1 Interactions involving parasites.](image-url)
the impact of one species on another is mediated by the action of a third species. Abrams (1992) distinguishes two types of indirect effect: those mediated through population density effects propagated through an intervening species and those resulting from behavioural responses by one or more species to the presence or density of another. In principle, both types of indirect effect can be mediated by parasites, and in this review we will discuss potential examples of each. A classic example of the former, density-mediated indirect effect is apparent competition (Holt 1977), where two species that share a natural enemy have reciprocal negative effects (−) on each other’s population density via the positive numerical response of the enemy population. Behaviourally mediated indirect effects that affect the shared predation module include those generated by a predator’s aggregative response to patchily distributed prey and its functional response within patches, or prey refuge-seeking behaviour and reduced foraging in the presence of predators (Holt & Kotler 1987; Abrams 1992; Abrams & Matsuda 1996). Density and behaviourally mediated indirect effects are likely to act at different timescales (Holt & Kotler 1987; Abrams 1992). Density-mediated indirect effects result from changes in the density of one species affecting the demographic response of others, and so should take longer to propagate to other species than the effects of direct interactions between species. Behavioural effects occur on the same, or shorter, timescale as direct effects (Holt & Kotler 1987; Abrams 1992). Holt & Kotler (1987) examine how behavioural responses of predators can generate (−) indirect effects between prey species; this ‘short-term apparent competition’ is quite distinct from the classical numerical response-induced variety elucidated by Holt (1977).

These short-term processes are of interest for several reasons. Firstly, they can produce a much greater variety of interactions than those resulting from density alone; for instance, in the two prey-one shared enemy module (Figs 1a,b) all the combinations of indirect interactions (−), (+−), (−+) and (++) between the prey are theoretically possible (Holt & Kotler 1987; Abrams 1992; Abrams & Matsuda 1996). Secondly, because these are relatively fast processes, they could potentially be powerful in structuring populations and communities (Bolker et al. 2003). Thirdly, the variety of (often, counter-intuitive) relationships generated can have knock-on effects for community structure and stability, for instance generating (++) relationships between top predators and middle predators (Abrams 1992). There has been a recent resurgence of interest in short-term effects, now termed trait-mediated indirect effects (TMIEs), which include morphological, physiological and life-history changes as well as behaviour (e.g. Bolker et al. 2003; Werner & Peacor 2003). Most of the theoretical and empirical research on TMIEs has concentrated on predator–prey relationships, in part because there are clear examples of predator/prey foraging responses known from the discipline of behavioural ecology. Less work has focused on TMIEs in parasite–host relationships; one reason being that concepts from foraging theory and behavioural ecology have seldom been applied to parasites. For instance, there is no obvious analogue for parasites of handling time or predator functional response. Nevertheless, as parasites rarely kill their host (at least, not immediately), host population density is not the only characteristic that parasites can influence, so there should be great scope for TMIEs in parasitism modules. The degree to which short-term effects are important in parasite modules will thus depend on the type of TMIEs, if any, induced by parasites; we will also discuss possible examples of these.

In this review we take a bottom-up approach to understanding the role of parasites in community structure, concentrating on their role in community modules (see Holt & Dobson 2006 for a similar approach). We restrict our review to interactions involving parasites and pathogens (organisms that feed on a host individual, usually living on or in it and often causing harm but not immediate death), and do not include predators, parasitoids or herbivores (unless they are part of a parasite module). Interactions involving these other natural enemies and their victims have been very well reviewed elsewhere (Chase et al. 2002; Bolker et al. 2003; van Veen et al. 2006). Because our aim is to examine how parasites influence interactions between other species, the interactions considered here necessarily involve three (or more) species and we ignore the large and important literature on simple pairwise host–parasite interactions. We consider how parasites enter into and influence interactions for all the modules depicted in Fig. 1, reviewing the theoretical and empirical state of understanding for each. Apparent competition via a shared parasite (Fig. 1a), resource competition between hosts of a shared parasite (Fig. 1b), and a specialist parasite of one of a competing pair (Fig. 1c) are considered under the heading ‘Parasites and competitors’; in the section ‘Parasites and Predators’ we examine predator–prey interactions where the parasite infects the prey (Fig. 1d) or predator (Fig. 1e), or both (Fig. 1f). We also consider plant, plant pathogen and herbivore interactions (structurally similar to Fig. 1d) and IGP with a shared parasite (Fig. 1g). Whilst we do not examine them in general, simple tri-trophic food chains are represented by the prey–predator–parasite module of Fig. 1e and omnivory by the IGP module of Fig. 1g. Where theory or empirical examples allow, we also extrapolate to community-level outcomes of these module-level interactions. One omission is the competition module of parasite–parasite–host (for a recent overview see Holt & Dobson 2006); another omission is the simple food chain host–parasite–hyperparasite, which has received relatively little
research and undoubtedly deserves more (Holt & Hochberg 1998). We cover interactions involving plants and their pathogens and endophytes, but not in as much depth as we cover animal–parasite interactions. We go on to discuss the potential significance of parasites for community structure with reference to a relatively well-studied situation, namely the on-going 'natural experiments' represented by biological invasions. We conclude with a discussion of future research challenges, identifying a number of areas where theoretical and empirical data might be integrated to further our understanding of parasite influences on community structure.

**PARASITES AND COMPETITORS**

**Apparent and parasite-mediated competition**

A classic controlled study of the effects of parasites on competition was conducted by Park (1948), who demonstrated that infection by the shared parasite *Adelina triboli* reversed the outcome of competition between two species of flour beetle (*Tribolium castaneum* and *T. confusum*). Two terms have been used to describe this type of interaction. Two species that do not compete directly for resources can have deleterious effects on each other’s population sizes via a shared natural enemy; this is termed apparent competition (Holt 1977; Fig. 1a). Parasites, predators and parasitoids can be responsible for this effect (Holt 1977; Holt & Pickering 1985; Holt & Lawton 1993, 1994). More recently, parasite-mediated competition has been used to describe the situation when two species compete with one another, and a parasite (which may or may not be shared in both species) influences this competitive interaction (Fig. 1b,c). If the parasite has differential effects on the fitness of competing species, relative competitive strengths and hence population outcomes can be altered (Price et al. 1986; Hudson & Greenman 1998).

It might be argued that real systems will tend to lie somewhere on a continuum, with varying relative strengths of indirect effect mediated by the natural enemy and direct competition. Apparent competition was initially defined as a reciprocal negative relationship (–) between prey species (Holt 1977), but a recent analysis suggests that it is frequently asymmetric (Chaneton & Bonsall 2000). Although the original theoretical formulation of apparent competition assumes no direct competition between the victim species sharing a natural enemy (Holt 1977), in principle the process can nevertheless be important in modules with direct competition. Deconstructing systems into direct and indirect effects helps to clarify the problem: two competing species that share a parasite interact directly via use of the shared resource and indirectly via effects of the parasite on the other's population size. In some cases (when resources are not depleted), the system is dominated by the indirect effects and resembles pure apparent competition (Fig. 1a). In contrast, when competition is severe because resources are strongly limiting, the module is dominated by the direct interaction (which could, nonetheless, be strongly influenced by parasite modification of competitive strength: Fig. 1b). The term ‘parasite-mediated competition’ has not been explicitly defined and is often used when elements of apparent competition apply. In addition, it can be difficult to determine the strength of the direct competitive component, making it difficult to assess the relative importance of parasite-mediated apparent and direct competition (Hudson & Greenman 1998; Chaneton & Bonsall 2000). In this review, we will therefore use the term ‘apparent competition’ to refer to situations where the population processes involve indirect effects on population density mediated by the parasite, and the term ‘parasite-modified competition’ to describe situations where there is evidence of a direct effect of the parasite on the competitive ability of one (or more) of its hosts.

**Theoretical and empirical examples**

Apparent competition mediated by parasites was first considered explicitly by Holt & Pickering (1985), who examined two host species sharing one microparasite. The hosts did not compete intra- or inter-spezically, the aim being to examine whether the parasite alone could be responsible for regulating the host populations (Fig. 1a). The lack of any direct competition meant that, in the absence of the parasite, the host species would coexist (indeed, both populations would grow unchecked as they were not internally regulated). With the parasite present, three outcomes were possible, analogous to outcomes for interspecific competition: (i) repeatable exclusion of one host species (the one most affected by the parasite; if interspecific transmission is the same as or greater than intraspecific transmission and virulence differences between host species are of sufficient magnitude); (ii) both hosts coexist (if interspecific transmission is less than intraspecific transmission, but the two species are similarly affected by the parasite); (iii) exclusion of either species depending on initial (population density) conditions (this occurs when interspecific transmission is higher than intraspecific transmission). The two exclusion results capture the essence of apparent competition: the outcomes look like the result of interspecific competition, but no direct competition is involved. One host species acts as a reservoir for enemies of the other host and vice versa; the species that

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1Later studies by Park et al. (1965), however, showed that these species engaged in ‘cannibalistic predation’, what we would now call intraguild predation, and it is unclear if this latter interaction was parasite mediated in the original ‘competition’ experiments.
Anolis gingivinius is a stronger competitor than A. wattsi parasite sympatric populations are found only when the malarial infection where the parasite is not generally shared affects the excluded competitor to persist. Parasite-modified competition in infects the superior competitor, it can enable the otherwise initial conditions. If the parasite infects the inferior absence of the parasite, the standard three outcomes for which was also host to a microparasite (Fig. 1c). In the two species that compete intra- and interspecifically, one of number of simple community models, including the case for parasites cannot necessarily be subsumed within predator or parasitoid models; the dynamics of parasite systems of apparent competition are considerably richer.

Anderson & May (1986) reviewed and developed a number of simple community models, including the case for two species that compete intra- and interspecifically, one of which was also host to a microparasite (Fig. 1c). In the absence of the parasite, the standard three outcomes for interspecific competition apply: the superior competitor wins, there is stable coexistence, or the outcome depends on initial conditions. If the parasite infects the inferior competitor, there is no change in outcome. However, if it infects the superior competitor, it can enable the otherwise excluded competitor to persist. Parasite-modified competition where the parasite is not generally shared affects the distribution of Anolis lizards in the caribbean (Schall 1992). Anolis gingivinius is a stronger competitor than A. wattsi and sympatric populations are found only when the malarial parasite Plasmodium azurophilum is present. This parasite rarely infects A. wattsi but can be common in A. gingivinius. The parasite reduces the host’s competitive ability, permitting coexistence of the lizard species (Schall 1992). In this instance, the parasite appears to be responsible for maintaining species diversity. Holt & Dobson (2006) examine theoretically the conditions under which parasites play such pivotal (keystone) roles in a variety of community modules.

Begon et al. (1992) examined the case for a shared parasite, examining a microparasite and two host species with intraspecific competition but no interspecific competition. As above, repeatable exclusion, coexistence or exclusion dependent on initial conditions were the possible outcomes. The Begon et al. (1992) model is a more realistic representation of real communities as the hosts are regulated in absence of the parasite, but the similarity of general outcomes to Holt & Pickering’s (1985) simpler model is not surprising; the parasite simply imposes additional regulation onto that imposed by intraspecific competition. Bowers & Turner (1997) incorporated interspecific competition into this framework and showed that parasites could enhance or reverse the effects of direct competition. The outcome depends on which species can tolerate or support a higher prevalence of infection. If the parasite is more virulent in the superior competitor, it can reverse competitive outcomes, enabling the inferior competitor (which might otherwise have been excluded) to persist (i.e. it is a keystone parasite; Holt & Dobson 2006), or even excluding the superior competitor. If the parasite is more virulent in the inferior competitor, it can lead to elimination of that species. An empirical example of this is the replacement of the native Eurasian red squirrel in the UK by the introduced grey squirrel, which harbours a parapox virus that is avirulent in greys but highly virulent in reds (e.g. Tompkins et al. 2003). Model parameterization indicates that whilst competition alone cannot explain the speed and pattern of replacement, inclusion of the virus produces a very close fit to the data (Rushton et al. 2000; Tompkins et al. 2003). In field and laboratory manipulations of isopod crustaceans (Porellia saber and P. laevis), Grosholz (1992) discriminated between interspecific competition, intraspecific competition and viral parasitism to show that the shared virus caused apparent competition and reduced interspecific competitive ability in P. sabor. Many other empirical studies of microparasites (Schall 1992; Aliabadi & Juliano 2002; Juliano & Lounibos 2005) and macroparasites (Hanley et al. 1995) that have not been tested quantitatively appear to support these general findings. Several examples of pathogen-modified competition have been reported in plants (Alexander & Holt 1998; Power & Mitchell 2004; Malmstrom et al. 2005, 2006). Some grass species infected with Barley and Cereal Yellow Dwarf Viruses (B/CYDVs) suffer reduced competitive ability against other grasses that are less prone to the infection (Power & Mitchell 2004; Malmstrom et al. 2006). The replacement of native perennial bunchgrasses by invasive annual grasses has been attributed, in part, to the greater impact of B/CYDV on the competitive ability of the native (Malmstrom et al. 2005, 2006). Many plant–endophyte interactions show the reverse pattern: fungal endophytes that form symbioses with their hosts enhance the ability of the host in intra- and interspecific competition (reviewed in Clay & Schardl 2002). Such endophyte-induced effects can reduce plant diversity (Clay & Holah 1999) with important consequences for community structure at other trophic levels (Rudgers et al. 2004; discussed below).

Models for macroparasites bear out the general conclusions drawn for microparasites (Begon et al. 1992; Yan 1996; Greenman & Hudson 2000). However, macroparasite models tend to be tailored to specific systems owing to the peculiarities of parasite life history. Even relatively simple one host-one parasite systems can have dramatically
different dynamics depending on aspects of parasite lifecycle, so these models are best examined in the context of the systems they represent. A well-studied case is that of the shared nematode parasite (*Heterakis gallinarum*) which may underpin exclusion of the native UK grey partridge by the introduced ring-necked pheasant (Tompkins et al. 2000a). The decline in partridge numbers over the last 50 years has been attributed partially to apparent competition mediated by the parasite, which has a much larger impact on host fitness in the partridge. Parameterization of models revealed that the parasite cannot be maintained in partridge populations alone as its basic reproductive rate is too low in this host (Tompkins et al. 2000a). However, when the pheasant is present, it acts as a reservoir, boosting the density of susceptible hosts above the threshold required for parasite establishment (Tompkins et al. 2000b).

A frequently cited case where parasites change the outcome of inter-specific competition is that of the nematode *Paraphysoderma tenuis*, its gastropod intermediate hosts, its definitive host the white-tailed deer (*Odocoileus virginianus*) and at least two alternative hosts, moose (*Alces alces*) and caribou (*Rangifer tarandus*). Widespread declines in moose and caribou have been attributed to the parasite because it is largely benign in deer but usually fatal in moose and caribou, and the deer is thought to be an inferior competitor (e.g. Holt & Pickering 1985; Price et al. 1986). Schmitz & Nudds (1994) develop a detailed model of this system, deriving equations for the population dynamics of deer, moose, gastropods and three parasite life stages. The model has three basic outcomes reminiscent of the general microparasite models above: (i) competitive exclusion of deer (when parasitism is infrequent but competitive differences large); (ii) exclusion of moose (when parasite virulence and transmission are high and competitive differences small); (iii) stable or unstable coexistence of moose and deer (for other combinations of these factors). They conclude that although exclusion of moose is theoretically possible, co-existence is equally likely, and more robust estimates of parameters from empirical data are required to predict the outcomes of this interaction.

**Complexity in multiple host systems**

The potential complexity of systems is illustrated by the tick-borne louping ill virus in red grouse (*Lagopus lagopus scoticus*, Fig. 2). The virus is amplified in grouse, in which it causes substantial mortality. The virus is transmitted by a vector, the sheep tick (*Ixodes ricinus*) which feeds on a range of vertebrate species, but can only complete its lifecycle on larger mammals; virus can also be transmitted non-viraemically by co-feeding of ticks on mountain hare (e.g. Gilbert et al. 2001). On managed gamebird estates in upland Britain, three types of host are relevant: Red deer (*Cervus elaphus*) amplify the tick but do not amplify virus; mountain hares (*Lepus timidus*) amplify both tick and virus, and grouse amplify the virus but not the tick. A combination of models and empirical data have been used to investigate this system (Norman et al. 1999; Gilbert et al. 2001). Because of the asymmetries in vector/virus amplification, deer and grouse alone cannot maintain the virus. But in combination, the two host species can maintain viral infections. Likewise, the virus can be maintained in a hare–grouse community and is almost always maintained in the three-host species community. This system provides interesting insights into apparent competition: firstly, in agreement with Chaneton & Bonsall (2000), apparent competition can be asymmetric; for example, in the deer–grouse community, grouse suffer from apparent competition but deer do not. Secondly, vectors can also cause apparent competition, as deer amplify vector but not virus, but nevertheless their presence results in increased mortality for grouse. Vector-mediated apparent competition is also implicated in native/invasive grassland systems infected with B/CYDVs; in California, the invasive annual grass *Avena fatua* is more attractive to the aphid vectors and supports larger colonies than the more susceptible native perennial bunchgrass (*Malmstrom et al. 2005*). The louping ill system also serves to demonstrate some issues relevant to community composition. The suggestion from this system is that more complex communities of hosts are more likely to support a disease. This can be seen as an example of the reservoir effect of alternative host species. From the perspective of the grouse, hares and deer are reservoirs for the vector and hares are also a reservoir for the virus.

![Interaction web of the tick-borne louping ill virus and hosts of the virus and tick vector](image-url)
similar situation exists in B/CYDV and grass systems: *A. fatua*, a reservoir for the aphid vectors (Malmstrom et al. 2005), is also a reservoir for the virus (Power & Mitchell 2004). In experimental communities, these reservoir effects resulted in large increases in pathogen prevalence in other grass species when grown with infected *A. fatua* (Power & Mitchell 2004). This had knock-on effects for community composition via a combination of direct and pathogen-modified competition; adding BYDV reduced the competitive ability of *A. fatua* against a less susceptible grass, which then excluded two competitively inferior species (Power & Mitchell 2004).

In contrast to the reservoir effect, a dilution effect is possible, whereby the addition of more host species reduces the prevalence or likely persistence of a disease. This is also illustrated by loupung ill: very high densities of deer cause a dilution effect because infected ticks are more likely to bite deer, which do not transmit the infection, rather than grouse. In another tick-transmitted infection in the USA, Lyme disease (caused by the spirochaete *Borrelia burgdorferi*), higher species richness of small terrestrial mammals results in decreased disease risk as most of the mammals are incompetent hosts (Ostfeld & Keesing 2000). The relationship between dilution and reservoir effects is reviewed by Keesing et al. (2006), who make the point that the effect seen will depend on whether the most competent reservoir species is already present when a community undergoes change. If the best reservoir is already present, increasing species richness will result in dilution; if the current community has relatively incompetent hosts, adding more species, some of which are more competent hosts, will tend to increase disease incidence. This pattern is borne out by experimental manipulations of grassland communities showing that reduced species richness tended to increase foliar fungal pathogen load (Mitchell et al. 2002). Variation in outcomes could be attributed to a combination of species composition effects (pathogen load was higher in species-poor communities provided the more susceptible grasses were retained) and spatial effects on transmission (transmission of the more species-specific pathogens is more efficient in dense stands).

To examine conditions for parasite establishment in two-host communities, Holt et al. (2003) develop a graphical isocline approach where hosts are treated as resources. They show how the criterion for parasite establishment depends on a threshold community composition, an extension of the concept of the threshold host population size for invasion. The shape of the threshold depends on how the hosts interact (e.g. with respect to parasite transmission), and patterns analogous to resource-consumer isoclines are produced. Although not providing quantitative predictions for any particular system, this approach can be useful in classifying and comparing different systems and could be extended to communities with more host species. For instance, Holt et al. (2003) observe that, of the many possible isocline shapes, only one produces a dilution effect; this shape is only possible for vector-borne diseases or those with free-living infectious stages, bearing out the dilution phenomena found for loupung ill and Lyme disease. Recent analyses by Dobson (2004) and Rudolf & Antonovics (2005) incorporating frequency-dependent parasite transmission (the likely form for vector-borne diseases) also find a dilution effect. An interesting implication of these results is that, in contrast to apparent competition models with density-dependent transmission, frequency-dependent transmission can induce apparent mutualism (i.e. a ++ interaction between the host species; Abrams & Matsuda 1996), whereby the presence of one host species enables persistence of another host that would otherwise be driven extinct by the parasite (Rudolf & Antonovics 2005). The implications of this type of positive feedback for biodiversity are potentially important: reduced biodiversity could increase disease incidence, causing further extinctions in a cascading process (Dobson 2004; Keesing et al. 2006).

**Evidence for trait-mediated indirect effects**

Classically, parasite-mediated apparent competition acts via long-term density effects (Holt 1977), and the general models considered here (Holt & Pickering 1985; Begon et al. 1992; Holt et al. 2003) reflect that mechanism. However, in a community module context, parasite-induced modification of competitive ability can be seen as a TMIE altering the dominance relationship between competing species. This would appear to be the mechanism underlying several of the plant examples, where parasite-modified competition is documented (Power & Mitchell 2004; Malmstrom et al. 2005). It is less clear whether parasite modification of competition occurs between animal species, although it appears to be a factor for isopods (Grosholz 1992) and may play a role in the lizard (Schall 1992) and squirrel systems (Tompkins et al. 2003). The most widely applicable general population dynamic model for interspecific competition (Bowers & Turner 1997) does not incorporate this effect because parasitism only influences outcomes via its effects on mortality (a density-mediated effect); a model of parasite-modified competition should, strictly speaking, incorporate competition parameters that depend upon infection status. Other potential TMIEs do exist in the animal systems: parasite-induced effects on fecundity can be important and have been included in some models (Yan 1996; Greenman & Hudson 2000; Tompkins et al. 2000a); parasite-induced changes in intermediate host behaviour are another example (Schmitz & Nudds 1994). Other potential TMIEs arise in vector-borne parasite systems generally, where vector foraging behaviour may result in TMIEs common to many
other arthropod predators (such as a saturating functional response). For predator–prey systems, these types of short-term effect are known theoretically to result in counter-intuitive dynamics that could counteract long-term processes (Abrams & Matsuda 1996). It is interesting to note that the model of frequency-dependent transmission studied by Rudolf & Antonovics (2005) attributes to the parasite many of the characteristics that are required by Abrams & Matsuda (1996) to produce apparent mutualism, and a similar outcome is found.

PARASITES AND PREDATORS

In real systems, parasites affect interactions between species at the same and also at different trophic levels. Parasites may impact predatory interactions in different ways, depending on whether they infect the prey (Fig. 1d), the predator (Fig. 1c), or both predator and prey (Fig. 1f). Anderson & May (1986) examined two cases: parasite of prey only (Fig. 1d) and parasite of predator only (Fig. 1e). They assumed that a microparasite invades an established predator–prey interaction, under a range of assumptions for density dependence, immunity and fitness effects. When parasites infect prey, if predators hold the prey population below the threshold density for parasite invasion, the parasite cannot spread. At the other extreme, the parasite may regulate the host below the level required to sustain the predator, in which case the predator is excluded via direct competition with the parasite. Within these two limits, the parasite, prey and predator coexist, in some cases with oscillatory dynamics that would not have occurred in the absence of the parasite. Similar outcomes are also found for the case of a parasitoid and pathogen interacting with a shared arthropod host species (Hochberg et al. 1990). However, the situation is more complicated because dual infections within a single host can occur, and the pathogen and parasitoid can interact directly. Hochberg et al. (1990) included interference competition between the pathogen and parasitoid when infecting the same host individual, and found a wide range of population dynamic outcomes. Regulation of the host population by both enemies was possible, but in some cases addition of the second enemy had a destabilizing effect. Conditions enhancing regulation included long-lived external pathogen propagules, roughly symmetrical interference, and aggregated attack by both enemies. At the other extreme, if attacks by both enemies were distributed at random, introduction of a second enemy increased host population size (Hochberg et al. 1990). Dwyer et al. (2004) show how two enemies can have a differential impact depending on prey density, resulting in complex dynamics for the host. Their model was tailored to forest Lepidoptera such as the gypsy moth Lymantria dispar and their baculovirus pathogens; periodic outbreaks of various moth species result in large-scale defoliation. Previously, pathogens had been implicated as regulatory factors, but parasite–host models failed to predict the variable time between outbreaks of the moth. Predator–prey models also failed to describe the dynamics, predicting longer cycle times between outbreaks. However, generalist predators and a specialist pathogen acting together can explain variable periodic outbreaks quite well (Dwyer et al. 2004): there is a low-density equilibrium where the predator regulates the host and the pathogen is relatively unimportant, and a high-density equilibrium controlled by the pathogen where the predator is relatively unimportant; adding a small amount of environmental stochasticity drives the trajectory unpredictably between the two.

Anderson & May (1986) also investigated a parasite infecting the predator rather than the prey (Fig. 1e). In this case, either the parasite cannot be maintained (if the predator, in the absence of the parasite, is at a density lower than the threshold for parasite invasion), or all three species coexist (if the converse is true). As is the case for parasites of prey interacting with predators, previously stable predator–prey dynamics may become oscillatory when a parasite of the predator is introduced (Anderson & May 1986). A recent analysis of population census data demonstrates this effect and also shows that parasites can indirectly affect population dynamics for the host and its prey even after the disease outbreak has been extinguished (Wilmers et al. 2006). In the early 1980s, canine parvovirus (CPV) was introduced to wolves (Canis lupus) in Isle Royale, USA, resulting in a dramatic crash of the wolf population. Since then, the population density of moose (Alces alces) has shown increased variance with peaks and crashes characteristic of populations relatively free from predation. Regulation of the moose population appears to have shifted from top-down (wolf) to bottom-up control, principally through climatic effects on moose and their winter food resource (Wilmers et al. 2006). That pathogen outbreaks may be responsible for shifts in trophic control is an exciting advance that warrants further empirical and theoretical attention. Furthermore, these effects appear to be long-lasting; although CPV is no longer present on Isle Royale, wolf and moose populations remain in the post-1980s pattern, possibly because genetic bottlenecks were generated by the crash and these prevent recovery of the wolf population.

Short-term and trait-mediated indirect effects: empirical evidence

Historically, emphasis has been placed on the role of parasites in behavioural manipulation of prey intermediate hosts thereby facilitating parasite transmission to the predator definitive host (Moore 2002). These systems are examples of the community module where the parasite
infects both predator and prey (Fig. 1f). In a community context, these parasite-induced behavioural changes can be regarded as TMIEs that often result in increased exposure of hosts to predation (Werner & Peacock 2003; Mouritsen & Poulin 2005a). Although some changes in host behaviour may be a by-product of infection, for others there is strong evidence for adaptive manipulation by the parasite (Poulin 1995). For example, behavioural manipulation of the intermediate amphipod host, Gammarus pulex, by the acanthocephalan Polymorphus paradoxus coincides with the infective stage of the parasite and increases predation risk of the shrimps from the definitive duck hosts (Bethel & Holmes 1977).

Cezilly & Perrot-Minnot (2005) review a number of examples of trophically transmitted parasites where manipulation increases predation by both host and non-host species. For example, although the definitive hosts of the acanthocephalan Polymorphus minutus are aquatic birds, P. minutus increased the susceptibility of G. pulex to fish predators (Marriott et al. 1989). Decreased aggregation of the tapeworm Echinococcus granulosus in wild moose populations under high levels of wolf predation indicates selective predation on infected hosts (Joly & Messier 2004). Red grouse killed by predators were found to have higher burdens of the nematode Trichostonglyus tenius, and predator control lead to an increase in the numbers of heavily infected birds, indicating that predators selectively prey on infected birds (Hudson et al. 1992). In a direct test for parasite-induced predation, field populations of voles (Microtus townsendii) that were treated with anthelminthics suffered a 17% reduction in predation in comparison with untreated (infected) controls (Steen et al. 2002). Hence, parasitic manipulation may affect predator–prey relationships beyond those involved in the life-cycle of the parasite, producing TMIEs in modules such as that depicted in Fig. 1d; indeed, most theoretical treatments of this problem (described in the following section) examine systems in which the predators are not part of the parasite lifecycle.

A series of field studies have demonstrated the wide reaching effects of trematode-induced behavioural changes in the cockle, Austrovenus stutchburyi (Mouritsen & Poulin 2003, 2005a,b). The trematode Curturia australis inhibits the burrowing behaviour of infected cockles, thereby exposing them to predation by the avian definitive host (Mouritsen & Poulin 2003, 2005b). This effect also leads to increased non-lethal predation (foot cropping) of the cockles by fish (Mouritsen & Poulin 2003). Changes in cockle zonation as a result of the infection alter the distribution of anemones that live on the cockles as well as the distribution of whelk and limpet predators (Mouritsen & Poulin 2005b). Changes in cockle burrowing lead to alterations in sediment structure, an increase in the density of some species and an increase in biodiversity in the intertidal zone, leading Mouritsen & Poulin (2005a) to advocate this parasite as an ‘ecosystem engineer’.

Population dynamic and community consequences

Hudson et al. (1992) parameterized a mathematical model of the grouse–nematode system (an example of Fig. 1d) to examine the effects of predation. Predators were represented as a parameter (rather than having explicit equations to represent population numbers), so this model is more appropriate for long-lived or generalist predators whose population dynamics are unlikely to be coupled to that of a single host species. If predators were non-selective or preferred infected individuals, increased predation reduced mean parasite burden in the grouse population, resulting in a reduction in the amplitude of oscillations in grouse numbers. Hudson et al. (1992) argued that because selective predation removes a disproportionate fraction of parasites, it reduces the regulatory role of parasites (in this case, reducing parasite-driven oscillations caused by delayed density-dependent effects on host fecundity). In contrast, Ives & Murray (1997) found that parasitism and predation acting together increased oscillations in a model of snowshoe hare (Lepus americanus) populations. They argued that parasites tend to destabilize predator–prey dynamics as adding parasites to a Lotka–Volterra predator–prey model (here, predator dynamics were included) shifted damped oscillations towards long-term cycles or diverging oscillations, mirroring the results of Anderson & May (1986). There are a number of possible explanations for this discrepancy, based on the different perspectives taken: Hudson et al. (1998) add predators to a host–parasite system where the parasites have a strong regulatory influence on the host even in the absence of predation; Ives & Murray add parasites to a predator–prey system where cycles in prey numbers are thought to be largely driven by predation. Importantly, the models differ in their treatment of short-term effects; Hudson et al. concentrate on short-term effects, considering the numerical response of predator density to be independent of prey numbers (this is also a feature of several other models discussed below); Ives & Murray include long-term density-mediated effects via predator population dynamics, both models include TMIEs in the form of parasite-induced changes in fecundity and vulnerability to predation.

Two general theoretical papers examine the problem from these different perspectives with broadly consistent results. Packer et al. (2003) examine the effect of predation on host/parasite systems, in relation to strategies for population management. They show that predator removal, commonly regarded as a potential strategy for boosting prey numbers, can harm host populations that are also subject to parasitic infection; predator dynamics were not modelled.
explicitly. For microparasites, they found that increased predation always increases the abundance of healthy prey and reduces the prevalence of infection. Surprisingly, this occurs even if the predator is unselective. Selectivity is, however, important in determining the consequences for total population size (summing infected and uninfected individuals): if predators select infected hosts, they are predicted to increase total host population size; if they prefer uninfected hosts they will reduce population size and if they are unselective then total population size remains unaltered. For macroparasites that have an aggregated distribution among hosts, the effect is more extreme. Even a low level of predation could in theory result in extinction of the parasite if predators concentrate on infected prey. Packer et al. observe that selective predation effectively increases parasite-induced mortality, thus it tends to increase the impact of parasitism as a stabilizing/ regulatory force on host populations. If predator removal boosts parasite numbers, this can have implications for public health, as many managed populations of mammals and birds harbour zoonotic diseases (Ostfeld & Holt 2004). The alternative approach was taken by Hethcote et al. (2004) who modified a Lotka-Volterra type predator–prey model with a model for microparasite infection of the prey. In some cases, the greater vulnerability of infected prey can allow persistence of a predator which would go extinct in the absence of infection; in this model the infected prey provide an ‘easy’ resource for the (otherwise too inefficient) predators to exploit. Predation could also lead to extinction of a parasite that could otherwise persist in its absence. Like Packer et al.’s result, this can occur even if predators are unselective: predation reduces the host population below the threshold density required to sustain infection (Anderson & May 1986).

A detailed investigation of the interplay between predation and parasitism is presented in Hall et al. (2005). They model predator behaviour assuming that predators have a saturating (type II) functional response to prey density, predator density was not modelled explicitly; this, then, is a study of short-term indirect effects (and, arguably, TMIEs; cf. Bolker et al. 2003; Werner & Peacor 2003). Once a saturating response is included, predators that preferentially prey on infected hosts can introduce an Allee effect on the parasite driving it extinct if it falls below a threshold population size. Invading parasites not only require a minimum host population size for invasion, they must also exceed a critical propagule size of infected individuals. Parasites are well-known theoretically (e.g. Anderson & May 1981, 1986) and empirically (Hudson et al. 1998) to drive population oscillations in the absence of any other forces; the addition of a predator with saturating functional response can destabilize the system further with the result that oscillations become catastrophic for the parasite if the Allee critical point is reached. If, on the other hand, the predator is non-selective (or prefers uninfected hosts), the combined forces of parasitism and predation acting in synergy may drive the host to extinction. Why does a type II functional response have these effects? A saturating response means that although at high prey densities the relative impact of predation is lower (compared with a linear response), at low prey densities it is higher. This has a destabilizing effect: regulation at high densities is weaker, and at low densities the prey cannot recover from the pressure of predation. Hall et al. suggest that these dynamics might explain empirical observations of epidemic invasions with very sudden crashes and apparent extinction after only one or two oscillations. In many lakes in midwestern and north-eastern USA, the bluegill sunfish Lepomis macrochirus appears to control outbreaks of the bacterium Spirobacillus cienkowskii in Daphnia dentifera. Bluegills preying on daphniids exhibit a type II response and they prey selectively on infected hosts, characteristics that can generate Allee effects in the parasite. Epidemics occur in response to seasonal decreases in fish predation but die out after only a single oscillation (Duffy et al. 2005); if predation is sufficiently intense and selective, invasion of the parasite into host populations may be prevented (Duffy et al. 2005).

PLANT PATHOGENS/ENDOPHYES AND HERBIVORES

Plant pathogens can alter their host’s interactions with other natural enemies, chief among them insect herbivores. There are numerous empirical examples of negative (and some positive) direct and indirect effects of pathogens on phytophagous insects (reviewed in Hatcher 1995; Stout et al. 2006), but the population dynamic and community consequences of these interactions are less clear (Stout et al. 2006). One extensively studied system is that of dock (Rumex spp.), attacked by the rust Uromyces rumicis and the beetle Gastrophysa viridula. In laboratory manipulations, rust infection of Rumex substantially reduced growth, survivorship and fecundity of the beetle (Hatcher et al. 1994). Field and laboratory studies demonstrate that beetle grazing induces resistance to infection by Uromyces and two other fungal pathogens (Hatcher et al. 1994; Hatcher & Paul 2000). The existence of bidirectional inhibitory interactions makes it difficult to predict the effect of combined attack by multiple natural enemies on plants, a potentially important strategy in biocontrol. Pathogen-induced deleterious effects on insect herbivores are, in part, caused by reduced nutritional quality (and, in some cases, quantity) of infected plants. Another important mechanism mediating pathogen–herbivore negative effects is that of induced defence: although pathogens and herbivores may activate different signalling pathways in the plants they attack, there is
considerable overlap in responses such that plant responses to pathogen infection confer some resistance to herbivory and vice versa (reviewed in Hatcher et al. 2004; Stout et al. 2006).

Fungal endophytes also confer resistance to attack by herbivores. Many fungal endophytes have limited (or no demonstrable) direct negative effects on their hosts and are generally considered to be mutualists; however, in many cases the positive effects they impart on the host are apparent only when the host interacts with members of other species. Recent work suggests that endophyte-modified interspecific competition may have important consequences for community structure and ecosystem processes (Rudgers et al. 2004). Endophyte-infected grasses have higher alkaloid levels reducing plant nutritional quality and making them less attractive to insect herbivores (Clay & Schardl 2002). Such grasses may also support a lower abundance of aphids, with knock-on effects at higher trophic levels (Omacini et al. 2001). The combined effects of endophytes on interspecific competition and herbivory can alter the balance between trophic levels, implicating these microbes in extensive trophic cascades. Laboratory experiments suggest that endophytes can reduce plant diversity through dominance of endophyte-infected species in interspecific competition; this (together with the direct effects on insect feeding) reduces phytophagous insect abundance, resulting in reduced species richness of specialist predators and parasitoids of insect herbivores (Omacini et al. 2001). Field manipulations demonstrate reduced diversity in endophyte-infected communities (Clay & Holah 1999; Rudgers et al. 2004) and suggest that endophytes can also affect the composition of the detritivore and generalist predator assemblages (Lemons et al. 2005; Finkes et al. 2006). Pathogen and endophyte modification of plant quality and defensive strategy, and modification of interspecific competitive ability, can all be regarded as TMIEs.

PARASITES AND INTRAGUILD PREDATORS

Intraguild predation is predation among species that are also potential competitors (Polis et al. 1989; Holt & Polis 1997). Recent analyses of real food webs indicate that IGP is widespread and important in the structuring of communities (Arim & Marquet 2004; Bascompte & Melian 2005). It often occurs among closely related species and can be associated with (and perhaps predicted from) a cannibalistic tendency (see Polis et al. 1989; Dick et al. 1993), as the ability to kill and consume conspecifics may easily transcribe into predation on congenerics/confamilials (e.g. Dick et al. 1993). IGP appears to be particularly prevalent where competing species have age or stage structure; smaller stage classes are eaten by the larger classes of competing species that consume the same general class of resource; in such cases many species may be involved in IGP to varying degrees (Polis & Holt 1992). Disparate taxa also engage in IGP, as its definition extends to similarity of resources used regardless of taxonomy or mode of resource acquisition (Polis et al. 1989). For instance, pathogen–parasitoid interactions when both enemies attack the same host species, and the pathogen also infects the parasitoid, can be regarded as IGP (Rosenheim et al. 1995).

Intraguild predation can be asymmetric (i.e. A eats B only), symmetric or mutual (i.e. A eats B, B eats A), or mutual but with different strengths of interaction (Polis et al. 1989; Dick et al. 1993). Mathematical analyses have so far mainly considered the dynamics and energetics of two competing species with asymmetric IGP (the IG predator and IG prey) and their shared resource (Polis et al. 1989; Holt 1997; Holt & Polis 1997; Ruggieri & Schreiber 2005; but see Dick et al. 1993; Dick & Platvoet 1996 where mutual IGP is included). Theory predicts that IGP can produce complex patterns of population dynamics including oscillations and stable equilibria with species exclusion, co-existence or alternative stable states (Holt & Polis 1997). Outcomes depend on the relative strength of IGP, and the relative efficiency of exploitation of the basal resource by the competitors; for IGP to persist, the IG prey must be a more efficient utilizer of the shared resource than the IG predator (Holt & Polis 1997). The existence of alternative stable states implies that the eventual outcome depends on initial conditions such as the order of arrival of species or their initial propagule size; this might translate into patchwork distributions of particular IGP assemblages within structured habitats (Polis & Holt 1992).

Theory predicts that IGP can reverse the outcomes predicted for simple (linear) food chains (Polis & Holt 1992; Holt & Polis 1997). For instance, in a trophic cascade, an increase in top predator numbers decreases intermediate predator densities, allowing herbivores to increase, which depresses plant biomass. Adding IGP can prevent the cascade or even reverse it, such that top predators have a positive impact on plants via their effect on herbivores (Polis et al. 1989; Polis & Holt 1992). IGP can be disruptive in biological control, as target prey may be ‘released’ from predation due to IGP among bio-control species (Müller & Brodeur 2002), but it can also be an important factor in conservation, controlling ‘mesopredators’ that are otherwise detrimental to prey populations (Müller & Brodeur 2002).

Whilst IGP gains currency as an important factor in community structure, the role of parasites in mediating IGP has received less attention (Fig. 1g). This is important as all the direct and indirect effects of IGP as reviewed above may be changed by parasite modification of IGP interaction strength. The same features of hosts that make them vulnerable to behavioural manipulation by parasites may
render such hosts more susceptible to intraguild predators. Parasite-induced effects on host condition, behaviour or habitat use could, in principle, alter either the prey or predatory aspects of IGP relationships, whether or not the parasite is actually shared between species. Two species of parasite have been shown to influence both the prey and predatory components of IGP in a guild of native and invasive amphipods (MacNeil et al. 2003a,b). The freshwaters of Ireland have been invaded by three amphipod species: Gammarus pulex from mainland Europe and the North American G. tigrinus and Crangonyx pseudogracilis. The invaders have excluded the native G. duebeni celticus from some sites, but in others, natives and invaders co-exist (MacNeil et al. 2003a). Gammarus d. celticus sits midway in a hierarchy of IGP with the invading species (Fig. 3). Field and laboratory experiments show that parasitism alters the hierarchy of IGP and therefore has the capacity to affect the outcome of invasions. Infection by the microsporidian Pleistophora mulleri had no direct effect on the survival of the native G. d. celticus, but infected adults showed a reduced capacity to prey on the smaller species G. tigrinus and C. pseudogracilis and an increased vulnerability to predation by G. pulex, none of which are prone to this infection (MacNeil et al. 2003a). A second parasite, the acanthocephalan Echinorhynchus truttae is more prevalent in the invader Gammarus pulex than in the native G. d. celticus. Here, infection reduces predation by G. pulex on G. d. celticus and may therefore slow the invasion or promote coexistence of these amphipods (MacNeil et al. 2003b). Parasitism also alters inter-guild predation (Fielding et al. 2003) and cannibalistic interactions (MacNeil et al. 2003c) in this system. All the parasite-induced effects measured in this system are short-term (sensu Holt & Kotler 1987; Abrams 1992) and can be seen as TMIEs.

Several recent papers investigating apparent competition speculate that parasite-mediated IGP might be important and clearly more work is needed to study this directly (Hoogendoorn & Heimpel 2002; Juliano & Lounibos 2005). For example, in a laboratory study, Yan et al. (1998) investigated the effect of the tapeworm Hymenolepis diminuta on competition between Tribolium castaneum and T. confusum. Tribolium castaneum, the stronger competitor, suffers higher infection and fitness costs from parasitism and thus the parasite should be expected to reverse the outcome of competition. However, the reverse occurred with a faster exclusion of T. confusum than occurred in the absence of infection. The authors suggested that parasite-induced changes in surface seeking behaviour might lead to higher levels of IGP by T. castaneum. This is a fruitful area for further research, particularly where IGP systems have been well studied but parasites largely ignored; in this respect, well-characterized systems, such as Park’s (1948) and Park et al.’s (1965) Tribolium competition/cannibalistic predation experiments, may need to be revisited.

**Figure 3** Interaction web of native and invading Gammarus spp. (see MacNeil et al. 2003a,b). Shown are four amphipod species that engage in IGP, their common resource base; two parasite species of gammarids (the microsporidian, P. mulleri, has a direct lifecycle whereas the acanthocephalan, E. truttae, uses brown trout Salmo trutta as its definitive host); and a top predator of gammarids (S. trutta). Not shown are other top predators without direct links to these parasites or other guild members that do not participate in IGP with gammarids. Also not shown are probable links between E. truttae to G. d. celticus and cannibalistic interactions, which occur within each amphipod species and are also be modified by parasitism (MacNeil et al. 2003c). Indirect interactions include: P. mulleri-mediated IGP between (G. pulex, G. d. celticus) and (G. d. celticus, G. tigrinus); E. truttae-mediated IGP between (G. pulex and G. d. celticus); behavioural manipulation of G. pulex to enhance transmission of E. truttae to S. trutta is also documented.
& Mitchell 2004; but see Colautti et al. 2005). A meta-analysis of 473 plant species naturalized to the United States from Europe demonstrated that they have, on average, 84% fewer fungal pathogens and 24% fewer virus species than those from the native range, and those species with greater release from enemies were more likely to be reported as harmful invaders (Mitchell & Power 2003). The invasive European green crab Carcinus maenas experiences greatly reduced parasite diversity and prevalence in the invasive range and the greater biomass in invasive populations has been attributed to enemy release (Torchin et al. 2001). Several amphipod species have invaded British Isles waters and these invaders enjoy a lower diversity, prevalence and burden of parasites than does the native amphipod Gammarus d. celticus (Prenter et al. 2004). Infection of the native amphipod G. d. celticus with the microsporidian *P. mulleri* increases its vulnerability to invasions by amphipod species that are not host to this parasite (MacNeil et al. 2003a; detailed in previous section). However, invaders do not always benefit from enemy release and, in the same system, the shared acanthocephalan parasite *E. truttae* is more prevalent in the invading *G. pulex* causing a reduction in its predatory abilities and slowing invasion and replacement (MacNeil et al. 2003b). For plants, the impact of invasive species appears to be a function of release from enemies of the native range and accumulation of novel enemies in their naturalized range (Mitchell & Power 2003). In addition, the net effect of multiple enemies is likely to be temporally and spatially variable, with little correspondence between escape from different enemies, creating ‘invasion opportunity windows’ for introduced species (Agrawal et al. 2005).

Parasites that invade with their hosts or vectors experience opportunities for transmission to new species. For example, in New Zealand, introduction and range expansion of exotic mosquitoes is likely to increase incidence of avian malaria, threatening native birds (Tompkins & Poulin 2006). The parapox virus introduced to the UK with the grey squirrel is highly virulent to the native red squirrel and is an important factor in its decline (Tompkins et al. 2003). Similarly, extinction of populations of the native UK White Clawed crayfish, Austropotamobius pallipes has been attributed to crayfish plague (*Aphanomyces astaci*). This fungus is thought to have been introduced with the invader *Paaffastacus leniusculus* in the 1970s. The parasite is widespread in *P. leniusculus* where it is rarely lethal, but it can cause high mortality in *A. pallipes* (Holdich 2003). That the disease is more virulent in the native may be an evolutionary accident and hence intrinsically unpredictable. Alternatively, tests of local adaptation may be important in predicting the likely impact of shared parasites on invasions. For example, the acanthocephalan parasite *Pomphorhynchus laevis* is shared by both the native amphipod Gammarus pulex and the invading *G. roeseli* in rivers in France. Whilst the parasite induces photophilic behaviour in *G. duiveni*, it does not alter phototaxis in the recent invader, probably reflecting their short coevolutionary history (Bauer et al. 2000). The absence of behavioural manipulation is likely to lead to less intense predation of the invader and may therefore facilitate coexistence with or even exclusion of the native amphipod.

Parasites may determine the speed of invasion, as seen in the case of red squirrel replacement by greys (Rushton et al. 2000; Tompkins et al. 2003). This is particularly likely where ‘strong’ interactions that result in rapid ecological change, such as those involving IGP, are concerned. IGP tends to destabilize interactions between competitors; this can drive one species extinct faster or over more of the parameter space than predicted for exploitative competition alone (Polis et al. 1989; Dick et al. 1993). Classic traits that are measured as evidence of interspecific competition, such as growth and fecundity, may have no time to respond measurably before species exclusion as a result of faster processes such as IGP. For example, IGP among amphipods can lead to eliminations of the inferior IGP participant in weeks/months (Dick & Platvoet 2000), with the lifespans of animals measured in months/years. Some studies (e.g. Polis et al. 1989; Yasuda et al. 2004) show IGP to be a powerful interspecific interaction, but competitive effects to be minimal or absent. Thus, models of IGP and its parasite mediation may be realistic when excluding competitive effects because competition may not have the time or strength to manifest. Indeed, Dick et al. (1993) and Dick & Platvoet (1996) showed theoretically that even a heavy bias in favour of the competitive ability of an inferior IGP participant could not overturn the superior IGP abilities of the inferior competitor, leading to exclusion of the inferior IGP participant. Short-term experiments examining IGP have been criticized for ignoring alternative resources (Briggs & Borer 2005), however, they may be appropriate in the situations above where competition is of little consequence.

**FUTURE CHALLENGES**

**Mathematical tractability**

With the addition of more species or more realistic life-history assumptions, many models of apparent competition are analytically intractable, so a complete characterization of the system is not possible (Hudson & Greenman 1998). Most of the analyses use simulation to explore the model (some with mathematical proof for particular cases), and sometimes, despite extensive exploration of the parameter space, interesting behaviours are missed (Greenman & Hudson 1999). A more complete analysis requires the application of bifurcation theory or related mathematical

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techniques (Hudson & Greenman 1998; Greenman & Hudson 1999). The message from these analyses is that a great range of system behaviours is possible. Some parameter combinations can have alternative stable states, making quantitative prediction about system behaviour problematic. There has recently been a move to simplify the mathematics in order to obtain some general patterns (Holt et al. 2003). A promising alternative approach that circumvents the intractability of population dynamics for multiple species is to use techniques from network theory to analyse real food webs (reviewed in Proulx et al. 2005). This can provide a useful test of hypotheses about the relative contribution of processes at lower scales (such as module-level processes) in structuring food webs (for example, Arim & Marquet 2004; Bascompte & Melian 2005).

Parasite-modified IGP

Emerging from our review of this area there is a clear need for focused studies of the impact of parasitism on IGP and its outcomes for the community. There are only two documented cases of parasite-modified IGP (MacNeil et al. 2003a,b) and both provide evidence that parasitism can alter IGP and hence community structure. There are also several tantalizing speculations in the literature that demand further study (Yan et al. 1998; Hoogendoorn & Heimpel 2002; Juliano & Loumibos 2005) as well as studies that need to be revisited (e.g. Park 1948; Park et al. 1965). Interestingly, two recent food web analyses both find evidence for the importance of apparent competition and IGP in structuring communities (Bascompte & Melian 2005; van Veen et al. 2006). To our knowledge, theory for parasite-modified IGP has yet to be developed. There may be problems with theory tractability for IGP/parasite or predator/parasite/competitor models, although we may be able to make some simplifying assumptions by ignoring ‘slower’ processes (in this case, interspecific competition, as discussed above). Development of theory should parallel the empirical study of parasite-modified IGP in these systems.

Inclusion of resource base

This is important for our understanding of IGP (Holt 1997; Holt & Polis 1997; Briggs & Borer 2005), and for apparent competition (Holt 1997; Bonsall & Holt 2003). Most of the models considered in this review do not explicitly include resources, although Hall et al. (2005) examined the impact of productivity (by changing prey carrying capacity) in a predator–parasite–prey system. They showed that high productivity could destabilize the system as parasites overexploit the host resource causing oscillations that predators exacerbate. This mirrors the ‘paradox of enrichment’ predicted in other simple community modules (Holt 1977; Holt & Lawton 1993), although this pattern does not always prevail (Bonsall & Holt 2003). Anthropogenic alteration of resources may have knock-on effects on the community via parasitism. For instance, cultural eutrophication is implicated in increased parasitism by the flatworm, Rhinella ondatrace, in US amphibians via a predator-mediated shift in the planorbid snail intermediate hosts (Johnson & Chase 2004).

Population structure

Parasite/host and predator/prey dynamics and coexistence are well known to be contingent on spatiality. However, most of the models discussed in this review do not explicitly consider population structure and there is a need for greater incorporation of spatial structure in order to examine a number of issues ranging from the population dynamic consequences of TMIEs to the evolution of disease emergence in novel hosts. Metapopulation models of predators and parasitoids have been developed for apparent competition scenarios; habitat structure offers opportunity for refugia from a shared enemy which can promote coexistence (e.g. Holt & Lawton 1993, 1994; Bonsall & Hassell 2000). On an evolutionary timescale, however, structured populations may be subject to inter-demic selection which can complicate outcomes (see below). In a spatial context, parasite-modified or apparent competition can translate into range limitation as the negative impact of parasitism drives species extinct at the limits of their range (Case et al. 2005). Wodarz & Sasaki (2004), in a model of competing species each harbouring a parasite lethal to its competitor, showed that the parasites could enforce species boundaries, creating zones where a competitor cannot invade because of encounter with the lethal parasite. MacNeil et al. (2004) show that the prevalence of microsporidian infection in the native G. d. celarius is heterogeneous across habitat patches; models demonstrated a ‘bridgehead’ effect, whereby the invasive G. pulex could succeed by first replacing natives in infected patches; invasion would not succeed in a homogenous environment.

Evolutionary perspective

There is a paucity of work examining parasite-mediated multi-species interactions over an evolutionary timescale. Theoretical bases including host–parasite local adaptation and predator–prey/host–parasite arms races extended to three-species interactions should prove fruitful. Choo et al. (2003) show that including predation can have counterintuitive effects on the evolution of virulence. Brown & Hastings (2003) analyse apparent competition in an evolutionary context. For a plant species sharing a disease with a superior competitor, selection favours lower disease
resistance when pathogen transmission is spatially local thus enhancing its negative impact on the competitor. Similarly, Wodarz & Sasaki (2004) find that evolutionary outcomes for parasite-mediated apparent competition depend on the relative strengths of within- and between-population selection; for instance, selection on the host can favour reduced recovery time even though possession of the parasite provides a weapon against competitors. Empirical evidence suggests that, in biological invasions, local adaptation of the parasite to its native host may increase (Tompkins et al. 2003) or decrease (Bauer et al. 2000) its impact on novel hosts. More work is needed on the relationship between parasite transmission strategies, virulence and trophic interactions. Many parasites with complex life-cycles require predation, IGP or a vector for transmission between host species. IGP might also provide opportunities for disease transmission that could benefit generalist parasites. However, a high risk of infection could select for a reduction of IGP in potential hosts or for selective predation on uninfected hosts. During biological invasions, IGP might provide opportunities for the infection of new species of host, with the outcome dependent on the specificity of the parasite. It is interesting to note that, although P. mulleri is transmitted between G. d. celticus hosts via cannibalism, IGP of infected individuals did not lead to infection in any of the three sympatric invading gammarids (MacNeil et al. 2003a). Grosholz (1992) observed that P. scaber suffered higher parasite prevalence when in competition with P. laevis than in single species populations and suggested that intraguild aggression over food could provide a transmission route for the virus through the haemocoel.

### Emerging diseases

Both management strategies such as predator removal and changes in biodiversity have implications for public health where species that are zoonotic disease reservoirs are concerned (Ostfeld & Keeseing 2000; Packer et al. 2003; Ostfeld & Holt 2004; Collinge & Ray 2006). Increasing numbers of emerging infectious diseases of humans and wildlife appear to be associated with increased levels of anthropogenic change including human population expansion into wildlife habitat, intensive husbandry of domesticated species and introductions of exotic species and/or their pathogens (Daszak et al. 2000). Spillover of pathogens from reservoir populations (often managed domestic stock or introduced species) to native wildlife is a major threat to biodiversity (de Castro & Bolker 2005) and spill-back from wildlife to crops and managed stock has clear economic and public health costs (Daszak et al. 2000; Power & Mitchell 2004). In this context, there is increasing need to understand how trophic interactions affect the evolution of parasite transmission strategies and virulence (and vice versa). Dennehuy et al. (2006) examine the evolution of disease emergence in novel hosts; while adaptations to the new host undergo natural selection, the native host acts as a source and the novel host a sink for the disease. Emergence will therefore require periodic exposure to the native host whilst adaptations evolve and have time to be selected.

### CONCLUSIONS

From a community perspective, it is clear that we have more information about the potential role parasites play in some modules than in others. Apparent competition (Fig. 1a,b) is relatively well-studied, both empirically and theoretically. However, to our knowledge there is no general theoretical treatment of parasite-modified competition, or how it might interact with apparent competition. More work is needed empirically, especially in animal systems, on the extent to which parasites do modify competition interaction strengths. Of the predator–prey modules, there are several thorough combined theory and empirical studies of parasites of prey only (Fig. 1d), but few theoretical treatments of Fig. 1f, where both predator and prey are host to the parasite. This module is of particular interest with regard to behaviourally mediated interactions, which we know from the studies of module 1d to have potentially strong influences. The role of parasites in IGP (Fig. 1g) is also understudied, theoretically and empirically. Equally, within modules, there is the potential for a great range of population dynamic or community outcomes (for instance, for Fig. 1a, compare short vs. long-term apparent competition; or for Fig. 1d compare the effects of animal parasites, plant pathogens and endophytes). Hence, if we are to determine the effects of parasites within whole communities, we need more information about the relative frequency of these different modules and the types and strengths of interactions within them.

The addition of parasites within modules, or as links between modules, might be key to understanding some outstanding problems in community ecology. For instance, a recurring theme in IGP theory is the observation that in many instances, IGP should be unstable (Polis & Holt 1992; Holt 1997); despite this, IGP appears to be a frequent component in many ecosystems (Polis et al. 1989; Bascompte & Melian 2005). Another frequently cited problem in studies of apparent competition concerns the continued coexistence of both the prey species; in predator- or parasitoid-driven systems one prey species should, theoretically, be excluded (Holt 1977). Spatial structure is likely to be important in explaining these discrepancies (Bonsall & Hassell 2000). Alternatively, interactions between modules might explain persistence in these cases; one way in which modules can be linked is via shared parasitism. Parasite diversity within trophic levels is also likely to be important.
(Memmott et al. 2000; Lafferty et al. 2006). Most species are host to numerous parasite species and how this diversity is maintained, and its consequences for community structure, is as yet poorly understood (Holt & Dobson 2006). A combination of theoretical and empirical approaches is required to examine how parasite interactions within and between modules scale up to community-level processes. The concluding message of Anderson & May (1986), namely that we need quantitative data on many population parameters in order to assess the effects of parasites on communities, remains salient today. Indeed, with increasing pressures on biodiversity and anthropogenic influences on the environment and species introductions, the need to understand how parasites affect communities has never been greater.

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