Competition Between Kingdoms

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Although studies of interspecific competition have traditionally been concerned with interactions between closely related species, ecological systems teem with examples of competition between representatives not only from different phyla, but even from different kingdoms. Indeed, interkingdom competition may be one of the commonest forms of interaction in nature; particularly prevalent are competitive interactions for shared hosts between insect parasitoids and pathogens from four other kingdoms. Ecologists have barely started to explore the ecological and evolutionary implications of interkingdom competition.

The literature on interspecific competition and its consequences is replete with studies on closely related species: Park on *Tribolium*[1], Lack on ecological isolation in birds[2], and MacArthur on *Dendroica* warblers[3] are classical examples. Nevertheless, there is growing awareness that taxonomically very distant species may compete for limiting resources, although many ecologists probably still feel that most of the examples are ecological and evolutionary oddities. In fact, competition between organisms in different kingdoms may be one of the most pervasive forms of interspecific competition in nature, albeit the most rarely studied, and certainly one of the most poorly understood. Our primary concern here is with competition between insect parasitoids and disease-causing microbes in shared hosts. We commence by placing interkingdom competition in context with other examples of competition between distantly related taxa.

Examples of interclass and interphyletic competition

Definitions of 'closely' and 'distantly' related are, of course, arbitrary. For example, nobody would be surprised to find evidence for interspecific competition between two different genera of birds within the same family. Things get more interesting when organisms from different classes in the same phylum compete. Possible examples involve exploitation competition between aquatic birds and fish for shared food resources[4–6]. Birds may also get involved in exploitation competition with lizards[7], and in interference competition with bats[8].

Interphyletic competition has also been reported. It is certainly important in some plant–pollinator systems in which birds and insects compete for floral nectar. Hummingbirds show both exploitation and interference competition with hawkmoths and bees[9,10]. There is some irony in the likelihood of competitive interactions between *Xylocopa* bees and *Geospiza* ground finches for nectar on the Galápagos[11], given Darwin's suggestion that 'the [competitive] struggle will generally be more severe between species of the same genus'[12]. Other examples of interphyletic competition include interactions between graminivorous ants and rodents[13] in desert ecosystems, and between frogs and insects[14] in small pools. It is unclear how common and widespread such interactions are.

Without question, one of the easiest places to see interphyletic competition is in rocky, shallow marine and intertidal habitats (e.g. Ref. 15), as animals from totally different parts of the ark compete for space. This struggle for space also involves members of different kingdoms: algae and a variety of encrusting animal taxa.

Interkingdom interactions

Is competition for space the only way in which members of different kingdoms are likely to compete? The answer is an emphatic 'no'. As Janzen[16] pointed out, one of the least appreciated types of interspecific competition is that between microbes and animals for a variety of prominent remains: fruits, seeds and newly dead animals. 'Fruits rot, seeds mould and meat spoils because that is the way microbes compete with bigger organisms.' This ubiquitous form of interkingdom competition may have been a potent selective force on microbial metabolism; it is much less clear what effect it has had on the animals, and it remains very poorly studied.

Indeed, interkingdom competition involving microbes may be one of the commonest, albeit most poorly studied, of all competitive interactions. The principal players are phytophagous insects that serve as resources for competitors from at least four kingdoms[17]. The competitors are insect parasitoids (Hymenoptera and Diptera), the larvae of which feed on the living tissues of their hosts, and agents of disease in the form of bacteria, protozoa and fungi, attacking the same hosts. Viruses sit uneasily on the interface of the living and nonliving worlds (we opt for the former), and if they are to be incorporated into the general scheme they constitute another kingdom[17] of disease-causing agents.

How many competitive interkingdom interactions are there? A very crude calculation is both instructive and astonishing. There are at least 3×10^5 species of higher plants. If every species of plant is fed on by just ten species of phytophagous insect[18], each of which is host for five species of parasitoid[19]

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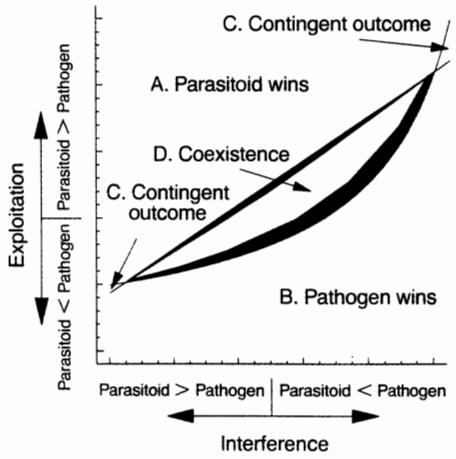
Box 1. Host–parasitoid–pathogen population dynamics

A formal theory of interkingdom competition has recently been developed[20] for hosts exhibiting discrete and nonoverlapping generations (as would be the case for many temperate insect populations) attacked by a parasitoid and an infectious disease (e.g. a virus, bacterium or fungus). Incorporated into the model are realistic assumptions about the biologies of the two natural enemies. For instance, the distribution of parasitoid attacks can range from highly aggregated to random. The pathogen is assumed to be transmitted by external stages, which can span periods when the host is rare or absent.

The model generates a wide range of dynamics. The persistence of each competitor in the system depends crucially on the finite rate of increase of the host, the distribution of parasitoid attacks, interference between the natural enemies within the host, and their relative exploitative potentials (e.g. attack rates, and number of offspring produced per attack).



This figure illustrates how relative levels of interference competition and exploitation competition may influence the outcome of competition. Interference competition (horizontal axis) measures the relative ability of each competitor to oust the other from co-infected host individuals. The relative measure of exploitation competition (vertical axis) includes both search rates for the host and the number of offspring produced per host for each parasitoid and pathogen. One of three general competitive outcomes may occur:

(1) Unilateral exclusion of one competitor from the system (regions A and B). Here, no matter what the initial densities of host, parasitoid and pathogen, one competitor excludes the other from the system. Although re-invasion of the inferior competitor is possible, it will be re-excluded in time.

(2) Competitive elimination contingent on initial population densities (region C). In this case, the initial densities determine which natural enemy wins out in competition. This type of interaction is most likely to occur when one competitor is completely dominant in interference competition, and when the distribution of attacks by the parasitoid tends to random.

(3) Competitive coexistence (region D and shaded area). Coexistence becomes more likely as the differences in the exploitative and interference abilities of the competitors lessens. Competitive coexistence, however, does not require that one competitor be better at exploitative competition whereas the other is better at interference; situations may arise in which the pathogen is better at both but the two enemies nevertheless coexist.

Competitive coexistence may be manifested by either constant (unshaded area) or oscillatory (shaded area) dynamics. Long-period cycles ensue as parasitoid search tends to become random, whereas more ragged, short-term cycles occur as intergeneration survival of the pathogen decreases. Intriguingly, in some cases, if the interaction between one competitor and the host is characterized by oscillatory populations, then the other competitor may either periodically or aperiodically re-invade the system. In such cases, no three-species equilibrium exists, leading to the suggestion that frequent colonizations and extinctions may sometimes drive nonequilibrium dynamics for host–parasitoid–pathogen systems in the field.

and one infectious disease, the potential for interkingdom competitive interactions between parasitoids and disease agents is vast (≈ 15000000). (Logically, we could also study competition between any combination of Monera, Protista, Fungi and viruses, vastly escalating the number of interkingdom interactions; we have chosen not to do this, focusing instead on parasitoids and diseases as a whole.)

Animals competing with disease agents: parasitoids versus pathogens

There is no doubt that parasitoids and the agents of disease do compete for hosts. This competition generally occurs in (but is by no means limited to) the larval stages of arthropod hosts, such as temperate Lepidoptera. For competition to take place, the natural enemies need not be physically present in the same host individual, or even in the host population at the same time; indeed, as is the case for interspecific competition between more closely related species, some avoidance is the surest means of coexistence.

A formal theory of the dynamics of interkingdom competition, involving a population of host insects attacked by parasitoids and pathogens, is summarized in Box 1. Important features of the ecology of macroparasites (e.g. parasitoids) and microparasites (i.e. disease agents) are presented in Table I.

As with conventional notions of competition involving closely related taxa, parasitoids and pathogens compete via mutual exploitation of a common resource (i.e. the host) and/or through interference whilst attempting to exploit the resource. Below, we briefly discuss some of these interactions in the context of parasitoids and pathogens.

Parasitoids against viruses

Some of the most studied competitive interactions are those involving parasitoids (Hymenoptera and Diptera) and viruses (especially nuclear polyhedrosis viruses) within lepidopteran hosts. These interactions can be intense and rich in detail. The outcome of competition frequently appears to be determined by the timing of infection and attack. For instance, if exposure of *Heliothis virescens* caterpillars to a nuclear polyhedrosis virus (NPV) occurs more than 48 h after attack by the parasitoid *Campoletis sonorensis*, then some of the parasitoids survive to maturity; otherwise, the virus essentially eliminates *Campoletis* larvae[22]. The NPV appears to have no effect on the developmental period of surviving parasitoids. The effect of relative exposure times on the competitive outcome is similar when NPV and the parasitoid *Hypositer exiguae* attack caterpillars of *Trichoplusia ni*[23]; here, however, the virus reduces the developmental time of the parasitoid. In still other systems, viruses can infect both host and parasitoid – as in the case of the hypertrophy strain of the NPV of *Pseudaletia unipuncta*, which adversely affects both host and parasitoid (*Glyptapanteles militaris*) tissues[24]. Susceptibility of parasitoid larvae to the virus is essentially identical to 'intraguild' predation observed between potentially competing animals[25].

Parasitoids can also have a substantial negative impact on pathogens. For example, larvae of the gregarious endoparasitoid *Apanteles glomeratus* may reduce by as much as 28% the potential resource for a competing granulosis virus to exploit in penultimate instar hosts of the large white butterfly, *Pieris brassicae*[26]. Interestingly, larger broods of the parasitoid can actually increase the weight of the host, and hence the potential amount of virus produced; here, the act of parasitism is detrimental to the virus, but

less so in larger broods than in smaller ones.

Parasitoids against bacteria, fungi or protozoa

Broadly similar qualitative interactions have been noted involving pathogens other than viruses attacking insect hosts. For example, the relative timing of attacks also appears to determine the competitive outcome of various parasitoid–fungus[27] and parasitoid–protozoan[28] systems. Furthermore, the competitive impact of a given pathogen can differ substantially between parasitoid species, as can the relative impacts of different pathogen species on a parasitoid. For instance, infection by the microsporidian *Nosema pyrausta* resulted in fewer *Macrocentrus grandi* parasitoids surviving to adulthood in *Ostrinia nubalis* hosts[29]. The same microsporidian had no significant effect on the ability of another parasitoid (*Lydella thompsoni*) to eclose, but a congeneric microsporidian did[30]. This is apparently because the second *Nosema* directly infects the larvae of *Lydella*, whereas *N. pyrausta* does not.

Although sharing hosts with a pathogen usually reduces the performance of a parasitoid, this outcome is not inevitable. For example, rates of parasitism of gypsy moth (*Lymantria dispar*) caterpillars by *Apanteles melanoscelus* are higher when the population is subject to infection by *Bacillus thuringiensis*[31]. In this case, parasitoids prefer to oviposit in small caterpillars, and benefit because hosts infected with *B. thuringiensis* grow more slowly. However, once the host is jointly infected, the parasitoid and bacterium still compete in the conventional way.

Table 1. Some general life history characteristics of pathogens and parasitoids (modified from Ref. 21)

Characteristic	Pathogen	Parasitoid
Number of offspring per host	Many (up to and exceeding 10^{10} per host)	Few (from one to about 1000)
Number of attacks required to infect host	Anywhere from one to billions, depending upon host susceptibility	One or few, depending on parasitoid choice and host immune system
Search and dispersal	Passive	Active
Ratio of average lifespan to that of host	Highly variable, from seconds to years depending on exposure to vagaries of environment	Approximately equal
Ratio of body sizes	Much smaller than hosts	Similar to (solitary) or smaller than (gregarious) hosts
Intrinsic growth rate of population	Much faster than hosts	Comparable, but usually slightly slower than hosts
Effect of the above interaction on the host individual	Mildly deleterious to lethal	Eventually fatal
Interaction with individual hosts, in natural populations	One host usually supports a number of populations of different species	One host can support one or several individuals of one (or rarely two) species
Ratio between numbers of species, at the population level	Many species of pathogens recorded from each member of host population	Most host species support several parasitoid species, both specialist and generalist (but only a proportion of hosts are actually attacked)
Degree of overlap of the ranges of the two species	Occur as diffuse foci throughout host's range	Usually present throughout host's range
Genotypes per host or prey	Single or multiple	Single or multiple sibships

Parasitoids mediate the transmission of their competitors

Interactions between parasitoids and pathogens commonly involve parasitoids aiding the dispersal of pathogens. The actual mechanisms involved are numerous (see Refs 32 and 33), although they can be conveniently divided into two categories. First, the insect may simply be a mechanical vector, or translocation agent, and the pathway of infection of the pathogen is similar to transmission in the absence of the parasitoid. There is a plethora of such examples from both the laboratory[22,23,28,34] and the field[26,35]. Second, the parasitoid may aid in both the translocation *and* infection of the pathogen, acting as a 'flying hypodermic needle' (in the case of an endoparasitoid) by inserting a contaminated ovipositor into a healthy host. Very few studies, however, have adequately substantiated claims that infection actually originates from injected pathogens.

The impact of parasitoids in disseminating pathogen inoculum in the field remains unclear, in part because of the difficulties involved in assessing spatial and temporal changes in the pathogen population and the contribution of the parasitoid to translocation. Hence, many studies on host–pathogen–parasitoid interactions in the field have relied on spatial and temporal correlations in the abundances of parasitized and infected hosts as a measure of parasitoid-aided dispersal, and, more generally, competition between the natural enemies. For example, there are significant positive correlations in the incidences of a NPV and two different parasitoid species, *Apanteles melanoscelus* and *Parasetigena silvestris*, attacking *Lymantria dispar*[36]; this is consistent with, but by no means proves, that these parasitoids aid translocation of the pathogen. Purely competitive interactions between parasitoids and the pathogen might be expected to generate negative correlations in their distributions[27,29].

In general, field experiments will be required to reveal the role played by insects in the transmission of pathogens. A model example is provided by the work of Young and colleagues[35], who have investigated the role of *Anticarsia gemmatalis* parasitoids in the dispersal of a NPV using controlled caged experiments in the field. The effects of such dispersal on the population dynamics of host–parasitoid–pathogen interactions remain to be addressed.

Amensalism

It is noticeable that many of the examples outlined in previous sections appear to be highly asymmetrical; pathogens reduce substantially the survival of parasitoids but not vice versa. The situation in which species A has a competitive effect on species B, but B has no

effect on A, is usually referred to as amensalism. Interestingly, amensalism is not necessarily the rule in pathogen–parasitoid interactions (e.g. Refs 26, 27, 31), and we suspect that apparent strong asymmetries in many of the reported cases may be as much due to observer bias as to biology. Nevertheless, the problem deserves more attention.

In a recent brief review of competition between unrelated taxa, Diamond[37] postulated that behavioural, population dynamic and evolutionary interactions between competitors become more asymmetrical as the players become taxonomically more distant. Schluter[11] similarly believes that the evolutionary effects of competition, in the form of character displacement, will be more asymmetrical between more distantly related taxa. However, conventional competitive interactions between related species tend to be amensal more often than they are symmetrical[38,39]. It is currently unclear whether competitive interactions between microorganisms and parasitoids are any more asymmetrical than those between related species.

Conclusion and extensions

Both quantitatively and qualitatively, the picture we paint here is far from complete. The conjecture that interkingdom competition must be one of the most prevalent forms of interspecific competition in nature is based on the assumption that most arthropod taxa that are subject to parasitism are also subject to infectious disease. On present evidence this does not seem an unreasonable assumption. Janzen's rotting examples[16] aside, we have, moreover, ignored other common forms of interkingdom competition. Not only do predators compete with parasitoids for insect prey, but they must also compete with pathogens; diseased caterpillars rapidly become unavailable as lunch for hungry nestlings. Some of the dynamic consequences of victim–predator–pathogen interactions have been explored by Dobson[40]. Once again, the potential for subtle, and as yet poorly understood, dynamic effects is great. A good example is provided by Weiser[41], who showed that the protozoan parasite *Nosema steinhaus* rendered individuals of the arthropod *Tryophagus noxius* more susceptible to predation by other insects. Here the insects both compete with the disease and (indirectly) consume it. It is unclear where the balance of advantage lies.

One final example of interkingdom competition also deserves comment. It has been known for some time that plant pathogens induce defensive biochemical responses in infected plants. More recently, ecologists have realized that feeding damage caused by phytophagous insects can induce similar changes in plant tissues[42]. A clear potential therefore exists for insects and pathogens to interact via the host, if the underlying biochemical response of the plant to attack by members of either kingdom is the same. At least in cotton this appears to be the case[43]. The fungal pathogen *Verticillium dahliae* was less likely to cause symptoms of verticillium wilt on seedlings that had been previously exposed to attack by *Tetranychus* spider mites. Conversely, populations of spider mites grew less rapidly on seedlings infected with *Verticillium*. The significance of induced chemical changes in plant foliage for the population dynamics of most insect herbivores remains unclear; nevertheless, if only a small proportion of insect species are involved in interactions with pathogens via the host plant, the absolute number of interkingdom competitive interactions will again be very large.

Ecologists have studied only a tiny number of examples of interkingdom competition, and we are only just beginning to fathom out its dynamic consequences for the immediate participants. The consequences of interkingdom competition for the evolution of various taxa and the structure and dynamics of larger assemblages, food chains, food webs, guilds or even entire communities, are uncharted territory. A moment's thought suggests, for example, that competition between pathogens and parasitoids for hosts has probably played a crucial role in the evolution of parasitoid life cycles; particularly in where, when and what to attack. Familiar representations of feeding links in food webs may be grossly misleading because they ignore four entire kingdoms of organisms and their role in the community dynamics! To what extent are the presence, absence and abundance of large, conspicuous and 'important' species such as birds due to interkingdom competition with microbial diseases for insect prey? The list of questions is endless; the answers may transform our understanding of communities.

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