

Opinion piece



Cite this article: Hasik AZ, King KC, Hawlena H. 2023 Interspecific host competition and parasite virulence evolution. *Biol. Lett.* **19**: 20220553.
<https://doi.org/10.1098/rsbl.2022.0553>

Received: 23 November 2022

Accepted: 17 April 2023

Subject Areas:

evolution, ecology

Keywords:

host–parasite interactions, interspecific competition, virulence evolution, virulence–transmission trade-off, virulence–recovery trade-off, specialist–generalist trade-off

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Evolutionary biology

Interspecific host competition and parasite virulence evolution

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Virulence, the harm to hosts caused by parasite infection, can be selected for by several ecological factors acting synergistically or antagonistically. Here, we focus on the potential for interspecific host competition to shape virulence through such a network of effects. We first summarize how host natural mortality, body mass changes, population density and community diversity affect virulence evolution. We then introduce an initial conceptual framework highlighting how these host factors, which change during host competition, may drive virulence evolution via impacts on life-history trade-offs. We argue that the multi-faceted nature of both interspecific host competition and virulence evolution still requires consideration and experimentation to disentangle contrasting mechanisms. It also necessitates a differential treatment for parasites with various transmission strategies. However, such a comprehensive approach focusing on the role of interspecific host competition is essential to understand the processes driving the evolution of virulence in a tangled bank.

1. Introduction: the evolution of virulence

Virulence can be defined as the harm to host fitness caused by parasite infection [1]. Since virulence is evolutionarily dynamic, a goal of evolutionary studies is to reveal the factors enhancing or impeding virulence of a particular parasite over time [2] and to understand why some parasites are more virulent than others across communities [3] and environments [4,5].

The classic virulence–transmission trade-off states that the benefit of host exploitation and parasite replication, which enhances parasite transmission, comes at the cost of higher host mortality (see references [1,2,6] for thorough reviews of the evolution of virulence). Because host mortality and parasite transmission are both functions of increasing parasite replication, a second trade-off arises between host recovery rate and virulence-induced host mortality rate [6]. High virulence lessens host recovery rate (i.e. increasing host mortality and reducing infection length), with shorter infections decreasing parasite transmission, though the field lacks evidence from empirical studies [7]. Accordingly, transmission is expected to be highest at intermediate virulence, balancing costs (virulence) and benefits (transmission) of high replication [2].

When there are multiple host species, parasites may face an additional trade-off. They could specialize in one host species (i.e. benefit of increased exploitation) carrying the cost of being maladapted to other host species. They could alternatively be generalists (i.e. benefit of transmission to multiple host species) [8]. Empirical results suggest that specialists are expected to evolve increased virulence—as they evolve to better exploit their hosts—while generalists evolve reduced virulence in a broader array of hosts [9,10]. Importantly, this trade-off is

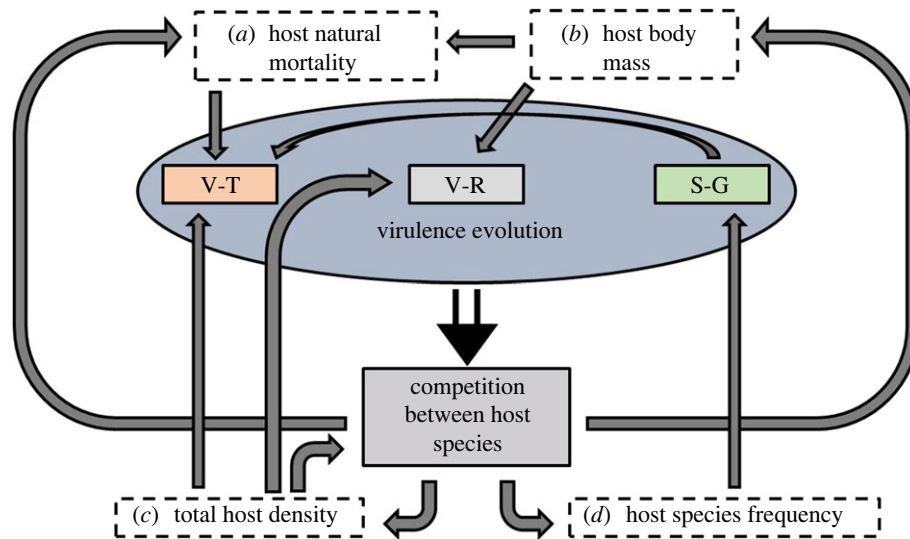


Figure 1. Interspecific competition between hosts is predicted to affect virulence evolution through changing the core trade-offs that shape virulence evolution and by introducing a new trade-off. The trade-off hypothesis states that two main trade-offs shape virulence evolution: the trade-off between virulence (V) and transmission (T) and the trade-off between virulence and recovery (R). In addition, in heterogeneous host communities, one additional trade-off emerges: the trade-off between being a host specialist (S) or generalist (G). Competition between host species is expected to modify these three trade-offs through influencing the natural mortality rates of the hosts, host body mass, and the frequency and density of each competing host species. The influences are indicated by single arrows, whereas the double arrow represents the feedback effect of virulence on interspecific host competition.

expected to change/disappear if a specialist parasite acquires mutations allowing it to infect both its typical host and a novel host [11].

Understanding virulence evolution requires not only a consideration of within-host dynamics, but of between-host processes [2,12–14], as some ecological factors at various biological scales directly affect virulence evolution. For example, theoretical [15] and empirical studies [16] suggest that changes in abiotic and biotic factors within the local environment can drive the evolution of virulence. Friman *et al.* [17] established the first step towards exploring the influence of interrelated ecological factors, finding that avian predators of a moth host induced a cascading effect on virulence evolution. Specifically, they showed experimentally that host investment in anti-predator defences resulted in decreased immune defence [17], subsequently relaxing selection for heightened parasite virulence via the virulence–recovery trade-off [1,2]. Since then, additional studies have also investigated how other interrelated ecological factors such as co-infection [18] and competition between protective microbes and pathogens [19] affect virulence evolution.

Like predation, interspecific host competition for shared resources is another central factor that affects many aspects of host life history such as growth [20], mortality [21] and immune function [22]. In this opinion piece, we highlight the network of effects between and among these life-history traits, which are expected to shape virulence evolution.

2. Interspecific host competition as a driver of virulence evolution through a network of effects

Much of the theoretical literature on virulence evolution has focused on processes affecting members of the same species (e.g. [15,23]), yet here we focus on competitive interactions between different species. We do this because interspecific

host competition is expected to modify the three trade-offs (virulence–transmission, virulence–recovery and specialist–generalist). Specifically, interspecific host competition increases host mortality rate, decreases host body mass and total host density, and changes host species frequencies (figure 1).

(a) Host natural mortality rate

A primary consequence of interspecific competition between hosts, as in empirical studies, is an increase in host mortality ([24,25], figure 1a). Theory suggests a greater host mortality rate reduces transmission within populations [26]. This negative relationship has the effect of increasing the benefits of transmission and reducing the costs of virulence, thereby favouring more virulent parasites [2]. Empirical evidence is equivocal, with experimental evolution studies reporting either increased [27,28] or decreased [29] virulence in response to elevated host mortality. In general, elevated mortality of infected hosts reduces transmission by removing infection sources, while greater mortality among non-infected hosts reduces transmission by removing susceptible hosts. However, this prediction can become more complicated if host mortality is not random. In cases where mortality from interspecific host competition occurs mostly in infected hosts (e.g. [30]), host mortality may further increase with parasite virulence. Such increased mortality quickly reduces infected host numbers and removes parasite propagules, subsequently reducing any further transmission in a manner similar to self-shading [31–35], the process whereby highly virulent parasites rapidly deplete the number of susceptible, non-infected hosts [36].

Future studies could therefore investigate two outstanding questions. First, how do competition-mediated mortality rates differ among infected and non-infected hosts [37]? Second, what are the general effects of host mortality on virulence evolution? Whether the effects of competition-mediated mortality rates on virulence evolution differ from the effects of mortality from other sources also remains unclear.

(b) Host body mass

The loss of host body mass is another consequence of competition in empirical studies ([20], figure 1*b*). Individuals with lower body mass tend to have higher mortality rates [38,39] and reduced immunocompetence [40,41], including experimental evidence for reduced fat reserves driving a reduction in immune defences [22]. While a heightened natural mortality rate increases the benefit of transmission (see above), a weaker immune response prolongs infection length (for non-obligate-killer parasites), thereby increasing the cost of virulence in experimental studies [42,43]. The effect of competition through host body mass could therefore be expected to indirectly affect virulence evolution through two opposing processes. One process increases virulence (i.e. increased natural mortality) and another decreases virulence (i.e. reduced body mass), where the overall effect is determined by the relative strength of each [20,24,25,30,44].

To understand the overall effect of body mass loss (due to interspecific host competition) on virulence evolution, future research could differentiate between these two opposing effects of body mass loss. Studies using an experimental evolution approach with a fully factorial experimental design manipulating food supplementation (to mitigate mortality via body mass loss) and host immunocompetence could be useful for disentangling these opposing processes.

(c) Host population density

Competition between multiple species can result in a decrease in the population density of one of the competing host species in experimental studies (e.g. [45], figure 1*c*). Theory predicts that decreases in host population density reduce the likelihood of parasite transmission [23], which selects for increased virulence [34]. However, empirical evidence for density-dependent transmission is controversial and likely host-parasite system-specific [46–48]. Selection for increased virulence in low-density host populations may thus be minimal or context-dependent, occurring in systems exhibiting density-dependent transmission. Further, high host population density can instead favour increased virulence. Ugelvik *et al.* [49] showed experimentally that salmon lice sourced from high-density salmon farms have evolved increased virulence relative to those sourced from low-density populations of wild salmon.

Future empirical research is therefore needed to assess the role of total host density on the evolution of parasite virulence. Such studies could also explore the less-studied consequences of host density reduction that may further complicate the evolution of virulence. For example, host density reduction may also decrease intraspecific competition, and thereby reduce host mortality, selecting for reduced virulence (see above). Even during situations of asymmetric interspecific host competition, which lead to reduced densities of inferior competitor species (e.g. *Anopheles stephensi* consistently outcompeted by *Aedes aegypti* [50]), it is not certain that parasites of the inferior competitor will become more virulent. Effects of low host densities on parasite evolution may conflict with the release from intraspecific competition [20,51].

(d) Host frequencies

In addition to effects on total host population density, empirical evidence suggests that interspecific host competition can shift host species frequency ([45,52,53], figure 1*d*). This may affect virulence evolution by determining whether a parasite

will adapt to infect one or more host species [54]. In general, populations/communities with multiple host species occurring at similar relative abundances should favour generalists, while those dominated by a single species should favour specialists [55]. However, the specialist-generalist trade-off may also depend on host quality. Parasites may evolve to be generalists in communities if high-quality host species are less abundant, as seen with *Philornis* spp. flies and their passerine bird hosts [56]. Under this scenario, the cost of specializing on the superior host type becomes too high given its limited numbers, while the benefit of transmission among multiple species increases. By contrast, if a high-quality host species occurs at a high relative abundance, parasites are expected to become specialists on these hosts. As a result, virulence evolution is predicted to either increase due to specialization or decrease due to generalism, depending on the exact effects of competition on host species frequency.

Indirectly, effects of host frequency on parasite evolution may also depend on host compatibility for the parasite (hereafter 'host' and 'non-host', respectively). Epidemiological models predict that effects of interspecific host competition with non-hosts will reduce host densities and subsequent transmission [26]. Increased virulence should evolve. Empirical evidence has shown that this process can generate a dilution effect [57], though the authors in that study did not test for virulence evolution. Further, theory and empirical evidence suggest that the impact of host/non-host frequency change depends on the nature of interspecific host competition, as hosts that are strong competitors can limit non-host populations [57]. Phylogenetic relatedness of the competing host species may also affect the impact of host/non-host frequency change. Closely related host species are more likely to harbour the same parasites [58], which may reduce or even eliminate the impact of any change in the host/non-host frequencies, particularly if the parasite can successfully infect both host species without any reductions in transmission.

Thus, future field studies should assess two related points to understand the influence of host frequency on virulence evolution. First, studies are needed to quantify the strength and asymmetry of competition between hosts and non-hosts. Second, evolutionary experiments should explore effects of host community composition and host/non-host ratio on virulence evolution. Experiments could use similar designs to Strauss *et al.* [57], crossing competitive ability with transmission ability, yet continue the experiment for multiple host and parasite generations. Ancestral hosts could be infected with evolved parasites to quantify changes in virulence (as in [59]). This approach would directly test for a link between competition, species frequencies, and virulence evolution. Evidence linking dilution effects to virulence evolution is equivocal, with empirical studies demonstrating that host diversity drove the evolution of increased [60] or decreased [8] virulence.

Taken together, the multi-farious effects of interspecific host competition on parasite virulence predict multiple, sometimes contrasting outcomes. Of further interest is how virulence evolution can then feed back into community dynamics (double arrow in figure 1). Parasites not only decrease host fitness [61], but can also affect the strength of host interspecific host competition [37]. Virulence evolution mediated by interspecific host competition may drive a subsequent coevolutionary response in hosts (due to parasite-mediated effects on host fitness) and/or modify virulence evolution (due to parasite-mediated effects on interspecific host competition).

3. Towards establishing a competition–virulence framework

The above predictions point to a complex network of direct and indirect pathways through which interspecific host competition can shape virulence evolution. Our conceptual framework provides an initial foundation for studying the relationship between interspecific host competition and virulence evolution. Yet, more nuanced models are needed for individual host–parasite systems, as contingency rules nature [37,62]. For example, rising temperatures due to climate change have driven the earlier onset of breeding in great tits (*Parus major*), which compete with pied flycatchers (*Ficedula hypoleuca*) for nesting sites [63,64]. Great tits breed earlier and occur at greater population densities [63], which increases the frequency of fatal competitive interactions for pied flycatchers [63,64]. Our conceptual framework predicts that, under this scenario, parasites of great tits could become less virulent due to climate change-mediated increases in host population density. By contrast, parasites of pied flycatchers may become more harmful due to host competition-mediated increased host mortality. Subtle modifications (e.g. inclusion of a term for the onset of breeding) would serve to improve the usefulness of the conceptual framework for this system.

Future experimental work could expand upon this conceptual framework to include parasite transmission mode and differential mortality rates among infected and non-infected competitors, in addition to considering the influence of simultaneous effects on virulence evolution (i.e. competition for space affecting both total host density and frequency). It will also be informative to consider effects of population density on immune defences generated by density-dependent prophylaxis [65–67], although this phenomenon is not ubiquitous [68,69]. Because immune function increases with host density in systems exhibiting density-dependent prophylaxis, the benefit of virulence increases with host density as opposed to our original predictions. Such an effect would result in an emerging conflict between the virulence–transmission and virulence–recovery trade-offs, where parasites must evolve to balance the costs and benefits of virulence. Another important consideration for future work is how selection can change over time as parasites infect novel hosts. For example, at the beginning of an epidemic, high transmission rates may not affect virulence evolution due to a high density of susceptible hosts [70]. Relating this scenario to our conceptual framework, a parasite infecting a population of susceptible novel hosts may evolve high transmission rates without affecting virulence evolution. That is, at least at the beginning of this novel host–parasite association. This situation would remove the expectations of the virulence–transmission trade-off and likely change the outcomes proposed in our conceptual framework.

An additional interesting possibility is the potential for change in the relative timescales for interspecific host competition and virulence evolution. The nature of competitive interactions among hosts can operate on similar timescales to virulence evolution [42,71], with bacteria and viruses undergoing generations worth of selection during a single generation of competing hosts. Yet, the relative timescales of competition and virulence evolution may vary, depending on the host–parasite system investigated. Future studies could therefore consider how shifts in the relative timescales of these two processes affect virulence evolution.

Evolution experiments using species such as the red flour beetle (*Tribolium castaneum*) and its numerous parasites (e.g. *Beauveria bassiana* [42], *Paranosema whitei* [71] or *Hymenolepis diminuta* [72]) could be useful for testing the interrelated effects of interspecific host competition on virulence evolution. Parasites are known to affect interspecific host competition in this system by making *T. castaneum* a superior competitor to its congener *T. confusum* [72], and virulence evolution can occur within a relatively short timeframe [42,71].

4. Conclusion

Theory and empirical evidence suggest that competition among host species can be a strong source of selection on parasites. However, the multi-faceted nature of species interactions within food webs requires consideration and experimentation to disentangle these mechanisms. By studying the varying effects of host competition on parasite virulence, we can gain increased understanding of the processes driving ecological and evolutionary dynamics in nature. Such dynamics have ramifications for predicting future impacts of parasites and disease on host communities.

Data accessibility. This article has no additional data.

Authors' contributions. A.Z.H.: conceptualization, visualization, writing—original draft and writing—review and editing; K.C.K.: conceptualization, visualization and writing—review and editing; H.H.: conceptualization, visualization and writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

Funding. A.Z.H. is supported by the Zuckerman STEM Leadership Program and benefited from the musical inspiration of Trivium. K.C.K. is supported by a European Research Council Starting Grant (grant no. COEOPRO 802242) and Nature Environment Research Council UK Standard Grant (grant no. NE/X000540/1).

Acknowledgements. We thank three anonymous reviewers for their helpful comments. All authors contributed to the conceptualization of this opinion. A.Z.H. wrote the initial draft, and all authors contributed substantially to revisions.

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