

Consumers indirectly increase infection risk in grassland food webs

Elizabeth T. Borer^{a,1}, Charles E. Mitchell^b, Alison G. Power^c, and Eric W. Seabloom^a

^aDepartment of Zoology, Oregon State University, Corvallis, OR 97331; ^bDepartment of Biology, University of North Carolina, Chapel Hill, NC 27599; and ^cDepartment of Ecology and Evolutionary Biology, Cornell University, Ithaca, NY 14853

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Most pathogens exist within complicated food webs of interacting hosts, vectors, competitors, and predators. Although theory has demonstrated a variety of mechanisms by which predation and competition in food webs can indirectly control infection risk in hosts, there have until now been no experimental tests of this theory. We sampled the effect of long-term exclusion of large vertebrate herbivores on the prevalence of infection by a group of aphid-vectored viruses that infect grasses (barley and cereal yellow dwarf viruses) in an oak savannah in central California. We found that pathogen prevalence was \approx 4-fold higher in the presence of consumers than in areas where they were excluded. Vertebrate consumers did not directly alter infection rates by this aphid-vectored pathogen group, but rather increased infection risk by increasing the relative abundance of highly-competent hosts in the grassland community. This large-scale experiment, measuring changes in host abundance and infection risk in response to altered consumption rates, confirms theoretical predictions that consumers can indirectly increase infection risk by altering the composition of whole communities. Most importantly, these results demonstrate that, even in complex natural communities, alterations to food web composition such as consumer invasion or extinction can lead to significant impacts that cascade throughout entire communities, including changes in infection risk.

apparent competition | dilution effect | disease ecology | grazing | healthy herds

Human activities worldwide are redistributing predators and other consumers via selective removal and deliberate or accidental introduction (1–3). Although altered consumer communities are known to impact biodiversity, ecosystem productivity, and stability and facilitate further biological invasions (4, 5), there is mounting concern about the effects of these food web alterations on host–vector–pathogen dynamics (6–9). Theory predicts that direct predation on a host population generally reduces pathogen prevalence regardless of whether predators preferentially target infected hosts or susceptible hosts or show no preference for either host type (9–11). In addition, the effect of predators and herbivores on infection prevalence in their prey may be mediated by impacts on other aspects of the community such as the abundance of either highly-competent host species or nonhosts or the search efficiency of a vector, leading to either increased or decreased infection risk (7, 12). For example, selective removal of nonhosts or hosts minimally viable for a pathogen may increase communitywide infection risk, whereas removal of highly competent “spillover” host species generally leads to reduced infection by generalist pathogens (7, 13). Predators or herbivores also may alter host diversity, generally leading to enhanced infection with reduced host diversity (7).

Although the theoretical exploration of the direct and indirect role of predators in altering disease risk has focused on directly-transmitted diseases in which transmission is a function of infected host density, theory demonstrates that the results are typically robust to transmission formulations (7, 9). These the-

oretical predictions about the role of predators in altering disease risk have been explored by using comparative studies and extensive observations in systems with directly- and vector-transmitted pathogens (e.g., refs. 9, 14, and 15). However, despite repeated calls for replicated, experimental assessments of the effects of consumers on host community composition and infection rates, none yet exist (7, 11). Here, we present results from a long-term experiment quantifying the effect of vertebrate consumers on the prevalence of a globally-distributed, economically-important vector-borne group of pathogens, the barley and cereal yellow dwarf viruses (B/CYDV).

B/CYDV, a group of generalist pathogens of grasses, infects well over 100 crop and noncrop species (16) and is among the most prevalent groups of all viral pathogens globally (17). This RNA virus complex, obligately transmitted by aphids, reduces longevity, growth, and seed production in infected hosts (16, 18). B/CYDV infection can cause extreme yield reductions in crops (16), and recently has been implicated in shifting the competitive balance between native and invasive grasses to allow the invasion and domination of >9 million ha of California’s native perennial grassland by Mediterranean annual grasses (19, 20). Thus, this pathogen group has relevance for both global cereal crop production and conservation of the world’s remaining grasslands. In contrast to most human and wildlife diseases, plant–pathogen interactions, such as those between the B/CYDV group and their hosts, also provide excellent model systems for testing theoretical predictions about the effects of consumers as the drivers of host community composition on pathogen spread, because consumer, host, vector, and pathogen populations can be experimentally manipulated at relevant spatial scales in the field (12).

Host life history appears to play an important role in disease transmission in West Coast grasslands; aphid vectors prefer and perform better on annual grasses compared with perennials, and some annuals cause substantially elevated transmission (i.e., “spillover”) throughout grassland communities (12, 19). In this system, nonhosts (i.e., forbs) do not support either aphid vectors or B/CYDV, but they compete with host species for shared soil and light resources. Perennial grasses are, as a group, low-quality hosts that serve as between-season pathogen reservoirs but sustain lower aphid populations and have relatively low reservoir competence, whereas annual grasses are high-quality hosts that support elevated aphid populations and have relatively high reservoir competence (19–21). Because B/CYDV require an aphid vector for transmission and cannot be directly or vertically transmitted, vertebrate herbivores cannot serve as a mode of

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¹To whom correspondence should be addressed. E-mail: borer@science.oregonstate.edu.

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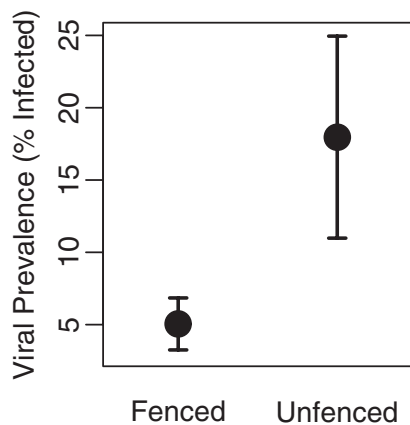


Fig. 1. In concordance with theory, the presence of consumers increases infection risk. Vertebrate herbivores caused a 12.9% increase in infection of an annual grass host by a group of aphid-vectored grass pathogens, the B/CYDV, compared with paired plots with no large vertebrate access.

pathogen transmission. However, vertebrate herbivore diet preferences can control grassland plant composition (e.g., selective removal of forbs) (22, 23), and plant regrowth stimulated by grazing can increase aphid colonization (24).

In a replicated, long-term experimental exclusion of large vertebrate herbivores, we examined whether and how vertebrate consumers indirectly control B/CYDV prevalence. Specifically, we examine the following questions:

1. Do consumers decrease host infection in this community (i.e., healthy herds hypothesis; ref. 11)?
2. Do consumers alter host community diversity leading to increased infection rates in low diversity communities (i.e., dilution effect, ref. 7; i.e., dilution effect, ref. 13)?
3. Do consumers control the composition of the host community (i.e., abundance of high-quality hosts, low-quality hosts, or nonhosts), leading to increased infection in communities dominated by high-quality hosts (e.g., refs. 7 and 12)?

Results and Discussion

Our results confirm theoretical predictions that consumers can indirectly control pathogen prevalence: B/CYDV-infection rates increased 3.6-fold, from 5% inside of exclosures to 18% in the presence of large vertebrate herbivores ($P = 0.04$; Fig. 1). Although this effect of consumers on infection is striking, it is not concordant with the healthy herds hypothesis, i.e., direct regulation of infected hosts leads to lower infection in the presence of consumers (11). These results are unlikely to arise from direct consumer control of pathogen prevalence because the direct pathway nearly always results in a reduction in prevalence in the presence of consumers (9, 11).

Theory predicts that changes in the species diversity or composition of hosts and nonhosts can control infection risk (7, 13). Although theoretical predictions suggest that reduced species richness can alter the prevalence of infectious disease (7), here, vertebrate consumers did not change host species richness, evenness, or Shannon diversity ($P > 0.05$). Thus, our results are not consistent with the dilution effect hypothesis (7).

Consumer alteration of the relative abundance of nonhosts, low-quality hosts, and high-quality hosts does appear to control infection in this community. Vertebrate consumers increased the total cover of high-quality hosts ($P = 0.006$; Fig. 2A), and these high-quality annual grasses comprised 80% (12/15) of the host species that increased outside of exclosure fences. In contrast, the cover of nonhosts (Fig. 2B) and

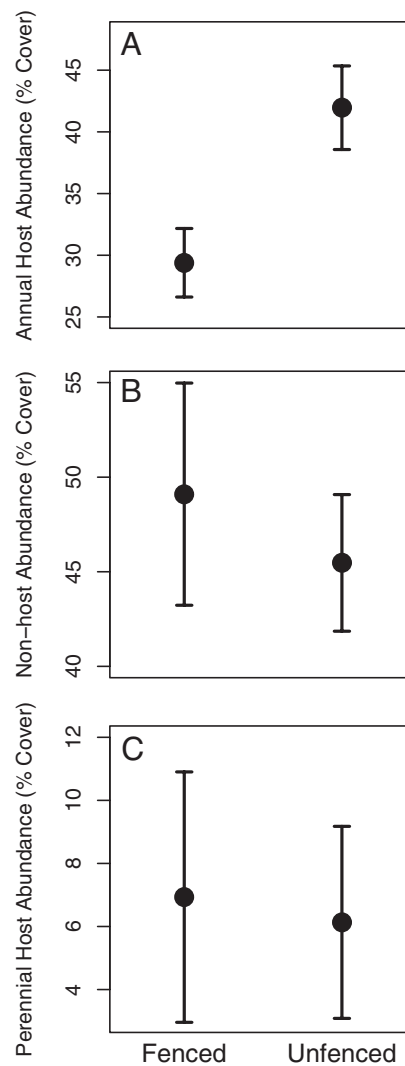


Fig. 2. Large vertebrate consumers increase the relative abundance of highly-competent hosts. (A) The long-term removal of large vertebrate herbivores shifted grassland plant composition, strongly reducing the cover of high-quality hosts (i.e., 13% reduction of annual grass cover). (B and C) In contrast, removal of large herbivores did not alter the cover of nonhosts (i.e., forbs) (B) or low-quality hosts (i.e., perennial grasses) (C). Cover estimates were independent for each species (i.e., do not sum to 100%). Here, we present means and 2 SE for the summed cover of each host and nonhost type.

low-quality hosts (Fig. 2C) remained constant across fence lines ($P = 0.51$). The relative frequency of host types in a community (proportional abundance) can be as important as absolute frequency (total abundance) for pathogen transmission, especially for vectored pathogens in which transmission may either be independent of host density or (more realistically) vary nonmonotonically with host density (10, 25). Because high-quality host cover increased, whereas the cover of nonhosts and low-quality hosts was unchanged by consumers, high-quality hosts were also relatively more abundant outside of exclosure fences ($P = 0.03$). Thus, large vertebrate consumers shifted the community context to enhance a group of hosts known to increase B/CYDV prevalence (12, 19), providing support for the host quality hypothesis (7).

The fact that consumers increased both abundances of high-quality hosts and pathogen prevalence suggests that host compositional shifts caused by consumers are the most likely

drivers of change in disease risk in this system. To determine whether the change in host composition caused by consumers was the driver of increased B/CYDV prevalence, we included annual grass cover as a covariate in our regression model. The inclusion of annual grass cover in this model of infection removed the significance of the enclosure treatment as a factor ($P = 0.62$), suggesting that there is no additional, independent effect of vertebrate exclusion after accounting for host context. This finding supports the conclusion that increased abundance of high-competence hosts is the mechanism linking consumption by vertebrate herbivores and increased virus prevalence.

In addition to testing these three hypotheses based on general theory, we tested whether several factors more specific to our study system might provide alternative explanations of our results. Experimentally simulated herbivory of infected individuals can increase survivorship in some B/CYDV-infected perennial grasses (26), providing one potential mechanism by which consumers could directly increase virus prevalence (11). However, half of the perennial species (4/8) were never found outside of enclosures, perennial host cover did not differ across fence lines ($P = 0.60$), and change in infection prevalence in our focal annual grass host was unrelated to the intensity of recent herbivory (difference in plant biomass inside and outside of each enclosure fence; $P = 0.23$). In addition, perennial grasses can serve as among-season pathogen reservoirs; however, our data do not support this mechanism for controlling the increased B/CYDV prevalence: perennial cover was not higher outside of enclosures (Fig. 2C). Alternatively, aphids can exhibit strong population responses to increased soil fertility, which can increase pathogen transmission rates (27, 28). Here, soil resource pools did not differ across fence lines ($P > 0.05$), despite high studywide variation in several resources (Table S1). Thus, infection risk was increased by consumers, but was not associated with removal of uninfected hosts or changes in plant biomass (11, 26), nor was infection risk associated with differences in soil fertility (29).

Our results experimentally confirm the theoretical prediction that consumers can dramatically alter infection risk in a complex natural community, but do not support the theoretical prediction that consumption should generally reduce infection prevalence (11). Using theory to inform our interpretation, these results suggest two likely mechanisms by which consumers may substantially increase infection risk in this host community. First, consumers may enhance the abundance of hosts that maximize virus replication and/or vector reproduction, thereby enhancing the average transmission competence, and hence infection risk, across the host community (7). Second, by shifting the community to reduce the relative abundance of nonhosts and poor-quality hosts, consumers may improve the probability that a searching vector will encounter a high-quality host plant, thereby elevating encounter rates between vectors and susceptible hosts (7, 12).

Models of vectored pathogen transmission often assume that transmission depends only on the proportion of infected individuals in the population (i.e., ratio- or frequency-dependent transmission) (10). However, pure frequency dependence is unlikely for most vectored pathogens, because the host-finding behavior of most vectors will be affected by host density and vector mobility (25). Our results suggest that elevated relative abundance of nonhosts and poor hosts may reduce the search efficiency of aphid vectors, reducing the encounter probability with susceptible hosts. However, these results point most strongly to the enhancement of high-quality hosts by consumers, leading to elevated virus and/or aphid reproduction and infection risk (7, 19).

This long-term, replicated field experiment demonstrates that consumers can indirectly control pathogen prevalence: consumers increased the absolute abundance of high-quality

hosts and decreased the relative abundances of nonhosts and low-quality hosts, indirectly increasing communitywide infection risk. These results provide an experimental demonstration that infection risk in natural communities can critically depend on the community context of hosts, which can be, in turn, dynamically controlled by consumers. In addition, these results extend the implications of theoretical predictions from general models motivated by predation (11) and host composition (7) to a broader variety of consumers. Specifically, we have experimentally demonstrated the presence of an indirect pathway in which consumers indirectly alter pathogen prevalence by increasing the abundance of highly competent hosts (12). This indirect pathway is relevant for multihost pathogens in which hosts differ in competence, a scenario common across many vectored pathogens (7, 12, 30). Thus, anthropogenic addition or removal of predator or herbivore consumers is likely to cause far-reaching effects throughout entire communities, including dramatic changes in infection risk.

Methods

To assess the direct and indirect influence of consumers on host composition and infection risk in natural communities, we sampled soils and plants from each side of fenced herbivore exclusion areas at Hastings Natural History Reservation (Carmel Valley, CA, 36.39° N 121.55° W) that had excluded large vertebrate herbivores for an average of 28 years from areas averaging 1,338 m² (Table S2). Large vertebrate herbivores at the site included mule deer (*Odocoileus hemionus*) and European wild boar (*Sus scrofa*), both of which have catholic diets that include both grasses and forbs. All fences were intact and effectively reduced access for many vertebrate consumers; 10/11 fenced areas had fences high enough to substantially deter access by mule deer. The abundance and richness of small and medium mammals were also lower inside of enclosure fences intended to exclude them (Table S2), and wild boars were completely excluded; boar disturbance was never present inside of fenced areas (31). Thus, fences effectively reduced consumption by mammals, consistently removing mule deer and wild boars; however, for simplicity, we refer to the areas inside and outside of each fence as “grazed” and “ungrazed.”

We collected 20 individuals of a common and widespread annual grass host, *Bromus hordeaceus*, from each of the paired grazed and ungrazed areas in May 2006, for a total of 440 host individuals. We assayed each host individual for infection by any 1 of the 5 major B/CYDV species via ELISA (antibodies from Agdia). In each grazed and ungrazed replicate, we quantified plant biomass by clipping, drying, and weighing 2 10-cm × 1-m strips, visually estimated the cover of each plant species in 2 1/2-m × 1-m quadrats and collected 3 10-cm soil cores that were analyzed for 13 different soil characteristics (Table S1). Soil assays were performed at A & L Western Agricultural Laboratories, Modesto, CA.

Pathogen prevalence and host-community responses to long-term experimental manipulation of large vertebrate consumers were analyzed by using a mixed-effects regression model in which each of the 11 grazed–ungrazed pairs was treated as a random effect (lme package in R, version 2.5.1). Treatment (fenced or unfenced) was included as a fixed effect to test for overall effects of consumers (P values reported in *Results and Discussion*). Enclosure type (large vertebrate, burrowing vertebrate, climbing vertebrate; Table S2), enclosure age, and four soil variables (phosphorous, nitrate nitrogen, sand percentage, and clay percentage; Table S1) also were included as covariates. The effect of consumer removal on infection did not depend on the enclosure age, soils, or suite of consumers excluded by each fence ($P > 0.05$; see Table S2). Thus, our results point to consistent compositional shifts and elevated infection caused by reduced consumer access, particularly implicating mule deer and wild boars, the most common large vertebrates excluded from virtually all fenced areas. Data used in this paper are available on the Knowledge Network for Biocomplexity (KNB) at <http://knb.ecoinformatics.org/> by searching on keywords seabloom and bydv.

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