Pathogen Spillover in Disease Epidemics

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ABSTRACT: In field experiments manipulating generalist pathogens and host community composition, the presence of a highly susceptible reservoir species drove disease dynamics in multiple nonreservoir species, sometimes decreasing their abundance through apparent competition. The dynamics of generalist pathogens in multispecies host communities remain a major frontier for disease ecology. Of particular interest are how host community structure controls pathogen transmission and how disease spread feeds back to influence the host community. Pathogen spillover occurs when epidemics in a host population are driven not by transmission within that population but by transmission from a reservoir population. Here we review examples of spillover in pathogens infecting humans, domesticated animals, and crops, noting that most empirical evidence for spillover results from nonmanipulative, observational studies. We then present results from two field experiments utilizing an experimentally tractable model system of annual wild grasses and a generalist virus, the barley yellow dwarf virus. In these experiments, the presence of a highly susceptible reservoir species, *Avena fatua* (wild oats), greatly increased pathogen prevalence in several other species. This result demonstrates pathogen spillover and illustrates the crucial role of host community structure in controlling the dynamics of generalist pathogens. Further, pathogen spillover from *A. fatua* decreased the abundance of two other host species through pathogen-mediated apparent competition. Thus, our results provide experimental support for theoretical predictions of strong feedbacks between host community structure and generalist disease dynamics.

Keywords: disease, plant pathogen, virus, transmission, epidemiology.

Although most epidemiological theory has focused on pathogens that infect a single host species (e.g., Anderson and May 1991), there is growing interest in understanding disease in multiple host systems (Dobson and Foufopoulos 2001; Holt et al. 2003). Pathogens with multiple hosts are common in both plant and animal systems (Woolhouse et al. 2001; Power and Flecker 2003), and understanding their dynamics has become increasingly important for human health, for domestic animal production, and for wildlife and plants of conservation significance. While some of these generalist pathogens may have host-specific strains or races, many are true generalists.

In most multiple host systems, all host species are not created equal. Species inevitably vary in key epidemiological traits such as resistance, tolerance, and vector preference (Daszak et al. 2000; Woolhouse et al. 2001; LoGiudice et al. 2003). As a result, the rates of transmission of a generalist pathogen within and between different host species are commonly highly heterogeneous and asymmetric (Woolhouse et al. 2001). In some host populations, epidemic or endemic disease may be primarily driven not by intraspecific pathogen transmission within that population but by transmission from a reservoir species that maintains a relatively high pathogen population. In such a case, the pathogen typically reaches high prevalence in the reservoir and then spills over into the other host, a process called "the spillover effect" or "pathogen spillover" (Daszak et al. 2000). Thus, we define pathogen spillover as the driving of disease dynamics in one host population by contact with pathogen propagules (regardless of transmission mode) from another host population as a result of high pathogen abundance in this reservoir population.

As a result of pathogen spillover, understanding generalist disease dynamics within any one species, especially nonreservoir hosts, requires considering the community context in which the host and pathogen are embedded. Both the species richness and species composition of the community can influence disease spread, and in fact their effects on disease dynamics are inherently intertwined. The effects of species richness and community composition on the spread of generalist pathogens have been considered primarily for animal pathogens, with most current knowledge coming from studies using Lyme disease as a model system (Ostfeld and Keesing 2000*a*, 2000*b*; Schmidt and Ostfeld 2001; LoGiudice et al. 2003).

In this article, we review examples of pathogen spillover in the best-studied host systems: those involving humans and their domesticated animals and plants. We then describe two field experiments carried out with a generalist pathogen of wild grasses to explore the role of pathogen

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spillover in disease dynamics of multiple host systems. We argue that, because of their tractability, plant disease systems are ideal for experimental studies of generalist pathogens.

Pathogen Spillover by Human Pathogens

Research suggests that pathogen spillover is likely to be a common factor in emerging human diseases. A recent analysis indicated that 61% of human pathogens are transmitted between humans and animals and that such pathogens are more likely to be classified as "emerging" pathogens than are pathogens that have no animal hosts (Taylor et al. 2001). The capacity of these pathogens to infect multiple host species sets the stage for pathogen spillover.

There are two major ways spillover can occur, depending on the traits of the nonreservoir host species. First, the rate of intraspecific transmission in nonreservoir hosts may be too low to support epidemic or endemic disease. In this case, sustained transmission from a reservoir population is necessary to maintain disease in the nonreservoir population. Some of the best-studied cases are pathogens of humans for which human-to-human and human-to-animal transmission are epidemiologically negligible, so epidemics in humans are sustained chiefly by repeated transmission from animals, either wild or domestic. For example, direct transmission from animals is the primary source of infection in humans for rabies (RNA virus in the genus *Lyssavirus*), brucellosis (*Brucella melitnis*), and bovine tuberculosis (*Mycobacterium bovis*; Taylor et al. 2001). One of the best-documented cases is that of Lyme disease (*Borrelia burgdorferi*), which is transmitted from animals to humans by deer ticks (Barbour and Fish 1993).

Second, there may be potential for sufficient intraspecific transmission to support a pathogen epidemic, but the pathogen is absent from the population when exposed to transmission from a reservoir. At least three factors can cause a pathogen to be absent from a susceptible population: lack of prior exposure, human intervention to extirpate the pathogen, or the pathogen previously depleted the population of susceptible hosts and drove itself extinct. Assuming the supply of susceptible hosts has been replenished, epidemics in these cases can be sparked by isolated interspecific transmission events. For example, transmission among humans can sustain epidemics of severe acute respiratory syndrome, so transmission from animals is only required to start epidemics in uninfected host populations (Riley et al. 2003).

Pathogen Spillover between Domestic and Wild Animals and Plants

It has long been known that wild animals and plants can harbor disease agents that may be detrimental to domestic animals and crops. Pathogen movement between wild and domestic animals presents some particularly important examples of spillover (Daszak et al. 2000). A recent study concluded that 77% of the pathogens infecting mammalian livestock were generalists that infected multiple host species, while 90% of the pathogens infecting domestic mammalian carnivores (cats and dogs) infected multiple hosts (Cleaveland et al. 2001). In the United Kingdom, for example, badgers may serve as a reservoir for tuberculosis that infects cattle (Donnelly et al. 2003). Similarly, infected bison may transmit brucellosis to cattle in the United States (Dobson and Meagher 1996). Wild suids, such as warthogs, serve as a reservoir of African swine fever, a devastating disease of domestic pigs in sub-Saharan Africa (Plowright et al. 1969).

In agriculture, weeds and natural plant populations may serve as reservoirs of plant pathogens that move into crops. In Australia, crown rust and stem rust of oats move from wild oats, *Avena fatua* and *Avena barbata*, to cultivated oats (Burdon et al. 1983; Oates et al. 1983). Similarly, wild legumes serve as significant reservoirs of soybean rust, and riparian vegetation contains numerous reservoir hosts for *Xylella fastidiosa*, which causes Pierce's disease of grapes (Purcell and Saunders 1999). A variety of viruses are known to move to crops from overwintering sites in weeds and other wild hosts (Brunt et al. 1996). For plant pathogens, the annual life cycle of most crops means that perennial wild hosts may often be a major overwintering reservoir, along with dormant inocula in the soil or litter.

Despite this widespread recognition of the movement of pathogens from wild populations to domestic ones, until recently, little attention has been paid to the movement of pathogens in the opposite direction. That is, the role of domestic animals and crops as reservoirs of pathogens that infect wild plants and animals has largely been ignored (but see Gilbert and Hubbell 1996; Daszak et al. 2000; Gog et al. 2002). Yet an increasing number of examples suggests that pathogens of domestic organisms may have significant impacts on wild hosts, including endangered or threatened species (table 1). For example, populations of both the endangered African wild dog and the endangered Ethiopian wolf have been decimated by rabies spilling over from domestic dogs (Gascoyne et al. 1993; Sillero-Zubiri et al. 1996; Laurenson et al. 1997). Domestic dogs were also the source of the 1994 epidemic of canine distemper virus that devastated vulnerable lion populations in the Serengeti (Roelke-Parker et al. 1996).

There are fewer well-documented examples of pathogen

Table 1: Infectious diseases transmitted from domestic species to wildlife

Wild host	Conservation status[a]	Domestic host	Disease	Transmission mode	Reference
African buffalo	Low risk, conservation dependent	Cattle	Brucellosis	Direct	Worthington and Bigalke 2001
African buffalo	Low risk, conservation dependent	Cattle	Rinderpest	Direct	Kock et al. 1999
African buffalo	Low risk, conservation dependent	Cattle	Tuberculosis	Direct	Bengis et al. 1996
African wild dog	Endangered	Dog	Rabies	Direct	Gascoyne et al. 1993; Kat et al. 1995
Bighorn sheep	Endangered[b]	Sheep	Scabies, *Pasturella*	Direct	Jessup et al. 1991
Caspian seal	Vulnerable	Dog	Canine distemper virus	Direct	Kennedy et al. 2000
Ethiopian wolf	Critically endangered	Dog	Rabies	Direct	Sillero-Zubiri et al. 1996
Harbor seals	Low risk	Dog	Canine distemper virus	Direct	Bengston 1991
Lion	Vulnerable	Dog	Canine distemper virus	Direct	Roelke-Parker et al. 1996
Ostrich	Not threatened	Poultry	Newcastle disease	Direct	Alexander 2000
Sea otter	Endangered	Cat	Toxoplasmosis	Consumption	Cole et al. 2000
Wolf	Not threatened	Dog	Canine parvovirus	Direct	Mech and Goyal 1993

[a] Conservation status based on the 2000 International Union for Conservation of Nature and Natural Resources Red List of Endangered Species.
[b] Some subspecies endangered, some critically endangered, and some at low risk but conservation dependent.

spillover from crops to wild plants, but this is probably in part because of the relative paucity of data on pathogens in natural plant populations (Jarosz and Davelos 1995). In previous work, we found that the prevalence of some strains of the barley yellow dwarf virus in wild grasses is highly dependent on their proximity to crop hosts of the virus (Power and Remold 1996). Although none of the grasses in our study were endangered, this finding illustrates the potential for spillover of generalist plant pathogens from crops to wild plants.

What are the characteristics of domestic species that make them a good source of pathogens for wild plants and animals? Human activities dramatically modify the environment, genetics, and population and community structure of domestic organisms. Domesticated plants and animals that are utilized by humans for commercial agricultural purposes are typically maintained in continuous, high-density monocultures at high nutritional status.

How do these characteristics of domestics affect disease epidemiology? High host densities are likely to increase transmission rates both within the domestic populations and between domestics and wild species (Burdon and Chilvers 1982; Anderson and May 1991; Gilbert 2002). As density increases, contact rates between hosts will increase, allowing more opportunities for pathogen transmission. Moreover, the probability that a pathogen will die out in a host population becomes very small at high population densities (Anderson and May 1991). Population densities are likely to be relatively independent of mortality due to pathogens, since dead individuals may be replaced even under conditions of relatively high mortality. Thus, high-density populations of domestic species are likely to sustain pathogen populations consistently over time.

Genetic homogeneity in the domestic host population may also contribute to high pathogen prevalence. The agricultural crop literature is replete with examples of the damping effects of genetic mixtures on disease epidemics (Wolfe 1985; Power 1991; Garrett and Mundt 1999; Zhu et al. 2000; Mundt 2002). Although this damping effect is particularly strong for directly transmitted pathogens that have highly strain-specific associations with different host genotypes (Garrett and Mundt 1999), such effects have also been demonstrated for vector-borne generalist plant pathogens (Power 1991). In the latter case, the mechanism appears to depend on changes in vector behavior in response to host plant heterogeneity. Although host genetic homogeneity for disease resistance has been demonstrated in domestic animals such as sheep (e.g., Stear et al. 1998), the impacts of this homogeneity on disease spread in domestic animals is less well documented.

Despite the fact that domestic host populations are commonly bred and treated to reduce infection prevalence, pathogen prevalence can still be high. Thus, they are also commonly bred or treated to increase tolerance to, and reduce morbidity from, pathogens. These human interventions can allow domestic populations to sustain high pathogen prevalences over long periods of time, making them sources of generalist pathogens for wild organisms, including threatened and endangered species.

Measuring Pathogen Spillover in Plant Communities

While pathogen spillover has been widely documented, the fact that most examples involve humans or domesticated species limits insight into the general importance of spillover for wild species, since the nature of domesticated

populations increases the potential for pathogen spillover. Moreover, most of what we know is based on observational studies, limiting the degree of inference that can be obtained from empirical studies. Lack of experimental control also makes it difficult to quantify the ecological consequences of pathogen spillover, such as apparent competition. Apparent competition results when pathogen spillover reduces the abundance of the nonreservoir species. Mounting evidence supports theoretical predictions that pathogen-mediated apparent competition can control the outcome of interactions between species sharing a natural enemy (Holt and Lawton 1994; Alexander and Holt 1998; Hudson and Greenman 1998). However, this body of theory has scarcely been tested using field experiments, perhaps because of the difficulty of manipulating most multiple host/pathogen systems in the field (but see Grosholz 1992, a study of an isopod virus). In our work, we have taken advantage of the experimental tractability of annual plants to address pathogen spillover and its ecological consequences using a field mesocosm approach.

We use a community of common annual wild grasses that serve as hosts of a vector-transmitted generalist virus as a model system. The barley yellow dwarf virus (BYDV) is a phloem-limited luteovirus that is obligately transmitted in a persistent manner by several species of grass-feeding aphids to cultivated and wild grasses (Miller and Rasochova 1997). In New York, BYDV is endemic and periodically epidemic, causing significant yield losses in small grains (Miller and Rasochova 1997). The most important vectors in New York are the corn leaf aphid *Rhopalosiphum maidis*, the bird cherry-oat aphid *Rhopalosiphum padi*, and the English grain aphid *Sitobion avenae*. The experimental host range of BYDV includes more than 100 species of grasses, though the prevalence of the virus varies significantly among different species (Griesbach et al. 1990; Power and Remold 1996). Grass species used in our experiments in Ithaca, New York, have included *Avena fatua*, *Bromus tectorum*, *Digitaria sanguinalis*, *Echinochloa crus-galli*, *Lolium multiflorum*, *Panicum capillare*, and *Setaria lutescens*. We continually propagate the aphid vector *R. padi* and a PAV strain of BYDV isolated from local field sources in controlled environments, and we use these lab cultures of known origin in the field experiments.

Experiment 1: Virus Prevalence in Multihost Communities

Initially, we tested the hypothesis that increasing host species richness in a community should reduce disease spread for a generalist pathogen, as it has been shown to do for most specialist pathogens (e.g., Power 1987; Boudreau and Mundt 1997; Garrett and Mundt 1999; Mitchell et al. 2002; Mitchell et al. 2003). We examined the spread of BYDV in multihost communities of grasses using *Avena fatua*, *Bromus tectorum*, and *Setaria lutescens* as our focal species (A. E. Jolles and A. G. Power, unpublished data). We knew from preliminary data that *A. fatua* was a particularly suitable host of BYDV, exhibiting high susceptibility to infection and harboring high concentrations of virus when infected. During the course of this experiment, the phenomenon of pathogen spillover from *A. fatua* emerged as a significant factor shaping the effect of host diversity on virus spread.

Methods

In 1999, we established an initial experiment with six grass species to explore the role of host diversity in virus prevalence. Plant community treatments were crossed with two virus treatments and replicated in three randomized blocks. Treatments included monocultures of *A. fatua* (A), *B. tectorum* (B), and *S. lutescens* (S); each two-species mixture (AB, AS, BS); the three-species mixture (ABS); a distinct three-species mixture of *Digitaria sanguinalis*, *Echinochloa crus-galli*, and *Panicum capillare*; and a six-species mixture including all grasses from the three-species mixtures. These nine community treatments were crossed with the presence or absence of virus for a total of 18 plots per block. All communities received a total of 600 seeds divided equally among all species planted, and plots were caged at plant emergence in order to assure that virus-free plots were not inadvertently infected from field aphid populations. Three weeks later, plots in the virus inoculation treatment were experimentally inoculated by releasing 100 viruliferous aphids into each plot. Virus-free plots were mock inoculated with 100 nonviruliferous aphids to control for effects of aphid feeding on plant performance.

Each replicate block was harvested in a single day in late September at the end of the growing season. At harvest, we estimated virus prevalence for each species in each inoculated plot by subsampling 20 plants of each grass species in each plot and testing these plants for the presence of BYDV using enzyme-linked immunosorbent assay (Rochow 1986). Virus prevalence of each community was calculated as the average prevalence across all component species, weighting each species' prevalence by the number of individuals in its population. Data on virus prevalence in inoculated plots were analyzed using ANOVA and are reported here. Because some species (*D. sanguinalis*, *E. crus-galli*, and *P. capillare*) were planted only in higher diversity treatments because of limitations on the size of the experiment, host species richness was confounded with community composition in our design. Therefore, we performed separate two-way ANOVAs using either richness

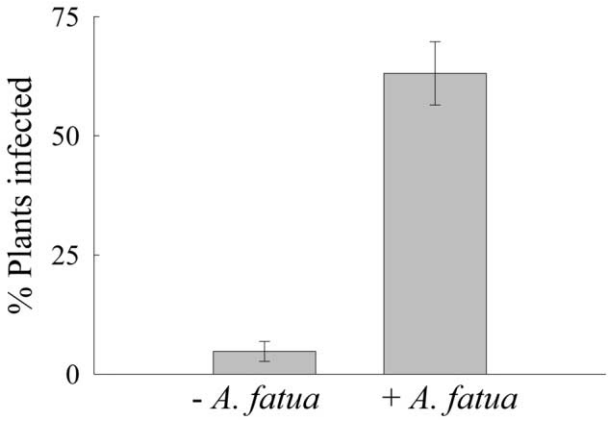

Figure 1: Community virus prevalence was higher in experimental communities planted with *Avena fatua* than in communities lacking *A. fatua* ($P < .001$). Data shown are means ± SE.

or community composition along with block as our main effects.

At harvest, we also measured for each host species in each plot the number of plants, the total aboveground vegetative biomass, and the total reproductive biomass. These data will be reported elsewhere (A. E. Jolles and A. G. Power, unpublished data).

Results

There was no significant effect of the number of host species (richness) in the community on the prevalence of BYDV in our experimental communities according to an ANOVA with host species richness and block as main effects ($F = 0.24$, df $= 3, 21$, $P > .10$). However, an ANOVA with plant community and block as main effects indicated that virus prevalence differed significantly among host communities ($F = 23.58$, df $= 8, 16$, $P < .0001$). In particular, a planned contrast indicated that communities that contained *A. fatua* had significantly higher virus prevalence than communities without *A. fatua* (fig. 1; $F = 148.50$, df $= 1, 16$, $P < .0001$). This result suggested that host diversity was less important for virus epidemiology than the presence of one highly suitable host species.

To test whether the amount of *A. fatua* in the community, and not simply the presence or absence of this species, influenced community level virus prevalence, we computed the proportion of the community made up by *A. fatua* in each experimental community at time of planting and examined the response of virus prevalence to this factor. We found that community virus prevalence increased significantly as the proportion of *A. fatua* in the community increased ($F = 40.06$, df $= 4, 20$, $P < .0001$). Virus prevalence in *A. fatua* itself ranged from 85% to 100% in the different treatments and did not differ significantly among communities ($F = 1.14$, df $= 5, 18$, $P > .10$). In communities that included *A. fatua*, the proportion of the community made up by *A. fatua* had no significant effect on virus prevalence among all other species. However, the simple presence of *A. fatua* did influence virus prevalence in other species. Virus prevalence in species other than *A. fatua* was more than five times greater in communities planted with *A. fatua* than in other communities (27.5% ± 8.8% vs. 4.78% ± 2.1%), although this trend was not statistically significant when species were pooled ($P > .10$). However, virus prevalence in the competitive dominant *S. lutescens* was significantly greater in communities with *A. fatua* than those without, indicating the occurrence of substantial pathogen spillover to this species (fig. 2; $F = 10.26$, df $= 1, 10$, $P = .009$). No significant effect of the presence of *A. fatua* was detected for *B. tectorum*, in which virus prevalence ranged from 2% to 20% in the different treatments.

Experiment 2: The Role of *Avena fatua*

Because of the central role that *Avena fatua* appeared to play in driving virus prevalence in the experimental plant communities, we designed a second experiment to further investigate the potential for and consequences of virus spillover from this host. We began with a three-species mixture as our base community and included treatments with and without the addition of *A. fatua*, crossed with

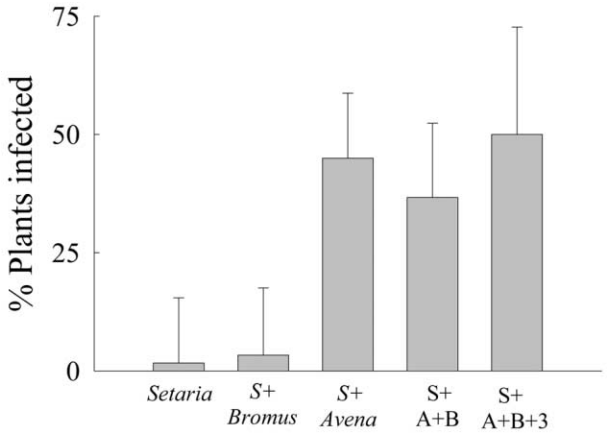

Figure 2: Virus prevalence in *Setaria lutescens* was greater in experimental communities planted with *Avena fatua* than in communities lacking *A. fatua* ($P < .009$), demonstrating pathogen spillover. Each experimental community is identified by the species included, where *S* represents *S. lutescens*, *B* represents *Bromus tectorum*, *A* represents *A. fatua*, and *3* represents *Digitaria sanguinalis*, *Echinochloa crus-galli*, and *Panicum capillare*. Data shown are means ± SE.

the presence or absence of the virus. We also included a nitrogen addition treatment; the results from the nitrogen addition are presented elsewhere (C. E. Mitchell and A. G. Power, unpublished manuscript).

Methods

In 2001, we established a factorial experiment to test the joint effects of the inclusion of *A. fatua* in the host community, virus inoculation, and nitrogen addition on pathogen spread and plant community structure. Methods were similar to those employed in previous years, except as follows. The experiment used a blocked split-plot design, with the nitrogen addition treatment applied at the whole-plot level and the community and virus treatments completely crossed at the split-plot level. Here we present only results from the split-plot level treatments; these effects were tested against the model residual error. There were no significant interactions between the effects we report and the whole-plot nitrogen treatment. Analyses were conducted in Systat version 9 (SPSS).

Thirty-two experimental communities (1-m^2 subplots) were established, comprising eight whole plots. The whole plots were arranged in four replicate blocks. Whole plots were separated by 3 m and subplots by 1 m. All experimental communities were planted with *Digitaria sanguinalis*, *Lolium multiflorum*, and *Setaria lutescens*. Half the communities were additionally planted with *A. fatua*. All communities received a total of 600 seeds divided equally among all species planted. Plots were planted and caged on June 13, 2001. Three weeks later, each community was either virus inoculated or mock inoculated, and communities in the nitrogen addition treatment received 4 g N m^{-2} in the form of NH_4NO_3 fertilizer. An identical second dose of nitrogen was applied 18 days after the first. Each replicate block was harvested in a single day over a 10-day period at the end of the growing season. As before, at harvest, we measured the number of plants, the total aboveground vegetative biomass, and the total reproductive biomass for each host species in each plot. We also estimated virus prevalence for each species in each plot by subsampling 20 plants of each grass species in each plot and testing for BYDV using enzyme-linked immunosorbent assay. At harvest, *D. sanguinalis* was missing from one plot in which it had been planted (i.e., no individuals were found, presumably because of poor germination), and *L. multiflorum* was similarly missing from another plot. We used standard least squares estimates of these data points in the analysis of virus prevalence.

Results

The presence of *A. fatua* led to higher virus prevalence across the three less heavily infected species, demonstrating pathogen spillover (fig. 3; MANOVA: $F = 3.59$, df $= 3, 16$, $P = .037$). Spillover from *A. fatua* approximately doubled virus prevalence in all three other species, increasing it by 76%–114%. Pathogen spillover from *A. fatua* was stronger for *L. multiflorum* and *S. lutescens* than for *D. sanguinalis* on the basis of canonical loadings of the MANOVA (0.62, 0.53, and 0.15, respectively).

Pathogen spillover from *A. fatua* resulted in apparent competition between *A. fatua* and both *L. multiflorum* and *D. sanguinalis*. Comparing the effects of *A. fatua* presence on their biomass in inoculated and uninoculated communities reveals the negative effects of apparent competition. Specifically, the presence of *A. fatua* increased the aboveground vegetative and reproductive biomasses of *L. multiflorum* and *D. sanguinalis* in uninoculated communities but decreased their biomasses in inoculated communities (fig. 4; MANOVA *A. fatua* × BYDV interaction; vegetative: $F = 4.6$, df $= 3, 16$, $P = .016$; reproductive: $F = 3.7$, df $= 3, 16$, $P = .035$). This indicates that the negative effects of *A. fatua* on *L. multiflorum* and *D. sanguinalis* were mediated by the presence of the shared pathogen, demonstrating apparent competition. Apparent competition from *A. fatua* had a stronger impact on *L. multiflorum* than *D. sanguinalis* and a negligible effect on *S. lutescens* (fig. 4; canonical loadings; vegetative: 0.70, 0.39, and 0.007, respectively; reproductive: 0.66, 0.45, and 0.007, respectively). The positive effect of *A. fatua* presence

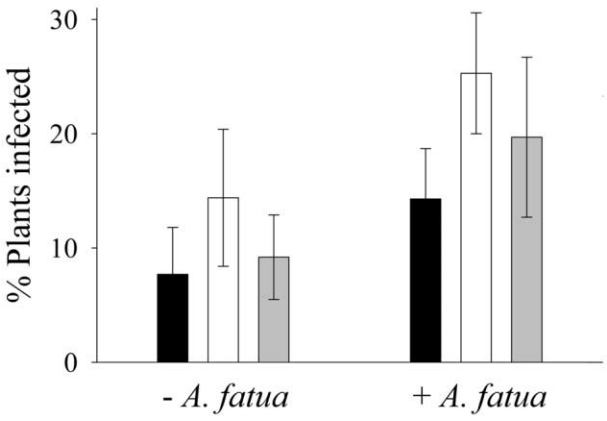

Figure 3: Virus prevalence across *Digitaria sanguinalis* (*black bars*), *Lolium multiflorum* (*white bars*), and *Setaria lutescens* (*gray bars*) in experimental communities planted with *Avena fatua* or lacking *A. fatua* in 2001. The presence of *A. fatua* increased virus prevalence across the other three species ($P < .05$), demonstrating pathogen spillover. Data shown are means ± SE

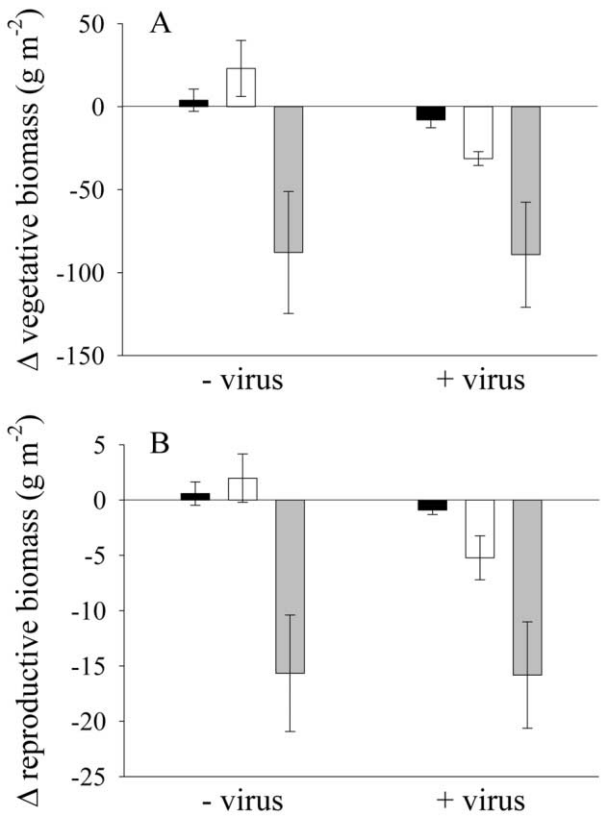

Figure 4: Effect of adding *Avena fatua* on (*A*) aboveground vegetative and (*B*) reproductive (seedhead) biomass of *Digitaria sanguinalis* (*black bars*), *Lolium multiflorum* (*white bars*), and *Setaria lutescens* (*gray bars*) in experimental communities in 2001. Communities were either inoculated with the virus (+virus) or mock inoculated using nonviruliferous aphids (−virus). Adding *A. fatua* decreased *D. sanguinalis* and *L. multiflorum* biomass only in virus-inoculated communities ($P < .05$), demonstrating virus-mediated apparent competition.

on *L. multiflorum* and *D. sanguinalis* biomass in uninoculated communities indicates that *L. multiflorum* and *D. sanguinalis* benefited more indirectly from *A. fatua*'s strong negative competitive effect on *S. lutescens* than they were harmed by direct competition with *A. fatua*. Thus, two types of indirect effects, one mediated by a shared pathogen and another mediated by a shared competitor, both controlled the biomass of these two species.

Pathogen-mediated apparent competition also influenced the distribution of plant species biomasses, as measured by community evenness. *Avena fatua* presence increased evenness of uninoculated communities ($P = .039$) but not inoculated communities (fig. 5; Tukey HSD: $P = .83$). *Avena fatua* increased evenness of uninoculated communities by increasing the biomass of the two subdominant species, *L. multiflorum* and *D. sanguinalis* (fig. 4), apparently by decreasing competition from the dominant species, *S. lutescens*. In contrast, *A. fatua* had no net effect on evenness of inoculated communities because these positive indirect effects of *A. fatua* on the two subdominant species were outweighed by its negative effects on them via pathogen spillover and apparent competition (fig. 4). This suggests that community evenness was also controlled jointly by two types of indirect effects.

Consequences of Pathogen Spillover in Plant Communities

These results provide experimental evidence for the occurrence and ecological importance of pathogen spillover in field populations of nondomesticated species. While pathogen spillover has been widely reported in the past (Daszak et al. 2000), its operation has generally been inferred post hoc. In this system, experimentally manipulating the presence of a heavily infected reservoir grass species, *Avena fatua*, dramatically altered disease dynamics in a suite of nonreservoir grasses. Virus prevalence in other host species was generally greater in communities including *Avena* than in other communities. In some cases (e.g., *Setaria lutescens* in 1999), the presence of *Avena* made the difference between almost undetectably low prevalences (<4% infected) and full-blown epidemics (>40% infected). Thus, infection prevalence in nonreservoir species was primarily determined by the community context in which they were embedded, specifically whether the community included the reservoir species. In a broader ecological context, the occurrence of pathogen spillover reinforces the general perspective that understanding the ecological dy-

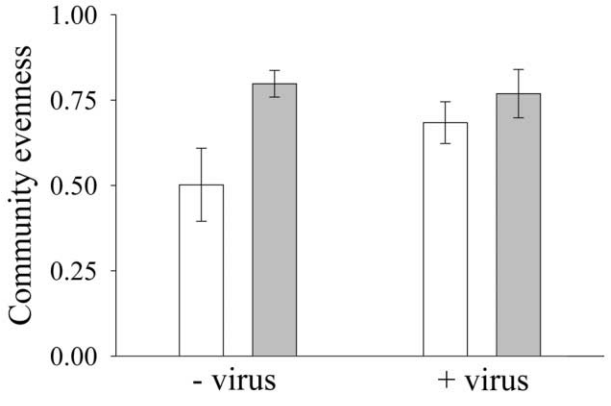

Figure 5: Evenness of experimental communities either including *Avena fatua* (*gray bars*) or not including *A. fatua* (*white bars*) in 2001. All communities included *Digitaria sanguinalis*, *Lolium multiflorum*, and *Setaria lutescens*. Communities were either inoculated with the virus (+virus) or mock inoculated using nonviruliferous aphids (−virus). Adding *A. fatua* increased community evenness in the absence, but not presence, of apparent competition.

namics of a focal population, community, or ecosystem may often require consideration of the dynamics in connected populations, communities, or ecosystems (e.g., Polis et al. 1997; Hoopes and Harrison 1998).

Pathogen spillover resulted in apparent competition among host species. Apparent competition mediated by a shared natural enemy has rarely been demonstrated in field experiments (Holt and Lawton 1994; Muller and Godfray 1997; Alexander and Holt 1998; Hudson and Greenman 1998; Morris et al. 2001, 2004). In this case, inoculating experimental communities with the shared virus converted what was a positive effect of *Avena* on *Digitaria* and *Lolium* biomass in uninoculated communities to a negative effect. This fundamental change in the sign of these interactions from facilitation to inhibition resulted from the presence of the shared virus, demonstrating apparent competition. The fact that *Setaria* received pathogen spillover from *Avena* but did not experience negative effects of apparent competition suggests that it is more tolerant of infection than *Digitaria* and *Lolium*. Thus, tolerance may be a key determinant of the outcome of pathogen-mediated apparent competition.

Presumably, presence of *Avena* increased *Digitaria* and *Lolium* biomass in uninoculated communities through further indirect effects mediated by *Setaria*. In the absence of *Avena*, *Setaria* was competitively dominant. Our results suggest that, when *Avena* was added to uninoculated communities, the benefit to *Digitaria* and *Lolium* from reduced competition from *Setaria* outweighed the disadvantages of additional competition from *Avena*. We are conducting further experiments to quantify the interaction strength of all the direct and indirect effects among these four grasses and their shared virus to determine their relative importance in controlling community structure.

The results of the *Avena* addition experiment suggest that pathogen-mediated apparent competition was a major force structuring communities that included *Avena*. As a result of its differential effects on the relative abundances of *Digitaria*, *Lolium*, and *Setaria*, apparent competition influenced the evenness of the plant community. To our knowledge, this is the first field evidence for such a community-level effect of apparent competition. Further experiments are examining the importance of apparent competition relative to resource competition in structuring this suite of grass species.

Experimentally manipulating the presence of the reservoir species *Avena* may have been essential to detecting pathogen spillover and apparent competition. In unmanipulated communities, apparent competition resulting from pathogen spillover could mask the appearance of pathogen spillover. That is, apparent competition could reduce the abundance of the nonreservoir species, perhaps even excluding them from habitat occupied by the reservoir. In such a case where past spillover and apparent competition have excluded or reduced the abundance of nonreservoir hosts, spillover and apparent competition would appear to be weak forces. Essentially, all that would remain is the "ghost" of apparent competition past, similar to the "ghost of competition past" (Connell 1980). Because apparent competition should be typical of interactions with generalist pathogens, this line of reasoning suggests that experimental manipulations of host populations will greatly facilitate quantification of rates of interspecific pathogen transmission. Communities of annual plants can be manipulated on small spatial and temporal scales, making them excellent experimental model systems for testing theoretical predicted dynamics of multihost systems, such as pathogen spillover and apparent competition.

Implications of Spillover for Disease Epidemiology and Conservation

Pathogen spillover is likely to be an important force structuring populations and communities at a variety of scales. The results of our experimental work suggest that spillover at relatively small scales can result in pathogen-mediated apparent competition that shapes community structure and community attributes like evenness. A growing body of observational studies suggests that spillover at larger scales may be a potent force shaping the population dynamics of nondomesticated species. In particular, spillover of pathogens from domestic animals can have enormous impacts on wildlife populations, including endangered ones (Gascoyne et al. 1993; Roelke-Parker et al. 1996; Sillero-Zubiri et al. 1996; Laurenson et al. 1997; Daszak et al. 2000). Although pathogen spillover from agricultural crops has received less attention, it is likely to be equally important for some plant species (Gilbert and Hubbell 1996). Our increasing ability to detect and monitor disease in wild populations is likely to reveal many additional cases of pathogen spillover shaping populations and communities.

This growing recognition of the frequency of pathogen spillover in nature requires increased attention to the development of epidemiological theory for pathogens with multiple hosts (e.g., Holt et al. 2003). At the same time, we need to identify easily manipulated experimental systems in which to test the predictions of this developing theory, since many disease systems are logistically intractable. Here we argue that annual plants and their pathogens may serve as useful model systems for exploring the epidemiology of generalist pathogens and their role in shaping ecological communities.

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