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EFFECTS OF GRASSLAND PLANT SPECIES DIVERSITY, ABUNDANCE, AND COMPOSITION ON FOLIAR FUNGAL DISEASE

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Abstract. In an experiment that directly manipulated grassland plant species richness and composition, decreased plant species richness (''diversity'') increased pathogen load (the percentage of leaf area infected by species-specific foliar pathogens across the plant community) in 1998. Pathogen load was almost three times greater in the average monoculture than in the average plot planted with 24 grassland plant species, an approximately natural diversity. Eleven individual diseases increased in severity (percentage of leaf area infected by a single disease) at lower plant species richness, and severity of only one disease was positively correlated with diversity. For 10 of the 11 diseases whose severity was negatively related to diversity, disease severity was positively correlated with host abundance, and in six of these cases, species diversity had no effect on disease severity after controlling for the effects of host abundance. These results suggest that increased abundances of individual host species at lower species diversity increased disease transmission and severity. In 1996 and 1997, similar results for a smaller number of diseases sampled were found in this experiment and another similar one. Although the effect of diversity on disease was highly significant, considerable variance in pathogen load remained among plots of a given diversity level. Much of this residual variance was explained by community characteristics that were a function of the species composition of the communities (the identity of species present vs. those lost). Specifically, communities that lost less disease-prone species had higher pathogen loads; this effect explained more variance in pathogen load than did diversity. Also, communities that lost the species dominant at high diversity had higher pathogen loads, presumably because relaxed competition allowed greater increases in host abundances, but this effect was weak. Among plant species, disease proneness appeared to be determined more by regional than local processes, because it was better correlated with frequency of the plant species' populations across the region than with local abundance or frequency across the state. In total, our results support the hypothesis that decreased species diversity will increase foliar pathogen load if this increases host abundance and, therefore, disease transmission. Additionally, changes in community characteristics determined by species composition will strongly influence pathogen load.

Key words: biodiversity and ecosystem functioning; functional composition; fungal pathogens; global change; habitat simplification; host density; macroecology; parasites; plant community; plant pathogens; polyculture vs. monoculture; species richness.

INTRODUCTION

Decreased local species diversity is a widespread impact of human activity (Groombridge 1992, Pimm et al. 1995, Vitousek et al. 1997), and may result in decreased primary production (Naeem et al. 1994, Tilman et al. 1996, 1997*a*, Hector et al. 1999). The two major proposed mechanisms for this effect of diversity on productivity are that lower species richness decreases the probability that species with key traits will be present in the community (the sampling effect; Aarssen 1997, Huston 1997, Tilman et al. 1997*b*), and that a less diverse community of competing species would utilize resources less completely (niche complementarity; Naeem et al. 1994, Tilman et al. 1996, 1997*b*, Hector et al. 1999). In addition to these mechanisms resulting from altered competitive interactions, losses of species diversity may alter interactions such as mutualism, predation, herbivory, or infectious disease (Bond 1993, McNaughton 1993, Chapin et al. 1997, 2000) in ways that decrease primary production, but this possibility has received little attention. In this paper, we report a test of the long-standing hypothesis (Elton 1958, van der Plank 1963) that decreased plant species diversity increases the severity of diseases, particularly those caused by specialist plant pathogens. This hypothesis is well supported for small numbers of plant species or genotypes (Wolfe 1985, Hagle and Goheen 1988, Burton et al. 1992, Gilbert and Hubbell 1996, Boudreau and Mundt 1997, Finckh and Wolfe 1997, Garrett and Mundt 1999, Zhu et al. 2000), but whether losses of diversity when diversity is near nat-

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ural (high) levels influence disease severity has not been tested experimentally. We tested this hypothesis using two well-replicated, large-scale field experimental communities, containing up to 32 plant species, in which plant species richness (hereafter, ''diversity'') was directly controlled (Tilman et al. 1996, 1997*a*).

The hypothesis that decreased plant species diversity should increase the severity of diseases caused by specialist pathogens (the diversity–disease hypothesis) is based on fundamental epidemiological and ecological principles. Because of relaxed interspecific competition (Aarssen 1997, Huston 1997, Tilman et al. 1997*b*), decreased plant species richness should, on average, increase abundances of one or more remaining species within the local community, and thus the abundance of one or more hosts for specialist pathogens. A basic premise of epidemiology is that increased host abundance (''host density'' in plant pathology) increases disease transmission, both initially into a population and subsequently within it, resulting in increased disease severity (Anderson and May 1979, Burdon 1987, Antonovics et al. 1995). This provides a simple mechanism for the diversity–disease hypothesis: a decreased number of competing plant species may allow the abundances of other species to increase locally, facilitating the spread of their diseases (Burdon and Chilvers 1976, Chapin et al. 1997, Knops et al. 1999). A necessary condition for ecological effects of species diversity is that species differ in some capacity (Tilman and Lehman 2002); in this case, species differ in the diseases to which they are susceptible. However, this local mechanism is unlikely to explain differences in disease level at larger spatial scales because host abundance will also be controlled by numerous other biotic and abiotic factors that differ among communities. Additionally, in communities that decrease in diversity, host abundance is not the only possible mechanism linking diversity and disease; other factors that change as a result of species loss, such as microclimate and host competitive status, may also influence disease levels (Boudreau and Mundt 1992, 1994, 1997, Zhu et al. 2000).

The published studies most relevant to the diversity–disease hypothesis come almost entirely from agriculture and silviculture. One observational study of natural communities found that higher diversity communities did not have lower disease levels (Kranz 1990), but the high-diversity communities were forests and pastures, and the low-diversity communities were meadows and agricultural fields, so species diversity was confounded with species composition, microclimate, and numerous other variables. In studies of agronomic intercrops (multiple crop species in a field), increased diversity decreases disease more often than increases it, particularly for fungal diseases. However, these studies are few and often inconsistent (reviewed by Boudreau and Mundt 1997). Experimental variety mixtures or multilines (multiple genotypes of one crop species in a field) reduce disease severity more consistently than intercrops, particularly for diseases caused by aerially dispersed fungi (Wolfe 1985, Boudreau and Mundt 1997, Finckh and Wolfe 1997, Garrett and Mundt 1999, Zhu et al. 2000). In general, reduced host abundance is the most important mechanism by which diversity reduces disease severity, particularly for fungal diseases, in agricultural systems (Burdon and Chilvers 1977, 1982, Chin and Wolfe 1984, Wolfe 1985, Alexander et al. 1986, Burdon 1987, Boudreau and Mundt 1997, Garrett and Mundt 1999, Zhu et al. 2000). However, in intercrops, changes in microclimate and host competitive status can also strongly affect disease severity, either positively or negatively (Boudreau and Mundt 1992, 1994, 1997, Zhu et al. 2000). In forestry, it is well established that increases in the abundance of host species, as result of decreased diversity or other changes, promote disease spread (Hagle and Goheen 1988, Burton et al. 1992, Gilbert and Hubbell 1996, Gerlach et al. 1997). In contrast to nonvectored foliar fungal pathogens, spread of plant viruses can be inhibited by increased plant genetic or species diversity as a result of altered vector abundance or behavior (Power 1987, 1991). Although agricultural and silvicultural studies overwhelmingly support the diversity–disease hypothesis, extrapolation from managed to natural ecosystems is uncertain because these systems generally differ in epidemiologically relevant ways, such as species diversity and physical heterogeneity.

A community that loses species through human actions is altered in species composition (i.e., the identity of species present) as well as richness. The effect of losing a given number of species on disease level across a plant community may differ markedly depending on the identity of the species lost, because their identity will determine the community's subsequent species composition. Species composition may influence community disease level by determining two aggregate characteristics of the community: disease proneness and dominant species presence. For instance, the loss of species that are less disease prone will leave species that are more disease prone, thereby increasing community disease proneness. Therefore, we hypothesized that the loss of species that are less disease prone would increase disease level across the plant community more than would the loss of more disease-prone species, all else being equal. This effect would not occur if the loss of diversity altered the relative disease proneness of the species. Similarly, the loss of dominant species, (i.e., a decrease in dominant species presence) should allow remaining species to increase in abundance more than would the loss of rare species. Therefore, we hypothesized that the loss of more dominant species would increase the pathogen load of species-specific diseases more than if rare species were lost, all else being equal (i.e., dominant species presence and disease severity across the plant community will be neg-

atively correlated). However, in both of these scenarios, all else may not be equal, because more abundant species may also be more disease prone (Arneberg et al. 1998), in which case the effects of losing dominant and disease-prone species on pathogen load would be countervailing. Alternately, a trade-off between competitive ability and disease resistance would cause these effects to magnify each other if abundance were determined by competitive ability. Additionally, because species abundance may be correlated with extinction risk (Tilman et al. 1994, Simberloff 1998), a correlation between abundance and disease proneness would predict a correlation between extinction risk and disease proneness. These effects are analogous to processes occurring in agricultural mixtures, but such studies have generally focused on disease levels of each mixture component rather than across the entire mixture of species or genotypes (Knott and Mundt 1990, Newton et al. 1997).

Because differences in species richness necessarily imply differences in species composition, composition can explain large amounts of the variance in ecosystem properties when diversity is varied (Tilman et al. 1997*a*, 2002, Hooper and Vitousek 1997, 1998, Symstad 1998). These previous studies have generally tested the effects of the presence or absence of specific functional groups on ecosystem processes, with functional groups constructed by grouping species that share a relatively discrete trait, such as symbiotic nitrogen fixation or C_4 photosynthesis. In contrast, understanding the effects of more continuous characteristics on ecosystem properties may best be accomplished by calculating the mean value of that characteristic for each experimental or observed ecosystem. We have used this approach to test the effects of community disease proneness and dominant species presence on disease severity across the plant community (pathogen load).

Here, we test the diversity–disease hypothesis using experimental communities of perennial grassland plants in which species diversity was directly controlled (Tilman et al. 1996, 1997*a*), an approach complementary to past experiments with few species of annual crop plants and observations of managed forests. We quantified the severity of all foliar diseases found in the experiment in 1998 and developed a measure of disease severity across the entire plant community. Our experimental species richness treatments ranged from approximately natural levels to monoculture. We tested the effect of host abundance (percent cover), the major proposed mechanism linking diversity and disease, on disease severity. Additionally, we tested the effects of two community characteristics, disease proneness and dominant species presence, on disease level across the plant community. Finally, we tested whether more dominant or widespread species were more disease prone. Initial results for four diseases sampled from one of our experiments in 1997 supported the diversity–disease hypothesis and its proposed mechanism (Knops et al. 1999). Here, we report results for 29 plant diseases sampled over three years in two experiments.

Materials and Methods

Study system

We tested the diversity–disease hypothesis using naturally occurring foliar plant pathogens, almost all of which were fungal. Foliar fungal pathogens of wild plants are generally specific to one or a few plant species in a given community, and almost all wild plant species are host to leaf-infecting pathogenic fungi (Farr et al. 1989). The known species of fungal plant pathogens are orders of magnitude more diverse than the known viral and bacterial plant pathogens (Agrios 1988). Furthermore, fungal pathogens have greater impacts on agriculture than any other type of pathogen, causing losses roughly equal to those from insect herbivores (Cramer 1967). Because of their economic importance, fungal pathogens of crops are well studied, providing a base of knowledge that facilitates the taxonomic identification and quantification of wild populations (Farr et al. 1989, Campbell and Madden 1990).

Experimental setup

We performed our experiments using perennial grassland plants at Cedar Creek Natural History Area, Minnesota, USA, ~50 km north of Minneapolis/St. Paul. We assessed disease in two well-replicated experiments in which plant species richness was directly manipulated. The smaller experiment, Biodiversity I (described in Tilman et al. 1996), consisted of 147 plots, each 3×3 m, separated by 1-m walkways. Each plot was randomly assigned a species richness treatment of 1, 2, 4, 6, 8 (20 replicates each), 12 (23 replicates), or 24 (24 replicates) species. The species composition of each plot was determined by separate random draws from a pool of 24 perennial grassland species (see Table 1 for species identities). Thus, the species composition of plots with richness <24 species was a random subset of the plots with richness of 24 species, so species richness was randomized across species composition, allowing their effects to be separated (Tilman and Lehman 2002). In fall 1993, existing vegetation was killed with herbicide and burned, the top ~7 cm of soil were removed to reduce the seedbank, and the area was plowed and thoroughly disked. In May 1994, each plot was seeded with 10 g/m^2 seed to achieve approximately natural total plant density; this total was divided equally among the species planted. To maintain the treatment species richness and composition, plots were hand-weeded from elevated boardwalks throughout each growing season. Density of each species planted was allowed to vary naturally after initial planting. All plots received ~2.5 cm of water per week during each growing season, with supplemental

TABLE 1. Foliar fungal diseases sampled, by experiment and year, in Biodiversity I and II grassland plots in Minnesota.

Plant species name, code†	Pathogen species	Disease type	Disease severity range‡	Years sampled	
				Bio I	Bio II
Achillea millefolium, 501	unidentified	leaf necrosis	0.0–34.3	1998	N/A
Agropyron smithii, 108	unidentified	fungal leaf spot	0.2–1.8	1998	N/A
Andropogon gerardi, 102	*Phyllosticta* sp.	fungal leaf spot	1.3–12.4	1998	N/A
Anemone cylindrica, 504	*Mycosphaerella* sp.	fungal leaf spot	0.2–26.0	1998	N/A
Asclepias tuberosa, 510	*Septoria* sp.	fungal leaf spot	0.0–7.2	1996, 1998	N/A
Aster azureus, 512	*Septoria* sp.	fungal leaf spot	0.0–9.8	1996, 1998	N/A
Astragalus canadensis, 411	unidentified	fungal leaf spot	0.1–10.7	1998	N/A
Bouteloua gracilis, 139	*Bipolaris* sp.	fungal leaf spot	0.3–8.8	1998	N/A
Buchloe dactyloides, 130	unidentified	fungal leaf spot	0.0–0.4	1998	N/A
Coreopsis palmata, 518	unidentified	bacterial leaf spot	0.0–11.5	1998	N/A
Coreopsis palmata, 518	unidentified	fungal leaf spot	0.0–18.6	1998	N/A
Elymus canadensis, 140	unidentified	fungal leaf spot	1.0–9.2	1998	N/A
Euphorbia corollata, 523	*Puccinia emaculata*	heteroecious rust	2.7–58.0	1996–1998	N/A
Koeleria cristata, 145	*Puccinia liatridis*	heteroecious rust	0.4–5.5	1998	N/A
Lespedeza capitata, 403	*Uromyces lespedezae-procumbentis*	autoecious rust	0.3–5.2	1996, 1998	1996
Lespedeza capitata, 403	*Colletotrichum* sp.	fungal leaf spot	0.0–10.4	N/A	1996
Liatris aspera, 538	*Puccinia liatridis*	heteroecious rust	0.0–5.0	1998	N/A
Liatris aspera, 538	*Septoria liatridis*	fungal leaf spot	0.0–13.4	1996, 1998	N/A
Monarda fistulosa, 533	*Erysiphe cichoracearum*	powdery mildew	0.0–41.3	N/A	1996
Panicum virgatum, 114	*Puccinia emaculata*	heteroecious rust	0.6–10.4	1997–1998	N/A
Panicum virgatum, 114	unidentified	fungal leaf spot	0.0–17.3	1998	N/A
Petalostemum purpureum, 652	unidentified	fungal stem spot	0.0–16.8	1998	N/A
Poa pratensis, 117	unidentified	fungal leaf spot	0.0–1.6	1998	N/A
Rudbeckia hirta, 552	*Septoria rudbeckiae*	fungal leaf spot	0.0–34.5	1998	N/A
Schizachyrium scoparium, 103	*Colletotrichum* sp.	fungal leaf spot	2.8–11.7	1996, 1998	1996
Solidago nemoralis, 561	*Cercospora* sp.	fungal leaf spot	0.0–9.0	1998	N/A
Sorghastrum nutans, 120	*Colletotrichum* sp.	fungal leaf spot	1.7–14.1	1996, 1998	N/A
Sporobolus cryptandrus, 121	*Helminthosporium* sp.	fungal leaf spot	0.6–4.6	1998	N/A
Vicia villosa, 406	unidentified	fungal leaf spot	0.0–0.1	1998	N/A

Notes: For taxonomic authorities, see Farr et al. (1989) and Kartesz (1994). N/A, not applicable.

† All species planted in Biodiversity I were sampled for disease. Species planted in Biodiversity II not sampled for disease are listed in Tilman et al. (1997*a*).

‡ Range is based on minimum and maximum plot averages in Biodiversity I in 1998, except for two diseases (*Lespedeza* leaf spot, *Monarda* powdery mildew) not sampled there then, for which the range is for Biodiversity II in 1996.

watering by sprinklers performed weekly if natural rainfall was <2.5 cm. After planting, abundances of planted species in each plot were allowed to vary naturally; by 1998, community structure in the plots planted with 24 species closely resembled that of nearby naturally occurring grasslands (Tilman et al. 2002).

The larger experiment, Biodiversity II (described in Tilman et al. 1997*a*), was similar to Biodiversity I, except as follows. It consisted of 342 plots, each 13 × 13 m. Of these, 167 were randomly assigned species richness treatment levels of 1, 2, 4, 8, or 16 species (29–35 replicates each) chosen from a pool of 18 species. Another 46 plots were seeded with 32 species randomly drawn from a 34-species pool consisting of the previous 18 species plus 16 other species. The plots were seeded in May 1994 and then reseeded at half the original rate in May 1995. To maintain the species richness treatment, plots were hand-weeded from within and sprayed with selective herbicides if compatible with the treatment species composition. Supplemental watering was performed as in Biodiversity I. The entire experiment was subject to a controlled burn each spring.

Sampling

Almost all plant species in both experiments were naturally colonized by populations of foliar fungal pathogens by 1996. Disease severity was defined as the percentage of leaf area of each plant species visibly infected by the pathogen. The percentage of leaf area infected is often proportional to the effects of the disease on the host, and was estimated visually in the field using cards with digitized images of leaves of known disease severity for reference, a standard technique in plant pathology (James 1971, Campbell and Madden 1990). We do not report disease incidence (proportion of plants or leaves infected) because this was consistently near 1 for most of the diseases. The one exception to this approach was the rust on *Euphorbia* in 1996, for which we estimated disease severity as the proportion of plant leaves infected, because infected plant leaves varied little in the degree of infection. We quantified the severity of each disease as close to its annual peak severity as possible. Because diseases peaked at different times, sampling occurred multiple times each season, and was nearly continuous from June through

September in 1998. In 1996, we quantified disease severity in four transects parallel to each plot edge. In 1997 and 1998, we quantified disease severity in 1 × 0.5 m permanent sampling quadrats (two per plot) where percent cover was also measured in order to better correlate disease severity and host abundance. In each plot, we sampled five plants of each species, as regularly spaced as possible in each quadrat or transect, by blindly choosing (looking away while outstretching a hand until a leaf, infected or not, was encountered) and visually inspecting five leaves per broadleaf stem or grass bunch. For plant species infected by more than one disease, each was quantified separately. In 1996, we sampled the 11 most prevalent diseases across both experiments. In 1997, we sampled the four most prevalent diseases in Biodiversity II (Knops et al. 1999) and the most prevalent disease in Biodiversity I. In 1998, we sampled all foliar diseases found on all plant species seeded into Biodiversity I. Table 1 describes the diseases sampled; in total, >100 000 leaves were sampled.

In all years, the percent cover of each species in the 0.5-m^2 permanent sampling quadrats was visually estimated (Tilman et al. 1996, 1997*a*); we used this as our primary estimate of host abundance. As an additional measure of host abundance in 1997 and 1998, we counted the number of that year's forb stems and grass bunches in each quadrat for each species that we assessed. Results using this measure differed little from those using percent cover, so we report only analyses using percent cover to facilitate comparison among years. Species richness observed in the percent cover permanent quadrats was strongly correlated with the species richness treatment (Tilman et al. 1996). The two were not perfectly correlated because our sampling quadrats only sample a portion of each plot, weeds could not be completely eradicated, and some planted species did not establish uniformly.

To test the correlation between disease proneness and host abundance at larger spatial scales, we also estimated the geographic extent of each species, both across the Great Plains and across Minnesota, by counting the number of counties in which each plant has been collected, based on published floristic atlases (Barker and Barkley 1977, Ownbey and Morley 1991).

Analysis

Disease severity of a plant species was defined as the percent leaf area visibly infected by a pathogen. Disease severity of all leaves of a given species sampled in a plot was averaged, and analyses were conducted on plot averages. In 1998, quantifying disease severity on all species planted in Biodiversity I allowed us to estimate community-wide disease severity. We call community-wide disease severity the pathogen load, *l*, because it is essentially the percentage of leaf area infected by disease across the entire plant community and, thus, is analogous to herbivore load, the ratio of herbivore to plant biomass (Root 1973, Andow 1991). We distinguish pathogen load from disease severity because pathogen load allows comparison among communities differing, in whole or part, in either the diseases or hosts present. Therefore, pathogen load is unlikely to correspond to effects on the plants as directly as severity of a single disease on a single host. We calculated pathogen load for each plot as a weighted average of the disease severity of each species planted in that plot, with disease severity of each species being weighted by the percent cover of that species in that plot:

$$l = \frac{\sum_{i=1}^{n} s_i c_i}{\sum_{i=1}^{n} c_i} \tag{1}$$

where s_i is disease severity of the *i*th species, c_i is cover of the *i*th species, and n is the number of species planted in the plot.

Because communities of equal diversity differed in species composition (the identity of species present) in ways that could influence pathogen load, we tested the influence of two community characteristics, disease proneness and dominant species presence, on pathogen load in Biodiversity I in 1998. We therefore calculated community indices of disease proneness and dominant species presence for each plot in the experiment based on the disease severity and percent cover, respectively, of each species at the approximately natural level of diversity. To do this, we first estimated how disease prone and dominant each species was at approximately natural levels of diversity by determining its mean disease severity and percent cover, respectively, across the plots planted with 24 species. Then, we calculated community disease proneness, *p*, for each plot by calculating a weighted average of the disease proneness of all species planted in the plot, with each species' disease proneness weighted by its percent cover in that plot:

$$p = \frac{\sum_{i=1}^{n} a_i c_i}{\sum_{i=1}^{n} c_i} \tag{2}$$

where a_i is the average disease severity in the plots planted with 24 species of the *i*th species. The index of dominant species presence, *d*, was calculated similarly, except that an unweighted average was used instead of a weighted average:

$$d = \frac{\sum_{i=1}^{n} h_i}{n} \tag{3}$$

where h_i is the average cover in the plots planted with 24 species of the *i*th species. Because both community

disease proneness and dominant species presence are direct functions of species composition, we view their effects as effects of species composition. Dominant species presence does not directly measure evenness, so we also tested whether community evenness influenced pathogen load. We based these parameters on disease proneness and dominance at approximately natural levels of diversity because, due to random chance, not all species were planted in monoculture, and because this method is more easily applied to habitat management: it is easier to measure disease in the field than to plant monocultures. In this analysis, one species, *Buchloe dactyloides*, was ignored because we did not encounter *Buchloe* in the sampling quadrats of any plots planted with 24 species, and therefore could not determine its contribution to disease proneness or dominant species presence. Consequently, the single *Buchloe* monoculture plot was excluded from this analysis, reducing degrees of freedom by one.

All regressions were unweighted ordinary least squares regressions. We transformed disease severity, pathogen load, and/or species richness treatment by the natural logarithm or square root, as necessary, to better satisfy the assumptions of constant variance and linearity, based on inspection of residual plots. When testing the same hypothesis with multiple diseases, we report both sequential Bonferroni-adjusted *P* values (Rice 1989) and unadjusted *P* values in the tables, but for simplicity we focus on unadjusted *P* values in the text. Across all analyses, 71% of regressions significant without the sequential Bonferroni adjustment were still significant with the adjustment. Whenever a few highly influential data points were detected, the analysis was similarly conducted with those points excluded; except when otherwise reported, the influential points did not alter the significance of the predictor variables. Analyses were performed in SYSTAT for Windows, Version 5.05 (SYSTAT 1994).

Results

Effects of species diversity and composition on pathogen load

Our broadest test of the diversity–disease hypothesis was to perform a linear regression of pathogen load, our measure of disease severity across the plant community, on plant species richness treatment for Biodiversity I in 1998. Pathogen load increased significantly as the natural log of plant species richness treatment decreased (Fig. 1A). Pathogen load increased from a mean of 4.3 in the plots planted with 24 species to a mean of 11.7 in the monocultures, a 2.7-fold increase. Maximum pathogen load increased dramatically across our experimental gradient, from 7.0 in the plots planted with 24 species to 34.3 in monoculture, a 4.9-fold increase. This large increase in the upper bound, combined with a small decrease in the lower bound from 0.97 to 0.27, led to a greater coefficient of variation in pathogen load as plant diversity decreased (Fig. 1B).

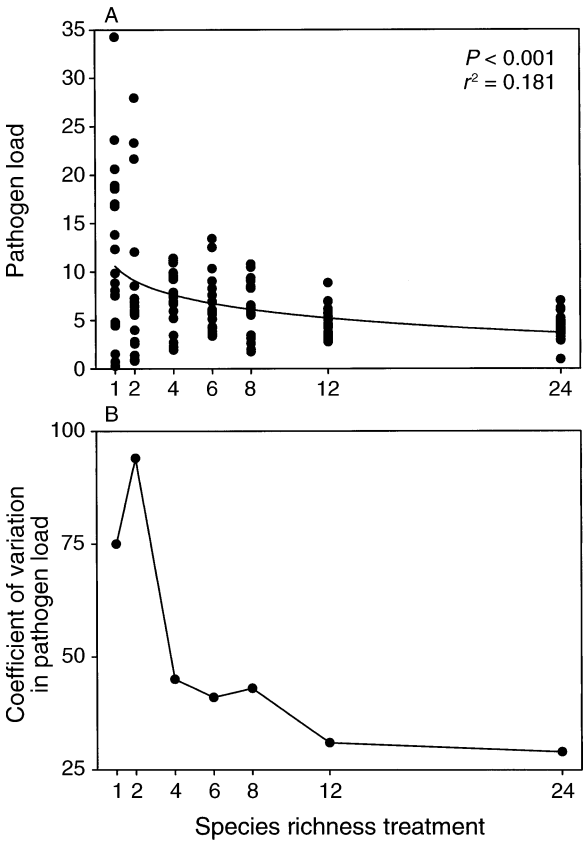

FIG. 1. The effect of plant species richness treatment (note log scale) on (A) pathogen load (community-wide disease severity) and (B) coefficient of variation in pathogen load in Biodiversity I experimental grassland plots in 1998.

Because large amounts of variance in pathogen load remained after controlling for diversity, we tested the simultaneous effects of diversity and species composition, in terms of disease proneness and dominant species presence, on pathogen load in Biodiversity I in 1998. In a multiple regression, the effect on pathogen load of these community characteristics was greater than that of diversity. Square-root transformed pathogen load was significantly positively related to the log of disease proneness, and significantly negatively related to both the log of dominant species presence and the log of species richness treatment (overall $r^2 = 0.407$; Fig. 2). In this multiple regression, disease proneness explained 25% of the total variance in pathogen load, dominant species presence explained 5%, and diversity explained 10%. Adding community evenness to this multiple regression model did not significantly increase r^2 (doing so by <0.001) and changed the effects of the other variables by minuscule amounts. Because disease proneness and dominant species presence were calculated using data from the plots planted with 24 species, we excluded these plots from these

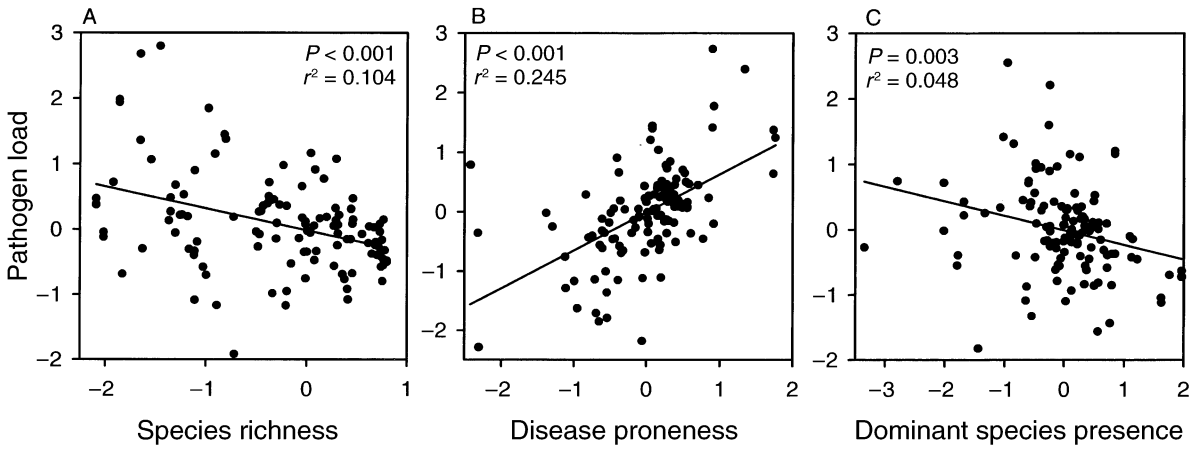

FIG. 2. Added-variable plots showing the simultaneous effects of ln(species richness treatment) and two community characteristics influenced by species composition, ln(disease proneness) and ln(dominant species presence), on the square root of pathogen load. Reported *P* values and coefficients of determination are for partial regression coefficients. (A) The effect of ln(species richness treatment) on the square root of pathogen load, after controlling for the effects of both ln(disease proneness) and ln(dominant species presence). (B) The effect of ln(disease proneness) on the square root of pathogen load, after controlling for the effects of both ln(species richness treatment) and ln(dominant species presence). (C) The effect of ln(dominant species presence) on the square root of pathogen load, after controlling for the effects of both ln(species richness treatment) and ln(disease proneness).

analyses, although doing so altered the results only slightly (not shown).

Effects of species diversity on disease severity

We further tested the diversity–disease hypothesis by performing a linear regression of disease severity on plant species richness treatment for each disease sampled in each year.

In 1998, the severity of 11 of 27 diseases increased significantly as plant species richness decreased (Table 2A). Additionally, three other diseases (the fungal leaf spots of *Andropogon*, *Coreopsis*, and *Panicum*) had a similar, although not quite significant, response ($P < 0.07$), and seven others had negative, although nonsignificant, correlations. In contrast, disease severity was significantly positively correlated with plant species richness for only one disease (the fungal leaf spot of *Elymus*), and positively, although not significantly, correlated with plant species richness in four other cases. Inspection of the single significantly positive correlation revealed that disease severity was significantly negatively correlated with species richness treatment over most of our experimental gradient (when plots planted with 24 species were excluded from analysis, $y = 5.621–0.357x$; $r^2 = 0.382$, $F_{1,10} = 6.192$, $P = 0.032$).

In 1997, severity of the rust disease on *Euphorbia* increased significantly as plant species richness decreased. Severity of the rust disease on *Panicum* was not significantly influenced by species richness treatment (Table 2B). Other diseases sampled in 1997 were reported in Knops et al. (1999).

Seven of the 11 diseases sampled in 1996 significantly increased in severity as plant species richness decreased (Table 2C). One other disease (the fungal leaf spot of *Andropogon*) showed a similar, but not quite significant, response ($F_{1,53} = 3.625$, $P = 0.062$). For no disease was severity positively correlated, even nonsignificantly, with species diversity that year. Note that only in 1998 were diseases sampled on all plant species; therefore, sample sizes differ among sections A, B, and C of Table 2.

Effects of host abundance and diversity on disease severity

To test the hypothesis that effects of plant species richness on disease severity were caused by increased host species abundances at lower species richness, we performed a multiple regression with disease severity as the response and plant species richness and percent cover as predictors for each disease sampled in all three years. Because we were interested not only in the effects of host abundance independent of species richness, but also in the effects of host abundance due ultimately to species richness treatment, we used Type I sums of squares with host percent cover entered in the model before species richness. Therefore, in the multiple regressions, tests of the effects of species richness treatment were conditional on host percent cover, but not vice versa. For heteroecious rusts (those that must sequentially infect two plant species each season, i.e., the *Euphorbia–Panicum* rust and the *Liatris–Koeleria* rust), host abundance is not easily defined. For simplicity of presentation, we present results only using percent cover of grass hosts, except when the effects of percent cover of forb hosts were significant. In all cases, the abundance of the host for which we do not

TABLE 2. Regressions for each foliar disease sampled, with disease severity as the response variable and species richness treatment (''diversity'') as the predictor.

Disease	Intercept	Diversity	r^2	N
A) 1998 Biodiversity I				
Achillea leaf necrosis	20.404***	−0.431**	0.128	57
Agropyron fungal leaf spot	N/A	N/A	N/A	2
Andropogon fungal leaf spot	6.731***	−0.070 NS	0.066	51
Anemone fungal leaf spot	16.488***	−0.527***+	0.369	52
Asceplias fungal leaf spot‡	−1.149***	−0.011 NS	0.008	57
Aster fungal leaf spot†,‡	2.350***	−0.624***+	0.510	43
Astragalus fungal leaf spot	2.132**	0.008 NS	0.001	49
Bouteloua fungal leaf spot	4.287***	−0.104***+	0.253	41
Buchloe fungal leaf spot	0.183*	−0.008 NS	0.042	14
Coreopsis bacterial leaf spot‡	1.361***	−0.041*	0.192	30
Coreopsis fungal leaf spot‡	1.106***	−0.031 NS	0.117	30
Elymus fungal leaf spot	2.530*	0.175*	0.304	14
Euphorbia§–*Panicum* rust	21.417***	0.035 NS	0.001	56
Euphorbia–*Panicum*§ rust	6.497***	−0.142**	0.227	29
Lespedeza rust (telia)	2.482***	−0.019 NS	0.016	56
Liatris§–*Koeleria* rust	3.440**	0.004 NS	0.000	43
Liatris–*Koeleria*§ rust	2.081***	−0.005 NS	0.001	48
Liatris fungal leaf spot	3.814***	−0.105**	0.164	46
Panicum fungal leaf spot‡	1.591***	−0.030 NS	0.123	29
Petalostemum fungal stem spot†,‡	1.712***	−0.503***+	0.376	43
Poa fungal leaf spot	0.411*	0.011 NS	0.043	38
Rudbeckia fungal leaf spot	8.092***	−0.059 NS	0.004	45
Schizachyrium fungal leaf spot	7.503***	−0.002 NS	0.000	50
Solidago fungal leaf spot†	6.460***	−1.843***+	0.307	52
Sorghastrum fungal leaf spot	9.184***	−0.167***+	0.247	62
Sporobolus fungal leaf spot†	3.356***	−0.921*	0.409	13
Vicia fungal leaf spot	0.034 NS	−0.001 NS	0.042	6
B) 1997 Biodiversity I				
Euphorbia§–*Panicum* rust	2.533***	−0.027*+	0.102	56
Euphorbia–*Panicum*§ rust	6.803***	−0.096 NS	0.059	29
C) 1996 Biodiversity I and II				
Andropogon fungal leaf spot	2.539***	−0.026 NS	0.064	55
Asclepias fungal leaf spot	0.697***	0.000 NS	0.000	58
Aster fungal leaf spot‡	1.179***	−0.037***+	0.346	50
Euphorbia§–*Panicum* rust†	0.024***	−0.006***+	0.185	55
Lespedeza rust (aecia)†,‡	1.186***	−0.261*	0.113	57
Lespedeza rust (telia)	8.582***	−0.019 NS	0.006	51
Lespedeza fungal leaf spot‡	1.361***	−0.027***+	0.241	51
Liatris fungal leaf spot‡	0.358 NS	−0.077***+	0.328	48
Monarda powdery mildew‡	1.910***	−0.040***+	0.150	74
Schizachyrium fungal leaf spot	5.053***	−0.054**+	0.132	57
Sorghastrum fungal leaf spot	1.179***	0.000 NS	0.000	62

Note: The fitted intercept, the regression coefficient, the coefficient of determination (r^2), and sample size (N) are shown, grouped by year. N/A, not applicable.

* $P \leq 0.05$; ** $P \leq 0.01$; *** $P \leq 0.01$; NS $P > 0.05$. A "+" indicates $P \leq 0.05$ after sequential Bonferroni adjustment by year.

† Species richness treatment was transformed by the natural logarithm.

‡ Disease severity (or disease severity + 1) was transformed by the natural logarithm.

§ Plant species on which disease severity was quantified.

report detailed results was not significantly correlated with disease severity ($P > 0.1$).

Severity was significantly positively correlated with host percent cover for 10 of the 11 diseases that increased in severity as species richness decreased in 1998 (Table 3A, Fig. 3). Severity of three other diseases also increased significantly with host percent cover; no disease significantly decreased in severity as host percent cover increased. For only four of the 27 diseases was the severity negatively correlated, even nonsignificantly, with host percent cover. For five diseases, severity was significantly negatively related to species richness treatment after controlling for the effects of host percent cover.

In 1997, severity of rust disease on *Euphorbia* increased significantly with host percent cover, whereas disease severity of rust on *Panicum* was independent of host percent cover. In both cases, plant species richness had no significant effect, after controlling for the effects of host percent cover (Table 3B).

Severity increased significantly with host percent cover for five of the seven diseases that were decreased

TABLE 3. Multiple regressions for each disease sampled with disease severity as the response variable and species richness treatment (''diversity'') and host abundance (''abundance'') as predictors.

Disease	Intercept	Abundance	Diversity	r^2	N
A) 1998 Biodiversity I					
Achillea leaf necrosis	18.912***	0.215 NS	−0.374*	0.137*	57
Agropyron fungal leaf spot	N/A	N/A	N/A	N/A	2
Andropogon fungal leaf spot	7.729***	0.026 NS	−0.103 NS	0.080 NS	51
Anemone fungal leaf spot	13.884***	0.177***	−0.444***	0.387***+	52
Asceplias fungal leaf spot‡	−1.355***	0.034 NS	−0.005 NS	0.027 NS	57
Aster fungal leaf spot†,‡	1.391***	0.073***	−0.340*	0.570***+	43
Astragalus fungal leaf spot	1.999***	0.028 NS	0.008 NS	0.002 NS	49
Bouteloua fungal leaf spot	1.528***	0.056**	−0.016 NS	0.389***+	41
Buchloe fungal leaf spot	0.180**	0.000 NS	−0.007 NS	0.042 NS	14
Coreopsis bacterial leaf spot‡	0.771***	0.155***	−0.020 NS	0.436***+	30
Coreopsis fungal leaf spot‡	0.353***	0.199***	−0.005 NS	0.529***+	30
Elymus fungal leaf spot	1.676***	0.192 NS	0.217*	0.391 NS	14
Euphorbia§–*Panicum*‖ rust	21.711***	8.732***	−0.111 NS	0.272***+	56
Euphorbia–Panicum§,‖ rust	5.681***	0.052*	−0.102 NS	0.270*	29
Lespedeza rust (telia)	2.624***	−0.019 NS	−0.022 NS	0.017 NS	56
Liatris§–*Koeleria*‖ rust	2.248***	0.303**	0.014 NS	0.173*	43
Liatris–Koeleria§,‖ rust	2.515***	−0.029 NS	−0.020 NS	0.053 NS	48
Liatris fungal leaf spot	2.700***	0.318**	−0.064 NS	0.207**	46
Panicum fungal leaf spot‡	1.399***	0.012 NS	−0.021 NS	0.151 NS	29
Petalostemum fungal stem spot†,‡	0.767***	0.121***	−0.232*	0.563***+	43
Poa fungal leaf spot	0.532***	−0.017 NS	0.008 NS	0.075 NS	38
Rudbeckia fungal leaf spot	6.539***	0.540 NS	−0.011 NS	0.018 NS	45
Schizachyrium fungal leaf spot	6.098***	0.030 NS	0.042 NS	0.041 NS	50
Solidago fungal leaf spot†	−0.804***	0.287***	0.298 NS	0.672***+	52
Sorghastrum fungal leaf spot	7.244***	0.078***	−0.099*	0.346***+	62
Sporobolus fungal leaf spot†	1.205***	0.174***	−0.058 NS	0.711**+	13
Vicia fungal leaf spot	0.036 NS	−0.037 NS	0.001 NS	0.157 NS	6
B) 1997 Biodiversity I					
Euphorbia§,‖–*Panicum* rust	−9.627***	0.363***	−0.053 NS	0.293***+	56
Euphorbia–Panicum§,‖ rust	6.187***	0.040 NS	−0.068 NS	0.071 NS	29
C) 1996 Biodiversity I and II					
Andropogon fungal leaf spot	2.374***	0.008 NS	−0.021 NS	0.071 NS	55
Asclepias fungal leaf spot	0.600***	0.009 NS	0.004 NS	0.039 NS	58
Aster fungal leaf spot‡	0.773***	0.076***	−0.020*	0.465***+	50
Euphorbia†,§–*Panicum* rust	0.778***	0.126***	−0.013 NS	0.274***+	55
Lespedeza rust (aecia)†,‡	1.417***	−0.010 NS	−0.319*	0.119*	57
Lespedeza rust (telia)	7.407***	0.107 NS	0.018 NS	0.043 NS	51
Lespedeza fungal leaf spot‡	0.873***	0.044***	−0.012 NS	0.369***+	51
Liatris fungal leaf spot‡	−0.218***	0.168***	−0.056**	0.380***+	48
Monarda powdery mildew‡	1.283***	0.292***	−0.017 NS	0.360***+	74
Schizachyrium fungal leaf spot	6.004***	−0.038 NS	−0.078**	0.157**+	57
Sorghastrum fungal leaf spot	−0.110 NS	0.006 NS	0.008 NS	0.020 NS	62

Note: The fitted intercept, partial regression coefficients, the coefficient of determination (r^2), and sample size (N) are shown, grouped by year. N/A, not applicable.

* $P \leq 0.05$; ** $P \leq 0.01$; *** $P \leq 0.001$; NS $P > 0.05$ (unadjusted). A "+" indicates $P \leq 0.05$ after sequential Bonferroni adjustment by year.

† Species richness treatment was transformed by the natural logarithm.

‡ Disease severity (or disease severity + 1) was transformed by the natural logarithm.

§ Plant species on which disease severity was quantified.

‖ Plant species abundance used as predictor variable.

in severity by species richness in 1996. For four of these seven diseases, effects of species diversity on severity were still significant after controlling for host percent cover. For nine of the 11 diseases, severity was positively correlated with host percent cover, without respect to statistical significance (Table 3C).

Disease proneness among species

To test the hypothesis that host abundance and disease proneness are positively correlated among species, we performed three simple regressions of disease proneness (the average severity of each disease in 1998 in the Biodiversity I plots planted with 24 species) against (1) host species' regional geographic extent (number of counties in which the plant species was reported across the Great Plains), (2) host species' state geographic extent (number of counties in which the plant species was reported across Minnesota), and (3) host species' local abundance (average percent cover in the Biodiversity I plots planted with 24 species). We

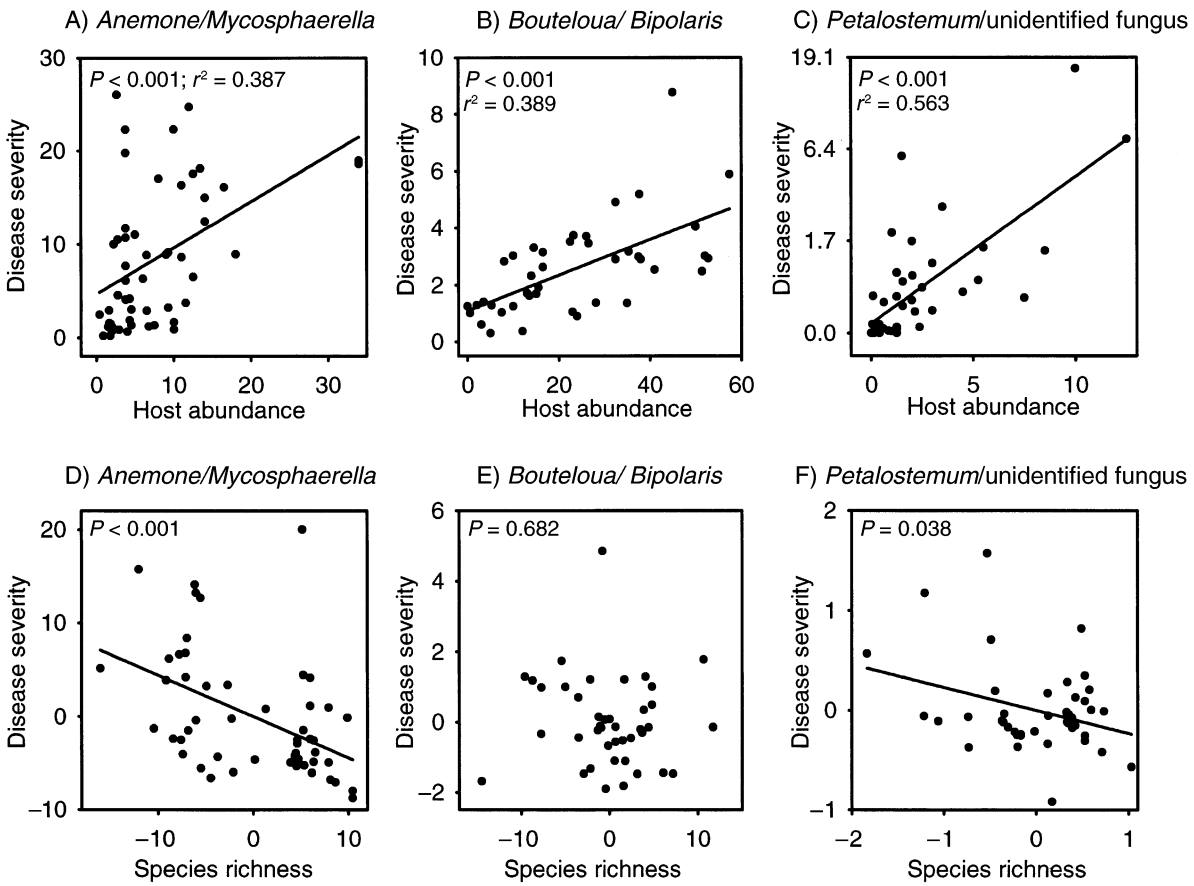

FIG. 3. The effects on disease severity of host abundance (A–C), and diversity (D–F: added-variable plots) after controlling for the effects of host abundance. Reported P values are for partial regression coefficients; reported r^2 is for each overall regression. The three species shown were the most abundant nonleguminous forb, C_4 grass, and leguminous forb, respectively, for which disease severity decreased with species richness in 1998. No C_3 grass fit this criterion. See Table 3 for a summary of similar analyses for all diseases sampled. In (A), when the two influential points (abundance >30) are excluded, $r^2 = 0.336$, host abundance $P = 0.002$, and species richness treatment $P = 0.001$. In (C), note the logarithmic y-axis scaling.

followed Arneberg et al. (1998) by taking the logarithm of all variables and excluding pathogens requiring more than one host species (the heteroecious rusts). Disease proneness was significantly positively correlated with Great Plains geographic extent (Fig. 4A). Disease proneness was also significantly positively correlated with Minnesota geographic extent (Fig. 4B). However, removal of one influential point (*Vicia*, the least abundant and least diseased plant species), caused this second relationship to be nonsignificant ($r^2 = 0.149$, $F_{1,17} = 2.978$, $P = 0.103$). Disease proneness was not significantly correlated with local abundance (Fig. 4C).

DISCUSSION

The diversity–disease hypothesis

In our experiments, lower plant species diversity increased the pathogen load (percentage of leaf area infected) experienced by the plants remaining in the community, supporting the diversity–disease hypothesis first suggested by Elton (1958). Over our range of treatments, from approximately natural levels of species diversity to monoculture, pathogen load increased from 4.3 to 11.7, a factor of 2.7 (Fig. 1A). Furthermore, the increase in average disease severity across species was not caused by just a few highly diseased species. Rather, about half of the plant species experienced significantly greater levels of disease at lower plant species richness (Table 2A). Although some diseases did not decrease in severity as plant species richness increased, there were no unambiguous cases in which the opposite occurred. Pathogen load increased logarithmically as diversity decreased. Both theoretical and empirical studies of the effects of crop genetic diversity on disease have found logarithmic decreases in disease as genetic diversity increases (Leonard 1969, Mackenzie 1979, Chin and Wolfe 1984, Mundt and Leonard 1986, Mundt 1994, Newton et al. 1997, Garrett and Mundt 1999, Zhu et al. 2000). Our study indicates that this relationship also holds for species diversity, and continues to higher levels of diversity than previously observed.

Disease dynamics are often patchy and stochastic in

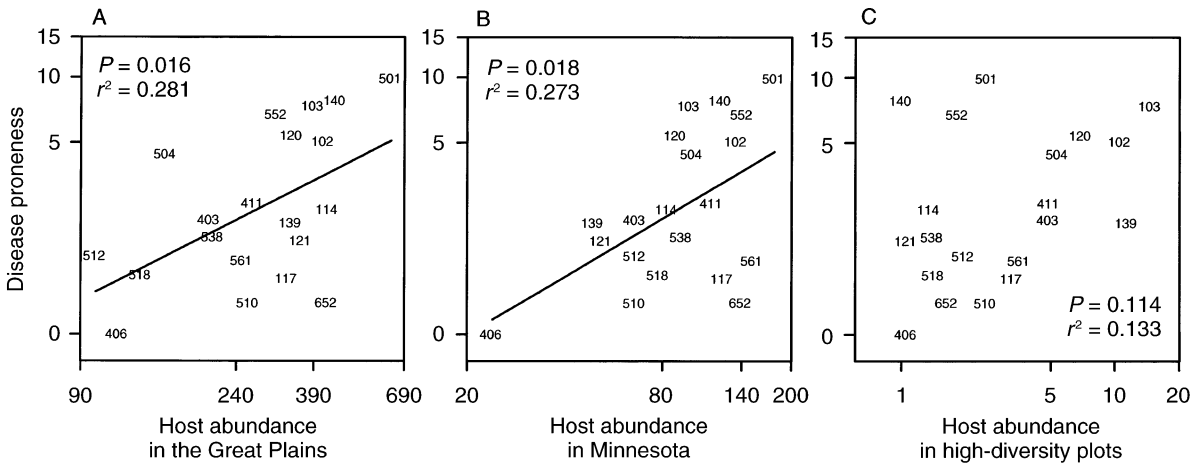

FIG. 4. The dependence among plant species of disease proneness on host abundance at three different scales: (A) the Great Plains, (B) Minnesota, and (C) local experimental plots with an approximately natural level of diversity. Disease proneness (percentage of leaf area infected) was measured in 1998 in the Biodiversity I plots planted with 24 species, as was local host abundance. Host abundances (geographic ranges) in Minnesota and in the Great Plains are the number of counties in which the plant species have been reported. See Table 1 for plant species code numbers. Note the logarithmic axis scaling.

space and time (Shaw 1994, Kleczkowski et al. 1997), but despite the variation due to these factors, the general pattern of greater disease severity under experimentally reduced species diversity was clear. Also, within our experiments, dispersal of pathogens between plots or from neighboring areas could have homogenized disease severity across treatments (''interplot interference'' in plant pathology), but this effect should weaken when losses of diversity occur at larger spatial scales (Wolfe 1985, Mundt 1994, Garrett and Mundt 1999, Zhu et al. 2000). Thus, the effect of diversity on disease may be even stronger in naturally occurring ecosystems than in our experimental ones because nonexperimental losses of diversity will probably occur at larger spatial scales than our experimental plots. In sum, our results support the idea that preventing anthropogenic losses of species diversity from ecosystems can help to maintain host-specific plant diseases at their natural levels, rather than increasing in severity (Chapin et al. 1997, Knops et al. 1999).

Host abundance and disease severity

Why did decreased plant species richness increase disease severity? Essentially, greater diversity diluted the abundance of hosts for a given disease because each disease was specific to only one or two plant species in our experimental communities. In almost every case, when disease severity was negatively related to species diversity, it was positively related to host abundance (percent cover). For the 27 diseases sampled in 1998, we found that severity of no disease was significantly negatively correlated with host abundance, whereas severity of 13 diseases was significantly positively correlated with host abundance, and severity of 10 others was positively, but not significantly, correlated with host abundance (Table 3). These results suggest that the increased abundance of host plant species at lower levels of species richness was a common and important mechanism underlying the observed relationship between species richness and disease severity in this experiment.

This dilution effect linking species diversity and disease is not directly analogous to either of the two major mechanisms linking diversity and productivity: niche complementarity and the sampling effect. The sampling effect for productivity requires that the best competitors are also the most productive species, and therefore most abundant (Aarssen 1997, Huston 1997, Tilman et al. 1997*b*, Tilman and Lehman 2002). For a sampling effect to contribute to the observed negative relationship between pathogen load and diversity would require more abundant species to be less disease prone; in contrast, there was no detectable relationship between abundance and disease proneness (Fig. 4C). Niche complementarity positively links diversity and productivity when different species utilize resources differently, e.g., temporally or spatially (Naeem et al. 1994, Tilman et al. 1996, 1997*b*, Hector et al. 1999, Tilman and Lehman 2002), and is therefore not analogous to the dilution effect observed for disease.

For five of the 27 diseases sampled in 1998, species diversity had significant negative effects on disease severity in addition to the effects of host abundance (Table 3), suggesting that mechanisms other than host abundance may also influence certain diseases. Such mechanisms may include diversity-dependent differences in microclimate or host plant nutrient status, and interference with spore dispersal by non-host plants (Trenbath 1977, Chin and Wolfe 1984, Wolfe 1985, Burdon 1987, Boudreau and Mundt 1992, 1994, 1997,

Zhu et al. 2000). However, the small number of these cases and the preponderance of strong correlations between disease severity and host abundance suggest that altered host abundance is the most important mechanism underlying effects of plant species diversity on disease severity, as is generally the case in agricultural mixtures (Burdon and Chilvers 1977, 1982, Chin and Wolfe 1984, Wolfe 1985, Alexander et al. 1986, Burdon 1987, Boudreau and Mundt 1997, Garrett and Mundt 1999, Zhu et al. 2000).

Effects of species composition on disease

In addition to the effects of species diversity, species composition, via its effects on community disease proneness and dominant species presence, also strongly influenced pathogen load (Fig. 2). In a multiple regression, these two community characteristics explained more of the total variance in pathogen load (29%) than did diversity (10%), agreeing with past studies of other ecosystem properties (Tilman et al. 1997*a*). When controlling for these effects, we found no significant effect of community evenness on pathogen load. Of the two community characteristics that we quantified, disease proneness had much stronger effects on pathogen load than did dominant species presence (respectively explaining 25% and 5% of the total variance). The negative dependence of pathogen load on dominant species presence (Fig. 2C) indicates that the loss of species dominant at approximately natural levels of diversity (i.e., a smaller index of dominant species presence) tended to increase pathogen load more than did the loss of relatively rare species. Similarly, the positive dependence of pathogen load on community disease proneness (Fig. 2B) supports the hypothesis that the loss of more disease-prone species will increase pathogen load less than if less disease-prone species are lost, on average. This effect is not surprising, requiring only that the rank of species in disease severity be relatively constant across diversity levels, but it is a real effect of differences in species composition. Therefore, community disease proneness can be an important determinant of pathogen load when species composition is altered, whether or not there is a net change in diversity.

Increased variation in species composition at low diversity may explain the increased plot-to-plot variation in pathogen load at low diversity (Fig. 1B), as has been suggested for several other ecosystem properties (McGrady-Steed et al. 1997, Naeem and Li 1997, Tilman et al. 1997*b*). The dependence of pathogen load on the community characteristics of disease proneness and dominant species presence (Fig. 2B, C) supports this hypothesis for disease, because species composition inevitably increases in variance as diversity decreases. These results suggest, for example, that a small number of low-diversity communities had extremely low pathogen loads because they consisted of species that were minimally disease prone. However, variance in pathogen load still increased at lower species diversity, even after we controlled for species composition (Fig. 2A). This suggests that factors other than species composition, at least in terms of disease proneness and dominant species presence, also contribute to this pattern. One important factor may be microclimate, which can profoundly influence the spread of foliar fungal disease (McCartney 1997).

More dominant species may be more disease prone (Arneberg et al. 1998), in which case the effects of losing dominant and disease-prone species on pathogen load would be countervailing. The lack of correlation between species disease proneness and abundance at the plot scale (Fig. 4C), does not support this hypothesis, suggesting that the effects of disease proneness and dominant species presence on pathogen load are independent. Because species abundance is often correlated with extinction risk (Tilman et al. 1994, Simberloff 1998), this also suggests that disease proneness and extinction risk should be uncorrelated. However, for the same set of species, disease proneness was significantly, but weakly, positively correlated with geographic range at the state scale (Fig. 4B), and disease proneness was strongly positively correlated with geographic range at the regional scale (the Great Plains; Fig. 4A). This scale dependence suggests that disease proneness among species may be more a function of processes occurring at larger spatial scales, perhaps such as long-term coevolutionary dynamics or greater regional inoculum loads (Thrall and Burdon 1997, 2000), than processes at a local spatial scale.

In total, our results indicate that decreased plant species diversity, when it strongly influences species abundances, increases foliar fungal pathogen load, and that species composition influences pathogen load when species differ in disease proneness. What further effects might an altered pathogen load have on an ecosystem? In a Cedar Creek, Minnesota grassland, experimentally reducing peak foliar fungal pathogen load from ~9% (comparable to an average biculture in Biodiversity I in 1998) to ~0.5% greatly increased belowground plant biomass by increasing leaf longevity and photosynthetic capacity (Mitchell 2001). Therefore, altered pathogen load may influence belowground biomass; specifically, increases in pathogen load as a result of decreased diversity may inhibit belowground production. This could result simply from increased pathogen load, but might be magnified by the lack of less diseased competitors to compensate for heavily diseased species. Therefore, reduction of disease severity by species diversity may, along with many other factors, contribute to the positive relationship observed between plant species diversity and belowground biomass (Reich et al. 2001). Finally, if the diseases that we observed decrease the abundance of their hosts by decreasing fitness, they could regulate host population size, promote coexistence, or cause oscillations in the abundances of their host species (Chilvers and Brittain

1972, Gates et al. 1986, Hudson et al. 1998), which could feed back to alter the effect of diversity on disease over time.

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