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LETTER

Diversity inhibits foliar fungal diseases in grasslands: Potential mechanisms and temperature dependence

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Abstract

A long-standing debate exists among ecologists as to how diversity regulates infectious diseases (i.e., the nature of diversity-disease relationships); a dilution effect refers to when increasing host diversity inhibits infectious diseases (i.e., negative diversity-disease relationships). However, the generality, strength, and potential mechanisms underlying negative diversity-disease relationships in natural ecosystems remain unclear. To this end, we conducted a large-scale survey of 63 grassland sites across China to explore diversity-disease relationships. We found widespread negative diversity-disease relationships that were temperature-dependent; non-random diversity loss played a fundamental role in driving these patterns. Our study provides field evidence for the generality and temperature dependence of negative diversity-disease relationships in grasslands, becoming stronger in colder regions, while also highlighting the role of non-random diversity loss as a mechanism. These findings have important implications for community ecology, disease ecology, and epidemic control.

KEYWORDS
encounter reduction, host regulation, intraspecific variation, pathogen, species turnover

INTRODUCTION

Climate change and human activities have accelerated diversity loss (Pimm et al., 2014) and exacerbated infectious diseases worldwide (Altizer et al., 2013; Laine, 2023; Rohr et al., 2011), making a thorough understanding of how diversity impacts infectious diseases critical (Keesing et al., 2010; Rohr et al., 2020). One possible avenue, known as a dilution effect, occurs when increasing host diversity inhibits infectious diseases (i.e., negative diversity-disease relationships) (Keesing et al., 2006; Keesing & Ostfeld, 2021). The generality of negative diversity-disease relationships is a key question for disease ecology (Johnson et al., 2015; Ostfeld & Keesing, 2012). If negative diversity-disease relationships are common, the goal of protecting diversity can be combined with that of controlling infectious diseases (e.g., the 'One Health' approach) (Hernando-Amado et al., 2019). Negative diversity-disease relationships have been described for many diseases (e.g., LoGiudice et al., 2003; Kilpatrick et al., 2006; Johnson et al., 2013; Lacroix et al., 2014), and meta-analyses have confirmed that dilution effects occur in agroecosystems (Wan et al., 2022), grasslands (Liu et al., 2020), and natural animal communities (Civitello et al., 2015). These findings are not universal, however, and other studies have identified neutral (Wood et al., 2017), idiosyncratic (Salkeld et al., 2013), or even positive diversity-disease relationships (Power & Mitchell, 2004; Wood & Lafferty, 2013), suggesting that negative diversity-disease relationships can be spatial-scale (Halliday & Rohr, 2019; Magnusson et al., 2020; Liu et al. 2023) and/or system dependent (Liu

et al., 2020). It is therefore important to understand the generality of negative diversity-disease relationships in natural communities and then use this information to predict when and where infectious diseases will outbreak.

Biotic mechanisms operating at local scales, including host regulation and encounter reduction (Keesing et al., 2006), as well as non-random diversity loss (Halliday et al., 2020; Johnson et al., 2013; Lacroix et al., 2014), are prerequisites for negative diversity-disease relationships (Huang et al., 2016; Keesing & Ostfeld, 2021). Infectious diseases can be suppressed by high host diversity ('host regulation'), as interspecific competition among hosts reduces the abundance of each host in substitutive host community assembly scenarios (i.e., community-level total abundance does not change with diversity) (Keesing et al., 2006), negatively impacting host-specific pathogens (Keesing et al., 2006; Rudolf & Antonovics, 2005). In diverse communities, the presence of low-competence hosts can decrease pathogen transmission, reducing the probability of effective transmission, a phenomenon known as 'encounter reduction' (Keesing et al., 2006). Both host regulation and encounter reduction rely on specific biotic interactions that occur at relatively local spatial scales. These mechanisms have been shown to shape negative diversity-disease relationships for foliar fungal pathogens in agroecosystems (Zhu et al., 2000), alpine meadows (Liu et al., 2016), European grasslands (Rottstock et al., 2014), and old fields (Mitchell et al., 2002); these pathogens have relatively narrow host ranges and tend to infect plants from the same species, genus, or family (Gilbert & Webb, 2007).

Non-random diversity loss has also been recently suggested as a prerequisite for negative diversity-disease relationships (Halliday et al., 2020; Huang et al., 2016; Johnson et al., 2013). Diversity loss generates predictable shifts in community competence, generally fostering a higher proportion of susceptible individuals as resistant individuals are more likely to be lost due to growth-defence trade-offs (Liu et al., 2017), although it has also been argued that susceptible individuals are lost at higher rates (e.g., Alexander, 2010; Power & Mitchell, 2004). This process emphasizes the role of low-competence host species loss in shaping negative diversity-disease relationships (Johnson et al., 2019), while other processes (e.g., abundance shifts in low-competence species in the community) can also contribute to negative diversity-disease relationships by altering host community competence (Liu et al., 2017). Meanwhile, both host regulation and encounter reduction rely on specific biotic interactions (Keesing & Ostfeld, 2021). According to the framework described by Lepš et al., 2011, the role of non-random diversity loss can be partly distinguished from that of specific biotic interactions (i.e., host regulation and encounter reduction) in shaping negative diversity-disease relationships. Specifically, variation in pathogen load (as weighted disease severity) along a diversity gradient can be caused by species turnover (i.e., changes in community composition) and intraspecific variation (i.e., variation among component species in disease susceptibility). Beyond species turnover effects, community nestedness can also be used to characterize non-random diversity loss (Liu et al., 2018). A high degree of nestedness indicates that lower diversity communities are subsets of higher diversity communities (Naeem et al., 1994). While species turnover captures the contributions of both host species loss/gain and shifts in component species abundance (Lepš et al., 2011; Liu et al., 2017), community nestedness emphasizes the role of host species loss/gain (Wolf & Zavaleta, 2015). At the same time, specific biotic interactions are partly reflected by intraspecific variation. However, little is known about their relative importance in natural communities.

Climatic factors, including temperature and precipitation, may regulate the direction and strength of diversity-disease relationships (Altizer et al., 2013; Laine, 2023), patterns of non-random diversity loss (Halliday et al., 2021; Mori et al., 2015), and interactions among hosts and pathogens (Roy et al., 2004; Van Dyke et al., 2022). However, how diversity-disease relationships (and their underlying mechanisms) shift along temperature and/or precipitation gradients remains unknown. To this end, we comprehensively evaluated how plant species richness affects foliar fungal diseases in natural grasslands across China. Our survey sites spanned 3300 km, a mean annual temperature ranged from −5.1 to 8.5°C, and a mean annual precipitation ranged from 77 to 595 mm. Our goal was to evaluate the direction and strength of relationships between diversity (measured as species richness) and foliar fungal diseases, and to test whether temperature and precipitation moderate these relationships. In terms of mechanisms, we evaluated the relative importance of non-random diversity loss (as partly reflected by 'species turnover' in the Lepš et al., 2011 framework) and specific biotic interactions (i.e., host regulation and encounter reduction; referred to as 'intraspecific variation' in the Lepš et al., 2011 framework) in shaping negative diversity-disease relationships along a climatic gradient. To further explore the climate-dependence of negative diversity-disease relationships, we characterized variation in community nestedness and pathogen load along the climatic gradient.

MATERIALS AND METHODS

Study area

In July 2023, we conducted a large-scale field survey of 63 grassland sites across China to explore the generality of grassland diversity-disease relationships (28.17° N to 44.88° N and 88.54° E to 114.96° E; Figure 1); the mean distance between adjacent grassland sites was 83.22 ± 30.96 km, with a range of 40.98 to 215.43 km. The mean annual temperature (MAT) and precipitation

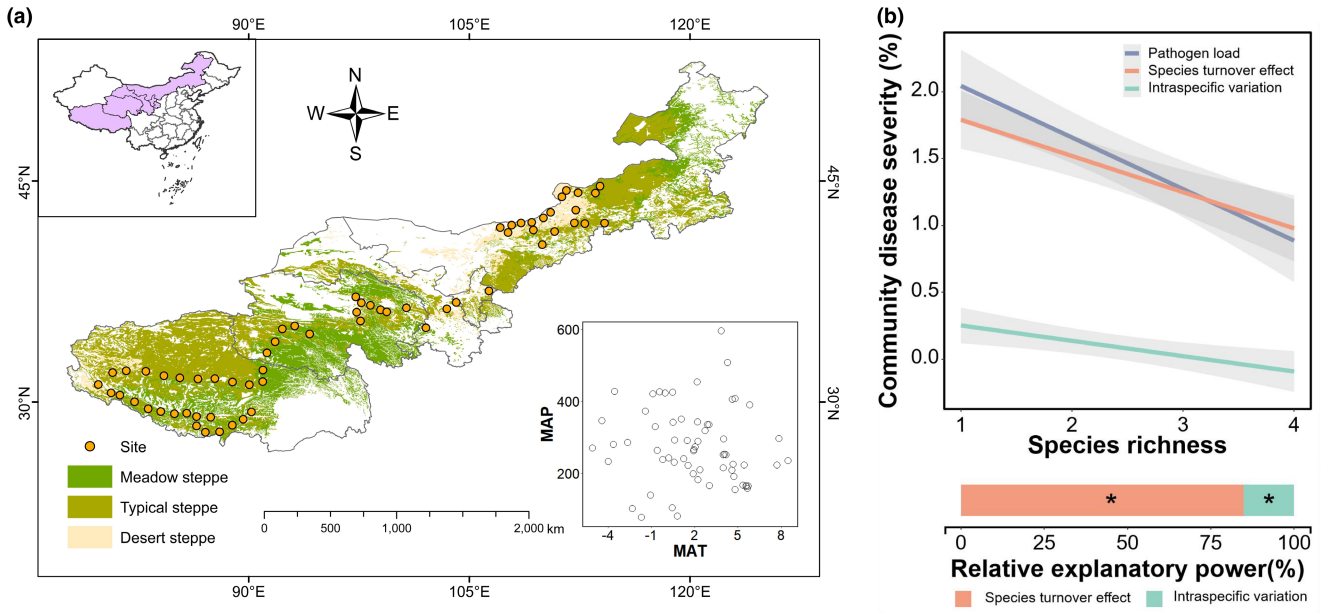

FIGURE 1 Overview of the dataset and key results. (a) Map showing the geographical distribution of survey sites across natural grasslands in China; inset shows the mean annual temperature (MAT) and precipitation (MAP) for each site. There was no correlation between MAT and MAP. (b) Effect of species richness on community disease severity and variability in pathogen load, species turnover, and intraspecific variation effects explained by species richness. Shading indicates 95% confidence intervals.

(MAP) across the survey sites ranged from −5.1 to 8.5°C and from 77 to 595 mm, respectively (Supplementary Data S1). Vegetation types represented across the survey sites included meadow steppe, typical steppe, and desert steppe. Survey sites were at least 500 m away from major roads and received little human interference.

Field survey

At each site, we surveyed the local vegetation which showed natural variation in species richness. Using a ring with a diameter of 0.3 m (negative diversity-disease relationships are more likely to occur at small spatial scales; Liu et al. 2023), we delineated 18 survey plots per site, allowing us to quantify variation in diversity-disease relationships across natural ecosystems. The rings were randomly placed multiple times in natural grasslands to select plots until there were one, two, or four species in the rings. Plots represented three species richness (SR) levels with six replicates per richness level; adjacent plots were at least 5 m apart. A total of 1134 plots (63 sites × 18 plots; Supplementary Data S2) were surveyed. In each plot, we recorded foliar fungal disease severity for each plant species and collected the aboveground plant biomass. We focused on foliar fungal diseases, including leaf blight, leaf spot, powdery mildew, and rust, as fungal diseases are the most common grassland diseases; in this system, more than one pathogen species may co-infect a single host. For all sites, we also recorded geographic data (i.e., altitude, latitude, and longitude) and extracted climatic data (i.e., MAT and MAP) from the WorldClim database (http://www.worldclim.org; Table S1). As grassland fungal disease pressure can result from multi-year cumulative effects, we averaged the climatic data from 1970 to 2000 before exploring the potential effects of climatic variables. Previous studies have shown that temperature and precipitation are important factors affecting fungal diseases (Liu et al., 2016, 2019); these two variables are also highly collinear with other climatic variables, and we therefore included only MAT and MAP as climatic variables in this study.

Community pathogen load

We calculated pathogen load and divided it into two components: species turnover and intraspecific variation (Lepš et al., 2011). For each plant species in each plot, we recorded species-level disease severity using randomly collected leaf samples (25 leaves from at least five individuals). For those species with less than 25 leaves, we examined all available leaves. We quantified disease severity for each leaf as the percent of the total leaf area covered by fungal lesions, then calculated the species-level disease severity (*vi*) by averaging these percentages across all 25 leaves. Next, we calculated the pathogen load (*PL*), which has been widely used as an indicator of community fungal disease severity in disease ecology (Liu et al., 2016; Mitchell et al., 2002). Community pathogen load was calculated at the plot-level and was defined as (Mitchell et al., 2002):

$$PL_{j,k} = \frac{\sum_{i=1}^{S} a_{ijk} v_{ijk}}{\sum_{i=1}^{S} a_{ijk}}$$

where $PL_{j,k}$ is community disease severity in plot j of site k, S the total number of plant species in plot j of site k, a_{ijk} the biomass of host plant species i in plot j of site k, and v_{ijk} the disease severity of species i in plot j of site k.

Species turnover and intraspecific variation effects in plot j of site k were calculated as (Johnson et al., 2013; Lepš et al., 2011; Liu et al., 2019):

$$\text{Species turnover effect}_{j,k} = \frac{\sum_{i=1}^{S} a_{ijk} p_{ik}}{\sum_{i=1}^{S} a_{ijk}}$$

$$\text{Intraspecific variation}_{j,k} = PL_{j,k} - \text{Species turnover effect}_{j,k}$$

where S is the total number of host plant species in plot j of site k, a_{ijk} the biomass of plant species i in plot j of site k, and p_{ik} the mean disease severity for species i in all 18 plots of site k. Pathogen load represents the actual community disease severity in a given plot. Species turnover effects represent the expected pathogen load based on host plant species composition, which was measured independently of the actual disease severity in a given plot (Liu et al., 2017); these effects can be partly linked to non-random diversity loss. Intraspecific variation effects are calculated as the difference between pathogen load and the species turnover effect, and represent differences in disease severity among component species; these effects result from specific biotic interactions.

For each site, we also calculated the matrix temperature (T; Rodríguez-Gironés & Santamaría, 2006), with a low T value indicating a high degree of nestedness. We repeated the calculation of T 500 times for each site and took the average value as the mean T (Wolf & Zavaleta, 2015; Table S2). Nestedness focuses on species identity effects and is calculated based on the presence or absence (0/1) of individual species, while species turnover considers variation in species composition and is calculated based on patterns of species abundance. Together, nestedness and species turnover more completely characterize the role of non-random diversity loss in negative diversity-disease relationships.

Statistical analysis

To evaluate the shape of diversity-disease relationships, we used linear mixed-effects models (LMMs) testing how species richness affected community disease severity (with pathogen load, species turnover, and intraspecific variation as response variables) across all sites. In order to address non-normality in the model residuals, we used a Fisher Z-transformation for the three response variables; this approach also accounted for the fact that community disease scores are ratios (e.g., intraspecific variation varies from −1 to 1). For the LMMs, we used the *lmer* function in the *lme4* package (Bates et al., 2015) in R, with site treated as a random effect.

To assess how species turnover and intraspecific variation effects shaped pathogen load across sites, we calculated their relative contributions to negative diversity-disease relationships using a decomposition method for community-level trait variances (Kichenin et al., 2013; Lepš et al., 2011). This method is based on the decomposition of the total sum of squares (SS_{total}) of the pathogen load related to an independent variable (here species richness) into 'species turnover' (SS_{fixed}), 'intraspecific' ($SS_{\text{intraspecific}}$), and 'covariation' ($SS_{\text{cov}}$) effects, so that $SS_{\text{total}} = SS_{\text{fixed}} + SS_{\text{intraspecific}} + SS_{\text{cov}}$. Briefly, for all sites, we ran three LMMs, one for each of 'pathogen load', 'species turnover', and 'intraspecific' effects, with species richness as an explanatory variable; next, we extracted the sum of squares for each of the three community disease severity measures (SS_{total}, SS_{fixed}, and $SS_{\text{intraspecific}}$) as explained by species richness. We took the ratio of SS_{fixed} to SS_{total} as the proportion of variation in the richness-disease severity relationship explained by species turnover effects; similarly, the ratio of $SS_{\text{intraspecific}}$ to SS_{total} was taken as the proportion of variation explained by intraspecific variation. Finally, we calculated the relative explanatory power of species turnover and intraspecific variation effects (i.e., SS_{fixed} / $SS_{\text{intraspecific}}$).

For each site, we conducted a series of general linear models (LMs) to test how species richness affected community disease severity (as pathogen load, species turnover, and intraspecific variation effects), extracting the slope from each model to quantify the strength of the relationship. Next, we calculated the log response ratio (LRR) for species turnover and intraspecific variation effects (SS_{fixed} / $SS_{\text{intraspecific}}$) at each site, using the decomposition method described above. To test if diversity-disease relationships were dependent on climatic factors, we again used LMs (for MAT and MAP) and calculated the LRRs. To determine if grassland type affected the model slope, we used an ANOVA and Tukey's HSD tests for multiple comparisons ($p < 0.05$) to evaluate pair-wise differences; these were implemented using the *glht* function in the R package *multcomp*. To distinguish whether diversity-disease relationships were driven by species nesting versus the environmental dependence of disease severity, we used LMs and LMMs to quantify how climatic factors affected T and pathogen load, respectively. We also developed LMs to test how T affected the slope of the diversity-disease relationship.

To further explore diversity-disease relationships and the factors underlying these relationships, we utilized structural equation modelling (SEM) as implemented in the *piecewiseSEM* package (Shipley, 2000) in R. As significant interactions occurred between species richness and MAT/MAP, we added circular arrows from MAT/MAP to the paths representing species richness effects on species turnover and intraspecific variation (Figure S1). We calculated the standardized path coefficients (scaled by their mean and standard deviation), standard errors,

and corresponding significance (*p*-values) for each path in the piecewise SEMs. To evaluate the overall fit of the piecewise SEMs, we calculated model AIC and *P*-values. In addition, we created partial regression plots (accounting for random effects) using the *partialResid* function in the *piecewiseSEM* package in R; these were used to evaluate the relationship between the residuals of species turnover and intraspecific variation effects, the interaction between species richness and MAT, and the interaction between species richness and MAP. All analyses were performed in R (version 4.2.3).

RESULTS

We collected data on the local climate, foliar fungal disease severity, and plant species richness for a total of 1134 plots nested in 63 sites across China's grasslands (Supplementary Data S1; Table S1). There was no correlation (correlation coefficient $r=-0.088$, $p=0.494$) between MAT and MAP across the study sites. Among the survey sites, we identified 146 plant species from 32 families, including the Amaryllidaceae, Asteraceae, Cyperaceae, Fabaceae, Poaceae, and Rosaceae. For all plant species, the overall foliar fungal disease severity ranged from 0.67% to 29.33% across all study plots, and observed diseases included leaf blight (0.67%–21.15%), leaf spot (0.67%–23.08%), and rust (1.19%–29.33%). Using a series of LMMs, we found that species richness significantly reduced community disease severity when measured as pathogen load ($F_{1,1068}=46.76$, $p<0.001$), as well as species turnover ($F_{1,1068}=53.51$, $p<0.001$) and intraspecific variation effects ($F_{1,1068}=9.41$, $p=0.002$; Figure 1b, Table S3). Using a variance decomposition approach, species turnover effects (84.91%) were found to have greater explanatory power than intraspecific variation effects (15.09%). These results suggest that negative diversity-disease relationships are common in natural grasslands, with both non-random diversity loss and specific biotic interactions (i.e., host regulation and encounter reduction) underlying these relationships.

To explore the environmental dependence of negative diversity-disease relationships, we performed a series of general linear models (LMs) to extract slope and variance estimates (Table S4) for species richness effects on pathogen load, species turnover, and intraspecific variation across all 63 study sites. Next, we used LMs to test how environmental factors (climate) affected these slopes. MAT significantly increased the slope of the relationship between species richness and pathogen load ($F_{1,61}=9.44$, $p=0.003$), species turnover ($F_{1,61}=5.24$, $p=0.026$), and intraspecific variation ($F_{1,61}=10.77$, $p=0.002$); MAP marginally decreased the slope of the relationship between species richness and species turnover ($F_{1,61}=3.57$, $p=0.046$), but not that between species richness and pathogen load ($F_{1,61}=2.13$, $p=0.149$) or intraspecific variation ($F_{1,61}=0.43$, $p=0.516$; Figure 2a, b, Table S5). In addition, grassland type affected the slope of the relationship between species richness and pathogen load ($F_{2,60}=10.61$, $p<0.001$) (Table S6); specifically, negative diversity-disease relationships were more common in meadow steppe (Figure S2). Moreover, MAT ($F_{1,49}=5.24$, $p=0.026$), but not MAP ($F_{1,49}=0.55$, $p=0.464$), increased the log response ratio (LRR) for species turnover and intraspecific variation effects (SS_{fixed} / $SS_{\text{intraspecific}}$) (Figure 2c, d, Table S5). Thus, the observed negative diversity-disease relationships were temperature-dependent and more likely to occur in cooler areas; species turnover played a greater role than intraspecific variation in driving negative diversity-disease relationships as temperature decreased.

We conducted a series of LMs and LMMs to test how climatic factors affected plant community nestedness and pathogen load. The MAT had a negative effect on community nestedness (a low *T* value indicates a high nestedness; $F_{1,61}=4.70$, $p=0.034$) and a negative effect on pathogen load ($F_{1,1069}=20.22$, $p<0.001$), while MAP only had a positive effect on pathogen load ($F_{1,1069}=5.66$, $p=0.021$; Figure 3, Table S7). We also found that *T* marginally increased the slope of the diversity-disease relationship ($F_{1,187}=3.73$, $p=0.055$; Figure S3). These results suggest that the temperature dependence of negative diversity-disease relationships was primarily driven by the negative effects of temperature on pathogen load.

Finally, we utilized structural equation modelling (SEM) to further explore the temperature dependence of negative diversity-disease relationships. We found that MAT directly decreased species turnover (standardized path coefficient $\beta=-0.352$, $p<0.001$) and intraspecific variation effects ($\beta=-0.091$, $p=0.003$), while the interaction of MAT and species richness positively affected species turnover ($\beta=0.068$, $p<0.001$) and intraspecific variation effects ($\beta=0.135$, $p<0.001$). In contrast, MAP directly increased species turnover effects ($\beta=0.228$, $p=0.011$), but not intraspecific variation effects ($\beta=-0.022$, $p=0.453$), and the interaction between MAP and species richness negatively affected species turnover effects ($\beta=-0.055$, $p=0.002$), but not intraspecific variation effects ($\beta=-0.018$, $p=0.551$; Figure 4a, Table S8). These interactions suggest that the negative effects of species richness on species turnover and intraspecific variation effects increased with decreasing temperature, while the negative effects of species richness on species turnover effects increased with precipitation. In addition, partial regression plots also revealed that the interaction between MAT and species richness positively affected species turnover ($F_{1,1130}=15.11$, $p<0.001$, $R^2=0.012$) and intraspecific variation effects ($F_{1,1130}=9.83$, $p=0.002$, $R^2=0.008$); the interaction of MAP and species richness negatively affected species turnover effects ($F_{1,1130}=21.19$, $p<0.001$, $R^2=0.018$), but not intraspecific variation effects ($F_{1,1130}=0.36$, $p=0.550$, $R^2<0.001$; Figure 4b, Table S9). Specifically, species richness had a negative effect on species turnover and

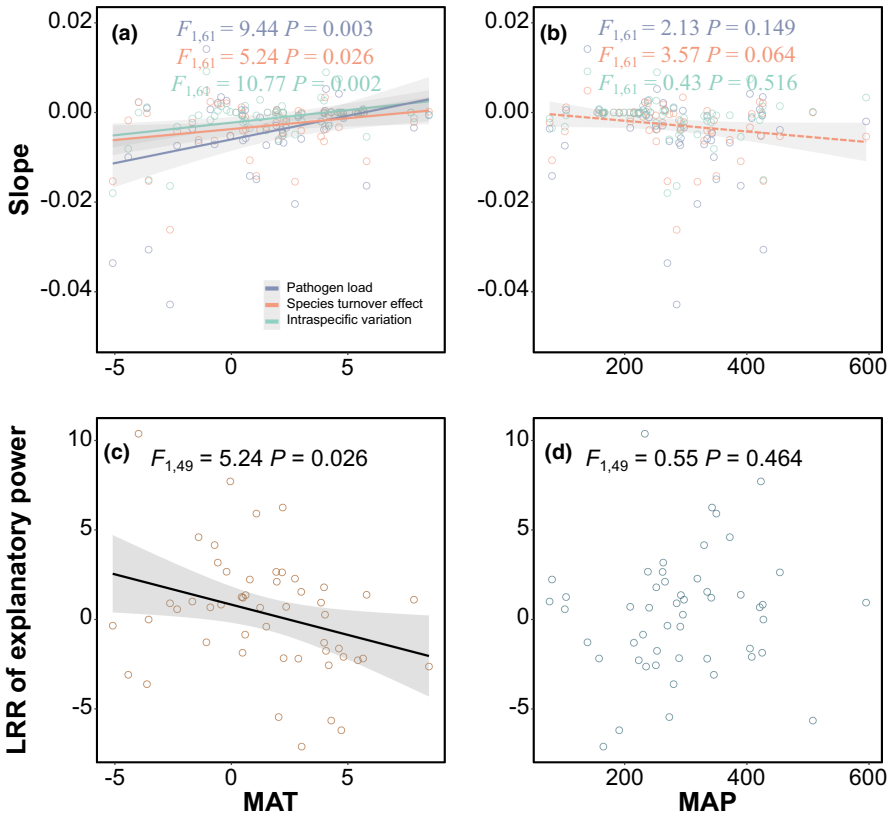

FIGURE 2 Temperature- and precipitation dependence of species richness effects and variability in pathogen load, species turnover, and intraspecific variation effects explained by species richness. Effects of mean annual temperature (MAT) (a) and precipitation (MAP) (b) on the slope of diversity-disease relationships (including estimates of species richness effects on pathogen load, species turnover, and intraspecific variation effects); each point represents one site. Effects of MAT (c) and MAP (d) on the log response ratio (LRR) for species richness (i.e., explanatory power of species richness for species turnover and intraspecific variation effects); each point represents one site. The solid and dotted lines represent significant and marginal effects, respectively. Shading indicates 95% confidence intervals.

intraspecific variation effects when MAT was low, but this negative effect gradually weakened as MAT increased (Figure 4b), indicating that negative diversity-disease relationships were stronger in colder regions.

DISCUSSION

Our study demonstrated the generality of negative diversity-disease relationships across China's grasslands using fine-scale surveys. Previous studies based on diversity-ecosystem function (BEF) experiments (Liu et al., 2016; Rottstock et al., 2014) and meta-analyses (Liu et al., 2020; Wan et al., 2022) have revealed the generality of negative diversity-disease relationships in plant communities. Interestingly, a previous meta-analysis reported that studies using a manipulative design (e.g., diversity-ecosystem function experiments, such as Mitchell et al., 2002; Rottstock et al., 2014; Liu et al., 2016) showed negative diversity-disease relationships, but observational studies did not; this is likely because potentially confounding factors, such as soil nutrients, may mask this relationship in observational studies (Liu et al., 2020). In contrast, our field survey demonstrated an overall negative diversity-disease relationship, likely because our sample plot size (0.3 m diameter) was smaller than that of previous observational studies, and dilution effects are stronger at smaller scales where biotic interactions operate (Magnusson et al., 2020; Liu et al. 2023). Together, these findings supporting negative diversity-disease relationships in grasslands provide solid evidence for the critical role of diversity in disease inhibition, thus protecting ecosystem and human health (Keesing & Ostfeld, 2021).

In this study, we examined the relative strength of specific biotic interactions (i.e., host regulation and encounter reduction) (Keesing et al., 2006; Liu et al. 2023) and non-random diversity loss (Halliday et al., 2020; Johnson et al., 2013) operating across grassland types (i.e., meadow steppe, typical steppe, and desert steppe) for the first time, finding that non-random diversity loss played the most important role. Previous studies have tended to underestimate the strength of such negative effects due to spatial-scale dependence (Mitchell et al., 2002; Liu et al. 2023) and by ignoring non-random host species losses (Halliday et al., 2020). Host life history strategies underlie non-random diversity loss, because of potential growth-defence trade-offs (Cappelli et al.,

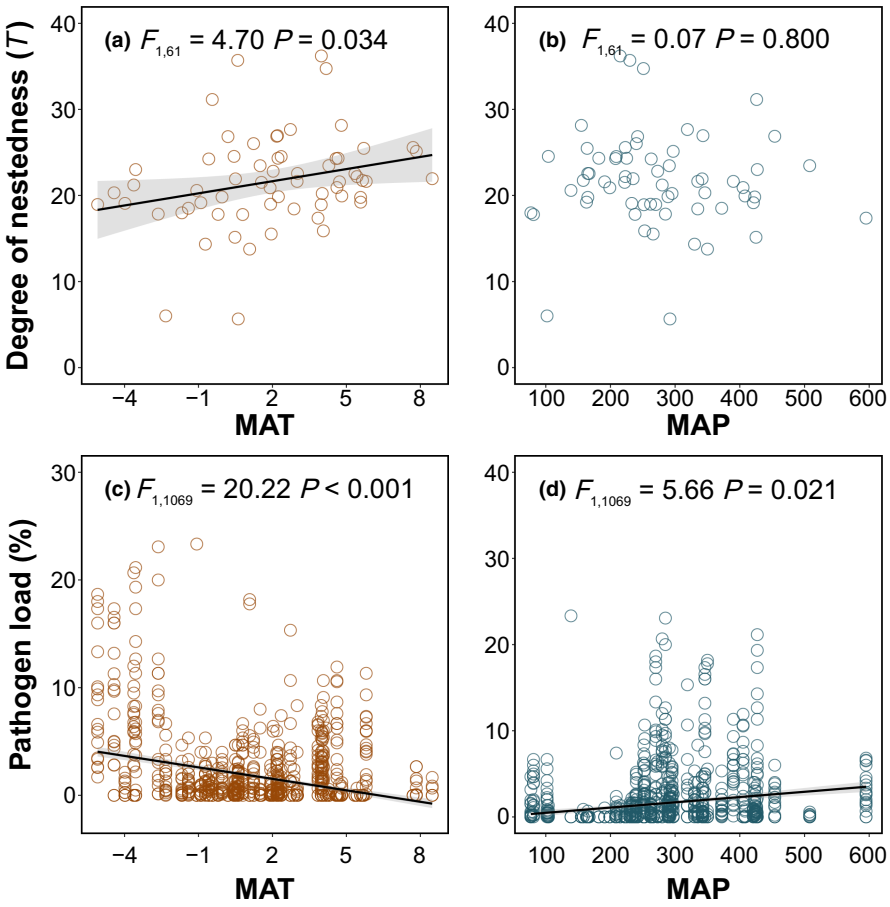

FIGURE 3 Effects of temperature and precipitation on community nestedness and pathogen load. Effects of mean annual temperature (MAT) (a) and precipitation (MAP) (b) on community nestedness (a low *T*-value represents a high degree of nestedness); each point represents one site. Effects of MAT (c) and MAP (d) on pathogen load; each point represents one plot. Solid lines represent significant effects. Shading indicates 95% confidence intervals.

2020). Host plant species that favour growth over building physical and chemical pathogen defence systems (i.e., high-competence species) often show greater resistance to anthropogenic disturbances (e.g., habitat fragmentation, nitrogen deposition, or urbanization; Lacroix et al., 2014; Liu et al., 2017). As plant species diversity is lost, high-competence species thus come to dominate in species-poor communities, leading to a negative relationship between diversity and community competence, and therefore a negative diversity-disease relationship (Halliday et al., 2020; Keesing & Ostfeld, 2021). This pattern has been confirmed for amphibian trematode parasites (Johnson et al., 2013, 2019), barley yellow dwarf virus (Lacroix et al., 2014), and Lyme disease (LoGiudice et al., 2003), as well as described by theoretical models (Joseph et al., 2013) and meta-analyses (Halliday et al., 2020). This further highlights the importance of protecting vulnerable species, as they are often low-competence species and therefore play a disproportionate role in maintaining ecosystem health under the One Health framework (Civitello et al., 2015).

Unlike previous studies that have focused on a single pathogen (e.g., Kilpatrick et al., 2006; LoGiudice et al., 2003), in this study, pathogen load is representative of the overall disease burden, rather than single pathogen-related disease risk (Wang et al., 2019), as more than one pathogen species may co-infect a single host in our system. Differences in how pathogen load is calculated among studies can obscure the relationship between plant diversity and foliar fungal diseases. For example, several studies of foliar fungal pathogens have supported the "diversity begets diversity" hypothesis (e.g., Liu et al., 2016; Rottstock et al., 2014), which states that pathogen diversity largely depends on host diversity, through both evolutionary covariation and/or host availability effects (Kamiya et al., 2014). This pattern may exacerbate pathogen spillover among hosts, especially in diverse communities, which may weaken negative diversity-disease relationships (Liu et al., 2016). However, plant species may utilize the same set of conserved defence traits (e.g., peroxidases and tannins) for a variety of pathogens (Gilbert & Parker, 2016). Hence, the coexistence of multiple pathogens can induce general pathogen resistance (Hanssen et al., 2010), especially for obligate biotrophs (e.g., rusts), which underlie a considerable proportion of diseases in our system. This

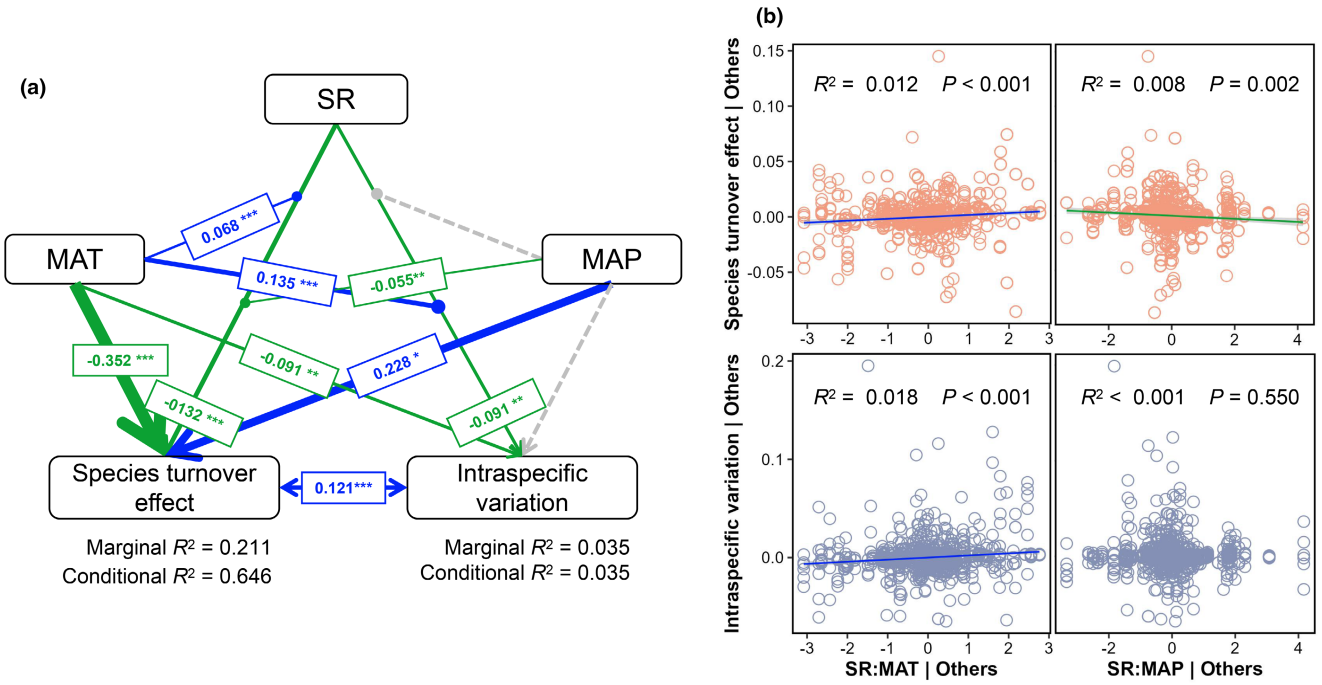

FIGURE 4 Structural equation model and partial regression plots depicting the temperature dependence of negative diversity-disease relationships. (a) Structural equation model describing the effects of mean annual temperature (MAT), precipitation (MAP), species richness (SR), and their interactions (i.e., SR:MAT and SR:MAP) on species turnover and intraspecific variation effects. Solid and dashed lines indicate significant and insignificant effects, respectively. Blue and green lines represent positive and negative effects, respectively. Double-headed arrows indicate correlations. The circular arrows that point from MAT and MAP to the paths by which species richness affects species turnover and intraspecific variation effects represent these interactions. (b) Partial regression plots describing the relationships among the residuals for species turnover and intraspecific variation effects, the interaction of species richness and MAT, and the interaction of species richness and MAP. When MAT was low, the relationship between species richness and species turnover/intraspecific variation was negative, while this negative effect gradually weakened as MAT increased. Marginal R^2 and p-values are shown.

phenomenon is also known as cross-protection in plant pathology (Hanssen et al., 2010) and can contribute to negative diversity-disease relationships.

The strength of the negative diversity-disease relationships observed here was temperature-dependent, a pattern which is usually relatively weak in warm geographical regions. One reason for this discrepancy may be that there was little evidence of foliar fungal diseases in our warmer survey sites (Figure 3c), dampening dilution effects on fungal diseases. The negative effect of temperature on disease severity is inconsistent with the conclusions from several other case studies (Liu et al., 2019; Roy et al., 2004) and data syntheses (Větrovský et al., 2019). In these, the prevalence of plant diseases depended on the factors outlined in the disease triangle (Agrios, 2005; Termorshuizen, 2017): host plants, pathogens, and environmental suitability. Temperature may promote the growth and reproduction of some pathogens (Ayres, 1993; Harvell et al., 2002), but this effect can be offset or even reversed by simultaneous increases in host resistance (Garrett et al., 2006), tolerance (Paseka et al., 2020), immunity (Cavieres et al., 2014), and/or defence (Hu et al., 2022). For example, warming stimulates the expression of plant resistance genes (Yang et al., 2023) and hormone signalling transduction (Samaradivakara et al., 2022) in legumes, thereby reducing disease severity (Yan et al., 2023). Moreover, high temperatures are unfavourable for the growth and reproduction of some pathogens, including rusts (Agrios, 2005), which are among the most common pathogens in China's grasslands. Rust urediniospores are not resistant to high temperature spikes in the growing season (Zhao et al., 2016), and high temperatures may therefore inhibit the spread of rust diseases. In summary, complex plant-pathogen interactions and interspecific variation in host plant and pathogen responses to higher temperatures together led to the observed negative effects of temperature on fungal disease severity.

Another potential explanation for the observed temperature-dependence of grassland negative diversity-disease relationships relies on the fact that temperature decreases community nestedness (Figure 3a). Community nestedness may be driven by variation in colonization and extinction along environmental or biological gradients (Ulrich et al., 2009). Dispersal limitation results in fewer species being able to spread to cooler regions than to warmer ones (Fang & Yoda, 1991; HilleRisLambers et al., 2012), thus exacerbating the non-random colonization of cooler regions (McAbendroth et al., 2005). Environmental filtering also plays a larger role in cooler areas (Hawkins, 2001), where there may be more non-random extinctions

(Wethered & Lawes, 2005), resulting in few or even a single dominant species. Conversely, in warmer regions, biotic interactions are more important (Haeuser et al., 2017; Speißer et al., 2022) and dominant species may coexist (Liu et al., 2016), leading to the possibility of greater species co-occurrence in our study plots. Finally, large historical climatic fluctuations (e.g., Quaternary climate change) may also affect contemporary community nestedness (Arroyo et al., 1982; Dalsgaard et al., 2013). Here, most cooler survey sites were located on the Qinghai-Tibet Plateau, where quaternary temperature changes were minimal and contemporary precipitation low, resulting in high community nestedness (Arroyo et al., 1982; Dalsgaard et al., 2013).

As a caveat to our findings, differences in phenology among study sites with varied climates may potentially bias our results. As with other studies involving extensive spatial scales (e.g., Wang et al., 2014; Liu et al. 2023), our study represents a single snapshot in time for foliar fungal diseases. The following three issues may have led us to over- or underestimate the strength of negative diversity-disease relationships and/or potentially increased model residuals. First, plants in cold regions usually experience short growing seasons; due to long-term coevolution, the generation time for obligate biotrophs may also be brief (Gilbert & Parker, 2016). This may have led to overestimation of the disease load for certain pathogens, as at the time of our survey, these pathogens may have been towards the end of their first generation or have already begun secondary infections; this is especially true for rusts, which were responsible for a large proportion of the surveyed diseases (Duplessis et al., 2021). At the same time, we may have underestimated diseases caused by pathogens that grow slowly in cold regions. Second, as it is difficult for plants to heal once they are infected, a single survey may reflect the cumulative disease impact on a given host over the preceding year (Grulke, 2011). The cumulative disease progress can be captured by using the area under the disease progress curve (AUDPC) approach (Shaner & Finney, 1977). However, the AUDPC approach is time- and labour-intensive, as it integrates disease severity over time through repeat sampling. Third, to overcome the potential impact of phenology, we started the survey in Lanzhou (central among our sites), proceeded south to Tibet, then north to Inner Mongolia (most northeastern sites), and finally back to Lanzhou. We did not expect phenology to bias our main results, but the survey's one-month time span may have increased the variation recorded in disease severity among sample sites (Faticov et al., 2020), leading to an increase in model residuals that cannot be explained by our independent variables.

Our comprehensive field survey across China's grasslands confirmed the general prevalence and temperature-dependence of negative diversity-disease relationships, while also highlighting the role of non-random diversity loss in driving these patterns. Building on previous studies, we explored the relative importance of specific biotic interactions versus community composition as underlying mechanisms for the first time. In summary, our study provides new insights into the temperature dependence of negative diversity-disease relationships and their underlying mechanisms. By emphasizing the need for endangered species conservation even at fine spatial scales, our study offers both a theoretical basis and empirical evidence for diversity conservation and disease control. In short, more attention should be paid to biodiversity conservation in colder regions, as they play a disproportionate role in maintaining ecosystem health.

AUTHOR CONTRIBUTIONS

X. Liu conceived this study. P. Zhang and H. Jiang collected the data. P. Zhang performed the analyses. The manuscript was drafted by P. Zhang and X. Liu. All co-authors provided input on subsequent drafts.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

PEER REVIEW

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DATA AVAILABILITY STATEMENT

All data and R code are available in Zenodo: https://doi.org/10.5281/zenodo.11044795 and GitHub: https://github.com/zhangpeng17/Biodiversity-Inhibits-Diseases-in-Grasslands.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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