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ARTICLE

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Coexistence is stabilized by conspecific negative density dependence via fungal pathogens more than oomycete pathogens

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Abstract

Plant pathogens are often hypothesized to promote species coexistence by generating conspecific negative density dependence (CNDD). However, the relative importance of fungal versus oomycete pathogens in maintaining plant species coexistence and community composition remains unresolved, despite their recognized effects on plant performance. Here, we use fungicide application to investigate how fungal versus oomycete pathogens affect plant species coexistence in an alpine meadow. We found that the severity of foliar fungal disease was density-dependent at both intra- and interspecific levels. Fungal pathogen-exclusion treatment successfully decreased the severity of foliar fungal diseases, with no detectable effects on root colonization by arbuscular mycorrhizal fungi or on soil chemical properties. Fungal pathogens were important factors shaping CNDD across 25 coexisting plant species. Exclusion of fungal pathogens significantly reduced plant species richness and Shannon's evenness. Treatments that excluded fungal pathogens also led to significant shifts in plant community composition toward more Poaceae and Cyperaceae. These results indicate that fungal pathogens, especially those affecting aboveground plant parts, may play a larger role in maintaining species coexistence and shaping community composition than has been previously recognized.

KEYWORDS
alpine meadow, negative density dependence, pathogen, species coexistence, stabilizing mechanism, Tibetan Plateau

INTRODUCTION

Understanding species coexistence and what regulates abundance is a central focus of ecology (Chesson, 2000; Hutchinson, 1957). These issues are of increasing importance in the context of the rapid degradation of natural ecosystems, global climate change, and accelerated species extinctions during the Anthropocene. Classical coexistence theory emphasizes the contributions of resource niche partitioning to community assembly (Hutchinson, 1957; MacArthur, 1969; Tilman, 1982). However, natural enemies such as pathogens may also play an important role in regulating plant populations (van der Heijden et al., 2008) and help facilitate plant species coexistence (Alexander, 2010; Bagchi et al., 2014; Bever et al., 2015; Kempel et al., 2018).

Pathogens can reduce plant performance through their negative effects on plant photosynthesis, growth, survival, or reproduction, thereby modifying outcomes of plant competition (Fisher et al., 2012). Specialized pathogens, in particular, tend to increase in abundance and cause greater disease severity as their host plants become more common, thereby differentially limiting plant growth and seedling survival of more abundant species (Bagchi et al., 2010). Many tests of the Janzen–Connell hypothesis (Connell, 1971; Janzen, 1970; Liang et al., 2016; Liu et al., 2012; Packer & Clay, 2000) and negative plant–soil feedbacks (Mills & Bever, 1998) have provided evidence supporting such a role for pathogens as stabilizing mechanisms (Chesson, 2000; Mordecai, 2011). These hypotheses emphasize that plant performance declines at greater pathogen abundance, leading to conspecific negative density dependence (CNDD). Some studies on the dilution effect (sensu Keesing et al., 2006) of host plant diversity on foliar fungal disease severity (Liu et al., 2016; Mitchell et al., 2002) also provide evidence that disease transmission increases with the abundance of a given plant species. Pathogens, especially soil-borne pathogens, are considered an important factor driving stabilizing mechanisms in a range of ecosystems, including tropical and subtropical forests (Liu et al., 2012), temperate forests (Packer & Clay, 2000), grasslands (Petermann et al., 2008), and old fields (Mills & Bever, 1998).

In contrast to stabilizing mechanisms, average fitness differences among species reflect variation in overall competitive ability that can lead to competitive exclusion in the absence of niche differences among neighboring species (Chesson, 2000). On the one hand, pathogens may reduce differences in average fitness by disproportionately damaging the competitively dominant species; this is an equalizing mechanism (sensu Mordecai, 2011) that can slow progression toward competitive exclusion. For instance, a growth-defense trade-off in which fast-growing species invest less in defense may lead them to harbor more pathogens (Parker & Gilbert, 2018); this is expected to reduce fitness differences among species, which in turn can enhance diversity by preventing competitive exclusion (Cappelli et al., 2020; Coley, 1987). On the other hand, pathogens can promote competitive exclusion if they reduce the fitness of inferior or equal competitors (Burdon, 1993).

Accumulated empirical evidence supports pathogens as potential factors affecting plant species coexistence in both forest and grassland ecosystems (e.g., Allan et al., 2010; Liu et al., 2012; Mangan et al., 2010; Mitchell, 2003; Packer & Clay, 2000; Parker & Gilbert, 2018; Petermann et al., 2008; Spear & Mordecai, 2018; Whitaker et al., 2017). Although plant pathogens are often considered a single ecological factor, they comprise both aboveground and belowground guilds and include deeply diverged lineages. Though historically lumped together because of their similar growth forms, fungal and oomycete pathogens differ in numerous critical ways. Oomycetes (in the Stramenopiles, within the Eukaryotic supergroup SAR) are phylogenetically distinct from fungi (kingdom Fungi, in the Eukaryotic supergroup Opisthokonta) with different physiologies and life cycles. Many oomycetes, especially those from the order Peronosporales, are virulent plant pathogens; most attack belowground plant parts and can directly reduce plant survival, whereas a smaller number affect aboveground parts and reduce plant growth and reproduction (Delavaux et al., 2020; Domínguez-Begines et al., 2021). Fungi comprise the greatest number of plant pathogens and can affect any plant parts, but many fungal pathogens mainly damage aboveground tissues, reducing productivity (Liu et al., 2019). Thus, oomycete pathogens might be inherently different from fungal pathogens in their effects on plant species coexistence.

Here we implement two experiments in an alpine meadow to test how fungal versus oomycete pathogens affect plant species coexistence (Figure 1). There was only one aboveground oomycete pathogen (caused downy mildew) in our site, but most of the aboveground diseases were caused by fungi (Liu et al., 2019). First, we conduct a factorial experiment with neighbor-removal and fungicide-application treatments (population-level experiment) to test how fungal versus oomycete pathogens affect CNDD (i.e., the slope of the relationship between conspecific density and growth) and plant fitness (i.e., plant growth without neighbors) across each of 25 co-occurring alpine meadow species (see Appendix S1: Table S1 for plant species and their associated foliar pathogens). Second, we use a community-level experiment to test whether aboveground disease severity is density dependent at both intraspecific (across plots within a species) and interspecific (across species in the community) levels and to evaluate the effect of

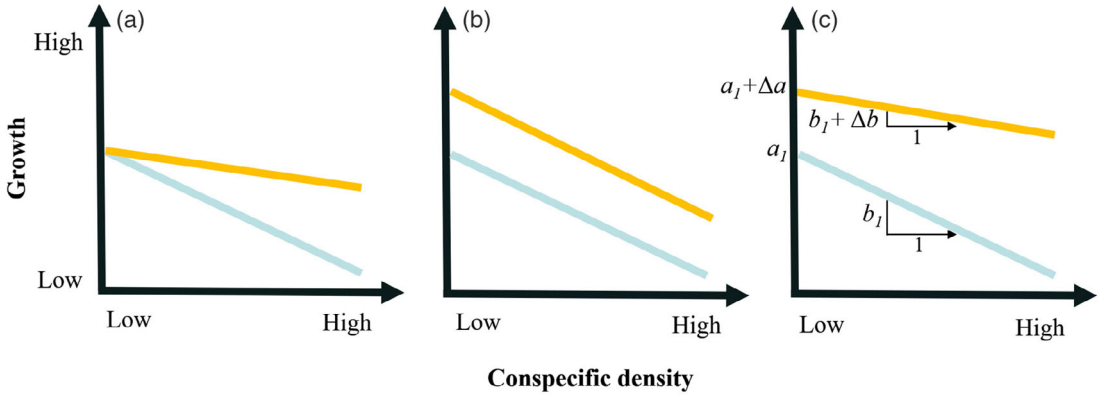

FIGURE 1 A framework for how pathogens affect plant species coexistence, showing how plant growth changes with conspecific host density and with fungicide treatment (yellow) relative to control (blue). We assume intraspecific competition, such that a plant will produce more aboveground biomass at lower conspecific density. The strength of conspecific negative density dependence (CNDD) is indicated by the slope of the line showing the relationship between the amount of growth and conspecific density. For any given species, we expect (a) if pathogens generate CNDD, application of fungicides to reduce pathogens should lead to a shallower slope of the line; (b) if pathogens decrease host fitness across all densities, fungicide application should lead to a greater *y*-intercept; (c) if pathogens generate CNDD as well as decrease host fitness, fungicide application should cause both a shallower slope of the line and a higher *y*-intercept. Here, a_1 is intercept for control (blue line), Δa is the difference in intercept between the control and fungicide treatments, b_1 is the standardized effect size (i.e., regression coefficient) for control, and Δb is the difference in standardized effect size between control and fungicide treatments.

fungicides on the severity of foliar fungal diseases. Finally, we examine the ecological consequences of pathogen removal on plant diversity and community composition.

MATERIALS AND METHODS

Study site

The field experiments were conducted in the northeastern part of Qinghai-Tibetan Plateau, at the Haibei Alpine Meadow Ecosystem Research Station of Northwest Institute of Plateau Biology, Chinese Academy of Sciences, in Menyuan County, Qinghai Province, People's Republic of China (101°19′ E, 37°36′ N; 3215 m above sea level). The mean annual temperature is −1.2°C, and mean annual precipitation is 489.0 mm, approximately 80% of which falls during the short growing season (May–September) (Liu et al., 2019). The soils are classified as "Mat-Gryic Cambisols" (Chinese Soil Taxonomy) and as "borolls" (US Department of Agriculture [USDA] Soil Taxonomy), with 60-cm thickness, and are colimited by both nitrogen and phosphorus (Niu et al., 2016). The grassland vegetation is a typical alpine meadow, and the plant community is dominated by perennial herbaceous species of Poaceae, Asteraceae, and Ranunculaceae, such as *Anemone obtusiloba, Elymus nutans*, *Gentiana macrophylla*, *Poa annua*, *Potentilla bifurca*, and *Saussurea nigrescens*. The dominant animals include horses, sheep, yaks, plateau pikas (*Ochotona curzoniae*), zokor (*Myospalax* spp.), and ants.

Population-level experiment (Experiment 1)

Experimental design

On 14 June 2019, we established a neighbor-removal and pathogen-exclusion experiment (i.e., population-level experiment) in a 50 × 70-m rectangle within a flat area. We selected 25 focal plant species that varied in rarity (Appendix S1: Table S1), phylogeny, and traits, with 60 individuals for each focal species, resulting in 1500 focal individuals. The 60 individuals of the focal species were located as close as possible to minimize differences in resource availability and microclimate but far apart enough (>50 cm) from each other to minimize the impact of adjacent treatments (especially fungicide application). The 60 individuals of the same plant species were selected to represent a gradient in conspecific density within a circular plot with a radius of 15 cm, from low to high density. To estimate initial biomass, we first derived unique equations for each species relating biomass to measured functional traits (e.g., number of leaves [NL] and plant height [H] for *Astragalus polycladus*; Appendix S1: Table S2). At the start of the experiment, we measured traits on each focal individual and then estimated its initial biomass ($B_{i,t}$) using the best estimation equation for that species (Appendix S1: Table S2 and Section S1).

The 60 individuals of each plant species were divided into 10 blocks based on their initial biomass (e.g., the six individuals with the smallest biomass were included in the same block) to control for the effect of initial size

on response to treatments. Then we randomly assigned each of the six treatments to the six individuals in each block. Treatments were implemented in circular plots of 15-cm radius around each focal individual. Treatments included (F) fungal pathogen exclusion, (O) oomycete pathogen fungicide, or (C) an equal amount of water; (F + R) fungal pathogen exclusion and neighbor removal, (O + R) oomycete pathogen exclusion and neighbor removal, or (C + R) an equal amount of water and neighbor removal.

The fungicides used to control fungal and oomycete pathogens were applied every 2 weeks from mid-June to early September 2019. The fungal pathogen-exclusion treatment was composed of Yangcai (Syngenta Crop Protection, Nantong, Jiangsu, China), a leaf spray fungicide with a combination of Azoxystrobin (7.0%; active ingredient [a.i.], 6.31×10^{-2} g m^{-2} year^{-1}) and Propiconazole (11.7%; a.i., 0.11 g m^{-2} year^{-1}) (Seabloom et al., 2017). This fungicide primarily targets fungi in the Ascomycota and Basidiomycota and has been used widely in both agricultural and natural ecosystems to prevent aboveground foliar fungal diseases. The oomycete pathogen-exclusion treatment was composed of Guoguang (Shaanxi Xiannong Biotechnology Co., Weinan, Shaanxi, China), a soil-drench fungicide of Mefenoxam (30.0%; a.i., 7.21×10^{-2} g m^{-2} year^{-1}) (Seabloom et al., 2017) that targets oomycetes, rather than fungi (Domínguez-Begines et al., 2021). As a control, we sprayed the same amount of water on nonfungicide plots (Bagchi et al., 2014).

Neighbor-removal treatments (750 plots) were implemented by removing the aboveground biomass of all neighboring conspecific and heterospecific individuals within a radius of 15 cm around the focal individual (Chu et al., 2008). We clipped all aboveground parts and removed as much of the roots as possible, taking care not to damage the focal plant. Given the rapid growth of plants during the short growing season, we had to repeat the removal treatment five times (mid-June, late June, mid-July, late July, and early August).

Sampling and plant growth calculation

To estimate the effects of neighboring conspecific density on focal individual growth, we recorded conspecific density (measured as the number of conspecific individuals per circle plot) just after the field experiment setup, in mid-June. After 80 days (growth period u), in early September, we harvested the aboveground biomass of each focal individual, dried them at 65°C, then weighed them to 0.0001 g for final biomass ($B_{i,t+u}$). There was a total of 1292 (86.13%) focal individuals harvested, and 208 (13.87%) individuals died or experienced severe herbivory (mainly from plateau pikas or zokor). Growth of each focal individual was calculated as final biomass minus (estimated) initial biomass: $G_i = B_{i,t+u} - B_{i,t}$.

We recorded foliar disease severity on leaf replicates by visual estimation, referencing cards with digitized images of leaves of known disease severity, in early September 2019. We recorded leaf-level disease severity (percentage of the leaf area covered by fungal lesions) and visually assessed the presence of pathogen groups on all leaves from the 1292 individuals available. Then we calculated the foliar disease severity index (V_i) as the average disease severity of all leaves for each individual we checked. The taxonomic groups of the pathogens (i.e., fungus-caused leaf-spot disease, rusts, powdery mildews, and downy mildews) were confirmed in the lab using an OLYMPUS CX33 light microscope (OLYMPUS, Tokyo, Japan). Identification also benefitted from extensive experience gained during previous studies at this site (e.g., Zhang, 2009).

Community-level experiment (Experiment 2)

Experimental design

An experiment crossing fungicide application with nutrient addition (hereafter labeled the community-level experiment) was established in August 2017, at a site adjacent to the population-level experiment. The entire experiment was a full four-way factorial experiment with a randomized complete block design ($N = 6$ per treatment combination). Nitrogen addition (N), phosphorus addition (P), fungal pathogen exclusion (F), and oomycete pathogen exclusion (O) were the four main factors ($2 \times 2 \times 2 \times 2 = 16$ treatments). However, for this study, fertilization was not considered, so only 24 plots (four treatments: control, F, O, and F + O [fungal and oomycete pathogen exclusion]) were included. A total of 96 3 × 3 m plots were arranged on a flat meadow with a 2-m buffer zone between plots and a 3-m buffer zone between blocks. Grazing (mainly sheep) was permitted only in winter. The pathogen-exclusion treatments were the same as the population-level experiment. The fungicides were applied every 2 weeks from mid-April to mid-September annually from 2018.

In early September (before most plants began to senesce) from 2017 to 2020, we delimited a random 0.5 × 0.5 m subplot at least 0.5 m from any plot edge. We harvested all the stems and leaves in each subplot and then sorted them by species. We dried the samples at 65°C until they stabilized at constant weight and weighed them to 0.001 g to estimate aboveground biomass. In addition,

to monitor shifts in plant species richness and percentage cover, we established a 0.5 × 0.5-m permanent quadrat at the center of each plot and visually estimated percentage cover (from an iron grid with 25 0.1 × 0.1-m^2 cells). Specifically, the percentage cover for each plant species in each quadrat was calculated as the average cover over the 25 cells.

Foliar disease evaluation

We recorded foliar disease severity on 25 leaves, with five from each of five randomly selected stems, for each plant species in each plot. For species with no more than five stems, we examined all available individuals. Then we calculated the population-level foliar disease severity index (V_i) as the average disease severity of the 25 leaves for each plant species in each plot we checked. We also collected five to 10 samples of infected plant tissue per plant species at the same study site in September 2019 to confirm the groups of the pathogens (i.e., fungus-caused leaf-spot disease, rusts, powdery mildews, and downy mildews) in the lab using an OLYMPUS light microscope.

We defined community pathogen load (l) as

$$l = \frac{\sum_{i=1}^{S} a_i V_i}{\sum_{i=1}^{S} a_i}$$

where S is the total number of host plant species, a_i is the biomass of plant species i, and V_i is the severity index. Community pathogen load (l) has been widely used in plant disease ecology and is considered a good indicator of community fungal diseases severity (Liu et al., 2019; Mitchell et al., 2002).

Statistical analyses

Population-level experiment (Experiment 1)

For the population-level experiment, linear mixed-effects models, with "block" nested in "species" as a random effect, were used for testing the effects of pathogen-exclusion and neighbor-removal treatments on foliar fungal disease severity and plant growth across the 25 plant species. We dropped the potential interactions between pathogen-exclusion and neighbor-removal treatments in the following analyses, given the fact that all these interactive effects were not significant. Independent variables were standardized (i.e., we subtracted the mean and divided by the standard deviation) to facilitate comparisons. The standardized effect sizes and 95% confidence intervals (CIs) of fixed effects were then extracted from the linear mixed-effects models. Furthermore, at the population level, for each of the 25 focal plant species, we used linear mixed-effects models with "block" as random effect to test how the pathogen-exclusion and neighbor-removal treatments affected foliar fungal disease severity and plant growth, respectively. We used diagnostic plots to verify the normality and homoscedasticity of the residuals. The aforementioned linear mixed-effects models were built using the **`lmer`** function in the R package **`lme4`** (Bates et al., 2015) with the restricted maximum likelihood method. The effect sizes of each fixed effect and their corresponding 95% CIs were extracted. The effect size was considered to be significant if the 95% CI of the estimated value did not include zero. This method was considered reasonable, given the fact that there was no significant interaction between the fixed effects.

For the population-level experiment, the strength of CNDD was defined as the standardized effect size (i.e., regression coefficient) of the relationship between density and plant growth. To examine whether fungicides reduced the effects of conspecific density, we fit a linear mixed-effects model to the plant growth data from the nonremoval plots, with fungicide treatments (F and O), conspecific density, and their interaction included as predictors and "block" nested in "species" as random effect.

Community-level experiment (Experiment 2)

For the community-level experiment, we tested the relationship between foliar fungal disease severity and host plant biomass, first within species across plots, and then across species. At the intraspecific level, we used the biomass (log-transformed to meet normality assumptions) by species across six control plots to predict corresponding disease severity in linear mixed-effects models across 3 years (i.e., 2018–2020). Plot and species were treated as random effects, after checking the normality of residuals and homogeneity of variance. At the interspecific level, we tested the relationship between average biomass (log-transformed) of each plant species across six control plots and the average foliar disease severity in linear mixed-effects models with "species" as random effect (one species that appeared in only one plot, *Elsholtzia ciliata*, was excluded). Similarly, using the subset of control plots, we evaluated the relationship between foliar fungal disease severity and host plant biomass at both intra- and interspecific levels to test the density dependence of foliar fungal disease severity. Moreover, linear mixed-effects models were also used to

test the effect of fungal and oomycete pathogen exclusion and their interaction on community pathogen load in 24 plots of the community-level experiment across 2018–2020, with cross-nested random structure (i.e., [1|block/plot] + [1|year]). We also tested the target effect of fungicides at the individual species level, with "year," "species and plot" nested in "block" as random structure (i.e., [1|block/plot] + [1|year] + [1|species]). We built a series of linear mixed-effects models, with "block" as a random effect to test the effects of fungal and oomycete pathogen exclusion and their interaction on plant species richness and Shannon's evenness index across 24 plots in the community-level experiment.

Permutational multivariate analysis of variance (PERMANOVA) in the R package vegan (Oksanen et al., 2020) was used to evaluate whether overall plant community composition after 3 years (i.e., 2020) differed across fungal and oomycete pathogen-exclusion treatments and blocks. Moreover, we classified all the plant species we found in the subplots into one of the four taxonomic groups: Poaceae/Cyperaceae, Asteraceae, Fabaceae, and others (Liu et al., 2015). Linear mixed-effects models were used to test how treatments affect the absolute biomass of each group, with "block" as a random effect. All statistical analyses were performed using R version 3.6.0 (R Development Core Team, 2020).

RESULTS

Pathogen-induced CNDD

Both fungal pathogen-exclusion treatment and the removal of neighboring conspecific plants reduced foliar fungal disease. The fungal pathogen-exclusion treatment (standardized effect size [ES] = −0.223, 95% CI = −0.338 to −0.109, $t_{1288} = -3.818$, $p < 0.001$) and neighbor removal (ES = −0.251, 95% CI = −0.345 to −0.158, $t_{1288} = -5.276$, $p < 0.001$), but not the oomycete pathogen-exclusion treatment (ES = 0.022, 95% CI = −0.092 to 0.137, $t_{1288} = 0.384$, $p = 0.701$), decreased foliar fungal disease severity significantly in the population-level experiment (Figure 2a; Appendix S1: Table S3). Neighbor removal significantly increased plant growth (ES = 0.285, 95% CI = 0.203–0.367, $t_{1288} = 6.798$, $p < 0.001$), but there was no effect of either fungal (ES = −0.030, 95% CI = −0.123 to 0.070, $t_{1288} = -0.585$, $p = 0.559$) or oomycete pathogen exclusion on plant growth (ES = 0.014, 95% CI = −0.087 to 0.115, $t_{1288} = -0.270$, $p = 0.787$) (Figure 2b; Appendix S1: Table S4).

Our results confirmed that fungal pathogens induced CNDD. Taking 25 plant species collectively, we found a marginally significant negative effect of conspecific density in control plots (ES = −0.123, 95% CI = −0.260 to 0.014, $t_{605} = -1.768$, $p = 0.078$ for nonremoval plots) (Figure 2c and Appendix S1: Figure S1). Among 25 species, 21 species (84.0%) experienced CNDD for plant growth (derived from the nonfungicide plots in the population-level experiment), including five species that experienced significant CNDD (Appendix S1: Table S5), indicating CNDD is universal in the alpine meadow. We further found that CNDD was not significantly related to the species' log-transformed biomass in the control plots ($R^2 = 0.027$; $t_{23} = 0.766$; $p = 0.453$) (Appendix S1: Figure S2), indicating that the strength of CNDD did not differ between rare and dominant species, which is inconsistent with several previous studies (e.g., Comita et al., 2010; Mangan et al., 2010). Fungal pathogens were important drivers of the density-dependent reduction in growth, and CNDD was neutralized by fungal pathogen exclusion (interaction effect between fungal pathogen exclusion and conspecific density: ES = 0.146, 95% CI = −0.020 to 0.313, $t_{605} = 1.717$, $p = 0.087$ for nonremoval plots) rather than oomycete pathogen-exclusion treatment (interaction effect between oomycete pathogen exclusion and conspecific density: ES = 0.081, 95% CI = −0.070 to 0.232, $t_{605} = 1.051$, $p = 0.294$ for nonremoval plots) (Figure 2c; Appendix S1: Tables S5–S7).

Fungal pathogens maintain plant species diversity and composition

Consistent with our expectations, foliar fungal disease severity was density-dependent at both intraspecific (effect size [ES] = 2.293×10^{-3}, 95% CI = 4.667×10^{-4}–4.113×10^{-3}, $t_{149} = 2.464$, $p = 0.014$) and interspecific (ES = 2.242×10^{-3}, 95% CI = 3.112×10^{-5}–4.453×10^{-3}, $t_{555} = 1.988$, $p = 0.050$) levels, based on linear mixed-effects models of control plots in the community-level experiment (Figure 3a,b). Interestingly, for community pathogen load, we found that fungal pathogen exclusion significantly decreased pathogen load by 41.72% (ES = −1.622, 95% CI = −2.440 to −0.804, $t_{66} = -3.886$, $p < 0.001$) across 3 years of treatment, whereas oomycete pathogen exclusion (ES = −0.538, 95% CI = −1.355 to 0.280, $t_{66} = -1.288$, $p = 0.202$) and the interaction effect of fungal and oomycete pathogen exclusion (ES = 0.412, 95% CI = −0.744 to 1.569, $t_{66} = 0.699$, $p = 0.487$) had no significant effect on pathogen load (Appendix S1: Table S8). Analyses at the individual species level showed a similar pattern (Appendix S1: Table S9).

Based on the data from 2020, fungal pathogen exclusion significantly decreased plant species richness by

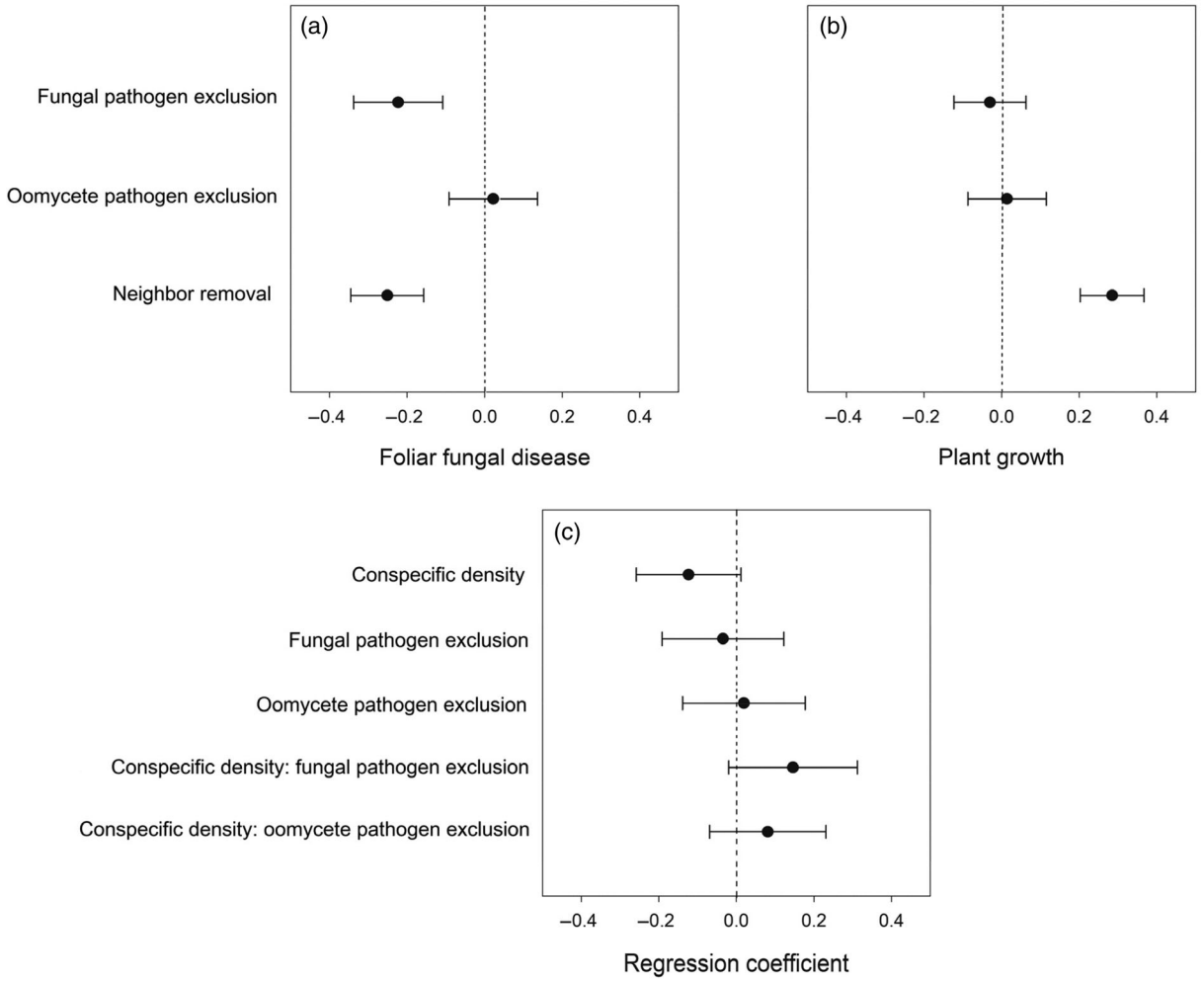

FIGURE 2 Standardized effect sizes (filled circles) with 95% confidence intervals across 25 focal plant species from the population-level experiment, analyzed with linear mixed-effects models. Shown are the effects of fungal and oomycete pathogen exclusion as well as neighbor removal treatment on (a) foliar disease severity and (b) plant growth (increase in biomass). Neighbor removal treatment was treated as a fixed effect, and "block" was nested in "species" as a random effect. (c) The effect of conspecific negative density dependence was weakened by fungal pathogen exclusion. Shown are the regression coefficients derived from a linear mixed-effects model, with fungicide treatments, conspecific density, and their interaction included as predictors and "block" nested in "species" as a random effect.

9.14% (ES = −2.833, 95% CI = −5.517 to −0.149, $t_{20} = -2.069$, $p = 0.052$), but no significant effect was found for oomycete pathogen exclusion (ES = −2.167, 95% CI = −4.851 to 0.517, $t_{20} = -1.582$, $p = 0.129$) or the interaction effect (ES = 2.833, 95% CI = −0.962 to 6.629, $t_{20} = 1.463$, $p = 0.159$) (Table 1a). Moreover, fungal pathogen exclusion significantly decreased Shannon's evenness index by 6.45% (ES = −0.192, 95% CI = −0.338 to −0.046, $t_{20} = -2.574$, $p = 0.021$). As with richness, oomycete pathogen exclusion alone had no significant effect on evenness (ES = −0.104, 95% CI = −0.251 to 0.042, $t_{20} = 1.400$, $p = 0.182$) (Table 1b). However, unlike with richness, there was a significant interaction between the effects of fungal and oomycete pathogen exclusion on evenness (ES = 0.227, 95% CI = 0.020–0.433, $t_{20} = 2.149$, $p = 0.048$). The interaction term reflects that fungal pathogen exclusion decreased evenness on its own, but not when combined with oomycete pathogen exclusion, which may indicate complex compensatory interactions among plant competitors.

Fungal pathogens had an important effect on plant community composition. Particular taxonomic groups showed stronger responses than others; for example, fungal pathogen exclusion significantly increased the absolute biomass of Poaceae and Cyperaceae by 32.94% (ES = 17.877, 95% CI = 3.824–31.930, $t_{20} = 2.493$, $p = 0.025$) and "others" by 44.37% (ES = 8.808, 95% CI = 1.917–15.699, $t_{20} = 2.505$, $p = 0.024$) (Appendix S1: Tables S10 and S11). Based on PERMANOVA, we found that fungal pathogen exclusion ($F_{1,16} = 2.350$, partial

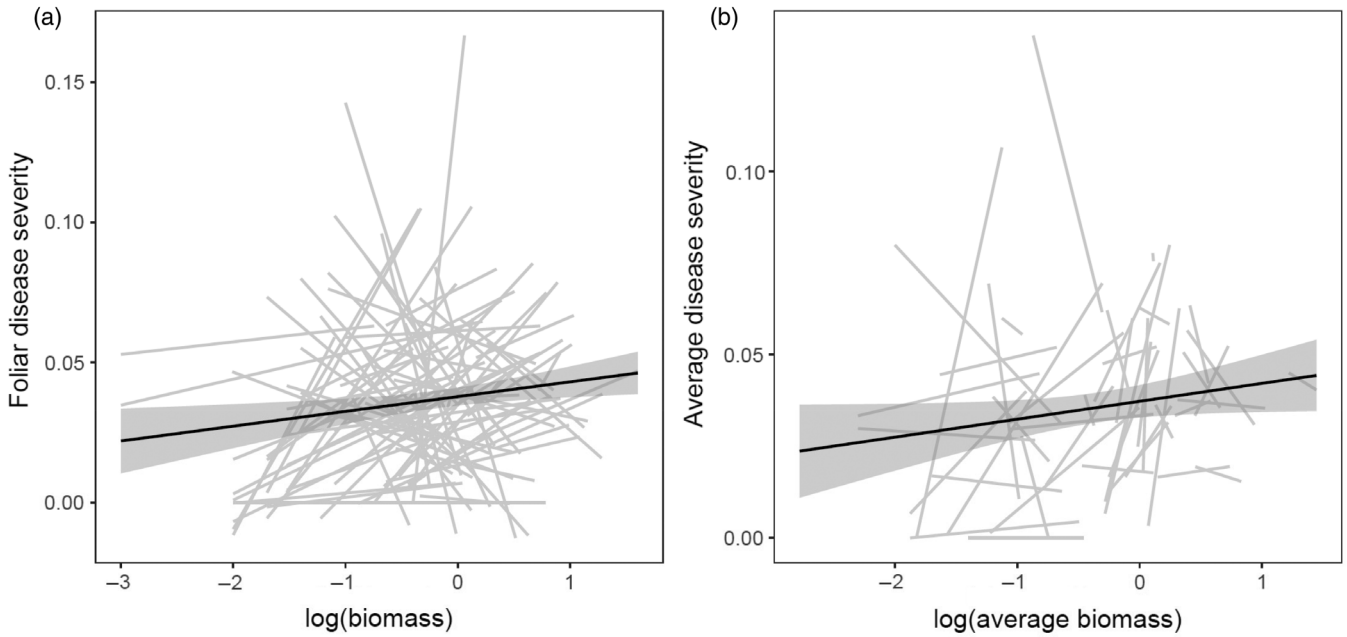

FIGURE 3 Relationships between foliar disease severity and biomass at intra- and interspecific levels in the community-level experiment. (a) The biomass of each plant species in the linear mixed-effects model was used to predict foliar disease severity in six control plots, with "plot" and "species" as random effects. Each gray line represents the regressions for individual plant species in one plot across 3 years. (b) Relationship between average biomass across six control plots and average foliar disease severity, with "species" as random effect in the linear mixed-effects model. Each gray line represents one plant species; one species (*Elsholtzia ciliata*) that appeared in only one plot was removed.

TABLE 1 Linear mixed-effects model results for the effects of fungal and oomycete pathogen exclusion and their interaction effect on (a) plant species richness and (b) plant Shannon's evenness index in 24 plots of the community-level experiment in 2020. Fungal and oomycete pathogen exclusion and their interaction effects were treated as fixed effects and "block" as a random effect. Shown are the estimated regression coefficient, *t*-value, and *p* value for each fixed effect.

Fixed effect	Estimated regression coefficient	*t* value ($df = 20$)	*p* value
(a) Plant species richness			
Fungal pathogen exclusion	**−2.833**	**−2.069**	**0.052**
Oomycete pathogen exclusion	−2.167	−1.582	0.129
Fungal: Oomycete pathogen exclusion (interaction term)	2.833	1.463	0.159
(b) Plant Shannon's evenness index			
Fungal pathogen exclusion	**−0.192**	**−2.574**	**0.021**
Oomycete pathogen exclusion	−0.104	−1.400	0.182
Fungal: Oomycete pathogen exclusion (interaction term)	**0.227**	**2.149**	**0.048**

Note: The significant results are in bold.

$R^2 = 0.086$, $P = 0.004$) and "block" ($F_{5,16} = 1.592$, partial $R^2 = 0.292$, $p = 0.002$) significantly affected plant community composition in 2020, whereas there was no significant effect of oomycete pathogens ($F_{1,16} = 0.952$, partial $R^2 = 0.035$, $p = 0.485$) (Appendix S1: Table S12).

DISCUSSION

Overall, CNDD was common in the alpine meadow, that is, plants produced more biomass when the species was rare than when it was common. One mechanism for CNDD can be strong intraspecific competition for limiting resources (i.e., bottom-up forces) (Hutchinson, 1957; MacArthur, 1969). An alternative mechanism for CNDD is the effects of natural enemies such as herbivores and soil pathogens (i.e., top-down forces as described by the Janzen–Connell hypothesis; Janzen, 1970; Connell, 1971). Soil pathogens in particular have been shown to accumulate disproportionately under high host density (Liang et al., 2016; Mangan et al., 2010) and to play a key role as a stabilizing mechanism for the maintenance of plant diversity via CNDD (Chesson, 2000; Mordecai, 2011). Our results show clear evidence that (mostly foliar) fungal pathogens, more than soil-borne oomycete pathogens, produced CNDD in the alpine meadow, which can serve as a stabilizing mechanism for plant diversity.

We found evidence for density-dependent foliar disease severity, indicating that foliar fungal disease development is generally proportional to host density (Mitchell et al., 2002). This is consistent with previous studies in both agricultural (Burdon & Chilvers, 1982; Zhu et al., 2000) and wild systems (Mitchell et al., 2002; Rottstock et al., 2014; Parker et al., 2015; Bayandala & Seiwa, 2017; but see Spear & Mordecai, 2018; Kendig et al., 2020). The density responsiveness of disease severity may reflect a preponderance of host specialists in the pathogen community (Gilbert & Webb, 2007; reviewed in Gilbert & Parker, 2016). Density-dependent foliar disease severity may influence plant growth in two ways; pathogens may reduce leaf area and photosynthetic activity directly (Allan et al., 2010), or plant growth may be suppressed in the presence of pathogens because of tradeoffs between growth and induced defense (Lind et al., 2013). Through the combined study of foliar fungal disease severity and host plant density both within and across species, we revealed how the negative effects of aboveground pathogens on host plants contribute to the CNDD we observed in the alpine meadow.

We provide mechanistic evidence that fungal pathogens (mainly foliar fungal pathogens) can promote coexistence via CNDD in a species-rich alpine meadow dominated by perennials. This type of experimental study has not been commonly done with foliar fungal pathogens. The previous studies we are aware of provided mixed evidence for how foliar fungal pathogens affect plant species richness, from negative effects (pathogen-exclusion experiment in Peters & Shaw, 1996), no effects (Dawkins, 1988; Mitchell, 2003; Parker & Gilbert, 2018; Schmidt et al., 2020; Spear & Mordecai, 2018), to positive effects (pathogen augmentation experiment in Peters & Shaw, 1996; Allan et al., 2010; Whitaker et al., 2017). Possible explanations for these differences among studies include the duration of fungicide treatment (long term vs. short term) (Dawkins, 1988), vegetation type (grass-dominated grassland vs. forb-dominated meadow) (Allan et al., 2010; Schmidt et al., 2020), pathogen life history (biotroph vs. necrotroph), and plant origin (native vs. nonnative). Others have found that native grasses experienced greater disease severity (Kendig et al., 2020), and biotrophic pathogens were more sensitive to fungicides (Cappelli et al., 2020); thus, grasses with more biotrophic pathogens might benefit the most under fungal pathogen-exclusion treatment.

We note that belowground oomycete pathogens can also shape CNDD and maintain plant species coexistence in the alpine meadow. The phenylamide fungicide Mefenoxam was used to control soil-borne diseases caused by oomycetes (Seabloom et al., 2017). Previous studies also used the fungicide Mefenoxam to successfully remove pathogens in temperate forests (Jia et al., 2020). Like these studies, we found that Mefenoxam could decrease plant species richness by reducing soil-borne oomycete pathogens, highlighting the important role of these pathogens in the ecosystem (Delavaux et al., 2020). However, our comparative study suggests that (foliar) fungal pathogens could play an even greater role than the oomycete pathogens that have received much more attention (Domínguez-Begines et al., 2021). This is partly consistent with the results of Bagchi et al. (2014), suggesting that fungi, more than oomycetes, shape plant diversity in tropical forest.

Fungicide application can affect both fungal and oomycete pathogens simultaneously. However, oomycetes tend to be belowground pathogens, whereas foliar pathogens (except downy mildew) are mainly fungi. For example, at our site there was only one aboveground oomycete pathogen, a downy mildew that caused limited disease severity (Liu et al., 2019). We confirmed in our experiment that a combination of Azoxystrobin and Propiconazole (directed at fungi), but not Mefenoxam (directed at oomycetes), significantly decreased foliar fungal diseases (Appendix S1: Tables S8 and S9). At the same time, we found no detectable effects on belowground mycorrhizal fungi in the plants treated with Azoxystrobin and Propiconazole (Appendix S1: Section S1), although saprotrophic Ascomycete fungi in the soil or litter layer may have been sensitive to them. Together, these lines of evidence suggest that the fungi were the main pathogen drivers of CNDD, helping shape community-level plant diversity and composition. The weaker effects of the oomycete removal treatment point to fungi being more important than oomycetes in causing CNDD in this system; however, if the efficacy of fungicides was not as efficient in complex soil media as on leaf surfaces, the effect of oomycetes might have been underestimated.

Pathogens are ubiquitous in natural ecosystems. They can have a range of functions (Kohli et al., 2020; Mordecai, 2011), but their ecological effects are difficult to determine in situ (Peters & Shaw, 1996). Using fungicides "is probably the only practical method of studying the overall role of these organisms in natural vegetation" (Paul et al., 1989). However, nontarget direct effects of fungicides on plant growth or on other plant-associated organisms like mycorrhizal fungi can complicate the interpretation of results. Our supplementary experiments confirmed the target specificity of the fungicides we used. The "Azoxystrobin and Propiconazole" treatment significantly decreased foliar fungal diseases, with no detectable effects on mycorrhizal fungi or soil chemical properties. The greenhouse experiment further produced no evidence of direct effects of the fungicides on the growth of four plant species (Appendix S1: Table S13).

Our experiments highlight the support for natural enemy–mediated CNDD driven by (foliar) fungal pathogens, promoting species coexistence and structuring the plant community. The effects of pathogen removal might be expected to accumulate over time, so in the absence of pathogens we would expect a sustained reduction in plant diversity in our perennial alpine meadow. Similar experiments in other ecosystems are needed to evaluate the relative importance of (foliar) fungal pathogens and oomycete pathogens and to understand the generality and context dependence of natural enemy–mediated CNDD as an explanation for species coexistence.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data (Liu et al., 2022) are available in Zenodo at https://doi.org/10.5281/zenodo.6867708.

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