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Research

Warming affects foliar fungal diseases more than precipitation in a Tibetan alpine meadow

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Summary

- The effects of global change on semi-natural and agro-ecosystem functioning have been studied extensively. However, less well understood is how global change will influence fungal diseases, especially in a natural ecosystem.
- We use data from a 6-yr factorial experiment with warming (simulated using infrared heaters) and altered precipitation treatments in a natural Tibetan alpine meadow ecosystem, from which we tested global change effects on foliar fungal diseases at the population and community levels, and evaluated the importance of direct effects of the treatments and community-mediated (indirect) effects (through changes in plant community composition and competence) of global change on community pathogen load.
- At the population level, we found warming significantly increased fungal diseases for nine plant species. At the community level, we found that warming significantly increased pathogen load of entire host communities, whereas no significant effect of altered precipitation on community pathogen load was detected.
- We concluded that warming influences fungal disease prevalence more than precipitation does in a Tibetan alpine meadow. Moreover, our study provides new experimental evidence that increases in disease burden on some plant species and for entire host communities is primarily the direct effects of warming, rather than community-mediated (indirect) effects.

Introduction

Plant fungal diseases play a key role in determining the rate and magnitude of ecosystem function and service delivery across multiple spatial scales (Fisher *et al.*, 2012). Owing to the sometimes strong negative effects of diseases on plant photosynthesis and growth (Fisher *et al.*, 2012), fungal diseases can modify plant competition, affect the assembly of natural plant communities, and ultimately influence evolution, speciation, and extinction of plant species (Bever *et al.*, 2015; Parker *et al.*, 2015; Ricklefs, 2015). Under the background of global change (Collins *et al.*, 2013), it is critical to know how global change factors (e.g. warming, altered precipitation, and nitrogen deposition) influence plant species interactions, and especially fungal diseases (Altizer *et al.*, 2013).

Warming is generally expected to affect fungal diseases directly in plant communities (e.g. Tapsoba & Wilson, 1997; Pfender & Vollmer, 1999; Hannukkala *et al.*, 2007; Siebold & Tiedemann, 2013; Launay *et al.*, 2014). On the one hand, warming is expected to increase pathogen fitness and transmission (Siebold & Tiedemann, 2013), by increasing growth rates and spore production, promoting mycelium growth, and extending the lengths of growth and reproduction times (Harvell *et al.*, 2002; Launay *et al.*, 2014). On the other hand, plant host resistance and tolerance might also benefit from warming simultaneously (Cavieres *et al.*, 2014). Hence, the net outcome of warming on plant diseases depends on the independent responses of plant and pathogen populations under a warming environment (Garrett *et al.*, 2006), which might be species specific.

Global change is comprised of a number of other environmental changes beyond temperature change. Precipitation patterns are influenced by global change (Piao *et al.*, 2010) and are potentially relevant for community disease dynamics. Understandably, fungal pathogens are likely to benefit from increased moisture, which can promote fungal spore germination, mycelium growth, and initiation of infection (Woods *et al.*, 2005; Strengbom *et al.*, 2006). In addition, the dispersal of spores could be affected by precipitation over short distances both positively (dispersal

through rain-splash droplets) and negatively (removing spores from air and leaf surfaces before they have attached) (Gigot *et al.*, 2014). For host plants, altered precipitation can alter fungal infections. Most notably, drought stress can promote fungal disease because of physiological stress responses that alter host plant resistance, which has been demonstrated in several agro-ecosystems (e.g. Clover *et al.*, 1999; Mcelrone *et al.*, 2003). However, few experiments have explicitly tested whether the conclusions derived from artificial ecosystems might change in natural systems given the differences in complexity (Garrett *et al.*, 2006; Chakraborty, 2013).

Compared with disease dynamics in managed agro-ecosystems, fungal diseases in natural plant–pathogen systems are subject to more complex influences and interactions (Gilbert & Parker, 2016). While we might not expect climate change to necessarily change the diversity of agricultural systems, warming is predicted to change the composition and diversity of natural plant assemblages and affect the host density (i.e. community evenness) and the phylogenetic structure of communities (Klein *et al.*, 2004; Ma *et al.*, 2017). Similarly, altered precipitation can also drive shifts in host density and community composition (Yang *et al.*, 2011; Yan *et al.*, 2015).

Such changes in host communities can affect fungal diseases through several mechanisms. For example, increases in host species richness can decrease specialist fungal diseases through the dilution effect (Mitchell *et al.*, 2003; Keesing *et al.*, 2006; Ostfeld & Keesing, 2012). In particular, previous studies found that hosts with lower proneness to diseases were more likely to be extirpated in natural communities following anthropogenic changes, leading to increases in community competence (Lacroix *et al.*, 2014). Moreover, the phylogenetic structure of host communities can mediate diseases, given the probability that a pathogen can infect two host species decreases with their phylogenetic distance (Gilbert & Webb 2007, Parker *et al.*, 2015). For individual plant species in a density-dependent transmission system (e.g. plant-foliar fungal pathogens), shifting host density can influence fungal diseases independent of global change (Mitchell *et al.*, 2002; Ostfeld & Keesing, 2012). However, few studies have explicitly tested the relative importance of these mechanisms across global change factors.

To address this knowledge gap, we implemented a 6-yr factorial experiment with warming and altered precipitation in an alpine meadow of the Tibetan Plateau to test global change effects on the foliar fungal diseases at the population and community levels, and to evaluate the relative importance of warming and altered precipitation on both population-level disease severity and community pathogen load based on data from the final year (i.e. 2016) of this long-term experiment. We focused on foliar fungal pathogens, given their high prevalence in our study area and in terrestrial ecosystems globally (Fisher *et al.*, 2012; Liu *et al.*, 2016). Moreover, fungal pathogens are thought to be relatively sensitive to global change (Garrett *et al.*, 2006), providing an ideal system to test for the effects of warming and precipitation on ecological dynamics in grasslands.

In addition, we defined the effects of changes in plant community composition (and thus also changes in percentage cover of individual plant species), species richness, evenness, and phylogenetic diversity as the 'community-mediated (indirect) effects', which act on the fungal diseases though plant community diversity (Hantsch *et al.*, 2014; Liu *et al.*, 2017). We measured host plant community composition, diversity, and proneness (i.e. the expected community pathogen load according to community constituent hosts) in each experimental treatment to investigate the following: first, the influence of experimental warming and altered precipitation on foliar fungal diseases of both individual host plant species (population level) and entire plant communities (community level) after 6 yr of exposure; and second, the importance of community-mediated (indirect) effects and direct effects of experimental warming and altered precipitation in driving the foliar fungal diseases at the population and community level.

Materials and Methods

Study site

We performed our field experiment in the eastern part of Qinghai–Tibetan Plateau, at the Haibei National Field Research Station in the Alpine Grassland Ecosystem in Qinghai Province, People's Republic of China (101°19′E, 37°37′N; 3215 m above sea level). The mean annual temperature is −1.1°C, and the mean annual precipitation is 488 mm (minimum 353 mm and maximum 610 mm over the past 30 yr of precipitation monitoring; Zhao & Zhou, 1999), 80% of which falls during the short growing season (May–September) (Ma *et al.*, 2017). The nitrogen-limited soils are classified as 'Mat-Cryic Cambisols' (Chinese Soil Taxonomy) or 'borolls' (USDA Soil Taxonomy) soils (Lin *et al.*, 2016). The grassland vegetation is a typical alpine meadow, which is dominated by some genera of perennial herbaceous plants, such as *Elymus*, *Gentiana*, *Kobresia*, *Poa*, *Saussurea*, and *Stipa* (see Supporting Information Table S1 for the entire plant species list). The vegetation height is quite variable, with the tallest species being some of the grasses (*c.* 60 cm, e.g. *Elymus nutans* and *Helictotrichon tibeticum*) and the shortest species being some of the forbs (*c.* 3 cm, e.g. *Viola kunawarensis*), and the mean vegetation height is *c.* 30 cm in control plots. The dominant animals are heavily influenced by anthropogenic land use and include sheep, yaks, horses, and ants.

Our study system contains foliar fungal pathogens with different host ranges from a single plant species to an entire family (Zhang, 2009; Liu *et al.*, 2016, 2017; Table S1). Fungal leaf spot is the most common group of foliar fungal diseases, and the whole pathogen community is dominated by some genera of *Ascomycota* and *Basidiomycota*, such as *Ascochyta*, *Puccinia*, and *Trichometasphaeria* (Table S1).

Experimental design

A full description of our experiment is provided in Lin *et al.* (2016), Zhang *et al.* (2016), Ma *et al.* (2017), and Liu *et al.* (2018), but we provide a brief synopsis here. We established 36 2.2 m × 1.8 m plots with a 4-m buffer zone between the plots in July 2011, and grazing was permitted only during winter. Our experiment is a randomized complete block design ($N = 6$ per

treatment combination) with temperature and precipitation as main treatment factors; the plots are designated as control (i.e. dummy installations; see later), decreased precipitation by 50%, increased precipitation by 50%, warming at *c.* 2.0°C, warming with decreased precipitation, or warming with increased precipitation (six treatments for each block), resulting in six treatments × six replicates (blocks) to give 36 plots, and the treatments were applied throughout the year (even in winter). Methodologically, many studies have simulated warming using transparent, reinforced-plastic, open-top chambers (e.g. Klein *et al.*, 2004), but open-top chambers themselves might directly decrease foliar fungal diseases by blocking the transmission of spores (Thompson & Drake, 1994; Liu *et al.*, 2016). For this reason, we used two parallel infrared heaters (1200 W, 220 V, 1 m long, and 0.22 m wide) hung 1.5 m above the ground (*c.* 120 cm above the vegetation) to increase the soil temperature in the top 5 cm layer by *c.* 2.0°C above ambient temperature throughout the year, resembling warming scenarios for this area by 2050–2100 AD (Collins *et al.*, 2013), and infrared heaters increased air temperature by *c.* 0.8°C (air flow could take away lots of heat) on average at 30 cm from the soil surface. Infrared heaters can warm the plot with few edge and coverage effects (Kimball, 2005).

Four V-shaped transparent Panlite® sheet channels (covering 50% of the plot base area) were set above the infrared heaters in the decreased precipitation plots. The rainwater collected (50% of the ambient precipitation, which reflected the lower and upper bounds of mean annual variations in precipitation from the past 30 yr) from decreased precipitation plots was added to the increased precipitation plots manually after each precipitation event by spraying bottle. Dummy installations of heaters (no warming) and V-shaped transparent channels (with bottom opening) were used all together in control plots and other nontarget plots (plots without a certain treatment; e.g. V-shaped transparent channels with bottom opening were also used in experimental warming only plots) to control for the shade effects from installations themselves. In addition, 50 cm iron sheets were buried along the edge of each plot to prevent the underground movement of water and reduce surface run-off between plots (Lin *et al.*, 2016; Ma *et al.*, 2017; Liu *et al.*, 2018).

Sampling

Sampling was conducted in the sixth year of this long-term warming × precipitation experiment, which should be sufficient time for the effects of experimental treatments on foliar fungal diseases to emerge. In August (the peak of the growing season) 2016, we established a $0.5 \times 1.5\,m^2$ quadrat (with 12 $0.25 \times 0.25\,m^2$ grid cells) at the center of each plot to detect individual plant species percentage cover and species richness, since the percentage cover was more relevant to fungal pathogen transmission than the number of individuals was (Zhu *et al.*, 2000). Then we recorded the percentage cover of each plant species and species richness in each grid cell by visual estimation. Thus, the individual plant species percentage cover in each plot was calculated as the average relative cover of 12 grid cells, so the sum of total percentage cover in each plot might exceed 100% mainly because of the plant species overtopping, and the community-level species richness was calculated as total number of species across grid cells.

We observed a total of 54 plant species across 36 plots in our experiment in 2016 (Table S1). From this, the 38 most abundant plant species that constituted > 90% of the cover were measured for disease severity, although their appearance in every plot was not guaranteed. A previous study showed that experimental warming and decreased precipitation had negative effects on plant species richness and positive effects on plant species dominance, and experimental warming also lowered plant community biomass stability by reducing the degree of species asynchrony rather than plant species diversity over time (Ma *et al.*, 2017).

We recorded disease severity following the methods provided in Liu *et al.* (2016). In brief, we recorded disease severity (estimated visually using cards with digitized images of leaves of known disease severity) on leaf replicates. For the 38 most abundant plant species in our study site, we recorded leaf-level disease severity (percentage of the leaf area covered by fungal lesions) and visually assessed the presence of pathogen groups (Table S1) on 25 leaves, with five from each of five randomly selected stems, for each plant species in each plot (measured from the entire $2.2 \times 1.8\,m^2$ plot). For species with no more than five individuals or 25 leaves, we examined all the leaves available. Then we calculated population-level 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 5–10 samples of infected plant tissue per plant species in the same study site in August 2016 to confirm the groups of the pathogens (i.e. fungus-caused leaf-spot disease and blights, rusts, smuts, powdery mildews, and downy mildews) in the laboratory using an Olympus light microscope (see Table S1 for preliminary results); taxonomy mainly followed previous studies in this site (e.g. Zhang, 2009).

Measures of community pathogen load

We defined community pathogen load l as follows:

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

where S is the total number of host plant species and a_i is the percentage cover 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 (Mitchell *et al.*, 2002; Hantsch *et al.*, 2013, 2014; Liu *et al.*, 2016, 2017).

Within warming and altered precipitation treatments, communities might differ in composition (the identity of certain host plant species present), which could influence community pathogen load. To test how much variation in community pathogen load was explained by the variation in plant species composition between plots and to avoid any confounding effects of warming or altered precipitation, we defined a 'disease proneness index' (hereafter referred to as P_i) as the average severity

index V_i in the six control plots of the specific plant species i. We then calculated a 'community proneness index' (hereafter referred to as p) for each plot by calculating a host percentage cover-weighted average of the P_i over all plant species per plot (Mitchell *et al.*, 2003):

$$p = \frac{\sum_{i=1}^{S} a_i P_i}{\sum_{i=1}^{S} a_i}$$

p is the expected community pathogen load according to community constituent hosts, and a high p means a high community capacity to support diseases (Mitchell *et al.*, 2003). Although p is not exactly the same as community competence because of the possible changes in plasticity of host plants and pathogens under various treatments, it is rational to use community proneness index to evaluate community-mediated (indirect) effects (due to changes in plant community only) of global change on fungal diseases (Liu *et al.*, 2017).

Analysis

Population-level disease severity To evaluate the differences of each plant's V_i (disease severity indices of individual plant species) among different treatments, we employed Tukey's honest significant difference (HSD) test for multiple comparisons at the $P < 0.05$ level (Table S2). Then, general linear mixed-effects models were used to evaluate focal plant's percentage cover (Cover), warming (W), precipitation (P) and their interaction effects on each plant's V_i, with 'block' as random effect. These general linear mixed-effects models were built using the LMER function in the R package LME4, and the corresponding F- and P-values were derived from the ANOVA function.

For each plot, we calculated host plant species richness S_h and Shannon's evenness index H_h' for host plant community evenness using the function DIVERSITY in the R package VEGAN. Then we estimated plant community phylogenies based on *rbcL* and *matK* sequences, following the methods provided in Liu *et al.* (2015). In brief, we transformed the phylogeny to an ultrametric tree with the CHRONOS function in the APE package (Paradis *et al.*, 2004) and then calculated three measures of phylogenetic diversity in the PICANTE package (Kembel *et al.*, 2010): Faith's PD (hereafter PD), mean pairwise distance (MPD), and mean nearest taxonomic distance (MNTD). Among these three measures, PD was the best predictor for community pathogen load l, so we only included PD in the following analysis, given the collinearity between them.

To select the best variable in predicting V_i with a multimodel inference approach, we employed general linear mixed-effects models for disease severity index V_i of each plant species as a function of host plant species richness S_h, Shannon's evenness index for host community H_h', community proneness p, phylogenetic diversity PD, focal plant percentage cover (Cover), warming treatment W, altered precipitation P, and combination of warming treatment and altered precipitation (W × P). We had to avoid multiple variables appearing in the same model, given collinearity between those variables (Fig. S1). In particular, only 21 (see Table 1 for species name) out of 38 plant species (Table S3) could be analyzed (i.e. certain species found in at least nine plot-level replicates; otherwise, statistical parameters cannot be calculated) with the multimodel inference approach. We calculated the information-theoretic Akaike information criterion corrected for small sample sizes (AIC_c) using the AICC function to evaluate relative model support for each model we built. We calculated the likelihood-ratio based pseudo-R^2 (Pseudo R^2) as a measure of the model's goodness-of-fit for each model (Nakagawa & Schielzeth, 2013), and the pseudo-R^2 was derived from the R.SQUAREDLR function in the MUMIN package.

Community pathogen load To evaluate the differences of community pathogen load l among different treatments, we employed Tukey's HSD test for multiple comparisons at the $P < 0.05$ level. General linear mixed-effects models were used to evaluate warming, altered precipitation and their interaction effects on l (log-transformed to achieve normality), in which warming W and precipitation P were treated as fixed effects, and 'block' as random effect using the aforementioned methods. We also plotted the l relative to the different treatments using boxplots. In addition, general linear mixed-effects models were also used to evaluate warming, precipitation, and their interaction effects on the various community-level indices (S_h, H_h' and p) with the aforementioned methods.

Given the collinearity between various community-level indices (S_h, H_h', PD and p) and treatments (warming and altered precipitation) (Fig. S1), we also tested the relationships between the community pathogen load l and the various community-level indices in the six control plots. We set l as the response variable and the various community-level indices as the independent variable in a series of simple linear models. We used the information-theoretic evidence ratio $ER = wAIC_c[\text{slope model}] : wAIC_c[\text{intercept-only model}]$, where $wAIC_c$ is the AIC_c weight, as an index of relative support for the linear slope model vs the intercept-only (null) model; when $ER > 1.5$, we deemed that there was evidence to support the slope model (Burnham *et al.*, 2011). We also calculated the AIC_c, $wAIC_c$ based on the aforementioned methods for each linear model, and De, the percentage deviance explained in the response variable, as an index of each model's goodness-of-fit (Burnham *et al.*, 2011).

Factors influencing community pathogen load To identify the most parsimonious model (i.e. greatest explanatory power for the fewest number of predictors according to $wAIC_c$) between community pathogen load l and the predictors (S_h, H_h', p, PD, warming treatment, and altered precipitation), we constructed a series of generalized linear mixed-effects models with 'block' as random effect, and S_h, H_h', p, PD, warming, and precipitation as fixed effects using the GLMER function in the R package LME4. We calculated the Spearman rank–order correlation between indices we considered using the COR.TEST function. We validated the use of a 'gamma' family (link = 'log') for the modelled error distribution based on the normalized scores of standardized residual deviance. We calculated AIC_c and the likelihood-ratio based pseudo-R^2

(marginal R^2) as a measure of the model's goodness-of-fit for each model (Nakagawa & Schielzeth, 2013).

We constructed a piecewise structural equation model (piecewise SEM; Lefcheck, 2016) to test for the direct treatment effects and indirect effects through changes in the host plant communities on pathogen load l (Fig. S2). Before fitting the piecewise SEM, community pathogen load l was log-transformed to achieve normality (Laforest-Lapointe *et al.*, 2017). Our piecewise SEM comprised a series of linear mixed-effects models, with 'block' as random effect. The full piecewise SEM included the warming, altered precipitation, and their interaction (by dummy-coding the interaction) effects on four host plant community mediators (S_h, H_h', p, PD), and both the direct treatment effects and host plant community-mediated (indirect) effects on community pathogen load l.

In addition, we compared AIC_c (ΔAIC_c) between full and final (reduced) piecewise SEMs and calculated the standardized path coefficients (scaled by their mean and standard deviation) and corresponding significance (P values) for both full and final models. We evaluated the overall fit of both full and final models using the χ^2 test and AIC_c in the R package piecewiseSEM. All statistical analyses were performed using R v.2.15.1 (R Development Core Team, 2015).

Data accessibility

The data supporting this article are available to all interested researchers upon request.

Results

Population-level disease severity

Based on general linear mixed-effects models with focal plant percentage cover (Cover) as a covariate, warming treatment increased nine out of 38 most abundant plant species' V_is; by contrast, altered precipitation increased only one species' V_i (Tables 1, S3). For the multimodel inference approach, warming treatment (W) was the best predictor of V_i for eight out of 21 (38.10%) host plant species, whereas focal plant percentage cover (Cover) was the best predictor of V_i for only one (4.8%) species (Table S4).

Community pathogen load

Warming outweighed altered precipitation in driving community pathogen load l (Table 2). The community pathogen loads l of the warming, warming with decreased precipitation, and warming with increased precipitation treatments were higher than controls, and there were no significant differences of l between decreased precipitation, increased precipitation, and control based on Tukey's HSD test for multiple comparisons (Figs 1, S3). Warming treatment significantly increased l ($P < 0.001$, $F = 18.03$), and there was no significant effect of altered precipitation ($P = 0.322$, $F = 1.19$), whereas there was a marginally significant effect of their interaction on community pathogen load ($P = 0.098$, $F = 2.56$) based on general linear mixed-effects models (Table 2). In plots that were not warmed, increased precipitation and decreased precipitation resulted in 69% and 139% greater pathogen load respectively than in the control. However, warmed plots exhibited a nearly 201% increase in l, regardless of the precipitation treatment (Fig. S3).

At the community level, in the general linear mixed-effects models we built, both warming and altered precipitation decreased S_h, H_h', and PD, whereas there were no effects of the interaction between warming and precipitation on diversity measures (Table 2). In particular, plant species richness ranged from 26.33 ± 1.45 in warming × decreased precipitation plots to 35.83 ± 0.87 in increased precipitation plots, compared with 35.67 ± 0.92 in control plots. In addition, warming, but not precipitation, increased the community proneness index p ($P = 0.011$, $F = 7.54$; Table 2).

Factors influencing community pathogen load

In generalized linear mixed-effects models, only PD ($AIC_c = 145.858$, $wAIC_c = 0.343$, marginal $R^2 = 0.04$) provided a slightly better fit than the intercept-only (null) model ($AIC_c = 146.746$, $wAIC_c = 0.221$) in explaining community pathogen load l (Table 3; phase 1: community-mediated (indirect) effects). However, this pattern disappeared in the piecewise SEM analysis after accounting for the direct effects of warming and altered precipitation treatments (Fig. 2). Furthermore, there were no significant relationships between community pathogen load l and various community-level indices (S_h, H_h', PD, and p) based on simple linear models across six control plots (Fig. S4), indicating that community composition also has no significant effect on community pathogen load in control plots. These results confirmed that community-mediated (indirect) effects of global change factors played a relatively poor role in driving community pathogen load l. The most parsimonious treatment predictor was warming treatment ($AIC_c = 139.558$, $wAIC_c = 0.740$, marginal $R^2 = 0.197$), followed by '$\sim P + W + P \times W$' (marginal $R^2 = 0.393$), but altered precipitation was worse than the intercept-only (null) model, indicating experimental warming outweighs altered precipitation in driving l (Table 3; phase 2: treatment effects).

In the full piecewise SEM (standardized path coefficients are given in Table S5), warming and altered precipitation did not interactively affect either community pathogen load l or host plant community mediators (S_h, H_h', PD, and p) significantly (Fig. S5). We then reduced the full model by removing this interaction term, yielding the final (reduced) model (Fig. 2, Table S6), which adequately fitted the data: $\chi^2 = 5.84$, $df = 4$, $P = 0.211$, $AIC_c = 66.17$, $\Delta AIC_c = 9.68$ (compared with the full model), and explained *c.* 38% of the variance of l (marginal $R^2 = 0.38$). In the final model, warming treatment (standardized path coefficient $\beta = 0.652$, $P = 0.004$), rather than altered precipitation ($\beta = 0.065$, $P = 0.747$), increased l (Fig. 2).

Table 1 General linear mixed-effects model results for the effects of warming treatment, altered precipitation, and their interaction on disease severity index of each plant species.

	Warming		Precipitation		Warming × Precipitation	
Plant species	*F*	*P* value	*F*	*P* value	*F*	*P* value
Aster tataricus	29.40	**< 0.001**	0.08	0.784	2.46	0.137
Saussurea pulchra	6.26	**0.020**	0.56	0.464	0.76	0.392
Medicago ruthenica	0.41	0.528	2.83	0.106	6.29	**0.020**
Tibetia himalaica	8.92	**0.007**	1.06	0.316	1.38	0.254
Thermopsis lanceolata	1.65	0.265	0.00	0.951	0.35	0.582
Gentiana farreri	1.24	0.280	0.51	0.486	4.30	0.053
Gentiana pudica	0.05	0.826	0.23	0.638	0.35	0.567
Gentiana straminea	0.61	0.447	0.56	0.463	0.27	0.611
Elymus nutans	0.01	0.936	3.81	0.072	1.23	0.286
Festuca rubra	21.02	**< 0.001**	0.71	0.406	0.04	0.835
Helictotrichon tibeticum	1.95	0.177	5.85	**0.024**	5.38	**0.030**
Kobresia humilis	14.36	**< 0.001**	0.06	0.804	1.15	0.292
Koeleria litvinowii	0.03	0.873	0.00	0.962	0.01	0.906
Poa annua	5.29	**0.036**	0.57	0.463	6.18	**0.028**
Anemone obtusiloba	0.26	0.627	0.19	0.670	0.11	0.756
Thalictrum aquilegiifolium	7.49	**0.011**	0.31	0.584	0.35	0.560
Potentilla anserina	3.51	0.08	1.62	0.225	0.25	0.621
Potentilla bifurca	5.12	**0.033**	0.00	0.986	0.14	0.715
Potentilla nivea	1.61	0.220	0.03	0.872	0.72	0.408
Euphrasia regelii	5.56	**0.028**	0.00	0.978	0.23	0.635
Viola kunawarensis	0.79	0.399	0.00	0.986	0.85	0.385

Warming, precipitation, and their interaction were treated as fixed effects, focal plant percentage cover as covariate, and 'block' as random effect. Twenty-one of the relationships could be tested (i.e. sufficient degrees of freedom). The full table is shown in Supporting Information Table S3. Red and blue backgrounds indicate significant positive and negative effects, respectively. Significant effects ($P < 0.05$) are given in bold.

Table 2 General linear mixed-effects model results for the effects of warming treatment, altered precipitation, and their interaction on various community-level indices.

	Warming		Precipitation		Warming × Precipitation	
Variable	*F*	*P* value	*F*	*P* value	*F*	*P* value
l	18.03	**< 0.001**	1.19	0.322	2.56	0.098
S_h	26.71	**< 0.001**	10.29	**< 0.001**	1.92	0.168
H_h'	35.63	**< 0.001**	16.32	**< 0.001**	1.67	0.209
PD	6.05	**0.021**	4.35	**0.024**	0.70	0.505
p	7.54	**0.011**	0.58	0.567	1.17	0.328

Warming and precipitation were treated as fixed effects, and 'block' as random effect using the methods noted in the text (model sequence: '~ W + P + W : P + (1|Block)'). Shown are community pathogen load l, host plant species richness S_h, Shannon's evenness index for host community H_h', community proneness p, and phylogenetic diversity PD. Significant effects ($P < 0.05$) are given in bold.

Although both warming treatment and altered precipitation significantly changed S_h and H_h', with the warming treatment increasing community proneness p significantly ($\beta = 0.343$, $P = 0.010$), S_h ($\beta = -0.050$, $P = 0.895$), H_h' ($\beta = 0.294$, $P = 0.451$), PD ($\beta = -0.337$, $P = 0.115$), and p ($\beta = -0.127$, $P = 0.447$) did not significantly influence l after accounting for the direct effects of warming and altered precipitation treatments (Fig. 2). The full piecewise SEM and the final (reduced) model are qualitatively similar, which further revealed that warming affected l more than precipitation did and that the direct effects of warming treatment, rather than indirect effects of shifts in host plant community composition, drove l.

Discussion

By integrating information on community composition and foliar fungal diseases in a 6-yr factorial experiment in an alpine meadow, we provide strong empirical evidence that warming increases disease prevalence on nine individual plant species (population level) and entire host communities (community level). Further, we show that there is no significant effect of altered precipitation on community pathogen load. We conclude that experimental warming outweighs altered precipitation in driving foliar fungal diseases under the background of global change in Tibetan alpine meadows where our study site is located. Although both warming and altered precipitation significantly

changed plant community diversity, shifts in plant community under treatments play a relatively weak role in driving community pathogen load.

Warming has a larger effect than precipitation on fungal diseases

The effect of experimental warming was more intense than that of altered precipitation on fungal diseases. At the population level, experimental warming had a positive effect on disease severity for nine out of 21 species that could be tested based on general linear mixed-effects models. Specifically, warming increased diseases on three plants in Poaceae (i.e. *Festuca rubra*, *Kobresia humilis*, and *Poa annua*), with fungal leaf spot and rusts (*Puccinia* genus) increasing most obviously. At the host community level, warming plots always exhibited a higher community pathogen load than precipitation did (both decreased and increased precipitation). The increases in foliar fungal diseases with warming at both population (at least for a portion of the plant species) and community levels are consistent with previous studies from agricultural and natural ecosystems (e.g. Tapsoba & Wilson, 1997; Pfender & Vollmer, 1999; Harvell *et al.*, 2002; Roy *et al.*, 2004; Hannukkala *et al.*, 2007; Siebold & Tiedemann, 2013; Launay *et al.*, 2014).

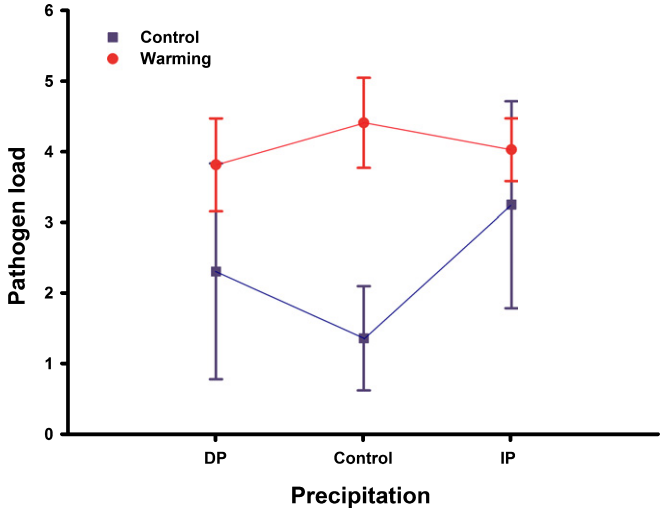

Fig. 1 Mean and standard deviation of community pathogen load by treatment. Warming consistently increased community pathogen load. Shown are mean ± 95% confidence interval. DP, decreased precipitation; IP, increased precipitation.

In contrast to warming, altered precipitation had no significant effect on community pathogen load, and it influenced the disease severity of only a very limited number of plant species (altered precipitation only significantly increases disease severity for one plant species based on general linear mixed-effects models). Our results contrast with several studies that have found effects of rainfall/moisture on fungal diseases in different systems (e.g. Madden, 1997; Clover *et al.*, 1999; McElrone et al., 2003; Strengbom *et al.*, 2006; Swinfield *et al.*, 2012; Prevéy & Seastedt, 2015). For instance, a previous study found that increased precipitation had a higher community pathogen load than communities with lower precipitation did (Strengbom *et al.*, 2006), likely because soil moisture and air moisture promote spore germination, which increases transmission (Woods *et al.*, 2005). Analogously, Prevéy & Seastedt (2015) found that increased winter precipitation decreased the abundance of invasive *Bromus tectorum* by increasing native fungal pathogen *Ustilago bullata*'s spore production, and thus transmission.

Table 3 Generalized linear mixed-effects model (family = gamma, link = log) results for community pathogen load *l* as a function of host plant species richness S_h, Shannon's evenness index for host community H_h', community proneness *p*, phylogenetic diversity PD, warming treatment W, altered precipitation P, and combination of warming treatment and altered precipitation W × P.

Model	LL	*k*	AIC_c	ΔAIC_c	$wAIC_c$	Marginal R^2
Phase 1: community-mediated effects						
~ PD	−68.284	3	145.858	0	0.343	0.040
~ 1 (null)	−69.998	2	146.746	0.878	0.221	−0.058
~ S_h	−68.792	3	146.875	1.007	0.207	0.012
~ H_h'	−69.142	3	147.573	1.705	0.146	−0.008
~ P	−69.725	3	148.740	2.872	0.082	−0.042
Phase 2: treatment effects						
~ W	−65.134	3	139.558	0	0.740	0.197
~ P + W + P × W	−60.251	5	141.836	2.277	0.237	0.393
~ 1 (null)	−69.998	2	146.746	7.188	0.020	−0.058
~ P	−69.462	3	150.924	11.365	0.003	−0.027

Phases 1 and 2 examine the relative support for all possible combinations (plant community and treatment predictors respectively) and the intercept-only (null) model. Shown are the estimated number of model parameters *k*, maximum log-likelihood LL, the information-theoretic Akaike's information criterion corrected for small samples AIC_c, change in AIC_c relative to the top-ranked model ΔAIC_c, AIC_c weight $wAIC_c$ (equal to model probability), and the likelihood-ratio-based pseudo-R^2 (marginal R^2) as a measure of the model's goodness-of-fit. Row outlines highlight single-predictor models.

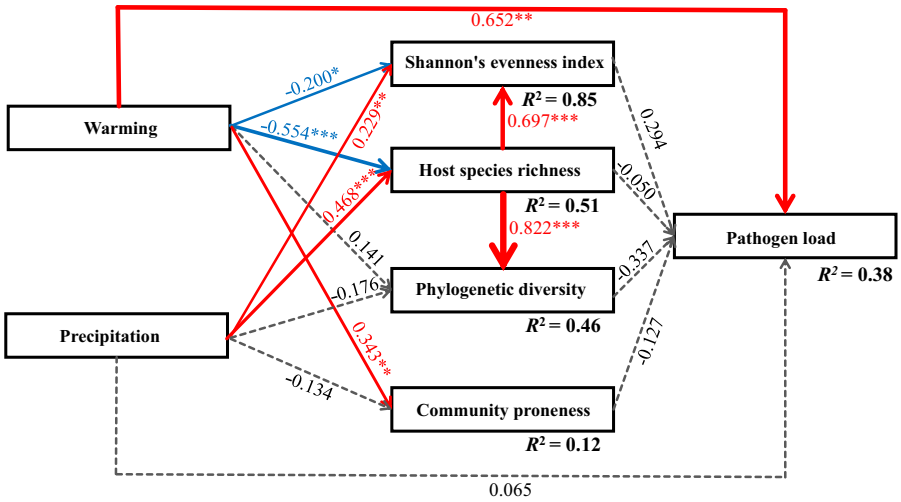

Fig. 2 The final (reduced) piecewise structural equation model results. The final (reduced) model adequately fitted the data: $\chi^2 = 5.84$, df = 4, $P = 0.211$, $AIC_c = 66.17$. Numbers on arrows are standardized path coefficients (scaled by their mean and standard deviation), and asterisks indicate statistical significance (***, $P < 0.001$; **, $P < 0.01$; *, $P < 0.05$). Red arrows, evidence for positive relationships; blue arrows, evidence for negative relationships; gray arrows, insufficient statistical evidence for path coefficients ($P > 0.05$). Width of the arrows shows the strength of the causal relationship, and R^2 is the marginal R^2, which indicates the variance explained by fixed effects in the mixed model.

In our system (alpine meadow), the mean annual precipitation is relatively high compared with other types of grassland, such as Inner Mongolia grassland (488 mm vs *c.* 200–300 mm) (Yang *et al.*, 2011). Hence, we speculate that the role of warming in the production of fungal spores and stimulated mycelium growth was stronger than that of precipitation in our system (Garrett *et al.*, 2006). We believe that temperature is a more limiting factor, with the extremely low temperatures (mean annual temperature is −1.1°C and growing season is only 4 months) (Zhao & Zhou, 1999), that limits pathogen fitness, whereas high temperatures limit plant resistance expression. In addition, generation time tends to be driven by temperature for most plant pathogens (Helfer, 2014), and having an increase of even a single additional generation can have large impacts on pathogen load. Overall, our results suggest that the effects of increased temperature or altered precipitation might depend on how those two variables currently act to affect pathogen and host plant.

The community-mediated effects of global change components on fungal diseases

In our system, our results suggest that increases in community pathogen load with warming are not mediated by shifts in host community diversity or composition. There were relatively small contributions of shifts in host percentage cover under various treatments on population-level disease severity, since there is no relationship between host percentage cover and corresponding V_i (disease severity indices of individual plant species) across all plots. We attribute the lack of predictive power of host percentage cover for fungal diseases to the extremely high plant species richness in our study site (*c.* 30–40 plant species in a 0.5 × 1.5 m^2 quadrat in control plots, and > 20 plant species under various treatments even after an experimental duration of 6 yr), where the percentage cover for each plant species is relatively low (96.30% of a total of 54 plant species had percentage cover estimates < 5%) (Ma *et al.*, 2017). Given that some (nearly 50%) of the fungal pathogens in our system are relatively specific to one genus (Zhang, 2009; Liu *et al.*, 2016), the physical isolation of the interception of spores by nonhosts may play an important role and overwhelm the effect of shifts in host density on fungal diseases (Zhu *et al.*, 2000).

Shifts in the identity of dominant plant species under treatments also play a relatively weak role in driving community pathogen load, mainly because there were no significant changes in percentage cover of the species that showed significant changes of V_i under warming or altered precipitation. This *post hoc* analysis provides strong evidence for the direct effects of warming treatment, rather than indirect effects of shifts in host plant community composition, driving community pathogen load. Plant species that showed significant compensatory increases (abundance of a certain plant species increases with total species richness decrease in treatment plots) in percentage cover under treatments were those with a low disease severity index V_i, such as *Medicago ruthenica* in Leguminosae. *M. ruthenica* has the second lowest V_i (0.27 ± 0.16) in control plots than any other plant in our system, and it did not exhibit any significant increases in V_i under warming and altered precipitation. On the contrary, for plant species with significant increases in fungal disease under our treatments (e.g. *F. rubra*, *K. humilis*, and *P. annua*), the relatively high disease severity could affect plant fitness, and thus competitive ability (Fisher *et al.*, 2012), resulting in no significant compensatory increase in percentage cover. Hence, we conclude that treatment-induced changes in community pathogen load are unlikely to have been driven by the responses of specific host populations. It is similarly unlikely that shifts in pathogen community composition contributed to observed increases in community pathogen load, as nearly 50% of the pathogens in our system are relative specialists (one genus) (Zhang, 2009; Liu *et al.*, 2016). Moreover, loss of species from warming might result in phylogenetically

overdispersed plant communities in alpine meadows (Liu *et al.*, 2016); therefore, we would expect lower pathogen spillover between closely related plants. Phylogenetically overdispersed plant communities might offset the effects of pathogen-mediated apparent competition to some extent, given the relatively narrow phylogenetic host range of pathogens in our system (Zhang, 2009; Liu *et al.*, 2016), and might promote the frequent positive interactions between plant species observed in alpine meadows like ours (Chu *et al.*, 2008; Lyu *et al.*, 2017).

Conclusions

Our results demonstrate that warming affects fungal diseases more than precipitation does in alpine meadows and suggests that increased temperature may additionally threaten ecosystem functions and services by increasing disease risk. We also provide new experimental evidence that the increases in disease burden on some plant species and for entire host communities are driven by global change directly rather than shifts in community composition. Our study expands our knowledge of the interface between community ecology, global change biology, and disease ecology, and clarifies the direct and indirect effects of warming on plant disease of terrestrial ecosystems under global change.

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Author contributions

J-SH and SZ conceived and designed the study. XL, ZM, and FC collected the data. XL and SZ analyzed the data. XL, MWC and SZ wrote the manuscript, and all authors approved the final manuscript.

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Supporting Information

Additional Supporting Information may be found online in the Supporting Information section at the end of the article:

Fig. S1 Correlation matrix of variables.

Fig. S2 *A priori* piecewise structural equation model.

Fig. S3 Community pathogen load varied in different warming and altered precipitation treatments.

Fig. S4 No evidence for a relationship between community pathogen load and various community-level indices in six control plots.

Fig. S5 The full piecewise structural equation model results.

Table S1 Host species list for the 54 species observed in our study, as well as observed diseases and pathogens, and corresponding disease proneness index.

Table S2 Disease severity index of 38 plant species we recorded between treatments.

Table S3 General linear mixed-effects model results for the effects of warming treatment, altered precipitation, and their interaction on disease severity index of 38 plant species.

Table S4 General linear mixed-effects model results for disease severity index of each plant species for 38 plant species with multi-model inference approach.

Table S5 Coefficient estimates from the full piecewise structure equation model.

Table S6 Coefficient estimates from the final (reduced) piecewise structure equation model.

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