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Intraspecific trait variation and changing life-history strategies explain host community disease risk along a temperature gradient

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Abstract:

Predicting how climate change will affect disease risk is complicated by the fact that changing environmental conditions can affect disease through direct and indirect effects. Species with fast-paced life-history strategies often amplify disease, and changing climate can modify life-history composition of communities thereby altering disease risk. However, individuals within a species can also respond to changing conditions with intraspecific trait variation. To test the effect of temperature, as well as inter- and intraspecifc trait variation on community disease risk, we measured foliar disease and specific leaf area (SLA; a proxy for life-history strategy) on >2500 host (plant) individuals in 199 communities across a 1101-meter elevational gradient in southeastern Switzerland. There was no direct effect of increasing temperature on disease. Instead, increasing temperature favored species with higher SLA, fast-paced life-history strategies. This effect was balanced by intraspecific variation in SLA: on average, host individuals expressed lower SLA with increasing temperature, and this effect was stronger among species adapted to warmer temperatures and lower latitudes. These results demonstrate how impacts of changing temperature on disease may depend on how temperature combines and interacts with host community structure, while indicating that evolutionary constraints can determine how these effects are manifested under global change.

Introduction

Infectious disease is strongly influenced by host community structure and abiotic conditions [1,2], both of which are changing at an unprecedented rate due to human activities [3]. Yet, predicting how these biotic and abiotic conditions interact to drive the emergence and spread of infectious disease remains a major research challenge, in part because several mechanisms can operate simultaneously, making it difficult to tease apart their relative contributions to realized disease risk. Thus, in order to predict disease under climate change will require an understanding of interactions among hosts, parasites, and the environment [4–6].

Recently, ecologists have proposed that shifting distributions of host species will be a key driver of disease risk under global change (including climate change) [7–9]. This proposal largely stems from two observations: First, particular characteristics of species (i.e., functional traits) influence how much those species contribute to disease risk in communities that they occupy (i.e., "host quality") [1,10–12]. Specifically, higher quality host species are often characterized by fast-growing, poorly defended tissues and short lifespans, [13–22]. Second, these functional trait values often underlie ecological and evolutionary tradeoffs related to host growth and defense, resource acquisition and allocation, and survival and reproduction (i.e., life history), resulting in higher levels of host competence (the contribution of a host species to disease transmission) [19,23–30]. The host species that stand to benefit the most from human disturbance (including climate change) often possess the same traits that make them more competent, high-quality hosts [11,31–33]. Although there has been some experimental evidence in support of these ideas [1,34,35], other empirical tests across environmental gradients have been inconclusive [2,36,37], suggesting that the traits of host species may be insufficient to predict how disease risk will shift under climate change.

While there is compelling evidence that human disturbance can shift host communities toward fast-pace-of-life species, thereby changing disease risk [33,38–41], how within-species trait variation responds to changing climate and how these jointly affect disease risk remains largely unknown. Within a species, individuals can show remarkable variation in their contribution to disease risk (i.e., host quality) [42–46] and in the expression of functional traits, generating intraspecific trait variation (ITV) [47–50]. The consequences of this ITV may be substantial, especially along environmental gradients, where ITV can be greater than trait variation between species [51,52]. For example, warming environmental conditions might favor

72 species characterized by a fast-paced life history strategy, increasing disease in warmer
73 conditions, but this effect may be overwhelmed if ITV is greater than variation between species
74 [2]. This suggests that incorporating ITV into trait-based models of disease might provide a
75 useful framework for predicting disease in the face of climate change.
76 How increasing temperature associated with climate change drives ITV may also depend
77 on how particular host species are adapted to their current abiotic conditions, thereby defining
78 which species will respond most strongly to a changing environment. There is some evidence
79 that, within a species, higher-altitude populations may exhibit lower levels of phenotypic
80 plasticity than populations from lower altitudes, suggesting that among temperate species,
81 adaptation to warmer environmental conditions could be associated with greater levels of
82 phenotypic plasticity [53] [but see 54]. This loss of phenotypic plasticity may be a consequence
83 of a stress response syndrome, relationships among plasticity and growth rate, or due to costs
84 associated with plasticity in extreme environments [53,55–57]. Scaling these observations up
85 across host species suggests that species adapted to warm environmental conditions might
86 exhibit higher levels of plasticity across environmental gradients than species adapted to cooler
87 environmental conditions [58] with broad implications for high elevation species to cope with
88 warming climate [59]. Yet, the implications of this variation in plasticity for ITV and disease are
89 rarely considered [60]. If warm-adapted species also exhibit greater levels of ITV than cold-
90 adapted species, this could amplify or offset the effect of changing host species composition
91 along environmental gradients (Fig 1). How ITV is expressed across species might therefore alter
92 the role that individual host species play in driving disease risk under climate change.
93 This study explores how intraspecific variation in specific leaf area (SLA), a proxy for
94 host pace-of-life, might combine with changes in the abundance and distribution of host species
95 to determine the relationship between host traits and disease under changing environmental
96 conditions. We explore this by measuring plant community trait variation and foliar fungal
97 disease along a 1101-meter elevation gradient in Southeastern Switzerland. Our results show that
98 increasing temperature favors species with high SLA and fast-paced life history strategies,
99 increasing disease, but that this effect is balanced by intraspecific trait variation in SLA in warm-
100 adapted host species. These results therefore underscore the pressing need to consider both inter-
101 and intraspecific variation in how host communities and their constituent members respond to

changing temperature, in order to predict how temperature will drive disease risk in a changing world.

Methods

Study system

To explore the relationship between host pace-of-life and disease in the context of changing temperature, we surveyed 199, 0.5 m-diameter vegetation communities, that were established as part of the Calanda Biodiversity Observatory [CBO; 2]. The CBO consists of five publicly owned meadows located along a 1101 m elevational gradient (648 m to 1749 m) below tree-line on the south-eastern slope of Mount Calanda (46°53′59.5″N 9°28′02.5″E) in the canton of Graubünden [2]. The soil in the area is calcareous with low water retention [61,62], and the five meadows are variable in size (roughly 8 to 40 Ha) and separated by forests. Meadows are maintained through grazing and mowing, a typical form of land use in the Swiss Alps [63].

The CBO consists of a nested set of observational units. Each meadow contains 2–7, .25 ha sites (n = 22 sites), each of which contains a grid of nine evenly spaced, 4 m^2 large-plots, with the exception of one site (I3), which is 100 m x 25 m and contains 10 large plots due to its shape (n = 199 large plots). In each site, large plots are arranged in a grid with the center of each plot separated by at least 20 m distance from its nearest neighbor. One 50 cm-diameter, round small plot was placed at random inside of each large plot to serve as the unit of observation used in this study.

Quantification of plant species abundance

In July 2020, we recorded the identity and visually quantified the percent cover of all plant taxa in each 50 cm-diameter small plot following a modified Daubenmire method (n = 199) [2], in which two researchers searched the entire plot for all rooted vascular plants present in the plot, before jointly estimating the total percent cover of each species. Plant individuals were only included in the survey if they were rooted in the small plot. The survey started at the lowest elevation and continued higher in order to survey the meadows approximately at the same phase of the growing season in relation to one another.

Quantification of disease

In August 2020, a survey of foliar disease severity was carried out by visually estimating the percent leaf area damaged on one mature non-senescing leaf of twenty randomly selected host individuals (n = 3980 host leaves across 199 small plots) following the plant pathogen and invertebrate herbivory protocol in the ClimEx Handbook [64]. Host individuals were selected by placing a grid of 20 equally spaced grill sticks into the ground, with every stick having a distance of 10 cm to its nearest neighbor and then sampling the 20 plant individuals that were most touching the sticks. The survey was carried out on leaves, because symptoms are highly visible and easily grouped into parasite types on leaves. On each leaf, we estimated the leaf area (%) that was covered by disease symptoms. Disease was then quantified for each small plot using the community weighted mean leaf area damaged by parasites (i.e., parasite community load) [2,65], calculated as the mean leaf area damaged by parasites on a plant species in a plot, multiplied by the relative abundance of that plant species from the vegetation survey, and then summed across all plant species in the plot.

Quantification of community pace-of-life

Community pace-of-life was calculated using a single trait – Specific Leaf Area (SLA) – which is often highly correlated with other metrics of host life-history strategy along natural environmental gradients [14,30,35,36,66], including along the elevation gradient of the CBO [2]. We quantified SLA at two levels: at the host species level and using local measurements in each small plot.

At the host species level, SLA was quantified using the TRY database [67]. Unknown taxa that could be identified to the genus level were assigned genus-level estimates for SLA, by taking the mean of the trait value for all members of that genus that had been observed on Mount Calanda during extensive vegetation surveys. Genus-level estimates were not substantially different, on average from species-level estimates with respect to locally measured values (Fig S1).

To test the assumption that SLA is a good proxy for host species pace-of-life, we also constructed a single functional trait axis representing a hosts' pace-of-life following Halliday et

al [2]. Briefly, we constructed a single axis representing covariation in the functional traits associated with the worldwide leaf economics spectrum [30] by performing full-information maximum-likelihood factor analysis on five functional traits extracted from the TRY database (leaf chlorophyll content, leaf lifespan, leaf nitrogen content, leaf phosphorus content, and specific leaf area) using the umxEFA function in r-package umx [68]. To facilitate comparisons, we transformed the factor score into units of SLA by first multiplying each species' factor score by the square-root sum of squares of the factor loadings and then multiplying this value by the standard deviation of SLA and adding the resulting value to the mean of SLA. Supporting the assumption that SLA is a good proxy for host species pace-of-life, this pace-of-life functional trait axis was highly correlated with host species SLA, and results using species-level trait estimates were qualitatively similar regardless of whether we used a species' position along this trait axis or SLA as a predictor (Appendix A).

Local SLA measurements were obtained on a subsample of plants that were surveyed during the disease survey. This included one individual for each unique species that was touching a grill stick, and no fewer than 10 individuals per small plot (n = 2537). SLA was then recorded on one mature, non-senescing leaf on each individual by photographing that leaf in the field using standard lighting, then immediately drying the leaf using silica gel. Silica gel was replaced regularly until the leaves reached a stable mass, at which point the leaf's dry mass was measured using an analytical balance. Specific leaf area was computed as the ratio of leaf area to dry mass.

We calculated a single value for each SLA measurement in a small plot (n = 199) using the community-weighted mean of SLA (hereafter community SLA). The community weighted mean (CWM) was calculated as:

$$CWM = \sum_{i=1}^{Nsp} p_i \, x_i$$

where Nsp is the number of taxa within a plot with a SLA trait value in the dataset, p_i is the relative abundance of taxon, *i*, in the plot, and x_i is SLA value for taxon, *i* in that plot or at the species level.

Quantification of temperature

As described in Halliday et al [2] soil temperature (6 cm below the soil surface), soil surface temperature, air temperature (12 cm above the soil surface), and soil volumetric moisture content were recorded at 15 minute intervals in the central large plot of each site during the time between grazing activities, (n = 22) using a TOMST-4 datalogger [69]. In the CBO, mean soil, soil surface, and air temperature all strongly and consistently decrease with increasing elevation [2]. Here, we report analyses using soil-surface temperature following Halliday et al [2].

Statistical analysis

All statistical analyses were performed in R version 3.5.2 [70]. We tested how increasing temperature associated with lower elevations influenced community SLA by fitting two linear mixed models with an identity link and Gaussian likelihoods using the lme function in the nlme package [71]. CWM SLA (either using local measurements or species means) was included in each model as the response variable and mean soil surface temperature as the explanatory variable. In order to meet assumptions of normality and homoscedasticity, we added an identity variance structure (varIdent function) for each site, which based on visual inspection of residuals of each model, exhibited considerable heteroscedasticity [71,72]. Each model included sites and meadows as nested random intercepts to account for non-independence among observations due to the sampling design of the CBO.

We tested how increasing temperature affects the expression of SLA within a species (i.e., ITV) using 93 species with at least five observations across the elevational gradient. To do this, we first estimated the Pearson correlation coefficient for each species SLA in response to temperature along the gradient. We converted the Pearson correlation coefficient into a standardized effect size by computing the Fisher's z-transformed correlation coefficient using the escalc function in the metafor package [73], and then tested whether SLA commonly increased or decreased within a species with increasing temperature by fitting an intercept-only model using the rma.mv function in the metafor package. This approach is similar to performing a meta-analysis on our own field collected data, thereby allowing us to account for variation in sampling intensity, effect size, and variance across these 93 different host species in the CBO. To test whether characteristics of species related to their habitat distribution might influence the direction and magnitude of intraspecific variation in SLA, we fit two additional models with continuous predictors of the Fisher's z-transformed correlation coefficient using the rma.mv

function. These models are the equivalent of meta-regressions performed on our own field collected data. The first model included the northern and southern range limit of each species, which was available for 56 species in the ClimPlant database, a database of estimated realized climatic niches of vascular plants based on climatic tolerances of European plant species [74]. The second model included the minimum and maximum elevation limit of a species observed in the CBO (n = 93 species).

We then tested how changing host community structure and shifting ITV among species that are adapted to particular environmental conditions could synergistically or antagonistically affect the distribution of life-history strategies in host communities. To test this, we first extracted the Ellenberg indicator values (i.e., indicator values for species' ecological optima) [75] for temperature adaptation for each species in the CBO using the Flora Helvetica [76], and then fit two linear mixed-effects models with data from every individual that was surveyed along the gradient (n = 2333 after excluding 200 individuals that could not be assigned an Ellenberg indicator value for temperature), with an identity link and Gaussian likelihoods using the lme function. Each model included temperature and the Ellenberg indicator value for temperature-adaptation of a species as interactive explanatory variables, and included species, plots, sites, and meadows as nested random intercepts to account for non-independence among observations due to the sampling design of the CBO. We used Ellenberg indicator values in this analysis because they were estimated independent of host distributions in the CBO and had greater species coverage than range-limits in the ClimPlant database, allowing us to include in this analysis nearly every measurement in the CBO. The first model included SLA as the response variable, and included random slopes for each species, because species exhibited different intraspecific responses to increasing temperature, while the second model included the relative abundance of each species as the response variable.

Finally, we tested how temperature and host community SLA affected disease by fitting a mixed model with square-root transformed community parasite load as the response, and temperature, host CWM SLA using local measurements, and host CWM SLA using species-level estimates as explanatory variables. To estimate whether the effect of host community SLA depends on temperature, we also included in the model the pairwise interactions between each measure of host community SLA and temperature as additional explanatory variables. To aid the interpretation of temperature effects in the model, we mean-centered soil-surface temperature, so

that mean temperature was used as the reference value for interpreting the other variables' independent effects. Like models of SLA, we added an identity variance structure for each site, and included sites and meadows as nested random intercepts to account for non-independence among observations due to the sampling design of the CBO. To avoid problematic correlations among parameter estimates, non-significant interactions among fixed-effects were removed from models using likelihood ratio tests [following 72,77], and overall impacts of variables were determined by evaluating the parameter estimates from the reduced model.

Results

First, we tested the hypothesis that increasing temperature could alter the distribution of host species based on specific leaf area, using species-level trait data from the TRY database, leveraging the strong and consistent relationship between increasing elevation and temperature (Fig 2a). Overall, increasing temperature marginally changed the species composition of host communities, favoring species characterized by higher SLA ($p = 0.045$, $R^2 = 0.14$, Fig 2b; Fig S2a), supporting the hypothesis that warming environmental temperatures might support higher quality host species.

We next tested the hypothesis that increasing temperature might also alter the expression of SLA within a species using 93 species with at least five observations across the elevation gradient. The model tested whether SLA commonly increased or decreased with increasing temperature within species, after accounting for differences in sampling intensity across host species within the study. In this model, individuals within a species generally exhibited lower SLA with increasing temperature ($p < 0.0001$; Fig 3a; Fig S3). Thus, even though increasing temperature favored species characterized by a faster pace-of-life, individuals within a species often exhibited lower overall levels of SLA.

Although increasing temperature commonly reduced SLA within a species, many species either did not respond to increasing temperature or responded in the opposite direction (Fig S2c; Fig S3). We therefore next tested whether characteristics of species related to their habitat distribution might influence the direction and magnitude of intraspecific variation in SLA, by assessing whether (a) the northern or southern range limit of a species and (b) the high or low-elevation limit of a species affected SLA responses to increasing temperature. We hypothesized that species adapted to warmer climates might experience stronger intraspecific responses to

284 increasing temperature. Consistent with this hypothesis, the strongest intraspecific responses of
285 SLA to increasing temperature were observed among species that were able to colonize the
286 lowest elevation meadows ($p=0.013$; Fig 3b), and species that were closest to their northern
287 range limit ($p = 0.015$; Fig 3c).
288 We then tested whether the combination of changing host community structure and
289 shifting ITV among species that are adapted to particular environmental conditions (using
290 Ellenberg indicator values for temperature) could synergistically or antagonistically affect the
291 distribution of life-history strategies in host communities (measured as CWM SLA). In this
292 model, SLA declined with increasing temperature, but only among species adapted to warmer
293 environmental conditions (i.e., with high Ellenberg indicator values; $p < 0.0001$; Fig 4a). In
294 contrast, the relative abundance of species adapted to cold environmental conditions declined
295 with increasing temperature ($p = 0.003$; Fig 4b). Consequently, changes in relative abundance
296 and the expression of specific leaf area counterbalanced one another, resulting in no net change
297 of community-weighted-mean SLA across the environmental gradient ($p = 0.35$; Fig 4c; Fig
298 S2b).
299 Finally, we tested how temperature, host community-weighted-mean SLA (using both
300 local measurements including ITV and species-level estimates excluding ITV), and their
301 interaction affected disease. Consistent with past studies, there was some evidence that
302 communities dominated by species characterized by high SLA (e.g., with fast-paced life history
303 strategies), experienced more disease, but only at high temperature (full model: $p = 0.058$;
304 reduced model: $p = 0.035$). In contrast, there was strong evidence for a consistent increase in
305 disease in communities dominated by hosts expressing higher levels of SLA in their local
306 environment (full model: $p = 0.0002$, reduced model: $p = 0.0003$), and this effect was
307 independent of increasing temperature (full model: $p = 0.60$, marginal $R^2 = 0.14$, conditional R^2
308 $= 0.39$; reduced model: $p = 0.086$, marginal $R^2 = 0.13$, conditional $R^2 = 0.40$; Table S1; Fig 5).
309 All together, these results reveal a complex relationship between environmental temperature,
310 community-level life history strategies, the expression of key functional traits, and disease risk.
311 Increasing temperature favored host species with faster life history strategies, which increased
312 disease risk, but only in warmer environments. However, this effect was balanced by
313 intraspecific trait variation in warm-adapted species, resulting in no net effect of increasing
314 temperature on local measurements of host community pace-of-life (Fig 4c) or disease (Fig S4),

despite a strong positive relationship between host pace-of-life and disease (Fig 5). These results highlight the importance of both inter- and intraspecific variation in driving host community responses to changing temperature and their impacts on disease risk.

Discussion

Climate change is increasing disease risk, with that effect only expected to intensify over time [7]. Yet, predicting how climate change will affect disease risk in host communities is complicated by the fact that changing environmental conditions can affect disease through a wide variety of direct and indirect effects [1,2,37,78–80]. This phenomenon creates challenges for predicting the impacts of climate change on infectious disease: species distributions are changing in response to temperature, and the effect of individual species on disease is shifting, but we lack a framework for integrating these ideas. This study leverages a fundamental concept from disease ecology, within-species host heterogeneity [81], to address this challenge (Fig. 1). After incorporating heterogeneity among individuals within a species, we find strong evidence of a consistent relationship between host traits and disease across environmental conditions. However, the effect of temperature on the distributions of species and the traits that individuals express countered one another (e.g., Fig 1a), resulting in no net effect of increasing temperature on disease risk in host communities. Thus, only by integrating shifts in host community structure with intraspecific changes in the expression of functional traits, were we able to unravel the influence of changing climate on disease risk across the 1101-meter elevation gradient.

In temperate climates, increasing temperature is often expected to increase the risk of infection by foliar parasites by increasing parasite growth and reproduction, [82–86], overwintering success [87,88], or by extending the duration of the growing season [89] and thereby allowing parasites to produce more generations during a single season [85]. However, in contrast with a past survey of disease along this environmental gradient [2], we did not detect a net effect of increasing temperature on disease in this survey. These results suggest that the effect of climate on disease might be sensitive to seasonal variation in biotic or abiotic environmental conditions, highlighting the need for long-term data on biotic and abiotic environmental conditions along climatic gradients [90].

Our results also highlight a pressing need to integrate information about the abiotic environment, species distributions, and phenotypic plasticity in order to predict disease risk

under climate change. Although a warming climate can directly influence disease risk in host communities [91], a warming climate can also indirectly influence disease risk by altering the composition of host or vector communities required for sustained parasite transmission [79,91,92]. ITV can further exacerbate this problem, and it can be particularly pronounced under variable environmental conditions that alter trait covariation, and hence limit the ability of raw trait values to predict infection [36]. Thus, traits of host species that are associated with low host competence in one environment could increase host competence under environmental change, due to ITV. Our results suggest that how ITV translates into disease risk might further depend on host adaptation to environmental conditions, with warm-adapted species showing stronger patterns of ITV than cold-adapted species.

In this study, we used SLA as a proxy for host pace-of-life and disease risk, but we did not measure covariance among SLA, other metrics of host-pace-of-life, and disease risk in the field. There is compelling evidence from the published literature that SLA is a useful proxy for host pace-of-life and disease risk at the host species level [14,15,34], but few studies have explored the link between SLA, pace-of-life, and disease within species. Studies of viral infection in California grasses suggest that SLA is a good predictor of host susceptibility across environmental conditions [15,22,36]. However, the relationship between SLA and host pace-of-life is more complicated. In one study, SLA and other traits became uncoupled when plants were grown under novel resource conditions, suggesting a breakdown in the host pace-of-life syndrome [36], while two other studies in the very same system consistently observed covariance among traits across resource conditions [15,22]. These results suggest that SLA might be a good proxy for how hosts contribute to disease risk (i.e., host quality), but that the relationship between SLA and host pace-of-life can change, depending on environmental conditions. Across scales and study systems, the degree to which particular traits and host quality correlate with each other remains an active research question [21,93], as do questions related to the generality of the pace-of-life syndrome itself [94]. In plants, a few studies have explored patterns of covariance in the traits underlying host pace-of-life across environmental gradients, albeit without the link to infectious disease risk, often coming to contradictory conclusions about whether interspecific relationships among traits are consistent with intraspecific relationships [95–97]. Consequently, even though local measurements of SLA consistently predicted disease risk in host communities, we cannot rule out the possibility that intraspecific variation in SLA

occurred independent of host pace-of-life in this study. Addressing this research gap will require future studies to link multiple traits associated with pace-of-life to disease across environmental gradients, both within and across species.

Across the studied elevation gradient, warmer temperatures favored host species with higher SLA and faster life-history strategies, consistent with past studies [e.g., 98], and communities dominated by faster-paced host species experienced more disease, but only at high temperature. This result is consistent with a past survey that was conducted in a different year along this same elevational gradient [2], suggesting that the effect of host community pace-of-life on disease may depend on environmental context. We hypothesized that ITV might explain the statistical interaction between host community pace of life and disease observed in that study, and found evidence to support that hypothesis. Although warmer temperatures favored host species with higher SLA, intraspecific changes in SLA among warm-adapted host species balanced this effect (e.g., Fig 1a). Specifically, among warm-adapted species, SLA commonly declined with increasing temperature associated with lower elevations. This result is in contrast with a recent meta-analysis that found the opposite effect across a global species pool [99]. Instead, our results suggest that ITV responses to environmental gradients may depend on other characteristics of species or study systems [51,52]. In this system, patterns of ITV were linked to species range-limits and adaptation to warm environmental temperatures, under the expectation that species adapted to warmer conditions and species closer to the margins of their range limits would show higher levels of phenotypic plasticity and consequently ITV [53,58,100]. These results therefore suggest that how species evolved in the face of past environmental constraints can influence how those species will contribute to disease in changing environments. Identifying mechanisms of these contrasting patterns of intra- and interspecific trait variation is an exciting avenue for future research.

Our results suggest that patterns of disease can be strongly influenced by inter- and intraspecific variation in SLA, but relationships between SLA and biotic interactions are being increasingly recognized as multidirectional: not only can host pace-of-life influence biotic interactions, but biotic interactions can reciprocally influence intraspecific variation in host pace-of-life. For example, across 101 species embedded in alpine communities, SLA decreased with increasing herbivory [101], within eight common tundra plant species, SLA increased when mammalian herbivory was excluded [102], and across twenty species in a biodiversity

manipulation, fungicide application reduced SLA within species [103]. Together with the results of this study, these experimental results highlight the potential for important feedbacks between ITV and disease risk in host communities experiencing climate change. These feedbacks, in turn, could alter relationships between functional traits and host competence over evolutionary timescales. For example, a reduction in infection severity with cooling temperatures could weaken the importance of investment in disease resistance [104,105], so that host species could still form trade-offs in pace-of-life for growth and survival, but the link between pace-of-life and disease severity could weaken. Future studies could explore these complex feedbacks using manipulative experiments (like experimental fungicide applications) across environmental gradients.

Together, the results of this study highlight the value of integrating among- and within-host differences in order to explain how environmental gradients shape disease risk [21,22,36]. Specifically, in this study, the strongest predictor of disease was host SLA, rather than the abiotic environment, and changes in temperature along the elevation gradient only influenced disease through its relationship with SLA. These results are consistent with a growing body of literature suggesting that the impacts of changing temperature on disease may depend on how temperature combines and interacts with the structure of host communities [2,78,79,91], while indicating that evolutionary constraints on individual host species can determine how these effects are manifested. These results therefore suggest that predicting how global change will influence disease may depend on complex relationships among global change drivers, the structure of host communities, and the evolutionary and ecological processes that affect individual hosts within those communities.

Figure Legends:

Figure 1. Hypothesized pathways through which changing host species composition and intraspecific trait variation can mediate changing disease risk across a temperature gradient. a) High levels of intraspecific trait variation (ITV) in warm-adapted species offset changes due to shifting species composition, resulting in similar levels of community functional traits and disease across the gradient. b) There is no ITV, so only changes in species composition alter disease. c) High levels of ITV in warm-adapted species amplify changes in community functional traits and disease due to shifting species composition. The colors represent an individual's placement along a functional trait axis from fast to slow. Beside the color bar, species are arranged according to their mean trait value and colored according to the range of traits that each species can express. Black points represent disease severity.

Figure 2. Relationships between elevation, mean soil surface temperature, and community weighted mean (CWM) specific leaf area (SLA). a) Relationship between elevation and mean-soil-surface temperature in the CBO. b) Results from a model testing how CWM SLA, calculated using species mean traits, is influenced by increasing mean soil surface temperature. Lines are model-estimated means and ribbons are model-estimated 95% confidence intervals. Open circles in (a) are the raw data for individual sites. Open circles in (b) are the raw data for individual host communities (i.e., small plots). Soil surface temperature and elevation are collinear ($r = -0.95$), and CWM SLA calculated using species means increases with increasing soil surface temperature.

Figure 3. Results from the analysis testing whether increasing temperature consistently affects intraspecific changes in specific leaf area (SLA). The y-axis is a standardized effect size (Fisher's Z), with values below zero corresponding to a negative effect of increasing temperature on SLA. Solid points and error bars (a) and solid lines and ribbons (b & c) represent model-estimated means and 95% confidence intervals. Raw data are represented in Figure S3. a) Results from an intercept-only model. b) Results from a model exploring lower-elevation limits of a species within the CBO (omitting the non-significant effect of higher-elevation limits). c) Results from a model exploring northern range-limits of a species (omitting the non-significant effect of

southern range-limits). On average, SLA becomes lower (i.e., leaves become thicker, more well defended) as temperature increases, with that effect being stronger for species able to colonize the lowest elevation meadows and for species with lower-latitude northern range limits.

Figure 4. Results from models comparing how (a) changing values of specific leaf area (SLA), (b) changing relative abundances of species, and (c) community weighted mean (CWM) SLA calculated using local trait measurements are influenced by increasing temperature. Lines are model-estimated means and ribbons are model-estimated 95% confidence intervals, with colors representing an aggregate of Ellenberg indicator scores for species thermal preference from the Flora Helvetica (Alpine = 1, 2, and 2+; Subalpine = 3 and 3+; Montane = 4 and 4+). Open circles in (c) are the raw data for individual host communities (i.e., small plots). As temperature increases, the average SLA of montane species declines and the relative abundance of alpine species declines, resulting in no net change in CWM SLA across the gradient.

Figure 5. Results from the models testing how community weighted mean (CWM) specific leaf area (SLA) and soil surface temperature jointly influence community-level disease. (a) Results from a model fit using species-level means to calculate CWM SLA. (b) Results from a model using local estimates to calculate CWM SLA. Lines represent the model-estimated effect of CWM SLA estimated at one standard deviation above the mean (orange), one standard deviation below the mean (purple), and at the mean temperature (fuchsia). Points represent the raw data colored by soil surface temperature of the site. Increasing CWM SLA increased disease, but only at high temperature when CWM SLA was calculated using species means (a). In contrast, increasing CWM SLA consistently increased disease when CWM SLA was calculated using local estimates (b).

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