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The effect of warming on seagrass wasting disease depends on host genotypic identity and diversity

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Open Research: The raw sequence reads of the leaf microbial communities are archived with the National Center for Biotechnology Information (NCBI) in Bioproject PRJNA716355 at https://www.ncbi.nlm.nih.gov/bioproject/PRJNA716355. All other data associated with this manuscript are permanently archived with the National Science Foundation Biological and Chemical Oceanography Data Management Office (BCO-DMO) Schenck, et al. (2022a,b,c,d) at https://doi.org/10.26008/1912/bco-dmo.883070.1; https://doi.org/10.26008/1912/bco-dmo.883055.1; https://doi.org/10.26008/1912/bco-dmo.879749.1; and https://doi.org/10.26008/1912/bco-dmo.883037.1, respectively. Complete code for microbial analyses (Kardish, 2022) is available in Zenodo at https://doi.org/10.5281/zenodo.7433397. Complete code for all other analyses (Schenck, 2022) is available in Zenodo at https://doi.org/10.5281/zenodo.7129648.

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Abstract

Temperature increases due to climate change have affected the distribution and severity of diseases in natural systems, causing outbreaks that can decimate host populations. Host identity, diversity, and associated microbiome can affect host responses to both infection and temperature, but little is known about how they could function as important mediators of disease in altered thermal environments. We conducted an 8-week warming experiment to test the independent and interactive effects of warming, host genotypic identity, and host genotypic diversity on the prevalence and intensity of infections of seagrass (*Zostera marina*) by the wasting disease parasite (*Labyrinthula zosterae*). At elevated temperature, we found genotypically diverse host assemblages had reduced infection intensity, but not reduced prevalence, relative to less diverse assemblages. This dilution effect on parasite intensity was the result of both host composition effects as well as emergent properties of biodiversity. In contrast to the benefits of genotypic diversity under warming, diversity actually increased parasite intensity slightly in ambient temperatures. We found mixed support for the hypothesis that a growth-defense tradeoff contributes to elevated disease intensity under warming. Changes in the abundance (but not composition) of a few taxa in the host microbiome were correlated with genotype-specific responses to wasting disease infections under warming, consistent with the emerging evidence linking changes in the host microbiome to the outcome of host-parasite interactions. This work emphasizes the context dependence of biodiversity-disease relationships and highlights the potential importance of interactions among biodiversity loss, climate change, and disease outbreaks in a key foundation species.

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Keywords: climate change; host-parasite; *Labyrinthula zosterae*; microbiome; seagrass; temperature; wasting disease; *Zostera marina*

Introduction

As a result of climate change, many host-parasite interactions are occurring in modified environments (Jones et al. 2008, Harvell et al. 2009). Temperature change, in particular, has been implicated as a causal factor in numerous disease outbreaks (Harvell et al. 2002, Rohr & Raffel 2010). Changes to the thermal environment may induce trait changes in the host that leaves them more susceptible to parasite infection (Burge et al. 2013, Burge et al. 2014). For example, common aspen (*Populus tremula*) experiencing elevated temperatures are more susceptible to disease because of temperature-triggered investment in growth at the expense of parasite defenses (i.e., the growth-defense tradeoff; Randriamanana et al. 2015, Sobuj et al. 2018). Beyond the traits of the host itself, host-associated microbes can either facilitate or inhibit parasites, with the direction and magnitude of this effect varying with temperature (Peixoto et al. 2017, Zaneveld et al. 2017). Temperature change could also contribute to disease outbreaks because of the predicted discrepancy between the broader thermal tolerances of parasites relative to their larger host organisms, leading to greater susceptibility of hosts when conditions shift from their thermal optima (i.e., the thermal mismatch hypothesis; Cohen et al. 2017). To effectively manage diseases in this era of global change, there is a pressing need to understand interactions among the thermal environment, hosts, host microbiomes, and parasites (Rohr et al. 2011, Sullivan et al. 2018).

Because of variation both within and among species in thermal tolerance (Deutsch et al. 2008, Dubois et al. 2019), host identity is a potentially important contributor to disease outbreaks in altered thermal environments. For example, variation in thermal tolerance across amphibian

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host species is thought to contribute to disease outbreaks of chytrid fungus that occur due to thermal mismatch in both cool and warm conditions (Rohr et al. 2011). Along with host identity, host interspecific and intraspecific genetic diversity have documented effects on the host response to thermal stress, with higher diversity necessary to sustain population, community, and ecosystem processes as temperatures changes (Reusch et al. 2005, Perkins et al. 2015, DuBois et al. 2020). In addition, host interspecific and intraspecific genetic composition and diversity can strongly influence disease risk (Johnson et al. 2015, Riess & Drinkwater 2018, Ekroth et al. 2019, Rohr et al. 2020). Whether and how host diversity interacts with environmental change to influence disease risk is an important question, because both biodiversity and climate are changing simultaneously in many ecosystems (Bellard et al. 2012, Urban et al. 2016).

The seagrass *Zostera marina* (eelgrass) provides an ideal system to test how host diversity and environmental change interact to determine disease risk. Eelgrass is a key species in estuaries throughout the northern hemisphere, where it provides habitat for numerous invertebrates, fishes, and birds, enhances primary productivity, and contributes to nutrient cycling and sediment stabilization (Williams & Heck 2001). Eelgrass was nearly extirpated on the Atlantic coast of North America due to an outbreak of wasting disease in the 1930s (den Hartog 1987), and chronic infections and isolated outbreaks continue to occur throughout the current eelgrass range (Bockelmann et al. 2013, Groner et al. 2016). Wasting disease is caused by infections of the parasitic marine protist *Labyrinthula zosterae* (Muehlstein et al. 1991). Temperature change has long been linked to increases in wasting disease in eelgrass (Rasmussen 1977, Kaldy 2014; Dawkins et al. 2018, Brakel et al. 2019, Graham et al. 2021, Groner et al. 2021). In addition, eelgrass has genetic variation for key functional traits related to thermal tolerance (Reusch et al. 2005, Reynolds et al. 2016, DuBois et al. 2021, 2022), as well as traits

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such as the production of chemical defenses and the relative investment in growth vs. storage that can affect plant carbon balance (Hughes et al. 2009; Abbott et al. 2018; DuBois et al. 2019, 2020). Additionally, eelgrass microbiomes vary as a function of temperature and disease status (Beatty et al. in. revision), and potentially plant genotype (J. Stachowicz, unpublished data), suggesting a potential direct or indirect role for microbiota in host-parasite dynamics. All of these processes potentially affect wasting disease prevalence and/or intensity (Groner et al. 2016, Zidorn 2016), suggesting that individual genotypes may vary in disease susceptibility. Further, eelgrass genetic and genotypic diversity increase plant density and biomass (Williams 2001, Hughes & Stachowicz 2004, 2011) and this effect becomes more pronounced under temperature stress (Reusch et al. 2005, DuBois et al. 2020). This benefit of diversity under temperature stress could lead to reduced disease susceptibility, or the higher biomass of more diverse assemblages could increase disease transmission and risk. Thus, it is an open question how host diversity, microbiome community, warming, and disease may interact in this system.

To test whether eelgrass genotypic diversity could mediate disease in a warming climate, we experimentally manipulated seawater temperature and the genotypic identity and diversity of eelgrass assemblages in outdoor mesocosms and documented their effects on the establishment of *Labyrinthula* that colonized naturally from seawater. We then assessed the independent and interactive effects of these factors, as well as the relationships of leaf growth rate and microbiome variation, with wasting disease prevalence and intensity. As discussed above, prior research indicates that *L. zosterae* prevalence and intensity could either increase or decrease in polycultures relative to monocultures. Regardless of the direction of the effect, we expected that trait variation among eelgrass genotypes that influences interactions among eelgrass, the eelgrass microbiome, *L. zosterae*, and temperature (Appendix S1: Table S1) could be used to predict the

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observed changes. For example, growth-defense tradeoffs would result in genotypes with higher growth rates having reduced defenses and thus increased *L. zosterae* infection (Herms and Mattson 1992, Steele et al. 2005, Trevathan-Tackett et al. 2015), particularly at higher temperature (Vergeer et al. 1995). In addition, genotypes with longer leaves would be expected to have higher *L. zosterae* prevalence due to greater leaf-to-leaf contact and resulting increased parasite transmission (Muehlstein 1992). Finally, we hypothesized that particular microbial taxa previously documented to be associated with disease status in the field would be associated with *L. zosterae* infection and more prevalent at warmer temperatures (Beatty et al. in revision).

Materials and Methods

Mesocosm experiment. We used a substitutive design to test the effects of eelgrass genotypic identity (eight genotypes), diversity (monocultures of 1 genotype vs. polycultures of 4 genotypes), and temperature (ambient or + 3.2° C) on the prevalence and intensity of *Labyrinthula* over eight weeks in an array of flow-through 120-L mesocosms at the Bodega Marine Laboratory in Bodega Bay, CA. In July 2015, we created ten unique polyculture combinations of four genotypes (4 genotypes per experimental pot) randomly drawn from a pool of eight genotypes; all eight genotypes were also grown in monoculture (1 genotype per pot). We filled pots (8.9 x 8.9 cm) with coarsely sieved sediment collected from Bodega Harbor, and planted 4 shoots per pot, matching the lower range of average field densities reported for Bodega Harbor (Ha and Williams 2018) to allow for growth during the experiment. Plants were originally collected in Bodega Harbor, CA in 2012, confirmed to be unique genotypes using 11 DNA microsatellite loci developed specifically for *Z. marina* (Abbott et al. 2018), and propagated in separate flow through mesocosms at BML. We previously characterized traits of

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each genotype relating to growth rate, morphology, nutrient content, and chemical defense in common garden experiments at ambient temperature from July 2013 to August 2014 (Abbott et al. 2018). We selected the 8 genotypes used in this experiment to encompass the range of trait values determined for this population of eelgrass measured when the common garden was experiencing marine heatwave conditions (DuBois et al. 2019).

We assigned ten pots - two unique polyculture combinations and each of the eight monocultures - to each of ten mesocosms, with five mesocosms per temperature treatment (see DuBois et al. 2021 for a diagram of the experimental set up). All mesocosms received sand-filtered flow-through seawater at a rate of approximately 0.8-1.0 L min^{-1}. We allowed the plants to acclimate for one month prior to initiating the temperature treatments. We maintained an ambient temperature treatment by cooling flow-through seawater in a head tank by approximately 1˚C using an Aqua Logic Delta Star in-line titanium chiller. Seawater in the elevated temperature treatment was raised approximately 3˚C above the ambient treatment in a separate header tank using Process Technologies titanium immersion heaters (Appendix S1: Fig. S1). This level of warming mimicked the 2014-15 extreme warming events in the Northern Pacific called “The Blob”, which raised summer ocean temperatures three standard deviations above the long-term average (Sanford et al. 2019).

At the end of the experiment (10 weeks), we estimated lesion percent cover of the third rank leaf of the terminal shoot of each transplant (i.e., focal leaf) to measure the signs of wasting disease (Burdick et al. 1993). We recorded lesions as either absent, <1%, <10%, or ≥10% cover. When lesions were ≥10% cover, we also recorded a numerical estimate of lesion percent cover. We then collected and preserved the top half of the focal leaf in individual plastic bags sealed with 30 ml of silica (Flower Drying Art Silica Gel; Activa) for subsequent DNA extraction and

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quantitative PCR to estimate *Labyrinthula zosterae* cells as a proxy for infection (Bergmann et al. 2011, Bockelmann et al. 2013, Groner et al. 2021; see Appendix S1 for details).

At one-month intervals over the course of the experiment, we measured leaf growth rate of the terminal shoot of each transplant using the "hole-punch" method (Williams and Ruckelshaus 1993). *L. zosterae* infection can be affected by plant defenses (Steele et al. 2005, Trevathan-Tackett et al. 2015) and these defenses may trade off with plant growth rate, resulting in a positive relationship between growth rate and infection. Alternatively, *L. zosterae* infection can result in reduced leaf growth rate (Graham et al. 2021), leading to a negative relationship. *L. zosterae* prevalence can also be affected by plant size (Groner et al. 2016), due to greater leaf-to-leaf contact and resulting increased parasite transmission (Muehlstein 1992), so we measured the length of the focal leaf of each transplant at the end of the experiment. We tested the direction and relative importance of these potential relationships using structural equation models (see below).

We also collected 2 cm of the focal leaf from directly below the midpoint and stored the tissue at -80˚C for later microbiome analyses to assess whether particular leaf microbial taxa changed in association with *Labyrinthula* presence on the focal leaf. From these samples, we selected a subset of 84 leaf microbial samples evenly distributed across temperature treatments and across genotypes in monoculture. We did not assess leaf microbiomes in genotypic polycultures. We extracted the surface community of these leaf segments with a modified protocol for the DNeasy Powersoil Kit (Qiagen) and sequenced the V4-V5 region of the 16S rRNA gene on an Illumina MiSeq to analyze differences in the leaf microbiome between treatments and among genotypes (see Appendix S1 for details).

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Analysis. Multiple metrics can be used to inform host-parasite interactions and resulting disease risk. Here, we present data on both wasting disease lesions and *L. zosterae* cells. Wasting disease lesions, the signs of infection, are often correlated with *L. zosterae* presence, and like a footprint, can also remain after *L. zosterae* cells, the causative agent of infection, are no longer detectable (Bockelmann et al. 2013, Brakel et al. 2014, Groner et al. 2021). *L. zosterae*, an opportunistic parasite, may also be present in plant tissue without causing lesions or prior to lesion development (Bockelmann et al. 2013, Burge et al. 2013). Thus, we used *L. zosterae* cells and lesions to estimate the presence of the *L. zosterae* parasite in plant tissue at the time of sampling and the extent of wasting disease infection over the course of the experiment, respectively. We also examined the relationship between the two metrics using generalized linear models, with focal leaf as the unit of replication in these models.

We further parsed our analyses of each disease response into prevalence (a binary variable characterizing presence or absence) and intensity (a continuous variable representing the number of parasites in infected hosts or the proportion of tissue damaged; Bush et al. 1997). We analyzed prevalence and intensity independently to examine whether temperature and genotypic diversity affected the probability of infection and the intensity of infection differently. In order to analyze lesion intensity using a parallel framework to *L. zosterae* cell intensity analyses, we converted our categorical lesion percent cover estimates into numerical estimates using the following method: We coded all lesions in the <1% category as 0.5, all lesions in the <10% category as 5, and we used the numerical estimates of lesion percent cover for lesions in the ≥10% category.

To account for potential environmental differences due to the spatial layout of mesocosms (e.g., variation in flow resulting from distance from the header tank), we paired hot

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and cold mesocosms based on location along the inflow raceway and included this blocking factor as a random effect. We treated pot as the unit of replication in all models.

We used generalized linear mixed models to examine the independent and interactive effects of host genotypic diversity and temperature on wasting disease intensity (i.e., lesion percent cover and cell density) and prevalence (i.e., lesion presence and cell presence). For the *L. zosterae* cell intensity analyses, we excluded one mesocosm pair due to the absence of *L. zosterae* cells in elevated-temperature polyculture treatments, because we were specifically interested in these patterns when *L. zosterae* was present. Because we found an interactive effect of host genotypic diversity and temperature on *L. zosterae* intensity, we further tested for additive vs emergent effects of host genotypic diversity by comparing the data we observed in our experimental polycultures to the data we expected should there be no interactions between genotypes (i.e., an additive effect; Johnson et al. 2006). We created expected data sets for the response variables by substituting the observed response of each plant in polyculture with the appropriate value for that genotype in monoculture (see Appendix S1 for details). For lesion prevalence, the model results for the diversity by temperature interaction failed to converge due to the quasi-complete separation of lesion prevalence data in elevated temperature polycultures, so we plotted these data rather than formally interpreting them.

For host genotypic identity, we also used generalized linear models examining the independent and interactive effects of identity and temperature on wasting disease prevalence and intensity. For the *L. zosterae* cell intensity analyses, we grouped mesocosm-pairs into two statistical blocks (1-3 and 4-5) based on distance from the header tanks and used these blocks as main effects in the model in order to maintain all combinations of genotype by temperature treatment in each block. Because we were interested in these patterns when *L. zosterae* was

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present, we excluded one genotype (labeled "green") from the *L. zosterae* cell intensity analyses and figures due to the absence of *L. zosterae* cells in the cold treatment. As above, the model results for the genotype main effect and the genotype by temperature interaction for lesion prevalence failed to converge, so we plotted these data but did not formally interpret them.

For all analyses, we fit response variables to error distributions based on the process that generated the distributions. Specifically, we fit *L. zosterae* cell and lesion prevalence with logistic regressions (binomial GLM/GLMER with logit link), *L. zosterae* cell intensity with a generalized linear regression (Poisson GLM/GLMER with a log link), and square-root transformed lesion percent cover prior to fitting with a linear regression (LMER). Model analyses was performed in the R package 'lme4' (Bates et al. 2015). We applied the 'bobyqa' optimizer function to generalized linear models as necessary to achieve model convergence (Bates et al. 2015). We used likelihood ratio tests following model simplification via a stepwise backwards-elimination procedure to test for significant treatment effects (Pinheiro & Bates 2004).

We used observed-variable structural equation modeling (SEM) to test for hypothesized causal relationships between temperature, traits of eelgrass genotypes in monoculture, and metrics of wasting disease infection. When the linear models described above indicated genotypic variation in wasting disease in response to temperature, we divided genotypes into two groups: genotypes that experienced significant increases in wasting disease under warming and genotypes that experienced significant decreases in wasting disease under warming. We excluded genotypes that did not experience temperature-dependent differences in wasting disease response, because we wanted to investigate how traits mediated wasting disease infections. Specifically, to test the growth-defense trade-off hypothesis for *L. zosterae* cell intensity, we ran

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one SEM with genotypes that experienced increases in *L. zosterae* cell intensity with warming: brown, orange, red, white, and yellow. We then ran a separate SEM with genotypes that experienced decreases in *L. zosterae* cell intensity with warming: blue and purple. We excluded the green genotype from both SEMs, because it did not exhibit temperature-dependent differences in *L. zosterae* cell intensity. To test the growth-defense trade-off hypothesis for lesion intensity, we included all genotypes in a single SEM, because there was no interactive effect of eelgrass genotype and temperature on lesion intensity in our prior analyses. To test our hypothesis that leaf length mediates the effects of temperature on *L. zosterae* cell prevalence, we ran an SEM on the two genotypes that experienced increases in *L. zosterae* cell prevalence with warming: brown and green. No genotypes experienced significant decreases in *L. zosterae* cell prevalence with warming. Finally, we did not use SEM to test the hypothesis that leaf length mediates the effects of temperature on lesion prevalence, because we were unable to assess possible interactive effects of genotype and temperature on lesion prevalence in our linear models. For all SEMs, we treated pot as the unit of replication. SEMs were performed in the R package 'lavaan' (Rosseel & Lavaan 2012; see Appendix S1: Fig. S2 for covariance plots).

To examine the potential contribution of the microbiome to host-parasite-warming interactions in this system, we ran 16S sequence data through a dada2 pipeline to de-noise sequence data, estimate error rates, identify amplicon sequence variants (ASVs) and remove chimeric sequences (see Appendix S1 for details; Callahan et al. 2016). We compared ASV richness among treatments and genotypes using the number of observed ASVs and number of ASVs when rarified to 12,149 sequences (see Appendix S1 for details). All other analyses were done on unrarefied data. We compared the microbiome of two groups: genotypes that showed reduced parasites when warmed vs those that showed increased parasites when warmed (Fig.

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1B). To identify differences among groups, we computed the Aitchison distance between samples (Aitchison et al. 2000; Gloor et al. 2017). We computed a permutational ANOVA on group-level differences with adonis2 in the R package 'vegan' (Oksanen et al. 2019). To identify which bacteria of the 2407 ASVs that we observed varied between genotypes with reduced vs. increased parasites when warmed, we built negative binomial models based on the geometric means of ASV counts in DESeq2 (Love et al. 2014). We separately assessed which ASVs varied (1) between treatments and (2) between genotype_group + temperature_treatment + genotype_group:treatment using a Wald test. We then applied a Benjamini-Hochberg correction to all reported p-values. All data analyses were performed in R (version 3.6.1, www.R-project.org).

Results

The probability of detecting *L. zosterae* and the intensity of these detections increased with lesion percent cover (Fig. 2A-B, Appendix S1: Table S2).

Effects of genotypic diversity

Water temperature and genotypic diversity interactively affected *L. zosterae* cell intensity, but only water temperature affected *L. zosterae* cell prevalence (Fig. 3A-B, Appendix S1: Table S3). At elevated water temperature, mean infection intensity in monoculture was 22% more than in polyculture, whereas at ambient water temperature mean infection intensity in monoculture was 13% less than in polyculture. In addition, the mean infection intensity we observed at elevated temperature in polycultures was 26% less than we expected given the infection intensities of the respective genotypes in monoculture ($\chi^2_1 = 242.43$, $P < 0.0001$; a priori contrasts, $P < 0.05$; Fig. 3C). In contrast, the observed mean infection intensity at ambient

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temperature in polycultures was 21% more than expected (a priori contrasts, $P < 0.05$; Fig. 3C). Thus, the effects of diversity we observed on infection intensity were not explained solely by the identity of infected genotypes in polycultures, but rather were the result of an emergent property of genotype interactions in polyculture.

In contrast to the *L. zosterae* cell results, water temperature but not genotypic diversity affected lesion intensity and prevalence (Fig. 3D-E, Appendix S1: Table S3). Lesions were more prevalent and intense at elevated water temperature.

Effects of genotypic identity

Among monocultures, water temperature and genotypic identity interactively affected *L. zosterae* cell intensity and prevalence (Fig. 1A-B, Appendix S1: Table S4). *L. zosterae* intensity increased in elevated water temperature in five genotypes (brown, orange, red, white, and yellow), but decreased in two genotypes (blue and purple; a priori contrasts, $P < 0.05$). *L. zosterae* prevalence increased in elevated water temperature in two genotypes (green and brown; a priori contrasts, $P < 0.05$), but it did not vary by temperature in the other six genotypes (blue, orange, purple, red, white, and yellow; a priori contrasts $P > 0.05$).

Water temperature, but not genotypic identity or the interaction of genotypic identity and water temperature, affected variation in wasting disease lesion intensity (Fig. 1C, Appendix S1: Table S4) among monocultures. Water temperature also affected lesion prevalence (Fig. 1D, Appendix S1: Table S4). Lesions were more intense and more prevalent at elevated water temperature.

Our structural equation models (SEMs) provided mixed support for our hypothesis that changes in eelgrass growth rates mediated the effects of temperature on *L. zosterae* cell and lesion intensity. For the five eelgrass genotypes that experienced greater *L. zosterae* cell intensity

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under warming, we found a direct positive relationship between temperature and eelgrass growth rate, and a weak direct positive relationship between growth rate and *L. zosterae* cell intensity (Fig. 4A). For the two genotypes for which warming reduced *L. zosterae* cell intensity, we found a weak direct positive relationship between temperature and eelgrass growth rate, but no effect of growth rate on *L. zosterae* cell intensity (Fig. 4B). For all genotypes, we found a direct positive relationship between temperature and eelgrass growth rate, and a direct positive relationship between growth rate and lesion intensity (Fig. 4C).

We did not find support for our hypothesis that changes in eelgrass length mediated the effects of temperature on *L. zosterae* cell prevalence. For the two eelgrass genotypes that experienced greater *L. zosterae* cell prevalence under warming, we found no relationship between temperature and eelgrass length, although eelgrass length had a weak direct positive effect on *L. zosterae* cell prevalence (Fig. 4D).

Effects of the host microbiome

Warming caused substantial differences in microbial community composition (Fig. 5A), increased bacterial alpha diversity on warmed leaves (t-test, df = 57.9, t = 3.7796, $p<0.001$, Appendix S1: Fig. S3B), and significant changes in the relative abundance of 79 taxa (Appendix S1: Fig. S4; http://lod.bco-dmo.org/id/dataset/883070). Effects of genotype group (those with reduced parasites when warmed vs increased parasites when warmed) were more subtle. We did not find effects of genotype group by itself or in an interaction with warming on alpha or beta diversity of bacterial taxa (Fig. 5A, Appendix S1: Fig. S3A). However, eighteen ASVs did differ in their relative abundance between genotype groups (Fig. 5B, http://lod.bco-dmo.org/id/dataset/883070). In ambient conditions, two taxa (*Arenicella* sp. and *Rhodopirellula* sp.) were more abundant in the genotype group that showed an increase in parasites when

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warmed; in warmed conditions, two other ASVs (*Cellvibrionaneae spp.* and *Tateyamaria sp.)* were more abundant in this group. A *Polibacter_2 sp.* ASV was more abundant in both temperature treatments in those genotypes that had greater parasites when warmed. In the genotype group with reduced parasites under warming, there was a greater abundance of 8 taxa in ambient and 4 taxa in warmed treatments. Notably, the relative abundance of only one ASV, *Maribacter sp.*, reversed across temperature treatments: it was more abundant in genotype groups that showed a reduction in parasites under the respective temperature conditions.

Discussion

Host genotypic diversity dampened the positive effect of warming on parasite intensity, but not prevalence, in our experiment. Notably, many empirical examples of the effects of host diversity on disease outcomes in natural systems are a function of host composition rather than host diversity per se (Johnson et al. 2015), yet the reduction we observed in disease intensity in polyculture was greater than expected based on genotypic composition (Johnson et al. 2006), similar to results found in crop studies (Reiss & Drinkwater 2018). Thus, emergent effects of biodiversity such as complementarity or facilitation among genotypes contributed to the reduction in parasite intensity at elevated temperature in our study. However, the benefits of diversity for disease risk were only manifest under warming: at ambient temperature, diversity actually increased parasite intensity slightly. There is also evidence that these infections had consequences for plant fitness; eelgrass biomass in this experiment was reduced in polycultures relative to monocultures at ambient temperature, but was greater in polycultures relative to monocultures under warm conditions, despite an overall negative effect of warming on biomass in monocultures (DuBois et al. 2020).

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The context-dependent effects of host diversity on wasting disease are likely due to the combined strong influence of temperature on *L. zosterae* and on the defensive capabilities of the host. Elevated temperatures are often correlated with increases in wasting disease in the field (Rasmussen et al. 1977, Bockelmann et al. 2013, Aoki et al. 2022), and experiments have demonstrated a positive relationship between temperature and parasite growth (Young 1943, Dawkins et al. 2018) and pathogenicity (Kaldy 2014), and a negative relationship between temperature and eelgrass chemical defense (Vergeer et al. 1995). Further, eelgrass genotypes show complementary resource use (Hughes & Stachowicz 2011), and mixtures are more resistant and resilient to disturbance or stress (Hughes & Stachowicz 2004, 2011), suggesting diversity mediates eelgrass condition. Because parasite growth is increased by temperature in this system, the role of diversity-mediated changes in host competence is expected to increase in importance as temperature increases (Dawkins et al. 2018, Brakel et al. 2019), as observed in our warming treatment. The effects of host diversity are similarly dependent on elevated disease risk in agricultural crop cultivars (Reiss & Drinkwater 2018) and amphibian-trematode systems (Johnson et al. 2013a). Thus, greater consideration of the broader ecological context under which host-parasite interactions are evaluated may help resolve conflicting results regarding relationships between host identity or diversity and disease risk.

We also found the effects of warming on disease varied among eelgrass genotypes, with genotypes falling into two groups: genotypes for which warming increased disease intensity and genotypes that had greater disease intensity at ambient temperatures. For genotypes that experienced increased parasite intensity under warming, we found some support for our hypothesis that eelgrass growth rate mediates the relationship between temperature and parasite intensity through growth-defense trade-offs (Lind et al. 2013). Warming also increased parasite

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prevalence in certain host genotypes, and there was some indication that longer leaves were more likely to be infected (Groner et al. 2016), but the prevalence-temperature relationship was not mediated by increased host leaf length as predicted. We were unable to include polycultures in these analyses due to lack of replication of specific genotype combinations.

The majority of the eelgrass leaf microbiome was unchanged among those plants that showed a reduction in parasites when warmed vs those that showed an increase in parasites when warmed. However, some specific ASVs showed variation between genotype groups. In warmed conditions, ASVs that were more abundant in genotypes that showed reduced parasites under warmed conditions could play some role in reducing parasite intensity, but of the taxa identified there is no specific evidence that these might be directly or indirectly antagonistic to *L. zosterae*. Taxa that were more abundant in genotypes with increased parasites could potentially facilitate *L. zosterae,* or negatively impact the host in ways that would enhance *L. zosterae* infection, or may be taxa that colonize opportunistically in diseased plants. For example, *Cellvibrionaceae* are cell-wall degraders that could either enhance *L. zosterae* spread or respond to the presence of dead cells (Egan and Gardiner 2016). ASVs in this family were consistently associated with wasting disease lesions in large-scale surveys across the west coast of the US (Beatty et al. in revision, mSystems), suggesting they are worthy of additional attention in the temperature-eelgrass-*Labyrinthula* interaction. It is also worth noting that similar but distinct strains likely have different effects: for example, one strain of *Maribacter* differed among genotype groups, but seven other ASVs identified as *Maribacter* showed no differences. More targeted approaches looking at the microbiome within lesions and during lesion development across genotypes might yield more information about microbiome-pathogen-genotype interactions (Hurtado-McCormick et al. 2021, Beatty et al. in revision). We did not sequence microbiome samples from

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polycultures; thus, we cannot address whether microbiome-related mechanisms (e.g. a reservoir of putatively protective microbes in resistant genotypes or an exchange of microbes among resistant and susceptible genotypes) could underlie the observed dilution effect under warming (Barbosa et al. 2009). Nevertheless, differences in relative abundance of microbial ASV between genotypes that do and do not show increased parasite load with warming suggest that microbes could play some role in variation in the susceptibility or response of eelgrass to *L. zosterae*, as has been suggested for the outcome of host-parasite interactions more broadly (Zaneveld et al. 2017, Wilkins et al. 2019).

As expected, the probability of detecting *L. zosterae* and the intensity of these detections increased with lesion percent cover, suggesting the lesions we observed are indeed signs of wasting disease. However, there was also considerable variation in this relationship. For example, the vast majority of leaves had lesions at the time of sampling, indicating most leaves had experienced infection over the course of the lifetime of that leaf. *L. zosterae* cells, in contrast, were only detected in half of leaves with lesions and in approximately a third of leaves without lesions at the end of the experiment. This variation supports the value of analyzing and interpreting both metrics, as they provide different information about infections: lesions provide a record of wasting disease infection over the lifetime of a leaf, whereas *L. zosterae* cells provide insight into active infections at the time of sampling. We detected warming effects on both lesion and cell-based metrics, but only the cell-based metrics were influenced by genotypic diversity and composition effects. The coarse, categorical nature of our lesion metrics may have limited our ability to detect genotype effects on that response. Alternatively, genotype differences may primarily affect the severity of active infections yet have effects that are more

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challenging to detect over the lifetime of a leaf. Overall, this difference does suggest that warming effects are stronger or of greater magnitude than genotypic effects on disease metrics.

Our experimental design mimicked random community assembly, which may have limited the likelihood of detecting diversity effects on disease prevalence that often occur via composition-based mechanisms (Schmidt and Ostfeld 2001). For example, if susceptible genotypes are more common than resistant genotypes, diversity may reduce disease prevalence as a result of correlations between host competence and community assembly (Joseph et al. 2013, Mihaljovic et al. 2014). Indeed, host community assembly is known to drive parasite dynamics in other systems (Previtali et al. 2012, Johnson et al. 2013b), and the same mechanisms behind these effects in communities with multiple host species (e.g., life-history trade-offs, parasite adaptation, and compensatory community assembly) are also applicable at the intraspecific level. Given the importance of eelgrass to the conservation and restoration of coastal ecosystems (Orth et al. 2006, McGlathery et al. 2007), exploration of potential diversity-disease interactions in the field, where both genotypic diversity and relative abundance vary (Hughes and Stachowicz 2009, Kollars and Stachowicz 2022), is a clear next step.

Intriguingly, considerable variation in eelgrass genotypic diversity and identity exists at very fine scales (e.g. m^2 plots) within natural meadows, and thus our results may have important implications for eelgrass-disease dynamics in the field (Kamel et al. 2012, Hays et al. 2020, Kollars and Stachowicz 2022). For example, minor changes in eelgrass diversity or identity could affect the spatial distribution of disease susceptibility within meadows, and these small-scale effects may be especially relevant in restoration contexts when changes to the method of collection and deployment of eelgrass may greatly impact the diversity and identity of genotypes. In addition, parasite diversity generally increases with host diversity (Kamiya et al. 2014,

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Rottstock et al. 2014) and, in some instances, disease risk associated with individual parasites is reduced in more diverse parasite communities due to competition among parasites (Rottstock et al. 2014, Johnson et al. 2012, Johnson et al. 2013b). There is some evidence of intraspecific variation among *L. zosterae* strains (Brakel et al. 2014, Dawkins et al. 2018), yet the link between eelgrass diversity and *L. zosterae* diversity and the consequences for wasting disease risk remain unexplored in this system. Finally, the variation in susceptibility we observed among genotypes suggests wasting disease may to some degree differentially affect genotypes, leading to the possibility that disease may contribute to diversity maintenance in natural eelgrass meadows via frequency dependent selection (Lively & Dybdahl 2000, Lively 2010).

Prior research on the effects of temperature on host-parasite interactions has largely ignored the potential importance of host identity and diversity. Our study addresses this knowledge gap by demonstrating that effects of temperature interact with host genotypic identity and diversity to determine disease risk. In addition, we found evidence that these effects are mediated through changes to host physiology and possibly associations with particular microbial taxa. Although our results serve as a warning that host biodiversity losses coupled with environmental change could increase disease risk, they also provide hope, as certain host genotypes and more diverse assemblages of hosts were capable of dampening disease when environmental conditions promoted parasites.

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Tarik Gouhier provided statistical advice. This is contribution 425 from the Northeastern University Marine Science Center.

Statement of Authorship: FRS and ARH conceived of the idea for the study. KD, MRK, and JJS conducted the experiment. FRS led the wasting disease analyses and MRK led the microbiome analyses. FRS wrote the first manuscript draft and all authors contributed substantially to revisions.

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Figure Legends

Figure 1. Eelgrass genotypic identity and seawater temperature moderate wasting disease risk. (A) Effects of eelgrass genotypic identity and temperature on average *L. zosterae* cell intensity. Infected replicates in ambient (bl (blue): n = 4; br (brown): n = 2; gr (green): n = 0; or (orange): n = 4; pu (purple): n = 4; re (red): n = 4; wh (white): n = 4; ye (yellow): n = 4) and warmed (bl: n = 4; br: n = 4; gr n = 0; or: n = 4; pu: n = 5; re: n = 4; wh: n = 3; ye: n = 5). (B) Effects of eelgrass genotypic identity and seawater temperature on *L. zosterae* cell prevalence. Open points represent ambient temperature treatments and solid points represent elevated temperature treatments. Replicates per unique genotype-temperature treatment combination (n = 5). (C) Effects of eelgrass genotypic identity and temperature on average lesion percent cover intensity (n = 5 per unique genotype-temperature treatment combination). (D) Effects of eelgrass genotypic identity and seawater temperature on lesion prevalence (n = 5 per unique genotype-temperature treatment combination). All values are means ± SEM and range trimmed violin plots.

Figure 2. *L. zosterae* cell prevalence and intensity are positively correlated with lesion percent cover. (A) *L. zosterae* cell prevalence by lesion percent cover group: 0% (n = 22), <1% (n = 45), <10% (n = 258), ≥10% (n = 53). (B) *L. zosterae* cell intensity by lesion percent cover group: 0% (n = 8), <1% (n = 13), <10% (n = 134), ≥10% (n = 31). All values are means ± SE.

Figure 3. Eelgrass genotypic diversity and seawater temperature moderate wasting disease risk. (A) Effects of eelgrass genotypic richness and temperature on average *L. zosterae* cell intensity. Infected replicates in ambient-monoculture (n = 24), warmed-monoculture (n = 31), ambient-polyculture (n = 6), warmed-polyculture (n = 8). (B) Effects of eelgrass genotypic richness and temperature on *L. zosterae* cell prevalence. Replicates in ambient-monoculture (n = 40),

warmed-monoculture (n = 40), ambient-polyculture (n = 10), warmed-polyculture (n = 10). (C) Comparison of the observed *L. zosterae* intensity from polycultures to the expected *L. zosterae* intensity if there were no interactions among genotypes (i.e. additive effects). Open points represent ambient temperature treatments and solid points represent elevated temperature treatments. Infected replicates in ambient-monoculture (n = 24), warmed-monoculture (n = 31), ambient-polyculture (n = 6), warmed-polyculture (n = 8). (D) Effects of eelgrass genotypic richness and temperature on lesion percent cover intensity. Replicates in ambient-monoculture (n = 40), warmed-monoculture (n = 40), ambient-polyculture (n = 10), warmed-polyculture (n = 10). (E) Effects of eelgrass genotypic richness and temperature on lesion prevalence. Replicates in ambient-monoculture (n = 40), warmed-monoculture (n = 40), ambient-polyculture (n = 10), warmed-polyculture (n = 10). All values are means ± SE and range trimmed violin.

Figure 4. Structural equation models depicting the relationships between seawater temperature, *Z. marina* leaf growth or leaf length, and disease intensity and prevalence in monoculture. (A) *L. zosterae* cell intensity in genotypes (brown, orange, red, white, and yellow) that experienced increased intensity under warming (n = 38), (B) *L. zosterae* intensity in genotypes (blue and purple) that experienced decreased intensity under warming (n = 18), (C) lesion intensity in genotypes (all) that experienced increased intensity under warming (n = 80), and (D) *L. zosterae* cell prevalence in genotypes (brown and green) that experienced increased prevalence under warming (n = 20). Coefficients of determination are shown in bold by endogenous variables. Path regression coefficients are standardized. Paths with solid black lines are significant at alpha <0.05, paths with dashed black lines are significant at alpha < 0.1, and paths with grey lines are not significant. See Appendix S1: Fig. S2 for metrics of model fit.

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Figure 5. Effects of genotype group and seawater temperature on community composition and abundance of bacteria isolated from *Z. marina* leaf surfaces. (A) Bacterial community composition varied by temperature (PERMANOV, $p < 0.001$), but variance in composition between and within temperature treatments were not different as calculated through multivariate dispersion in betadisper (vegan R package, $p = 0.49$). Open shapes are ambient temperature treatments ($n = 25$); solid shapes are elevated temperature treatments ($n = 36$). Triangles are genotypes that showed an increase in *L. zosterae* intensity when warmed ($n = 16$) and circles are genotypes that showed a decrease in *L. zosterae* intensity when warmed ($n = 45$). (B) Log2 fold change of the 18 bacterial taxa of 2407 tested that differ in their relative abundance between genotype groups by DESeq2 ($P < 0.05$). Only taxa that varied significantly between genotypes with reduced vs. increased parasites when warmed are shown. Positive values indicate greater abundance in the genotype group that showed an increase in *L. zosterae* when warmed and negative values indicate greater abundance in the genotype group that showed a decrease in *L. zosterae* when warmed. Open points are differences between genotype groups in ambient temperature treatments; solid points are differences between genotype groups in elevated temperature treatments. Values are means ± SE. The right of the figure includes the rank abundance of the bacterial taxa (out of 2407) and its relative abundance as the average percentage seen across our samples.

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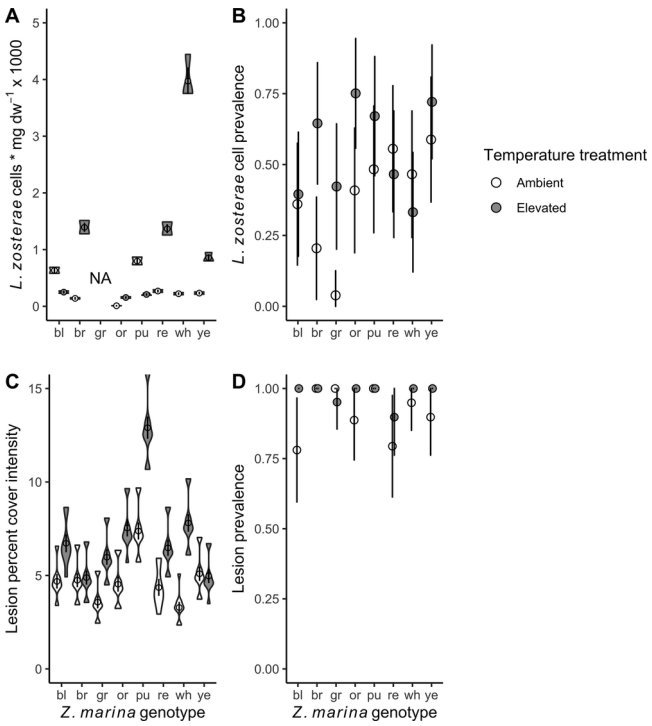

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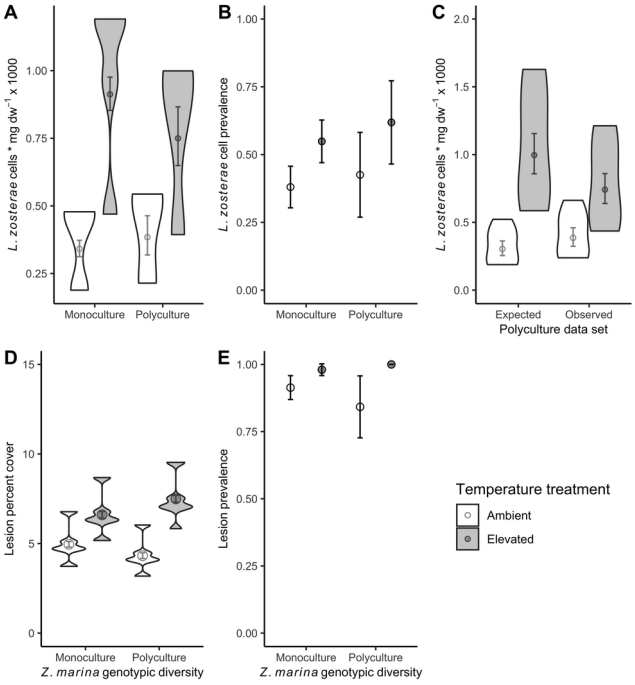

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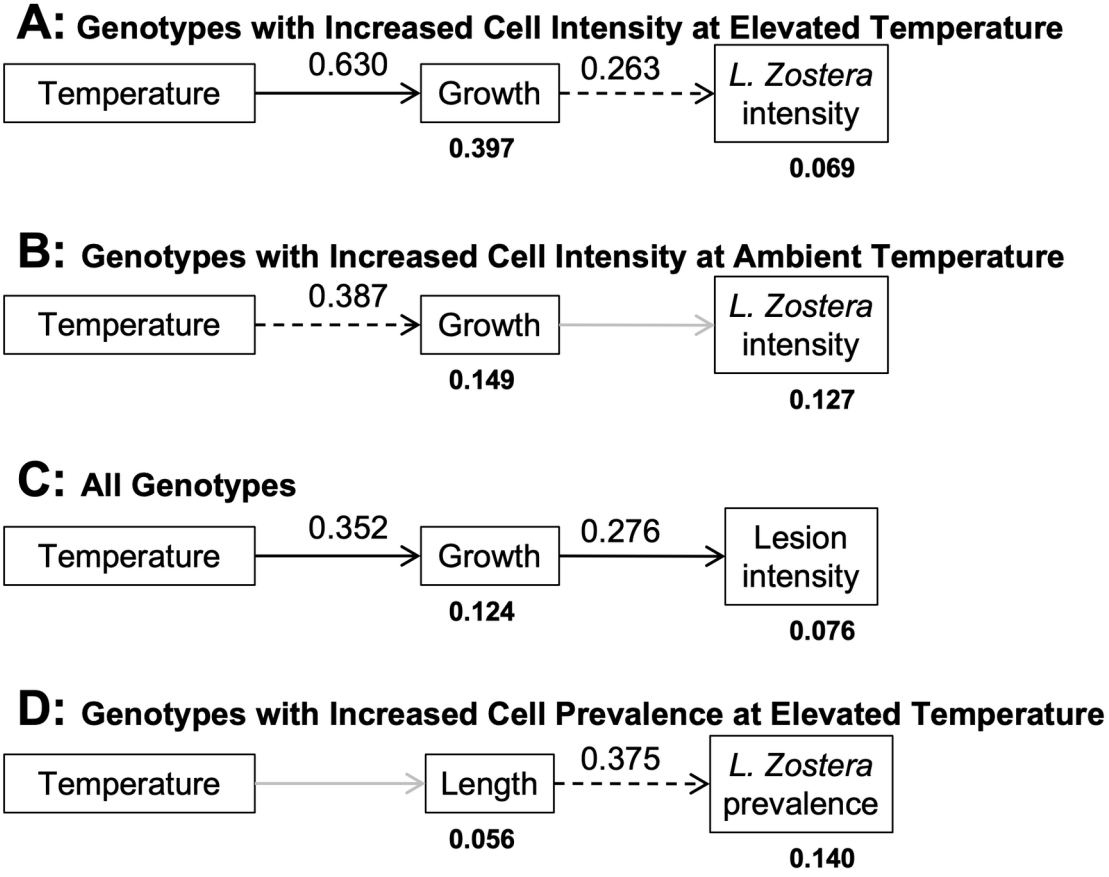

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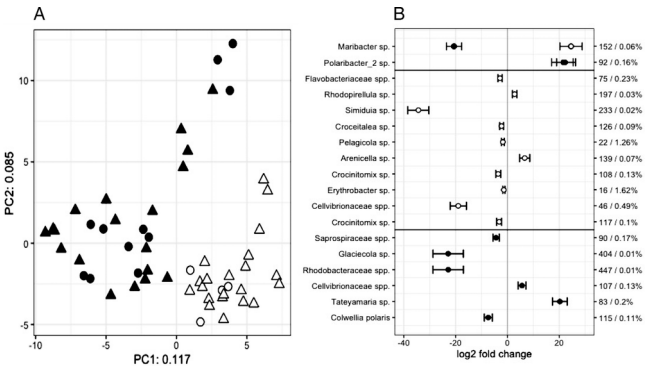

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