Received: 19 September 2024 | Accepted: 13 November 2024

DOI: 10.1002/ecy.4532

ARTICLE

ECOLOGY
ECOLOGICAL SOCIETY OF AMERICA

Seagrass wasting disease prevalence and lesion area increase with invertebrate grazing across the northeastern Pacific

Lillian R. Aoki[1,2] | **Carmen J. Ritter**[3] | **Deanna S. Beatty**[4] | **Lia K. Domke**[5,6] | **Ginny L. Eckert**[5] | **Olivia J. Graham**[1] | **Carla P. Gomes**[7] | **Collin Gross**[8] | **Timothy L. Hawthorne**[9] | **Eliza Heery**[3,10,11] | **Margot Hessing-Lewis**[12] | **Kevin Hovel**[13] | **Karl Koehler**[14] | **Zachary L. Monteith**[12] | **Ryan S. Mueller**[15] | **Angeleen M. Olson**[12] | **Carolyn Prentice**[12] | **Brendan Rappazzo**[7] | **John J. Stachowicz**[4] | **Fiona Tomas**[16] | **Bo Yang**[17] | **C. Drew Harvell**[1] | **J. Emmett Duffy**[3]

[1]Department of Ecology and Evolutionary Biology, Cornell University, Ithaca, New York, USA

[2]Environmental Studies Program, University of Oregon, Eugene, Oregon, USA

[3]Tennenbaum Marine Observatories Network, Smithsonian Environmental Research Center, Edgewater, Maryland, USA

[4]Department of Evolution and Ecology, University of California, Davis, California, USA

[5]College of Fisheries and Ocean Sciences, University of Alaska Fairbanks, Juneau, Alaska, USA

[6]Alaska Fisheries Science Center, NOAA, National Marine Fisheries Service, Juneau, Alaska, USA

[7]Department of Computer Science, Cornell University, Ithaca, New York, USA

[8]Department of Biology, Stanford University, Stanford, California, USA

[9]Department of Geosciences, Auburn University, Auburn, Alabama, USA

[10]School of Interdisciplinary Arts and Sciences, University of Washington, Tacoma, Washington, USA

[11]Science and Mathematics Division, School of Interdisciplinary Arts and Sciences, University of Washington, Tacoma, Washington, USA

[12]Hakai Institute, Campbell River, British Columbia, Canada

[13]Department of Biology, Coastal and Marine Institute, San Diego State University, San Diego, California, USA

[14]Maine Department of Marine Resources, Boothbay, Maine, USA

[15]Department of Microbiology, Oregon State University, Corvallis, Oregon, USA

[16]Instituto Mediterraneo de Estudios Avanzados (UIB-CSIC), Esporles, Spain

[17]Department of Environmental Studies, University of California, Santa Cruz, California, USA

Correspondence
Lillian R. Aoki
Email: laoki@uoregon.edu

Funding information
Maria de Maeztu Exellence Unit, Grant/Award Number: CEX2021-001198; Hakai Institute in British Columbia;

Abstract

Disease is a key driver of community and ecosystem structure, especially when it strikes foundation species. In the widespread marine foundation species eelgrass (*Zostera marina*), outbreaks of wasting disease have caused large-scale meadow collapse in the past, and the causative pathogen, *Labyrinthula zosterae*, is commonly found in meadows globally. Research to date has mainly

National Science Foundation, Grant/Award Numbers: OCE-1635716, OCE-1600230, OCE-1829890, OCE-1829921, OCE-1829922, OCE-1829992; RESIGRASS, Grant/Award Number: CGL2014-58829-C2-2-R; MarineGEO and Tennenbaum Marine Observatories Network

Handling Editor: Matthew E. Bracken

focused on abiotic environmental drivers of seagrass wasting disease, but there is strong evidence from other systems that biotic interactions such as herbivory can facilitate plant diseases. How biotic interactions influence seagrass wasting disease in the field is unknown but is potentially important for understanding dynamics of this globally valuable and declining habitat. Here, we investigated links between epifaunal grazers and seagrass wasting disease using a latitudinal field study across 32 eelgrass meadows distributed from southeastern Alaska to southern California. From 2019 to 2021, we conducted annual surveys to assess eelgrass shoot density, morphology, epifauna community, and the prevalence and lesion area of wasting disease infections. We integrated field data with satellite measurements of sea surface temperature and used structural equation modeling to test the magnitude and direction of possible drivers of wasting disease. Our results show that grazing by small invertebrates was associated with a 29% increase in prevalence of wasting disease infections and that both the prevalence and lesion area of disease increased with total epifauna abundances. Furthermore, these relationships differed among taxa; disease levels increased with snail (*Lacuna* spp.) and idoteid isopod abundances but were not related to abundance of ampithoid amphipods. This field study across 23° of latitude suggests a prominent role for invertebrate consumers in facilitating disease outbreaks with potentially large impacts on coastal seagrass ecosystems.

KEYWORDS

disease ecology, eelgrass wasting disease, epifauna, herbivory, mesograzers, plant-herbivore interactions, plant-pathogen interactions, plant-pathogen-herbivore interactions, *Zostera marina*

INTRODUCTION

Pathogens are ubiquitous and strongly influence ecological dynamics. Diseases that affect foundation species have particularly strong ecological impacts. For example, white band disease caused widespread mortality of reef-building corals in the Caribbean in the 1980s, facilitating conversion to algal-dominated reefs with flattened habitat complexity and reduced fish biodiversity (Alvarez-Filip et al., 2009). In North America, white pine blister rust and chestnut blight caused continental-scale losses of whitebark pine and American chestnut respectively since the early 1900s, altering forest canopy structure, stream hydrology, and carbon sequestration (Ellison et al., 2005). Abiotic conditions and local stressors can alter host susceptibility and pathogen abundance and virulence, resulting in spatially variable vulnerability to disease (Maynard et al., 2015). Organismal interactions also influence disease, such as bark beetles vectoring the fungus causing Dutch elm disease when feeding on elms (Santini & Faccoli, 2015). Understanding the ecological consequences of disease requires investigating the network of biological interactions and environmental drivers that facilitate or suppress disease.

Herbivory is a key interaction that affects the ecology of plant disease through multiple mechanisms. Physical scars from grazing can facilitate infection (Boyd et al., 2022; Silliman & Newell, 2003), although grazing that consumes whole leaves can remove pathogens and reduce disease (Liu et al., 2021). Grazing can alter plant community structure, in turn altering pathogen abundances and disease prevalence (Li et al., 2024); grazing can also stimulate plant responses that defend against both herbivory and disease and may influence infection intensity (Thaler et al., 2010). Commonalities are emerging across herbivore-plant-pathogen systems, with infections promoted by grazing scars from crustaceans in salt marshes (Daleo et al., 2009), insects in tropical forests (García-Guzmán & Dirzo, 2001), and mammals in grasslands (Liu et al., 2021). Yet system-specific aspects, such as the extent and mode of herbivory, will affect whether grazing increases or decreases disease (Cappelli & Koricheva, 2021).

Both herbivory and disease strongly influence the ecology of seagrasses, including eelgrass (*Zostera marina*), a widespread foundation species of temperate Northern Hemisphere coasts whose meadows support diverse faunal communities, improve water quality, and sequester carbon (Duffy et al., 2013). Eelgrass meadows are susceptible to seagrass wasting disease, caused by the globally distributed protist *Labyrinthula zosterae* (Muehlstein, 1992). This disease has strongly affected meadow dynamics in the past, especially in

the 1930s when outbreaks reportedly wiped out 90% of eelgrass along the Atlantic coast of North America (Thayer et al., 1984). More recently, wasting disease was linked to localized meadow declines in the western Atlantic (Short et al., 1987) and the eastern Pacific (Groner et al., 2016). Temperature and salinity likely influence the disease, but despite both field (Bockelmann et al., 2012; Graham et al., 2023; Groner et al., 2016) and laboratory studies (e.g., Schenck et al., 2023), the drivers of the disease are not well understood. Biological interactions, including grazing, that may facilitate or suppress seagrass wasting disease have received little attention, although these interactions have wide-ranging effects on other aspects of seagrass ecology (Duffy et al., 2013).

Herbivores play important functional roles in eelgrass meadows with multiple potential mechanisms linked to wasting disease. Epifaunal invertebrate grazers include snails and crustaceans that can strongly influence marine plant communities and help to control growth of epiphytic algae on eelgrass leaves, which can shade plants and restrict productivity (Baden et al., 2010; Duffy et al., 2013; Reynolds et al., 2014). High epiphyte loads were associated with increased wasting disease prevalence in surveys in the northeastern Pacific (Groner et al., 2016); grazer consumption of epiphytes might therefore reduce wasting disease. Other studies found higher rates of wasting disease infection in eelgrass leaves exposed to grazing by epifauna (Graham et al., 2025; Murray et al., 2024), suggesting that physical wounding by grazers increases infection risk. Grazers may transfer the pathogen through feces, accelerating infection, as occurs with snails in salt marshes (Silliman & Newell, 2003). Finally, grazers may preferentially consume diseased tissue (Graham et al., 2025; Murray et al., 2024; Reynolds et al., 2018), decreasing the standing stock of diseased tissue and limiting transmission (Muehlstein, 1992). These mechanisms overlap in nature, and initial laboratory studies suggest that different grazer taxa have different effects on disease (Graham et al., 2025).

In this study, we explored the association of grazing epifaunal invertebrates with seagrass wasting disease in meadows across 23° of latitude in the northeastern Pacific. The study captured latitudinal trends in abundances of grazers and substantial variation in eelgrass shoot densities, canopy heights, wasting disease, and environmental conditions. We used structural equation modeling (SEM) to compare a direct pathway from epifaunal grazers to wasting disease with an indirect pathway in which epifauna affected disease by reducing epiphyte loads. We further quantified the effects of temperature and seagrass structure on epifauna and disease to explore the network of linkages that influence wasting disease in eelgrass meadows.

METHODS

Field surveys

We conducted surveys annually in midsummer to assess shoot densities, plant morphology, epifaunal communities, and seagrass wasting disease at 32 eelgrass meadows distributed across 23° of latitude from 2019 to 2021. We selected sampling locations based on the presence of continuous eelgrass at the mean lower low water line in 2019 within six geographic regions (Alaska, AK; British Columbia, BC; Washington, WA; Oregon, OR; Bodega Bay, California, BB; San Diego, California, SD, Appendix S1: Figure S1, Table S1). At each meadow, we established six 20-m intertidal transects, three in the upper and three in the lower intertidal. We resampled the same transects each year and always collected samples from the seaward side of the transect and counted shoot densities on the landward side.

For morphology and disease measurements, we sampled five shoots along each transect by harvesting an individual plant from the seaward side of the transect at 4-m intervals ($n = 30$/meadow). We bagged shoots individually and transported them on ice to the laboratory for further measurements. To characterize seagrass structure, we counted shoot densities in four replicate quadrats (0.0625–0.36 m^2) placed at 4-m intervals on the landward side the transect ($n = 24$/meadow). For epifauna measurements, we collected two grab samples per transect, at meter 4 and meter 16 ($n = 12$/meadow), by gently lowering a 0.5-mm mesh bag over a clump of eelgrass and associated macroalgae and breaking off shoots at the base into the bag. We repeated surveys in 2020 and 2021, with modifications at a few sites due to loss of meadow area and limitations resulting from the COVID-19 pandemic (Appendix S1: Section S1: Supplemental methods).

Eelgrass and wasting disease measurements

After returning to the laboratory, we measured canopy height (longest leaf length plus sheath length) and number of leaves for each shoot. We then separated the third-rank leaf at the top of the sheath bundle and measured epiphyte load on this leaf by gently scraping epiphytes onto pre-weighed foil tins and drying samples to constant mass at 60°C. After cleaning, we examined leaves visually and recorded the presence of invertebrate grazing scars, which were readily apparent on the cleaned leaves and indicated recent consumption of eelgrass tissue. Although gastropods and crustaceans leave distinct scars due to different modes of grazing, we did not distinguish between scar types and recorded only presence or absence of any grazing scars on

each leaf. We then placed the leaves between two sheets of transparent plastic film and scanned them at 600 dpi to create high-resolution images for disease analyses. We used the third-rank leaf because it integrates recent environmental conditions and has not deteriorated as much as older leaves that are too fragile to process. In Oregon, where third-rank leaves are often too fouled by epiphytes for imaging, infection rates were consistent between second- and third-ranked leaves (Aoki et al., 2022), so we measured epiphytes and disease on the second-rank leaf.

We used the Eelgrass Lesion Image Segmentation Application (EeLISA) to measure wasting disease in the images consistently across sites. EeLISA uses a convolutional neural network to classify healthy and diseased eelgrass tissue; we used the classifications to calculate disease prevalence (presence or absence of diseased tissue) and lesion area for each leaf. EeLISA was previously calibrated with samples across the geographic range of this study and validated with qPCR verification of pathogen identity (Aoki et al., 2022).

Epifaunal community measurements

We processed epifauna samples immediately after collection by emptying each bag into a sorting tray and carefully removing the mobile epifauna from macrophytes. We sorted macrophytes into eelgrass and macroalgae, gently squeezed to remove excess water, and recorded the wet mass. We passed the remaining contents of the tray through a mesh sieve (0.5- or 1.0-mm mesh depending on the site and year; Appendix S1: Table S1) and transferred material remaining on the sieve to a plastic 20-mL scintillation vial filled with 70% ethanol until further processing.

We size-sorted the epifauna by pouring vials through eight nested sieves of decreasing mesh size (5.6–0.5 mm) followed by gentle rinsing with fresh water. We carefully removed animals from the sieves with forceps, placed them into petri dishes of 70% ethanol, and counted and identified them using a dissecting microscope. For consistency across sites that used different mesh sizes in the initial processing, we excluded animals in the last two sieves (0.71- and 0.5-mm mesh) from this study. We identified each invertebrate to the lowest level of taxonomic certainty (species where possible). One individual made all identifications except for samples from Bodega Bay, where epifauna were also not size-sorted. We standardized counts by the combined wet biomass of eelgrass and macroalgae for total epifauna and for three eelgrass grazers of interest: *Lacuna* spp. gastropods, idoteid isopods, and ampithoid amphipods. These taxa are known to consume live eelgrass (Table 1), as well as eelgrass detritus, leaving visible grazing scars, and are widely distributed in meadows across the northeastern Pacific. Recent laboratory studies have shown that *Lacuna* spp., the amphipod *Ampithoe lacertosa*, and two isopods, *Pentidotea wosnesenskii* and *Pentidotea resecata*, consume both live and diseased eelgrass, creating grazing scars comparable to the scars observed in our surveys. In these studies, wasting disease infections established faster and more frequently in grazer-wounded tissue than in ungrazed tissue (Graham et al., 2025; Murray et al., 2024). While other taxa, including limpets, also graze eelgrass, we focused on these taxa as common across the study range and having the link between grazing and wasting disease validated through laboratory studies.

Characterization of pathogen loads in field-collected lesions

At a subset of meadows, we used qPCR to assess presence of *L. zosterae* DNA in 5–10 lesion samples (see Appendix S1 for methods). For lesion samples with detectable *L. zosterae* DNA, we also analyzed paired green tissue samples. In 2020, we analyzed lesions from six sites across three regions; in 2021, we analyzed lesions from 22 sites (at least three sites per region).

Sea surface temperature

To investigate the effect of temperature on eelgrass disease, we accessed 1-km gridded daily sea surface temperature (SST) records. These records are reasonable proxies for temperature differences between sites, and long-term records enabled calculation of thermal anomalies (Aoki et al., 2022). We accessed two products: MUR (JPL MUR MEaSUREs Project, 2015) and G1SST (Chao et al., 2009). By combining MUR and G1SST, we retrieved SST records for 27 of 32 sites in 2019 but only 21 sites in 2020 and 2021 (Appendix S1: Table S1); coastal pixels were often masked as land, especially for enclosed estuaries in Oregon and California. Due to these limitations, we ran SEMs with temperature as a predictor on a subset of the data and ran a regression analysis on the full dataset without temperature as a predictor.

Statistical analyses

Piecewise SEMs with temperature (2-year dataset)

We tested a network of proposed causal relationships among components of the eelgrass ecosystem using piecewise SEM (Grace, 2006; Lefcheck, 2016). Specifically, we tested whether epifaunal grazers influenced wasting disease directly or indirectly via grazing on epiphytes, and

TABLE 1 Summary of studies confirming consumption of live, diseased, and detrital eelgrass tissue by *Lacuna* snails, idoteid isopods, and ampithoid amphipods; NA indicates no assessment of that food type.

Reference	Location	Assay type[a]	Species	Consumption of offered eelgrass		
				Live	Detrital	Diseased
McConnaughey and McRoy (1979)	Izembek Lagoon, AK, USA	Stable isotope analysis	*Lacuna variegata*[b]	Yes	Yes	NA
Thom et al. (1995)	Puget Sound, WA, USA	No-choice feeding trial	*Idotea resecata*[b]	Yes	NA	NA
Tomas et al. (2011)	Bodega Bay, CA, USA	No-choice feeding trial	*Idotea resecata*[b]	Yes	NA	NA
Best and Stachowicz (2013)	Bodega Bay, CA, USA	No-choice feeding trial	*Ampithoe dalli*	Yes	No	NA
			Ampithoe sectimanus	No	Yes	NA
			Ampithoe valida[b]	Yes	Yes	NA
			Ampithoe lacertosa[b]	Yes	Yes	NA
			Idotea resecata[b]	Yes	Yes	NA
Reynolds et al. (2018)	Bodega Bay, CA, USA	Choice feeding trial	*Ampithoe lacertosa*[b]	Yes	Yes	NA
			Idotea resecata[b]	Yes	Yes	NA
Hernán et al. (2020)	Coos Bay, Yaquina Bay, OR, USA	No-choice feeding trial	*Pentidotea resecata*[b]	Yes	NA	NA
Namba and Nakaoka (2021)	Akkeshi Lagoon, Notoro Lagoon, Hokkaido, Japan	No-choice feeding trial	*Lacuna decorate*	Yes	NA	NA
Murray et al. (2024)	Bodega Bay, CA, USA	Choice feeding trial	*Pentidotea resecata*[b]	Yes	NA	Yes
Graham et al. (2025)	San Juan Islands, WA, USA	Choice feeding trial	*Pentidotea wosnesenskii*	Yes	NA	Yes
			Ampithoe lacertosa[b]	Yes	NA	Yes
			Lacuna sp.[b]	Yes	NA	Yes

[a]No-choice trials indicate only one type of biomass (live/detrital/diseased) was provided at a time; choice trials indicate more than one type of biomass was provided at the same time.
[b]The species/taxon was positively identified in the epifaunal samples analyzed in this study.

whether these effects varied between grazer taxa. The SEMs also included effects of temperature and seagrass structure that influence wasting disease. Following the multilevel study design, we tested the effects of temperature and seagrass structure on epifauna abundances at the meadow level, and we tested the effects of all predictors on leaf area, epiphyte load, grazing scar presence, and disease at the leaf level. Temperature predictors for each year were mean temperature in June (a metric of variation across latitudes) and cumulative positive temperature anomaly in June (a metric of warming previously associated with disease, Aoki et al., 2022). Seagrass predictors were shoot density and canopy height. The SEMs included exogenous variables of year and tidal height; component models were linear and generalized linear mixed-effects models with random intercepts for meadow and geographic region. Lesion area, leaf area, epiphyte load, and epifauna abundances were log-transformed to meet normality assumptions. Shoot density was log-transformed to improve model convergence.

Due to missing SST data, we limited SEM data to 44 meadow-year combinations from 2019 to 2021 ($n = 1307$ leaves, Appendix S1: Table S1). We constructed separate SEMs for disease prevalence (modeled with logistic regression) and lesion area; the lesion area model included only diseased leaves ($n = 572$) to isolate the effects of predictors on lesion area. For each disease metric, we constructed separate SEMs for each of four grazer abundances: total epifauna, *Lacuna* snail, ampithoid amphipod, and idoteid isopod. We modeled causal paths from the grazer abundances, and from the presence of grazing scars, to disease metrics to assess the direct effect of grazers on disease. Paths from grazer abundances to epiphyte load to disease indicated indirect effects. For each SEM, we assessed the global goodness-of-fit based on a χ^2 distributed Fisher's C statistic and tests of directed separation (d-sep); models had adequate fit with $p > 0.05$ for Fisher's C and d-sep tests showing no significant missing paths (Shipley, 2009; Appendix S1: Table S2). For models with adequate fit, we calculated standardized

path coefficients to compare the relative importance of predictors within that model; individual paths were considered significant for $p < 0.05$. We visualized effects of grazers on disease using partial effect plots and we ran post hoc comparison assess the effects of grazing scars within component models. Finally, we also ran SEMs using total grazer richness, rather than abundance; these models found no associations with disease metrics and results are not further reported.

Leaf-level SEM to test grazing direction

Given the multiple mechanisms likely linking disease and grazing, we used a simplified leaf-level SEM to test the direction of the relationship between grazing scar presence and disease. We compared models with the relationship directed from grazing scar presence to disease metrics to alternate models with the relationship directed from disease metrics to grazing scar presence. Leaf area was a predictor of both disease and grazing scar presence, and tidal height and year were predictors of all variables. Models also included random effects for region and meadow. We assessed the SEMs as above (Appendix S1: Table S11) and compared the alternate models based on the global Akaike information criterion (AIC), standardized coefficients, and variation explained by fixed effects.

Taxa comparison without temperature (3-year dataset)

Across the full dataset, we modeled epifauna relationships with disease prevalence using logistic regression and with lesion area using linear regression. These models included only seagrass structure, epifauna abundances, and individual leaf area as predictors, allowing us to include all epifauna and disease data ($n = 2370$ leaves from 82 site-by-year combinations in the prevalence model; $n = 1088$ diseased leaves from 78 site-by-year combinations in the lesion area model). The models also included year as a fixed effect and meadow and region as random effects. We compared candidate models (Appendix S1: Tables S12 and S13) using AIC to determine the best-fitting models, for which we calculated standardized coefficients to compare effect sizes of predictors.

RESULTS

SEMs with temperature (2-year dataset)

The SEMs found strong associations between epifaunal invertebrate grazers and disease across latitudes. Direct positive paths linked total epifauna abundance to both wasting disease prevalence and lesion area (Figure 1). Furthermore, the taxon-specific SEMs showed that paths between grazer abundances and disease varied between epifaunal grazers (Figure 2; Appendix S1: Figure S3). Disease prevalence and lesion area increased with abundances of both *Lacuna* snails and idoteid isopods. Ampithoid amphipod abundance was not clearly linked to either metric, though partial residuals showed a marginal pattern of decreasing lesion area with increasing amphipod abundance.

Across meadow-scale SEMs, disease metrics increased with grazing scar presence (Appendix S1: Tables S3–S10). Leaves with grazing scars were 1.8× more likely to be infected than ungrazed leaves (probability of infection was 35% for ungrazed leaves and 64% for grazed leaves, Figure 3; proportion of infected leaves increased by $29 \pm 6\%$ (95% CI) with grazing, $\chi^2 = 93.054$, $df = 1$, p-value < 0.0001). Post hoc comparison showed that infected leaves with scars had larger lesions than infected leaves without scars ($t = -3.13$, $df = 560$, $p = 0.0018$); lesion area was 0.94 ± 0.37 mm^2 and 0.61 ± 0.23 mm^2 on grazed and ungrazed leaves, respectively (mean ± SE). Grazing scars on individual leaves were more common where *Lacuna* snails were abundant but were not sensitive to abundances of other taxa (Appendix S1: Figure S2a,b).

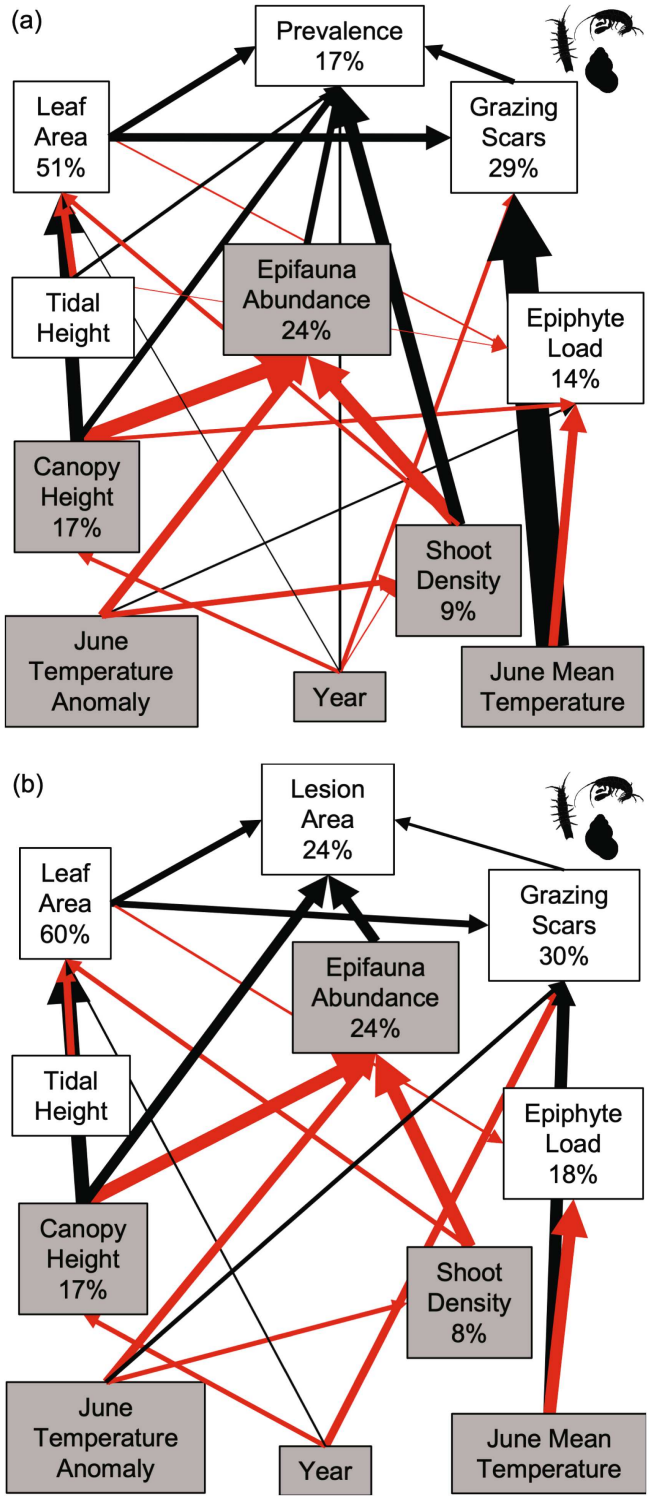
The leaf-level SEMs were more consistent with the hypothesis that the presence of grazing scars increased disease prevalence than the reverse (Appendix S1: Table S11, Figure S3). Directing the relationship from grazing scars to prevalence had a lower AIC than the reverse direction, indicating a better fit to the data ($\Delta AIC = 3.68$). The model with the path directed from grazing to prevalence also explained more variation in prevalence than the amount of variation in grazing scars explained by the model with the path reversed. Standardized coefficients were similar between the two models. For lesion area, AIC was equivalent, and variance explained and standardized coefficients were similar between the alternate models. Thus, while the positive relationship between grazing scars and lesion area is clear, the direction of causation is not.

In contrast to direct paths from epifauna abundances and grazing scars to disease, we found no evidence that epifauna affected disease indirectly by reducing epiphytes. Total epifauna abundance did not affect epiphyte load (Figure 1; Appendix S1: Table S3), likely because many epifauna species do not consume seagrass epiphytes (see *Epifauna distributions*). In the taxon-specific SEMs, *Lacuna* snails, ampithoid amphipods, and idoteid isopod abundances all weakly but significantly reduced epiphyte load (Appendix S1: Figure S2, Tables S4–S6). However, the path from epiphyte load to disease was never significant.

In the total epifauna SEM, epifauna abundances per g of macrophytes decreased with increasing seagrass

structure variables (larger canopy height and greater shoot density) while disease prevalence increased. Lesion area was only sensitive to canopy height. Abundances of specific taxa were not sensitive to either seagrass structure metric in this analysis (Appendix S1: Figure S2), which may result from standardizing abundances across combined seagrass and macroalgae wet mass.

Temperature had taxon-specific effects on epifauna. Ampithoid amphipod abundances increased with June mean temperature and decreased with June temperature anomaly, and this analysis likely underestimates their temperature sensitivity due to lack of SST data in 2021 at some sites with high amphipod abundances. *Lacuna* and idoteid abundances were not associated with either temperature metric. Given latitudinal distributions of these taxa (see below), lack of temperature effects may result from limited SST data, especially in the warmer regions (see *Methods*). Total epifauna abundance decreased with June temperature anomaly. The strongest effect across models was the increase in grazing scar presence with June mean temperature, while neither temperature metric had direct effects on disease prevalence or lesion area.



Grazer comparison without temperature (3-year dataset)

For disease prevalence, the best-fitting model included effects of seagrass structure, leaf area, and epifauna taxa abundances, while for lesion area, the best-fitting model included only leaf area and epifauna abundances (Appendix S1: Tables S12 and S13). For prevalence, leaf area, shoot density, and *Lacuna* snail abundance had comparable effect sizes, indicating an increase in disease risk of similar magnitude associated with the observed variation in each predictor. For lesion area, *Lacuna* snail abundance and leaf area had similar effect sizes, while idoteid isopod abundance had a smaller effect (Figure 4). Decreasing lesion area was marginally associated with increasing amphipod abundance. Disease varied significantly among years. Models explained ~12% of variation through fixed effects; with random effects, the models explained 41% and 25% of variation in prevalence and lesion area, respectively.

FIGURE 1 In structural equation modeling path analysis, total epifaunal abundance was a significant and positive predictor of both (a) disease prevalence ($n = 1307$ leaves) and (b) lesion area ($n = 597$ leaves), indicating that wasting disease increased with larger populations of epifauna. Gray variables were measured at the meadow scale; white variables were measured at the leaf scale. Solid lines indicate significant paths ($p < 0.05$), with positive and negative coefficients in black and red, respectively. Line width is proportional to the standardized coefficients (values in Appendix S1: Tables S3 and S7). Variance explained is shown for each endogenous variable. Images adapted from Phylopic.

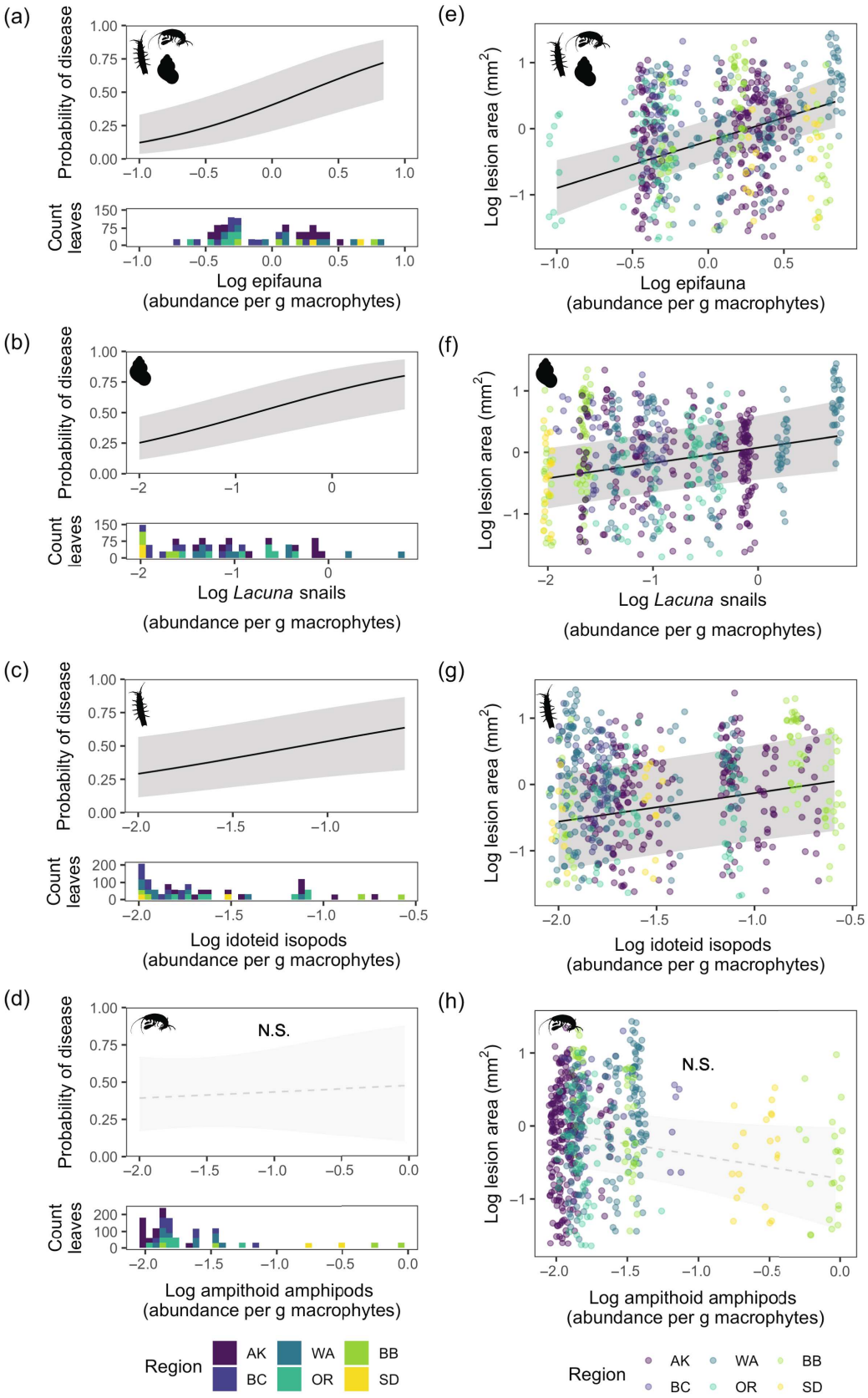


FIGURE 2 Legend on next page.

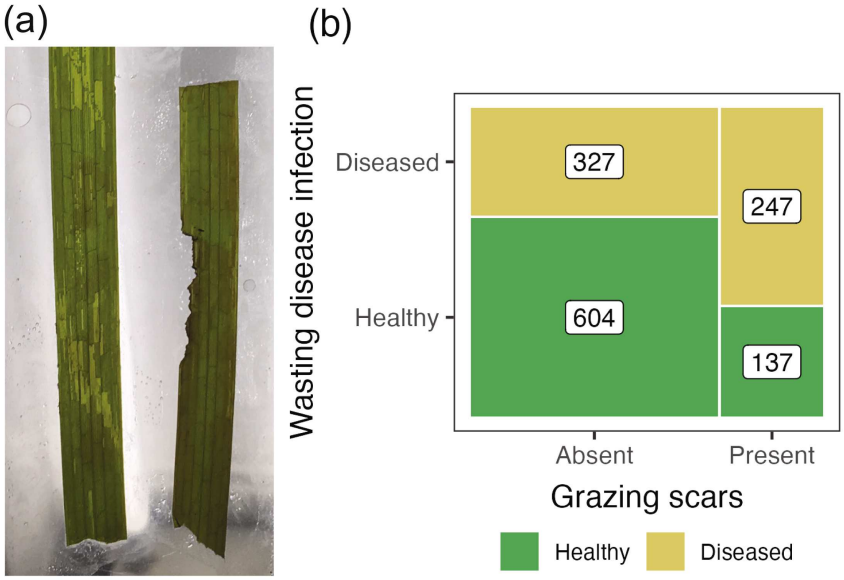


FIGURE 3 (a) Evidence of snail grazing (left) that damages leaf surfaces in contrast to crustacean grazing (right) that consumes the full thickness of the leaf tissue (photo credit Lillian R. Aoki). (b) Across all meadows and years, leaves with grazing scars were more likely to be diseased; labels show counts of leaves in each category and box widths are proportional to the count (total $n = 1351$).

Epifauna distributions

Populations of invertebrate eelgrass grazers showed contrasting geographic trends (Appendix S1: Figure S4). *Lacuna* snails were more abundant at higher latitudes while ampithoid amphipods were more abundant at lower latitudes. Idoteid isopods abundances were lower and variable throughout the range, with maximum abundances at meadows in the Bodega Bay region. Besides these three taxa of interest, epifauna included many other amphipods, gastropods, and polychaetes. Across all meadows and years, *Lacuna* snails, idoteid isopods, and ampithoid amphipods were on average 25% of the total epifauna community by number of individuals (Appendix S1: Figure S5). Meadows where these three taxa of eelgrass grazers were >50% of the total epifauna count generally had less than one animal per g of macrophytes.

Pathogen loads in lesioned tissue

We detected *L. zosterae* DNA in lesioned tissue samples collected in every region (Appendix S1: Figure S8). Similar to prior studies (Bockelmann et al., 2012; Schenck et al., 2023), detectable *L. zosterae* DNA was not present in every lesion sample, likely due to nonuniform pathogen distribution in lesion tissue (Muehlstein, 1992). The mean pathogen load was 71.4 ± 22.4 cells mg dry mass^{-1}; pathogen loads were highest in the Bodega Bay region and were also high at sites in Washington and Oregon. No *L. zosterae* DNA was detected in green tissue samples.

DISCUSSION

Our results show that the wasting disease widespread in the coastal foundation species eelgrass is positively associated with grazing by small invertebrates across an extensive geographic range (Figure 5). Both prevalence and lesion area of infections increased with increasing abundances of total epifauna, *Lacuna* snails, and idoteid isopods. The presence of invertebrate grazing scars on eelgrass leaves was associated with an 80% greater likelihood of disease (Figure 3), and path analysis supported a direct pathway from grazing scars to increased

FIGURE 2 Partial correlations from the taxon-specific structural equation modeling component models showed different relationships between wasting disease and abundances of specific grazer taxa. Plots show the estimated effect of the grazer abundance while other predictors of disease are held fixed; gray area indicates the 95% CI. For prevalence (a–d), distributions of grazer abundances are shown in the lower panel; for lesion area (e–h), partial residuals are plotted with jitter for improved visibility. N.S. and dashed line indicate nonsignificant correlations ($p > 0.05$). AK, Alaska; BB, Bodega Bay, California; BC, British Columbia; OR, Oregon; SD, San Diego, California; WA, Washington.

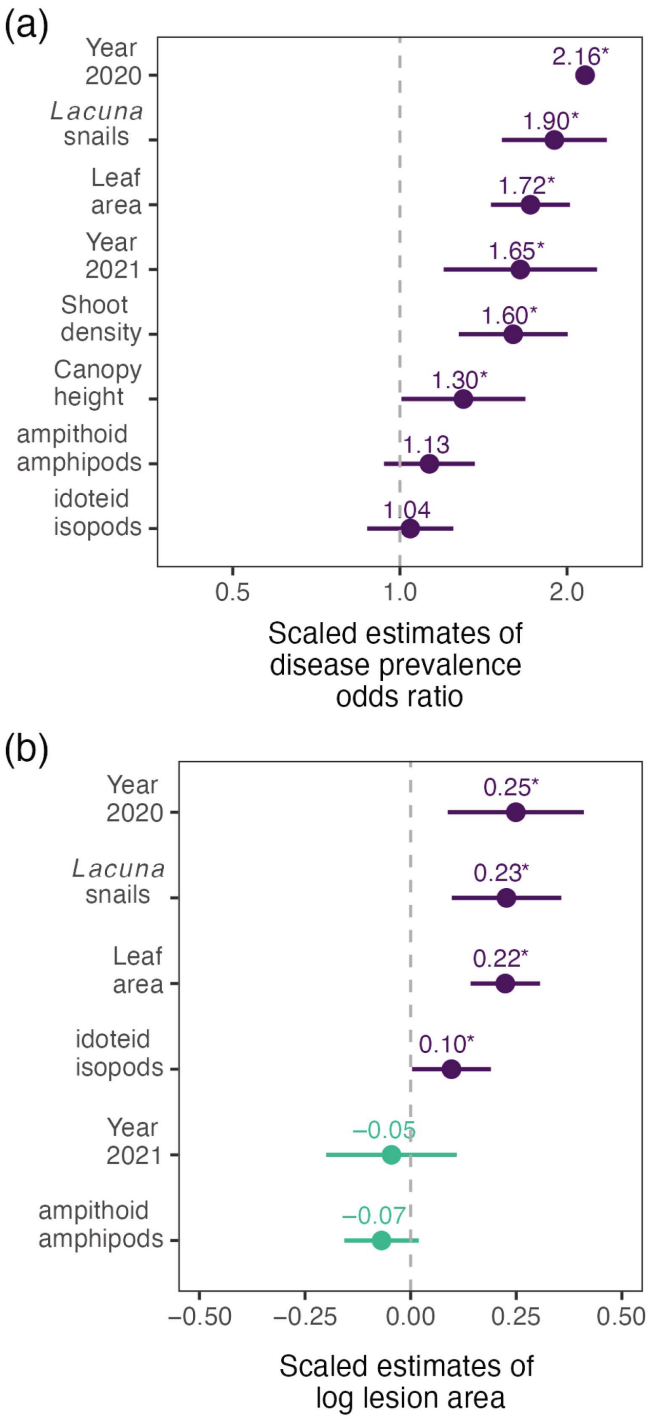


FIGURE 4 Standardized effect sizes for best-fitting models of disease prevalence (a) and lesion area (b); points indicate the positive (purple) or negative (teal) modeled value with the 95% CI. Gray dashed lines indicate zero effect size; effects are significant if CIs do not overlap with the zero line (asterisks). Year effects indicate change relative to 2019. Images adapted from Phylopic.

prevalence. This association between grazing scar presence and disease is particularly informative because it indicates that grazer consumption of eelgrass tissue is a relevant predictor of wasting disease in the field. Path analysis also confirmed a positive association between grazing scars and lesion area but did not confirm whether larger lesions were a cause or consequence of grazing. These results demonstrate that invertebrate grazers likely influence the extent of wasting disease in eelgrass meadows and highlight the complex interactions between plants, pathogens, and herbivores that warrant more attention in a changing world.

Multiple mechanisms likely link epifaunal grazers and wasting disease, including physical damage creating entry points for the pathogen and altered resource allocation in response to herbivory increasing susceptibility to infection (Boyd et al., 2022; Thaler et al., 2010). Distinguishing these mechanisms is challenging, but recent evidence confirms that invertebrate grazers can facilitate wasting disease infection. Field surveys in the Washington region revealed that grazing scar presence increased disease prevalence by a factor of 1.7 (Graham et al., 2025), similar to the increase by a factor of 1.8 across a broader geographic range in this study. Leaf tissue exposed to snail and isopod grazing and inoculated with wasting disease in lab incubations developed infections that were more common, were more intense, and established more quickly compared with ungrazed leaves (Graham et al., 2025; Murray et al., 2024). Live *L. zosterae* has also been cultured from herbivore feces (Graham et al., 2025), suggesting another pathway that may alter transmission. These lab experiments demonstrated that wounding by grazers facilitates subsequent infection, and this study confirms a consistent, positive association between grazing and disease levels across 23° of latitude.

A key finding from this study is that grazer effects on seagrass wasting disease varied by grazer taxon. *Lacuna* snails had the strongest association, as shown by the larger effect sizes in both the SEMs and regression models. Differentiating grazers is important because feeding modes likely affect disease transmission. Snails rasp the surface of leaves, weakening tissue and creating opportunities for infection. Isopods and amphipods, in contrast, feed along the edges of leaves and consume the full thickness of the leaf, which may not provide the same entry opportunities for the pathogen (Figure 3). Grazing scars were positively correlated with snail abundances in this study, but not with other taxa abundances, reinforcing snails' stronger disease effect compared with other taxa. Preferential wounding of green tissue by *Lacuna* snails (Graham et al., 2025) could contribute to the stronger association between snail abundances and disease by creating new opportunities for infection. The stronger association between *Lacuna* snails and disease compared with other taxa suggests that taxon-specific traits and feeding behaviors, as well as abundances, influence disease dynamics.

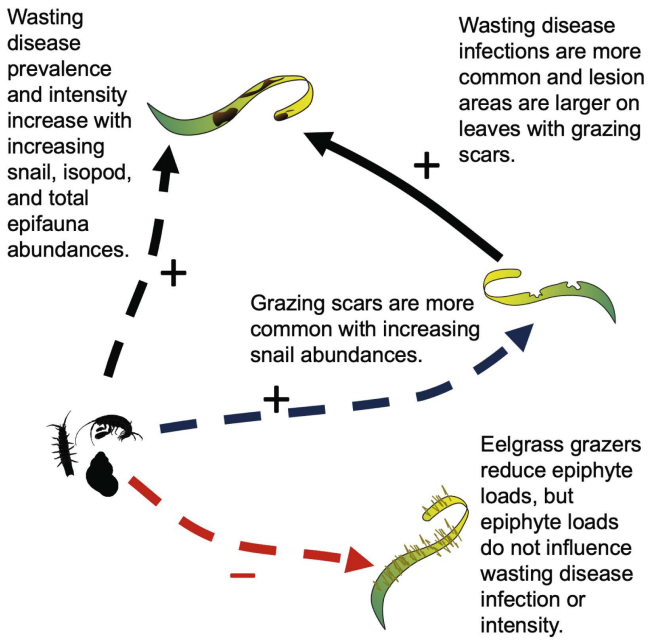


FIGURE 5 Conceptual model showing the key pathways through which grazer abundances influence seagrass wasting disease through both positive (black) and negative (red) relationships. Relationships that were consistent across analyses of combined total epifauna abundances and each individual taxon are solid lines; relationships that varied by taxon are dashed lines. Images adapted from Phylopic and Diana Kleine, Marine Botany, UQ (ian.umces.edu/media-library).

Differential effects of different grazer taxa likely contributed to geographic variation in eelgrass infection rates since *Lacuna* snails were more abundant at northern latitudes while ampithoid amphipods were more abundant at lower latitudes (Appendix S1: Figure S4). The overall greater abundance of *Lacuna* snails, up to 64× greater than amphipods at specific sites, likely explains their stronger relationship with both disease prevalence and lesion area across all years and sites compared with other taxa (Figures 2 and 4). Thus, in meadows with low abundances of *Lacuna* snails, such as the southern sites from 32° to 38° N in this study, grazing may have less influence on disease compared with sites with high snail abundances above 38° N. The pattern of increasing gastropod and decreasing crustacean abundance with increasing latitude occurs in eelgrass beds throughout the Atlantic and Pacific oceans (Gross et al., 2024), suggesting that grazer effects on eelgrass disease might vary with latitude in general. Unpacking this relationship requires a better understanding of how grazer traits like feeding mode and tissue preference are distributed for a broader range of taxa that consume eelgrass tissue beyond the focal taxa of this study.

While our results suggest that grazing facilitates disease, grazers may also seek out infected tissue and decrease disease load. Senescent or lesioned eelgrass tissue attracts epifauna due to reduced toughness and decreased phenolic content (Murray et al., 2024; Reynolds et al., 2018); lesions also offer enhanced nutritional quality through high concentrations of fatty acids, produced by *L. zosterae* (Yoshioka et al., 2019). However, not all grazers prefer diseased tissue (Graham et al., 2025). Determining causality from this dataset is challenging; one limitation is that epifauna were collected from intact shoots, including senescent leaves that might attract grazers, while disease was measured only on third-rank leaves, largely green tissue except for wasting disease lesions. While our data support a directed association from grazing scars to prevalence, indicating disease facilitation as confirmed in laboratory experiments, our data equally support both directions of the association between grazing scars and lesion area. Both preferential feeding on lesions and grazer-caused infections would contribute to the observed positive associations between grazer abundances, scars, and lesion area in the field. Plant palatability and defense strategies also vary among plant genotypes and with latitude (Hernán et al., 2020; Reynolds et al., 2018), therefore likely affecting grazer feeding preferences (Tomas et al., 2011). Further understanding of the role of epifaunal grazers in controlling seagrass wasting disease will require assessment of variation among both grazers and plants across spatial scales.

Contrary to our expectations, this study did not provide evidence that grazing on epiphytes influenced wasting disease. Invertebrate grazing of epiphytes reduces shading and facilitates eelgrass growth (Baden et al., 2010; Östman et al., 2016; Reynolds et al., 2014), and this analysis found weak negative correlations between specific grazer abundances and epiphyte load (Appendix S1: Figure S2), confirming that grazing generally reduced epiphytes. However, there was no association between epiphyte load and either disease prevalence or lesion area, suggesting that epiphyte overgrowth was not a driver of disease across latitudes. Therefore, conditions that alter epiphyte load, such as nutrient availability, do not necessarily translate to altered disease levels. Instead, the analysis supports a direct pathway from epifauna abundances to disease, reinforcing the positive relationship between the presence of grazing scars (consumption of eelgrass tissue) and disease (Figure 5).

In this study, disease prevalence increased with seagrass shoot density and canopy height across latitudes, likely due to increased physical contact between diseased and healthy tissue facilitating transmission in denser and/or taller meadows (Muehlstein, 1992). However, seagrass structure also indirectly affected disease by negatively affecting epifauna counts per unit of shoot biomass; that is, with increased shoot density,

there were fewer animals per gram of leaf tissue. This negative relationship led in turn to a negative indirect effect of seagrass shoot density and canopy height on disease prevalence. Contrasting pathways perhaps explain why shoot density has been both positively (Groner et al., 2016) and negatively (Graham et al., 2023) correlated with wasting disease infections in prior studies. The balance of these direct and indirect effects of seagrass structure may depend on the strength of other biotic interactions, including grazing.

Temperature influenced grazing in eelgrass meadows, as evidenced by lower total epifauna abundances associated with warm temperature anomalies, with potential consequences for disease. Ampithoid amphipods were more sensitive to warming than other taxa; warming might therefore accentuate taxon-specific influence on disease. The strong positive association between mean June temperatures and grazing scar presence suggests greater consumption of eelgrass tissue in warmer conditions, which in turn could compound increases in disease under warming. In general, this analysis highlighted the indirect effects of temperature on wasting disease, with temperature influencing seagrass structure, epifauna, and grazing scars but not directly influencing disease (Figure 1). This result is somewhat surprising, given prior findings linking disease levels to summer temperature anomalies in this region (Aoki et al., 2022), as well as increased wasting disease in mesocosm warming experiments (Breiter et al., 2024; Kaldy, 2014). However, this temperature analysis is limited, while the SST records accommodated the use of temperature anomalies, some warmer sites lacked appropriate SST data in 2021 (see *Methods*). The 1-km SST pixels also did not account for differences in exposure to air temperatures at low tide between sites. Furthermore, temperature relationships with disease are likely complex. Seasonal temperatures beyond the specific metrics tested here affect seagrass growth, wasting disease, and epifauna abundances (Graham et al., 2023; Groner et al., 2021; Ha & Williams, 2018); *L. zosterae* growth dynamics are underexplored in the field but are sensitive to temperature in laboratory studies (Dawkins et al., 2018). While this analysis suggests that a biological interaction, that is, grazing, had a stronger direct effect on wasting disease compared with the direct temperature effect, temperature does impact each component of this plant-pathogen-herbivore system. Given the network of effects documented here, changes to grazing rates and eelgrass morphology under warming are likely to propagate to affect wasting disease, in addition to any direct temperature effects not captured here.

This study highlights the role of epifaunal grazers in facilitating eelgrass wasting disease in the field, showing for the first time how grazing contributes to diminished eelgrass health under natural conditions. Prior studies have emphasized how epifaunal grazers benefit eelgrass by removing epiphytic algae that shade the plants (Reynolds et al., 2014). However, this study demonstrates a contrasting ecological role, with herbivory by epifauna associated with the spread of wasting disease. These different ecological roles occur simultaneously, as demonstrated in this analysis with higher grazer abundances associated with both lower epiphyte loads and higher disease levels (Appendix S1: Figure S3). Local context likely determines the balance between these contrasting pathways; in particular, robust abundances of *Lacuna* snails at higher latitudes appear to drive stronger disease amplification from epifaunal grazing in those locations. Local conditions, including abiotic factors like climate but also biotic characteristics such as eelgrass genetic diversity and the presence of invasive grazer species, shape eelgrass response to stressors (DuBois et al., 2022; Schenck et al., 2023); these factors likely modulate the grazing-disease interaction. Although the total proportion of eelgrass biomass consumed by epifaunal grazers is low, the epifauna grazing interaction with eelgrass disease is ecologically relevant because it affects a foundation species' health.

By demonstrating the positive association between grazing and disease in eelgrass, this study motivates a more complete understanding of plant-pathogen-herbivore dynamics in a critical habitat. The causative agent of seagrass wasting disease is distributed in eelgrass meadows globally (Martin et al., 2016), yet the conditions that trigger disease outbreaks and related meadow declines remain poorly understood. Recent laboratory experiments showed that epifauna grazing facilitates disease (Graham et al., 2025; Murray et al., 2024), and this analysis further suggests that grazer identity, feeding mode, and diet preference, affect the interaction. Assessment of eelgrass grazing, such as by quantifying grazing scars, may therefore provide insight into the conditions that enable wasting disease outbreaks and the vulnerability of specific meadows to infection. These insights are needed to advance regional conservation goals, such as the prioritization of resilient meadows in protected area networks (Graham et al., 2024), and to develop a predictive understanding of wasting disease in ecologically valuable seagrass meadows.

Across ecosystems, consumer identity, feeding mode, and food preferences alter disease ecology. In coral reefs, many corallivores are associated with increased levels of disease, but some such as butterflyfish are also associated with decreased disease, likely due to their feeding mode and food preferences (Renzi et al., 2022). In salt marshes, both snails and crabs are associated with increased fungal disease, but only snails "farm" the fungus by

inoculating scars with their fecal pellets (Daleo et al., 2009; Silliman & Newell, 2003). In grasslands, different food preferences and grazing behaviors between cattle and sheep lead to contrasting effects on plant-pathogen loads, largely through indirect effects on plant communities (Li et al., 2024). Across these disparate habitats and taxonomic diversity of consumers, herbivory (or corallivory) remains a key biotic interaction influencing foundation species' disease levels, with consequences for ecosystem health. As we demonstrated for eelgrass meadows, grazing effects on disease are sensitive to environmental drivers and constrained by local factors. In an era of rapid environmental change, better understanding of the relative importance of grazing compared with other drivers of disease is needed to predict, identify, and respond to disease outbreaks.

AUTHOR CONTRIBUTIONS

J. Emmett Duffy, C. Drew Harvell, Ginny L. Eckert, Timothy L. Hawthorne, Margot Hessing-Lewis, Kevin Hovel, Fiona Tomas, and John J. Stachowicz conceptualized the study, with contributions from Lillian R Aoki, Olivia J. Graham, and Ryan S. Mueller. Brendan Rappazzo and Carla P. Gomes developed the AI tools. Lillian R Aoki, Deanna S. Beatty, J. Emmett Duffy, Lia K. Domke, Ginny L. Eckert, Olivia J. Graham, C. Drew Harvell, Eliza Heery, Margot Hessing-Lewis, Kevin Hovel, Karl Koehler, Zachary L. Monteith, Ryan S. Mueller, Angeleen M. Olson, Carolyn Prentice, Carmen J. Ritter, Collin Gross, John J. Stachowicz, Fiona Tomas, Bo Yang collected field data. Carmen J. Ritter led epifauna identifications. Lillian R Aoki led data analysis and drafted the manuscript. All authors contributed to the final version.

ACKNOWLEDGMENTS

We thank the many volunteers and field crews who assisted with data collection. South Slough National Estuarine Research Reserve supported sampling in Oregon. The Tula Foundation supported sampling by the Hakai Institute in British Columbia. We conducted this research on lands and waters of Kumeyaay, Coast Miwok, Tlingit and Haida, Heiltsuk and Wuikinuxv Nations, Coast Salish, the Confederated Tribes of Coos, Lower Umpqua and Siuslaw Indians, the Confederated Tribe of Siletz Indians, and other Indigenous peoples. This work was supported by the National Science Foundation (awards OCE-1829921, OCE-1829922, OCE-1829992, OCE-1829890, OCE-1635716, OCE-1600230, and GRFP). IMEDEA is an accredited "Maria de Maeztu Excellence Unit" (Grant CEX2021-001198, funded by MICIU/AEI/10.13039/501100011033). Fiona Tomas was supported by RESIGRASS (CGL2014-58829-C2-2-R). This is contribution number 150 from the Smithsonian's MarineGEO and Tennenbaum Marine Observatories Network.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data and code (lillian-aoki, 2024) are available in Zenodo at https://doi.org/10.5281/zenodo.10892511.

ORCID

Lillian R. Aoki https://orcid.org/0000-0002-2792-0523
Carmen J. Ritter https://orcid.org/0000-0002-6912-9262
Deanna S. Beatty https://orcid.org/0000-0002-4930-5505
Lia K. Domke https://orcid.org/0000-0003-1155-989X
Ginny L. Eckert https://orcid.org/0000-0002-7538-7081
Olivia J. Graham https://orcid.org/0000-0003-3834-264X
Carla P. Gomes https://orcid.org/0000-0002-4441-7225
Collin Gross https://orcid.org/0000-0002-0896-8476
Timothy L. Hawthorne https://orcid.org/0000-0003-4565-3745
Eliza Heery https://orcid.org/0000-0002-5318-8890
Margot Hessing-Lewis https://orcid.org/0000-0002-9956-955X
Kevin Hovel https://orcid.org/0000-0002-1643-1847
Zachary L. Monteith https://orcid.org/0000-0003-4963-8374
Ryan S. Mueller https://orcid.org/0000-0001-9565-5912
Angeleen M. Olson https://orcid.org/0000-0001-8443-1868
Carolyn Prentice https://orcid.org/0000-0002-9644-8496
Brendan Rappazzo https://orcid.org/0000-0003-3417-2372
John J. Stachowicz https://orcid.org/0000-0003-2735-0564
Fiona Tomas https://orcid.org/0000-0001-8682-2504
Bo Yang https://orcid.org/0000-0001-7439-192X
C. Drew Harvell https://orcid.org/0000-0002-8905-9847
J. Emmett Duffy https://orcid.org/0000-0001-8595-6391

REFERENCES

Alvarez-Filip, L., N. K. Dulvy, J. A. Gill, I. M. Côté, and A. R. Watkinson. 2009. "Flattening of Caribbean Coral Reefs: Region-Wide Declines in Architectural Complexity." *Proceedings of the Royal Society B: Biological Sciences* 276: 3019–25.

Aoki, L. R., B. Rappazzo, D. S. Beatty, L. K. Domke, G. L. Eckert, M. E. Eisenlord, O. J. Graham, et al. 2022. "Disease Surveillance by Artificial Intelligence Links Eelgrass Wasting Disease to Ocean Warming across Latitudes." *Limnology and Oceanography* 67: 1577–89.

Baden, S., C. Boström, S. Tobiasson, H. Arponen, and P.-O. Moksnes. 2010. "Relative Importance of Trophic Interactions and Nutrient Enrichment in Seagrass Ecosystems: A Broad-Scale Field Experiment in the Baltic–Skagerrak Area." *Limnology and Oceanography* 55: 1435–48.

Best, R. J., and J. J. Stachowicz. 2013. "Phylogeny as a Proxy for Ecology in Seagrass Amphipods: Which Traits Are Most Conserved?" *PLoS One* 8: e57550.

Bockelmann, A., K. Beining, and T. Reusch. 2012. "Widespread Occurrence of Endophytic *Labyrinthula* spp. in Northern European Eelgrass *Zostera marina* Beds." *Marine Ecology Progress Series* 445: 109–116.

Boyd, A. D., N. S. Walker, S. R. Valdez, Y. S. Zhang, A. H. Altieri, V. Gulis, C. Crain, and B. Silliman. 2022. "Invertebrate Grazing on Live Turtlegrass (*Thalassia testudinum*): A Common Interaction that May Facilitate Fungal Growth." *Frontiers in Marine Science* 8: 789380.

Breiter, A. L., C. T. Sokoloski, S. Yang, and J. L. Gaeckle. 2024. "Effect of Prolonged Seawater Warming on *Zostera marina* Ecotypes of the Northeast Pacific." *Journal of Experimental Marine Biology and Ecology* 578: 152036.

Cappelli, S. L., and J. Koricheva. 2021. "Interactions between Mammalian Grazers and Plant Pathogens: An Elephant in the Room?" *New Phytologist* 232: 8–10.

Chao, Y., Z. Li, J. D. Farrara, and P. Hung. 2009. "Blending Sea Surface Temperatures from Multiple Satellites and In Situ Observations for Coastal Oceans." *Journal of Atmospheric and Oceanic Technology* 26: 1415–26.

Daleo, P., B. Silliman, J. Alberti, M. Escapa, A. Canepuccia, N. Peña, and O. Iribarne. 2009. "Grazer Facilitation of Fungal Infection and the Control of Plant Growth in South-Western Atlantic Salt Marshes." *Journal of Ecology* 97: 781–87.

Dawkins, P., M. Eisenlord, R. Yoshioka, E. Fiorenza, S. Fruchter, F. Giammona, M. Winningham, and C. Harvell. 2018. "Environment, Dosage, and Pathogen Isolate Moderate Virulence in Eelgrass Wasting Disease." *Diseases of Aquatic Organisms* 130: 51–63.

DuBois, K., K. N. Pollard, B. J. Kauffman, S. L. Williams, and J. J. Stachowicz. 2022. "Local Adaptation in a Marine Foundation Species: Implications for Resilience to Future Global Change." *Global Change Biology* 28: 2596–2610.

Duffy, J. E., A. R. Hughes, and P.-O. Moksnes. 2013. "Ecology of Seagrass Communities." In *Marine Community Ecology and Conservation*, edited by M. D. Bertness, J. F. Bruno, B. R. Silliman, and J. J. Stachowicz, 271–297. Sunderland, MA: Sinauer Associates, Inc.

Ellison, A. M., M. S. Bank, B. D. Clinton, E. A. Colburn, K. Elliott, C. R. Ford, D. R. Foster, et al. 2005. "Loss of Foundation Species: Consequences for the Structure and Dynamics of Forested Ecosystems." *Frontiers in Ecology and the Environment* 3: 479–486.

García-Guzmán, G., and R. Dirzo. 2001. "Patterns of Leaf-Pathogen Infection in the Understory of a Mexican Rain Forest: Incidence, Spatiotemporal Variation, and Mechanisms of Infection." *American Journal of Botany* 88: 634–645.

Grace, J. B. 2006. *Structural Equation Modeling and Natural Systems*. Cambridge: Cambridge University Press.

Graham, O. J., L. R. Aoki, C. Burge, and C. D. Harvell. 2025. "Invertebrate Herbivores Influence Seagrass Wasting Disease Dynamics." *Ecology* 106: e4493.

Graham, O. J., D. Harvell, B. Christiaen, J. Gaeckle, L. R. Aoki, B. Ratliff, A. Vinton, B. H. Rappazzo, and T. Whitman. 2024. "Taking the Pulse of Resilience in Conserving Seagrass Meadows." *Integrative and Comparative Biology* 64: 816–826.

Graham, O. J., T. Stephens, B. Rappazzo, C. Klohmann, S. Dayal, E. M. Adamczyk, A. Olson, et al. 2023. "Deeper Habitats and Cooler Temperatures Moderate a Climate-Driven Seagrass Disease." *Philosophical Transactions of the Royal Society, B: Biological Sciences* 378: 20220016.

Groner, M., C. Burge, C. Kim, E. Rees, K. Van Alstyne, S. Yang, S. Wyllie-Echeverria, and C. Harvell. 2016. "Plant Characteristics Associated with Widespread Variation in Eelgrass Wasting Disease." *Diseases of Aquatic Organisms* 118: 159–168.

Groner, M., M. Eisenlord, R. Yoshioka, E. Fiorenza, P. Dawkins, O. Graham, M. Winningham, et al. 2021. "Warming Sea Surface Temperatures Fuel Summer Epidemics of Eelgrass Wasting Disease." *Marine Ecology Progress Series* 679: 47–58.

Gross, C. P., J. E. Duffy, K. A. Hovel, P. L. Reynolds, C. Boström, K. E. Boyer, M. Cusson, et al. 2024. "A Latitudinal Cline in the Taxonomic Structure of Eelgrass Epifaunal Communities Is Associated with Plant Genetic Diversity." *Global Ecology and Biogeography* 33: e13918.

Ha, G., and S. L. Williams. 2018. "Eelgrass Community Dominated by Native Omnivores in Bodega Bay, California, USA." *Bulletin of Marine Science* 94: 1333–53.

Hernán, G., M. J. Ortega, J. Henderson, J. Alós, K. Boyer, S. Cimon, V. Combes, et al. 2020. "Latitudinal Variation in Plant Defence against Herbivory in a Marine Foundation Species Does Not Follow a Linear Pattern: The Importance of Resource Availability." *Global Ecology and Biogeography* 30: 220–234.

JPL MUR MEaSUREs Project. 2015. "GHRSST Level 4 MUR Global Foundation Sea Surface Temperature Analysis." Ver. 4.1. PO. DAAC, CA, USA.

Kaldy, J. E. 2014. "Effect of Temperature and Nutrient Manipulations on Eelgrass *Zostera marina* L. from the Pacific Northwest, USA." *Journal of Experimental Marine Biology and Ecology* 453: 108–115.

Lefcheck, J. S. 2016. "PIECEWISESEME: Piecewise Structural Equation Modelling in R for Ecology, Evolution, and Systematics." *Methods in Ecology and Evolution* 7: 573–79.

Li, T., E. Allan, S. Yang, Y. Liu, M. Inbar, D. Wang, and Z. Zhong. 2024. "Contrasting Effects of Sheep and Cattle Grazing on Foliar Fungal Diseases by Changing Plant Community Characteristics." *Functional Ecology* 38: 1172–84.

lillian-aoki. 2024. "lillian-Aoki/EelgrassEpifaunaDisease: Accepted for Publication (v1.1)." Zenodo. https://doi.org/10.5281/zenodo.14201457.

Liu, M., T. D. Mipam, X. Wang, P. Zhang, Z. Lin, and X. Liu. 2021. "Contrasting Effects of Mammal Grazing on Foliar Fungal Diseases: Patterns and Potential Mechanisms." *New Phytologist* 232: 345–355.

Martin, D. L., Y. Chiari, E. Boone, T. D. Sherman, C. Ross, S. Wyllie-Echeverria, J. K. Gaydos, and A. A. Boettcher. 2016. "Functional, Phylogenetic and Host-Geographic Signatures of Labyrinthula spp. Provide for Putative Species Delimitation and a Global-Scale View of Seagrass Wasting Disease." *Estuaries and Coasts* 39: 1403–21. https://doi.org/10.1007/s12237-016-0087-z

Maynard, J., R. van Hooidonk, C. M. Eakin, M. Puotinen, M. Garren, G. Williams, S. F. Heron, et al. 2015. "Projections of Climate Conditions that Increase Coral Disease Susceptibility and Pathogen Abundance and Virulence." *Nature Climate Change* 5: 688–694.

McConnaughey, T., and C. P. McRoy. 1979. "13C Label Identifies Eelgrass (*Zostera marina*) Carbon in an Alaskan Estuarine Food Web." *Marine Biology* 53: 263–69.

Muehlstein, L. K. 1992. "The Host – Pathogen Interaction in the Wasting Disease of Eelgrass, *Zostera marina*." *Canadian Journal of Botany* 70: 2081–88.

Murray, N. A., K. DuBois, and J. J. Stachowicz. 2024. "Herbivores Can Benefit both Plants and Their Pathogens through Selective Herbivory on Diseased Tissue." *Journal of Ecology* 112: 1413–24.

Namba, M., and M. Nakaoka. 2021. "Increased Salinity Stress Changes Plant Productivity and Biomass by Altering the Top-Down Controls in Eelgrass Beds." *Ecosphere* 12: e03852.

Östman, Ö., J. Eklöf, B. K. Eriksson, J. Olsson, P.-O. Moksnes, and U. Bergström. 2016. "Top-Down Control as Important as Nutrient Enrichment for Eutrophication Effects in North Atlantic Coastal Ecosystems." *Journal of Applied Ecology* 53: 1138–47.

Renzi, J. J., E. C. Shaver, D. E. Burkepile, and B. R. Silliman. 2022. "The Role of Predators in Coral Disease Dynamics." *Coral Reefs* 41: 405–422.

Reynolds, L. K., K. M. Chan, E. Huynh, S. L. Williams, and J. J. Stachowicz. 2018. "Plant Genotype Identity and Diversity Interact with Mesograzer Species Diversity to Influence Detrital Consumption in Eelgrass Meadows." *Oikos* 127: 327–336.

Reynolds, P. L., J. P. Richardson, and J. E. Duffy. 2014. "Field Experimental Evidence that Grazers Mediate Transition between Microalgal and Seagrass Dominance." *Limnology and Oceanography* 59: 1053–64.

Santini, A., and M. Faccoli. 2015. "Dutch Elm Disease and Elm Bark Beetles: A Century of Association." *iForest - Biogeosciences and Forestry* 8: 126–134. https://doi.org/10.3832/ifor1231-008

Schenck, F. R., K. DuBois, M. R. Kardish, J. J. Stachowicz, and A. R. Hughes. 2023. "The Effect of Warming on Seagrass Wasting Disease Depends on Host Genotypic Identity and Diversity." *Ecology* 104: e3959.

Shipley, B. 2009. "Confirmatory Path Analysis in a Generalized Multilevel Context." *Ecology* 90: 363–68.

Short, F. T., L. K. Muehlstein, and D. Porter. 1987. "Eelgrass Wasting Disease: Cause and Recurrence of a Marine Epidemice." *The Biological Bulletin* 173: 557–562.

Silliman, B. R., and S. Y. Newell. 2003. "Fungal Farming in a Snail." *Proceedings of the National Academy of Sciences* 100: 15643–48.

Thaler, J. S., A. A. Agrawal, and R. Halitschke. 2010. "Salicylate-Mediated Interactions between Pathogens and Herbivores." *Ecology* 91: 1075–82.

Thayer, G. W., W. J. Kenworthy, and M. S. Fonseca. 1984. "The Ecology of Eelgrass Meadows of the Atlantic Coast: A Community Profile." Report.

Thom, R., B. Miller, and M. Kennedy. 1995. "Temporal Patterns of Grazers and Vegetation in a Temperate Seagrass System." *Aquatic Botany* 50: 201–5.

Tomas, F., J. M. Abbott, C. Steinberg, M. Balk, S. L. Williams, and J. J. Stachowicz. 2011. "Plant Genotype and Nitrogen Loading Influence Seagrass Productivity, Biochemistry, and Plant–Herbivore Interactions." *Ecology* 92: 1807–17.

Yoshioka, R., J. Schram, and A. Galloway. 2019. "Eelgrass Pathogen *Labyrinthula zosterae* Synthesizes Essential Fatty Acids." *Diseases of Aquatic Organisms* 135: 89–95.

SUPPORTING INFORMATION

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

How to cite this article: Aoki, Lillian R., Carmen J. Ritter, Deanna S. Beatty, Lia K. Domke, Ginny L. Eckert, Olivia J. Graham, Carla P. Gomes, et al. 2025. "Seagrass Wasting Disease Prevalence and Lesion Area Increase with Invertebrate Grazing across the Northeastern Pacific." *Ecology* 106(1): e4532. https://doi.org/10.1002/ecy.4532