PHILOSOPHICAL TRANSACTIONS B

royalsocietypublishing.org/journal/rstb

Research

Cite this article: Tomamichel M, Walters T, Fox E, Rivera A, O'Hare M, Minniefield A, Braun M, Hall RJ, Frischer M, Byers J. 2026 Changes in transmission rates drive seasonal patterns of shrimp Black Gill disease. *Phil. Trans. R. Soc. B* **381**: 20240327.
https://doi.org/10.1098/rstb.2024.0327

Received: 30 January 2025
Accepted: 29 September 2025

One contribution of 16 to a theme issue 'Managing infectious marine diseases in wild populations'.

Subject Areas:
ecology

Keywords:
Hyalophysa lynni, disease mortality, marine infectious disease, marine parasites, disease prevalence, mechanistic model, disease model, disease transmission, disease in fisheries, manage marine disease

Author for correspondence:
Megan Tomamichel
e-mail: megan.tomamichel@gmail.com

Electronic supplementary material is available online at https://doi.org/10.6084/m9.figshare.c.8286581.

THE ROYAL SOCIETY PUBLISHING

Changes in transmission rates drive seasonal patterns of shrimp Black Gill disease

Megan Tomamichel[1,2], Tina Walters[5], Elianna Fox[1], Ashly Rivera[5], Mikaela O'Hare[5], Anita Minniefield[5], Max Braun[5], Richard John Hall[1,2,3], Marc Frischer[5,4] and James Byers[1,5]

[1]Odum School of Ecology, [2]Center for the Ecology of Infectious Disease, [3]Department of Infectious Diseases, College of Veterinary Medicine, and [4]Department of Marine Sciences, University of Georgia, Athens, GA 30602, USA
[5]Skidaway Institute of Oceanography, Savannah, GA 31411, USA

MT, 0000-0002-7903-6633; JB, 0000-0001-9240-0287

Quantifying the processes affecting disease dynamics is critical for informing management strategies. We present the results of a series of experiments and mechanistic models that disentangle the roles of disease transmission and host density, mortality and recovery in driving seasonal prevalence of shrimp Black Gill (sBG) disease. We first quantified seasonal sBG transmission to uninfected hosts deployed into the environment. Next, we manipulated temperature and infection status in a laboratory experiment to quantify drivers of host mortality and recovery. Finally, we utilized these experiments to parameterize a series of mechanistic models to determine whether disease dynamics were driven by host density-dependent or -independent processes. Transmission was highest during the summer, with 75–91% of hosts acquiring infection, but declined substantially in all other seasons (0–10%). In our laboratory experiment, we observed little disease-induced mortality and complete recovery from infection in all treatments. Our models revealed that host density did not drive disease dynamics. Together, this suggests that high transmission rates in the summer owing to seasonal changes in the environment, followed by gradual recovery when transmission rates are low, cause variation in sBG prevalence. Our methodology provides a framework to quantify drivers of seasonal variation in disease prevalence in fisheries.

This article is part of the theme issue 'Managing infectious marine diseases in wild populations'.

1. Introduction

Infectious diseases in marine systems can have large impacts on keystone and commercially fished species [1,2]. These impacts are predicted to increase in some systems owing to anthropogenic stressors and climate change, with critical implications for ecosystem function and human economics [3,4]. Seasonal changes in infectious disease prevalence (i.e. the fraction of the host population infected) can result from environmentally driven fluctuations in processes that increase the numbers of infected individuals in a population (e.g. transmission) and in those that remove them (e.g. infection-induced mortality and recovery) [5]. The management of infectious disease in fisheries and aquaculture often involves reducing transmission or host mortality or increasing host recovery from infection [6]. Because each of these management strategies requires very different methodologies to execute, understanding the relative contributions of transmission, recovery and host mortality

to observed seasonal infection patterns is crucial for designing effective management plans.

Mechanistic models of infectious disease dynamics are powerful tools that can be used to untangle the roles of transmission, recovery and mortality in determining population-level disease prevalence patterns [6]. These models frequently incorporate data from experiments and field-collected data to estimate key disease processes and host parameters and to assess model fit [7]. Some additionally include time- or temperature-varying components to simulate seasonality in environmental conditions that affect transmission processes [8]. Most frequently, mechanistic models of disease in fisheries are used to analyse the efficacy of harvest-based strategies to mitigate the spread of infectious disease [6]. However, these efforts often assume density-dependent transmission, which is usually a requirement for culling-based disease control to be effective [7]. Therefore, to build a model that accurately captures seasonally variable disease dynamics to inform management, it is necessary to establish (i) whether disease processes (i.e. transmission, mortality and recovery) vary seasonally and (ii) whether seasonal variation in these disease processes is driven by changing environmental conditions and/or host population density.

In marine systems, environmental conditions that change seasonally can influence multiple components of transmission, from survival of external pathogen stages to host susceptibility and density. A hospitable environment can increase transmission by allowing infectious particles to travel large distances through oceanic currents to reach hosts that are heterogeneously distributed [9–11]. Conversely, unfavourable environmental conditions can prevent infectious disease from spreading by reducing the viability of external infectious particles, thereby preventing transmission to a susceptible host [12–14]. Environmental factors also influence host susceptibility to, and tolerance of, infection. Hosts experiencing environmental stress can become increasingly susceptible to infection owing to an insufficient immune response or from damage to host tissues allowing for pathogen entry [15,16]. The ability of hosts to tolerate severe infections can also be modified by the environment [17,18]. Highly tolerant infected hosts serve as sources of infectious agents and therefore can facilitate the transmission of disease [19,20]. The influx of susceptible individuals through birth pulses or congregations of hosts can increase transmission by increasing susceptible host density [21]. These influxes are often tied to seasonal environmental changes that serve as cues for migration or breeding [22].

Host immunity and recovery often exhibit annual or seasonal fluctuations as well. In marine systems, temperature can strongly influence host immunity, with an often narrow range of temperatures inducing an optimal immune response [23]. This is particularly important in crustaceans, whose innate immune system is sensitive to environmental conditions [24]. Increasing temperature can promote host recovery by accelerating the life cycle of a parasite and transitioning the parasite into its external, reproductive stages [25,26]. The thermal tolerance of external parasite stages also influences host recovery, since thermal refugia can offer hosts an opportunity to reduce parasite prevalence [27–29]. In crustaceans, thermal conditions can induce change in moulting rate that then alters the recovery rate from external and gill parasites or pathogens [30].

Infected host mortality is also influenced by seasonal changes in the environment [31]. Higher temperature increases parasite replication in many systems, which can overwhelm host defenses and increase the risk of host death [32–34]. Temperature can also modulate parasite genes that influence virulence and thus induce a greater rate of parasite-induced mortality [17,35–38]. Sub-lethal impacts on host physiology can increase infected host mortality by reducing the ability of hosts to evade predation or to compensate for environmental stressors—for example, low dissolved oxygen or temperatures outside of the host's thermal optimum [32,39,40].

Quantifying infection transmission, mortality and recovery rates and their integrated response to environmental conditions is crucial for developing predictive models that forecast disease impacts. However, in wild marine systems, these parameters can be difficult to estimate [6,26,41–43]. Furthermore, the extent to which changes in prevalence reflect environmental drivers (such as temperature-dependent host or parasite physiology) or seasonal changes in host density can be challenging to distinguish [5]. In this study, we used shrimp Black Gill (sBG) disease, caused by a ciliate parasite in an economically and ecologically important crustacean host, to disentangle the contributors to seasonal disease prevalence through empirical and mechanistic modelling approaches. We first determined whether sBG transmission varies over the course of a year by using a seasonal field deployment study (§2c, *Seasonal sentinel experiments*). We then manipulated infection status and temperature in a laboratory experiment to determine whether disease-induced mortality and/or host recovery varies according to seasonal changes in temperature (§2d, *Mortality and recovery experiment*). Finally, we utilized this information to build a mechanistic model to determine how seasonal variation in transmission, host mortality and recovery ultimately influences sBG prevalence and explored whether changes in prevalence are owing to host density-dependent or -independent mechanisms (§2e, *Mechanistic models*). Owing to this multifaceted approach, we provide an overview of our research in the main text and invite readers to view the online electronic supplementary material for detailed methods and results.

2. Methods

(a) Biology of the study system

Shrimp Black Gill disease in the South Atlantic Bight and the Gulf of Mexico is typically caused by an infectious apostome ciliate, *Hyalophysa lynni* [44], and is thought to affect many species of penaeid shrimp in the Southeastern United States. The most economically and ecologically significant host species are white (*Litopenaeus setiferus)* and brown (*Farfantepenaeus aztecus*) shrimp, and the ciliate appears to influence the physiology of both species similarly [45]. The prevalence of this disease is highly seasonal, with upwards of 80% prevalence in late summer and early autumn. The disease almost disappears during the cooler water months until prevalence rises again in the early summer [46–49]. The period when sBG prevalence is highest corresponds to both peak water temperatures and increases in local densities of white and brown shrimp [50]. Although

the association between sBG and the performance of the fishery is not clear, shrimp harvesters report sudden drops in the abundance of shrimp coincident with the seasonal emergence of sBG (personal communication with Georgia shrimp harvesters, 2013). Longitudinal analyses suggest that increases in sBG are correlated with reductions in harvest and that climate indices are associated with mean disease prevalence between years [46,51]. Similar to other apostome ciliates, *H. lynni* must transition from an initial colonization stage (phoront) to a feeding stage (trophont) and finally to a free-swimming, transmissible stage (swimming tomite) in order to reproduce [44]. Pathogenic sBG disease manifests when shrimp gill lamellae melanize as an immune response to *H. lynni* trophonts invading host tissue [44]. Host pathology results from the melanized nodules that impair respiratory function and haemolymph ion regulation [52,53]. Consequently, severely infected shrimp have reduced endurance and escape responses, and they are more heavily predated than their asymptomatic counterpart [40,47,54]. Infected shrimp can recover from the infection, but they gain no lasting immunity and shrimp are readily reinfected with the ciliate [55]. White and brown shrimp support the state of Georgia's largest commercial fishery and provide an important food source for numerous game fish species in the South Atlantic Bight [56]. Therefore, investigation into the potential threat of sBG disease is critically important for the economic and ecological health of the area.

(b) Diagnostics

For our empirical efforts, we determined sBG prevalence through a polymerase chain reaction (PCR) assay [46] and sBG severity by counting the number of trophonts per mm^2 of gill tissue microscopically (electronic supplementary material, appendix 1). The PCR assay is significantly more sensitive than the microscopic assay and detects every stage of the ciliate life cycle. However, the microscopic procedure quantifies the number of parasites inhabiting the gill tissue, which is an important metric of disease severity in macroparasites and provides information regarding host pathology that is not available from the PCR diagnostic alone [46,57]. Therefore, coupling these diagnostic metrics provides a more complete understanding of host–parasite interactions.

(c) Seasonal sentinel experiments

We determined the relative contribution of *H. lynni* transmission to seasonal disease dynamics through a manipulative field experiment. Seasonally (twice in summer and once in each other season), we suspended shrimp that had been treated to reduce *H. lynni* infections (following [26]) in the Wassaw estuary for 10 days and recorded the resulting prevalence and mean severity of *H. lynni* infections (electronic supplementary material, appendix 2). For every trial, approximately 200 shrimp were purchased from a bait shop that collected shrimp from the Vernon River in the Ogeechee Estuary (approx. 31.9° N, −81.1° W). These shrimp were collected daily and held in continuously flowing seawater for no more than 24 h prior to our purchase. Shrimp were treated with the antiparasitic medications as previously described [26]. Briefly, shrimp were treated with Metroplex™ (metronidazole 1.575 mg L^{-1}) and Cupramine™ (Cu + 0.25 mg L^{-1}) every other day for 14 days. The species, average length and sex ratio varied depending on the season, but all shrimp used in each experiment trial were of a single species (either *L. setiferus* or *F. aztecus*) and no greater than 12.41 mm standard deviation in length within each season (electronic supplementary material, table S1). After the antiparasitic medication protocol, shrimp were placed in experimental conditions. Between 13 and 26 shrimp were caged individually and in each experimental trial they were deployed for 10 days into the Wassaw Estuary at the Skidaway River (figure 1a). Concurrently, in the lab, between 11 and 16 shrimp were caged and held in plastic bins in artificial seawater as controls. These controls allowed us to quantify the level of uncured or latent infections that became apparent due to increased holding time. We surrounded the control bins in flowing estuary water to maintain the control shrimp at approximately equal temperatures (±1°C) as the shrimp deployed in the estuary. At the end of the experimental trial, all shrimp were euthanized and processed for sBG prevalence and severity (electronic supplementary material, appendix 1). We also compared the prevalence of *H. lynni* in wild shrimp collected from the Wassaw Sound at the time of each trial to the resulting *H. lynni* prevalence in the transmission experiments. Wild shrimp were collected through 15 min boat tows with either a 7.6 or 12.2 m flat shrimp net with 4.8 cm stretch mesh webbing. Severity data were not collected on wild shrimp due to logistical reasons. We evaluated if there were seasonal differences in infection presence and severity between our deployed, control and wild shrimp using logistic regression and the Kruskal–Wallis rank sum test, respectively (electronic supplementary material, appendix 2). These tests were chosen based on the binary nature of prevalence data and the non-normal distribution of our severity data. All analyses were performed in R using v. 4.3.1.

(d) Mortality and recovery experiment

We compared the survival and recovery of unmedicated and medicated shrimp over two ecologically relevant temperatures to determine the importance of recovery and host mortality to seasonal variation in sBG prevalence (electronic supplementary material, appendix 3). Approximately 400 white shrimp were collected from the Wassaw Sound Estuary in October. Half of these shrimp were subjected to the medication procedure, while the other half were maintained in flow-through tanks as an unmedicated treatment. Initial samples of both treatments were taken within 2 days of arrival (just prior to the medication procedure) and provided baseline estimates of infection prevalence and severity. These samples are referred to as the *pre-application medicated* and *pre-application unmedicated* samples (12 shrimp per sample).

After the medication procedure, 20 shrimp from each medication application group were sampled for *H. lynni* presence and severity, referred to as the *post-application medicated* and *post-application unmedicated* samples. Nineteen *previously medicated*

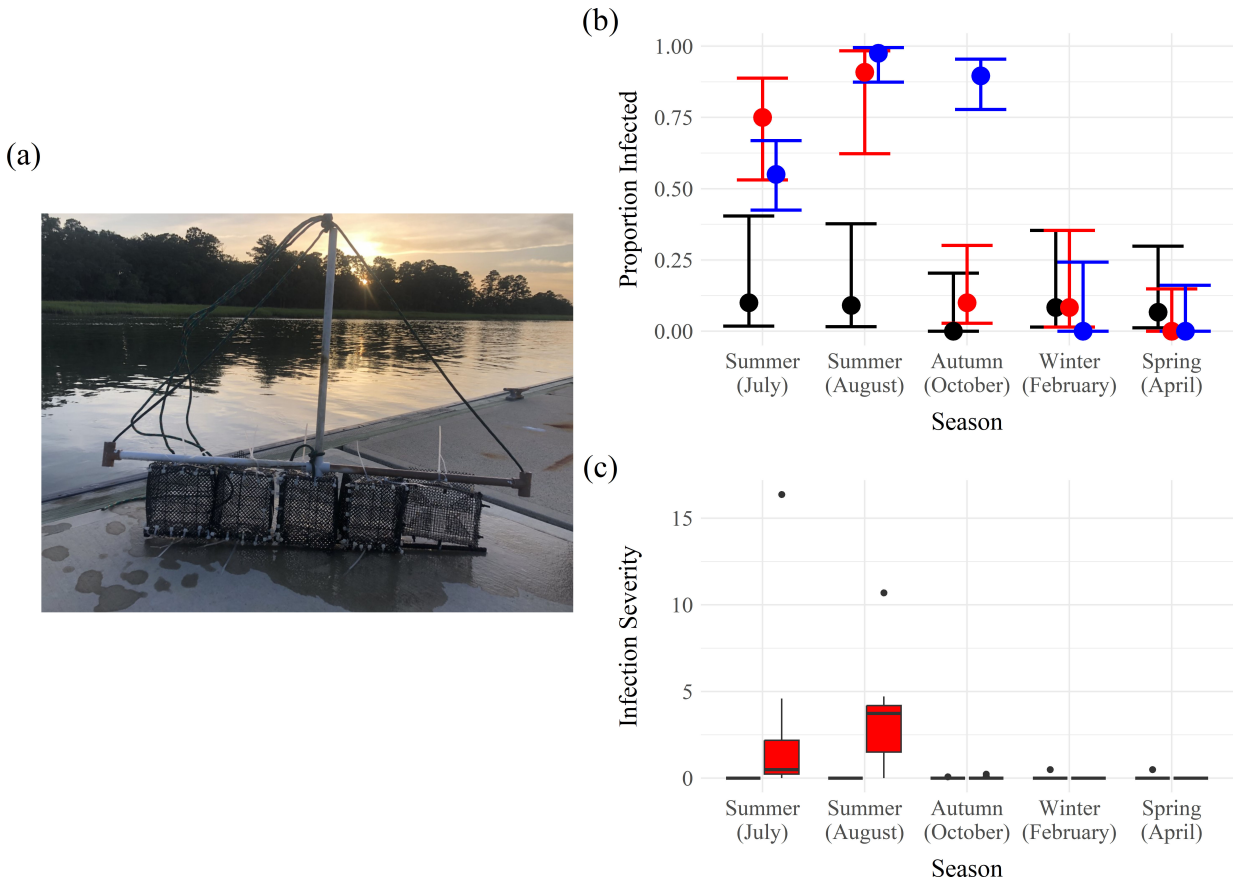

Figure 1. Seasonal Sentinel Experiments. (a) Field cage deployment. Medicated shrimp were held in individual mesh cages deployed in the Wassaw Estuary at the Skidaway River, which kept shrimp protected from predators while allowing shrimp full exposure to estuary water. (b) *H. lynni* infection prevalence (dots) and 95% confidence intervals (bars) in wild (blue), deployed estuary (red) and control (black) shrimp per experimental trial according to the month when it was conducted. (c) Final infection severity (trophonts mm^{-2} of gill tissue), inclusive of zeroes, in deployed estuary (red) and control (black) shrimp, where the solid horizontal line is the median infection intensity per experimental month, and the lower and upper hinges depict the first and third quantiles. The whiskers mark data within 1.5 times the interquartile range, and solid points display outliers. With the exception of July and August, all severity metrics were at or near zero.

shrimp and 20 *unmedicated* shrimp were acclimated to the *ambient temperature treatment* (23.5 ± 0.6°C), and 29 *previously medicated shrimp* and 30 *unmedicated shrimp* were acclimated to the *heated temperature treatment* (30.2 ± 0.7°C). The number of shrimp in the heated temperature treatment was higher to compensate for additional mortality caused by the high temperature. The ambient temperature simulated estuary water temperatures in the early spring and late autumn (when sBG prevalence is low), and the heated temperature treatment simulated late-summer estuary temperatures (when sBG prevalence is high [47,58]). The shrimp were then transferred into individual plastic aquaria filled with aerated artificial seawater (Instant Ocean) at the designated temperature treatment. We monitored shrimp daily for survival and moulting, because it is hypothesized that *H. lynni* infection may cause shrimp to moult more frequently, causing stress to shrimp hosts and inducing mortality [45,48]. Dead shrimp were collected daily and processed immediately for *H. lynni* presence and severity. After 8 days, 78% of the shrimp in the heated treatment had died; therefore, the experiment was terminated and all shrimp were processed for *H. lynni* infection metrics.

We used a series of logistic regression models and Kruskal–Wallis tests to determine whether recovery rates differed between our temperature and medication treatments, and a series of Cox-proportional hazard models to compare mortality risk across treatments (electronic supplementary material, appendix 3). The Cox proportional hazards model was chosen to analyse the time-to-event data collected during our survival experiment and the multiple factors contributing to differences in mortality [59].

(e) Mechanistic models

We developed a suite of mathematical models of host–pathogen dynamics, and their structure was informed by the results of our empirical research (see §3a,b). Our models explored whether the observed changes in transmission could be owing to fluctuations in local shrimp densities as part of white and brown shrimp annual migration cycles [50] or fishing depletion [60], and thus independent from physiological changes to hosts and/or parasites caused by abiotic factors [61]. We developed three classes of models to determine whether host density-dependent (*density-dependent transmission models*), host frequency-dependent (*frequency-dependent transmission models*) or density-independent (*reservoir transmission models*) transmission best explains the seasonal change in sBG prevalence on the Georgia coast. We crossed these model classes with a transmission function that either varied the transmission parameter value (β_D, β_F, β_R, §2f) over the course of the season or held it static. The use of static transmission parameter values reflects seasonality in transmission that is driven only by changes in host density, while variable transmission values allowed for seasonal variation in transmission to be independent of host density (e.g. temperature effects on

parasite infectivity). Model parameters (table 1) were estimated from a combination of our laboratory and field experiments, the published literature [62] and data on the abundance of wild shrimp from monthly trawls along the Georgia coast conducted by the Georgia Department of Natural Resources (electronic supplementary material, appendix 4).

(f) Transmission models

The differential equations below track the number of susceptible (S) and infected (I) shrimp through time, assuming a source of infectious *H. lynni* particles originating from the local penaeid shrimp community or an environmental reservoir. All rates are calculated at a daily time step (t) and the value of that rate is changed every month (m) when applicable (electronic supplementary material, appendices 4 and 5, and electronic supplementary material, figure S1). Owing to the seasonal migration of shrimp, different sets of equations were developed for positive and negative migration rates.

Positive migration rate:

$$\frac{dS}{dt} = \lambda_m - FS - \mu_m S + \gamma_m I \tag{2.1a}$$

$$\frac{dI}{dt} = FS - I(\mu_m + \alpha + \gamma_m) \tag{2.2a}$$

Negative migration rate:

$$\frac{dS}{dt} = \lambda_m \frac{S}{N} - FS - \mu_m S + \gamma_m I \tag{2.1b}$$

$$\frac{dI}{dt} = \lambda_m \frac{I}{N} + FS - I(\mu_m + \alpha + \gamma_m) \tag{2.2b}$$

The *Positive migration rate* equations (equations (2.1a) and (2.2a)) are used when the migration rate of shrimp into the estuary (λ_m) is positive (January–March, May and June), and we assume that all incoming shrimp enter the susceptible class. This rate is updated monthly and is estimated from fisheries-independent catch data. The *Negative migration rate* equations (equations (2.1b) and (2.2b)) are used when shrimp are migrating out of the estuary (i.e. λ_m is negative, April, July–December) and shrimp from both S and I classes are removed from the local population ($n = S + I$) in proportion to their relative abundances. The transmission function (F) is used to evaluate hypotheses regarding the nature of sBG transmission and is described in further detail in §2g. Shrimp hosts experience two sources of mortality: both a natural mortality rate that is updated monthly (μ_m) and a static disease-induced mortality (α). A static disease mortality rate was chosen based on the results from the recovery and mortality experiment, where disease-induced mortality was found not to vary with temperature. Infected shrimp can recover from sBG infection at a recovery rate of γ_m, then immediately enter the S class without any immune protection. The results of the recovery and mortality experiment contrasted with earlier work that indicated a difference in recovery with temperature [26]. Therefore, we added additional model runs that included variable recovery with season (electronic supplementary material, appendix 4 and figure S1) to evaluate the possible significance of variable recovery for sBG prevalence.

(g) Transmission functions

The transmission function represents our three potential mechanisms for the influence of host density:

$$\text{Reservoir:} \qquad F = \beta_R \tag{2.3a}$$

$$\text{Density-Dependent:} \qquad F = \beta_D I \tag{2.3b}$$

$$\text{Frequency-Dependent:} \qquad F = \beta_F \frac{I}{N} \tag{2.3c}$$

Each of the transmission parameters β_i ($i = R,D,F$) in equations (2.3a)–(2.3c) is either held constant for model simulations where host density is the only component of transmission that varies across the year, or is modified to update monthly (β_{im}) in simulations where transmission varies seasonally, independent of host density (see *Model execution and sensitivity analysis;* §2h).

The exact value and rate of change in the β_{im} parameter over the course of the year are unknown. Therefore, we generated candidate values using a Normal, Brière and Modified-Brière function [63] (referred to as β_{im} generating functions; electronic supplementary material, appendix 5). The parameters for the normal distribution are a constant controlling the curve amplitude, curve width (i.e. standard deviation) and date of peak transmission. For the Brière and modified-Brière functions, the parameters are a constant controlling the curve amplitude, the earliest month of non-zero transmission, the latest month of non-zero transmission and skew. The Brière function generates a left-skewed distribution, while the modified-Brière function generates a right-skewed distribution. We crossed each of these β_{im} generating functions with the aforementioned transmission functions. Static transmission model runs used the average value produced by the β_{im} generating functions as β_i. This process resulted in 18 different model formulations: 3 transmission functions × 3β_{im} generating functions × 2 transmission updating frequencies (β_i and β_{im}). We additionally crossed variable and static recovery with each of the above model formulations, totaling 32 models.

Table 1. Parameter symbols, definitions and sources for sBG mechanistic models. Fitted values refer to estimates derived by our model fitting procedures. The migration rate was estimated from data collected by the Georgia Department of Natural Resources Coastal Resources Division Ecological Monitoring Trawl Survey (GA DNR CRD EMTS). We used estimates of wild shrimp mortality rate to scale our lab-based estimates to that of local shrimp populations [62].

parameter	symbol	value or range	source
migration rate	λm	−3.618–5.958	GA DNR CRD EMTS
transmission rate	$\beta_F, \beta_D, \beta_R$	variable	fitted value
recovery rate	γm	0–0.681	*Mortality and recovery experiment*
mortality rate	μm	0–0.013	*Mortality and recovery experiment*; [62]
disease-induced mortality rate	α	0.008	*Mortality and recovery experiment*; [62]

(h) Model execution and sensitivity analysis

We evaluated whether density-independent or -dependent transmission mechanisms recreated seasonal sBG prevalence dynamics by simulating annual disease dynamics (*dede* function, *deSolve* package; [64]) for each of the three above-mentioned transmission functions (equation 2.3a–c). We first generated 10 000 parameter sets for the $\beta_{\mathfrak{F}}$ generating functions over a permissive range of values (electronic supplementary material, table S2) using latin hypercube sampling [65,66]. We then used our estimated values of λ_m, μ_m, α and either γ_m or γ and each β_{im} parameter set to simulate sBG dynamics during portions of the year when sBG is typically present (June–December). To evaluate model fit, we calculated the sum of squared errors (SSE) between the simulated sBG prevalence and the monthly mean and standard error of the wild sBG prevalence over the entire Georgia coast from the past 5 years (electronic supplementary material, appendix 5). The SSE was chosen as our fit metric because of its simplicity and ease of interpretation. We repeated this process with all 32 model formulations. The model formulation and parameter set with the lowest SSE was determined to be the best fit.

We conducted a sensitivity analysis on the best-fit model formulation (i.e. static or variable transmission generated by the Normal, Brière or Modified-Brière function and static or variable recovery) for the density-dependent, frequency-dependent and reservoir models to evaluate the influence of our parameter estimations on our choice of the best-fit model and our conclusions regarding the nature of sBG transmission [67]. We varied host mortality (both natural and disease-induced), migration, recovery and shrimp abundance independently by ±10% and ±20% and then ran the simulation over the 10 000 parameter values generated by the latin hypercube appropriate for that model formulation. We then compared models with real data using SSE as described above.

3. Results

(a) Seasonal sentinel experiments

We found substantial variation in *H. lynni* transmission across seasons (figure 1b,c). Between June 2020 and May 2021, we deployed a total of 97 shrimp and were able to recover and determine *H. lynni* infection presence after 10 days of exposure in 88% of these animals. Prevalence and mean severity peaked in July (prevalence: 75%, mean severity: 1.88 ± 0.82 (s.e.) ciliates per mm^2) and August (prevalence: 91%, mean severity: 3.38 ± 0.9 s.e.) when water temperatures were maximal (electronic supplementary material, table S1 and figure 1b,c). Concurrently, the control group had little to no infection (0–10% prevalence) over the course of their trials. In the summer, estuary-deployed shrimp experienced a significantly higher probability of infection presence than shrimp in the control treatment (estimate = 3.91, $z = 3.00$, $p = 0.003$; electronic supplementary material, appendix 6, figure 1b). Similarly, *H. lynni* severity was significantly higher in estuary-deployed shrimp than control shrimp in July ($H = 15.216$, d.f. = 1, $p < 0.001$) and August ($H = 15.556$, d.f. = 1, $p < 0.001$; figure 1c). No other seasonal deployment trials recorded a significant difference between the estuary-deployed and control treatments. In October, wild shrimp had a significantly higher probability of infection presence than estuary-deployed experimental shrimp (estimate = 4.35, $z = 4.93$, $p < 0.001$), where 90% of the wild shrimp sampled were infected with *H. lynni* compared with only 10% prevalence in the estuary-deployed shrimp during that season. There were no other statistically significant differences between wild and deployed shrimp (figure 1b; electronic supplementary material, appendix 6).

(b) Mortality and recovery experiment

We found that shrimp gradually recovered from *H. lynni* infection over the course of our laboratory experiment and that temperature did not influence recovery (table 2). Between our unmedicated pre-application and post-application samples 14 days later, there was a significant reduction in average *H. lynni* severity ($H = 10.07$, d.f. = 1, $p = 0.002$), but not in the probability of infection presence (estimate = 1.39, $z = 1.19$, $p = 0.23$). At the end of the experiment, no shrimp from any of the temperature or medication application treatment groups tested positive with PCR, and only eight shrimp (two in the unmedicated ambient, three in the unmedicated heated and three in the previously medicated heated treatments) were found to harbour ciliates

Table 2. Infection metrics for the *recovery and mortality* experiment. Each row presents the number of shrimp sampled, the proportion infected from each sample and the *H. lynni* infection severity of each sample taken during the medication procedure and temperature trial. 'Pre-application' refers to subsamples that were taken to measure initial infection levels. To verify how well drug exposure worked to cure infection, post-application subsamples were taken from both medication groups after 14 days in the lab, immediately prior to shrimp beginning the temperature treatments. 'Ambient' and 'heated' refer to the temperatures at which shrimp were held during the temperature trial. 'Severity' is the mean and s.e. of density of ciliate trophonts in the gills, inclusive of shrimp without infection. Cases where there is a non-zero severity metric but zero infected shrimp are owing either to ciliate misidentification microscopically or to already-degraded ciliate DNA that was impossible to detect through PCR.

treatment	medication	number sampled (*n*)	proportion infected	severity (trophonts mm^{-2}) mean ± s.e.
pre-application	medicated	12	1	4.46 ± 1.29
	unmedicated	12	1	1.83 ± 0.52
post-application	medicated	20	0	0.07 ± 0.05
	unmedicated	20	0.75	0.38 ± 0.21
ambient (23.5 ± 0.6°C)	previously medicated	19	0	0
	unmedicated	20	0	0.06 ± 0.05
heated (30.2 ± 0.7°C)	previously medicated	29	0	0.06 ± 0.04
	unmedicated	30	0	0.15 ± 0.11

microscopically. There were no statistically significant differences in *H. lynni* infection prevalence or severity between our medication or temperature treatments at this point (electronic supplementary material, appendix 7).

We found that the temperature and medication treatments significantly affected shrimp mortality, but there was no significant interaction between these two variables. Only 13% of shrimp died in our ambient temperature treatment, while 78% of shrimp in the heated treatment died (figure 2). Three Cox proportional hazard models that examined the variables responsible for mortality were found to be equivalent through Akaike information criteria corrected for small sample size (AICc) [68] and all three models found a significantly higher risk of mortality in the heated treatment compared with the ambient (table 3). All three models also identified the temperature treatment as the most important contributor to mortality hazard, as indicated by the large coefficient estimates in table 3. The interaction between temperature and medication treatment was only selected in one of our best-fit models, but even there, it was not significant. Similarly, moulting frequency was not significantly related to shrimp mortality. Thus, while shrimp in our unmedicated treatments did experience greater 'natural' mortality than shrimp in our medicated treatments, disease-induced mortality was not altered by temperature.

(c) Mechanistic models

We found that host density-independent models that included variable transmission rates most accurately recreated sBG prevalence dynamics (figure 3). All models required variable transmission rates to capture the seasonal rise and fall of sBG dynamics (figure 3). Both the frequency-dependent and reservoir models with variable transmission fit the data well (SSE = 0.192 and 0.178, respectively), while the best-fit density-dependent model with variable transmission resulted in higher values of prevalence than were observed (SSE = 0.964). Furthermore, the parameter estimates for the frequency-dependent and reservoir models matched the observed dynamics in the Seasonal sentinel experiments, with peak transmission values occurring during mid-August and the transmission season ending in October (electronic supplementary material, table S3). We found that the normal distribution was the best fit β_{im} generating function for the frequency- and density-dependent models, while the Brière β_{im} generating function was the best fit for the reservoir model. Monthly variation in recovery rate did not greatly alter model fit (electronic supplementary material, table S4), and static recovery rate fit the data better in the reservoir and frequency-dependent models, while variable recovery rate fit the data better in the density-dependent model. Our choice of best-fit model was not sensitive to our parameter estimations (electronic supplementary material, tables S5–S7). Regardless of the sensitivity analysis scenario, the density-dependent models fit relatively poorly, producing SSEs at least 3.75 times greater than the reservoir or frequency-dependent models. Our model fit was most sensitive to changes in recovery rate (γ), where reducing the static recovery rate by 10% improved model fit by 6.5% in the reservoir model, while increasing the recovery rate by 20% improved model fit by 23% in the frequency-dependent model.

4. Discussion

Evaluating the roles of transmission, host recovery and mortality with respect to seasonal changes in disease prevalence is critical to understanding and managing infectious disease in wild systems. Here, we demonstrated that a steep increase in transmission probably drives the sharp rise in sBG prevalence seen in the South Atlantic Bight in the late-summer months. As the water cools in the autumn, sBG transmission ceases and host shrimp gradually recover from infection. The force of infection appears low over the subsequent autumn, winter and spring. We found a high prevalence of infected wild shrimp in the autumn (October) despite low levels of transmission during that season. This is probably the result of wild shrimp

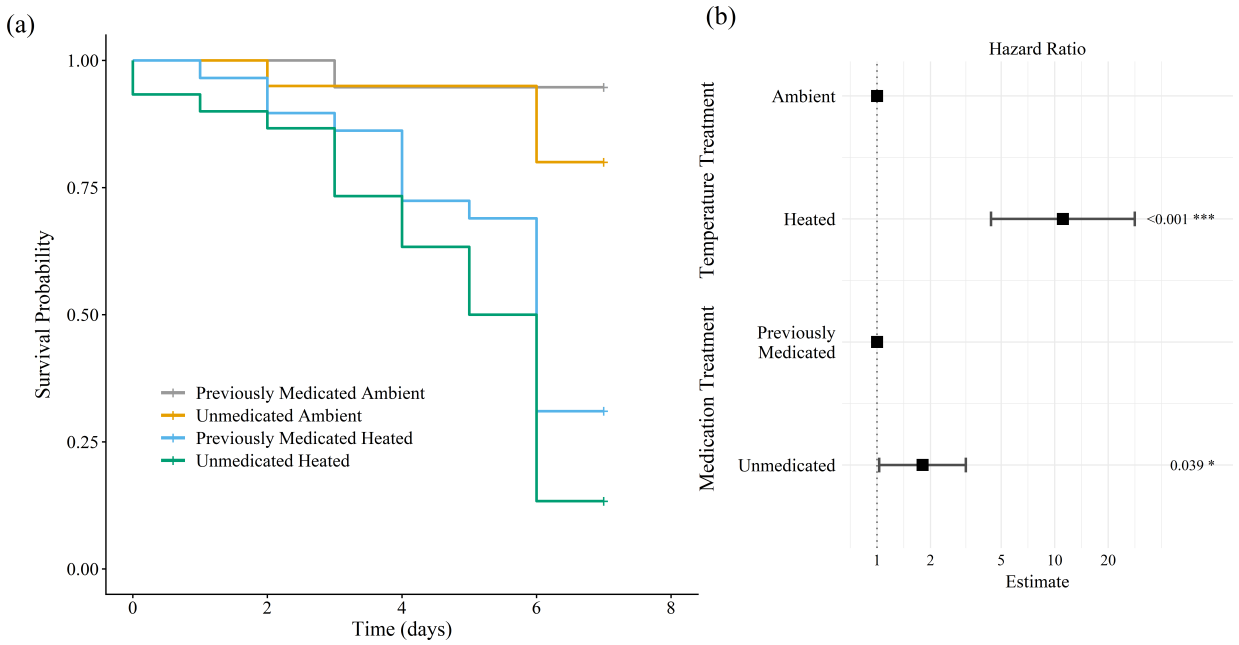

Figure 2. Survival probability and relative hazard estimates from the recovery and mortality experiment. (a) The Kaplan–Meier survival curve depicting the estimated mean survival probability in shrimp per each medication and temperature treatment combination. (b) Log of the relative hazard estimates of mortality from the most parsimonious Cox proportional hazard model (Model 1) with 95% confidence intervals. Estimates to the right of the dotted line indicate treatments with an increased risk of shrimp mortality compared with the treatments along the dotted line. The *p*-values of these estimates are also displayed (*$p \leq 0.05$, ***$p \leq 0.001$).

acquiring the infection earlier in the year and then requiring several weeks to recover. We found little evidence that sBG causes substantial host mortality in the laboratory or that this disease-induced mortality varied with temperature, suggesting that the decline in seasonal prevalence in the field is most likely owing to host recovery once high transmission rates abate. Finally, we demonstrated that seasonal variation in sBG transmission and prevalence is not owing to increases in local shrimp densities, but rather an environmentally driven variable transmission rate. Together, these results motivate additional research into the drivers of high summer sBG density-independent transmission.

Models with static transmission rates failed to reproduce field prevalence patterns, while models incorporating variable transmission drastically improved fit. Moreover, seasonally varying reservoir and frequency-dependent models fit the field-collected data much better than density-dependent models. These models additionally better captured the early-season dynamics and late-summer peak in transmission observed in the field (figure 3, electronic supplementary material, table S3). We were unable to discriminate whether the lack of density dependence indicated that transmission was driven by reservoir hosts, immigration of infectious particles from non-local shrimp or whether local density dependence was masked by high seasonal parasite production. However, our model does indicate that seasonal changes in environmental conditions that support parasite transmission are much more important than seasonal changes in host density for explaining sBG dynamics. This finding adds to the growing body of work suggesting that environmental effects, rather than local host density, may drive transmission in well-mixed marine systems [41,69–71].

The rise in transmission rate during the summer months could be owing to a temperature-associated acceleration of the *H. lynni* life cycle. The rate at which many parasites can complete their life cycle is strongly influenced by temperature [3]. Warm temperatures may also facilitate the survival of transmissible stages of the parasite, allowing for infectious propagules to reach more hosts and at a higher propagule pressure [25,70]. As temperatures continue to warm, there may be an increase in the duration of the sBG transmission season, allowing for periods of high prevalence to extend earlier into the spring and later into the autumn. However, extremely warm temperatures may reduce prevalence by decreasing host and parasite survival [12,63,72,73]. Therefore, sBG dynamics are likely to vary nonlinearly with temperature, and the fate of the system will depend on the degree of warming in the area.

Shrimp in our laboratory experiments may have begun recovering from infection upon their arrival. Although PCR prevalence remained high in the post-application unmedicated shrimp sample, there was a significant decline in sBG severity of infection between our pre- and post-application samples, indicating a reduction in ciliates over time. This result could have been caused by increased mortality in heavily infected shrimp, gradual recovery of infected shrimp, or both. Furthermore, every shrimp that entered the temperature treatments, regardless of whether they died during the experiment or were euthanized at the end, tested negative for *H. lynni* via PCR. We therefore did not find a significant difference in recovery between our temperature treatments, which indicates that shrimp are able to recover from infection in temperatures at or above our ambient temperature treatment of 23.5°C.

Unmedicated shrimp were significantly more likely to die in our laboratory experiment compared with previously medicated shrimp. This finding may indicate that sBG infection does increase shrimp mortality rate. However, it is difficult to attribute disease-induced mortality as an important factor contributing to seasonal changes in sBG prevalence because of the small effect size and the low severity of infection in our experiment. Furthermore, the interaction between temperature and mortality was not significant; thus, there was no evidence that disease-induced mortality varied with temperature. However, our laboratory

Table 3. Cox proportional hazard model summaries for recovery and mortality experiment. Three models were determined to be equivalent from our model selection procedure (within 2 ΔAICc points). Model predictor variables are listed (parameter column), as well as the factor levels (when applicable). The corresponding mortality hazard estimate (coef) is displayed directly under the model number, along with the standard error (s.e.), *z* value and the exponentiated coefficient (exp(coef)). Positive values indicate that mortality risk increases with the parameter, while negative values indicate a decline in risk. Significance values are indicated by asterisks next to the coefficient numbers (***$p \leq 0.001$, **$p \leq 0.01$, *$p \leq 0.05$).

parameter	factor level	model 1				model 2				model 3			
		coef	s.e.	*z*	exp(coef)	coef	s.e.	*z*	exp(coef)	coef	s.e.	*z*	exp(coef)
temperature	heated	2.41***	0.48	5.07	11.13	2.42***	0.48	5.08	11.23	2.98**	1.03	2.9	19.65
medication	unmedicated	0.59*	0.29	2.06	1.8	0.68*	0.29	2.36	1.98	1.4	1.12	1.25	4.04
moulting frequency	N/A					−0.49	0.34	−1.45	0.61	−0.48	0.34	−1.41	0.62
temp * medication	heated * unmedicated									−0.78	1.16	−0.67	0.46
ΔAICc		0.25				0				1.85			

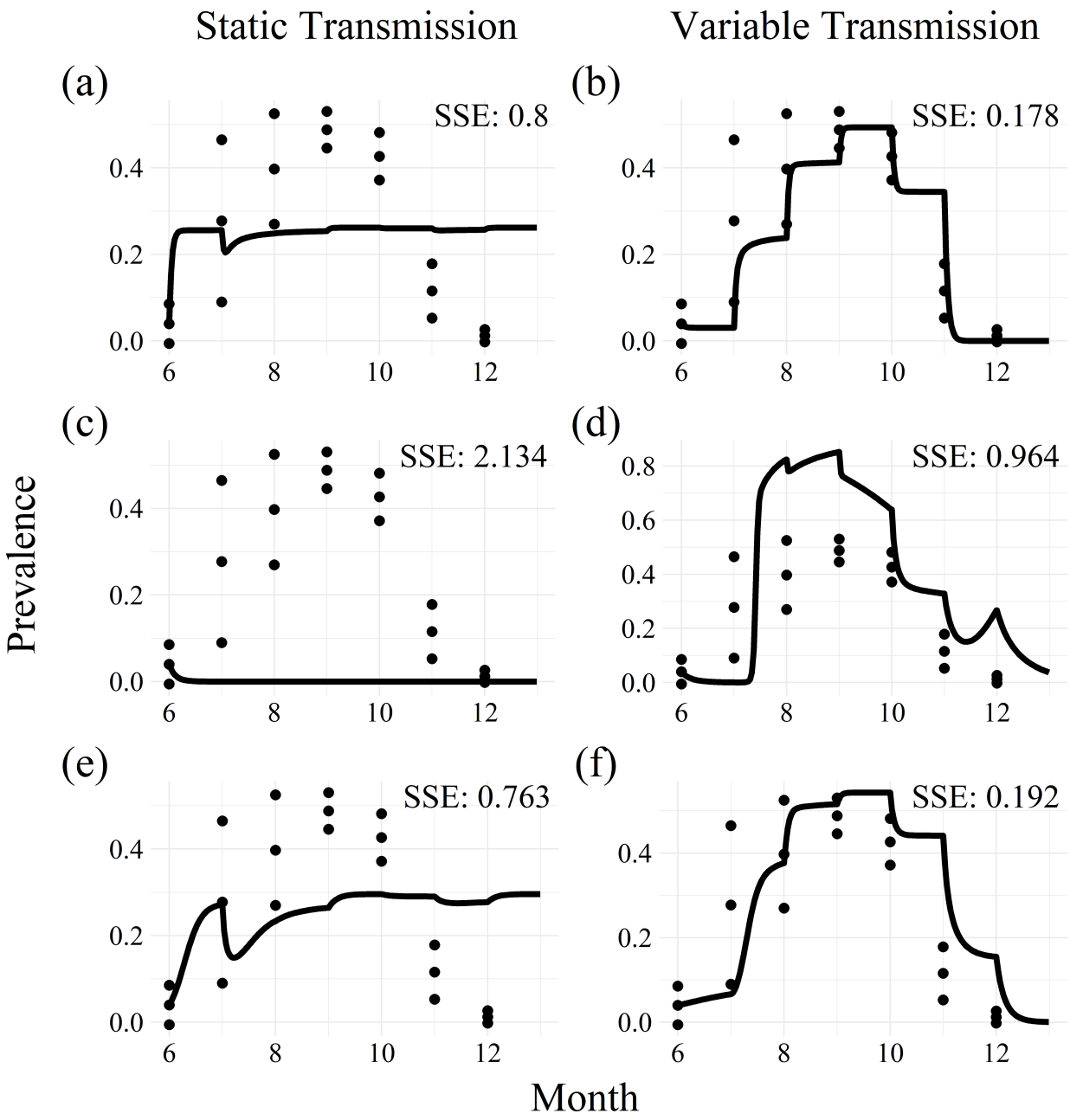

Figure 3. Simulated sBG dynamics from the three transmission functions with static and variable transmission rate**.** Solid lines show model output for infection prevalence through time for the best-fit models by transmission function (rows) and static versus variable transmission rates (columns). (a,b) Reservoir transmission; (c,d) density-dependent transmission; (e,f) frequency-dependent transmission. Models with static transmission rates (β_i, $i = R,D,F$) are along the first column, and models with variable transmission rates (β_{im}) are along the second column. The black dots depict the mean and mean ± s.e. of the last 5 years of sBG prevalence by month, which are used to evaluate model fit and calculate the sum of squares error (SSE). These values are weighted equally and treated as independent points for model fitting purposes (see electronic supplementary material, appendix 5 for more details).

conditions may have masked a more significant effect of disease-induced mortality. The survival of the previously medicated shrimp may have been altered by the medication application [26]. Cupramine™, according to its manufacturer, is known to cause additional mortality in crustaceans. Therefore, we may have inadvertently selected for the most resilient shrimp with the medication procedure, which could result in the previously medicated shrimp having higher survival. The heated temperature treatment, although ecologically relevant, is close to the thermal maximum of the shrimp (CT_{max} = 36–41.6°C [74]), resulting in thermal stress to these animals. This, coupled with the extended timeframewhich during these shrimp were held in captivity (22 days), could have resulted in an excessively high mortality rate in the heated temperature treatment that obscured additional mortality from sBG.

In some instances, shrimp tested negative for sBG through PCR, but sBG trophonts were detected microscopically, as well as the reverse, where *Hyalophysa* DNA was detected with PCR but no trophonts were found under the microscope (table 2). The former can be attributed either to misidentification under the microscope or to excessive degradation of *H. lynni* DNA making its amplification impossible. The shrimp immune response effectively destroys parasite DNA through the process of encapsulation and melanin production [55], which could maintain the shape of the ciliate but prevent the PCR primers from amplifying the ciliate DNA. The latter case is due to the ability of PCR to detect all life stages of the ciliate, while our microscopic procedure can detect only the trophont stage [46].

It is important to contextualize the results of our laboratory-estimated recovery and mortality rates when applying these findings to the natural system. The shrimp in the mortality and recovery experiment were held at low densities in either filtered or artificial seawater that was aerated vigorously. Therefore, infected shrimp were not exposed to secondary infections and did not struggle to sequester oxygen from a low dissolved oxygen environment [47]. Experimental shrimp were also protected from predators and fed daily. Thus, infected shrimp did not experience the increase in mortality that may result from sub-lethal changes in physiology induced by parasite infection [40]. Additionally, we focus on temperature as a primary driver in our system; however, other seasonally varying biotic and abiotic factors, such as predation, dissolved oxygen, salinity, etc., may also influence disease processes and affect parasite-induced mortality in the estuary.

Our findings are relevant to the many other aquatic disease systems where parasite transmission interacts with variable environmental conditions to yield changes in prevalence [12,24,75–78]. Diseases that are caused by parasites with extended environmental stages are likely to display these dynamics, particularly if the parasite exhibits 'degree-day'-like physiological responses to temperature [79]. Warm water temperatures compound degree-days rapidly and can accelerate the completion of parasite life cycles, thus increasing their prevalence [80–82]. Several notable aquatic pathogens experience degree-day responses to temperature, including sea lice (*Lepeophtheirus salmonis*) and *Ceratanova shasta*, a myxozoan parasite that causes severe disease in salmon and trout [83,84]. Opportunistic parasites and those that can maintain low levels of prevalence in latently infected hosts are also likely to exhibit a rapid rise in prevalence when the temperature reaches a specific critical threshold [8,31,85–89]. This relationship between increased temperature and parasite replication is especially relevant for parasites with single-host life cycles but may not apply to parasites with complex, multi-host life cycles when transmission chains are interrupted owing to reductions in the densities of host species [73]. Additionally, if temperatures are outside of the thermal limits of the parasite, a linear relationship between temperature and parasite prevalence will not manifest [63,90].

The finding that high transmission rates drive infection prevalence over a narrow time period has important implications for the management of infectious disease in marine systems. Disease has been a significant factor in the success of restoration efforts for coral [91–94], oysters [95–97] and eelgrass [98,99] and is likely to impact other conservation breeding efforts in aquatic species. Any restoration programmes that release individuals into the wild will need to ensure that release dates correspond to time periods of low transmission. Additionally, any management efforts attempting to 'fish out the parasite' by reducing host density to disrupt pathogen transmission should first determine whether the pathogen in fact relies on density-dependent transmission, and if so, decrease host density during, or just prior to, maximal transmission rates [7,60]. Our research further demonstrates the utility of using endemic 'sentinel' animals to deduce disease parameters in wild aquatic systems [43,100]. By deploying these susceptible animals in natural environments, we can move beyond sampling for disease prevalence and quantify transmission rates, which is critical for the development of process-based models that aid in conservation and management [6,101].

Infectious disease in aquatic systems is a result of highly connected interactions between host, parasite, environmental and anthropogenic processes. With the increasing influence of global temperature change, there is an urgent need to understand the temperature dependencies of these interactions. Here, we have demonstrated that changes in transmission are the critical factor driving seasonal patterns of prevalence in an aquatic pathogen of a host that is highly important to the economy, ecology and culture of the southeastern USA. We further provide a methodology to test specific hypotheses regarding transmission dynamics in marine systems. Additionally, our methodology allows for efficient and rapid development of mechanistic models that address information gaps in the system. The data required to accurately parameterize a model to capture disease dynamics precisely in marine systems are extensive and difficult to obtain [7]. However, mechanistic models that reproduce trends seen in empirical studies and address specific hypotheses about the underlying dynamics in the system can provide useful information both for the management and understanding of infectious disease in marine systems [102]. Thus, our work represents an important step to increasing our mechanistic understanding of disease dynamics and facilitating proactive management and conservation efforts [103].

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. The data and code used in this manuscript are publicly available on Dryad: [104].

Supplementary material is available online [105].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. M.T.: conceptualization, data curation, formal analysis, funding acquisition, methodology, validation, visualization, writing—original draft, writing—review and editing; T.W.: conceptualization, data curation, methodology, writing—review and editing; E.F.: data curation, writing—review and editing; A.R.: data curation, writing—review and editing; M.O'H: conceptualization, methodology, writing—review and editing; A.M.: data curation, writing—review and editing; M.B.: data curation, writing—review and editing; R.J.H.: conceptualization, methodology, writing—review and editing; M.F.: conceptualization, data curation, funding acquisition, methodology, writing—review and editing; J.B.: conceptualization, funding acquisition, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

Funding. This study was supported in part by an Institutional Grant to the Georgia Sea Grant College Program (no. NA180AR417008) from the National Sea Grant Office, National Oceanic and Atmospheric Administration and the University of Georgia Presidential Interdisciplinary Seed Grant program. Additional support was provided by an Institutional Grant no. NA16NOS4190165 to the Georgia Department of Natural Resources from the Office of Coastal Management, National Oceanic and Atmospheric Administration, U.S. Department of Commerce. M.T. was partially supported by the U.S. National Science Foundation Grant DGE-1545433 and by the University of Georgia Graduate School.

Acknowledgements. This experiment was performed on land originally inhabited by the Mvskoke (Muscogee), Yamasee and Guale indigenous peoples and later occupied by former enslaved people during the Reconstruction Era. We wish to thank the many students and volunteers who assisted with these studies, including Julie Blaze, Maxwell Kleinhans, Olivia Hair, Jordan McNally, Alannah Dodd, Hannah Hall, Roland Berg, Graham Hardy, Arlyn Valeria Santiago-Garcia, as well as three anonymous reviewers for reviewing early drafts of this manuscript. All views, opinions, findings, conclusions, and recommendations expressed in this material are those of the authors and do not necessarily reflect

the opinions of the Georgia Sea Grant College Program, the National Oceanic and Atmospheric Administration, The Georgia Department of Natural Resources, the University of Georgia, or the National Science Foundation.

References

1. Lafferty KD, Harvell CD, Conrad JM, Friedman CS, Kent ML, Kuris AM, Powell EN, Rondeau D, Saksida SM. 2015 Infectious diseases affect marine fisheries and aquaculture economics. *Annu. Rev. Mar. Sci.* **7**, 471–496. (doi:10.1146/annurev-marine-010814-015646)
2. Lafferty KD. 2017 Marine infectious disease ecology. *Annu. Rev. Ecol. Evol. Syst.* **48**, 473–496. (doi:10.1146/annurev-ecolsys-121415-032147)
3. Byers JE. 2020 Effects of climate change on parasites and disease in estuarine and nearshore environments. *PLoS Biol.* **18**, 1–12. (doi:10.1371/journal.pbio.3000743)
4. Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, Ostfeld RS, Samuel MD. 2002 Climate warming and disease risks for terrestrial and marine biota. *Science* **296**, 2158–2162. (doi:10.1126/science.1063699)
5. Altizer S, Dobson A, Hosseini P, Hudson P, Pascual M, Rohani P. 2006 Seasonality and the dynamics of infectious diseases. *Ecol. Lett.* **9**, 467–484. (doi:10.1111/j.1461-0248.2005.00879.x)
6. Irwin BJ, Tomamichel MM, Frischer ME, Hall RJ, Davis ADE, Bliss TH, Rohani P, Byers JE. 2023 Managing the threat of infectious disease in fisheries and aquaculture using structured decision making. *Front. Ecol. Environ.* **22**, e2695. (doi:10.1002/fee.2695)
7. Ben-Horin T, Lafferty KD, Bidegain G, Lenihan HS. 2016 Fishing diseased abalone to promote yield and conservation. *Phil. Trans. R. Soc. B* **371**, 20150211. (doi:10.1098/rstb.2015.0211)
8. Omori R, Adams B. 2011 Disrupting seasonality to control disease outbreaks: the case of koi herpes virus. *J. Theor. Biol.* **271**, 159–165. (doi:10.1016/j.jtbi.2010.12.004)
9. Shore A, Caldwell JM. 2019 Modes of coral disease transmission: how do diseases spread between individuals and among populations? *Mar. Biol.* **166**, 1–14. (doi:10.1007/s00227-019-3490-8)
10. Cantrell DL, Groner ML, Ben-Horin T, Grant J, Revie CW. 2020 Modeling pathogen dispersal in marine fish and shellfish. *Trends Parasitol.* **36**, 239–249. (doi:10.1016/j.pt.2019.12.013)
11. Amundrud TL, Murray AG. 2009 Modelling sea lice dispersion under varying environmental forcing in a Scottish sea loch. *J. Fish Dis.* **32**, 27–44. (doi:10.1111/j.1365-2761.2008.00980.x)
12. Byers JE. 2021 Marine parasites and disease in the era of global climate change. *Annu. Rev. Mar. Sci.* **13**, 397–420. (doi:10.1146/annurev-marine-031920-100429)
13. Coffey AH, Li C, Shields JD. 2012 The effect of salinity on experimental infections of a *Hematodinium* sp. in blue crabs, *Callinectes sapidus*. *J. Parasitol.* **98**, 536–542. (doi:10.1645/GE-2971.1)
14. Raffel TR, Romansic JM, Halstead NT, McMahon TA, Venesky MD, Rohr JR. 2013 Disease and thermal acclimation in a more variable and unpredictable climate. *Nat. Clim. Chang.* **3**, 146–151. (doi:10.1038/nclimate1659)
15. Burge CA *et al*. 2014 Climate change influences on marine infectious diseases: implications for management and society. *Annu. Rev. Mar. Sci.* **6**, 249–277. (doi:10.1146/annurev-marine-010213-135029)
16. Derome N, Gauthier J, Boutin S, Llewellyn M. Bacterial opportunistic pathogens of fish. In *The Rasputin effect: when commensals and symbionts become parasitic. advances in environmental microbiology* (ed. C Hurst), pp. 81–108. Cincinnati, OH: Springer. (doi:10.1007/978-3-319-28170-4_4)
17. Franke F, Armitage SAO, Kutzer MAM, Kurtz J, Scharsack JP. 2017 Environmental temperature variation influences fitness trade-offs and tolerance in a fish–tapeworm association. *Parasit. Vectors* **10**, 1–11. (doi:10.1186/s13071-017-2192-7)
18. Zeller M, Koella JC. 2017 The role of the environment in the evolution of tolerance and resistance to a pathogen. *Am. Nat.* **190**, 389–397. (doi:10.1086/692759)
19. Roy BA, Kirchner JW. 2000 Evolutionary dynamics of pathogen resistance and tolerance. *Evolution* **54**, 51–63. (doi:10.1111/j.0014-3820.2000.tb00007.x)
20. Horns F, Hood ME. 2012 The evolution of disease resistance and tolerance in spatially structured populations. *Ecol. Evol.* **2**, 1705–1711. (doi:10.1002/ece3.290)
21. Martinez-Bakker M, Helm B. 2015 The influence of biological rhythms on host–parasite interactions. *Trends Ecol. Evol.* **30**, 314–326. (doi:10.1016/j.tree.2015.03.012)
22. Staudinger MD *et al*. 2019 It's about time: a synthesis of changing phenology in the Gulf of Maine ecosystem. *Fish. Oceanogr.* **28**, 532–566. (doi:10.1111/fog.12429)
23. Burge CA, Hershberger PK. 2020 Climate change can drive marine diseases. In *Marine disease ecology* (eds DC Behringer, BR Silliman, KD Lafferty), pp. 83–94. New York, NY: Oxford University Press. (doi:10.1093/oso/9780198821632.003.0005)
24. Shields JD. Climate change enhances disease processes in crustaceans: case studies in lobsters, crabs, and shrimps. *J. Crust. Biol.* **39**, 673–683. (doi:10.1093/jcbiol/ruz072)
25. Barber I, Berkhout BW, Ismail Z. 2016 Thermal change and the dynamics of multi-host parasite life cycles in aquatic ecosystems. *Integr. Comp. Biol.* **56**, 561–572. (doi:10.1093/icb/icw025)
26. Tomamichel MM, Walters TL, Hall RJ, Byers JE, Frischer ME. A methodology to produce specific-pathogen-free penaeid shrimp for use in empirical investigations of parasite ecology. *J. Shellfish Res.* **42**, 133–142. (doi:10.2983/035.042.0114)
27. Blakeslee AMH *et al*. 2021 Invasion of the body snatchers: the role of parasite introduction in host distribution and response to salinity in invaded estuaries. *Proc. R. Soc. B* **288**, 20210703. (doi:10.1098/rspb.2021.0703)
28. Hofmann E *et al*. 2009 Understanding how disease and environment combine to structure resistance in estuarine bivalve populations. *Oceanography* **22**, 212–231. (doi:10.5670/oceanog.2009.110)
29. Tolley SG, Winstead JT, Haynes L, Volety AK. 2006 Influence of salinity on prevalence of the parasite *Loxothylacus panopaei* in the xanthid *Panopeus obesus* in SW Florida. *Dis. Aquat. Org.* **70**, 243–250. (doi:10.3354/dao070243)
30. Groner ML, Shields JD, Landers DF Jr, Swenarton J, Hoenig JM. 2018 Rising temperatures, molting phenology, and epizootic shell disease in the American lobster. *Am. Nat.* **192**, E163–E177. (doi:10.1086/699478)
31. Tomamichel MM, Lowe KI, Arnold KMH, Frischer MarcE, Irwin BJ, Osenberg CW, Hall RJ, Byers JE. 2025 Warmer is deadlier: a meta-analysis reveals increasing temperatures accentuate disease effects on fisheries hosts. *Ecol. Lett.* **28**, e70156. (doi:10.1111/ele.70156)
32. Rohr JR, Raffel TR, Blaustein AR, Johnson PTJ, Paull SH, Young S. 2013 Using physiology to understand climate-driven changes in disease and their implications for conservation. *Conserv. Physiol.* **1**, 1–15. (doi:10.1093/conphys/cot022)
33. Studer A, Thieltges D, Poulin R. 2010 Parasites and global warming: net effects of temperature on an intertidal host–parasite system. *Mar. Ecol. Prog. Ser.* **415**, 11–22. (doi:10.3354/meps08742)

34. Scharsack JP, Franke F, Erin NI, Kuske A, Büscher J, Stolz H, Samonte IE, Kurtz J, Kalbe M. 2016 Effects of environmental variation on host–parasite interaction in three-spined sticklebacks (*Gasterosteus aculeatus*). *Zoology* **119**, 375–383. (doi:10.1016/j.zool.2016.05.008)
35. Guijarro JA, Cascales D, García-Torrico AI, García-Domínguez M, Méndez J. 2015 Temperature-dependent expression of virulence genes in fish-pathogenic bacteria. *Front. Microbiol.* **6**, 1–11. (doi:10.3389/fmicb.2015.00700)
36. Pattanayak S, Priyadarsini S, Paul A, Kumar PR, Sahoo PK. 2020 Diversity of virulence-associated genes in pathogenic *Aeromonas hydrophila* isolates and their *in vivo* modulation at varied water temperatures. *Microb. Pathog.* **147**, 104424. (doi:10.1016/j.micpath.2020.104424)
37. Wolinska J, King KC. 2009 Environment can alter selection in host–parasite interactions. *Trends Parasitol.* **25**, 236–244. (doi:10.1016/j.pt.2009.02.004)
38. Thomas MB, Blanford S. 2003 Thermal biology in insect–parasite interactions. *Trends Ecol. Evol.* **18**, 344–350. (doi:10.1016/s0169-5347(03)00069-7)
39. Aalto EA *et al*. 2020 Models with environmental drivers offer a plausible mechanism for the rapid spread of infectious disease outbreaks in marine organisms. *Sci. Rep.* **10**, 1–10. (doi:10.1038/s41598-020-62118-4)
40. Gooding EL, Kendrick MR, Brunson JF, Kingsley-Smith PR, Fowler AE, Frischer ME, Byers JE. 2020 Black gill increases the susceptibility of white shrimp, *Penaeus setiferus* (Linnaeus, 1767), to common estuarine predators. *J. Exp. Mar. Biol. Ecol.* **524**, 151284. (doi:10.1016/j.jembe.2019.151284)
41. Harvell D *et al*. 2004 The rising tide of ocean diseases: unsolved problems and research priorities. *Front. Ecol. Environ.* **2**, 375–382. (doi:10.1890/1540-9295(2004)002[0375:TRTOOD]2.0.CO;2)
42. Glidden CK *et al*. 2022 Strategies for managing marine disease. *Ecol. Appl.* **32**, 1–28. (doi:10.1002/eap.2643)
43. Byers JE, Malek AJ, Quevillon LE, Altman I, Keogh CL. 2015 Opposing selective pressures decouple pattern and process of parasitic infection over small spatial scale. *Oikos* **124**, 1511–1519. (doi:10.1111/oik.02088)
44. Landers SC, Lee RF, Walters TL, Walker AN, Powell SA, Patel MK, Frischer ME. 2020 *Hyalophysa lynni* n. sp. (Ciliophora, Apostomatida), a new pathogenic ciliate and causative agent of shrimp black gill in penaeid shrimp. *Eur. J. Protistol.* **73**, 125673. (doi:10.1016/j.ejop.2020.125673)
45. Frischer ME, Landers SC, Walker AN, Powell SA, Lee RF. 2022 Black Gill in marine decapod crustaceans: a review. *Rev. Fish. Sci. Aquac.* **30**, 498–519. (doi:10.1080/23308249.2022.2047153)
46. Frischer ME *et al*. 2017 Causes, diagnostics, and distribution of an ongoing penaeid shrimp black gill epidemic in the U.S. South Atlantic Bight. *J. Shellfish Res.* **36**, 487–500. (doi:10.2983/035.036.0220)
47. Frischer ME, Fowler AE, Brunson JF, Walker AN, Powell SA, Price AR, Bulski K, Frede RL, Lee RF. 2018 Pathology, effects, and transmission of black gill in commercial penaeid shrimp from the south Atlantic bight. *J. Shellfish Res.* **37**, 149–158. (doi:10.2983/035.037.0113)
48. Gambill JM, Doyle AE, Lee RF, Geer PJ, Walker AN, Parker LG, Frischer ME. 2015 The mystery of black gill: shrimpers in the South Atlantic face off with a cryptic parasite. *Curr. J. Mar. Educ* **29**, 2–7. https://www.researchgate.net/profile/Maryellen-Timmons/publication/289378503_Current_The_Journal_of_Marine_Education/links/568c2bb908ae71d5cd04b589/Current-The-Journal-of-Marine-Education.pdf#page=4
49. Swinford J, Anderson J. 2023 Multi-year environmental trends of shrimp black gill (*Hyalophysa lynni*) prevalence in Texas gulf coast shrimp populations. *Gulf Caribb. Res.* **34**, 60–68. (doi:10.18785/gcr.3401.08)
50. Bailey-Brock JH, Moss SM. 1992 Penaeid taxonomy, biology and zoogeography. In *Developments in aquaculture and fisheries science, marine shrimp culture* (eds AW AFast, JL Lester), pp. 9–27. Amsterdam, The Netherlands: Elsevier Science. (doi:10.1016/B978-0-444-88606-4.50008-4)
51. Kendrick MR, Brunson JF, Frischer ME, Kingsley-Smith PR. 2021 Climate indices predict black gill prevalence in white shrimp *Penaeus setiferus* (Linnaeus, 1767) in South Carolina and Georgia, USA. *J. Shellfish Res.* **40** 145–151. (doi:10.2983/035.040.0114)
52. Martin GG, Quintero M, Quigley M, Khosrovian H. 2000 Elimination of sequestered material from the gills of decapod crustaceans. *J. Crustac. Biol.* **20**, 209–217. (doi:10.1651/0278-0372(2000)020[0209:eosmft]2.0.co;2)
53. Burnett KG, Burnett LE. 2015 Respiratory and metabolic impacts of crustacean immunity: are there implications for the insects? *Integr. Comp. Biol.* **55**, 856–868. (doi:10.1093/icb/icv094)
54. Burnett LE, Kendrick MR. 2024 The effects of black gill disease on the respiration of *Penaeus setiferus*, the Atlantic white shrimp, during activity and hypoxia: treadmill studies. *Biol. Bull.* **246**, 52–57. (doi:10.1086/733347)
55. Hauton C. 2012 The scope of the crustacean immune system for disease control. *J. Invertebr. Pathol.* **110**, 251–260. (doi:10.1016/j.jip.2012.03.005)
56. Gillett R. 2008 *Global study of shrimp fisheries*. Part II. Shrimp Fisheries in Selected Countries Shrimp Fishing in the United States of America, pp. 289–312. FAO Fisheries Technical Paper. No. 475. Rome, Italy: FAO. See https://www.fao.org/4/i0300e/i0300e00.htm.
57. Shaw DJ, Dobson AP. 1995 Patterns of macroparasite abundance and aggregation in wildlife populations: a quantitative review. *Parasitology* **111**, S111–S133. (doi:10.1017/s0031182000075855)
58. Verity PG, Borkman DG. 2010 A decade of change in the Skidaway River estuary. I. Hydrography and nutrients. *Estuaries* **25**, 944–960. (doi:10.1007/BF02691343)
59. Harrell FE. 2015 Cox proportional hazards regression model. In *Regression modeling strategies: with applications to linear models, logistic and ordinal regression, and survival analysis* (eds P Bickel, P Diggle, SE Feinberg, U Gather, I Olkin, S Zeger), pp. 475–519. Cham, Switzerland: Springer International Publishing. (doi:10.1007/978-3-319-19425-7_20)
60. Wood CL, Lafferty KD, Micheli F. 2010 Fishing out marine parasites? Impacts of fishing on rates of parasitism in the ocean. *Ecol. Lett.* **13**, 761–775. (doi:10.1111/j.1461-0248.2010.01467.x)
61. Ryder JJ, Webberley KM, Boots M, Knell RJ. 2005 Measuring the transmission dynamics of a sexually transmitted disease. *Proc. Natl Acad. Sci. USA* **102**, 15140–15143. (doi:10.1073/pnas.0505139102)
62. Baker R, Fujiwara M, Minello TJ. 2014 Juvenile growth and mortality effects on white shrimp *Litopenaeus setiferus* population dynamics in the northern Gulf of Mexico. *Fish. Res.* **155**, 74–82. (doi:10.1016/j.fishres.2014.02.026)
63. Gehman ALM, Hall RJ, Byers JE. 2018 Host and parasite thermal ecology jointly determine the effect of climate warming on epidemic dynamics. *Proc. Natl Acad. Sci. USA* **115**, 744–749. (doi:10.1073/pnas.1705067115)
64. Soetaert K, Petzoldt T, Setzer RW. 2010 Solving differential equations in R: package deSolve. *J. Stat. Softw.* **33**, 1–25. (doi:10.18637/jss.v033.i09)
65. Tang B. 1993 Orthogonal array-based latin hypercubes. *J. Am. Stat. Assoc.* **88**, 1392–1397. (doi:10.1080/01621459.1993.10476423)
66. Carnell R. 2024 lhs: Latin Hypercube Samples, 1.2.0. *R package, v. 1.2.0*. Vienna, Austria: R Foundation for Statistical Computing. See https://github.com/bertcarnell/lhs.
67. Hamby DM. 1994 A review of techniques for parameter sensitivity analysis of environmental models. *Environ. Monit. Assess.* **32**, 135–154. (doi:10.1007/bf00547132)
68. Hurvich CM, Tsai C. 1993 A corrected Akaike information criterion for vector autoregressive model selection. *J. Time Ser. Anal.* **14**, 271–279. (doi:10.1111/j.1467-9892.1993.tb00144.x)

69. Burke S, Pottier P, Lagisz M, Macartney EL, Ainsworth T, Drobniak SM, Nakagawa S. 2023 The impact of rising temperatures on the prevalence of coral diseases and its predictability: a global meta-analysis. *Ecol. Lett.* **26**, 1466–1481. (doi:10.1111/ele.14266)
70. McCallum H, Kuris A, Harvell C, Lafferty K, Smith G, Porter J. 2004 Does terrestrial epidemiology apply to marine systems? *Trends Ecol. Evol.* **19**, 585–591. (doi:10.1016/j.tree.2004.08.009)
71. Burge CA, Kim CJS, Lyles JM, Harvell CD. 2013 Special issue Oceans and humans health: the ecology of marine opportunists. *Microb. Ecol.* **65**, 869–879. (doi:10.1007/s00248-013-0190-7)
72. Shodipo MO, Duong B, Graba-Landry A, Grutter AS, Sikkel PC. 2020 Effect of acute seawater temperature increase on the survival of a fish ectoparasite. *Oceans* **1**, 215–236. (doi:10.3390/oceans1040016)
73. Wood CL *et al*. 2023 A reconstruction of parasite burden reveals one century of climate-associated parasite decline. *Proc. Natl Acad. Sci. USA* **120**, 1–9. (doi:10.1073/pnas.2211903120)
74. Díaz F *et al*. 2013 The effects of exposure to critical thermal maxima on the physiological, metabolic, and immunological responses in adult white shrimp *Litopenaeus vannamei* (Boone). *Mar. Freshw. Behav. Physiol.* **45**, 365–374. (doi:10.1080/10236244.2013.771911)
75. Poulin R. 2020 Meta-analysis of seasonal dynamics of parasite infections in aquatic ecosystems. *Int. J. Parasitol.* **50**, 501–510. (doi:10.1016/j.ijpara.2020.03.006)
76. Karvonen A, Rintamäki P, Jokela J, Valtonen ET. 2010 Increasing water temperature and disease risks in aquatic systems: climate change increases the risk of some, but not all, diseases. *Int. J. Parasitol.* **40**, 1483–1488. (doi:10.1016/j.ijpara.2010.04.015)
77. Lee RF, Walker AN, Landers SC, Walters TL, Powell SA, Frischer ME. 2019 Black spot gill syndrome in the northern shrimp, *Pandalus borealis*, caused by the parasitic ciliate *Synophrya* sp. *J. Invertebr. Pathol.* **161**, 40–46. (doi:10.1016/j.jip.2019.01.003)
78. Messick G, Shields J. 2000 Epizootiology of the parasitic dinoflagellate *Hematodinium* sp. in the American blue crab *Callinectes sapidus*. *Dis. Aquat. Org.* **43**, 139–152. (doi:10.3354/dao043139)
79. Bernal J, González D. 1993 Experimental assessment of a degree-day model for predicting the development of parasites in the field. *J. Appl. Entomol.* **116**, 459–466. (doi:10.1111/j.1439-0418.1993.tb01222.x)
80. Jenkins EJ, Veitch AM, Kutz SJ, Hoberg EP, Polley L. 2006 Climate change and the epidemiology of protostrongylid nematodes in northern ecosystems: *Parelaphostrongylus odocoilei* and *Protostrongylus stilesi* in Dall's sheep (*Ovis d. dalli*). *Parasitology* **132**, 387–401. (doi:10.1017/S0031182005009145)
81. Kutz SJ, Hoberg EP, Polley L, Jenkins EJ. 2005 Global warming is changing the dynamics of Arctic host–parasite systems. *Proc. R. Soc. B* **272**, 2571–2576. (doi:10.1098/rspb.2005.3285)
82. Stensgaard AS, Vounatsou P, Sengupta ME, Utzinger J. 2019 Schistosomes, snails and climate change: current trends and future expectations. *Acta Trop.* **190**, 257–268. (doi:10.1016/j.actatropica.2018.09.013)
83. Bartholomew JL, Alexander JD, Hallett SL, Alama-Bermejo G, Atkinson SD. 2022 *Ceratonova shasta*: a cnidarian parasite of annelids and salmonids. *Parasitology* **149**, 1862–1875. (doi:10.1017/s0031182022001275)
84. Groner ML, McEwan GF, Rees EE, Gettinby G, Revie CW. 2016 Quantifying the influence of salinity and temperature on the population dynamics of a marine ectoparasite. *Can. J. Fish. Aquat. Sci.* **73**, 1281–1291. (doi:10.1139/cjfas-2015-0444)
85. St-Hilaire S, Beevers N, Way K, Le Deuff R, Martin P, Joiner C. 2005 Reactivation of koi herpesvirus infections in common carp *Cyprinus carpio*. *Dis. Aquat. Org.* **67**, 15–23. (doi:10.3354/dao067015)
86. Yuasa K, Sano M. 2009 Koi herpesvirus: status of outbreaks, diagnosis, surveillance, and research. *Isr. J. Aquac. Bamidgeh* **61**, 169–179. (doi:10.46989/001c.20562)
87. Defoirdt T. 2016 Implications of ecological niche differentiation in marine bacteria for microbial management in aquaculture to prevent bacterial disease. *PLoS Pathog.* **12**, e1005843. (doi:10.1371/journal.ppat.1005843)
88. Declercq AM, Haesebrouck F, Van den Broeck W, Bossier P, Decostere A. 2013 Columnaris disease in fish: a review with emphasis on bacterium–host interactions. *Vet. Res.* **44**, 1–17. (doi:10.1186/1297-9716-44-27)
89. Holt RA, Sanders JE, Zinn JL, Fryer JL, Pilcher KS. 1975 Relation of Water Temperature to *Flexibacter columnaris* Infection in Steelhead Trout (*Salmo gairdneri*), Coho (*Oncorhynchus kisutch*) and Chinook (*O . tshawytscha*) Salmon. *J. Fish. Res. Board Can.* **32**, 1553–1559. (doi:10.1139/f75-182)
90. Molnár PK, Sckrabulis JP, Altman KA, Raffel TR. 2017 Thermal performance curves and the metabolic theory of ecology-a practical guide to models and experiments for parasitologists. *J. Parasitol.* **103**, 423–439. (doi:10.1645/16-148)
91. Moriarty T, Leggat W, Huggett MJ, Ainsworth TD. 2020 Coral disease causes, consequences, and risk within coral restoration. *Trends Microbiol.* **28**, 793–807. (doi:10.1016/j.tim.2020.06.002)
92. Toth KA, Buckley SF, Noren H, Neely KL, Walker BK. 2024 Broadscale coral disease interventions elicit efficiencies in endemic disease response. *Front. Mar. Sci.* **10**, 1–15. (doi:10.3389/fmars.2023.1302697)
93. Wilber MQ, Yang A, Boughton R, Manlove KR, Miller RS, Pepin KM, Wittemyer G. 2022 A model for leveraging animal movement to understand spatio-temporal disease dynamics. *Ecol. Lett.* **25**, 1290–1304. (doi:10.1111/ele.13986)
94. Miller MW, Lohr KE, Cameron CM, Williams DE, Peters EC. 2014 Disease dynamics and potential mitigation among restored and wild staghorn coral, *Acropora cervicornis*. *PeerJ* **2**, 1–30. (doi:10.7717/peerj.541)
95. Miller LS, La Peyre J, La Peyre M. 2017 Suitability of oyster restoration sites along the Louisiana coast: examining site and stock × site interaction. *J. Shellfish Res.* **36**, 341–351. (doi:10.2983/035.036.0206)
96. Powell EN, Ashton-Alcox KA, Kraeuter JN, Ford SE, Bushek D. 2008 Long-term trends in oyster population dynamics in Delaware Bay: regime shifts and response to disease. *J. Shellfish Res.* **27**, 729–755. (doi:10.2983/0730-8000(2008)27[729:ltiopd]2.0.co;2)
97. Ben-Horin T, Burge C, Bushek D, Groner M, Proestou D, Huey L, Bidegain G, Carnegie R. 2018 Intensive oyster aquaculture can reduce disease impacts on sympatric wild oysters. *Aquac. Environ. Interact.* **10**, 557–567. (doi:10.3354/aei00290)
98. Aoki LR *et al*. 2022 Disease surveillance by artificial intelligence links eelgrass wasting disease to ocean warming across latitudes. *Limnol. Oceanogr.* **67**, 1577–1589. (doi:10.1002/lno.12152)
99. Ward M, Beheshti K. 2023 Lessons learned from over thirty years of eelgrass restoration on the US West Coast. *Ecosphere* **14**, e4642. (doi:10.1002/ecs2.4642)
100. Shields JD, Huchin Huchin-Mian JP, O'Leary PA, Small HJ. 2017 New insight into transmission dynamics of the crustacean pathogen *Hematodinium perezi* (Dinoflagellata) using a novel sentinel methodology. *Mar. Ecol. Prog. Ser.* **573**, 73–84. (doi:10.3354/meps12175)
101. Peeler EJ, Taylor NGH. 2011 The application of epidemiology in aquatic animal health—opportunities and challenges. *Vet. Res.* **42**, 1–15. (doi:10.1186/1297-9716-42-94)

102. Russell RE, Katz RA, Richgels KLD, Walsh DP, Grant EHC. 2017 A framework for modeling emerging diseases to inform management. *Emerg. Infect. Dis.* **23**, 1–6. (doi:10.3201/eid2301.161452)
103. Chapman JM, Kelly LA, Teffer AK, Miller KM, Cooke SJ. 2021 Disease ecology of wild fish: opportunities and challenges for linking infection metrics with behaviour, condition, and survival. *Can. J. Fish. Aquat. Sci.* **78**, 995–1007. (doi:10.1139/cjfas-2020-0315)
104. Tomamichel M *et al*. 2025 Changes in transmission rates drive seasonal patterns of shrimp Black Gill disease. Dryad Digital Repository. (doi:10.5061/dryad.mw6m9068b)
105. Tomamichel M, Walters T, Fox E, Rivera A, O'Hare M, Minniefield A *et al*. 2026 Supplementary material from: Changes in Transmission Rates Drive Seasonal Patterns of Shrimp Black Gill Disease. Figshare. (doi:10.6084/m9.figshare.c.8286581)