ANNUAL REVIEWS

Annual Review of Marine Science

Marine Parasites and Disease in the Era of Global Climate Change

James E. Byers

Odum School of Ecology, University of Georgia, Athens, Georgia 30602, USA;
email: jebyers@uga.edu

ANNUAL REVIEWS CONNECT

www.annualreviews.org

- Download figures
- Navigate cited references
- Keyword search
- Explore related articles
- Share via email or social media

Keywords

enemy escape hypothesis, global warming, nonnative species, sea star wasting disease, shrimp black gill disease, transmission

Abstract

Climate change affects ecological processes and interactions, including parasitism. Because parasites are natural components of ecological systems, as well as agents of outbreak and disease-induced mortality, it is important to summarize current knowledge of the sensitivity of parasites to climate and identify how to better predict their responses to it. This need is particularly great in marine systems, where the responses of parasites to climate variables are less well studied than those in other biomes. As examples of climate's influence on parasitism increase, they enable generalizations of expected responses as well as insight into useful study approaches, such as thermal performance curves that compare the vital rates of hosts and parasites when exposed to several temperatures across a gradient. For parasites not killed by rising temperatures, some simple physiological rules, including the tendency of temperature to increase the metabolism of ectotherms and increase oxygen stress on hosts, suggest that parasites' intensity and pathologies might increase. In addition to temperature, climate-induced changes in dissolved oxygen, ocean acidity, salinity, and host and parasite distributions also affect parasitism and disease, but these factors are much less studied. Finally, because parasites are constituents of ecological communities, we must consider indirect and secondary effects stemming from climate-induced changes in host–parasite interactions, which may not be evident if these interactions are studied in isolation.

INTRODUCTION

All organisms have optimal temperature windows in which they thrive and outside of which they underperform or die (Pörtner & Farrell 2008). Part of the human body's defense system against infection is predicated on this physiological principle. For example, fever's function in endothermic animals, as in humans, is in part to ramp up temperature to an intolerable level for invading pathogens. Although the use of optimal temperature windows to frame the success or failure of pathogens seems quite logical, for a long time the thinking was that a warmer world equals a sicker world (e.g., Harvell et al. 2002). That is, a dominant expectation was that parasites and pathogens, and even biotoxins, would differentially benefit from warming temperatures relative to their hosts. Some prominent examples adhered to this pattern, such as *Vibrio*, coral black band disease, and sea star wasting disease (SSWD). However, if this pattern were universally true, it would mean that most pathogens around the world are living just below their thermal optima, and the warmer temperatures boost them into optimal conditions. More likely, rather than being a universal truism, we were seeing some conspicuous instances of pathogens moving closer to their thermal optima but not noticing others that were pushed outside of their optima and thus declining (Raffel et al. 2013, Carlson et al. 2017, Cizauskas et al. 2017). Particularly in the ocean, where many processes (especially microscopic or internal ones) are often out of sight and out of mind, it can be difficult to view these host–parasite systems with a balanced, holistic perspective.

To gain a proper perspective of temperature effects on marine disease, we should start by clarifying an important discrepancy in tallies of diseases. In an earlier review, Lafferty (2017) enumerated that there are only 102 notable disease-causing agents in marine systems recognized in the literature; specifically, he states, "The literature considers approximately 25 viruses, 33 bacteria, 23 protists, and 21 metazoans to cause notable marine diseases in plants, corals, molluscs, crustaceans, echinoderms, fishes, turtles, and mammals" (p. 474). Yet my colleague Al Camus, a veterinary pathologist, teaches a graduate course on fish disease and pathology where he indexes thousands of parasitic and pathogenic species that cause hundreds of diseases. (Admittedly, these numbers include both freshwater and marine fish species, but they are only for fish and do not include those hosted by myriad marine invertebrate, mammal, and reptile species.) The discrepancy between the two tallies arises from an important distinction: The bulk of Dr. Camus's material pertains to cultured fish species. In the wild, detection may be more difficult, particularly with some viral diseases. Many of the diseases he has catalogued affect young fish life stages that are unlikely to be observed in the act of dying in the wild. By the time any population effect manifests, possibly several years later, evidence of the disease is long past.

In addition to logistical reasons, disease detection in wild fish may be more difficult for biological reasons. Specifically, many disease agents may cause symptoms in individual wild fish but often are not widespread enough that we would notice a population-level effect. As host species come under stressful culture conditions (e.g., in aquaculture), diseases often emerge. Most are probably not new but existed in wild populations at a subclinical level or produced too few mortalities to be observed. The sobering thought to consider about this discrepancy is the latent ability for disease outbreak. That is, if this huge number of pathogens does exist subclinically in wild populations, how will climate change (along with other large, intensifying anthropogenic impacts, such as overfishing, environmental degradation, and diminishing feed sources) influence future outbreaks?

One illuminating example is mycobacteriosis. For years it was considered almost entirely a stress-associated disease of cultured fish, but it has developed into a substantial problem in striped bass (*Morone saxatilis*) in Chesapeake Bay as environmental quality and food sources have declined (Kane et al. 2007). Population-level impacts associated with mycobacteriosis were initially difficult to ascertain because the disease is chronic and disease data were limited. But lately, researchers

have shown that the disease compromises growth and increases mortality (e.g., Latour et al. 2012). Groner et al. (2018) measured mycobacteriosis incidence in mature fish consistently at more than 89% and mortality of diseased fish at approximately 80% each year. The authors modeled the effect of summer sea surface temperature on the fish, both healthy and infected, and found that mortality increased with temperature, to more than 90% in warm years. This indicates that striped bass populations are living close to their maximum thermal tolerance, which is driving increased disease and mortality.

Given the paucity of marine diseases adequately known in an ecological context, especially the potentially large number that may be latent, how can we generate a predictive framework for climate effects on parasites in marine systems? Although some insight could be gained by drawing generalities across taxa and comparing with other systems, several researchers have cautioned against that. Not only are comparisons with terrestrial systems difficult given the sampling imbalance, but marine systems may warrant their own approach due to life history differences and the taxonomic diversity of parasites and hosts (Strathmann 1990, McCallum et al. 2004, Lafferty 2017). For example, marine systems have more parasitic castrators than parasitoids and different transmission modes, such as a larger reliance on environmental transmission and less on biting vectors (Lafferty 2017). Marine organisms also have a preponderance of open population structure (i.e., with highly dispersive larval or adult stages), which likely makes environmental transport as a disease transmission mode far more common, including over long distances (McCallum et al. 2004). Most likely, progress in the near term will continue to be made in specific host–parasite systems where detailed data are readily (and ideally experimentally) gathered.

This review seeks to summarize our current state of knowledge, identify data gaps, and move the field in a predictive direction. To this end, first, I examine thermal performance curves (TPCs) as a promising first-step approach to predict the effects of changing temperature on parasites and disease. Second, I consider reasons why some disease pathologies may be intensified by climate change, supporting these reasons with examples from several prominent case studies. Third, although temperature is one of the biggest drivers of disease that is changing with climate, other important climate-related disease drivers, such as dissolved oxygen, salinity, ocean acidification (OA), and species distributions, are changing and may also play a role; I summarize the effects of these influential yet underrepresented variables in studies of climate change and disease. Finally, I propose that the responses of host–parasite dynamics to climate change require broader investigation than vital rates and transmission rates measured on the host–parasite pair in isolation from their community. Rather, it is often necessary to measure host–parasite dynamics in a natural context, because alterations of host–parasite interactions can ramify to affect the community, and some effects on the community can themselves feed back on host–parasite dynamics.

TEMPERATURE SENSITIVITY AND THERMAL PERFORMANCE CURVES

Similar to other organisms, parasites have temperature optima for their vital rates (Marcogliese 2001), and climate change can affect those rates directly. However, the local host environment buffers parasites from environmental change to some degree. As the condition, competency, and availability of a host become affected by environmental change, these changes to the host influence parasites (Lohmus & Bjorklund 2015). Thus, climate can indirectly affect parasites through alterations to host distribution, immunology, physiology, and behavior (Marcogliese 2001, Thieltges et al. 2008, Callaway et al. 2012), all of which are temperature sensitive. These indirect effects of climate on parasites may surpass the influence of direct effects, and they certainly ensure a strong dependent coupling of parasite responses to their hosts' reactions to climate change.
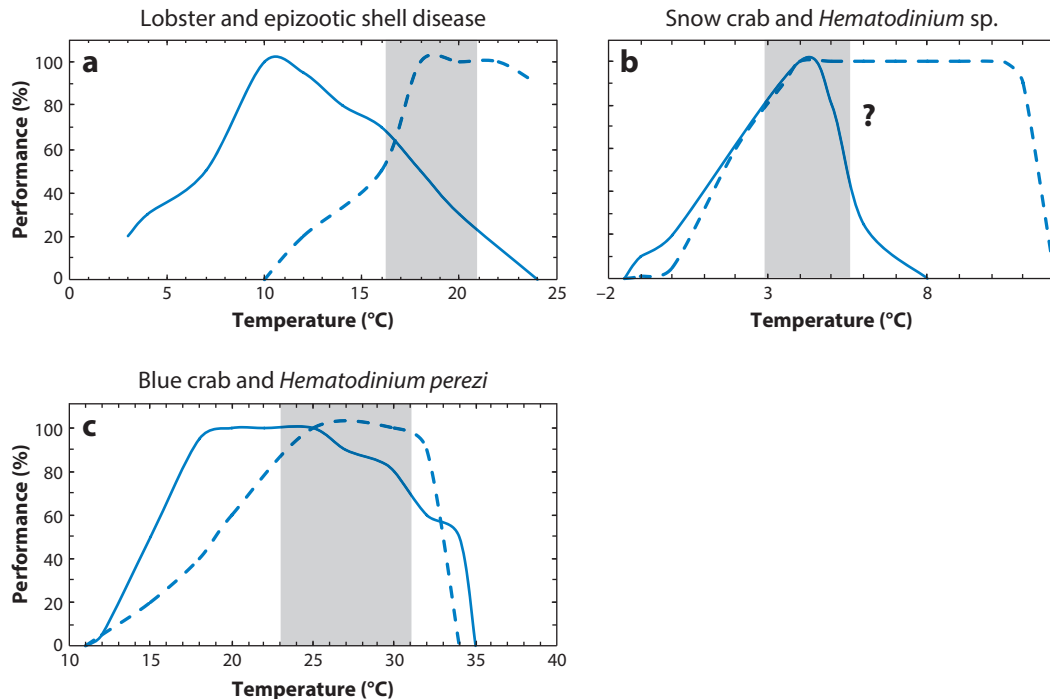
Figure 1

Overlays of thermal ranges for three crustacean hosts (*solid lines*) and their selected pathogens (*dashed lines*). Increases in temperature (*gray boxes*) above the host's optimum favor pathogens in these systems because their growth rates respond quickly to overwhelm innate host immune systems. Performance is given in relative terms of aerobic scope. (*a*) The American lobster (*Homarus americanus*) and the dysbiotic microbiome that causes epizootic shell disease. (*b*) The snow crab *Chionoecetes opilio* and the boreal species of *Hematodinium* sp. The question mark denotes that the temperature range for the parasite is not well known and has been pieced together from field and culture conditions from different hosts. (*c*) The blue crab *Callinectes sapidus* and the parasitic dinoflagellate *Hematodinium perezi*. Figure adapted with permission from Shields (2019); copyright 2019 Oxford University Press.

To predict effects of climate change on host–parasite systems, TPCs are an informative starting point. TPCs quantify the temperature sensitivities of host and parasite vital rates, which can then be compared to examine the relative consequences of altered temperature on each. Because most parasites are obligate, construction of these curves, at least for survivorship rates, usually means comparing the thermal responses of uninfected and infected hosts (the infected host's response typically represents the parasite's response since their fates are intertwined). Shields (2019) provided an example of a simple approach to TPC construction that yields much insight for predicting outbreaks, the emergence of pathogens, and general climate change influences. He compiled from the literature the effects of temperature on several crustacean host–parasite systems. Within each system, he then compiled and relativized the responses of the hosts and parasites into a single performance metric of aerobic scope, which allowed him to overlay the thermal ranges of the hosts with those of the pathogens (**Figure 1**). In three systems he examined, he was able to predict that rising temperatures should favor the parasite more than the host.

Widespread application of Shields's approach may be difficult because few studies have quantified the thermal responses of marine hosts and their pathogens over sufficient temperature ranges to resolve the nonlinearities in TPCs. However, fish aquaculture may be a rich area to look for data to construct TPCs. For example, Karvonen et al. (2010) examined the effects of natural variations

in temperature over 21 years on four diseases of salmon and trout in two fish farms in Finland. Their comparison of host and parasite responses to temperature yielded every possible result, with the prevalence of some pathogens benefiting from warmer conditions, while the prevalence of others decreased or remained unaffected. Also, even though the two studied farms were only 65 km apart, the effects were not spatially universal, emphasizing the importance of specific local conditions.

Although the TPC comparisons done in the previous examples provide good insight, they typically report on the survival, and possibly reproduction, of the host and parasite across temperature. Thus, they usually do not look at all relevant responses of the host–parasite dynamic and may fall short of enabling reliable predictions of temperature effects on the ultimate outcome of their interaction. To determine a net prediction of climate effects on host–parasite dynamics, a more comprehensive approach is often needed. The variables necessary for such an approach are those that influence the growth and spread of disease, including prevalence, intensity, transmission, demographics, abundance, and distribution. Collectively, these critical variables inform the population growth trajectory of a parasite (R_0) and determine whether it is increasing or decreasing. Temperature could induce change in any one of the component variables of R_0, so models can help sort them out, predict a bottom line, and test the significance and sensitivity of each variable (Lafferty & Holt 2003). To evaluate the net effect of these influential variables, TPC construction is a valuable start, but most likely a comprehensive model will be needed to compile the effects of the variables together and predict the net effects.

Several authors have been promoting this approach for parasites, emphasizing that care must be taken in how the TPCs are constructed to ensure they are done in standardized ways, in order to enable modeling approaches and subsequent cross-species comparisons (Molnár et al. 2017). Modeling using TPCs as a starting point has been applied frequently in terrestrial insects, in large part due to their short generation times (which facilitate the quantification of the insects' vital rates) and their importance as disease vectors (e.g., Mordecai et al. 2019). Many marine species, especially invertebrates, have similar characteristics to insects, such as ectothermy and short generation times, and would thus be good candidates for TPC construction and subsequent modeling. Additionally, much of what has been learned from insects may translate directly to crustaceans since they share the same phylum.

Gehman et al. (2018) provided an illuminating example, and their study happens to be one of the only marine examples of constructing and comparing host–parasite TPCs as an important step toward modeling population-level effects. It was conducted on a short-lived marine crab (*Eurypanopeus depressus*) and its rhizocephalan barnacle parasite (*Loxothylacus panopaei*) (**Figure 2**). After constructing TPCs, the authors used those data to help build an epidemiological model to forecast dynamical responses of the host–parasite system to plausible future climate-warming scenarios. In warming scenarios within the coastal southeastern United States, the model predicted declines in parasite prevalence, with local parasite extinction occurring with just 2°C warming. By predicting that the warming temperatures would be more detrimental for the parasite than for the host crab, this work exemplifies that system responses to increased temperature are not universally good for parasites and accentuates the need to measure host and parasite thermal performance to predict infection responses to climate change. In fact, more examples like these for marine host–parasite systems could enable better prediction and broader generalities.

Studer et al. (2010) provided another thorough example. The authors conducted laboratory experiments on the effects of temperature on all transmission steps of the intertidal trematode *Maritrema novaezealandensis* from its first intermediate snail host, *Zeacumantus subcarinatus*, to the second intermediate amphipod host, *Paracalliope novizealandiae*. Although the authors did not construct TPCs per se, their study has many of the same data needed for them. They also did not
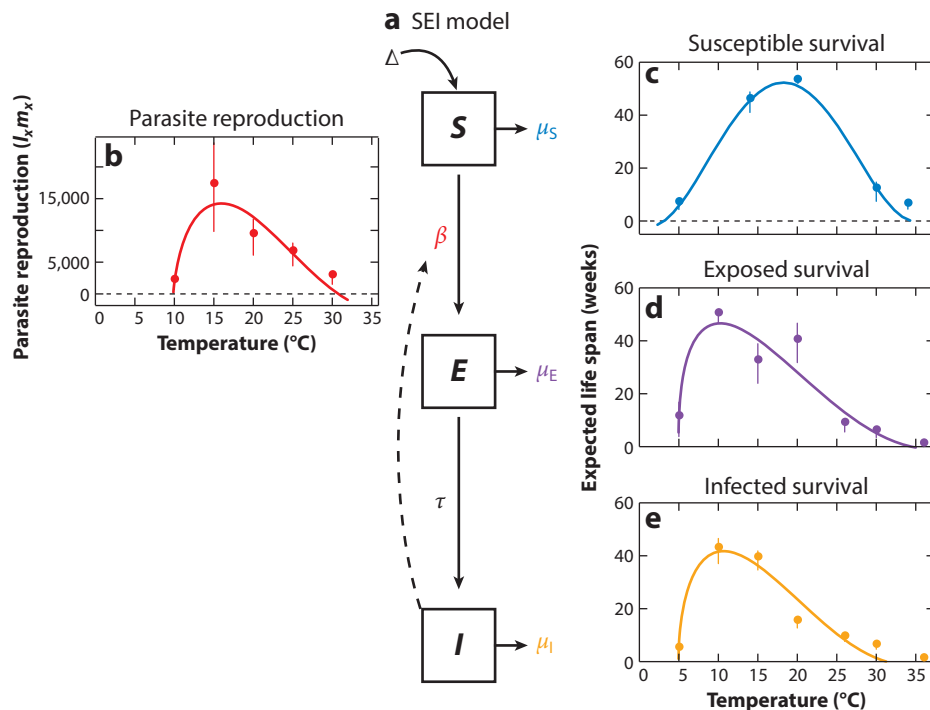
Figure 2

(*a*) A model schematic for transmission of the parasitic rhizocephalan *Loxothylacus panopaei* in the mud crab *Eurypanopeus depressus*. Susceptible hosts (*S*) are recruited at a constant weekly rate (Δ) during the recruitment period and die at per capita rate μ_S (1/expected life span). Susceptible hosts become parasitized at per capita rate βSI, where the transmission rate, β, is an agglomerate parameter assumed to be proportional to larval parasite production, and *I* is the number of infectious hosts with the reproductively mature parasite stage. Exposed hosts (*E*) are infected with reproductively immature parasites that develop at rate τ. Exposed and infectious hosts have mortality rates μ_E and μ_I, respectively. (*b–e*) Model parameters labeled in color are temperature dependent and are parameterized based on thermal performance curves fit to experimental measurements of parasite reproduction (lifetime reproduction, $l_x m_x$) (panel *b*) and host survival for each infection status: susceptible (panel *c*), exposed (panel *d*), and infected (panel *e*). Figure adapted from Gehman et al. (2018).

construct a formal model, but their study is rich in data, and their conclusions regarding the net effects of rising temperatures on the host–parasite dynamic are helped by the fact that one of their quantified rates (mortality of the second intermediate host) shows an obvious bottleneck (i.e., complete mortality at high temperature). Specifically, they found that increasing temperatures were favorable for the parasite, but only to a certain point—namely, when the mortality of the second intermediate host skyrocketed. Thus, temperature differentially affected the various steps of the transmission process, and the amphipod host was the most vulnerable component.

Finally, Lohmus & Bjorklund (2015) explored the sensitivity of host–parasite parameters to small changes in temperature. As a base, they used a model by Taylor et al. (2015) on a multihost trematode life cycle modified to include temperature dependence in four parameters: the reproductive rate of the first intermediate host, the reproductive rate of the parasite, the mortality of the first intermediate host, and the transmission rate of infection to the first intermediate host. They simulated modest temperature changes (0.5°C and 1°C) and three temperature-dependent

death rates of the host (LD_{50} shifted 1°C). Their results show that small temperature changes (0.5°C) and small changes in the sensitivity of host survival to temperature can readily move parasites from nonviability to optimum to extinction. That is, they found strong, qualitatively different outcomes for parasite abundance with small environmental changes. The heightened sensitivity of the responses in this model suggests that understanding net effects of temperature on host–parasite systems may often be difficult to do with only informal comparison (i.e., TPCs) and that a subsequent model may often be necessary.

MECHANISMS OF INCREASED TEMPERATURE-DEPENDENT INTENSITY AND PATHOLOGY

TPCs (and associated models) can help identify which parasites exceed their optimal temperature for growth and transmission as temperature increases. For those parasites for which increases in temperature do not exceed their lethal limits, biologically we might expect their intensity and pathology in infected hosts to increase for three reasons—namely, that higher temperatures lead to (*a*) increased metabolism of parasites, (*b*) increased oxygen stress on hosts, and (*c*) increased transmission windows.

First, because all invertebrates and most fish in the ocean are ectotherms, as a general rule, the metabolisms of hosts and parasites increase with temperature. Increased metabolism means increased feeding or replication of the parasite inside a host, accentuating damage (Fels & Kaltz 2006, Kirk et al. 2018). There are numerous instances of temperature-dependent disease pathologies in distinct host–pathogen systems, but three examples are found in three-spined sticklebacks, corals, and sea stars.

Three-spined sticklebacks (*Gasterosteus aculeatus*) are the intermediate host for a bird tapeworm (*Schistocephalus solidus*). Macnab & Barber (2012) experimentally infected fish and held them at 20°C and 15°C for two months. At 20°C, the tapeworm larvae in fish grew four times more than those held at 15°C, and 100% had sufficient biomass to make them competent to infect definitive host species. By contrast, none of the parasite larvae at 15°C had sufficient biomass to be competent. Because of the strong biomass-to-fecundity relationship in this species, enhanced tapeworm growth at higher temperatures predicts dramatically increased output of infective parasite stages. Higher temperatures therefore enhance rates of parasite feeding, and thus growth and development, which accentuates parasite transmission and parasite effects on the fish hosts (Macnab & Barber 2012).

Corals are particularly sensitive to stress from high temperatures, and many pathogens opportunistically infect them under stress (e.g., Harvell et al. 1999, Bruno et al. 2007, Burge et al. 2014). The fungus *Aspergillus sydowii* infects many taxa, including coral. Two severe outbreaks of *A. sydowii* on the fan coral *Gorgonia ventalina* have occurred during warm-water episodes that killed more than half of the sea fan tissue (Kim & Harvell 2004, Ward et al. 2007). The higher temperatures are thought to increase fungal growth and virulence and reduce host resistance (Ward et al. 2007). Thus, as warmer temperatures become increasingly common, they stress coral hosts and increase pathogen performance, both of which enhance the impacts of disease on the coral. Black band disease and its staghorn coral host (*Acropora muricata*) on the Great Barrier Reef seem to show exactly the same patterns of disease virulence increasing with temperature while the host resistance declines (Boyett et al. 2007).

SSWD has affected at least 20 species of sea stars, particularly along coastlines of the northwestern United States. Although uncertainties exist about the agent of disease and triggers of its outbreaks, one agreed-upon aspect is the diminished effects of SSWD in cooler water (e.g., Eisenlord et al. 2016). In a laboratory experiment, Kohl et al. (2016) tested whether cool

temperatures, representative of winter temperatures in Washington, could slow the progression of morbidity and even prevent SSWD mortality in the sea star *Pisaster ochraceus* compared with average summer temperatures. Cooler temperatures did not prevent SSWD mortality; however, sea stars in cooler water lived more than twice as long as those held at summer temperatures. These data are consistent with experimental studies and field observations during SSWD outbreaks that seem to occur during, or immediately following, temperature increases (Dungan et al. 1982, Bates et al. 2009) and support that SSWD pathology is temperature dependent (Kohl et al. 2016).

Second, a contributing factor to increased parasite intensity and pathology with temperature is accentuated environmental (oxygen) stress experienced by the host. The stress results from higher temperatures that boost the metabolism of ectothermic hosts and simultaneously drive down oxygen saturation in the water, thus increasing oxygen demand while decreasing environmental availability. The resulting oxygen stress deteriorates the host's resistance to infecting pathogens, typically because of reduced immunocompetence and altered physiology and behavior (Harvell et al. 1999, 2002; Bruno et al. 2007; Mann et al. 2013). TPCs, which are measured in controlled laboratory environments, often maintain O_2 at a high, stable level; thus, they might show more positive effects of rising temperatures on a host compared with a natural setting where O_2 negatively covaries with temperature. Low oxygen is particularly common during summer in shallow-water environments, especially if they are eutrophic (Lenihan et al. 2001).

The stress of low oxygen associated with increased temperature can weaken hosts and exacerbate disease impacts (e.g., Mikheev et al. 2014). In these cases, it is the interaction of the parasite with the temperature-dependent stressor that affects the hosts. For example, Mikheev et al. (2014) experimentally demonstrated that low oxygen made rainbow trout (*Oncorhynchus mykiss*) increase ventilation volume through their gills. Because the gills are an important penetration site for larval stages of trematode parasites, more trematodes were pumped over the host's gills in low-oxygen conditions, which increased infection intensity. The number of cercariae acquired by a fish host was positively correlated with ventilation amplitude and volume.

Another example comes from *Hyalophysa lynii*, an apostome ciliate that parasitizes penaeid shrimp in the southeastern United States (Landers et al. 2020). The ciliate lives in the gills of its shrimp host, which triggers an immune response by the host that results in melanized nodules in the gill tissue of infected shrimp, giving the disease its common name of shrimp black gill (**Figure 3**). The highest prevalence of the gill nodules, which are the intense, late-stage symptomatic expression of the disease, occurs in late summer as water temperatures peak (Frischer et al. 2017). The nodules interfere with oxygen exchange, reducing physical endurance and escape responses compared with shrimp without nodules (Frischer et al. 2018). As temperatures warm, oxygen concentration decreases, and the physical impairment intensifies stress on the host.

The third reason that parasite intensity might increase is the tendency of rising temperatures to increase parasite transmission windows, especially at high latitudes, where temperatures are expected to change the most (Galaktionov 2016). In many high-latitude regions, parasite transmission occurs during short summer periods (Karvonen et al. 2010), while the majority of the year is unsuitable for parasites (Hakalahti et al. 2006). For example, increasing temperatures in the Baltic Sea have coincided with the emergence of *Vibrio* infections in northern Europe, with the number and distribution of cases corresponding closely with the temporal and spatial peaks in sea surface temperatures (Baker-Austin et al. 2013). As another example, the transmission window is narrowly calibrated for trematodes, which are often incapable of transmission at low temperatures because physiologically they cannot reproduce. Working in the White Sea, Galaktionov et al. (2006) showed that the emission of trematode cercariae from infected snails is blocked when water temperature drops to 3–5°C; if the temperature remains low, the formation of embryos is suspended. Experiments attempting to infect *Hydrobia ulvae* snails by ingestion of *Bunocotyle*

Figure 3

White shrimp (*Litopenaeus setiferus*) in the southeastern United States are increasingly infected with *Hyalophysa lynii*, an apostome ciliate that parasitizes penaeid shrimp. The ciliate lives in the gills of its shrimp host, which triggers an immune response by the host that results in melanized nodules in the gill tissue of infected shrimp, giving the disease its common name of shrimp black gill. The nodules interfere with oxygen exchange and reduce physical endurance and escape responses compared with shrimp without nodules (Frischer et al. 2018). The top shrimp shows heavy melanized gill tissue due to infection; the bottom shrimp is healthy (or asymptomatic). Photo by Marc Frischer.

progenetica trematode eggs showed that when the water temperature was less than 4°C, the eggs passed through the snail gut intact, and miracidia did not hatch from them (Galaktionov et al. 2006).

Temperature's effect to enlarge transmission windows may be particularly influential through its effect on the hosts of parasites with complex, multihost life cycles. Local definitive host abundance can produce remarkable spatial consistency in infection prevalence in the first intermediate host populations across multiple host generations (Byers et al. 2016). However, climate change could readily alter this by changing the distribution patterns of these definitive hosts. For trophically transmitted parasites (like many trematodes), definitive hosts are often not present during lengthy winter seasons. For example, in the northeastern United States, trematode transmission to snails in the rocky intertidal zone presumably falls to zero during winter, when definitive bird hosts have migrated away (Wells 1994, Byers et al. 2015a). Lengthening summer in these higher-latitude systems may subsequently lengthen the exposure times of hosts to parasites and their vectors. Enlargement of the transmission window may thus dampen annual cycles of infection prevalence and intensity, especially by diminishing their typical winter declines. During winter, although parasite transmission shuts down, parasites often persist inside intermediate hosts. A portion of infected intermediate hosts die during the cold season, because the pathogenic and often intensity-dependent effects of parasitic infection make them more vulnerable to adverse environmental factors (Galaktionov 2016). As a result, the prevalence and intensity of parasites in intermediate hosts usually dips lowest just at the end of the cold season and peaks in late summer, after transmission has been in effect for its full seasonal extent (e.g., Pohley 1976, Kube et al. 2002). As winter warms, hosts will likely remain present longer, extending the intactness of the parasite life cycle and thus reducing the amplitude of annual cycling.

In sum, physiological mechanisms responsive to increasing temperatures suggest that for parasites not killed by rising temperatures, on average their intensity and pathology in infected hosts may increase. These predicted increases arise because of the deterministic ways in which temperature increases the metabolism of parasites, the oxygen stress on hosts, and the residency and activity levels of definitive hosts and vectors of parasites, especially at high latitudes.

OTHER CLIMATE CHANGE DRIVERS

Of all the global change variables, temperature is clearly perceived as the biggest driver of biological processes, receiving most of the attention. But global change alters more than just temperature, including covarying factors that also influence disease prevalence and intensity, such as the acidity, salinity, dissolved oxygen, and species distributions in the ocean (Marcogliese 2016). These four factors not only covary with but also interact with temperature, increasing the difficulty of elucidating the causal effect of global change variables on parasitism. Here, I address how changes in each are predicted to influence host–parasite dynamics.

Although not the focus here, it is worth noting that other influential anthropogenic factors that affect host–parasite dynamics are intensifying over time and thus correlate with climate change even though they are not directly linked with it, such as human fishing pressure, UV radiation, eutrophication, and pollution (Marcogliese & Cone 2001, Lafferty et al. 2004, Lohmus & Bjorklund 2015). For example, elevated ammonium, nitrite, and chlorophyll levels (in addition to temperature) were suggested to predispose farmed Atlantic salmon to amoebic gill disease (Bermingham & Mulcahy 2004). In addition, the deteriorating quality and eutrophication of estuarine waters, combined with higher temperatures, may increase outbreaks of the parasitic dinoflagellate *Pfiesteria* sp. (Burkholder & Glasgow 1997).

Oxygen

The most recent and comprehensive analysis of oxygen changes in the global oceans suggests that there has been an average 2% decline since 1960, mostly through the effects of increased temperature decreasing the solubility of O_2 in the ocean, altering ocean circulation, and changing biotic metabolism (Schmidtko et al. 2017). Because of its strong causal negative covariance with temperature, dissolved oxygen has already been discussed at length above (see the section titled Mechanisms of Increased Temperature-Dependent Intensity and Pathology). But it is important to recognize that oxygen can also be pushed to low levels by factors other than high temperatures. For example, heavy nutrient inputs in enclosed bays can drive eutrophication. Low wind speed and water turbulence can reduce water column mixing, which sets up stratification, causing oxygen depletion at the benthic boundary layer. These low-dissolved-oxygen conditions, even if not driven by or exacerbated by temperature, can influence disease.

As dissolved oxygen decreases, we might expect heightened disease impacts because hypoxia depresses immune function, increasing host susceptibility to disease and mortality from bacterial infections, especially in shrimp and crabs (Le Moullac et al. 1998, Holman et al. 2004, Tanner et al. 2006, Breitburg et al. 2009). In fish, hypoxia can temporarily reduce bactericidal activity, antibody levels, and the production of disease-fighting reactive oxygen species (Valenzuela et al. 2005, Welker et al. 2007). Hypoxia can increase papillomatosis tumors in the roach (*Rutilus rutilus*) (Korkea-aho et al. 2008) and the North Sea dab (*Limanda limanda*) (Mellergaard & Nielsen 1997) as well as outbreaks of mycobacteriosis in Chesapeake Bay striped bass (*Morone saxatilis*) (Vogelbein et al. 1999).

Breitburg et al. (2015) demonstrated that eutrophication in various portions of the Chesapeake Bay was driving hypoxia that was associated with increased infection rates and intensity of *Perkinsus*

marinus infections of oysters. Oysters reduced filtration during intense hypoxia, which should have decreased their exposure to the waterborne *P. marinus*. Experiments suggested that negative effects of hypoxia on the host immune responses were the likely cause of the elevated infections in oysters exposed to hypoxia (despite their lower parasite exposure). Because there is considerable spatial variation in the frequency and severity of hypoxia, the authors concluded that hypoxia likely contributes to landscape-level spatial variation in disease dynamics at the estuary scale.

Salinity

Sea surface salinity in the ocean is driven by the local balance of evaporation and precipitation; thus, salinity varies spatially, with higher salinities found in the evaporation-dominated midlatitudes. The surface waters of these and other saltier regions of the ocean increased in salinity by 4% between 1950 and 2000, a rate expected to increase with rising temperatures (Durack et al. 2012). The effects of sea surface salinity change may be most pronounced biologically in nearshore regions, where shallower depths permit changes to surface-level salinity to influence a greater portion of the water volume. Nearshore environments are also subject to land-based influences such as freshwater runoff, which can drive salinity lower.

The dinoflagellate *Hematodinium* sp. parasitizes the blue crab *Callinectes sapidus* throughout estuaries of the Atlantic coast of the United States. Infections in blue crabs have only been reported from areas where salinity is greater than 11 (Coffey et al. 2012). Experimental exposure of infective stages from in vitro cultures at salinities less than 15 quickly inactivated them (Coffey et al. 2012). Thus, it appears that the parasite is incapable of transmission at low salinities, which explains the lack of natural infections in blue crabs in less saline waters. Furthermore, large catches of blue crab are positively correlated with high river flow, suggesting that the availability of fresh water can benefit the health of the crab population (Sheppard et al. 2003). In fact, blue crabs are known to migrate seasonally up-estuary into fresh water (e.g., Posey et al. 2005), perhaps because this serves as a refuge from parasites that require high salinity, such as *Hematodinium*.

In 2002, the blue crab population in Georgia and the associated fishery crashed (Lee & Frischer 2004). In 2003, landings were depressed 80% compared with historic averages (GEPD 2003). An outbreak of *Hematodinium* sp. stimulated by increased salinities was thought to be a causal factor in the declines. In the early 2000s, extensive droughts throughout much of the southeastern United States increased temperatures and reduced stream inflow and precipitation in estuaries, greatly boosting their salinity. For example, in 2002 in Georgia, the drought increased the average salinity in several estuaries to more than 30 (Lee & Frischer 2004). Thus, the blue crab decline was postulated to be due to severe regional drought that increased coastal salinities, removing the crabs' freshwater refuges and allowing *Hematodinium* to overwhelm the population (GEPD 2003, Gandy et al. 2011). In 2003, the Georgia fishery was declared a disaster by the National Marine Fisheries Service due to drought and disease (GEPD 2003).

Hematodinium is not the only parasite that prefers high salinity. The parasitic copepod *Lepeophtheirus salmonis*, commonly known as sea louse, survives poorly if the salinity is less than 29 (Bricknell et al. 2006). Relatedly, high-freshwater-runoff events that cause low-salinity events seem to decrease sea lice abundance in aquaculture facilities (Callaway et al. 2012). On the Gulf Coast of Florida, the mud crab *Panopeus obesus* is infected by the parasitic castrator *Loxothylacus panopaei*, a rhizocephalan barnacle. Host crabs collected over multiple months along the salinity gradient of three estuaries showed that the prevalence of *L. panopaei* was lower upstream and after freshwater inflow and seasonal rains. Thus, the parasite prevalence seems to be regulated by spatiotemporal low-salinity refuges for its host (Tolley et al. 2006). As another example, *Cliona celata* is a boring sponge that lives in holes it makes in mollusc shells, damaging and even killing oyster hosts,

leading to local oyster population demise in areas with heavy infestations (Rosell et al. 1999, Carroll et al. 2015). *C. celata* and several of the more damaging boring sponges thrive in higher-salinity water (Hopkins 1956, Stubler et al. 2017). Even though oysters themselves also thrive in high-salinity water, they can tolerate lower salinity better than *C. celata*. Thus, North Carolina has been advised to restore oyster reefs in brackish areas of estuaries where developing oyster reefs can avoid *Cliona* infection and the bioerioson and mortality that it causes (Dunn et al. 2014).

It should be noted that of the above examples, all but *Hematodinium* are ectoparasites. Möller (1978) postulated that the high degree of contact of ectoparasites with the water makes them more susceptible to salinity variation. In fact, the stenohalinity (salt sensitivity) of ectoparasites, Möller argued, makes them good bioindicators for the migrations of their hosts into waters of differing salinities. By contrast, endoparasites are often buffered from environmental salinity. In a fascinating experiment, Möller (1978) held European flounder (*Platichthys flesus*) for 9 days in salinity treatments ranging from 0 to 32 and found that the osmolality of the fish's intestines was moderate and identical across all salinities. Unsurprisingly, parasites in the fish intestines were unaffected by changing external water salinities, because the internal salinity of the gut was so stable. However, Möller found that the free-living stages of many of those endoparasites could actually tolerate low salinity very well, including first-stage larvae of the nematode *Contracaecum aduncum*, which developed in salinities ranging from 32 down to 0, and the trematode *Cryptocotyle lingua*, which was infective at salinities down to 4. He also measured high survivorship of larvae of *Anisakis* nematodes from herring (*Clupea harengus*), held for more than 6 months across a full range of salinity treatments, including 0.

Thus, a balanced picture suggests that many marine endoparasites (but not all—e.g., *Hematodinium*) may be buffered from salinity variation. On the other hand, ectoparasites are highly exposed and may be more sensitive than their hosts, perhaps because their high surface-area-to-volume ratios stemming from their small size (relative to the host) increase the cost of osmoregulation. Even within these general rules, there are many examples of parasite species responding idiosyncratically based on their own salinity optima. For example, Koprivnikar et al. (2010) found that free-living stages of one trematode species in the California horn snail (*Cerithidea californica*) did best at very high salinities (40), while another preferred far lower salinities. Blanar et al. (2011) examined helminth and arthropod parasite communities in an estuarine fish host (*Fundualus heteroclitus*) across a strong salinity gradient at opposite ends of two tidal coastal rivers. The opposite ends of each had distinctly different parasite communities, and richness was lower at the higher-salinity ends. These findings suggest that different parasite species respond differently to salinity changes.

Acidification

Due to increasing concentrations of atmospheric CO_2, the pH of the ocean is expected to decrease 0.3–0.5 pH units by 2100, along with a concomitant drop in carbonate saturation states (IPCC 2007, Callaway et al. 2012). The most vulnerable organisms are thought to be species heavily dependent on calcification, such as coral, molluscs, and coccolithophores, principally through their diminished ability to produce and maintain calcium carbonate structures under declining ocean carbonate ion concentrations (Callaway et al. 2012). OA has been recognized as an issue of conservation concern since the early 2000s (Doney et al. 2009). Many experiments on single species followed (for a review, see Kroeker et al. 2013) and gradually led to examination of the effects of OA on ecological processes and interactions, such as predation and competition (Kroeker et al. 2014, Gaylord et al. 2015). Parasitism, however, has largely been neglected in these examinations. There seem to be fewer than two dozen publications on the influence of OA on parasites, and only

half of those are research articles. The majority come from only two research laboratories and focus on trematode parasites interacting with host snails (e.g., Harland et al. 2015, 2016; MacLeod & Poulin 2015; Guilloteau et al. 2016).

OA could influence parasitism by directly affecting parasites, especially the free-living transmissive stages, or could do so indirectly by stressing hosts and thereby altering their susceptibility to parasites (MacLeod 2017). In terms of direct effects, parasites exposed to OA would be challenged by similar problems as all organisms, namely decreased abilities to regulate ions and conduct calcification. In most of the few experimental studies that have been done, exposure to high-CO_2 seawater reduced the survival of free-living trematode cercariae and external metacercarial cysts (Harland et al. 2015, MacLeod & Poulin 2015, Guilloteau et al. 2016). However, some studies have shown mixed (Leiva et al. 2019) or relatively limited (Koprivnikar et al. 2010) responses of trematode cercaraie to lower pH. As with salinity, endoparasites may be better buffered from pH by having less direct contact with the environment. Only when the environmental pH has dropped low enough to affect the internal fluids of the host, presumably by overwhelming its ion regulatory abilities (Reipschlager & Pörtner 1996), would we expect the endoparasite to be directly exposed to a more acidic environment (MacLeod 2017).

In fact, the effects of OA stressing hosts and altering their susceptibility to parasites make the indirect effects of OA on parasitism more complicated to ascertain. For example, despite many direct negative effects of lower pH on trematodes, net transmission of trematodes to the second intermediate host in one of the above-mentioned studies was highest at the lowest tested pH (Harland et al. 2015). Harland et al. (2016) explained this seemingly counterintuitive pattern in a follow-up study that showed experimentally that lower pH produces different host and parasite responses that somewhat, but not completely, offset one another. That is, despite the high mortality of the transmitting cercarial stage of the trematode under low-pH conditions (MacLeod & Poulin 2015), the amphipod hosts were still more infected at lower pH because the susceptibility of amphipods increased sufficiently at low pH to outweigh the reduced life span of infective cercariae. Other studies also showed this pattern, where stress to the host from low pH seems to outweigh stress to the parasite. For example, Guilloteau et al. (2016) quantified higher reproductive output of the trematodes (*Parorchis* sp. and *Philophthalmus* sp.) when their host snail species were exposed to high-CO_2 (low-pH) seawater. Magalhães et al. (2018) experimentally found that low pH led to biochemical alterations in cockles that were linked to increases in trematode infections. McLean et al. (2018) found that lobsters held in elevated-CO_2 treatments for 3–4 months were more susceptible to shell disease. Multiple studies have shown that the immune systems of host organisms are compromised or suppressed under lower-pH conditions (e.g., Bibby et al. 2008, Ellis et al. 2015).

The effects of OA on parasitism may also exhibit reverse causality, i.e., with parasites altering the sensitivity of the host to OA. MacLeod & Poulin (2016b) demonstrated that trematode-infected and uninfected host snails exhibited different tissue glucose concentrations when exposed to pH treatments. Another study by MacLeod & Poulin (2016a) found that snail survival was reduced by low pH, but only in uninfected host snails. That is, the snails were sensitive to pH, but parasitic infection reduced the pH-related mortality of infected individuals relative to uninfected conspecifics.

Host and Parasite Distributional Shifts and Translocations

Climate change is altering distributions of hosts and parasites, affecting the incidence and dynamics of parasites in host populations (Dobson & Carper 1992, Marcogliese 2008). Above (see the section titled Mechanisms of Increased Temperature-Dependent Intensity and Pathology), I

primarily discussed the role of increasing temperatures to alter temporal or seasonal distributions of hosts in complex, multihost life cycles, which enlarges the transmission windows and subsequent intensity of parasites. Here, I focus on spatial distributional shifts, including those that result from multiple influences of global change, emphasizing the shifts that put host and parasite species in contact with novel communities.

Distributional shifts may result from an extension or shift in the parasite's range (Harvell et al. 2002) or their hosts' range (Phillips et al. 2010, Cizauskas et al. 2017). Unless a parasite is able to change host species, as is usually possible only for generalist parasites, the majority of parasites will be dependent on the presence of specific host species. Thus, it seems logical that parasite distributional shifts would often lag behind those of the host (Phillips et al. 2010, Carlson et al. 2017). Hopper et al. (2014) found that a marine snail that had expanded its range substantially northward along the California coast experienced a reduced prevalence of infection and had only 14% of the parasite species richness in the new portion of its range compared with its historical range. However, given sufficient time, parasites can spread to find the hosts in the range-expanding areas. For example, on the US East Coast, the oyster parasites *Perkinsus marinus* and *Haplosporidium nelsoni* now appear much farther north, thanks to warming temperatures that have enabled their spread to previously uninfected oyster populations (Ford 1996, Hofmann et al. 2001). Cohen et al. (2018) modeled how rising temperatures will affect polar sea ice extent, which in turn influences marine mammal distributions. Their modeling suggested greatly increased abundance and aggregations of seals, leading to a subsequent spike in the pinniped disease brucellosis, with a high possibility of spillover of this generalist bacteria disease to other hosts.

The most extreme range shifts are translocations that occur with the aid of human transport, both intentional and unintentional. Human transport of species is also accelerating with increasing globalization (Ruiz & Carlton 2003), and the rate of nonindigenous species (NIS) introductions continues to increase in marine environments (Cohen & Carlton 1998, Byers et al. 2015b). NIS can expose hosts to new parasites, which results either from native hosts transmitting parasites to NIS or from NIS bringing new parasites with them from their native ranges (e.g., Torchin et al. 2003, Paterson et al. 2012, Blakeslee et al. 2013). Movement of species for aquaculture has resulted in accidental parasite introductions, despite close monitoring (Ford 1996, Birkbeck et al. 2011, Krkošek 2017).

Climate change is expected to influence many aspects of NIS (Hellmann et al. 2008). Specifically, rising temperatures are expected to change the modes of introduction of NIS, routes and rates of introduction, and the probabilities and intensity of their post-introduction processes (establishment, spread, and impact) (Hellmann et al. 2008). Thus, we can expect temperatures to alter nonindigenous host and parasite distributions, perhaps quite substantially, with large ramifying effects on their interactions. In fact, in an analysis of viruses, Zell et al. (2008) found that the emergence of new diseases was linked not to global warming but to the translocation of pathogen-infected vectors or hosts. This finding suggests that temperature's largest effect on disease is not direct but rather indirect, occurring through alterations of host and parasite distributions, which control rates of exposure and transmission to new species.

Several marine examples demonstrate that translocation can alter parasite distributions, with climate variables affecting the aftermath. One prominent NIS parasite whose temperature-dependent effects have been well studied was discussed above (see the section titled Temperature Sensitivity and Thermal Performance Curves). Specifically, *Loxothylacus panopaei*, the rhizocephalan barnacle that parasitizes mud crabs on the US East Coast, was introduced from the US Gulf Coast in the 1960s (Van Engel et al. 1966, Kruse & Hare 2007). It is more prevalent and widely distributed in its invasive range compared with its native range (Hines et al. 1997, Kruse & Hare 2007). However, high temperatures in the future are predicted to negatively affect the

parasite more than its crab host, possibly driving the parasite extinct in parts of its introduced range (Gehman et al. 2018). Another NIS parasite with documented temperature-dependent impacts in its transplanted range is *Cryptocotyle lingua*, the numerically dominant trematode infecting the intertidal snail *Littorina littorea* in northeastern North America, where both the snail and the parasite are NIS (Blakeslee et al. 2008). The trematode accounts for more than 90% of all infections in *L. littorea* and also infects two native congeneric *Littorina* species, in which it is the second-most-prevalent trematode species (Blakeslee & Byers 2008, Byers et al. 2008). Temperature interacts with *C. lingua* infection to influence the grazing rate of its snail host (Larsen & Mouritsen 2009). This interaction is discussed in further detail in the following section.

INFLUENCES FROM AND ON THE WIDER ECOLOGICAL COMMUNITY

Just as we need to think broadly about climate-related drivers of host–parasite dynamics, we should also broaden our examination of responses beyond the host–parasite pair. An important response to consider is how far temperature effects on a host and its parasite ramify through a community. In some cases, responses may be pronounced if the community feeds back to affect the host–parasite interaction itself. Influences from and on the community may be difficult to predict a priori because more species are interacting than just the host and parasite. For example, although temperature often increases parasite production, it might also increase the feeding rates of organisms preying on infective parasite stages in the water column as they are released (Thieltges et al. 2008, Goedknegt et al. 2015). Thus, community interactions can be highly influential, offsetting or amplifying outcomes of the pairwise interaction between host and parasite. Soberingly, this suggests that predictions stemming from TPCs of a host and parasite, which are most often measured in isolation, need to be evaluated thoughtfully, since potentially influential community interactions are excluded.

Parasites can account for a substantial portion of biomass in marine systems (Kuris et al. 2008) and up to 78% of food web linkages (Lafferty et al. 2006), highlighting their connectedness within systems. In fact, their connectedness is what has led to the promotion of parasites as sensitive indicators of ecosystem change (Huspeni & Lafferty 2004, Byers et al. 2011, Altman & Byers 2014). Mechanistically, parasites' sensitivity stems from the fact that as numerically prominent, embedded members of communities, their abundance is often affected by direct and indirect biological interactions of the larger community (Marcogliese 2008, Gehman & Byers 2017). An example of the community influence on host–parasite dynamics is the dilution effect, which results when a high proportion of incompetent disease hosts deflect infection from competent hosts, thereby reducing disease transmission (Schmidt & Ostfeld 2001, Civitello et al. 2015). Any effect of climate change that affects biodiversity patterns in a community has the potential to accentuate or diminish the dilution effect. Although well researched in terrestrial and some freshwater systems, the dilution effect has far fewer marine examples (e.g., Thieltges et al. 2008, Kritsky et al. 2011, Samsing et al. 2014, Welsh et al. 2014).

The dilution effect is only one example of a community-level interaction. Other, simpler temperature-sensitive biotic interactions that affect host–parasite dynamics might occur more commonly; some are supported in the marine literature. For example, temperature-sensitive secondary effects of parasites arise when parasites interact with other species in the community to generate indirect effects, intensifying effects on the host or affecting the larger ecological community. In a multispecies community context, indirect or secondary effects mean that a parasite can have lethal effects on its host without directly killing it. For example, black gill disease, described above (see the section titled Mechanisms of Increased Temperature-Dependent Intensity
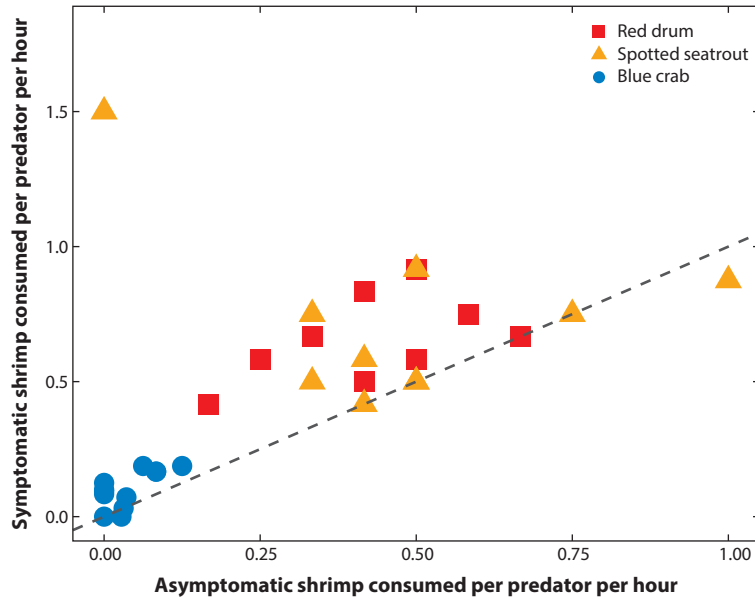
Figure 4

Comparison of predation rates of asymptomatic shrimp and shrimp symptomatic with the ciliate parasite
Hyalophysa lynii. The predator species evaluated were red drum (*Sciaenops ocellatus*), spotted seatrout
(*Cynoscion nebulosus*), and blue crab (*Callinectes sapidus*). Each point represents the predation rate calculated for
each predator species within a single mesocosm stocked with the same initial densities of asymptomatic and
symptomatic shrimp and conducted over a 4-h trial period. The dashed line represents the 1:1 line that
indicates equal consumption of both types of shrimp. Figure adapted with permission from Gooding et al.
(2020); copyright 2020 Elsevier.

and Pathology), compromises respiration of the infected shrimp host. When an infected shrimp is
resting, its oxygen demand is low enough that it can readily survive. However, rising temperatures
decrease oxygen levels, making the already hard job of oxygen extraction even harder. Further-
more, when infected shrimp are made to exert themselves in simulated predation trials, they tire
more quickly and exhibit fewer escape behaviors (Frischer et al. 2018).

    Gooding et al. (2020) probed this system further to examine the net effect of these isolated,
individual physiological responses to disease in a realistic, multispecies context. They exposed
symptomatic shrimp with black gill disease and asymptomatic shrimp to three different fish and
crab predator species in a prey-choice mesocosm experiment at high summer water temperatures.
The predators preferred the infected symptomatic shrimp 1.4–3 times more than asymptomatic
shrimp (Gooding et al. 2020) (**Figure 4**). Thus, the parasite compromised the host's physiol-
ogy, but this had limited consequences until the shrimp were embedded in a community set-
ting (i.e., with predators) at a warm temperature. Although the effect of temperature per se has
not been tested, the warm water temperature is probably key to driving the observed predation
differential between symptomatic and asymptomatic shrimp hosts because warmer temperatures
increase predator metabolism and oxygen stress on infected shrimp hosts. The predation differen-
tial also underscores the dramatic influence that secondary effects of parasites, manifested through
community-level interactions, can have on host–parasite interactions. In fact, one of the largest
effects of a parasite may be its enhancement of its host's vulnerability to predators—an effect that
would not be seen unless the host–parasite relationship is examined in a multispecies context.

The shrimp black gill example illustrates the feedback of the community on a host–parasite interaction, but these effects can also go the other direction, with the host and parasite affecting the community. On the northeastern coast of the United States, the intertidal snail *Littorina littorea*, which is the dominant herbivore in the system, ate 30% less algae when parasitized with the trematode *Cryptocotyle lingua* (Wood et al. 2007). As a consequence of their altered grazing, populations of infected snails in a field experiment created different algal communities compared with uninfected populations (Wood et al. 2007). In this same snail host, Larsen & Mouritsen (2009) experimentally demonstrated that temperature interacted with trematode infection to affect the rate of algal consumption by the snail host. Specifically, although parasite infection decreased a snail's grazing rate, temperature worked to increase it, both by increasing the snail's metabolism and by increasing the energy demands placed on the host by the parasite. Thus, temperature positively affected consumption by snails, but particularly did so for trematode-infected hosts. Higher temperatures therefore neutralized the negative impact that trematodes otherwise have on snail consumption. The temperature levels used were relevant because infected snails consumed less than uninfected ones at 18°C but consumed equal amounts at 21°C. Therefore, the interaction between parasitism and a realistic temperature increase that is in line with expected global warming within this century (3°C), may counteract the presently negative impact of trematodes on snail consumption.

Effects of parasites on communities may be particularly large if the parasites affect prominent consumers or habitat-forming species and ecosystem engineers because of these species' fundamental role in community structure (Byers et al. 2006, Hastings et al. 2007). For example, reef-building coral and oysters are ecosystem-engineering hosts that are increasingly succumbing to disease as temperature rises (as discussed above) and whose decreases will likely cascade strongly to affect the communities for which they play a critical role provisioning habitat (Bruno et al. 2007). As another example, unusually high water temperature (26°C) in the Wadden Sea in May 1990 stimulated a spike in trematode cercariae production in infected snails (*Hydrobia ulvae*), resulting in metacercarial hyperinfection of the second intermediate amphipod host (*Corophium volutator*) (Jensen & Mouritsen 1992, Mouritsen & Jensen 1997). Mass mortality of the amphipod population followed. Because the normally abundant amphipods are ecosystem engineers that build tubes from sand that stabilize the sediment, their absence resulted in substrate erosion and changes to the productivity and structure of benthic communities, including the loss of several macrofaunal groups (Mouritsen et al. 1998, Mouritsen & Poulin 2002). Mouritsen et al. (2005), based on their population model of this system, predicted that crashes of *C. volutator* will occur any time the monthly mean temperature rises 4°C above average. With predictions of increases in the air temperature of up to 6°C in the Wadden Sea area over the next 50 years (Christensen et al. 1998), it seems likely that parasite-induced crashes of this keystone species will occur frequently (Mouritsen et al. 2005).

CONCLUSIONS

The responses of parasites and their hosts to climate change will vary due to the tolerances of the species involved, and possibly even the genotypes involved (Berkhout et al. 2014). However, increasing observational and experimental attention to this issue has enabled insight into possible generalities in expected responses, as well as into useful study approaches. One promising universal approach to examine the response of a host–parasite system to increasing temperature is the construction of TPCs for the host and parasite, along with the resultant modeling they empower. These curves enable rough comparison of the relative effects of temperature on host and parasite vital rates. However, because TPCs are parameterized in isolated experiments or measurements,

it is important to temper their results in some instances. For example, they do not often incorporate transmission dynamics, which are a critically important parameter that is often temperature dependent; sometimes these unmeasured parameters may act in opposing directions to the measured ones. For instance, increased temperature might reduce survivorship of infected hosts, but harming the host might in turn decrease parasite transmission (Lafferty & Holt 2003). Also, temperature variation (as opposed to the mean) may strongly influence infection patterns, an aspect that requires more experimental attention (Ben-Horin et al. 2013).

For parasites that do not overshoot their thermal optima (which TPCs help us determine), we might expect increased intensity and pathology. Because almost all marine parasites and diseases and their hosts (except marine mammals and seabirds) are ectothermic, higher temperatures will lead to increased metabolism, meaning parasites feed faster and more heavily on their hosts. In addition, higher temperatures reduce dissolved oxygen levels, increasing oxygen stress and the resultant susceptibility in hosts, and increase transmission windows because hosts in high latitudes and in multihost parasite life cycles prolong their seasonal residency and activity.

As we progress in our understanding of marine parasites under climate change, it is important to broaden our studies in two ways. First, we should bolster studies that examine how host–parasite dynamics are influenced by other climate change variables, such as increased ocean acidity, lower dissolved oxygen, altered salinity, and altered species geographic distributions. These variables can strongly influence hosts and parasites, and they often act both independently and interactively with temperature. Second, we should examine host–parasite responses from a broader community perspective. With this broadened perspective, we can capture effects that manifest only in the presence of other species, such as those that involve indirect and secondary effects. In some cases, not only does the community feed back to affect the host–parasite interaction via indirect interactions, but the community itself can be affected by climate-induced changes to the host–parasite interaction. These cases underscore that parasites are integrated constituents of ecological systems. To build predictive capacity of host–parasite dynamics, we need to continue to examine the effects of climate change not only on differential tolerances of hosts and parasites but also on broader interactions of parasites within their communities and ecosystems.

DISCLOSURE STATEMENT

ACKNOWLEDGMENTS

LITERATURE CITED

Altman I, Byers JE. 2014. Large-scale spatial variation in parasite communities influenced by anthropogenic factors. *Ecology* 95:1876–87

Baker-Austin C, Trinanes JA, Taylor NGH, Hartnell R, Siitonen A, Martinez-Urtaza J. 2013. Emerging *Vibrio* risk at high latitudes in response to ocean warming. *Nat. Clim. Change* 3:73–77

Bates AE, Hilton BJ, Harley CDG. 2009. Effects of temperature, season and locality on wasting disease in the keystone predatory sea star *Pisaster ochraceus*. *Dis. Aquat. Organ.* 86:245–51

Ben-Horin T, Lenihan HS, Lafferty KD. 2013. Variable intertidal temperature explains why disease endangers black abalone. *Ecology* 94:161–68

Berkhout BW, Lloyd MM, Poulin R, Studer A. 2014. Variation among genotypes in responses to increasing temperature in a marine parasite: evolutionary potential in the face of global warming? *Int. J. Parasitol.* 44:1019–27

Bermingham ML, Mulcahy MF. 2004. Environmental risk factors associated with amoebic gill disease in cultured salmon, *Salmo salar* L., smolts in Ireland. *J. Fish Dis.* 27:555–71

Bibby R, Widdicombe S, Parry H, Spicer J, Pipe R. 2008. Effects of ocean acidification on the immune response of the blue mussel *Mytilus edulis*. *Aquat. Biol.* 2:67–74

Birkbeck TH, Feist SW, Verner-Jeffreys DW. 2011. *Francisella* infections in fish and shellfish. *J. Fish Dis.* 34:173–87

Blakeslee AMH, Byers JE. 2008. Using parasites to inform ecological history: comparisons among three congeneric marine snails. *Ecology* 89:1068–78

Blakeslee AMH, Byers JE, Lesser MP. 2008. Solving cryptogenic histories using host and parasite molecular genetics: the resolution of *Littorina littorea*'s North American origin. *Mol. Ecol.* 17:3684–96

Blakeslee AMH, Fowler AE, Keogh CL. 2013. Marine invasions and parasite escape: updates and new perspectives. *Adv. Mar. Biol.* 66:87–169

Blanar CA, Marcogliese DJ, Couillard CM. 2011. Natural and anthropogenic factors shape metazoan parasite community structure in mummichog (*Fundulus heteroclitus*) from two estuaries in New Brunswick, Canada. *Folia Parasitol.* 58:240–48

Boyett HV, Bourne DG, Willis BL. 2007. Elevated temperature and light enhance progression and spread of black band disease on staghorn corals of the Great Barrier Reef. *Mar. Biol.* 151:1711–20

Breitburg DL, Hondorp DW, Audemard C, Carnegie RB, Burrell RB, et al. 2015. Landscape-level variation in disease susceptibility related to shallow-water hypoxia. *PLOS ONE* 10:e0116223

Breitburg DL, Hondorp DW, Davias LA, Diaz RJ. 2009. Hypoxia, nitrogen, and fisheries: integrating effects across local and global landscapes. *Annu. Rev. Mar. Sci.* 1:329–49

Bricknell IR, Dalesman SJ, O'Shea B, Pert CC, Luntz AJM. 2006. Effect of environmental salinity on sea lice *Lepeophtheirus salmonis* settlement success. *Dis. Aquat. Organ.* 71:201–12

Bruno JF, Selig ER, Casey KS, Page CA, Willis BL, et al. 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. *PLOS Biol.* 5:e124

Burge CA, Eakin CM, Friedman CS, Froelich B, Hershberger PK, et al. 2014. Climate change influences on marine infectious diseases: implications for management and society. *Annu. Rev. Mar. Sci.* 6:249–77

Burkholder JM, Glasgow HB. 1997. *Pfiesteria piscicida* and other *Pfiesteria*-like dinoflagellates: behavior, impacts, and environmental controls. *Limnol. Oceanogr.* 42:1052–75

Byers JE, Altman I, Grosse AM, Huspeni TC, Maerz JC. 2011. Using parasitic trematode larvae to quantify an elusive vertebrate host. *Conserv. Biol.* 25:85–93

Byers JE, Blakeslee AMH, Linder E, Cooper AB, Maguire TJ. 2008. Controls of spatial variation in the prevalence of trematode parasites infecting a marine snail. *Ecology* 89:439–51

Byers JE, Cuddington K, Jones CG, Talley TS, Hastings A, et al. 2006. Using ecosystem engineers to restore ecological systems. *Trends Ecol. Evol.* 21:493–500

Byers JE, Holmes ZC, Blakeslee AMH. 2016. Consistency of trematode infection prevalence in host populations across large spatial and temporal scales. *Ecology* 97:1643–49

Byers JE, Malek AJ, Quevillon LE, Altman I, Keogh CL. 2015a. Opposing selective pressures decouple pattern and process of parasitic infection over small spatial scale. *Oikos* 124:1511–19

Byers JE, Smith RS, Pringle JM, Clark GF, Gribben PE, et al. 2015b. Invasion expansion: time since introduction best predicts global ranges of marine invaders. *Sci. Rep.* 5:12436

Callaway R, Shinn AP, Grenfell SE, Bron JE, Burnell G, et al. 2012. Review of climate change impacts on marine aquaculture in the UK and Ireland. *Aquat. Conserv.* 22:389–421

Carlson CJ, Burgio KR, Dougherty ER, Phillips AJ, Bueno VM, et al. 2017. Parasite biodiversity faces extinction and redistribution in a changing climate. *Sci. Adv.* 3:e1602422

Carroll JM, O'Shaughnessy KA, Diedrich GA, Finelli CM. 2015. Are oysters being bored to death? Influence of *Cliona celata* on *Crassostrea virginica* condition, growth and survival. *Dis. Aquat. Organ.* 117:31–44

Christensen OB, Christensen JH, Machenhauer B, Botzet M. 1998. Very high-resolution regional climate simulations over Scandinavia—present climate. *J. Clim.* 11:3204–29

Civitello DJ, Cohen J, Fatima H, Halstead NT, Liriano J, et al. 2015. Biodiversity inhibits parasites: broad evidence for the dilution effect. *PNAS* 112:8667–71

Cizauskas CA, Carlson CJ, Burgio KR, Clements CF, Dougherty ER, et al. 2017. Parasite vulnerability to climate change: an evidence-based functional trait approach. *R. Soc. Open Sci.* 4:160535

Coffey AH, Li CW, Shields JD. 2012. The effect of salinity on experimental infections of a *Hematodinium* sp. in blue crabs, *Callinectes sapidus*. *J. Parasitol.* 98:536–42

Cohen AN, Carlton JT. 1998. Accelerating invasion rate in a highly invaded estuary. *Science* 279:555–58

Cohen RE, James CC, Lee A, Martinelli MM, Muraoka WT, et al. 2018. Marine host-pathogen dynamics: influences of global climate change. *Oceanography* 31(2):182–93

Dobson AP, Carper R. 1992. Global warming and potential changes in host-parasite and disease-vector relationships. In *Global Warming and Biological Diversity*, ed. RL Peters, TE Lovejoy, pp. 201–17. New Haven, CT: Yale Univ. Press

Doney SC, Fabry VJ, Feely RA, Kleypas JA. 2009. Ocean acidification: the other CO_2 problem. *Annu. Rev. Mar. Sci.* 1:169–92

Dungan ML, Miller TE, Thomson DA. 1982. Catastrophic decline of a top carnivore in the Gulf of California rocky inter-tidal zone. *Science* 216:989–91

Dunn RP, Eggleston DB, Lindquist N. 2014. Oyster-sponge interactions and bioerosion of reef-building substrate materials: implications for oyster restoration. *J. Shellfish Res.* 33:727–38

Durack PJ, Wijffels SE, Matear RJ. 2012. Ocean salinities reveal strong global water cycle intensification during 1950 to 2000. *Science* 336:455–58

Eisenlord ME, Groner ML, Yoshioka RM, Elliott J, Maynard J, et al. 2016. Ochre star mortality during the 2014 wasting disease epizootic: role of population size structure and temperature. *Philos. Trans. R. Soc. B* 371:20150212

Ellis RP, Widdicombe S, Parry H, Hutchinson TH, Spicer JI. 2015. Pathogenic challenge reveals immune trade-off in mussels exposed to reduced seawater pH and increased temperature. *J. Exp. Mar. Biol. Ecol.* 462:83–89

Fels D, Kaltz O. 2006. Temperature-dependent transmission and latency of *Holospora undulata*, a micronucleus-specific parasite of the ciliate *Paramecium caudatum*. *Proc. R. Soc. B* 273:1031–38

Ford SE. 1996. Range extension by the oyster parasite *Perkinsus marinus* into the northeastern United States: response to climate change? *J. Shellfish Res.* 15:45–56

Frischer ME, Fowler AE, Brunson JF, Walker AN, Powell SA, et al. 2018. Pathology, effects, and transmission of black gill in commercial penaeid shrimp from the South Atlantic Bight. *J. Shellfish Res.* 37:149–58

Frischer ME, Lee RF, Price AR, Walters TL, Bassette MA, et al. 2017. Causes, diagnostics, and distribution of an ongoing penaeid shrimp black gill epidemic in the US South Atlantic Bight. *J. Shellfish Res.* 36:487–500

Galaktionov KV. 2016. Transmission of parasites in the coastal waters of the Arctic seas and possible effect of climate change. *Biol. Bull.* 43:1129–47

Galaktionov KV, Irwin SWB, Prokofiev VV, Saville DH, Nikolaev KE, Levakin IA. 2006. Trematode transmission in coastal communities – temperature dependence and climate change perspectives. In *11th International Congress of Parasitology – ICOPA XI*, pp. 85–90. Bologna, Italy: Medimond

Gandy RL, Crowley CE, Machniak AM, Crawford CR. 2011. *Review of the biology and population dynamics of the blue crab*, Callinectes sapidus, *in relation to salinity and freshwater inflow*. Rep., Fla. Fish Wildl. Conserv. Comm., St. Petersburg, FL

Gaylord B, Kroeker KJ, Sunday JM, Anderson KM, Barry JP, et al. 2015. Ocean acidification through the lens of ecological theory. *Ecology* 96:3–15

Gehman AM, Byers JE. 2017. Non-native parasite enhances susceptibility of host to native predators. *Oecologia* 183:919–26

Gehman AM, Hall RJ, Byers JE. 2018. Host and parasite thermal ecology jointly determine the effect of climate warming on epidemic dynamics. *PNAS* 115:744–49

GEPD (Ga. Environ. Prot. Div.). 2003. *Water quality in Georgia*. Rep., GEPD, Atlanta, GA

Goedknegt MA, Welsh JE, Drent J, Thieltges DW. 2015. Climate change and parasite transmission: how temperature affects parasite infectivity via predation on infective stages. *Ecosphere* 6:96

Gooding EL, Kendrick MR, Brunson JF, Kingsley-Smith PR, Fowler AE, et al. 2020. Black gill increases the susceptibility of white shrimp, *Penaeus setiferus* (Linnaeus, 1767), to common estuarine predators. *J. Exp. Mar. Biol. Ecol.* 524:151284

Groner ML, Hoenig JM, Pradel R, Choquet R, Vogelbein WK, et al. 2018. Dermal mycobacteriosis and warming sea surface temperatures are associated with elevated mortality of striped bass in Chesapeake Bay. *Ecol. Evol.* 8:9384–97

Guilloteau P, Poulin R, MacLeod CD. 2016. Impacts of ocean acidification on multiplication and caste organisation of parasitic trematodes in their gastropod host. *Mar. Biol.* 163:96

Hakalahti T, Karvonen A, Valtonen ET. 2006. Climate warming and disease risks in temperate regions – *Argulus coregoni* and *Diplostomum spathaceum* as case studies. *J. Helminthol.* 80:93–98

Harland H, MacLeod CD, Poulin R. 2015. Non-linear effects of ocean acidification on the transmission of a marine intertidal parasite. *Mar. Ecol. Prog. Ser.* 536:55–64

Harland H, MacLeod CD, Poulin R. 2016. Lack of genetic variation in the response of a trematode parasite to ocean acidification. *Mar. Biol.* 163:1

Harvell CD, Kim K, Burkholder JM, Colwell RR, Epstein PR, et al. 1999. Emerging marine diseases—climate links and anthropogenic factors. *Science* 285:1505–10

Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, et al. 2002. Climate warming and disease risks for terrestrial and marine biota. *Science* 296:2158–62

Hastings A, Byers JE, Crooks JA, Cuddington K, Jones CG, et al. 2007. Ecosystem engineering in space and time. *Ecol. Lett.* 10:153–64

Hellmann JJ, Byers JE, Bierwagen BG, Dukes JS. 2008. Five potential consequences of climate change for invasive species. *Conserv. Biol.* 22:534–43

Hines AH, Alvarez F, Reed SA. 1997. Introduced and native populations of a marine parasitic castrator: variation in prevalence of the rhizocephalan *Loxothylacus panopaei* in xanthid crabs. *Bull. Mar. Sci.* 61:197–214

Hofmann E, Ford S, Powell E, Klinck J. 2001. Modeling studies of the effect of climate variability on MSX disease in eastern oyster (*Crassostrea virginica*) populations. *Hydrobiologia* 460:195–212

Holman JD, Burnett KG, Burnett LE. 2004. Effects of hypercapnic hypoxia on the clearance of *Vibrio campbelli* in the Atlantic blue crab, *Callinectes sapidus* rathbun. *Biol. Bull.* 206:188–96

Hopkins SH. 1956. Notes on the boring sponges in Gulf Coast estuaries and their relation to salinity. *Bull. Mar. Sci.* 6:44–58

Hopper JV, Kuris AM, Lorda J, Simmonds SE, White C, Hechinger RF. 2014. Reduced parasite diversity and abundance in a marine whelk in its expanded geographical range. *J. Biogeogr.* 41:1674–84

Huspeni TC, Lafferty KD. 2004. Using larval trematodes that parasitize snails to evaluate a saltmarsh restoration project. *Ecol. Appl.* 14:795–804

IPCC (Intergov. Panel Clim. Change). 2007. Summary for policymakers. In *Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*, ed. S Solomon, D Qin, M Manning, Z Chen, M Marquis, et al., pp. 1–18. Cambridge, UK: Cambridge Univ. Press

Jensen KT, Mouritsen KN. 1992. Mass mortality in two common soft-bottom invertebrates, *Hydrobia ulvae* and *Corophium volutator*—the possible role of trematodes. *Helgol. Meeresunters.* 46:329–39

Kane AS, Stine CB, Hungerford L, Matsche M, Driscoll C, Baya AM. 2007. Mycobacteria as environmental portent in Chesapeake Bay fish species. *Emerg. Infect. Dis.* 13:329–31

Karvonen A, Rintamaki 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.* 40:1483–88

Kim K, Harvell CD. 2004. The rise and fall of a six-year coral-fungal epizootic. *Am. Nat.* 164:S52–63

Kirk D, Jones N, Peacock S, Phillips J, Molnár PK, et al. 2018. Empirical evidence that metabolic theory describes the temperature dependency of within-host parasite dynamics. *PLOS Biol.* 16:e2004608

Kohl WT, McClure TI, Miner BG. 2016. Decreased temperature facilitates short-term sea star wasting disease survival in the keystone intertidal sea star *Pisaster ochraceus*. *PLOS ONE* 11:e0153670

Koprivnikar J, Lim D, Fu C, Brack SHM. 2010. Effects of temperature, salinity, and pH on the survival and activity of marine cercariae. *Parasitol. Res.* 106:1167–77

Korkea-Aho TL, Partanen JM, Kukkonen JVK, Taskinen J. 2008. Hypoxia increases intensity of epidermal papillomatosis in roach *Rutilus rutilus*. *Dis. Aquat. Organ.* 78:235–41

Kritsky DC, Bullard SA, Bakenhaster MD. 2011. First report of gastrocotylinean post-oncomiracidia (Platy-helminthes: Monogenoidea: Heteronchoinea) on gills of flyingfish (Exocoetidae), snapper (Lutjanidae), dolphinfish (Coryphaenidae), and amberjack (Carangidae) from the Gulf of Mexico: decoy hosts and the dilution effect. *Parasitol. Int.* 60:274–82

Krkošek M. 2017. Population biology of infectious diseases shared by wild and farmed fish. *Can. J. Fish. Aquat. Sci.* 74:620–28

Kroeker KJ, Kordas RL, Crim R, Hendriks IE, Ramajo L, et al. 2013. Impacts of ocean acidification on marine organisms: quantifying sensitivities and interaction with warming. *Glob. Change Biol.* 19:1884–96

Kroeker KJ, Sanford E, Jellison BM, Gaylord B. 2014. Predicting the effects of ocean acidification on predator-prey interactions: a conceptual framework based on coastal molluscs. *Biol. Bull.* 226:211–22

Kruse I, Hare MP. 2007. Genetic diversity and expanding nonindigenous range of the rhizocephalan *Loxothy-lacus panopaei* parasitizing mud crabs in the western North Atlantic. *J. Parasitol.* 93:575–82

Kube J, Kube S, Dierschke V. 2002. Spatial and temporal variations in the trematode component community of the mudsnail *Hydrobia ventrosa* in relation to the occurrence of waterfowl as definitive hosts. *J. Parasitol.* 88:1075–86

Kuris AM, Hechinger RF, Shaw JC, Whitney KL, Aguirre-Macedo L, et al. 2008. Ecosystem energetic implications of parasite and free-living biomass in three estuaries. *Nature* 454:515–18

Lafferty KD. 2017. Marine infectious disease ecology. *Annu. Rev. Ecol. Evol. Syst.* 48:473–96

Lafferty KD, Dobson AP, Kuris AM. 2006. Parasites dominate food web links. *PNAS* 103:11211–16

Lafferty KD, Holt RD. 2003. How should environmental stress affect the population dynamics of disease? *Ecol. Lett.* 6:654–64

Lafferty KD, Porter JW, Ford SE. 2004. Are diseases increasing in the ocean? *Annu. Rev. Ecol. Evol. Syst.* 35:31–54

Landers SC, Lee RF, Walters TL, Walker AN, Powell SA, et al. 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

Larsen MH, Mouritsen KN. 2009. Increasing temperature counteracts the impact of parasitism on periwinkle consumption. *Mar. Ecol. Prog. Ser.* 383:141–49

Latour RJ, Gauthier DT, Gartland J, Bonzek CF, McNamee KA, Vogelbein WK. 2012. Impacts of mycobac-teriosis on the growth of striped bass (*Morone saxatilis*) in Chesapeake Bay. *Can. J. Fish. Aquat. Sci.* 69:247–58

Le Moullac G, Soyez C, Saulnier D, Ansquer D, Avarre JC, Levy P. 1998. Effect of hypoxic stress on the immune response and the resistance to vibriosis of the shrimp *Penaeus stylirostris*. *Fish Shellfish Immun.* 8:621–29

Lee RFD, Frischer ME. 2004. The decline of the blue crab: Changing weather patterns and a suffocating parasite may have reduced the numbers of this species along the eastern seaboard. *Am. Sci.* 92:548–53

Leiva NV, Manriquez PH, Aguilera VM, Gonzalez MT. 2019. Temperature and pCO_2 jointly affect the emer-gence and survival of cercariae from a snail host: implications for future parasitic infections in the Hum-boldt Current system. *Int. J. Parasitol.* 49:49–61

Lenihan HS, Peterson CH, Byers JE, Grabowski JH, Thayer GW, Colby DR. 2001. Cascading of habitat degradation: oyster reefs invaded by refugee fishes escaping stress. *Ecol. Appl.* 11:764–82

Lohmus M, Bjorklund M. 2015. Climate change: What will it do to fish-parasite interactions? *Biol. J. Linn. Soc.* 116:397–411

MacLeod CD. 2017. Parasitic infection: a missing piece of the ocean acidification puzzle. *ICES J. Mar. Sci.* 74:929–33

MacLeod CD, Poulin R. 2015. Differential tolerances to ocean acidification by parasites that share the same host. *Int. J. Parasitol.* 45:485–93

MacLeod CD, Poulin R. 2016a. Parasitic infection: a buffer against ocean acidification? *Biol. Lett.* 12:20160007

MacLeod CD, Poulin R. 2016b. Parasitic infection alters the physiological response of a marine gastropod to ocean acidification. *Parasitology* 143:1397–408

Macnab V, Barber I. 2012. Some (worms) like it hot: fish parasites grow faster in warmer water, and alter host thermal preferences. *Glob. Change Biol.* 18:1540–48

Magalhães L, de Montaudouin X, Figueira E, Freitas R. 2018. Trematode infection modulates cockles biochemical response to climate change. *Sci. Total Environ.* 637:30–40

Mann W, Burge C, Mydlarz L. 2013. The effects of climate change on the immunocompetence of the Caribbean Sea fan coral. *Integr. Comp. Biol.* 53:E135

Marcogliese DJ. 2001. Implications of climate change for parasitism of animals in the aquatic environment. *Can. J. Zool.* 79:1331–52

Marcogliese DJ. 2008. The impact of climate change on the parasites and infectious diseases of aquatic animals. *Rev. Sci. Tech.* 27:467–84

Marcogliese DJ. 2016. The distribution and abundance of parasites in aquatic ecosystems in a changing climate: more than just temperature. *Integr. Comp. Biol.* 56:611–19

Marcogliese DJ, Cone DK. 2001. Myxozoan communities parasitizing *Notropis hudsonius* (Cyprinidae) at selected localities on the St. Lawrence River, Quebec: possible effects of urban effluents. *J. Parasitol.* 87:951–56

McCallum HI, Kuris A, Harvell CD, Lafferty KD, Smith GW, Porter J. 2004. Does terrestrial epidemiology apply to marine systems? *Trends Ecol. Evol.* 19:585–91

McLean EL, Katenka NV, Seibel BA. 2018. Decreased growth and increased shell disease in early benthic phase *Homarus americanus* in response to elevated CO_2. *Mar. Ecol. Prog. Ser.* 596:113–26

Mellergaard S, Nielsen E. 1997. Epidemiology of lymphocystis, epidermal papilloma and skin ulcers in common dab *Limanda limanda* along the west coast of Denmark. *Dis. Aquat. Organ.* 30:151–63

Mikheev VN, Pasternak AF, Valtonen ET, Taskinen J. 2014. Increased ventilation by fish leads to a higher risk of parasitism. *Parasite Vector* 7:281

Möller H. 1978. Effects of salinity and temperature on development and survival of fish parasites. *J. Fish Biol.* 12:311–23

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.* 103:423–39

Mordecai EA, Caldwell JM, Grossman MK, Lippi CA, Johnson LR, et al. 2019. Thermal biology of mosquitoborne disease. *Ecol. Lett.* 22:1690–708

Mouritsen KN, Jensen T, Jensen KT. 1997. Parasites on an intertidal *Corophium*-bed: factors determining the phenology of microphallid trematodes in the intermediate host populations of the mud snail *Hydrobia ulvae* and the amphipod *Corophium volutator*. *Hydrobiologia* 355:61–70

Mouritsen KN, Mouritsen LT, Jensen KT. 1998. Change of topography and sediment characteristics on an intertidal mud-flat following mass-mortality of the amphipod *Corophium volutator*. *J. Mar. Biol. Assoc. UK* 78:1167–80

Mouritsen KN, Poulin R. 2002. Parasitism, climate oscillations and the structure of natural communities. *Oikos* 97:462–68

Mouritsen KN, Tompkins DM, Poulin R. 2005. Climate warming may cause a parasite-induced collapse in coastal amphipod populations. *Oecologia* 146:476–83

Paterson RA, Townsend CR, Tompkins DM, Poulin R. 2012. Ecological determinants of parasite acquisition by exotic fish species. *Oikos* 121:1889–95

Phillips BL, Kelehear C, Pizzatto L, Brown GP, Barton D, Shine R. 2010. Parasites and pathogens lag behind their host during periods of host range advance. *Ecology* 91:872–81

Pohley WJ. 1976. Relationships among three species of *Littorina* and their larval digenea. *Mar. Biol.* 37:179–86

Pörtner HO, Farrell AP. 2008. Physiology and climate change. *Science* 322:690–92

Posey MH, Alphin TD, Harwell H, Allen B. 2005. Importance of low salinity areas for juvenile blue crabs, *Callinectes sapidus* Rathbun, in river-dominated estuaries of southeastern United States. *J. Exp. Mar. Biol. Ecol.* 319:81–100

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. Change* 3:146–51

Reipschlager A, Pörtner HO. 1996. Metabolic depression during environmental stress: the role of extracellular versus intracellular pH in *Sipunculus nudus*. *J. Exp. Biol.* 199:1801–7

Rosell D, Uriz MJ, Martin D. 1999. Infestation by excavating sponges on the oyster (*Ostrea edulis*) populations of the Blanes littoral zone (north-western Mediterranean Sea). *J. Mar. Biol. Assoc. UK* 79:409–13

Ruiz GM, Carlton JT, eds. 2003. *Invasive Species: Vectors and Management Strategies*. Washington, DC: Island. 2nd ed.

Samsing F, Oppedal F, Johansson D, Bui S, Dempster T. 2014. High host densities dilute sea lice *Lepeophtheirus salmonis* loads on individual Atlantic salmon, but do not reduce lice infection success. *Aquacult. Environ. Interact.* 6:81–89

Schmidt KA, Ostfeld RS. 2001. Biodiversity and the dilution effect in disease ecology. *Ecology* 82:609–19

Schmidtko S, Stramma L, Visbeck M. 2017. Decline in global oceanic oxygen content during the past five decades. *Nature* 542:335–39

Sheppard M, Walker A, Frischer ME, Lee RF. 2003. Histopathology and prevalence of the parasitic dinoflagellate, *Hematodinium* sp, in crabs (*Callinectes sapidus*, *Callinectes similis*, *Neopanope sayi*, *Libinia emarginata*, *Menippe mercenaria*) from a Georgia estuary. *J. Shellfish Res.* 22:873–80

Shields JD. 2019. Climate change enhances disease processes in crustaceans: case studies in lobsters, crabs, and shrimps. *J. Crustacean Biol.* 39:673–83

Strathmann RR. 1990. Why life histories evolve differently in the sea. *Am. Zool.* 30:197–207

Stubler AD, Robertson H, Styron HJ, Carroll JM, Finelli CM. 2017. Reproductive and recruitment dynamics of clionaid sponges on oyster reefs in North Carolina. *Invertebr. Biol.* 136:365–78

Studer A, Thieltges DW, Poulin R. 2010. Parasites and global warming: net effects of temperature on an intertidal host-parasite system. *Mar. Ecol. Prog. Ser.* 415:11–22

Tanner CA, Burnett LE, Burnett KG. 2006. The effects of hypoxia and pH on phenoloxidase activity in the Atlantic blue crab, *Callinectes sapidus*. *Comp. Biochem. Phys. A* 144:218–23

Taylor RA, White A, Sherratt JA. 2015. Seasonal forcing in a host-macroparasite system. *J. Theor. Biol.* 365:55–66

Thieltges DW, Bordalo MD, Hernandez AC, Prinz K, Jensen KT. 2008. Ambient fauna impairs parasite transmission in a marine parasite-host system. *Parasitology* 135:1111–16

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. Organ.* 70:243–50

Torchin ME, Lafferty KD, Dobson AP, McKenzie VJ, Kuris AM. 2003. Introduced species and their missing parasites. *Nature* 421:628–30

Valenzuela A, Silva V, Tarifeno E, Klempau A. 2005. Effect of acute hypoxia in trout (*Oncorhynchus mykiss*) on immature erythrocyte release and production of oxidative radicals. *Fish Physiol. Biochem.* 31:65–72

Van Engel WA, Dillon WA, Zwerner D, Eldridge D. 1966. *Loxothylacus panopaei* (Cirripedia, Sacculinidae) an introduced parasite on a xanthid crab in Chesapeake Bay, U.S.A. *Crustaceana* 10:110–12

Vogelbein WK, Zwerner DE, Kator H, Rhodes MW, Cardinal J. 1999. *24th Annual Eastern Fish Health Workshop, Atlantic Beach, NC, 1999*. Rep., Natl. Fish Health Res. Lab., Biol. Resour. Div., US Geol. Surv., Washington, DC

Ward JR, Kim K, Harvell CD. 2007. Temperature affects coral disease resistance and pathogen growth. *Mar. Ecol. Prog. Ser.* 329:115–21

Welker TL, McNulty ST, Klesius PH. 2007. Effect of sublethal hypoxia on the immune response and susceptibility of channel catfish, *Ictalurus punctatus*, to enteric septicemia. *J. World Aquacult. Soc.* 38:12–23

Wells JV. 1994. Correlates of the distribution and abundance of wintering gulls in Maine. *J. Field Ornithol.* 65:283–94

Welsh JE, van der Meer J, Brussaard CPD, Thieltges DW. 2014. Inventory of organisms interfering with transmission of a marine trematode. *J. Mar. Biol. Assoc. UK* 94:697–702

Wood CL, Byers JE, Cottingham KL, Altman I, Donahue MJ, Blakeslee AMH. 2007. Parasites alter community structure. *PNAS* 104:9335–39

Zell R, Krumbholz A, Wutzler P. 2008. Impact of global warming on viral diseases: What is the evidence? *Curr. Opin. Biotechnol.* 19:652–60

Contents

Errata

An online log of corrections to *Annual Review of Marine Science* articles may be found at
http://www.annualreviews.org/errata/marine