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Climate change enhances disease processes in crustaceans: case studies in lobsters, crabs, and shrimps

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ABSTRACT

Climate change has resulted in increasing temperature and acidification in marine systems. Rising temperature and acidification act as stressors that negatively affect host barriers to infection, thus enhancing disease processes and influencing the emergence of pathogens in ecologically and commercially important species. Given that crustaceans are ectotherms, changes in temperature dominate their physiological and immunological responses to microbial pathogens and parasites. Because of this, the thermal ranges of several crustacean hosts and their pathogens can be used to project the outcomes of infections. Host factors such as molting, maturation, respiration, and immune function are strongly influenced by temperature, which in turn alter the host's susceptibility to pathogens, further amplifying morbidity and mortality. Microbial pathogens are also strongly influenced by temperature, arguably more so than their crustacean hosts. Microbial pathogens, with higher thermal optima than their hosts, grow rapidly and overcome host immune defenses, which have been weakened by increased temperatures. Pathogen factors such as metabolic rates, growth rates, virulence factors, and developmental rates are often enhanced by rising temperature, which translates into increased transmission, dispersal, and proliferation at the population level, and ultimately emergence of outbreaks in host populations. Less well known are the effects of acidification and salinity intrusion on host-pathogen processes, but they operate alongside temperature, as multiple stressors, that impose significant metabolic and physiological demands on host homeostasis.

Key Words: crustacean fisheries, disease outbreaks, environmental stressors, epidemics, global climate change, global warming, pathogens, parasites, thermal range, ocean acidification, salinity

INTRODUCTION

Temperatures in terrestrial and aquatic systems have been increasing for several decades as a result of anthropogenically induced climate change. Increased climatic variability associated with climate change has resulted in many organisms experiencing episodic or periodic temperature shifts outside their normal thermal ranges (Pörtner *et al.*, 2014). One response to rising temperatures has been population-level latitudinal shifts northward, and this has been well documented in terrestrial systems, particularly among vector-borne pathogens of humans and livestock (Smith *et al.*, 2014). Populations of invertebrate vectors, primarily mosquitoes, kissing bugs, ticks, and snails, respond positively to changes in rainfall, humidity, temperature, and phenological shifts in seasonality. Latitudinal shifts in marine systems have been reported in several fish species as well as in the American lobster in the Gulf of Maine (Nye *et al.*, 2009; Pinsky *et al.*, 2013). Additionally, many marine and estuarine invertebrates have experienced increasingly detrimental influences from climate change, including weakening of their shells or exoskeletons due to the increased absorption of CO_2 into seawater (i.e., acidification), increased exposure to hypoxia, changes in salinity regimes, and the notable emergence of many pathogens and disease conditions in response to these stressors (Pörtner *et al.*, 2014).

The marine environment has experienced several changes induced by climate change, including rapid increases in temperature (fluctuations and sustained levels), acidification, increases in sea-level rise with consequent inundation of low-lying areas, enhanced flooding, salinity intrusion into freshwater regions, loss of Arctic ice cover, and an increase in the severity and frequency of storm events (Pörtner *et al.*, 2014). The effect of these stressors on marine organisms have several manifestations because of the

complex interrelationships between temperature, the solubility of gases (i.e., oxygen and CO2), and increasing levels of CO_2 in the atmosphere being absorbed into the oceanic carbonate cycle, as well as the disparate physiological and ecological downstream effects in marine organisms (Marcogliese, 2008).

Concurrent with climate change and effects on marine systems, disease outbreaks have been on the rise over the last several decades (Harvell *et al.*, 1999; Ward & Lafferty 2004; Shields, 2012). Given the increasing emergence and outbreaks of pathogens in marine system, it is useful to identify a unifying conceptual framework to address the emergence and spread of pathogens in marine ectotherms. Temperature plays a dominant role in the metabolism of ectothermic fishes and invertebrates, as well as their pathogens. Accordingly, studies on temperature effects in crustaceans have shown that rising temperatures can affect homeostasis resulting in metabolic stress and disease (Dove *et al.*, 2004, 2005), change host susceptibility to pathogens (Le Moullac & Haffner, 2000; Shields *et al.*, 2005, 2007), and spur proliferation of pathogens that are no longer kept in check by lower temperatures at which the host-pathogen relationships remain stable (Huchin-Mian *et al.*, 2018).

Here I review the effect of climate change on disease processes in crustaceans. The focus is on commercially important decapods, because they have received more attention with respect to physiological and ecological studies. Disease processes include host factors, such as susceptibility, morbidity, and mortality, as well as pathogen factors, such as transmission, dispersal, and proliferation. Thermal range is used as a conceptual framework, because it illustrates how temperature stress affects responses in the host-pathogen association, shifting the balance in favor of pathogens and ultimately leading to their outbreaks in host populations. The case studies are on commercially important crustaceans, particularly lobsters and crabs, because they are better studied than most crustaceans, their pathogens are mostly known, and their host-pathogen relationships can provide useful comparisons (Shields *et al.*, 2015a). It is noteworthy that the interrelationship between climate change and disease has received little attention in ecologically important taxa of crustaceans, such as copepods, amphipods, and krill, even though they are important to many biological processes because of their sheer biomass in marine systems.

Climate change, thermal ranges, and lobster diseases

Because an increase in temperature is the dominant effect of climate change, an examination of thermal range and homeostasis helps to establish the effects of thermal stress on an organism (Pörtner, 2002, 2012; Pörtner & Farrell, 2008). For example, animals living under near optimal conditions have a high scope for aerobic performance (Fig. 1). As temperatures change an organism approaches its pejus temperature, which is the temperature where metabolic performance declines and limits long-term tolerance. Between the pejus and critical temperatures (high and low ranges), increasing energy demands are placed on respiration to maintain adequate aerobic performance, i.e., conditions become stressful as more energy is diverted to maintain performance, and there is a limit to long-term tolerance. At critical temperatures, the animal's performance degrades as it transitions to anaerobic metabolism and survival can be compromised; finally, at denaturation temperatures there is a rapid onset of cell damage leading to death.

The effects of temperature on a host-pathogen system can be examined by overlaying the thermal ranges of the host with the pathogen of interest (Fig. 2). It is clear from this simple analysis that when the pathogen's optimal temperature is greater than the host's optimum, that the establishment or proliferation of the pathogen will likely overwhelm the host's ability to control the infection. Although simplistic, this overlay can be used to model population-level effects (Gehman *et al.*, 2018), but it does not address the underlying mechanisms. It nevertheless provides a framework for predicting outbreaks or establishing criteria for the

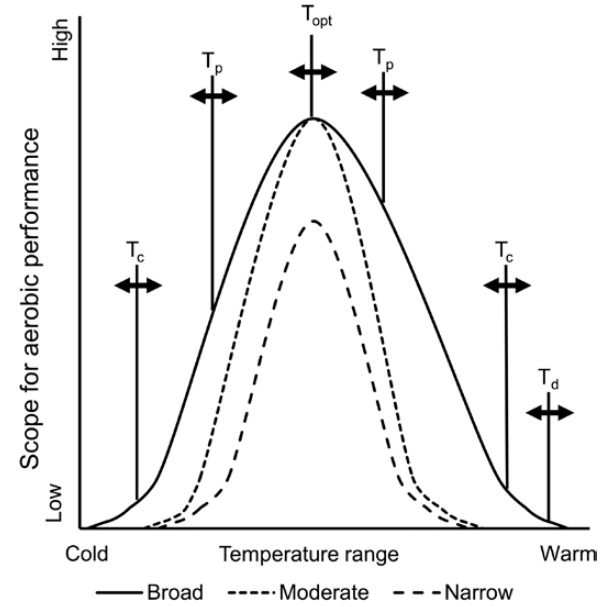

Figure 1. Scope for aerobic performance in relation to thermal range of an organism. Performance is at its maximum at an organism's optimum temperature threshold (T_{opt}). Also shown are the limits to long-term tolerance (pejus temperature, T_p), where extended time leads to loss in performance or population abundance, and critical temperatures (T_c), above and below which performance degrades rapidly as anaerobiosis. Denaturation temperatures (T_d) rapidly lead to cell death. Dashed lines refer to declines in performance in degraded systems such as hypoxia or ocean acidification. Redrawn from Pörtner, 2012, Pörtner *et al.*, 2014.

emergence of pathogens. The underlying mechanisms whereby rising temperatures influence host and pathogen factors in crustaceans will be examined with a few examples as they are modulated in different ways.

Finding data on the thermal range for crustacean hosts and their pathogens can be difficult because few studies have examined these for either set of organisms in much detail. The American lobster, *Homarus americanus* H. Milne Edwards, 1837, offers excellent physiological data on thermal range and other environmental ranges for a crustacean host (Fig. 3). The thermal range for the lobster extends broadly from 4 °C to 32 °C, but realistically the pejus temperature is between 4°C and 15 °C, with an optimum of around 12 °C, critical temperatures are below 4 °C and above 25 °C, and the denaturation temperature is below freezing, –1.4 °C, and above 32 °C (McLeese, 1956). Temperatures above 15 °C can result in immune dysfunction in this species in the form of reduced phagocytosis (Paterson *et al.*, 1976; Steenbergen *et al.*, 1978; Dove *et al.*, 2005), but the species can thrive at temperatures up to 18 °C. Temperatures above 18 °C–20 °C cause homeostatic imbalances in the form of a metabolic calcinosis, the deposition of calcium salts into soft tissues that can be fatal over long warming periods (Dove *et al.*, 2004). Temperature also affects lobster behavior, with much less agonistic behavior exhibited at 5 °C compared to 10 °C (Hoffman *et al.*, 1975), but few studies have examined this aspect at higher temperatures. American lobsters have a thermal optimum between 10 °C and 12 °C in the Gulf of Maine and southern Nova Scotia, Canada, but they live in temperatures over 20 °C in Long Island Sound, eastern USA, albeit maturing there at smaller sizes and rarely attaining large sizes as those seen in the Gulf of Maine (Landers *et al.*, 2002). Moreover, lobsters in Long Island Sound have experienced persistent disease issues in response to environmental stressors over the last three decades, in part due to temperature stress (Tlusty *et al.*, 2007; Shields, 2013).

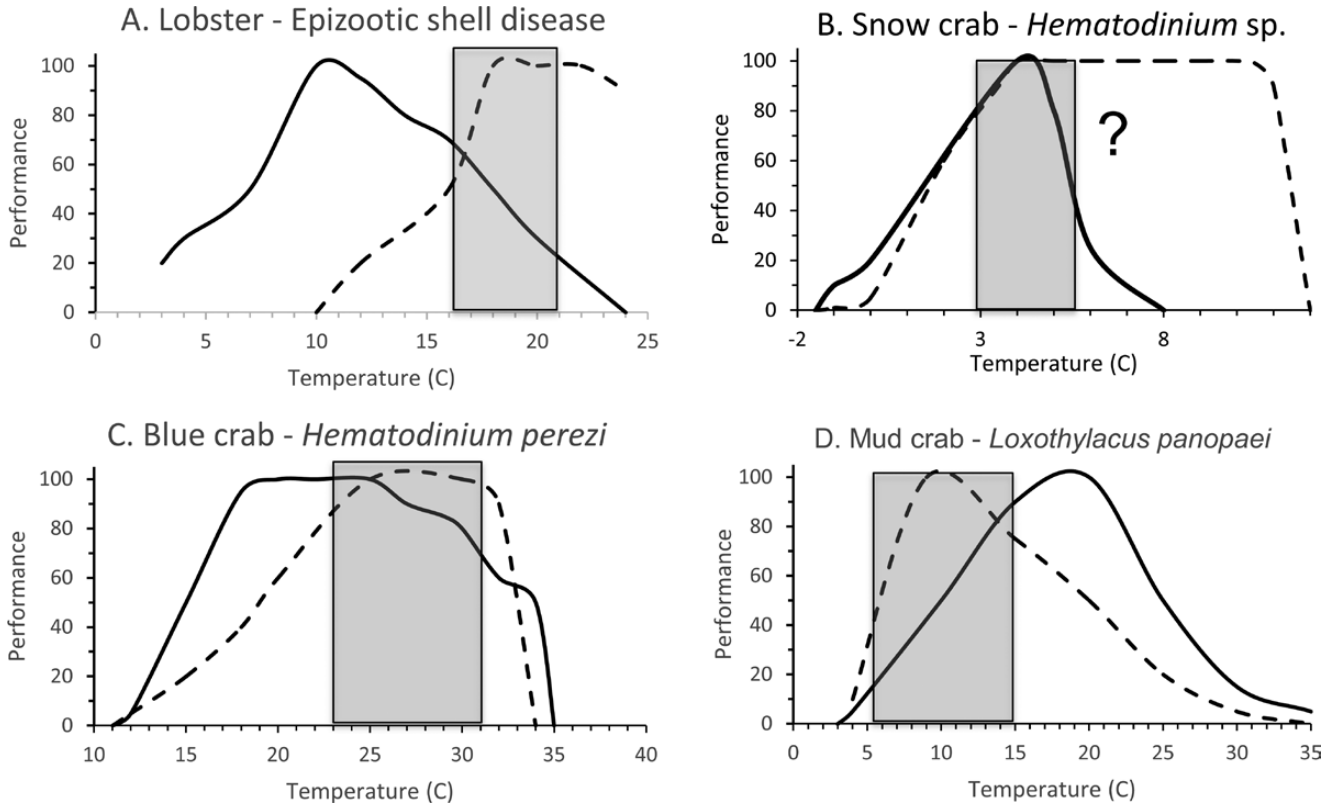

Figure 2. Overlays of thermal ranges for four crustacean hosts (solid lines) and their selected pathogens (dashed lines). Increases in temperature above the host's optimum (gray boxes) favor pathogens in three of these systems because their growth rates respond quickly to overwhelm innate host immune systems. Warmer temperatures favor the host in the fourth example. Performance is given in relative terms of aerobic scope. Redrawn with permission from Shields & Huchin-Mian (In press). The American lobster, *Homarus americanus*, and the dysbiotic microbiome that causes epizootic shell disease (estimated from McLeese, 1956; Glenn & Pugh, 2006; Barris *et al.*, 2018) (**A**). The snow crab *Chionoecetes opilio* and the boreal species of *Hematodinium* sp. (estimated from Meyers *et al.*, 1987; Appleton & Vickerman, 1998; Shields *et al.*, 2007). Question mark refers to the temperature range for the parasite not being well known and pieced together from field and culture conditions from different hosts (**B**). The blue crab *Callinectes sapidus* and the parasitic dinoflagellate *Hematodinium perezi* (estimated from Huchin-Mian *et al.*, 2017) (**C**). The mud crab *Eurypanopeus depressus* and the rhizocephalan barnacle *Loxothylacus panopaei* (from Gehman *et al.*, 2018). This is also the only metazoan parasite used in these examples (**D**).

Microbial pathogens, or microparasites, have well-known responses to temperature. These responses are often measured using the Q_{10} temperature coefficient, which is the rate of increase in cell growth, enzyme activity, respiration, or cell division over a 10 °C increase in temperature. Bacterial growth rates have an estimated Q_{10} of 3.9 in marine systems, whereas they are 2.1 in freshwater systems (White *et al.*, 1991). Because microbial pathogens often have different thermal ranges from that of their host, with optimum conditions for microbial growth at higher temperatures, the resultant rapid increase in pathogen density, or severity of infection, frequently outstrips the immune defenses in their ectothermic hosts. Host defenses are also regulated in part by temperature and by Q_{10} values for phagocytic activity, enzyme responses (e.g., superoxide dismutases), or other immune functions. These Q_{10} values are often less than 2.0, i.e., lower than that of most microbial pathogens in marine systems. Phagocytic activity in lobsters in fact declines markedly with increasing temperatures over 15 °C (Steenbergen *et al.*, 1978; Dove *et al.*, 2005), further highlighting the host's increased susceptibility to microbial pathogens with climate change. Thus, for ectotherms estimates of the pejus and critical temperatures for the host and pathogen can be used to project which temperature may lead to increased host susceptibility and which may enhance growth rates of their microbial pathogens.

With respect to lobster pathogens, an analysis of their thermal ranges explains in part the pathogenicity of *Aerococcus viridans* var. *homari* Williams, Hirsch & Cowan, 1953, the causative agent of gaffkemia, and the effects of epizootic shell disease (ESD) on lobster health. Temperatures below 4 °C–6 °C provide a refuge from gaffkemia, which becomes progressively more pathogenic to lobsters at 10 °C, and rapidly fatal to them over 15 °C (Stewart *et al.*, 1969a, 1980). The bacterium is clearly responding to temperature with increased growth rates and the lobster host cannot maintain an adequate defense, due in part to its reduced phagocytic activity and possibly due to a loss of glycogen and other metabolic resources. Gaffkemia is primarily a disease caused by handling stress. It is transmitted through open wounds in contact with contaminated benthos (Stewart *et al.*, 1969b). It was once widespread due to the practice of "pegging" the claws such that lobsters held in crowded conditions could not kill each other. Once this practice stopped, the prevalence of the pathogen declined considerably.

In contrast, ESD is a complicated environmental disease involving a dysbiotic, or altered, bacterial community, increasing temperature, and contaminants (Fig. 4; see review by Shields, 2013). Rising temperatures have impacted both host and pathogen factors in this system. Temperatures below 10 °C provide some protection against the establishment and proliferation of ESD, whereas the disease proliferates rapidly at temperatures above 16 °C–18 °C (Glenn & Pugh, 2006; Barris *et al.*, 2018). In the lobster, temperatures above 15 °C–16 °C stimulate rapid metabolic growth, molting, and maturation, but their immune defenses become increasingly compromised (Paterson *et al.*, 1976; Steenbergen *et al.*, 1978), which enhances their susceptibility to this suite of pathogens. ESD has a broad distribution centered in central Long

Island Sound and eastward into Buzzards Bay, MA, USA (Castro & Angell, 2000; Castro & Somers, 2012; Howell, 2012). It has a strong association with warming temperatures (Fig. 5) (Glenn & Pugh, 2006; Barris *et al.*, 2018) and, although it occurs in the Gulf of Maine, its prevalence and distribution are reduced there primarily due to lower temperatures (Glenn & Pugh, 2006; Reardon

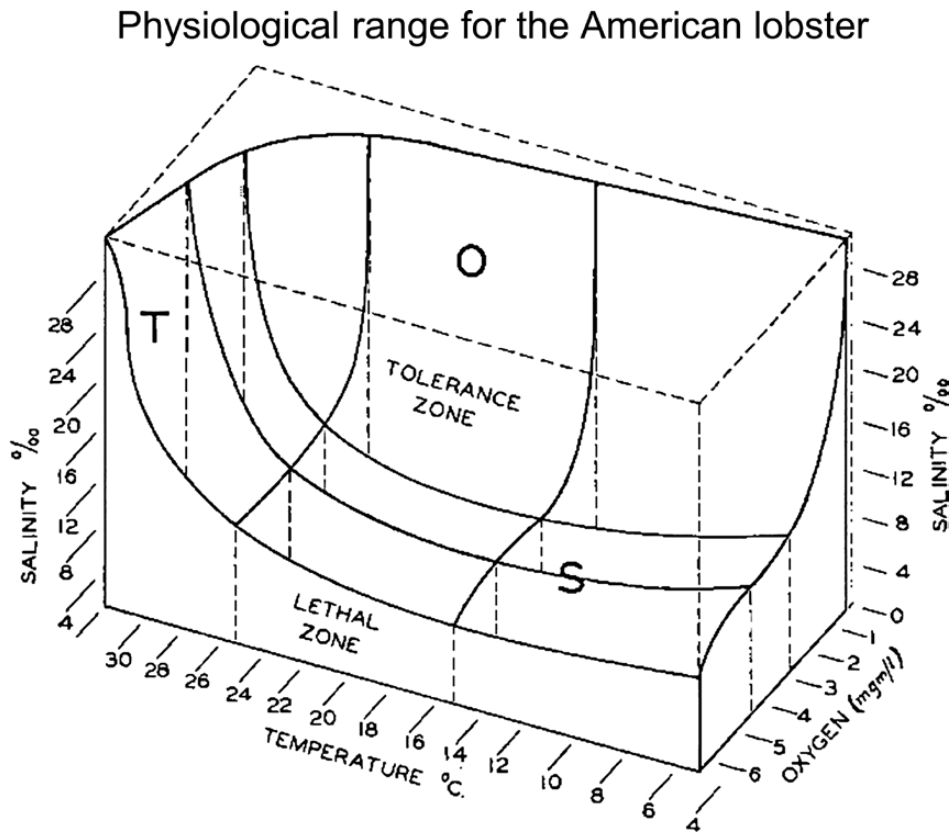

Figure 3. Physiological range as mortality of the American lobster in relation to temperature (T), salinity (S), and dissolved oxygen (O). From McLeese, 1956, with permission.

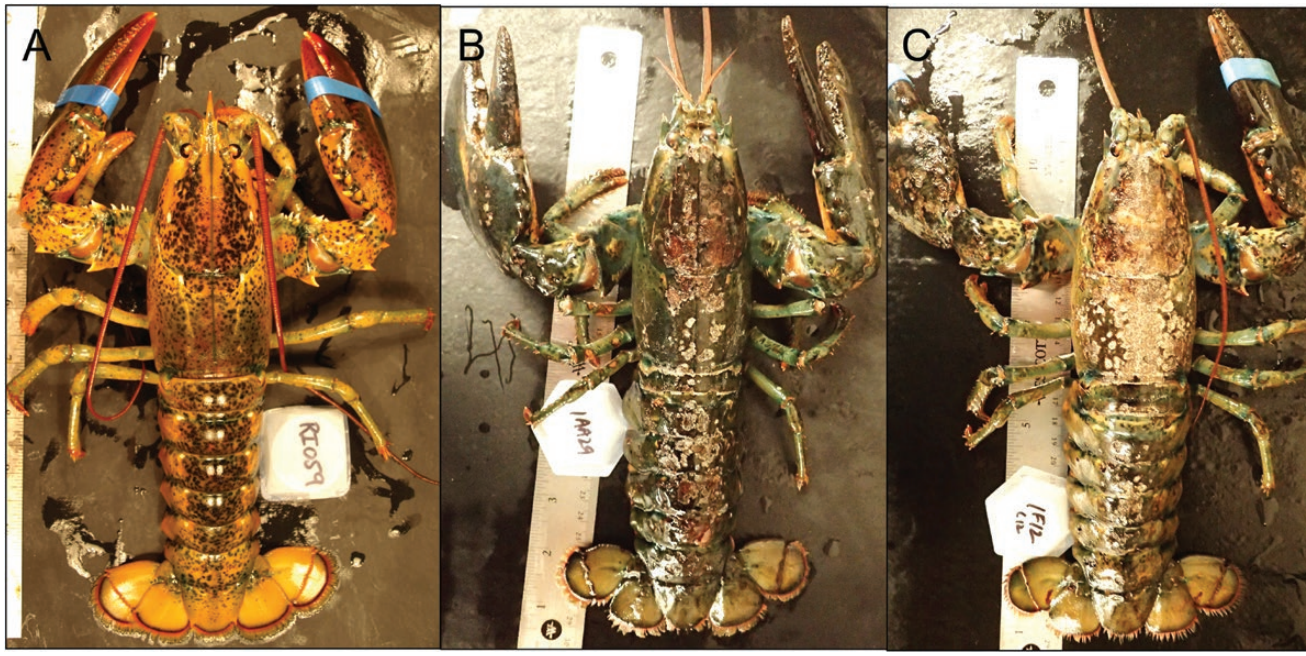

Figure 4. Healthy lobster exhibiting normal coloration of the carapace (**A**). Lobster with a moderate infection of epizootic shell disease on the dorsal carapace and abdomen (**B**). lobster with a heavy infection of epizootic shell disease covering much of the dorsal carapace (**C**). This figure is available in color at *Journal of Crustacean Biology* online.

et al., 2018). The continued expansion and dispersal of ESD has been modeled using thermal range data combined with projected increases in temperature, and longer warming periods, with the indication that lobsters in the Gulf of Maine will be at significant risk to this disease in less than 20 years (Maynard *et al.*, 2016).

With the widespread system-level responses to climate change, regime shifts in temperature patterns have occurred off southern New England, USA, including Long Island Sound as well as in the Gulf of Maine. These shifts have elicited changes in the phenology of molting and maturity of lobsters such that they are maturing at smaller sizes than previously reported (Landers *et al.*, 2002; Waller *et al.*, 2019). Regime shifts may have also contributed to the emergence of ESD in the lobster population in eastern Long Island Sound in the late 1990s (Castro & Angell, 2000). The establishment of ESD and its persistence and effects on the lobster population off southern New England are strongly correlated with the altered seasonal phenology due to climate change (Hoenig *et al.*, 2017; Groner *et al.*, 2018). Because ESD is associated with the host's shell, lobsters can effectively molt out of the disease, albeit the severity of the disease poses a significant risk to host survival (Hoenig *et al.*, 2017). Males and juveniles have less risk to ESD because they molt annually, i.e., the disease rarely progresses to a severe state on these animals (Fig. 6). In contrast, females have a two-year molt cycle, which makes them highly susceptible to an increased risk of severe infections and death. This has manifested as serious declines in recruitment to the lobster population in Long Island Sound

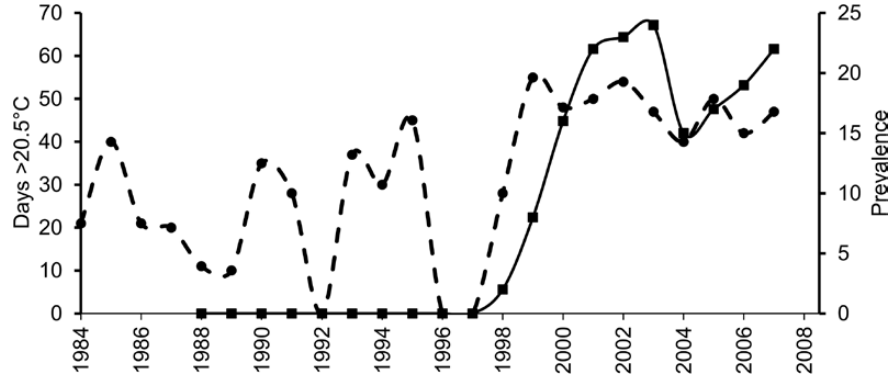

Figure 5. Temperature shift and prevalence (solid line) of epizootic shell disease in Long Island Sound, eastern USA associated with climate change. The number of days where the bottom temperature was > 20.5 °C (dashed line) indicates a significant regime change in summer temperatures in the region. Since 1987 there have been years where bottom temperatures have not reached 20.5 °C over long portions of the summer. Redrawn from Howell, 2012.

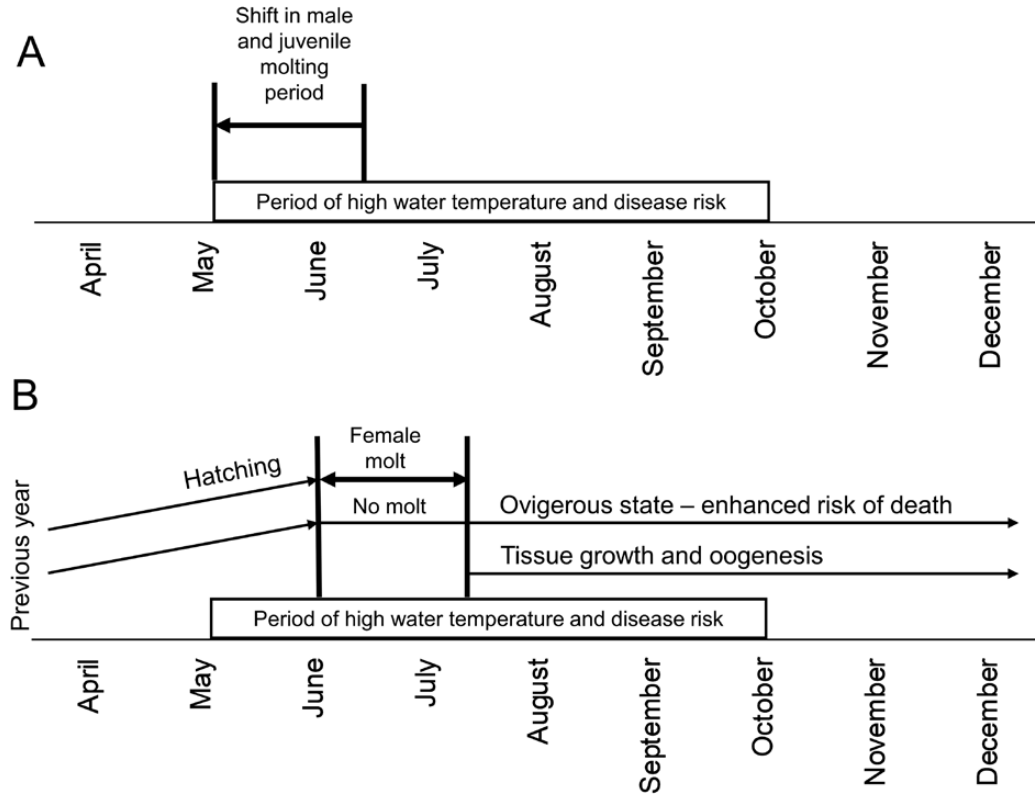

Figure 6. Phenological shift in molting activity of male and juvenile lobsters due to increased climatic warming leaves lobsters in Long Island Sound, eastern USA in higher temperatures with increased risk of developing epizootic shell disease (**A**). Mature females are at greater risk because they molt every two years, effectively placing them at higher temperatures over at least two annual cycles before molting out of what is typically a more advanced disease state (**B**).

(Wahle *et al.*, 2009, Howell, 2012). Furthermore, phenological shifts in molting due to climatic warming place new instars, which are susceptible to ESD, in warmer waters for a longer period (Fig. 6), effectively increasing the risk that they will develop more severe infections and death before molting again (Groner *et al.*, 2018). The combined effects of ESD on females, with their two-year molting cycle, and an altered molting phenology, has resulted in significant sex-specific mortality in this system and has contributed to a significant decline in the affected lobster population (Hoenig *et al.*, 2017; Groner *et al.*, 2018).

Temperature responses alter host-pathogen associations

Climatic warming has caused outbreaks of bitter crab disease (BCD) in the snow crab, *Chionoecetes opilio* (Fabricius, 1788), in the coastal bays of Newfoundland, Canada. Bitter crab disease is caused by a species of parasitic dinoflagellate, *Hematodinium* Chatton & Poisson, 1931, that lives in the hemocoel of its host. Infected crabs are inedible and bitter in flavor, hence represent lost value to the fishery. They also have a significantly high mortality rate compared to healthy crabs (Hoenig *et al.*, 2017). Annual monitoring of the biomass of snow crabs has been done for several decades and the parasite was not noticed there until the late 1980s. In the early 1990s the parasite was initially reported at very low prevalence levels in the coastal bays of Newfoundland (Taylor & Khan, 1995).

Beginning in 1997–1998 and again in 2002–2003 two epidemic outbreaks of BCD occurred in the coastal bays off Newfoundland, with the first centered in females and smaller juveniles, and the second centered in large males (Shields *et al.*, 2005, 2007). Snow crabs in the area normally live at temperatures ranging from –1.0 °C to 0.5 °C, but during the second outbreak water temperatures consistently increased above 0 °C. The resulting warming enhanced molting activity in larger snow crabs, which presumably made them more susceptible to infection by the parasite. In a typical cold year, the terminal molt to the long-claw morphotype would occur in approximately 20% to 30% of the males, whereas in the warmer years, the terminal molt occurred 60% to 85% in the male subpopulations (Fig. 7). Although the transmission of the pathogen in this system is not well understood, infections appear to have an annual cycle that aligns with host molting activity; 97% of the infections occur in new-shell crabs, i.e., crabs that molted that year (Shields *et al.*, 2005, 2007). In the large male crabs, prevalence levels jumped from < 1% in cold years to > 20% at the height of the temperature increase. From a logistic regression model, a 1 °C increase in water temperature was associated with a five-fold increase in disease prevalence in the large males in this system (Fig. 8). This relationship was not evident in cold years.

A single change in a host factor, molting activity, in relation to warming was evident in the snow crab-*Hematodinium* system. Although it may not fully explain the increase in transmission, this pattern in molting in relation to disease was revealed from analysis of fishery-independent monitoring data, which allowed for the examination of large-scale patterns in this system (Shields, 2017). The dynamics of BCD infection are quite complex and show differences in the transmission ecology between the coastal bays and open ocean areas off northern and eastern Newfoundland (Mullowney *et al.*, 2014). These differences in physiography likely limit the potential for more widespread damage due to this pathogen.

Changes in pathogen factors such as transmission, establishment, and proliferation have been observed in relation to increasing temperatures of a similar pathogen in the blue crab *Callinectes sapidus* Rathbun, 1896. Juvenile blue crabs in the high-salinity coastal bays of the mid-Atlantic region of the USA experience annual outbreaks of *Hematodinium perezi* Chatton & Poisson, 1931 during late summer and fall. Prevalence levels frequently reach 100% in juvenile crabs in the region (Messick, 1994; Messick & Shields, 2000; Small *et al.*, 2019). Temperature experiments with infected early-crab instars have revealed several important features of the host-parasite association. First, infections are stable at temperatures ranging from 10 °C –20 °C, but rapidly became fatal at temperatures of 4 °C and ≥ 25 °C (Fig. 9) (Huchin-Mian *et al.*, 2018). Surprisingly, infected crabs held at 25 °C produce dinospores, the presumptive infectious stage of the parasite and a stage rarely observed previously, and release them from crabs in mass sporulation events that are typically fatal. Mass sporulations did not occur at lower temperatures. Infections also appeared to retrograde in crabs surviving at 4 °C. Sentinel studies in the field have shown that hyper-endemic transmission occurs in late summer and early autumn, i.e., periods of warmest temperatures that coincide with juvenile recruitment (Shields *et al.*, 2017), and rapidly declines with increased storm activity and lower temperatures (Huchin-Mian *et al.*, 2017). Increasing water temperatures thus stimulate the development of the presumptive infectious stage of the parasite and enhance its transmission to highly susceptible juvenile hosts. Moreover, infected blue crabs die very quickly at 30 °C as do infected fiddler crabs, but the latter show

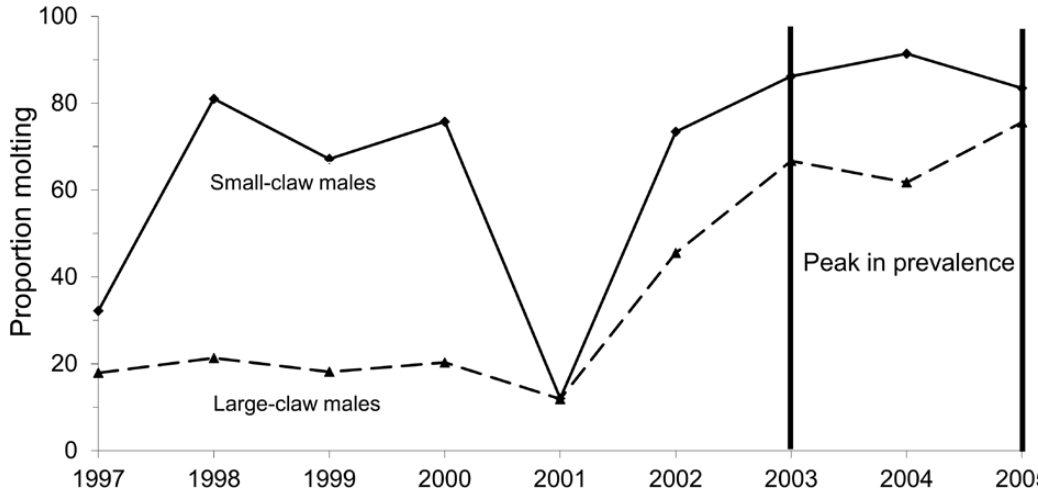

Figure 7. Proportion of male snow crabs caught in traps that had molted in Conception Bay, Newfoundland during two outbreaks of bitter crab disease. In the first outbreak the molting activity and prevalence was high in juvenile small-clawed males. In the second outbreak, warming temperatures caused an increase in molting activity in male crabs molting to the terminal large-claw status (dashed line) resulting in a higher prevalence in that subpopulation from 2003–2005. Redrawn from Shields *et al.*, 2007.

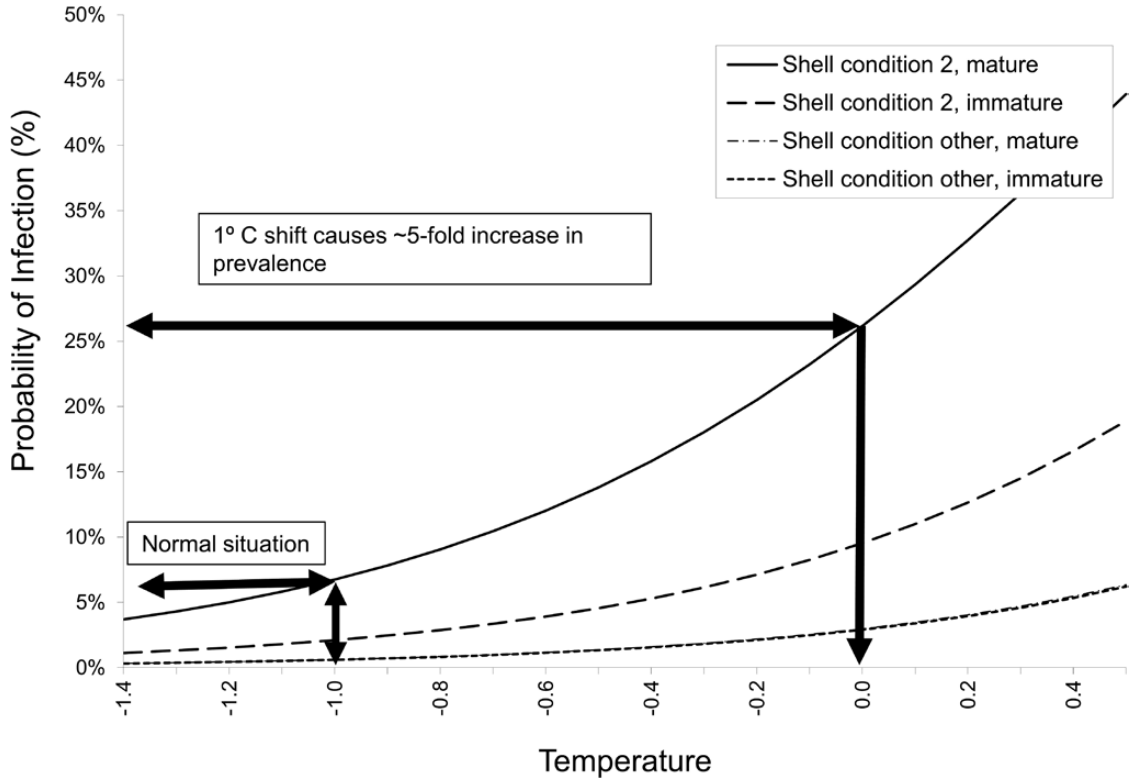

Figure 8. Probability of infection in relation to temperature for snow crabs in Conception Bay, Newfoundland during the 2003–2005 outbreak of *Hematodinium* sp. A 1 °C change in mean temperature was associated with a five-fold increase in prevalence in this system. Redrawn from Shields *et al.*, 2007.

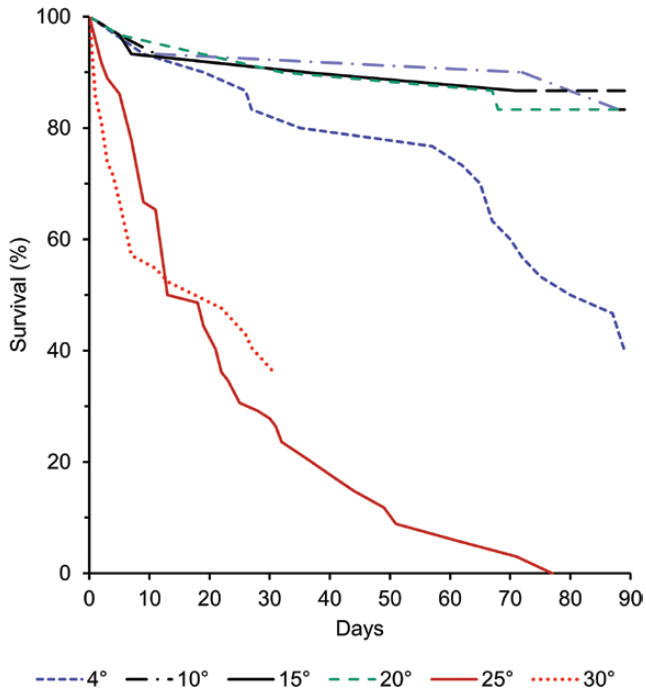

Figure 9. The effects of temperature on the survival of *Callinectes sapidus* naturally infected with *Hematodinium perezi*. Survival of uninfected controls is not shown. Mortality of infected crabs was greatest at 4 °C and 30 °C. Redrawn from Huchin-Mian *et al.*, 2018. This figure is available in color at *Journal of Crustacean Biology* online.

some resistance, with surviving crabs apparently losing their infections at this temperature (O'Leary, 2018), i.e., this is approaching the critical temperature for the parasite.

The blue crab-*H. perezi* system will likely exhibit more mortalities with climate change because the transmission and life cycle of the parasite occur very quickly at warmer temperatures and the infection dynamics are extended through mild winters. The parasite overwinters as active infections at temperatures over 6 °C–8 °C (Shields *et al.*, 2015b). Cold winters lower the prevalence through the mortality of infected crabs and possibly through retrograde loss of infections. The parasite thrives during mild winters, and can rapidly proliferate when temperatures increase to ≥ 15 °C. The warmer winter temperatures thus facilitate the survival of infected crabs and provide a reservoir of infection heading into the spring. This reservoir explains the rapid increases in prevalence observed in the spring (Small *et al.*, 2019).

Not all host-pathogen systems respond in the same way to temperature increases. The thermal ranges of the mud crab, *Eurypanopeus depressus* (Smith, 1869), and its rhizocephalan barnacle parasite, *Loxothylacus panopaei* (Gissler, 1884), indicate that the parasite has a lower range than that of its host (Gehman *et al.*, 2018). Notably, infected crabs have a reduced survival in relation to warming temperatures, particularly at temperatures above 15 °C (Fig. 2D). With increases in temperature, there is a predicted phenological mismatch between the survival of the host and parasite. The prevalence in the latter shifts from summer into cooler spring periods. More importantly, a model using the thermal range data predicted a decline in the prevalence of the parasite with 1 °C shifts in temperature that eventually lead to local extinctions of the barnacle. Although the model predicts localized extinctions, it was fit for only one host species, *E. depressus*, and the parasite is a host generalist that infects at least five other xanthid mud crab hosts with disparate overlapping distributions (Hines *et al.*, 1997); hence, the temperature relationship and possible extirpation may only be specific to one host species or it may be ameliorated by the presence of other host species. Further modeling of this parasite may prove very useful for predicting the effect of a pathogen on a multiple-host system.

Genetic underpinnings in host-pathogen responses to temperature

One of the best-known systems for studying the molecular aspects of disease in crustaceans is the host-pathogen relationship between two penaeid shrimps, *Penaeus vannamei* Boone, 1931 and *Penaeus monodon* Fabricius, 1798, and White Spot Syndrome Virus (WSSV). Both the shrimps and the virus have well known thermal ranges. There is little to no replication below 13 °C and above 32 °C for WSSV (Vidal *et al.*, 2001; Du *et al.*, 2006; Guan *et al.*, 2003; Granja *et al.*, 2006). Although low temperatures do not clear the virus from infected hosts (Korkut *et al.*, 2018a), temperatures at 32 °C and above can effectively clear shrimp of the virus. The nature of this sensitivity is based on at least two genes, ALDH, an aldehyde dehydrogenase gene, and hsp70, a heat-shock protein. Although the exact mechanism is unclear, Lin *et al.* (2011) designed an elegant experiment using dsRNA interference to knockdown four genes thought to be responsible for host susceptibility. Shrimp with knockdowns in ALDH and hsp70 genes were susceptible to WSSV infection at high temperatures, but control shrimp were not susceptible. Although the nature of this susceptibility is not clear (see discussion in Lin *et al.*, 2011), it does open the door to a more complete understanding of the effects of temperature in this system.

Host responses to different pathogens are known in crustaceans, but there are few unifying principles in which to place their responses into the scheme of thermal range. For example, interference RNA likely protects some crustaceans from the broad host generalist, WSSV, but few other viral pathogens have been as well studied. Although we know the thermal range of WSSV in shrimps, it is unclear how temperature increases will affect other infected hosts (e.g., lobsters; Clark *et al.*, 2013). Similarly, investigations into bacterial and fungal pathogens in crayfishes and other crustacean hosts have elucidated the phenoloxidase pathway in great detail, but there is more to be done with respect to defensins and other small anti-bacterial peptides and how these are affected by temperature. Investigations into protozoan pathogens have not received as much attention, but their infections are frequently not recognized by host immune defenses (Rowley *et al.*, 2015), which may lead to rampant infections as seen in *H. perezi* (Shields & Squyars, 2000; Huchin-Mian *et al.*, 2018). Investigations in the crayfish *Pacifastacus leniusculus* (Dana, 1852) with bacteria and WSSV confirm that cellular immunity is more effective at low temperatures in crayfishes (Korkut *et al.*, 2018a, b), and similar findings have been reported for amphipods (Labaude *et al.*, 2017), but with the few exceptions outlined above, we really have a cursory mechanistic understanding of how climate change will affect host immunity and susceptibility to pathogens. Pathogens (e.g., ESD, viral pathogens in shrimps) are clearly emerging in these systems, mostly likely due to complex environmental stressors associated with climate change; hence, more work is urgently needed in this area.

OCEAN ACIDIFICATION (OA)

Although rising temperature is the dominant stressor in climate change, particularly with respect to metabolism and growth in ectotherms, ocean acidification (OA) is also occurring at rapid rates and it may also affect the host-pathogen relationship. Ocean acidification impairs calcification by increasing the energetic cost of precipitating calcium carbonate in skeletons. The exoskeleton of lobsters contains as much as 17%–23% calcium carbonate (Boßelmann *et al.*, 2007). OA is known to influence the immune response and disease status of bivalves (Mackenzie *et al.*, 2014) and corals (Hoegh-Guldberg *et al.*, 2007). Few studies, however, have examined how OA operates as a stressor to alter disease processes in crustaceans. Crustaceans appear to have more variable responses to OA than molluscs, because they store bicarbonate in their cuticle and use it to buffer their hemolymph, giving them a large physiological pool of calcium bicarbonate (Qadri *et al.*, 2007; Spicer *et al.*, 2007; Whiteley, 2011). Hence, most negative effects have been noted for crustaceans at relatively strong acidification levels (i.e., pH 7.5), less so at modest pH levels (i.e., pH 7.81). Nonetheless, negative effects appear to affect the host in much the same way that increasing temperatures do (Hernroth *et al.*, 2018). For example, early benthic American lobsters are more susceptible to shell disease after exposure to OA conditions (McLean *et al.*, 2018). The Norway lobster *Nephrops norvegicus* (Linnaeus, 1758) shows significant immune suppression and protein damage when subjected separately to hypoxia and OA conditions (Hernroth *et al.*, 2012, 2015). The survival of the amphipod *Paracalliope novizealandiae* Ruffo & Paiotta, 1972 was not affected by OA, but transmission of one of its trematode parasites was significantly reduced, with possible effects to the host-parasite dynamics (Harland *et al.*, 2015). Metabolic and physiological effects underpin these results but they remain largely unexplored. The amplitude of effects in crustaceans will depend on duration of exposure, acclimation, differences in susceptibility between life history stages, and negative consequences to vital physiological processes.

SALINITY CHANGE

Temperature and ocean acidification have received the most attention with respect to climate change, but salinity is also changing. Rising sea levels shift salinity distributions in estuaries as seawater intrudes farther into freshwater reaches, but the outcomes are difficult to predict. For example, the salinity of Chesapeake Bay, one of the largest estuaries in the USA, has increased over time and is projected to continue increasing, but current models indicate a highly variable response due in part to predicted variability in seasonal rainfall and sea level rise (Hilton *et al.*, 2008; Najjar *et al.*, 2010). Moreover, salinity is expected to increase broadly over parts of the world's oceans in response to increased evaporation but the increase will be highly variable due to redistribution via precipitation and changing weather patterns (Curry *et al.*, 2003; Stott *et al.*, 2008). Although there are many exceptions, most marine invertebrates, including decapod crustaceans, are osmo-conformers, meaning that their internal osmolality, or osmotic pressure, is the same as seawater. One exception is the blue crab, which is an osmo-regulator at low (< 24 psu) and high (> 40 psu) salinities, keeping its hemolymph osmolality near the range of seawater (30–36 psu), but it is an osmo-conformer between those salinities (Lynch *et al.*, 1973). It is more susceptible to disease when subject to stressors that impose a metabolic cost (Brill *et al.*, 2015), such as hypoxia or osmoregulation, but more work is needed in the area of disease susceptibility with respect to salinity.

Changes in osmoregulation require metabolic energy; hence, the model of environmental stressors affecting the host-pathogen relationship should apply to changes in salinity, particularly for animals with narrow salinity requirements. Most studies on salinity and the host-pathogen relationship have focused on assessing immune functions in shrimps and crabs. For example, crabs and shrimps subject to low-salinity stress show alterations to several immunological factors: phagocytosis, phenoloxidase activity, total hemocyte densities, and several proteins (Ge *et al.*, 2017; Wang *et al.*, 2018). These constituents all responded with short-term changes, followed by return to normal levels after approximately 72 h. In essence, this represents an acclimation phase to a short-term stressor. The effect of long-term salinity stress is not apparent.

Conversely, pathogens may be subject to more lasting effects from salinity changes. Dinospores of the parasite *H. perezi*, for example, show an exponential decline in viability in relation to lower salinities (Coffey *et al.*, 2012). This response explains why the parasite has very low prevalence levels at salinities less than 20 psu in mid-Atlantic estuaries of the USA. Infected crabs can move into low salinity waters, but dinospores released into these waters have little chance of transmission to another host. Lower salinity

(≤ 15 psu) does not, however, affect the mortality rate of crabs infected with the parasite (Huchin-Mian *et al.*, 2018). Thus, the higher salinities expected with salinity intrusion into Chesapeake Bay may allow the parasite to spread more broadly into the bay. A similar phenomenon has been observed in oysters infected with *Perkinsus marinus* (Mackin, H.M.Owen & Collier, 1950) in the region (Burreson & Ragone, 1996). Unlike temperature, where there is a clear association with stress and enhancement of host susceptibility, salinity effects are likely to be different between host-pathogen systems. WSSV infections in the Chinese shrimp *Penaeus chinensis* (Osbeck, 1765), for example, have lower viral replication and infected hosts show better survival at 20 psu compared to higher salinities (Xue *et al.*, 2017). Infections in *Penaeus vannamei*, however, show increased mortality with abrupt exposure to lower salinities (to 5 psu) as might occur in a torrential rainfall (Van Thuong *et al.*, 2016) as well as high salinities that might occur with drought (Ramos-Carreño *et al.*, 2014). Thus, responses to salinity may be specific to the host-pathogen relationship.

CONCLUSIONS

Climate change is the greatest anthropogenic stressor of our time. It effectively alters the tripartite (host, pathogen, environment) framework of disease ecology, essentially becoming a major feature of the fourth component of that framework, anthropogenic effects (Shields, 2013). Climate change is a complex issue because it consists of multiple, interacting drivers that impose varying, sometimes conflicting, changes to both the host and the pathogen. Research in response to it is still in its infancy as demonstrated by the fact that most studies investigate the effects of single factors on disease, particularly temperature, as the sole stressor. Nonetheless, climate change modulates temperature, acidification, and salinity through multiple pathways which then affect host factors by changing host susceptibility, on pathogen factors by enhancing growth rates, and on environmental factors through broad alterations to ocean chemistry, facilitation of nutrient-driven hypoxia, and phenology. Placed into the context of a broad, system-level stressor, climate change, with its increasing environmental stress projected over the next century, should result in many more emergent pathogens affecting crustacean hosts. Thus, there is a need to more fully understand the effects of climate change on host-pathogen associations in commercially important hosts, because they supply important food sources to human populations. There is also a need to understand the effects of climate change on ecologically important copepods, amphipods, and krill, because these crustaceans support many marine ecosystems through the sheer size of their productive biomass.

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