Effects of Infectious Diseases on Population Dynamics of Marine Organisms in Chesapeake Bay

Jerelle A. Jesse1 · M. Victoria Agnew2 · Kohma Arai1 · C. Taylor Armstrong3 · Shannon M. Hood4 · Mariah L. Kachmar2 · Jessie T. Long4 · Alexandra J. McCarty4 · Morgan O. Ross4 · Kasondra D. Rubalcava5 · Jacob Shaner6 · Sakura Tanaka2 · Lindsay Wood7 · Eric J. Schott3 · Michael J. Wilberg1

Received: 6 March 2020 / Revised: 3 February 2021 / Accepted: 6 February 2021 / Published online: 24 March 2021
© The Author(s) 2021

Abstract
Diseases are important drivers of population and ecosystem dynamics. This review synthesizes the effects of infectious diseases on the population dynamics of nine species of marine organisms in the Chesapeake Bay. Diseases generally caused increases in mortality and decreases in growth and reproduction. Effects of diseases on eastern oyster (Crassostrea virginica) appear to be low in the 2000s compared to effects in the 1980s–1990s. However, the effects of disease were not well monitored for most of the diseases in marine organisms of the Chesapeake Bay, and few studies considered effects on growth and reproduction. Climate change and other anthropogenic effects are expected to alter host-pathogen dynamics, with diseases of some species expected to worsen under predicted future conditions (e.g., increased temperature). Additional study of disease prevalence, drivers of disease, and effects on population dynamics could improve fisheries management and forecasting of climate change effects on marine organisms in the Chesapeake Bay.

Keywords Host pathogen · Mycobacteriosis · Eastern oyster · Blue crab · Eelgrass · Wasting disease

Introduction

Infectious diseases can have substantial effects on population dynamics and communities of marine organisms (Andrews 1979; Muehlstein et al. 1991; Groner et al. 2018b). For this paper, infectious disease is defined as a disease caused by bacterial, fungal, viral, or parasitic infections that can be transmitted from one individual to another (simply referred to as diseases hereafter). Diseases can affect population dynamics by altering mortality, growth, and reproduction (Fig. 1). The most studied effect of disease is increased mortality, which causes decreased abundance of a population. Disease can have negative effects on growth and reproduction. For example, a population with a sustained low disease prevalence and severity should experience low population-level mortality rates, high growth, and reproduction sufficient to replace the individuals lost from mortality (Fig. 1a). Alternatively, high levels of a lethal disease (Fig. 1b) can increase mortality rates, decrease growth, and decrease reproduction resulting in a smaller population size than the low disease case. These population dynamic effects can interact with each other and can be affected by other stressors. Thus, the effects of disease on a population are complicated and likely change over time.

Diseases can have important effects on fisheries. For example, MSX, caused by the protozoan parasite Haplosporidium
nelsoni, resulted in the collapse of the fishery for eastern oyster (*Crassostrea virginica*) in Delaware Bay during the 1950s (Ford and Bushek 2012). The Chesapeake Bay is important for commercial and recreational fisheries with the total dockside commercial revenue of key species at just under $300 million in 2016 (National Marine Fisheries Service 2018). The Chesapeake Bay also serves as spawning and nursery habitat for many species in the western Atlantic Ocean. Therefore, it is essential to understand the effects of diseases that affect the population dynamics of marine species in the Chesapeake Bay.

The objective of this review is to describe the effects of disease on the population dynamics of nine species of marine organisms in Chesapeake Bay (Table 1). The organisms included in this review are some of the most ecologically and economically important species in the Bay. The review highlights the gaps in our knowledge about how diseases affect populations and is organized by disease effects on population dynamics processes (mortality, growth, and reproduction). Furthermore, we discuss how external factors, such as climate change and aquaculture, may alter the effects of diseases on these processes. Information on transmission of diseases was not included because there are knowledge gaps regarding transmission for most of the included species.

### Population Dynamics

#### Mortality

Mortality is the most well-studied effect of diseases of Chesapeake Bay marine organisms. Disease-driven mortality affects species throughout Chesapeake Bay, from the benthic environment to the pelagic zone (Table 1). While some diseases affect multiple species (Stine et al. 2010), many only affect a single species or family of species within Chesapeake Bay (e.g., Muehlstein et al. 1991; Ragone Calvo et al. 2003b). Although well-researched disease-driven mortalities are documented for many economically important species (Ragone Calvo et al. 2003b; Hoenig et al. 2017), disease may affect other species that are not commonly monitored. In addition, mortality is more easily observed in some species than others (e.g., fish kills or gaping oysters; Ford et al. 2006; Gauthier et al. 2008). Therefore, it is likely that some diseases (or perhaps many) have not yet been prioritized or studied. Prevalence of a disease and individual-level mortality are necessary to understand the population level impacts of disease, but often disease dynamics are studied at small spatial or temporal scales which inhibits the understanding of full population effects (Table 2). In the Chesapeake Bay, striped bass and oyster diseases are well studied at the population level, but the effects of disease on the population are not well studied for other species.

Striped bass (*Morone saxatilis*) in the Chesapeake Bay are affected by mycobacteriosis, a chronic, progressive disease which results in internal granulomatous inflammation and external ulcerative lesions (Jacobs et al. 2009). Mycobacteriosis was first detected in the population in 1997 and correlates with an increase in natural mortality in the Chesapeake Bay according to tagging estimates (Northeast Fisheries Science Center 2019). Prevalence of mycobacteriosis can vary among years and locations and can exceed 60% (Cardinal 2001; Gauthier et al. 2008; Hoenig et al. 2017; Groner et al. 2018b). The mortality of striped bass increases with increasing disease severity, with moderately and severely diseased individuals having 16% and 46% year$^{-1}$ higher mortality relative to healthy fish (Hoenig et al. 2017). Mortality among severely diseased striped bass is especially high, with average mortality from 56 to 87% year$^{-1}$ (Groner et al. 2018b). At the population-level,
Table 1  Summary of organisms, diseases and pathogens, and the population dynamics processes affected by disease in the Chesapeake Bay. Note some diseases do not have separate names from the pathogen. For these diseases, only the pathogen is listed.

| Organism                  | Disease (pathogen)                                                                 | Population processes affected                        | Citation                                                                                      |
|---------------------------|------------------------------------------------------------------------------------|------------------------------------------------------|-----------------------------------------------------------------------------------------------|
| Eastern oyster (Crassostrea virginica) | Dermo (Perkinsus marinus), MSX (Haplosporidium nelsoni), SSO (Haplosporidium costale) | Mortality, growth, reproduction                      | Paynter and Burreson 1991, Ben-Horin et al. 2018, Barber et al. 1988, Andrews 1979, Andrews and Castagna 1978, Ragone Calvo and Burreson 2000, Carnegie et al. 2004, Schulte 2017, Keppel et al. 2015 |
| Soft-shell clam (Mya arenaria) | Disseminated neoplasia, Perkinsus sp.                                              | Mortality, growth, recruitment                       | Farley et al. 1991, Farley et al. 1986, Glaupie et al. 2018                                  |
| Razor clam (Tagetes plebeius)   | Quahog parasite unknown                                                            | Mortality                                             | Glaupie et al. 2018, Farley et al. 1986                                                      |
| Hard clam (Mercenaria mercenaria) | Perkinsus sp., neoplasms                                                          | Growth                                                | Whyte et al. 1994, Smolowitz 2018, Ragone Calvo et al. 1998, Ragone Calvo et al. 2007, Ford et al. 2002, Christensen et al. 1974 |
| Small Saltwater Clam (Mactona balitica) | Perkinsus sp., neoplasms                                                          | Mortality, growth, recruitment                       | Small et al. 2019, Shields and Squyars 2000, Messick and Shields 2000, Messick 1994, Johnson 1977, Bowers et al. 2010, Messick and Kennedy 1990, Flowers et al. 2016, 2018, Spitznagel 2019, Newman and Ward 1973, Sprague 1970, Morado 2011 |
| Blue crab (Callinectes sapidus) | Bitter crab disease (Hematodinium perezi), Reovirus CsRV1, Vibrio sp., Paramoeba perniciosa, Lagenidium callinectes, Picorna-like virus | Mortality, growth, recruitment                       | Small et al. 2019, Shields and Squyars 2000, Messick and Shields 2000, Messick 1994, Johnson 1977, Bowers et al. 2010, Messick and Kennedy 1990, Flowers et al. 2016, 2018, Spitznagel 2019, Newman and Ward 1973, Sprague 1970, Morado 2011 |
| Striped bass (Morone saxatilis) | Mycobacteriosis                                                                     | Mortality, growth, recruitment                       | Gauthier et al. 2008, Latour et al. 2012, Hoenig et al. 2017, Groner et al. 2018, Matsche et al. 2010, Lapointe et al. 2014, Gervasi et al. 2019, Gervasi 2015 |
| Atlantic Menhaden (Brevoortia tyrannus) | Aphanomyces invadans, Kudoa sp., Mycobacteriosis                                   | Mortality, recruitment                               | Kiyu et al. 2003, Blazer et al. 1999, Webb et al. 2005, Kane et al. 2007, Stine et al. 2010 |
| Atlantic sturgeon (Acipenser oxyrinchus) | Nitzschiasturioni                              | Growth                                                | Matsche et al. 2010                                                                       |
| American eel (Anguilla rostrata) | Anguillicolides crassus                                                             | Growth, recruitment                                  | Zimmerman and Welsh 2012, Haro et al. 2000, Kirk 2003, Kaldy 2014, Brkal et al. 2019, Sullivan et al. 2018, Arnold et al. 2012, Arnold et al. 2017, Muchlstein et al. 1988, Muchlstein et al. 1991, Short et al. 1987 |
| Eelgrass (Zostera marina) | wasting disease (Labyrinthula zosterae)                                             | Mortality                                             | Kaldy 2014, Brkal et al. 2019, Sullivan et al. 2018, Arnold et al. 2012, Arnold et al. 2017, Muchlstein et al. 1988, Muchlstein et al. 1991, Short et al. 1987 |
## Table 2  
Studies included in review by population dynamic effect including information on type of study and location of study

| Citation                  | Study type | Study location                          |
|---------------------------|------------|-----------------------------------------|
| **Mortality**             |            |                                         |
| Hoenig et al. 2017        | Field      | Chesapeake Bay (MD and VA)              |
| Cardinal 2001             | Field      | Chesapeake Bay (VA)                     |
| Gauthier et al. 2008      | Field      | Chesapeake Bay (MD and VA)              |
| Groner et al. 2018b       | Field      | Chesapeake Bay (VA)                     |
| Blazer et al. 1999        | Field      | Chesapeake Bay (MD and VA)              |
| Kiryu et al. 2003         | Experiments| Chesapeake Bay (MD and VA)              |
| Webb et al. 2005          | Field      | Chesapeake Bay (VA)                     |
| Andrews et al. 1962       | Field      | Chesapeake Bay and coastal bays (VA)    |
| Andrews and Castagna 1978 | Field      | Chesapeake Bay to Delaware Bay          |
| Ragone Calvo et al. 2003b | Field      | Chesapeake Bay (VA)                     |
| Southworth et al. 2010    | Field      | Chesapeake Bay (VA)                     |
| Tarnowski 2019            | Field      | Chesapeake Bay (MD)                     |
| Ragone Calvo and Burreson | Field      | Chesapeake Bay (VA)                     |
| Carnegie et al. 2004      | Field      | Chesapeake Bay (VA)                     |
| Ford and Bushek 2012      | Field      | Delaware Bay                            |
| McCollough et al. 2007    | Field      | Chesapeake Bay (MD)                     |
| Paynter and Burreson 1991 | Field      | Chesapeake Bay (MD and VA)              |
| Reece et al. 2008         | Field      | Chesapeake Bay (MD and VA)              |
| Bushek and Ford 2016      | Field      | Delaware Bay                            |
| Dungan et al. 2002        | Field      | Chesapeake Bay (MD)                     |
| Farley et al. 1991        | Field      | Chesapeake Bay (MD)                     |
| Farley et al. 1986        | Field      | Chesapeake Bay (MD)                     |
| Johnson 1977              | Field and experiments | Chincoteague Bay, VA            |
| Newman and Ward 1973      | Field      | Chincoteague Bay, VA                    |
| Messick and Shields 2000  | Field      | Chesapeake Bay and coastal bays (MD and VA) |
| Shields and Squyars 2000  | Experiments| Chesapeake Bay (MD and VA)              |
| Bowers et al. 2010        | Field      | Chesapeake Bay (MD)                     |
| Flowers et al. 2018       | Field      | Chesapeake Bay (MD)                     |
| Flowers et al. 2016b      | Field      | Chesapeake Bay (MD) and east coast USA  |
| Zhao et al. 2020          | Field      | Western Atlantic (Massachusetts to Uruguay) |
| Short et al. 1987         | Field      | Atlantic and Pacific coasts USA         |
| Muehlstein et al. 1988    | Experiments| North American coasts, including MD and VA |
| Muehlstein et al. 1991    | Field      | North American coasts                   |
| **Growth**                |            |                                         |
| Latour et al. 2012        | Field      | Chesapeake Bay (MD and VA)              |
| Matsche et al. 2010       | Experiments| Chesapeake Bay (MD)                     |
| Zimmerman and Welsh 2012  | Field      | Shenandoah River, WV (upstream of Chesapeake Bay) |
| **Reproduction**          |            |                                         |
| Gervasi et al. 2019       | Field      | Chesapeake Bay (MD and VA)              |
| Barber et al. 1988        | Field      | Delaware Bay                            |
| Palstra et al. 2007       | Experiments| Hungary                                 |
| Kane et al. 2007          | Field      | Chesapeake Bay (MD)                     |
| Stine et al. 2005         | Field      | Chesapeake Bay (MD)                     |
| Small et al. 2019         | Field      | Chesapeake Bay and coastal bays (VA)    |
| Messick 1994              | Field      | Coastal Bays (MD and VA)                |
mycobacteriosis causes an approximate doubling of the natural mortality rate (Hoenig et al. 2017).

Atlantic menhaden (*Brevoortia tyrannus*) exhibit ulcerative skin lesions from multiple sources that may be connected with fish kills (Webb et al. 2005). The oomycete, *Aphanomyces invadans*, is thought to cause deep lesions and result in large-scale fish kills around the world. Atlantic menhaden from the Chesapeake Bay have been found with lesions similar to those caused by *A. invadans*, and preliminary testing found that the pathogen cultured from the lesions was an *Aphanomyces* spp. similar to *A. invadans* (Blazer et al. 1999). Atlantic menhaden showed increased mortality with increased injection dose of *A. invadans*, and fish treated with a realistic aqueous exposure developed 14% lesion prevalence and 11% mortality (Kiryu et al. 2003). Large mortality events for Atlantic menhaden have also been connected to other diseases. Atlantic menhaden with pre-ulcerative lesions tested positive for *Kudoa* sp. infections (a myxozoan parasite), while only fish with fully ulcerative lesions tested positive for other bacterial, fungal, and amphizoic amoebae. This suggests that

### Table 2 (continued)

| Citation             | Study type       | Study location                      |
|----------------------|------------------|-------------------------------------|
| Fenske et al. 2010   | Field            | Chesapeake Bay (MD)                 |
| Mann et al. 2009     | Field            | Chesapeake Bay (VA)                 |
| Gervasi 2015         | Model simulation | Chesapeake Bay (MD and VA)          |
| Lapointe et al. 2014 | Experiments      | Chesapeake Bay (VA)                 |
| Glaspie et al. 2018  | Field            | Chesapeake Bay (MD and VA)          |
| Hofmann et al. 2001  | Model simulation | Chesapeake Bay (MD and VA)          |
| Audemard et al. 2006 | Field            | Chesapeake Bay (VA)                 |
| Soniat et al. 2009   | Field            | Delaware Bay and Gulf of Mexico     |
| Gieseke 2001         | Field            | Chesapeake Bay (MD)                 |
| Christensen et al. 1974 | Field and experiments | Chesapeake Bay (MD) |
| Perrigault et al. 2011 | Experiments    | East coast USA                      |
| Kaldy 2014           | Experiments      | Pacific Northwest USA               |
| Brakel et al. 2019   | Experiments      | Baltic Sea                          |
| La Peyre et al. 2006 | Experiments      | Louisiana                           |
| Arnold et al. 2012   | Field            | Italy and Chesapeake Bay (MD)       |
| Groner et al. 2018a  | Experiments      | Washington                          |
| Ben-Horin et al. 2018| Model simulation | Chesapeake Bay (MD and VA) and Louisiana |
| Spitznagel 2019      | Field            | Chesapeake Bay (MD and VA)          |
| Messick and Kennedy 1990 | Experiment     | Chesapeake Bay (MD)                 |
| Sizemore et al. 1975 | Field            | Chesapeake Bay                      |
| Whyte et al. 1994    | Field            | Canada                              |
| Ragone Calvo et al. 1998 | Field      | Chesapeake Bay and coastal bays (VA) |
| Ford et al. 2002     | Field            | New Jersey                          |
| Ragone Calvo et al. 2007 | Field      | Chesapeake Bay (VA) and New Jersey  |
| Rothchild et al. 1994 | Stock assessment model | Chesapeake Bay (MD)                 |
| Wilberg et al. 2011  | Stock assessment model | Chesapeake Bay (MD)                 |
| Reed and Hovel 2006  | Field            | California                          |
| Lefcheck et al. 2017 | Field            | Chesapeake Bay                      |
| Kellogg et al. 2013  | Field            | Chesapeake Bay (MD)                 |
| Newell and Koch 2004 | Model simulation | Chesapeake Bay                      |
the causative agent for the lesions, and potentially the fish mortality events, was *Kudoa* sp. infection while the others were opportunistic secondary infections that took hold after the ulcer had formed (Webb et al. 2005).

Eastern oyster (*Crassostrea virginica*) populations in the Chesapeake Bay have been reduced by a combination of factors, including overfishing and infectious diseases (Rothschild et al. 1994). The most notable diseases are caused by the protozoan parasites *Haplosporidium costale* (SSO), *Haplosporidium nelsoni* (MSX), and *Perkinsus marinus* (Dermo). SSO disease has affected eastern oysters in high salinity areas of the Chesapeake Bay since 1959 (Andrews et al. 1962) causing mortality rates of 20−50% year\(^{-1}\) in the 1970s (Andrews and Castagna 1978) and is commonly found in association with MSX disease in the summer months (Andrews 1979). However, because SSO infections require high salinities (>30), they do not affect eastern oysters as severely as do MSX and Dermo disease. The combination of Dermo and MSX disease on eastern oysters in the Bay has caused high mortality since at least the 1960s (Andrews 1979; Ragone Calvo et al. 2003b; Southworth et al. 2010).

MSX disease was introduced into Delaware Bay in the 1950s before making its way to the Chesapeake Bay in 1959 (Andrews 1964). There have been four major epizootics of MSX disease since 1990 in association with increased salinities, three of which (1991−1992, 1995, 1999−2000) were associated with increases in mortality (Tarnowski 2019). All epizootics were arrested by increased freshwater inputs in following years, indicating that decreases in prevalence or severity is likely temporary and that a background reservoir of infection agents persist (Carnegie et al. 2004; Tarnowski 2005). In recent years, MSX-related eastern oyster mortality has declined, even when MSX prevalence is high (Tarnowski 2019), suggesting that population-level resistance may be developing in the Chesapeake Bay, as it did in Delaware Bay (Ford and Bushek 2012).

Dermo disease has likely led to oyster mortalities since the 1940s (Andrews 1979), causing mortality rates of up to 90% for adults (McCollough et al. 2007; Schulte 2017); Dermo remains prevalent throughout the Bay (Reece et al. 2008). In 2018, Maryland waters of the Chesapeake Bay had 40% mean infection prevalence, dropping from 69% in 2017, and below the 29-year average of 67% (Tarnowski 2019). However, similar to MSX, Dermo disease varies in prevalence spatially and over time throughout the Chesapeake Bay (Ragone Calvo and Burreson 2000; Maryland Department of Natural Resources 2001; Carnegie et al. 2004; Tarnowski 2005). Although low salinity may mitigate the negative effects of Dermo disease, it does not eradicate the pathogen (Paynter and Burreson 1991), indicating that future periods of high salinity may lead to further epizootics. Over the decades, there is evidence that natural populations of oysters become more tolerant of MSX and Dermo, presumably through natural selection (Bushek and Ford 2016).

Two parasites closely related to *P. marinus* (*P. andrewsi* and *P. chesapeaki*) and disseminated neoplasia (also referred to as sarcoma) cause infections and are associated with high mortality rates in soft clams (*Mya arenaria* and *Macoma balthica*) and razor clam (*Tagelus plebeius*) populations in Chesapeake Bay (Dungan et al. 2002; Reece et al. 2008). Almost 90% of *M. arenaria* clams infected with *Perkinsus* sp. were also infected with disseminated neoplasia, a transmissible leukemia-like disease of bivalves (Dungan et al. 2002). However, *T. plebeius* with *Perkinsus* sp. infections did not have evidence of lesions from disseminated neoplasia (Farley et al. 1986). Farley et al. (1986) also documented less than 0.1% prevalence of disseminated neoplasia in *M. arenaria* between 1969 and 1983, followed by 54% prevalence in 1984. Subsequent studies show that disseminated neoplasia persists in the Bay, with peak prevalence between 33 and 57% (Farley et al. 1991; Dungan et al. 2002).

Several pathogens may cause mortality in blue crabs (*Callinectes sapidus*) within the Chesapeake Bay. *Paramoeba perniciosa* is a parasitic amoeba that affects blue crabs in Chesapeake Bay and the Southeastern US Atlantic coast. *P. perniciosa* is thought to be limited to high salinity areas (Sprague 1970), although the low salinity threshold has not been defined (Morado 2011). Prevalence of *P. perniciosa* in Chesapeake Bay is unknown, but infections were detected in nearly 60% of blue crabs in nearby Chincoteague Bay (Johnson 1977). Heavily infected blue crabs (containing 95 *P. perniciosa* amoeba/100 hemocytes) experienced 100% mortality (Newman and Ward 1973), and *P. perniciosa* infection may lead to significant winter mortality among the blue crab population (Morado 2011). *Hematodinium perezi* is a parasitic dinoflagellate that causes blue crab mortality in the lower Chesapeake Bay. Mortality of infected blue crabs ranges from 86 to 100% (Messick and Shields 2000; Shields and Squyars 2000). Infection is linked to salinity, with no infected blue crabs found in low salinity waters (salinity < 11), limited infections in salinities of 11−18, and highest prevalence (38%) found in salinities of 26−30 (Messick and Shields 2000). There are recent reports on *H. perezi* alternate hosts, infections in early juveniles, and effects of climate change (Lohan et al. 2012; Huchin-Mian et al. 2018; Shields 2019).

Several viruses have been described in blue crabs in Maryland, including reo-like, picorna-like, rhabdo-like, and baculoviruses (Johnson 1983). The most well studied is the reo-like virus since re-named *C. sapidus* reovirus 1 (CsRV1). It is associated with mortality of blue crabs in soft-shell production (Bowers et al. 2010), and prevalence may be increased in wild crabs in close proximity to soft crab shedding facilities and in larger animals (Flowers et al. 2018). Originally described in 1977 (Johnson...
Diseases can negatively affect reproduction and recruitment by causing changes in maturation, fecundity, spawning behavior, and juvenile survival. At the population level, disease may reduce the effectiveness of spawning individuals or may lower recruitment if infections disproportionately affect the earliest life stages. Lower individual fecundity could reduce the productivity of populations in the Chesapeake Bay. Disease effects on reproductive processes have not been comprehensively studied for marine organisms in the Chesapeake Bay. However, the negative effects of disease, including the combination of decreased growth and increased mortality, can reduce reproductive success indirectly.

Maturation and fecundity can be negatively affected by disease. Striped bass with mycobacteriosis mature earlier and at smaller sizes than healthy fish (2.65 years and 322 mm for diseased, 2.94 years and 343 mm for healthy; Gervasi et al. 2019). No other reproductive metrics (oocyte development, oocyte size, fecundity, gonadosomatic index, or oocyte dry weight) were affected in diseased fish (Gervasi et al. 2019). In C. virginica, systemic infection by MSX disease decreased oyster fecundity by 81% (Barber et al. 1988). Decreased fecundity was a result of reduced food intake caused by infection and consequent use of metabolic reserves that would otherwise have been used for gamete production (Barber et al. 1988).

The ability to undertake spawning migrations may be affected by disease. A. crassus causes swim bladder pathology in American eels, including inflammation, lesions, hemorrhaging, and fibrosis (Lefebvre et al. 2011). This swim bladder degradation is thought to reduce swimming efficiency and migratory ability of infected individuals, and migrating silver stage eels with swim bladder impairment may be unable to reach their spawning grounds in the Sargasso Sea (Haro et al. 2000; Palstra et al. 2007).

Recruitment could be negatively affected by disease if it causes increased mortality in juveniles. Atlantic menhaden in the Chesapeake Bay experience mycobacteriosis, but it is unknown if the disease is fatal. Young-of-the-year Atlantic menhaden from Maryland tributaries in the Bay had 2–100% prevalence of mycobacteriosis, thought to be dependent on the water quality in the tributary (Stine et al. 2005; Kane et al. 2007). H. perezi may affect blue crab recruitment by increasing mortality of the early life stages. Fifty to 100% of early benthic juveniles were infected with H. perezi (Small et al. 2019). Higher prevalence in small crabs than in large crabs may indicate mortality of infected small crabs (i.e., the infected crabs may die at a higher rate than the non-infected crabs resulting in a lower prevalence at later ages; Messick 1994).

Reproduction and early life stages

Diseases can negatively affect reproduction and recruitment by causing changes in maturation, fecundity, spawning

Growth

Diseases can affect growth, but less is known about the effects of disease on growth than on mortality. In the Chesapeake Bay, few studies have directly evaluated the effects of disease on host growth, but those that do indicate reduced growth in diseased marine organisms. A study of striped bass collected from the Chesapeake Bay found that striped bass with mycobacteriosis had significantly slower growth than disease-negative fish (Latour et al. 2012). Furthermore, Atlantic sturgeon (Acipenser oxyrinchus) with skin and gill infections of the trematode, Nitzschia sturionis, lost weight compared to uninfected Atlantic sturgeon (Matsche et al. 2010). Similarly, American eel (Anguilla rostrata) infected with the swim bladder nematode, Anguillicoloides crassus, had a lower length-at-age than uninfected eels (Zimmerman and Welsh 2012). Dermo disease in the eastern oyster can slow growth and cause a reduction in condition index of the oysters (Paynter and Burreson 1991). Multiple mechanisms, including changes in behavior or physiological responses, could explain the reduced growth of infected individuals. To further complicate matters, the reduction in growth may be difficult to distinguish from size selective mortality (large individuals have a higher mortality rate than smaller individuals).

Interactions among mortality, growth, and reproduction

The effects of diseases are sometimes not easily separated into mortality, growth, and reproduction because diseases can affect multiple interacting population dynamics processes.
Decreased growth can lead to decreased reproductive potential because fecundity is positively related to size for most marine organisms (Hixon et al. 2014). Similarly, disease-induced mortality can cause a reduction in the number of large individuals in a population. Because larger individuals are typically the most fecund in the population, increases in mortality or decreases in growth may cause declines in population-level fecundity and decreased reproduction.

Slower growth caused by disease would be expected to also reduce fecundity. American eels that were previously infected with A. crassus had lower length-at-age than uninfected American eels in upper watersheds of the Chesapeake Bay (Zimmerman and Welsh 2012), although a similar pattern was not seen in the lower Potomac River (Fenske et al. 2010). However, the upper watershed may be an important juvenile habitat for female American eels (Goodwin and Angermeier 2003). Therefore, an effect of A. crassus on growth could result in lower reproductive potential because American eels are semelparous with only one opportunity to spawn and smaller American eels would likely have lower fecundity (Zimmerman and Welsh 2012; Jessop 2018). During 2004–2008, 10% of Atlantic sturgeon from the Chesapeake Bay had infections of N. sturionis, which caused reduced growth (Matsche et al. 2010). Smaller size caused by the trematode infection may reduce reproductive success of Atlantic sturgeon in the Chesapeake Bay due to the lack of sufficient nutrition for gonadal development.

Increased mortality due to disease is expected to reduce the abundance of older and large individuals in a population because an increase in mortality at any age causes a truncation of a population’s age structure (Quinn and Deriso 1999). For example, diseases have shortened the eastern oyster life expectancy and lifetime fecundity in the James and Wicomico Rivers, where oyster life expectancy is around 3 years compared to an estimated 10–20 years for a population without disease (Mann et al. 2009; Southworth et al. 2010). This is also demonstrated in M. arenaria, where sarcomas mostly occur in adult clams. When a severe outbreak occurs in larger clams, infection and subsequent mortalities of infected adult clams lowers the number of individuals available to reproduce. In years of severe infections, both adults and juveniles will be negatively affected, possibly reducing the reproducing population for many years (Farley et al. 1991).

Slower growth and increased mortality due to disease combine to affect lifetime reproductive success. Mycobacteriosis in striped bass has the potential for large reproductive effects through cumulative mortality and reduced growth. Compromised growth due to mycobacteriosis was estimated to cause a 3.3% decrease in individual lifetime reproductive potential for female striped bass (Gervasi 2015). At the population level, disease-associated increases in natural mortality were estimated to cause a 74.5% decrease in reproductive output. The combined effects caused a 75.1% decrease, indicating that mycobacteriosis likely reduces the lifetime reproductive potential of striped bass in the Chesapeake Bay by a substantial amount (Gervasi 2015).

**Climate Change**

As indicated by references to climate effects elsewhere in this review, many hosts and pathogens are responsive to climate-driven changes in salinity, temperature, and pH (Burge et al. 2014). Shifting environmental conditions may affect interactions between hosts and pathogens, which can tilt the balance toward or away from high disease states (Burge et al. 2014; Barbieri et al. 2018). Our understanding of how climate change will affect marine infectious diseases in the Chesapeake Bay is restricted to hosts for which there are long-term infectious disease datasets (Harvell et al. 2002), in particular oysters and striped bass (Hofmann et al. 2001; Bushek and Ford 2016; Groner et al. 2018b). Predicting responses to climate change is further complicated by the synergistic effects of populations responding to multiple climate variables and subsequent changes in the marine community (Harvell et al. 2002; Ward and Lafferty 2004). Although the potential effects of climate change are well documented, the response of organisms to these effects are understudied for many ecosystems and taxonomic groups (Poloczanska et al. 2016), including those in the Chesapeake Bay. Many studies of potential effects of climate change use data from other regions to forecast effects for a new region. We adopted a similar approach in relying on studies from a variety of locations to inform potential responses of Chesapeake Bay species to climate change.

**Temperature**

Warming temperatures are expected to cause complex changes in infectious disease dynamics (O’Connor and Bernhardt 2018; Rohr and Cohen 2020). The Chesapeake Bay surface water temperatures are likely to increase 2–5.5 °C by 2100 (Muhling et al. 2018). As the overall average temperature increases, so will the frequency and duration of marine heat waves, with temperatures regularly reaching numbers currently considered extreme events (Oliver et al. 2018). Warming can affect host–pathogen interactions by changing pathogen development and survival, disease transmission, and host susceptibility (Harvell et al. 2002). The effects of increased temperature on diseases in the Chesapeake Bay have already been observed in some cases. For example, average summer sea
surface temperatures are correlated with increased mortality for striped bass with mycobacteriosis (Groner et al. 2018b). The combination of hypoxic conditions, increased water temperature, and severe mycobacteriosis reduced the metabolic performance of striped bass more than any individual factor (Lapointe et al. 2014).

In some cases, increased temperature may positively influence the pathogen, negatively affecting the host. Populations of soft clams (M. arenaria) in the Chesapeake Bay are likely to decrease as both sarcomas and Perkinsus sp. have higher virulence with increased temperatures (Farley et al. 1986; Glaspie et al. 2018). In addition, the mean infection intensity of Perkinsus sp. in M. arenaria and T. plebeius increases as temperature and salinity increase and dissolved oxygen decreases (Glaspie et al. 2018). Similarly, the two pathogens that cause the most prevalent diseases affecting eastern oysters in the Chesapeake Bay, Dermo and MSX, proliferate at temperatures above 20 °C and are inhibited by cold winter temperatures (Hofmann et al. 2001; Audemard et al. 2006; Soniat et al. 2009). Unusually high winter temperatures may facilitate a high survival of over-wintering Dermo and MSX organisms, leading to increased infections in the spring and summer. An increase in infections was observed in 1999 when abnormally high winter and summer temperatures coupled with drought conditions led to increases in Dermo and MSX disease and consequent high mortality of oysters throughout the Bay (Ragone Calvo and Burreson 2000; Gieseker 2001). Similarly, in 2001–2002, 2005–2006, and 2007–2008 the Wicomico River had high winter temperatures (above 5 °C), followed by increased oyster mortality and Dermo severity (Southworth et al. 2010).

Alternatively, some host species may experience beneficial effects with increasing temperature. In some clam species (M. balthica and Mercenaria mercenaria) in Chesapeake Bay, the extended periods of warmer weather and water temperatures may decrease effects of disease (Christensen et al. 1979; Perrigault et al. 2011). For example, prevalence of quahog parasite unknown (QPX), a protistan parasite that infects both wild and aquaculture hard clams, in M. mercenaria clams was higher at colder temperatures, and clams in warmer temperatures had a higher rate of healing when exposed to QPX (Perrigault et al. 2011).

Climate change effects can be complicated, as temperature increases may have contrasting effects on disease dynamics depending on the overall rise in temperature. Eelgrass has growth rates that increase linearly with temperatures from 5 to 25 °C, but temperatures over 25 °C cause decreased photosynthesis and growth in eelgrass, likely leading to enhanced physiological stress and susceptibility to pathogens (Kaldy 2014; Sullivan et al. 2018). The relationship is further complicated as the effect of temperature on the host may be different than the effect on the pathogen. For example, Z. marina collected in the Pacific Northwest naturally infected with L. zosterae had increased wasting disease severity at 25 °C compared to plants kept at 10 °C (Kaldy 2014).

Salinity

The predicted future salinity of the Chesapeake Bay is complex and spatially dependent, with climate models projecting a freshening of water in the upper Bay but an increase in salinity in the lower Bay (Muhling et al. 2018). Changes in salinity will likely affect organisms’ susceptibility to pathogens and the virulence of pathogens. Most hosts and pathogens have a specific salinity niche where they are able to survive, and changes in salinity in the Chesapeake Bay could lead to new host/pathogen interactions or reduced exposure. For example, L. zosterae does not grow in salinities lower than 10, meaning outbreaks of wasting disease may retreat towards the lower Chesapeake Bay with climate change (Muehlstein et al. 1988). In addition, soft-shell clams infected with transmissible sarcoma have the highest prevalence in the Bay mainstem, and are not detected in areas of freshwater influence (Farley et al. 1991).

The pathogens that cause the most common oyster diseases (MSX, Dermo, and SSO) are strongly influenced by salinity. Specifically, P. marinus has high viability in moderate to high salinities of 15–35 (La Peyre et al. 2006), H. nelsonii prefers salinities of 15–25, and H. costale prefers high salinities (> 30; Andrews 1979). These salinity constraints result in shifts in infection prevalence and intensity associated with rainfall or other sources of freshwater input, and high winter salinities may lead to increased epizootics in the following spring (Southworth et al. 2010). Droughts resulting in raised salinity caused substantial increases in Dermo and MSX disease in the Chesapeake Bay in 1991–1995, and 1999–2000, along with a movement of MSX disease into the upper Bay (Gieseker 2001; Mann et al. 2009). High freshwater inputs into the Bay halted further disease expansion and decreased disease severity (Mann et al. 2009; Tarnowski 2019).

Ocean Acidification

By 2100, the atmosphere in the Chesapeake region will see increases in CO₂ concentration of 50–160% (Najjar et al. 2010). As excess CO₂ is absorbed into the Chesapeake Bay, it will change the pH and alkalinity of the waters resulting in acidification. Chesapeake Bay pH changes are likely to be spatially dependent; model simulations of the Chesapeake Bay found that pH increased in the upper Bay, decreased in the lower Bay, and was variable in the mid-Bay from 1986 to 2015 (Shen et al. 2020). These changes to water chemistry are expected to affect the physiology of many important Chesapeake Bay marine organisms. However, there is a general lack of information on the effects of acidification on disease susceptibility of estuarine organisms. One exception is research indicating that future acidification may increase the
susceptibility of seagrasses to wasting disease outbreaks. High carbon dioxide concentrations result in a ~ 95% loss of antimicrobial phenolic acids released by seagrass species (Arnold et al. 2012, 2017). The loss of phenolic acids may be problematic as they inhibit growth of the pathogen responsible for seagrass wasting disease. Alternatively, laboratory studies found that eelgrass experienced less severe wasting disease with higher CO₂, although the mechanism for this response is unknown (Groner et al. 2018a). The uncertainty of wasting disease outbreaks with ocean acidification and the expected decrease with warming temperatures and lower salinity further emphasizes the complexity of host–pathogen interactions with climate change and the need for multi-factor experiments.

**Aquaculture**

Aquaculture of marine species has increased globally due to declining wild populations combined with the increasing demand for seafood products (FAO 2018). When dense monospecies aggregations are created, aquaculture can exacerbate the spread of disease. Within the Chesapeake Bay, an aquaculture industry currently exists for eastern oyster, hard clam, and soft-shell blue crabs. The transportation of oysters and the introduction of new species for aquaculture have facilitated the spread of infectious diseases globally (Minchin 2007). *H. nelsoni* (MSX) was introduced to California with the transportation of a foreign species, *Crassostrea gigas*, from Japan in the early 1900s. Similarly, MSX was likely introduced to Chesapeake Bay in the 1960s via *C. gigas* introductions to Delaware Bay in the 1950s (Andrews 1964; Ragone Calvo and Burreson 2000). Although aquaculture has enabled the spread of disease, there are recent suggestions that it may have the potential to reduce disease by filtering pathogens. However, this capability depends on the pathogen, timing of harvest, and the aquaculture species (Burge et al. 2016). As suspension feeders, oysters remove particulates from the water column including parasites, bacteria, and viruses. For non-human pathogenic diseases such as Dermo disease, modeling work suggests that aquaculture oysters may reduce the abundance of pathogens in the water column. If aquaculture populations filter these pathogens from the water column and if they are harvested prior to onset of disease, they may reduce the effect of a pathogen (Ben-Horin et al. 2018). Increasingly, eastern oyster aquaculture in the Chesapeake Bay is conducted with disease resistant oyster strains from selective breeding (Ragone Calvo et al. 2003a; Frank-Lawale et al. 2014).

Within the Chesapeake Bay, blue crabs can be sold as value-added “soft crabs,” a colloquial term for the post molt stage of a crab. Many commercial crabbers set aside crabs which look to be nearing a molt (known as peelers), and place them in shedding facilities, which are shallow flow-through or recirculating aquaculture systems. Multiple diseases have been associated with soft crab shedding facilities. For example, infection by CsRV1 was the primary predictor of mortality within shedding facilities, documented in 75% of dead crabs (Bowers et al. 2010; Spitznagel 2019). Given the prevalence of CsRV1 in shedding facilities, concerns have been raised about the potential for flow-through systems to reintroduce virus to nearby wild populations, and indeed, higher prevalence of CsRV1 has been observed in wild crabs near such facilities (Flowers et al. 2018). Similar to CsRV1, *Vibrio* spp. (*Vibrio parahaemolyticus, V. vulnificus, V. cholerae*) have been identified in both wild and shedding facility blue crabs (Messick and Kennedy 1990). Injury- and capture-related stress inherent to soft-shell facilities may increase the intensity of infection from *Vibrio* spp. (Sizemore et al. 1975). It remains to be determined whether the association of disease with soft crab production is a consequence of stress related to molting, captivity, or both.

Hard clam aquaculture has a strong presence in the lower Chesapeake Bay and surpassed the wild clam harvest in 2017 (Hudson 2018). QPX infects both wild and aquaculture hard clams, with higher mortality observed in aquaculture settings (Whyte et al. 1994; Ragone Calvo et al. 1998; Smolowitz 2018). Ford et al. (2002) observed significantly higher QPX presence in clam beds planted at higher densities, but overall disease infection and mortality depended on the seed source; hard clams originating from South Carolina had higher prevalence than seed from New Jersey when grown in New Jersey. The northernmost clam stocks had the highest survival rates and lowest prevalence, suggesting that host genotypes are a determinant of QPX susceptibility in hard clams (Ragone Calvo et al. 2007).

**Ecosystem Effects**

Diseases can affect habitat, nutrient cycling, and trophic interactions in the Chesapeake Bay by reducing populations of keystone species or ecosystem engineers. Repercussions from reductions in abundance of ecosystem engineers in other systems are well documented, such as coral reef habitats and kelp forests, with the disappearance of primary habitat engineers leading to community phase shifts (Ward and Lafferty 2004; Burge et al. 2014).

The eastern oyster is an ecosystem engineer that forms complex reef structures and is the main source of hard-bottom habitat in the Chesapeake Bay. In addition to habitat production, eastern oysters filter seawater which improves water quality and is important for nutrient cycling (Fig. 2; Kemp et al. 2005; Grabowski and Peterson 2007). The abundance of eastern oyster has declined substantially due to the combined effects of overfishing, disease, and habitat loss (Rothschild et al.
Like eastern oyster, eelgrass provides important nursery and foraging habitat for economically valuable species in Chesapeake Bay, such as blue crabs and numerous commercially exploited finfish (Reed and Hovel 2006). Eelgrass mortality as a result of L. zosterae infection causes reduced abundance, resulting in benthic habitat fragmentation and isolated patches which affects numerous marine and some terrestrial species (Muehlstein 1989). Altered eelgrass habitat configurations can lead to reductions in faunal abundance, diversity, and survival (Reed and Hovel 2006). Such loss has been estimated to translate to approximately 236 Mt reduction in epifaunal biomass in the Chesapeake Bay during 1991–2006 (Lefcheck et al. 2017). For commercially exploited species, the valuation of reduced blue crab biomass associated with eelgrass losses would cost the fishery an estimated $28.6 to $76.8 million USD (Lefcheck et al. 2017).

Declines of ecosystem engineer species due to disease effects may also affect nutrient cycling processes. Oysters remove nitrogen from the water column through assimilation into their tissues and shells along with deposition and burial through production of feces and pseudofeces (Kellogg et al. 2013). This supports benthic-pelagic coupling and the production of benthic microalgae (Newell and Mann 2012). The deposition of nutrients in shallow waters decreases their availability in the deeper Bay where organic matter contributes to hypoxia (Kemp et al. 2005). Oysters also consume phytoplankton (Newell et al. 2007) and reduce turbidity (Newell and Koch 2004). A study of local eastern oyster restoration sites in the Chesapeake Bay shows bio-geochemical flux rates of O2, NH4+, NO2+3, an order of magnitude greater than unrestored control sites, indicating increased nutrient sequestration and conversion (Kellogg et al. 2013).

Fishery management implications

The Chesapeake Bay supports important commercial (2017 landings valued at $73.7 m for blue crab, $59.8 m for eastern oyster, $23.5 m for Atlantic menhaden, $13 m for striped bass) and recreational fisheries (striped bass support the largest recreational fishery in the USA with 38 million pounds harvested in 2017; National Marine Fisheries Service 2018). Diseases can have substantial effects on fishery sustainability, particularly for the eastern oyster (Wilberg et al. 2011). However, less is known about how fishing practices and regulations may interact with disease and how information on disease effects (mortality, reproduction, movement) may improve management.
Routine disease monitoring is not conducted for the majority of important fishery species in Chesapeake Bay, and fishery management does not currently use information on disease to modify regulations. Disease prevalence and intensity for oysters are monitored annually within Maryland (e.g., Tarnowski 2019) and periodically in Virginia (e.g., Carnegie et al. 2004), but harvest regulations do not change annually in response to these data. Mycobacteriosis is monitored annually for striped bass in Maryland and periodically in Virginia (Gauthier et al. 2008; Hoenic et al. 2017; Maryland DNR unpublished data). The most recent stock assessment for striped bass began the process of including increased natural mortality due to mycobacteriosis in the Chesapeake Bay in the assessment models, but that model is not currently used for management (Northeast Fisheries Science Center 2019). Monitoring disease-driven mortality is noted as a critical data and analysis need for blue crab (Chesapeake Bay Stock Assessment Committee 2019). Effects of disease on mortality, growth, and reproduction are difficult to estimate, but they can have large effects on population sustainability (Wilberg et al. 2011; Gervasi 2015). Incorporating disease effects in the management of Chesapeake Bay fisheries should be a priority.

Conclusion

Diseases have had substantial effects on the Chesapeake Bay ecosystem, and they likely have had similar effects in less well-studied estuaries and coastal systems. Despite these effects, there are few estimates of effects of diseases on populations in the Chesapeake Bay, stemming partially from a lack of routine monitoring (Carnegie et al. 2016). This lack of monitoring highlights the need for new monitoring systems. Models to forecast disease dynamics and effects are also rare (Groner et al. 2016). For the organisms with population models, disease is often not explicitly included (Fenske et al. 2011; Miller et al. 2011; Wilberg et al. 2011). While improving understanding of disease effects on marine populations in estuarine and coastal ecosystems is often listed as a high priority, there is still a lack of knowledge about the roles of disease on populations, fisheries, and ecosystems. Advances in the application of molecular methods to track disease agents should make this goal more attainable (e.g., Burge et al. 2016; Zhao et al. 2020). Effects of diseases on coastal ecosystems are expected to increase in the future including increased virulence of diseases, increased host stress, and changes in the ranges of diseases and hosts (Cohen et al. 2018). Because anthropogenic stresses are thought to exacerbate effects of diseases, estuaries and the coastal ocean are likely areas that will show the largest effects (Groner et al. 2016). Additional study of disease prevalence and effects on population dynamics is necessary to accurately incorporate the effects of disease in management for marine organisms in the Chesapeake Bay and other coastal systems.

Acknowledgments

We thank the associate editor and two anonymous reviewers for helpful comments that improved this manuscript.

Open Access

This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article’s Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article’s Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by/4.0/.

References

Andrews, J.D. 1964. Oyster mortality studies in Virginia. IV. MSX in James River public seed beds. Proceedings of the National Shellfisheries Association 53: 65–84.

Andrews, J.D. 1979. Oyster diseases in Chesapeake Bay. Marine Fisheries Review 41 (1–2): 45–53.

Andrews, J.D., and M. Castagna. 1978. Epizootiology of Myxobolus costalis in susceptible oysters in Seaside Bays of Virginia’s Eastern Shore, 1959–1976. Journal of Invertebrate Pathology 32 (2): 124–138.

Andrews, J.D., J.L. Wood, and H.D. Hoese. 1962. Oyster mortality studies in Virginia III. Epizootiology of a disease caused by Haplosporidium costale. Journal of Invertebrate Pathology 4: 327–343.

Arnold, T., C. Mealey, H. Lefahey, A.W. Miller, J.M. Hall-Spencer, M. Milazzo, and K. Maers. 2012. Ocean acidification and the loss of phenolic substances in marine plants. PLoS One 7 (4): e35107.

Arnold, T.M., R.C. Zimmerman, K.A.M. Engelhardt, and J.C. Stevenson. 2017. Twenty-first century climate change and submerged aquatic vegetation in a temperate estuary: the case of Chesapeake Bay. Ecosystem Health and Sustainability 3 (7): 1353283.

Audemard, C., L.M. Ragone Calvo, K.T. Paynter, K.S. Reece, and E.M. Burreson. 2006. Real-time PCR investigation of parasite ecology: in situ determination of oyster parasite Perkinsus marinus transmission dynamics in lower Chesapeake Bay. Parasitology 132 (6): 827–842.

Barber, B.J., S.E. Ford, and H.H. Haskin. 1988. Effects of the parasite MSX (Haplosporidium nelsoni) on oyster (Crassostrea virginica) energy metabolism. I. Condition index and relative fecundity. Journal of Shellfish Research 7 (1): 25–31.

Barbieri, M., C. Duncan, A.L. Haring, K.L. Pabilonia, T.C. Johanoss, T. Goldstein, S.J. Robinson, and C.L. Littman. 2018. Survey for placental disease and reproductive pathogens in the endangered Hawaiian monk seal (Neomonachus schauinslandi). Journal of Wildlife Diseases 54 (3): 564–568.

Ben-Horin, T., C.A. Burge, D. Bushek, M.L. Groner, D.A. Proestou, L.J. Huey, G. Bidegain, and R.B. Carnegie. 2018. Intensive oyster aquaculture can reduce disease impacts on sympatric wild oysters. Aquaculture Environment Interactions 10: 557–567.

Blazer, V.S., W.K. Vogelbein, C.L. Densmore, E.B. May, J.H. Lilley, and D.E. Zwerner. 1999. Aphanomyces as a cause of ulcerative skin
lesions of menhaden from chesapeake bay tributaries. Journal of Aquatic Animal Health 11 (4): 340–349.

Bowers, H.A., G.A. Messick, A. Hanif, R. Jagus, L. Carrión, O. Zmora, and E.J. Schott. 2010. Physicochemical properties of double-stranded RNA used to discover a reo-like virus from blue crab Callinectes sapidus. Diseases of Aquatic Organisms 93 (1): 17–29.

Brakel, J., S. Jakobsson-Thor, A.C. Bockelmann, and T.B.H. Reusch. 2019. Modulation of the eelgrass–Labyrinthula zosterae interaction under predicted ocean warming, salinity change and light limitation. Frontiers in Marine Science 6: 1–13.

Burge, C.A., C.M. Eakin, C.S. Friedman, B. Froelich, P.K. Hersherberger, E.E. Hofmann, L.E. Petes, K.C. Prager, E. Weil, B.L. Willis, S.E. Ford, and C.D. Harvell. 2014. Climate change influences on marine infectious diseases: implications for management and society. Annual Review of Marine Science 6 (1): 249–277.

Burge, C.A., C.J. Closek, C.S. Friedman, M.L. Groner, C.M. Jenkins, A. Shore-Maggio, and J.E. Welsh. 2016. The use of filter-feeders to manage disease in a changing world. Integrative and Comparative Biology 56 (4): 573–587.

Bushek, D., and S.E. Ford. 2016. Anthropogenic impacts on an oyster metapopulation: mutation introduction, climate change and responses to natural selection. Elementa: Science of the Anthropocene 4: 000119.

Cardinal, J. L. 2001. Mycobacteriosis in striped bass, Morone saxatilis, from Virginia Waters of Chesapeake Bay. College of William and Mary- Virginia Institute of Marine Science.

Carnegie, R. B., L. M. Ragone Calvo, and E. M. Burreson. 2004. Status and Trends of American Eel in the Chesapeake Bay, USA. Integrative and Comparative Biology 54 (2): 173–179.

Carnegie, R.B., I. Arzul, and D. Bushek. 2016. Managing marine mollusc diseases in the context of regional and international commerce: policy issues and emerging concerns. Philosophical Transactions of the Royal Society, B: Biological Sciences 371 (1689): 20150215.

Chesapeake Bay Stock Assessment Committee. 2019. 2019 Chesapeake Bay blue crab advisory report.

Christensen, D.J., A. Farley, and F.G. Kem. 1974. Epizootic neoplasms in the clam Macoma balthica (L.) from Chesapeake Bay. Journal of the National Cancer Institute 52 (6): 1739–1749.

Cohen, R., C. James, A. Lee, M. Martellini, W. Maura, S. Ortega, R. Sadowski, L. Starkey, A. Szczesicka, S. Timko, E. Weiss, and F. Franks. 2018. Marine Host-Pathogen Dynamics: Influences of Global Climate Change. Oceanography 31 (2): 182–193.

Dungan, C.F., R.M. Hamilton, K.L. Hudson, C.B. McCollough, and K.S. Reece. 2002. Two epizootic diseases in Chesapeake Bay commercial clams, Mya arenaria and Tagelus plebeius. Diseases of Aquatic Organisms 50 (1): 67–78.

FAO. 2018. The state of the world fisheries and aquaculture 2018- meeting the sustainable development goals. Rome.

Farley, C.A., S. Otto, and C. Reinsch. 1986. New occurrence of epizootic sarcoma in Chesapeake Bay soft shell clams, Mya arenaria. Fishery Bulletin 84 (4): 851–857.

Farley, C.A., D.L. Plutschak, R.F. Scott, C. Haven, and B. Neck. 1991. Epizootioloogy and distribution of transmissible sarcoma in Maryland softshell clams, Mya arenaria, 1984-1988. Environmental Health Perspectives 90: 35–41.

Fenske, K.H., D.H. Secor, and M.J. Wilberg. 2010. Demographics and parasitism of American eels in the Chesapeake Bay, USA. Transactions of the American Fisheries Society 139 (6): 1699–1710.

Fenske, K.H., M.J. Wilberg, D.H. Secor, and M.C. Fabrizio. 2011. An age- and sex-structured assessment model for American eels (Anguilula rostrata) in the Potomac River, Maryland. Canadian Journal of Fisheries and Aquatic Sciences 68 (6): 1024–1037.

Flowers, E.M., T.R. Bachvaroff, J.V. Warg, J.D. Neill, M.L. Killian, A.S. Vinagre, S. Brown, A.S. Almeida, and E.J. Schott. 2016a. Genome sequence analysis of CsRV1: a pathogenic reovirus that infects the blue crab Callinectes sapidus across its trans-hemispheric range. Frontiers in Microbiology 7: 126.

Flowers, E.M., K. Simmonds, G.A. Messick, L. Sullivan, and E.J. Schott. 2016b. PCR-based prevalence of a fatal reovirus of the blue crab Callinectes sapidus (Rathbun) along the northern Atlantic coast of the USA. Journal of Fish Diseases 39 (6): 705–714.

Flowers, E.M., A.F. Johnson, R. Aguilar, and E.J. Schott. 2018. Prevalence of the pathogenic crustin-type virus Callinectes sapidus reovirus 1 near flow-through blue crab aquaculture in Chesapeake Bay, USA. Diseases of Aquatic Organisms 129 (2): 135–144.

Ford, S.E., and D. Bushek. 2012. Development of resistance to an introduced marine pathogen by a native host. Journal of Marine Research 70 (2): 205–223.

Ford, S.E., J.N. Kraeuter, R.D. Barber, and G. Mathis. 2002. Aquaculture-associated factors in QPX disease of hard clams: density and seed source. Aquaculture 208 (1–2): 23–38.

Ford, S.E., M. Cummings, and E.N. Powell. 2006. Estimating mortality in natural assemblages of oysters. Estuaries and Coasts 29 (3): 361–374.

Frank-Lawale, A., S.K. Allen, and L. Dégremont. 2014. Breeding and domestication of eastern oyster (Crassostrea virginica) lines for culture in the mid-atlantic, USA: line development and mass selection for disease resistance. Journal of Shellfish Research 33 (1): 153–165.

Gauthier, D.T., R.J. Latour, D.M. Hisey, C.F. Bonzek, J. Gartland, E.J. Burge, and W.K. Vogelbein. 2008. Mycobacteriosisis-associated mortality in wild striped bass (Morone saxatilis) from Chesapeake Bay, USA. Ecological Applications 18 (7): 1718–1727.

Gervasi, C.L. 2015. The reproductive biology of striped bass (Morone saxatilis) in the Chesapeake Bay. College of William and Mary-VIMS.

Gervasi, C.L., S.K. Lowerre-Barbieri, W.K. Vogelbein, J. Gartland, and R.J. Latour. 2019. The reproductive biology of Chesapeake Bay striped bass with consideration of the effects of mycobacteriosis. Bulletin of Marine Science 95 (2): 117–137.

Gieseker, C. 2001. Year 2000 Maryland Oyster Disease Status Report.

Glaspie, C.N., R.D. Seitz, M.B. Ogbum, C.F. Dungan, and A.H. Hines. 2018. Impacts of predator, harvest, recruitment, and disease on softshell clams Mya arenaria and stout razor clams Tagelus plebeius in Chesapeake Bay. Marine Ecology Progress Series 603: 117–133.

Goodwin, K.R., and P.L. Angermeier. 2003. Demographic characteristics of American eel in the Potomac river drainage, Virginia. Transactions of the American Fisheries Society 132 (3): 524–535.

Grabowski, J.H., and C.H. Peterson. 2007. Restoring oyster reefs to recover ecosystem services. Pages 281–298 Ecosystem Engineers. Elsevier.

Groner, M.L., J. Maynard, R. Breyta, R.B. Carnegie, A. Dobson, C.S. Friedman, B. Froelich, M. Garren, F.M.D. Gulland, S.F. Heron, R.T. Noble, C.W. Revie, J.D. Shields, R. Vanderstichel, E. Weil, S. Wylie-Echeverria, and C.D. Harvell. 2016. Managing marine disease emergencies in an era of rapid change. Philosophical Transactions of the Royal Society, B: Biological Sciences 371 (1689): 20150364.

Groner, M.L., C.A. Burge, R. Cox, N.D. Rivlin, M. Turner, K.L. Van Alstyne, S. Wylie-Echeverria, J. Bucci, P. Standigel, and C.S. Friedman. 2018a. Oysters and eelgrass: potential partners in a high production system. Bulletin of Marine Science 92 (1): 168–180.

Groner, M.L., J.M. Hoening, R. Pradel, R. Choquet, W.K. Vogelbein, D.T. Gauthier, and M.A.M. Friedricks. 2018b. Dermal mycobacteriosisis and warming sea surface temperatures are associated with elevated mortality of striped bass in Chesapeake Bay. Ecology and Evolution 8 (18): 9384–9397.

Haro, A., W. Richkus, K. Whalen, A. Hoar, W.-D. Busch, S. Lary, T. Brush, and D. Dixon. 2000. Population decline of the American eel: implications for research and management. Fisheries 25 (9): 7–16.
Harvell, C.D., C.E. Mitchell, J.R. Ward, S. Altizer, A.P. Dobson, R.S. Ostfeld, and M.D. Samuel. 2002. Climate warming and disease risks for terrestrial and marine biota. *Science* 296 (5576): 2158–2162.

Hixon, M.A., D.W. Johnson, and S.M. Sogard. 2014. BOFFF:s on the importance of conserving old-growth age structure in fishery populations. *ICES Journal of Marine Science* 71 (8): 2171–2185.

Hoenig, J.M., M.L. Groner, M.W. Smith, W.K. Vogelbein, D.M. Taylor, D.F. Landers, J.T. Swenarton, D.T. Gauthier, P. Sadler, M.A. Matsu, A.N. Haines, H.J. Small, R. Pradel, R. Choquet, and J.D. Shields. 2017. Impact of disease on the survival of three commercially fished species. *Ecological Applications* 27 (7): 2116–2127.

Hofmann, E., E. Powell, and J. Klinck. 2001. Modeling studies of the effect of climate variability on MSX disease in eastern oyster (*Crassostrea virginica*) populations. *Hydrobiologia* 460 (1): 195–212.

Huchin-Mian, J.P., H.J. Small, and J.D. Shields. 2018. The influence of temperature and salinity on mortality of recently recruited blue crabs, *Callinectes sapidus*, naturally infected with *Hematodinium perezi* (Dinoflagellata). *Journal of Invertebrate Pathology* 152: 8–16.

Hudson, K. 2018. Virginia shellfish aquaculture situation and outlook report results of the 2017 Virginia shellfish aquaculture crop reporting survey. Page VIMS Marine Resource Report No. 2018-9. Virginia Sea Grant VSG-18-3.

Jacobs, J.M., M.R. Rhodes, A. Baya, Reimschuessel, H. Townsend, and R.M. Harrell. 2009. Influence of nutritional state on the progression and severity of mycobacteriosis in striped bass Morone saxatilis. *Diseases of Aquatic Organisms* 87 (3): 183–197.

Jessop, B.M. 2018. American eel fecundity and ovary maturation in *Callinectes sapidus*. *Journal of Experimental Marine Biology and Ecology* 453: 108–115.

Johnson, P. T. 1983. Diseases caused by viruses, rickettsia, bacteria and fungi. Pages 2–78 in A. J. Provenzano Jr., editor. *The Biology of Zostera marina*. Vol. 6. Academic Press, New York, NY.

Johnson, P.T. 1977. Paramoebiasis in the blue crab. *Journal of Fish Diseases* 9 (2): 169–189.

Kane, A.S., C.B. Stine, L. Hungerford, M. Matsche, C. Driscoll, and A.M. Baya. 2007. Mycobacteria as environmental portent in Chesapeake Bay fish species. Emerging Infectious Diseases 13 (2): 329–331.

Kellogg, M.L., J.C. Cornwell, M.S. Owens, and K.T. Paynter. 2013. Denitrification and nutrient assimilation on a restored oyster reef. *Marine Ecology Progress Series* 480: 1–19.

Kemp, W.M., W.R. Boynton, J.E. Adolf, D.F. Boesch, W.C. Boicourt, G. Brush, J.C. Cornwell, T.R. Fisher, P.M. Gibert, J.D. Hagy, L.W. Harding, E.D. Houde, D.G. Kimmel, W.D. Miller, R.I.E. Newell, M.R. Roman, E.M. Smith, and J.C. Stevenson. 2005. Eutrophication of Chesapeake Bay: historical trends and ecological interactions. *Marine Ecology Progress Series* 303: 1–29.

Keppel, A.G., D.L. Breitburg, G.H. Wikfors, R.B. Burrell, and V.M. Clark. 2015. Effects of co-varying diel-cycling hypoxia and pH on disease susceptibility in the eastern oyster *Crassostrea virginica*. *Marine Ecology Progress Series* 538: 169–183.

Kirk, R.S. 2003. The impact of Anguillicola crassus on European eels. *Fisheries Management and Ecology* 10 (6): 385–394.

Kiryu, Y., J.D. Shields, W.K. Vogelbein, H. Kator, and V.S. Blazer. 2003. Infectivity and pathogenicity of the oomycete *Aphanomyces invadans* in Atlantic menhaden *Brevoorta tyrannus*. *Diseases of Aquatic Organisms* 54 (2): 135–146.

La Peyre, M., S. Casas, and J. La Peyre. 2006. Salinity effects on viability, metabolic activity and proliferation of three Perkinsus species. *Diseases of Aquatic Organisms* 71 (1): 59–74.

Lapointe, D., W.K. Vogelbein, M.C. Fabrizio, D.T. Gauthier, and R.W. Brill. 2014. Temperature, hypoxia, and mycobacteriosis: effects on adult striped bass *Morone saxatilis* metabolic performance. *Diseases of Aquatic Organisms* 108 (2): 113–127.

Latour, R.J., D.T. Gauthier, J. Garland, C.F. Bonzek, K.A. Menamme, and W.K. Vogelbein. 2012. Impacts of mycobacteriosis on the growth of striped bass (*Morone saxatilis*) in Chesapeake Bay. *Canadian Journal of Fisheries and Aquatic Sciences* 69 (2): 247–258.

Lefcheck, J.S., D.J. Wilcox, R.R. Murphy, S.R. Marion, and R.J. Orth. 2017. Multiple stressors threaten the imperiled coastal foundation species eelgrass (*Zostera marina*) in Chesapeake Bay, USA. *Global Change Biology* 23 (9): 3474–3483.

Lefebvre, F., G. Fazio, and A.J. Crivelli. 2011. *Anguilluloides crassus*. Pages 310–316 Fish Parasites: Pathobiology and Protection.

Lohan, K.M., K.S.reece, T.L. Miller, K.N. Wheeler, H.J. Small, and J.D. Shields. 2012. The role of alternate hosts in the ecology and life history of *Hematodinium sp.*, a parasitic dinoflagellate of the blue crab (*Callinectes sapidus*). *Journal of Parasitology* 98 (1): 73–84.

Mann, R., M. Southworth, J.M. Harding, and J.A. Wesson. 2009. Population studies of the native Eastern oyster, *Crassostrea virginica*, (Gmelin, 1791) in the James River, Virginia, USA. *Journal of Shellfish Research* 28 (2): 193–220.

Maryland Department of Natural Resources. 2001. Maryland oyster population status report 1996–2000 fall surveys.

Matsche, M.A., J.R. Flowers, E.L. Markin, and C.P. Stence. 2010. Observations and treatment of *Nitzschia sturionis* on Atlantic sturgeon from Chesapeake Bay. *Journal of Aquatic Animal Health* 22 (3): 174–181.

McCoughlin, C.B., B.W. Albright, G.R. Abbe, L.S. Barker, and C.F. Dungan. 2007. Acquisition and progression of Perkinsus marinus infections by specific-pathogen-free juvenile oysters (*Crassostrea virginica* Gmelin) in a mesohaline Chesapeake Bay tributary. *Journal of Shellfish Research* 26 (2): 465–477.

Messick, G.A. 1994. *Hematodinium perezi* infections in adult and juvenile blue crabs *Callinectes sapidus* from coastal bays of Maryland and Virginia, USA. *Diseases of Aquatic Organisms* 19: 77–82.

Messick, G.A., and V.S. Kennedy. 1990. Putative bacterial and viral infections in blue crabs, *Callinectes sapidus* Rathbun, 1896 held in flow-through or a re-circulating system. *Journal of Shellfish Research* 9: 33–40.

Messick, G.A., and J.D. Shields. 2000. Epizootiology of the parasitic dinoflagellate *Hematodinium* sp. in the American blue crab *Callinectes sapidus*. *Diseases of Aquatic Organisms* 43 (2): 139–152.

Miller, T.J., M.J. Wilberg, A.R. Colton, G.R. Davis, A. Sharov, R.N. Lipcius, G.M. Ralph, E.G. Johnson, and A.G. Kaufman. 2011. *Labyrinthula zosterae* sp. nov., the causative agent of wasting disease of eelgrass, Zostera marina. *Marine Biology* 158 (2): 394–406.

Morado, J.F. 2011. Protistan diseases of commercially important crabs: a review. *Journal of Invertebrate Pathology* 106 (1): 27–53.

Muehlstein, L.K., D. Porter, and F.T. Short. 1988. Observations and treatment of *Lipschitzia sturionis* on Atlantic sturgeon from Chesapeake Bay. *Bulletin Marine Resources* 258: 197–29.

Muhling, B.A., C.F. Gaitán, C.A. Stock, V.S. Saba, D. Tommasi, and W.K. Vogelbein. 2012. The importance of conserving old-growth age structure in fishery populations. *Hydrobiologia* 673: 1–11.

Muhling, B.A., C.F. Gaitán, C.A. Stock, V.S. Saba, D. Tommasi, and K.W. Dixon. 2018. Potential salinity and temperature futures for the Chesapeake Bay using a statistical downscaling spatial disaggregation framework. *Estuaries and Coasts* 41 (2): 349–372.
Stine, C.B., A.M. Baya, J.D. Salierno, M. Kollner, and A.S. Kane. 2005. Mycobacterial infection in laboratory-maintained Atlantic menhaden. *Journal of Aquatic Animal Health* 17 (4): 380–385.

Stine, C.B., A.S. Kane, and A.M. Baya. 2010. Mycobacteria isolated from Chesapeake Bay fish. *Journal of Fish Diseases* 33 (1): 39–46.

Sullivan, B.K., S.M. Trevathan-Tackett, S. Neuhauser, and L.L. Govers. 2018. Review: host-pathogen dynamics of seagrass diseases under future global change. *Marine Pollution Bulletin* 134: 75–88.

Tarnowski, M. 2005. Maryland oyster population status report 2003 and 2004 fall surveys. Annapolis, MD.

Tarnowski, M. 2019. Maryland oyster population status report 2018 fall survey. Annapolis, MD.

Ward, J.R., and K.D. Lafferty. 2004. The elusive baseline of marine disease: are diseases in ocean ecosystems increasing? *PLoS Biology* 2 (4): e120 Public Library of Science.

Webb, S.R., G.C. Garman, S.P. McIninch, T.A. Nerad, M.T. Peglar, P.M. Gillevet, and B.L. Brown. 2005. Etiology of ulcerative lesions of Atlantic menhaden (*Brevoortia tyrannus*) from James River, Virginia. *Parasitology Research* 97 (5): 358–366.

Wylte, S.K., R.J. Cawthorn, and S.E. McGladdery. 1994. Co-infection of bay scallops *Argopecten irradians* with *Perkinsus karlssoni* (Apicomplexa, Perkinsea) and an unidentified coccidian parasite. *Diseases of Aquatic Organisms* 18 (1): 53–62.

Wilberg, M.J., M.E. Livings, J.S. Barkman, B.T. Morris, and J.M. Robinson. 2011. Overfishing, disease, habitat loss, and potential extirpation of oysters in upper Chesapeake Bay. *Marine Ecology Progress Series* 436: 131–144.

Zhao, M., D. Behringer, J. Bojko, A. Kough, L. Plough, C. Tavares, A. Aguilar-Perera, O. Reynoso, G. Seepersad, O. Maharaj, M. Sanders, D. Camales, G. Fabiano, D. Carmeivia, M. Freeman, N. Atherley, L. Medero-Hernández, and E. Schott. 2020. Climate and season are associated with prevalence and distribution of trans-hemispheric blue crab reovirus (Callinectes sapidus reovirus 1). *Marine Ecology Progress Series* 647: 123–133.

Zimmerman, J.L., and S.A. Welsh. 2012. Prevalence of Anguillicoloides crassus and growth variation in migrant yellow-phase American eels of the upper Potomac River drainage. *Diseases of Aquatic Organisms* 101 (2): 131–137.