# Climate Change Influences on Marine Infectious Diseases: Implications for Management and Society

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Annu. Rev. Mar. Sci. 2014. 6:249–77

First published online as a Review in Advance on June 27, 2013

The *Annual Review of Marine Science* is online at marine.annualreviews.org

This article's doi: 10.1146/annurev-marine-010213-135029



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## Keywords

epizootics, mass mortalities, health, oceans, ocean warming

## Abstract

Infectious diseases are common in marine environments, but the effects of a changing climate on marine pathogens are not well understood. Here we review current knowledge about how the climate drives host-pathogen interactions and infectious disease outbreaks. Climate-related impacts on marine diseases are being documented in corals, shellfish, finfish, and humans; these impacts are less clearly linked for other organisms. Oceans and people are inextricably linked, and marine diseases can both directly and indirectly affect human health, livelihoods, and well-being. We recommend an adaptive management approach to better increase the resilience of ocean systems vulnerable to marine diseases in a changing climate. Land-based management methods of quarantining, culling, and vaccinating are not successful in the ocean; therefore, forecasting conditions that lead to outbreaks and designing tools/approaches to influence these conditions may be the best way to manage marine disease.

## INTRODUCTION

**Infectious disease:** a disease caused by a transmittable agent (e.g., a virus, bacterium, parasite, fungus, alga, or prion) that infects the host tissues, leading to an identifiable illness or syndrome

**Pathogen:** a causative agent of disease; under certain conditions, parasites may also cause disease

Marine ecosystems are among the most valuable and heavily used natural systems worldwide (Staudinger et al. 2012) and provide critical ecosystem services, including shoreline protection, water filtration, nursery grounds, food from fisheries and aquaculture, and revenue from tourism (reviewed in Ruckelshaus et al. 2013). For example, coastal wetlands provide storm protection services estimated to be worth $23.2 billion per year in the United States alone (Costanza et al. 2008). Seafood currently provides vital jobs and ~15% of the animal protein intake for the world's population (Cooley et al. 2012). In addition to being economically and ecologically valuable, marine ecosystems and their interconnected web of organisms (e.g., from microbes to phytoplankton to zooplankton to shellfish to fish to marine mammals) are vulnerable to natural and human perturbations (Gilman et al. 2008). Marine ecosystems are influenced not only by the direct effects of stressors on populations and species but also by changes in species interactions, including competition, predation, parasitism, and mutualism (reviewed in Kordas et al. 2011). Preserving the health of marine ecosystems and managing them effectively require determining the effects of such stressors, including marine infectious disease and climate change.

Infectious diseases are important drivers within ecosystems. There are many well-documented terrestrial examples of ecosystems being reshaped by large-scale outbreaks of infectious disease, such as Dutch elm disease and chestnut blight (Anderson et al. 2004). Diseases may also impact marine ecosystems by influencing community structures, age distributions, trophic interactions, hydrodynamics, and biotic structures, as has been observed in relation to diseases in seagrasses (e.g., eelgrass wasting disease; reviewed in Burge et al. 2013), reef-building corals (Aronson & Precht 2001), oysters (Mann et al. 2009), and sea urchins (Lessios et al. 1984, Lauzon-Guay et al. 2009). Diseases have had large impacts on both cultured and wild harvests of commercially important species, such as salmon [e.g., *Ichthyophonus* infection in marine and anadromous fish (reviewed in McVicar 2011) and viral infections in Atlantic and Pacific salmon (reviewed in Kurath & Winton 2011)], abalone (e.g., withering syndrome; Friedman et al. 2000), and crustaceans (e.g., protozoan infections of natural populations and viruses in aquacultured species; reviewed in Stentiford et al. 2012). In both terrestrial and marine systems, interactions between hosts, pathogens, and the environment govern disease outbreaks, and a change in any of these components can shift the balance toward or away from a high-intensity disease state. As many host-pathogen interactions are highly vulnerable to changes in environment, climate change can alter the likelihood of disease outbreaks. Climate change has altered terrestrial agricultural disease risk (Garrett et al. 2012) and may be affecting human disease risk (Harvell et al. 2002, 2009; Lafferty et al. 2004; Baker-Austin et al. 2012). We are only beginning to understand the effects of infectious diseases in the ocean and how climate change will affect marine host-pathogen interactions, both of which are critical for informing conservation and management efforts (Harvell et al. 2009).

The effects of climate change and ocean acidification are being documented in oceans around the world (IPCC 2007, Doney et al. 2012). Here, we review aspects of climate change that could affect host-pathogen interactions, as these changes will have large impacts on disease outbreaks. Climate change is affecting ocean physical, chemical, and biological systems as well as human uses of ocean resources. Rising levels of atmospheric carbon dioxide ($CO_2$) are leading to increased global atmospheric and ocean temperatures; without significant near-term reductions in $CO_2$ levels, ocean warming is likely to continue (IPCC 2007). Increasing temperatures lead to physical impacts on ocean systems, including rising sea levels, increased ocean stratification, loss of sea ice, and altered oceanic circulation (Doney et al. 2012, Howard et al. 2013). Warming temperatures have already affected the survival, growth, reproduction, health, and phenology of marine organisms (Doney et al. 2012). For example, periods of thermal stress contribute to mass

coral bleaching (Hoegh-Guldberg et al. 2007) and disease outbreaks (Harvell et al. 2002, 2009). Increased periodicity of anomalous thermal events is reducing the capacity for recovery between events (Baker et al. 2008, Eakin et al. 2010).

In addition to temperature-related consequences, the increased $CO_2$ concentrations in the atmosphere are raising $CO_2$ concentrations in the ocean, causing chemical reactions that reduce ocean pH, a phenomenon termed ocean acidification (Doney et al. 2009). The ocean has become 30% more acidic over the past century (Feely et al. 2004), and the rate of acidification is accelerating (Gattuso & Hansson 2011), creating conditions unparalleled in the past 300 million years (Hönisch et al. 2012). Ocean acidification directly threatens the health of many calcifying organisms, including pteropods (Fabry et al. 2009), corals (Hoegh-Guldberg et al. 2007), and oysters (Barton et al. 2012).

Physical and chemical changes associated with climate change and ocean acidification are affecting the health of marine organisms and ecosystems (Harvell et al. 2009). At least five well-characterized syndromes of corals—white syndromes in the Caribbean and Pacific, white patch disease (also known as white pox disease or *Acropora* serriatosis) of threatened acroporids in the Caribbean, white plague, black band disease, and Caribbean yellow band disease—are temperature sensitive, and disease outbreaks are increasing with warming (Harvell et al. 2009). Other marine organisms, such as abalones, fish, seagrasses, and sea urchins, are also affected by warming oceans, as are humans. Many marine organisms, including marine pathogens, are shifting their distributions poleward as ocean temperatures warm (e.g., Parmesan & Yohe 2003, Nye et al. 2009, Baker-Austin et al. 2012). Outbreaks of *Vibrio* bacterial infections affecting humans have been recently reported as far north as the Baltic Sea (Baker-Austin et al. 2012) and Alaska (Martinez-Urtaza et al. 2010). Similarly, the ranges of some protistan diseases [e.g., Dermo disease (perkinsosis) and MSX (multinucleated sphere unknown) disease of the eastern oyster, *Crassostrea virginica*] have moved further north with changing temperatures (Burreson & Ragone Calvo 1996, Ford & Tripp 1996).

Environmental changes, including temperature increases, have been linked to enhanced disease expression (reviewed in Harvell et al. 2002). Climate shifts can impair the immune response of a host and increase the frequency of disease. This is especially true for ectothermic organisms such as shellfish (Travers et al. 2009), corals (Harvell et al. 2002), and finfish (reviewed in Bowden 2008). In the US states of Oregon and Washington, the reemergence of *Vibrio tubiashii*, a bacterial pathogen of larval Pacific oysters (*Crassostrea gigas*), was linked to climate-related changes, including thermal shifts and upwelling of low-pH waters (Elston et al. 2008). Changes in host species can also increase disease frequency. For example, inbreeding of the host species could favor the parasite over the host (e.g., Dionne et al. 2007). Conversely, changes in the parasite could result in increased virulence (enhancing the parasite's ability to infect and overcome the host immune response) or increased pathogenicity (increasing clinical disease associated with infection; e.g., Martenot et al. 2011). Alternatively, changes in the host, pathogen, or environment may favor the host, thereby reducing or eliminating a particular pathogen or disease in the affected population. For example, Pacific oysters selected for resistance to summer mortality were more resistant to a subsequent herpesvirus infection (Dégremont 2011).

Here, we consider the potential consequences of climate-related factors (e.g., temperature, rainfall, storms, and increased acidity) for marine infectious disease outbreaks (see **Figure 1**) as well as the known effects of climate on disease-related mass mortality events (**Table 1**). We review (*a*) the known and hypothesized impacts of climate change on the dynamics of marine infectious disease, focusing on effects on host resistance, pathogen virulence, and interactions between host resistance and pathogen virulence; (*b*) the known and potential consequences for human society; (*c*) the tools needed to fill future knowledge gaps associated with the impacts of climate change on

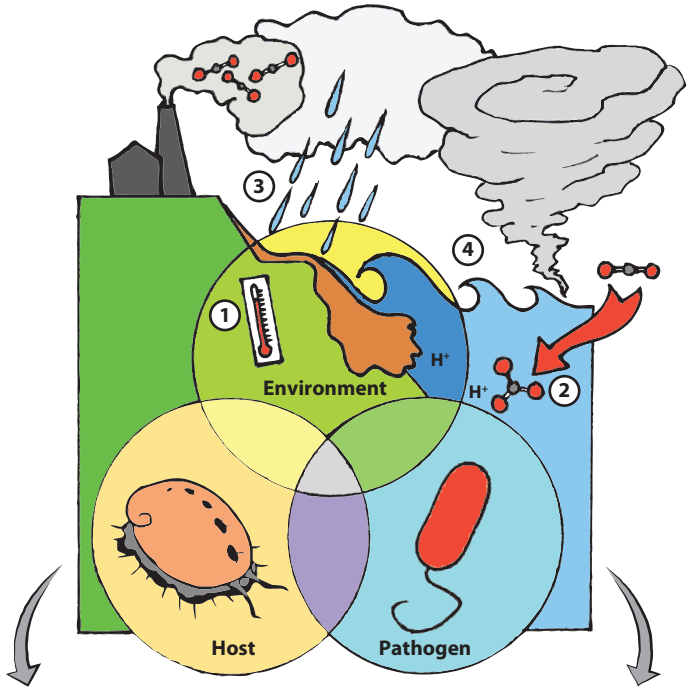
**Parasite:** an organism (often microscopic) that is metabolically dependent on its host and typically gains energy or food from its host, thus creating a negative association

**Virulence:** the characteristics of a pathogen that allow it to infect, multiply, and spread within or among hosts

**Pathogenicity:** the ability of a pathogen to cause disease in its host

**Resistance:** an organism's natural tolerance to infection

**Opportunistic pathogens:** pathogens that can survive outside a host and are often ubiquitous in the environment and within both healthy and diseased hosts, but that are pathogenic only under specific conditions




| Changes in host | Climate factor | Changes in pathogen |
|---|---|---|
| • Increased susceptibility<br>• Change in behavior<br>• Invasions<br>• Range shifts | ① | • Increased activity<br>• Change in virulence<br>• New emergence<br>• Invasions<br>• Range shifts |
| • Increased susceptibility (?)<br>• Change in larval survival/recruitment<br>• Change in behavior<br>• Reductions in diversity (?) | ② $H^+$ $2-$ $H^+$ | • Change in virulence<br>• Change in abundance |
| • Increased susceptibility<br>• Nutrient stress<br>• Salinity stress<br>• Contaminant stress | ③ | • New emergence<br>• Change in virulence<br>• Invasions<br>• Range shifts<br>• Increased nutrients |
| • Increased susceptibility<br>• Physical injury | ④ | • New emergence<br>• Invasions<br>• Range shifts<br>• Mixing/resuspension of particles |

**Figure 1**

Climate change impacts on marine host-pathogen-environment relationships. Shifts in the global environment are leading to physical ocean changes, including ① changes in temperature, ② increases in $CO_2$ concentrations/decreases in pH, ③ changes in precipitation (leading to changes in salinity), and ④ exposure to storms and cyclones. All of these factors are shifting the host-pathogen-environment equilibrium.

marine host-pathogen interactions; and (*d*) potential management actions to increase the resilience of ocean ecosystems, communities, and economies in the face of changes in disease. Specifically, we describe host-pathogen relationships where sufficient information exists, using case studies on the known and hypothesized effects of climate change on marine infectious diseases from invertebrate (corals, abalones, and oysters) and vertebrate (marine mammals, finfish, and humans) species.

## CASE STUDIES: IMPACTS OF CLIMATE CHANGE ON MARINE HEALTH

### The Decline of Coral Reef Ecosystems

Hermatypic corals have evolved in tropical oceans that in the recent past were characterized by relatively gradual changes in temperature and pH ranges. Over the past four decades, however, increasing environmental stress from rapidly changing climate-related and interacting climate-related and anthropogenic factors has disrupted the balance between hosts, agents, and the environment that underpins coral health. Disruption of coral-microbial symbioses and concomitant reduced resistance to opportunistic pathogens have been major factors in the deterioration of coral reef communities worldwide (e.g., Bruno & Selig 2007, Hoegh-Guldberg et al. 2007, Miller et al. 2009, Weil et al. 2009a, De'ath et al. 2012). Nowhere is coral reef deterioration more widespread and intense than in the wider Caribbean region, a disease hot spot characterized by frequent temperature-induced mass coral bleaching (loss of photosymbionts), the rapid emergence of a variety of new and virulent diseases, and typically higher disease prevalence than in other reef regions (reviewed in Harvell et al. 2007, Ruiz-Moreno et al. 2012). Alarming declines in coral cover have also been recorded on Indo-Pacific reefs (Bruno & Selig 2007, De'ath et al. 2012) along with the first records of several new diseases (Antonius 1999, Raymundo et al. 2003, Willis et al. 2004), highlighting the global nature of declining coral health.

Temperature-induced stress has been a key factor in mass mortalities associated with bleaching and infectious disease in coral reef organisms (Harvell et al. 1999, 2002, 2009; Hoegh-Guldberg et al. 2007; Carpenter et al. 2008; Croquer & Weil 2009a; McClanahan et al. 2009; Miller et al. 2009; Weil et al. 2009a; Rogers & Muller 2012). Significant warming of the Caribbean basin in the past 25 years (Chollet et al. 2012), including six major thermal anomalies and warmer winters, has coincided with coral bleaching events, disease emergence, and an increasing frequency of infectious disease outbreaks (Weil et al. 2009a, Eakin et al. 2010, Ruiz-Moreno et al. 2012). Similarly, on the Great Barrier Reef, high summer thermal anomalies and mild winter temperatures have been linked to outbreaks of tissue-loss coral diseases (e.g., white syndromes) (Bruno et al. 2007, Heron et al. 2010, Maynard et al. 2011). The host range and abundance of one of the most temperature- and nutrient-sensitive coral diseases, black band disease, have increased worldwide, likely reflecting the combined impacts of compromised host resistance and enhanced pathogen virulence associated with increasing seawater temperature and declining water quality (Voss & Richardson 2006; Sato et al. 2009, 2011).

Water quality, encompassing measures of turbidity, nutrient load, sediments, and pollutants, is a key factor in coral health that is influenced by a changing climate, with significant implications for the emergence and spread of coral diseases. Projected increases in the frequency of extreme weather events and associated increases in precipitation (Bender et al. 2010) could reduce coastal and ocean water quality through terrestrial inputs from runoff and resuspension events. Increases in terrestrial runoff lead to reduced salinity, enhanced sediments, increased human sewage pollution, and increased nutrients; this combination can compromise host resistance, as shown by links

**Table 1 Mass mortalities (>10%) of marine organisms associated with environmental correlates**

| Time period | Location | Host organism | Causative agent or syndrome | Environmental correlates | Source |
|---|---|---|---|---|---|
| 1913–present | North Atlantic and North Pacific | Finfish | *Ichthyophonus* sp. | ? | See herein; **Table 2** |
| 1931–present | North America and Europe | Marine plant (seagrass) | *Labyrinthula* sp. | HT, salinity | Burge et al. 2013 (review) |
| 1946–present | Gulf Coast and Northeast Atlantic | Bivalve (eastern oyster) | *Perkinsus marinus* (Dermo) | HT, salinity | See herein |
| 1958–present | Northeast Atlantic | Bivalve (eastern oyster) | *Haplosporidium nelsoni* (MSX) | HT, high salinity | See herein |
| 1960–present | Northeastern North America | Bivalve (hard clam) | Thraustochytrids (QPX) | HT, high salinity | Lyons et al. 2007, Burge et al. 2013 (review) |
| 1974–present | Europe | Bivalve (flat oyster) | *Marteilia refringens* | Salinity? | Elston & Ford 2011 (review) |
| 1975 | Western United States | Echinoderm (starfish) | ? | HT | Dungan et al. 1982 |
| 1979–1980 | New England, United States | Marine mammal (harbor seal) | Influenza A virus | HT | Geraci et al. 1982 |
| 1979–1983 | Caribbean | Corals (*Acropora* spp.) | White band disease | M-HT | Aronson & Precht 2001 |
| 1982 | Central America | Octocorals | Pathogen? | HT | Guzmán & Cortés 1984 |
| 1982–1986 | Australia | Gastropod (abalone) | *Perkinsus* sp. | HT | Villalba et al. 2004 (review) |
| 1983 | Caribbean | Corals | Black band disease | Seasonal | Ruetzler et al. 1983 |
| 1983 | Caribbean | Echinoderm (urchin) | Bacteria? | HT | Lessios et al. 1984 |
| 1985–present | Northeast Pacific | Gastropod (abalone) | "*Candidatus* Xenohaliotis californiensis" | HT | See herein |
| 1987–present | Florida | Marine plant (seagrass) | *Labyrinthula* sp. | HT, salinity | Robblee et al. 1991 |
| 1988 | Northwestern Europe | Marine mammal (harbor seal) | Phocine distemper virus | HT | Lavigne & Schmitz 1990 |
| 1990–1992 | Western Mediterranean | Marine mammal (striped dolphin) | Dolphin morbillivirus | HT | Aguilar & Raga 1993 |
| 1991–present | Worldwide | Bivalves (primarily Pacific oysters) | Oyster herpesvirus type 1 | Seasonal | Martenot et al. 2011 |
| 1992 | Northeastern New Zealand | Algae (kelp) | ? | High turbidity | Cole & Babcock 1996 |
| 1993–present | Florida | Corals (*Acropora* spp.) | White patch disease | HT | Patterson et al. 2002 |
| 1995 | Florida | Corals | White plague type II | HT | Richardson et al. 1998 |
| 1995–present | Caribbean | Octocorals (primarily sea fans) | *Aspergillus sydowii* and other fungi | HT | Burge et al. 2013 (review) |
| 1996-present | New England, United States | Crustacea (lobster) | Epizootic shell disease | HT, pollutants? | Castro et al. 2012 (review) |

| | | | | | |
|---|---|---|---|---|---|
| 1997 | Vancouver Island, Canada | Bivalve (Pacific oyster) | *Mikrocytos mackini* | LT | Bower et al. 1997 |
| 1998 | Florida | Octocorals | Cyanobacteria | HT | Harvell et al. 2002 |
| 1998–1999 | Central Visayas, Philippines | Corals | Porites ulcerative white spot disease | ? | Raymundo et al. 2003 |
| 1998–present | France | Gastropod (abalone) | *Vibrio harveyi* | HT | Travers et al. 2009 |
| 1999–2003 | Mediterranean Sea | Octocorals (gorgonian) | *Vibrio coralliilyticus* | HT | Bally & Garrabou 2007 |
| 2000 | Caspian Sea | Marine mammal (seal) | Canine distemper virus | HT | Kuiken et al. 2006 |
| 2000–2002 | Magnetic Island, Great Barrier Reef | Corals | Atramentous necrosis | Seasonal HT | Jones et al. 2004 |
| 2001 | New England, United States | Bivalve (Eastern oyster) | *Roseovarius* oyster disease | M-HT, pollution | Ford & Tripp 1996, Boettcher et al. 2005 |
| 2002–2003 | Great Barrier Reef | Corals | White syndrome | HT, mild winter temperatures | Willis et al. 2004, Bruno et al. 2007, Heron et al. 2010, Maynard et al. 2011 |
| 2003–2009 | Puerto Rico | Corals | Caribbean yellow band disease, white plague disease | HT-BLE | Bruckner & Hill 2009; **Figure 2** herein |
| 2005–2006 | Virgin Islands | Corals | Multiple diseases | HT-BLE | Miller et al. 2009 |
| 2005–2006 | Puerto Rico | Corals | Multiple diseases | HT-BLE | Weil et al. 2009a |
| 2006–2007 | US west coast | Bivalves (oysters) | *Vibrio tubiashii* | HT, nutrient enrichment | Elston et al. 2008 |
| 2006–2008 | Pelorus Island, Great Barrier Reef | Corals | Black band disease | Seasonal HT and light | Sato et al. 2009 |
| 2008–2009 | Great Barrier Reef | Corals | Atramentous necrosis | HT, low salinity, high particulate organic carbon | Haapkylä et al. 2011 |
| 2010–2011 | Los Roques, Venezuela | Corals | Black band disease, white plague disease | HT-BLE | Bastidas et al. 2012 |
| 2010–2011 | Curaçao | Corals | Multiple diseases | HT-BLE | E. Weil, unpublished data |
| 2010–2011 | Grenada | Corals | Multiple diseases | HT-BLE | E. Weil, unpublished data |

Abbreviations: BLE, coral bleaching; HT, high temperatures; M-HT, mid-to-high temperatures; LT, low temperatures. "Seasonal" indicates that mortalities occur seasonally with maximum temperatures.

**Epizootic:** an unexpected increase in disease or mortality in nonhuman hosts that occurs in an unusual time or place or at an unusually high frequency

with outbreaks of the coral disease atramentous necrosis (Haapkylä et al. 2011), and can potentially increase the frequency of opportunistic pathogens (Burge et al. 2013). Severe storms also compromise host resistance through direct breakage, abrasion, and surface injuries that provide entry points for pathogens. Additionally, declining seawater pH will undermine skeletal supporting structures, further increasing the susceptibility of corals to breakage injuries. Rising temperatures, intensifying storms, and falling pH adversely affect not only adult corals but also those at earlier life-history stages (e.g., Negri et al. 2007, Albright et al. 2010), which, in combination with reduced reproductive output caused by bleaching and disease (e.g., Szmant & Gassman 1990, Weil et al. 2009b), suggests recurrent reproductive and recruitment failures that will increasingly hinder future reef recovery.

Coral epizootics illustrate how a cascade of interacting environmental and anthropogenic disturbances can dramatically alter community structure in coral reef ecosystems, leading to the need for management and restoration. Populations of two important reef-building corals (*Acropora cervicornis* and *Acropora palmata*) in the Caribbean crashed in the early 1980s (Gladfelter 1982, Aronson & Precht 2001); this was followed more recently by outbreaks of white patch disease in the Florida Keys and the Virgin Islands (Rogers & Muller 2012) that were caused by the fecal endobacterium *Serratia marcescens* and linked with human sewage in the Florida Keys (Sutherland et al. 2010). Similarly, an unknown pathogen caused mass mortality of the black sea urchin (*Diadema antillarum*) (Lessios et al. 1984), a keystone species controlling macroalgal biomass, which led to algal overgrowth of reefs and reduced space for coral recruitment. *Acropora* and *Diadema* populations experienced >90% mortality throughout their Caribbean ranges and have not recovered even 30 years after the epizootics (Weil et al. 2005). Remaining reef-building genera and other important groups have been affected by severe storms, major widespread bleaching events (in 1998, 2005, and 2010), and outbreaks of a variety of biotic diseases. The consequence has been a cascade of dramatic decreases in coral cover, increases in macroalgal cover, and changes in community structure and function (e.g., Hughes 1994; Richardson et al. 1998; Aronson & Precht 2001; Carpenter et al. 2008; Bruckner & Hill 2009; Croquer & Weil 2009a,b; McClanahan et al. 2009; Miller et al. 2009; Weil et al. 2009b) (**Figure 2**). Disease outbreaks contributed to the 2006 listing of two Caribbean coral species as threatened under the US Endangered Species Act, and to a 2012 proposal to list seven species as endangered and two additional species as threatened.

Pathogen identification and environmental correlates of coral disease remain critical knowledge gaps for understanding how coral health can be managed in the context of changing climate. Clearly, managing water quality is important for reducing environmental burdens that interact with climate change. Evidence is accumulating that marine protected areas (MPAs) with intact reef communities and reduced fishing disturbances may help maintain coral health (Page et al. 2009, Raymundo et al. 2009). Although MPAs may not protect coral reefs against climate-related threats (e.g., Miller et al. 2009, Weil et al. 2009a, Selig & Bruno 2010), MPA networks may provide important mechanisms to increase ecosystem resilience (Keller et al. 2009), potentially reseeding nearby degraded reefs. MPAs will become increasingly important for managing the growing litany of unprecedented challenges to the long-term persistence of coral reef ecosystems, which are critical for developing and developed economies that depend on the ecosystem services provided by these systems. In the case of localized coral disease outbreaks and mass coral bleaching events (which are noninfectious in nature), there is potential for early detection to prepare for vulnerabilities and implement management plans. The US National Oceanic and Atmospheric Administration (NOAA) Coral Reef Watch program provides real-time bleaching forecasts based on temperature anomalies detected as the accumulation of degree heating weeks (available at **http://coralreefwatch.noaa.gov/satellite**). Building on this program, an *Acropora* white syndrome disease forecasting algorithm was developed based on the accumulation of warm

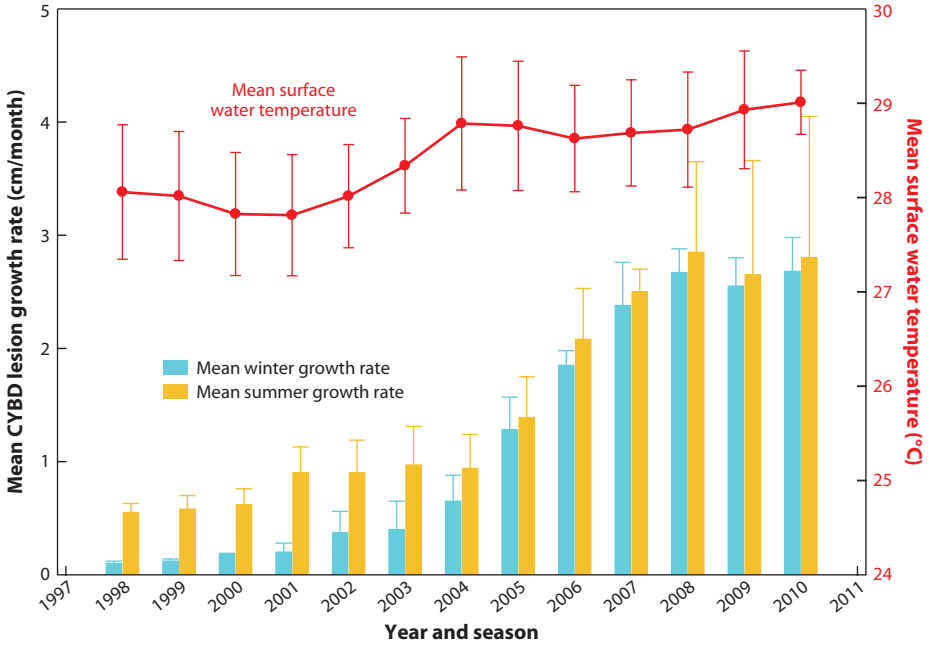



**Figure 2**

Seasonal (winter and summer) variability in mean linear growth rates of Caribbean yellow band disease (CYBD) lesions measured in tagged colonies of *Orbicella faveolata* in reefs off La Parguera, Puerto Rico, from 1998 to 2010. A positive ($r^2 = 0.82$) and significant ($p < 0.001$) correlation was found between mean lesion growth rates and mean yearly surface water temperatures.

temperature anomalies and the accumulated heat or cold stress from the preceding winter (Heron et al. 2010, Maynard et al. 2011). Other coral disease management actions that have been discussed include boosting coral immunity, cleaning off external signs of disease, performing phage therapy, and administering probiotics (summarized in Beeden et al. 2012).

## Marine Bivalve Diseases

Much is known about the influence of diseases on marine bivalve shellfish, especially those of economic concern. Examples include bonamiasis in the European flat oyster (*Ostrea edulis*) (Grizel et al. 1988), QPX (quahog parasite unknown) disease in the eastern quahog (*Mercenaria mercenaria*) (Lyons et al. 2007), MSX and *Roseovarius* oyster disease in the eastern oyster (*C. virginica*) (Ford & Tripp 1996, Boettcher et al. 2005), and Dermo disease in a variety of molluscs worldwide (Villalba et al. 2004). The best studied of the marine bivalve hosts affected by infectious disease are oysters, which experience a disproportionately high incidence of lethal disease compared with other commercial bivalves (Ford & Tripp 1996, Ford et al. 2012). Oysters are also of ecological importance owing to the services they provide via reef habitats, benthic-pelagic coupling, and water filtration and as prey for many organisms (e.g., Grabowski & Peterson 2007). Hence, in addition to being economically devastating, oyster diseases affect overall ecosystem productivity and health.

Two diseases, both caused by protozoans, severely impact eastern oyster populations in estuarine environments along the US east and Gulf of Mexico coasts (Ford & Tripp 1996). The first,

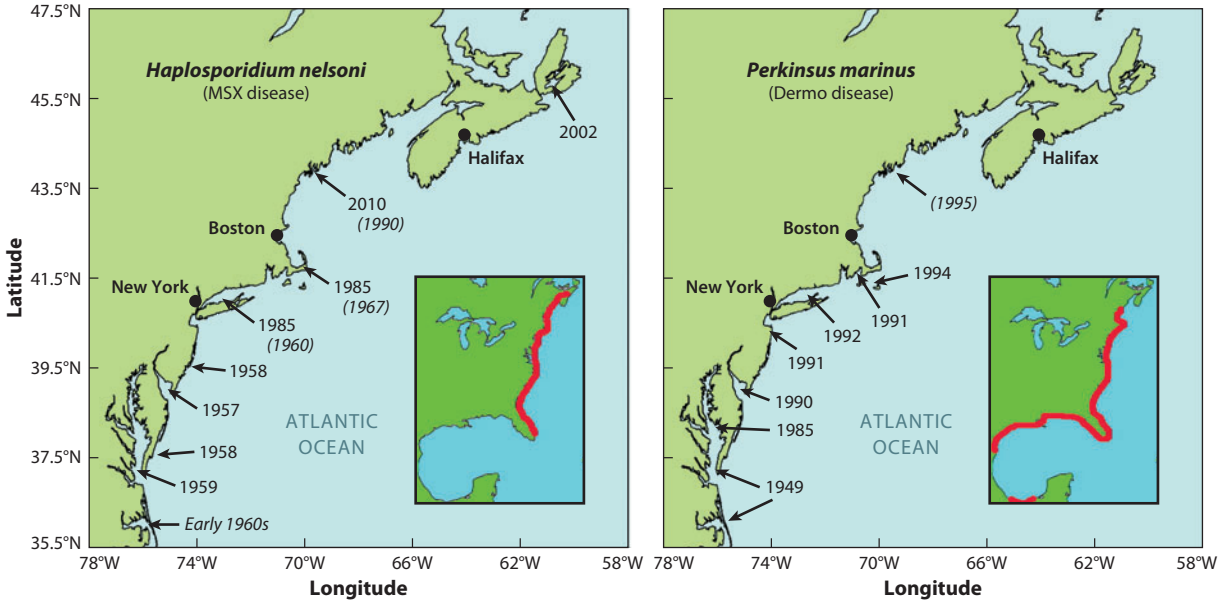



**Figure 3**

Range extension of oyster disease outbreaks in the northeastern United States and Canada. Years of first reported mortality are shown in roman type; when different, years in which the pathogen was first reported are shown in italic. No mortality has been associated with the northernmost report of *Perkinsus marinus* (in Maine, United States). The northward extension of *P. marinus* (Dermo disease) epizootics coincided with a pronounced winter warming period beginning in the mid-1980s, and range extension was especially pronounced between 1990 and 1992, when disease outbreaks occurred over a 500-km range from Delaware Bay, New Jersey, to southern Massachusetts. The period between 1989 and 1995 was also marked by consistently positive North Atlantic Oscillation anomalies. Insets show the parasites' entire ranges, including everywhere they have been reported.

Dermo disease, is caused by *Perkinsus marinus* (Mackin et al. 1950); it was identified in the late 1940s as the cause of extensive mortalities in the Gulf of Mexico, and was almost immediately found along the southeastern US coast and into lower Chesapeake Bay. In the mid-1980s, Dermo disease outbreaks intensified and spread northward (**Figure 3**), causing heavy oyster mortality as far north as Great Bay, New Hampshire. Although *P. marinus* has been found in Maine, it has not caused mortalities there (see review in Ford & Tripp 1996). The second disease, MSX disease, is caused by the protozoan *Haplosporidium nelsoni* (Haskin et al. 1966), an introduced parasite (Burreson et al. 2000). The first recorded MSX outbreak occurred in Delaware Bay, United States, in the spring of 1957 (Haskin et al. 1966), and additional devastating MSX epizootics occurred in oyster populations along the US mid-Atlantic coast and subsequently spread northward (**Figure 3**).

Temperature and salinity are known drivers of Dermo and MSX disease (Burreson & Ragone Calvo 1996, Ford & Tripp 1996); the prevalence and intensity of these diseases are subject to influence by cyclical climate patterns, such as the El Niño–Southern Oscillation (ENSO) and North Atlantic Oscillation, which modify regional and local temperature and rainfall (salinity) conditions. However, the relative influence of these climate cycles varies. Dermo disease prevalence and intensity in oyster populations along the Gulf of Mexico are affected by ENSO through its effect on salinity. The La Niña phase of ENSO produces warm, dry conditions throughout the Gulf of Mexico, which increases salinity and favors parasite proliferation (Powell et al. 1992, Soniat et al. 2005, Soniat et al. 2009). For oyster populations along the US mid-Atlantic coast, Dermo disease and subsequent mortality are influenced primarily by the warmer temperatures

during the positive phase of the North Atlantic Oscillation (Soniat et al. 2009, Bushek et al. 2012). For Dermo disease in eastern oysters, the prevalence and intensity along the Gulf of Mexico coast are regulated primarily by salinity, because the temperature is never low enough to inhibit *P. marinus* proliferation. Until the mid-1980s, temperature was considered the controlling factor along the east coast of the United States, restricting the parasite to waters of the southern United States for decades. Salinity does, however, exert local control on the parasite, limiting its spread into the upper reach of rivers and estuaries. Thus, the response of oysters, and possibly other organisms, to climate variability in a region is not an indicator of the response that occurs over the entire range of the species. The cyclic nature of climate cycles has been suggested as being responsible for the lack of development of resistance to Dermo disease in spite of high mortality rates and frequent epizootics (Powell et al. 2012).

Historically, Dermo disease has affected oysters along the US southeast and Gulf of Mexico coasts (Ford & Tripp 1996). However, since the early 1990s, the range over which Dermo disease occurs has expanded to include regions north of Chesapeake Bay along the US east coast (Cook et al. 1998, Ford & Smolowitz 2007). Several hypotheses have been put forward to explain this range expansion; an analysis of water temperatures showed that the northward expansion of Dermo disease was associated with long-term increases in winter water temperatures (Cook et al. 1998).

Similarly, MSX disease has been present in oyster populations along the US mid-Atlantic coast since the 1950s, but during the 1980s and 1990s it became established in populations further north along the US east coast (Haskin & Andrews 1988, Barber et al. 1997, Sunila et al. 1999, Burreson & Ford 2004), and it has now been found in populations in Canada (Burreson & Ford 2004). Environmental control of MSX disease by temperature and salinity is well documented (Ford et al. 1999, 2012). A modeling study of the effects of temperature and salinity variability on MSX-disease establishment indicated that warming winter temperatures facilitate the northward spread of MSX disease (Hofmann et al. 2001), and these results have been supported by observations (Burreson & Ford 2004). The link between the expansion of MSX disease and climate warming is not as clear as that for Dermo disease, as the MSX parasite has been present in the southeastern United States for decades without causing epizootics.

Variations in environmental conditions at seasonal, decadal, and longer-term scales interact to control the prevalence, intensity, and geographic distribution of the two primary diseases affecting eastern oyster populations. Recent studies have suggested that disease limits the ability to maintain oyster reefs because mortality rates are too high for reef accretion to occur over most of the estuarine salinity gradient where oysters exist, even in the absence of fishing (Mann & Powell 2007, Powell & Klinck 2007, Mann et al. 2009, Harding et al. 2010, Powell et al. 2012). Thus, increased mortality from disease has reduced the resiliency of oyster populations in the face of exploitation. Given the high prevalence and broad distribution of oyster diseases, management and control are difficult. Management of present-day oyster resources (through water management and fisheries management) must account for the effects of disease and environmental variations at a range of scales. For example, management of upstream freshwater input can help to manage downstream estuarine salinity and therefore disease-related oyster mortality (Petes et al. 2012). In addition, fisheries can apply adaptive management through techniques such as temporarily reducing harvest when high disease-related mortality occurs in order to ensure the long-term sustainability of oyster populations.

## Management of Natural and Aquaculture Abalone Stocks

Abalones, marine vetigastropods of the genus *Haliotis*, inhabit the nearshore rocky intertidal and shallow subtidal zones and are important herbivores in many ecosystems. Their sedentary nature,

gregarious distribution, accessible habitat, and economic value have resulted in overutilization. Declines in a number of the more than 50 *Haliotis* species worldwide are due in part to fishing pressure and disease (e.g., Hobday & Tegner 2000, Travers 2008). Significant alterations in abalone host–bacterial parasite dynamics in recent years are associated with increased seawater temperature. Climate change has been clearly linked to epizootics of two bacterial diseases: withering syndrome (WS) (Lafferty & Kuris 1993, Tissot 1995, Friedman et al. 2000, Moore et al. 2000, Braid et al. 2005, García-Esquivel et al. 2007) and vibriosis in adult European abalones (*Haliotis tuberculata*) (Travers et al. 2009).

WS is caused by the gastrointestinal rickettsial-like organism (RLO) "*Candidatus* Xenohaliotis californiensis" (Friedman et al. 2000) and leads to varying levels of disease among host species in the wild and in farms (Friedman et al. 1997, 2002, 2007; Moore et al. 2000, 2001, 2002; Tinajero et al. 2002; Wetchateng et al. 2010). WS was first observed in black abalone (*Haliotis cracherodii*) populations on the south shore of Santa Cruz Island in 1985, shortly after the strong 1982–1983 ENSO event (Tissot 1995, 2007). Subsequently, seasonal warm-water or ENSO events were associated with severe losses in farmed red abalones and enhanced clinical signs of disease in several wild abalone species (Moore et al. 2000; Césares-Martínez & Tinoco-Orta 2001; Friedman et al. 2002, 2003; Raimondi et al. 2002; Tinajero et al. 2002; García-Esquivel et al. 2007). Laboratory studies further confirmed a link between increased temperature ($\geq$18°C), food availability, and both transmission of the WS RLO and development of clinical disease (Moore et al. 2000, Braid et al. 2005, Vilchis et al. 2005). Because of both overfishing and WS, in 2009 the black abalone was listed as endangered under the Endangered Species Act. Both the black abalone and the endangered white abalone (*Haliotis sorenseni*) are highly susceptible to WS (Friedman et al. 1997, 2002, 2007), which jeopardizes their recovery (Moore et al. 2002).

Vibriosis in European abalones is caused by the gram-negative bacillus *Vibrio harveyi* (Gauger & Gómez-Chiarri 2002, Nicolas et al. 2002). Large losses of reproductively mature abalones have coincided with thermal maxima, and the relationship between temperature and vibriosis has been demonstrated in both laboratory trials and field studies (Travers et al. 2009). In laboratory experiments, a 1°C increase in temperature (from 17°C to 18°C) resulted in an increase in losses from 0% to 80% when abalones were exposed to the bacterium during their spawning season (Travers et al. 2009). Thus, susceptibility to this pathogen is driven by both climatic factors and reproductive physiology.

These climate-driven losses have resulted in changes in abalone community structures in intertidal and subtidal habitats, reduced recruitment of abalones, closures of abalone fisheries (e.g., Leet et al. 2001, pp. 70, 89–97; CDFG 2005), and increased costs of abalone aquaculture (e.g., Friedman et al. 2003). Rock reefs historically inhabited by substantial abalone populations may undergo changes in community structure because of the absence of abalone grazing activity, further reducing recruitment. Several approaches have been proposed to alleviate losses and their associated impacts. In the United States, California developed an Abalone Recovery and Management Plan that calls for specific actions to rebuild populations of red, pink, green, white, and black abalone (CDFG 2005). Methods to reduce or eliminate WS-RLO infections have been developed that use oral dosage of oxytetracycline (100 mg per kilogram of live body weight), but given the unusually long depletion duration (extending for many months), use of this therapeutant is likely limited to valuable brood stocks or endangered species (Friedman et al. 2003, 2007). Specific guidelines to detect and limit the transfer of marine diseases have been outlined by the World Organisation for Animal Health (**http://www.oie.int**). To limit WS in California, the state prohibited movement of infected abalone outside the known distribution of the disease (C.S. Friedman, personal observation). Methods to limit the impacts of cosmopolitan pathogens such as *V. harveyi* are more difficult to develop, but Travers (2008) suggested a link between

**Table 2 Reported ichthyophoniasis epizootics in wild fishes**

| Time period | Location | Affected fish | Impacts | Reference(s) |
|---|---|---|---|---|
| 1913–1914 | Gulf of St. Lawrence, Canada | Atlantic herring | Dead fish "in great numbers were washed ashore on beaches or sand reefs, skirting the coast, or in quiet coves" | Cox 1914 |
| 1931–1932 | Gulf of Maine | Atlantic herring | Infection prevalence reached 70% during the peak of the epizootic, then declined to 18% | Daniel 1933, Fish 1934 |
| 1940–1943 | British Isles | Mackerel | Infection prevalence was as high as 100%, and the disease was described as fatal | Sproston 1944 |
| 1947 | Gulf of Maine | Atlantic herring | No information | Scattergood 1948 |
| 1954–1956 | Gulf of Saint Lawrence, Canada | Atlantic herring | At least half of the mature herring in the western Gulf of St. Lawrence were killed | Sindermann 1956, Tibbo & Graham 1963 |
| 1966–1970 | Western North Atlantic | Yellowtail flounder | Infection prevalence was as high as 25–57%, and "there can be no question that many [affected flounder] must succumb directly to the infection" | Powles et al. 1968, Ruggieri et al. 1970 |
| 1991–1993 | Eastern North Atlantic (Denmark, Sweden, Norway, and Iceland) | Atlantic herring | Total *Ichthyophonus*-induced mortality in the North Sea was estimated at 12.8–36.8% | Rahimian & Thulin 1996, Mellergaard & Spanggaard 1997 |
| 1990s–2000s | Prince William Sound, Alaska | Pacific herring | The disease was a possible contributor to the population decline and failed recovery | Marty et al. 2010 |
| Early 2000s | Yukon River, Alaska and Canada | Chinook salmon | The disease was a possible contributor to prespawn mortality | Kocan et al. 2004 |
| 2007 | Columbia River, Washington and Oregon | American shad | Infection prevalence peaked at 72% and declined concomitantly with shad abundance | Hershberger et al. 2010 |

the pathogenicity of this bacterium and a possible plasmid; thus, limiting transfers of animals harboring the pathogen is recommended.

## Ichthyophoniasis in Marine and Anadromous Finfishes

The impacts of diseases on wild marine fishes are extremely difficult to enumerate, owing largely to the challenges of studying highly mobile organisms in large systems where direct observation is not feasible. Epizootics in marine finfishes are periodically reported, primarily in response to the appearance of massive fish kills that wash ashore or plug the nets of commercial fishers. Examples include herpesvirus disease in south Australia (Murray et al. 2003), viral hemorrhagic septicemia in the northeast Pacific (Meyers et al. 1999), and *Ichthyophonus* disease (ichthyophoniasis) throughout the Northern Hemisphere (reviewed in McVicar 2011). Of these diseases, ichthyophoniasis is arguably the most economically and ecologically significant, based on the well-documented magnitude, distribution, frequency, and effects of recurring epizootics (**Table 2**).

In addition to its population-level effects on marine fish resources (**Table 2**), ichthyophoniasis affects human societies by reducing the market value of fish. For example, epizootics in yellowtail flounder (*Limanda ferruginea*), European plaice (*Pleuronectes platessa*), alewife (*Alosa pseudoharengus*),

and mackerel (*Scomber scombrus*) from the Nova Scotian shelf resulted in unsightly and aromatic lesions in the skeletal muscles of infected fish, rendering the affected fillets unmarketable (reviewed in McVicar 2011). This clinical manifestation of the disease also affected commercial and subsistence fishers along the Yukon River (Kocan et al. 2004), leading to the culling of chinook salmon prior to human consumption, which further damaged remaining fish stocks as additional fishing effort was employed.

The linkage between ichthyophoniasis epizootics and climate change remains speculative, owing largely to issues of geographical and ecological scale as well as observational difficulties in quantifying disease impacts on wild marine fishes; however, both empirical and field observations suggest that such a linkage exists. The direct effects of temperature manipulations on ichthyophoniasis disease kinetics are well demonstrated. In *Ichthyophonus*-exposed rainbow trout, infection prevalence and cumulative mortality increase with temperature (Okamoto et al. 1987), as do the negative impacts of ichthyophoniasis on host swimming performance (Kocan et al. 2006, 2009). Field observations during the Yukon River epizootic revealed that elevated river temperatures likely contributed to disease progression (Kocan et al. 2006), and the most heavily diseased individuals were seen at the end of migration (Zuray et al. 2012).

The indirect effects of climate change are likely more important determinants of ichthyophoniasis. For example, the most parsimonious hypothesis accounting for natural routes of infection in planktivorous hosts involves consumption of an infectious-stage parasite through an intermediate host (Gregg et al. 2012); however, neither the infectious stage nor the intermediate host has been identified. Climate changes that affect ocean circulation patterns, nutrient regimes, and temperatures will likely alter zooplankton assemblages and the abundance and bloom dynamics of this proposed intermediate host.

Managing ichthyophoniasis epizootics in marine fishes involves understanding the climate-driven variables that influence the disease, including host physiological state, temperature, and natural route(s) of exposure and transmission. Ichthyophoniasis surveillance, combined with stock assessment surveys, is critical for understanding long-term infection and disease trends in affected populations. Once understood, these metrics can be developed into predictive tools to forecast the epizootics and then implemented as part of management strategies intended to mitigate disease impacts. For example, if an intermediate host for *Ichthyophonus* is identified, then its relative abundance may provide some predictive capability for future epizootics. A more pragmatic example involves options specific to the Yukon River, where water temperatures may be incorporated into adaptive fishery management strategies. Additional strategies may incorporate projected mortality from ichthyophoniasis into an effective population size. For example, management decisions for chinook salmon are based on in-season sonar counts and other indices near the mouth of the river, which are then used to designate proportions of the population for harvest and escapement in the United States and Canada. A more conservative approach to meeting escapement goals may involve allocating harvest and escapement quotas based on an effective population size that is calculated using the current population assessments minus any projected prespawn mortality from ichthyophoniasis. Interannual variability in projected prespawn mortality would likely be affected by climate-driven changes that directly and indirectly influence ichthyophoniasis.

## Noncholera *Vibrio* Diseases in Humans

Human infections caused by the marine bacteria *Vibrio vulnificus* and *Vibrio parahaemolyticus* are typically acquired through either ingestion of undercooked seafood or infection of existing wounds (Tantillo et al. 2004, Faruque & Nair 2006, Oliver 2006). Clinical signs from *Vibrio* infection can

present in as little as 7 hours, and the case fatality rate is greater than 50% (Oliver 2006). Disease can present as gastroenteritis or fulminating wound infections but can quickly progress to primary septicemia (Iida et al. 2006, Oliver 2006). Warm (20–30°C), mesohaline (<5–30‰) waters are the most hospitable for growth of pathogenic *Vibrio* bacteria (Tantillo et al. 2004). Because these bacteria are most prolific in warmer water, the vast majority of infections (~85%) occur between May and October, and infections rarely occur in regions with consistently low water temperatures (Kaysner et al. 1990, Rippey 1994, Oliver 2006). As global temperatures rise, the geographic and seasonal ranges of these bacteria may expand, along with the potential for infection as the relative numbers of *Vibrio* bacteria increase (Huq et al. 2005, Baker-Austin et al. 2010, Vezzulli et al. 2012). Storms or other extreme climatic events can further broaden the geographic range of these pathogens and lead to outbreaks of *Vibrio* disease (Lejeusne et al. 2010). Hurricanes, for example, can create storm surges that carry *Vibrio* bacteria, increasing the risk of human exposure, and can generate rain events that freshen full-strength marine waters, providing a more hospitable environment for these mesohaline bacteria. ENSO events can produce warmer conditions that potentially increase *Vibrio* abundance and can generate flooding that extends the boundaries of *Vibrio*-abundant waters (Harvell et al. 1999, Baker-Austin et al. 2010, Martinez-Urtaza et al. 2010).

Pathogenic *Vibrio* bacteria pose a significant human health risk. In the United States alone, there are approximately 4,600 cases of *Vibrio* infection each year, of which approximately 90 are *V. vulnificus* cases and 4,500 are *V. parahaemolyticus* cases (CDC 2009a,b). The consequences of climatological effects for pathogenic *Vibrio* infection risk can be either immediate (e.g., following intense storms) or long term (driven by increases in annual mean temperature and rainfall). Immediately after Hurricane Katrina struck the United States in 2005, 22 cases of *V. vulnificus* were reported, including 5 deaths (CDC 2005, Baker-Austin et al. 2010). The chronic effects of warming temperatures have been observed in northern Europe, where an unusually warm summer in 1994 coincided with the first reports of *V. vulnificus* infections in Germany along the Baltic Sea (Hoyer et al. 1995, Boer et al. 2012). Since those initial infections, cases of *Vibrio* disease have been repeatedly documented in the Baltic Sea, where water temperatures have increased markedly over the past several decades (Boer et al. 2012).

Historically, abnormally high temperatures in the Baltic Sea area coincide with unusually large numbers of reported *Vibrio* cases (Baker-Austin et al. 2012) (**Figure 4**). Furthermore, long-term sea-surface temperature increases are linked with elevated *Vibrio* infection risk. Waters warmed by approximately 0.5°C per century in the Baltic Sea from 1854 to 2010, but this rate increased to 5°C per century from 1980 to 2010 (Baker-Austin et al. 2012). Modeling efforts indicated that *Vibrio* illnesses increased by 1.93 times for every 1°C increase in annual maximum water temperature (Baker-Austin et al. 2012).

Because these infections can be acquired through ingestion as well as through wound infection, tackling the growing problem of *Vibrio* infection must also include addressing risks associated with seafood consumption. Most of the ingestion cases are the result of undercooked shellfish, such as oysters, which are often purposely consumed raw. Reducing the risk of infection requires a combination of effective seafood industry regulation and improved consumer awareness, particularly for at-risk populations (e.g., immunocompromised individuals). In the United States, *Vibrio* controls are in place that include a maximum time to refrigeration from harvest, or postharvest processing to reduce bacterial populations. Postharvest processing methods such as flash freezing and pasteurization are available for oysters and shellfish and provide safe seafood products for consumers, but these techniques kill the oysters in the process, leading many consumers to reduce their consumption of the product (Muth et al. 2000). As a newer alternative, it has been suggested that oysters be relayed to a high-salinity site for depuration of harmful *V. vulnificus* bacteria, which

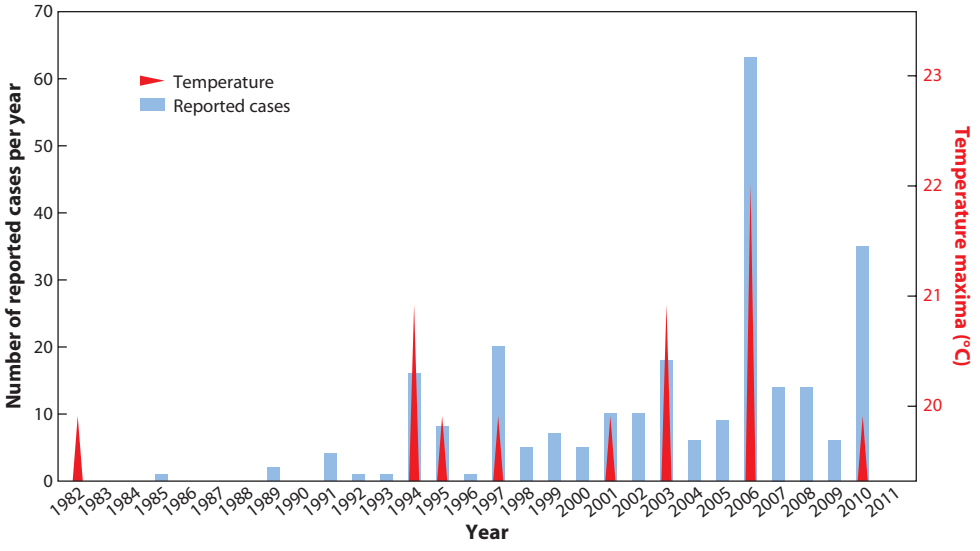



**Figure 4**

Number of reported *Vibrio* cases in the Baltic Sea area from 1982 to 2010. Figure provided by Craig Baker-Austin.

leaves the product both raw and alive (Audemard et al. 2011). Management of wound infections is also difficult, as elevated temperature is usually a driver of increased recreational water activity, exposing even more people to risk (Baker-Austin et al. 2012). Education of at-risk populations and the development of more accurate predictive models of abundance could mitigate wound-associated infections (B. Froehlich, J. Bowen, R. Gonzalez & R. Noble, manuscript in review).

## Marine Mammal Diseases

Climate change is clearly influencing infectious disease dynamics in the marine environment; however, no studies have shown a definitive causal relationship between any components of climate change and increases in infectious disease among marine mammals. This is due in large part to a lack of sufficient data and to the likely indirect nature of climate change's impact on these diseases. Climate change could potentially affect the incidence or prevalence of infection, the frequency or magnitude of epizootics, and/or the severity or presence of clinical disease in infected individuals. There are a number of potential proposed mechanisms by which this might occur. Ocean warming can change haul-out patterns (Lavigne & Schmitz 1990), thus changing pathogen transmission risk because of shifts in host density, duration at haul-outs, and/or contact with terrestrial hosts. Temperature changes can shift species' ranges, richness (Walther et al. 2002, Gilg et al. 2012), or assemblages (Benson et al. 2002), potentially changing the risk of contact between susceptible individuals and pathogen reservoirs. Climate change may shift the distribution and/or abundance of food sources, causing poor nutrition and immunosuppression (Latshaw 1991, Van Loveren et al. 2000). Changes in sea-surface temperature and salinity can alter pathogen survival (Kelly & Stroh 1988) by modifying pathogen persistence or range. Shifts in land-use or rainfall patterns can increase terrestrial pathogen flow to the coastal zone, increasing marine mammal exposure (Shapiro et al. 2010). And, finally, warming can affect pathogen evolution (Yan & Wu 2011),

potentially resulting in strains better adapted to infecting and persisting in marine mammals. However, attribution is difficult, as many effects on these diseases that could be attributed to climate change could also be attributed to changes in nonclimatic factors.

Although there is a lack of definitive evidence for the impacts of climate change on marine mammal infectious disease, insight can be gained from associations with climate variability and extreme weather and climate events. In addition, the potential effects of climate change on these diseases have been discussed for avian influenza in harbor seals (*Phoca vitulina*) (Geraci et al. 1982); *Toxoplasma gondii* in polar bears (*Ursus maritimus*), phocids, and cetaceans (Jensen et al. 2010); and *V. parahaemolyticus* in sea otters (*Enhydra lutris*) (Burek et al. 2008), among other examples. Abnormal climate events have also been associated with morbillivirus (canine distemper virus, phocine distemper virus, and cetacean morbillivirus) epizootics and mass mortalities: A 1988 phocine distemper virus outbreak in harbor seals in northern Europe was associated with unseasonably warm temperatures (LaVigne & Schmitz 1990); a 1990–1992 cetacean morbillivirus event in the western Mediterranean that affected multiple cetacean species, with the greatest impact on striped dolphins (*Stenella coeruleoalba*), was associated with high winter sea-surface temperatures, low rainfall, and reduced prey availability (Domingo et al. 1992, Aguilar & Raga 1993, Aguilar & Borrell 1994); and a 2000 canine distemper virus epizootic in Caspian seals (*Pusa caspica*) in the Caspian Sea was associated with warm temperatures and early disappearance of ice cover (Kuiken et al. 2006).

Obtaining data on the incidence and prevalence of infection in wild, free-ranging populations is logistically difficult; therefore, evaluating changes in long-term disease dynamics in marine mammal populations and identifying the role of climate change in these shifts can be quite difficult and is often impossible with the data currently available. To better understand the role of climate change in marine mammal infectious disease dynamics, improved in-depth baseline data acquired through specific and directed long-term marine health programs are needed. In addition, identifying sources of pathogen spillover can assist in mediation efforts. For example, protection and restoration of wetlands can reduce coastal contamination with pathogens that are transported in contaminated runoff, such as *T. gondii* (Shapiro et al. 2010). Vaccination, population control, and restriction of the movement of domestic dog populations in proximity to phocid haul-out sites may reduce the potential for transmission of pathogens such as canine distemper virus to susceptible marine mammals.

## FINAL THOUGHTS

### Key Knowledge Needs for Improving Understanding and Management of the Impacts of Climate Change on Marine Diseases

Although our ability to detect links between climate change/variability and marine infectious disease outbreaks has improved, the effects on most marine host-pathogen interactions are still poorly known. Knowledge gaps still exist regarding causative agents and transmission dynamics, and we need improved diagnostic methods and management strategies. Increasing outbreaks in some taxa are expected with climate change based on changes in physiological state for the host species (stressed or immunocompromised) and/or microbes (increased growth and virulence). The vulnerability and responses of marine organisms to climate change are highly variable, and certain species—including marine calcifiers, cold-adapted species, and rare, endemic, threatened, and endangered species—are particularly at risk from climate change (Howard et al. 2013), with potential implications for stress that could exacerbate sensitivity to disease. In contrast, species

with a high physiological tolerance for changes in environmental conditions will likely experience fewer climate-related effects (Howard et al. 2013), and less sensitivity could possibly translate into lower disease risk. For example, as discussed above, both tropical and temperate species (i.e., tropical corals and temperate abalones) may be highly endangered by climate-driven synergisms.

There are several reasons that more is known about certain host-pathogen-environment interactions compared with others in marine systems. Some of these systems may be particularly sensitive to climate variability and change and therefore respond strongly. Some systems may be more easily monitored (e.g., slow-moving or sessile taxa and taxa that show clear and distinguishable signs of infection) and therefore have more baseline data available. Commercial species (e.g., oysters) and those with human health consequences (e.g., *Vibrio* in seafood and water) are often well monitored owing to their direct economic and societal consequences. Some of the host-pathogen interactions described above (e.g., between abalones and WS RLO/*V. harveyi*, between oysters and Dermo/MSX disease, and between elkhorn coral and *Serratia marcescens*) could serve as sentinel host-pathogen systems, given that each of these diseases is relatively well studied and the hosts have a large latitudinal range.

In contrast, there are far fewer examples of climate-related effects on diseases in more mobile marine organisms (e.g., fish, crustaceans, and marine mammals) and in species with a low direct impact on economies and human health. Improved disease surveillance, particularly when linked with environmental/climate monitoring, is needed to improve understanding, early warning, and management of diseases in marine systems under climate change. We suggest that, for these taxa, disease detection is a problem that calls for new and better long-term health and population monitoring and forecasting. The most effective management strategy for mitigating disease involves developing adaptive disease management strategies. These strategies should be based on sound science, including long-term climatic and organismal monitoring, experimental work to test the effects of climatic stressors on host-pathogen interactions, and forecasts and decision-support tools to inform management. The sidebar (Key Science Needs for Marine Health in the Face of Climate Change) identifies several ways that we can improve our ability to manage climate-related impacts on marine diseases.

## KEY SCIENCE NEEDS FOR MARINE HEALTH IN THE FACE OF CLIMATE CHANGE

- Collect long-term data to understand host-pathogen interactions, including monitoring to understand how individual syndromes are affected by extreme events, climate variability, and climate change
- Develop sensitive and specific diagnostic tools, especially those that can be broadly applied and perhaps used in autonomous monitoring systems
- Develop new monitoring protocols to better understand the effects of disease in mobile vertebrates
- Identify the relative proportion of host-pathogen interactions affected by climate variability and climate change
- Improve understanding of the effects of climate change and ocean acidification on host-pathogen interactions across multiple life-history stages of the host (i.e., larval versus adult)
- Develop decision-support tools to improve the integration of climate-related disease considerations into marine ecosystem management (e.g., marine protected area design)
- Develop modeling tools to advance understanding and prediction of epizootics under different climate scenarios
- Enhance collaborations between natural resource managers, culturists, and researchers
- Increase public awareness of marine diseases and their consequences for people

## Challenges and Opportunities for Management of Marine Diseases Under Climate Change

Above, we discussed the impacts of climate variability and change on marine diseases and articulated potential management actions that could reduce disease risk. Management of marine diseases is difficult, as little is known about which management actions could be successfully applied in the ocean, particularly given that standard terrestrial strategies (e.g., culling, quarantining, and vaccinating) are not widely applicable. In addition to the strategies outlined in the case studies above, we suggest the following strategies to reduce climate-related disease risk:

- When possible, reduce exposure to and impacts of marine diseases by reducing nonclimatic stressors, such as coastal pollution (e.g., resulting from poor sewage management), habitat loss, translocation of pathogens, and overharvesting
- Improve forecasting programs to identify sensitive times and places for disease outbreaks, and improve monitoring and detection as a positive feedback to enhance the accuracy of forecasting programs (e.g., the integration of *Acropora* white syndrome into the Coral Reef Watch program; Maynard et al. 2011)
- Improve understanding of factors that contribute to disease resistance in marine organisms
- Encourage sharing of best practices and lessons learned, creating a community of practice among managers/culturists who control, manage, and/or respond to marine diseases

There is very little effort under way to integrate climate-related consequences for marine diseases into management approaches, such as ecosystem-based management, MPA design and implementation, and fisheries management. Factoring climatic and disease considerations into management will be key to ensuring the sustainability of ocean ecosystems, and the benefits they provide to people, for generations to come.

## DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

## ACKNOWLEDGMENTS

This work was conducted as part of the Ecology of Infectious Marine Disease Research Coordination Network (**http://www.eeb.cornell.edu/ecologymarinedisease/Home/Home.html**) funded by National Science Foundation (NSF) Ecology and Evolution of Infectious Diseases grant OCE-1215977 and by Atkinson Center for a Sustainable Future funds to C.D.H. R. Yoshioka and N. Rivlin provided technical help in developing **Figure 1** and **Table 1**. C.S.F. was funded in part by a grant from the National Sea Grant College Program, NOAA, US Department of Commerce, under project number R/FISH-208 through the California Sea Grant Program and the School of Aquatic and Fishery Sciences at the University of Washington. B.F. thanks Craig Baker-Austin for the Baltic *Vibrio* data and **Figure 4** and acknowledges support from the NSF/National Institutes of Health joint program in Ecology and Evolution of Infectious Diseases under grants OCE-0813147 and OCE-0812913. E.E.H. was supported by NSF Ecology and Evolution of Infectious Diseases grant OCE-1216220. K.C.P. acknowledges financial support from the Research and Policy for Infectious Disease Dynamics (RAPIDD) program of the US Department of Homeland Security's Science and Technology Directorate and the National Institutes of Health's Fogarty International Center. Partial funding for E.W.'s field and laboratory work has been provided by NOAA Coastal Ocean Programs grant NA17OP2919, the Global

Environment Facility/World Bank Targeted Research Program, and NSF grants OCE-1105143 and IOS-1017510; the Department of Marine Sciences at the University of Puerto Rico has also provided partial funding and logistical support. E.W. also thanks all the graduate students who have helped over the years. B.L.W. was supported by Australian Research Council (ARC) Discovery Projects funding administered through the ARC Centre of Excellence for Coral Reef Studies. S.E.F. has been funded by NOAA and the New Jersey Department of Environmental Protection (and its predecessors) as well as the US Department of Agriculture, which has supported long-term and geographically extensive data collection on oyster disease that allowed documentation of range extensions associated with climate warming. The article contents are solely the opinions of the authors and do not constitute a statement of policy, decision, or position on behalf of NOAA, the US Geological Survey, or the US government.

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Annual Review of
Marine Science

Volume 6, 2014

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**Errata**

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