

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

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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.

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

INTRODUCTION

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 (CO₂) are leading to increased global atmospheric and ocean temperatures; without significant near-term reductions in CO₂ 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 CO₂ concentrations in the atmosphere are raising CO₂ 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 (Burrenson & 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

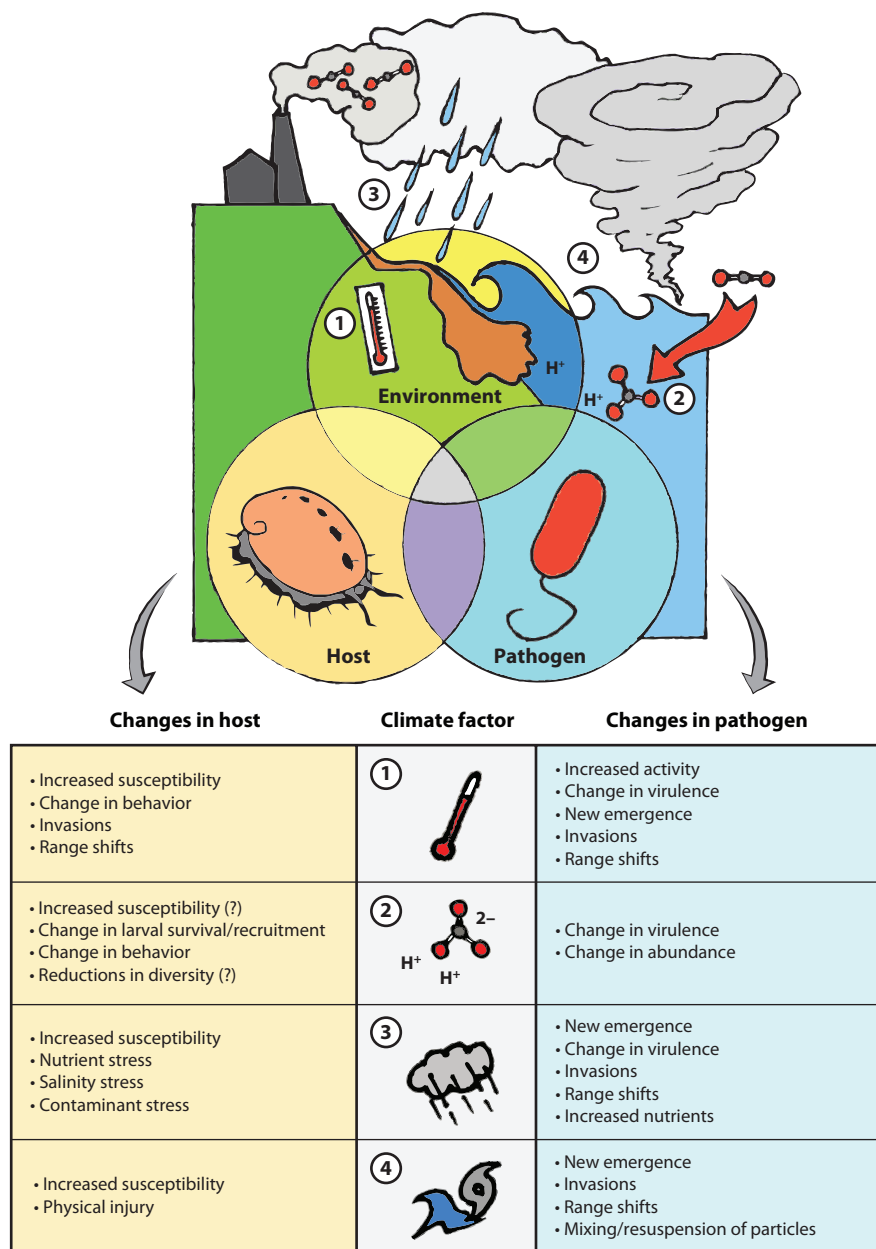


Figure 1

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	<i>Ichthyophonus</i> sp.	?	See herein; Table 2
1931–present	North America and Europe	Marine plant (seagrass)	<i>Labyrinthula</i> sp.	HT, salinity	Burge et al. 2013 (review)
1946–present	Gulf Coast and Northeast Atlantic	Bivalve (eastern oyster)	<i>Perkinsus marinus</i> (Dermo)	HT, salinity	See herein
1958–present	Northeast Atlantic	Bivalve (eastern oyster)	<i>Haplosporidium nelsoni</i> (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)	<i>Marteilia refringens</i>	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 (<i>Acropora</i> spp.)	White band disease	M-HT	Aronson & Precht 2001
1982	Central America	Octocorals	Pathogen?	HT	Guzmán & Cortés 1984
1982–1986	Australia	Gastropod (abalone)	<i>Perkinsus</i> 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)	“ <i>Candidatus</i> Xenohaliotis californiensis”	HT	See herein
1987–present	Florida	Marine plant (seagrass)	<i>Labyrinthula</i> 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 (<i>Acropora</i> 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)	<i>Aspergillus sydowii</i> 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)	<i>Mikrocystus mackini</i>	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)	<i>Vibrio harveyi</i>	HT	Travers et al. 2009
1999–2003	Mediterranean Sea	Octocorals (gorgonian)	<i>Vibrio coralliilyticus</i>	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)	<i>Roseovarius</i> 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)	<i>Vibrio tubiashii</i>	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

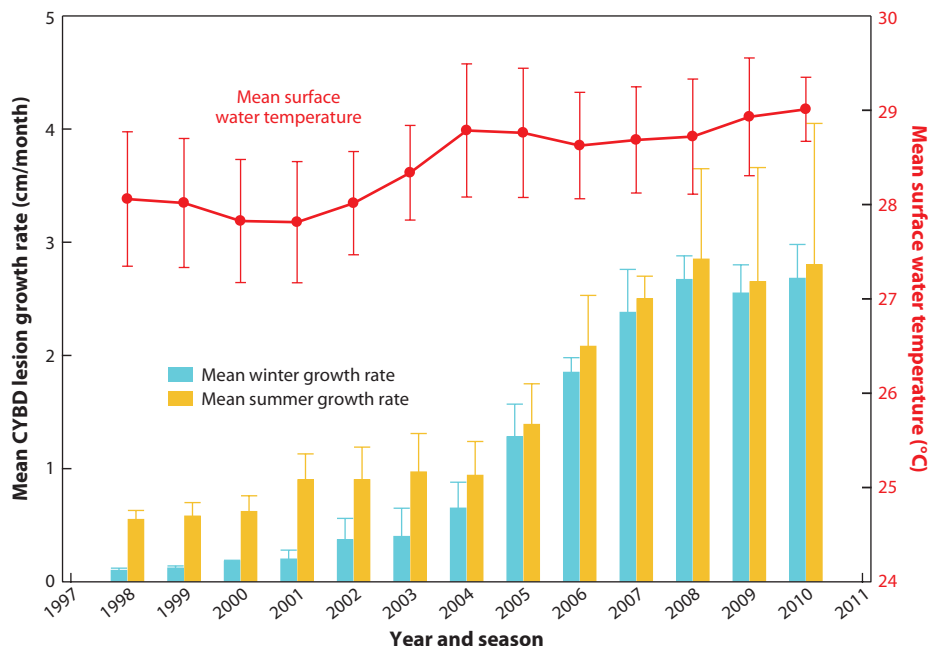


Figure 2

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,

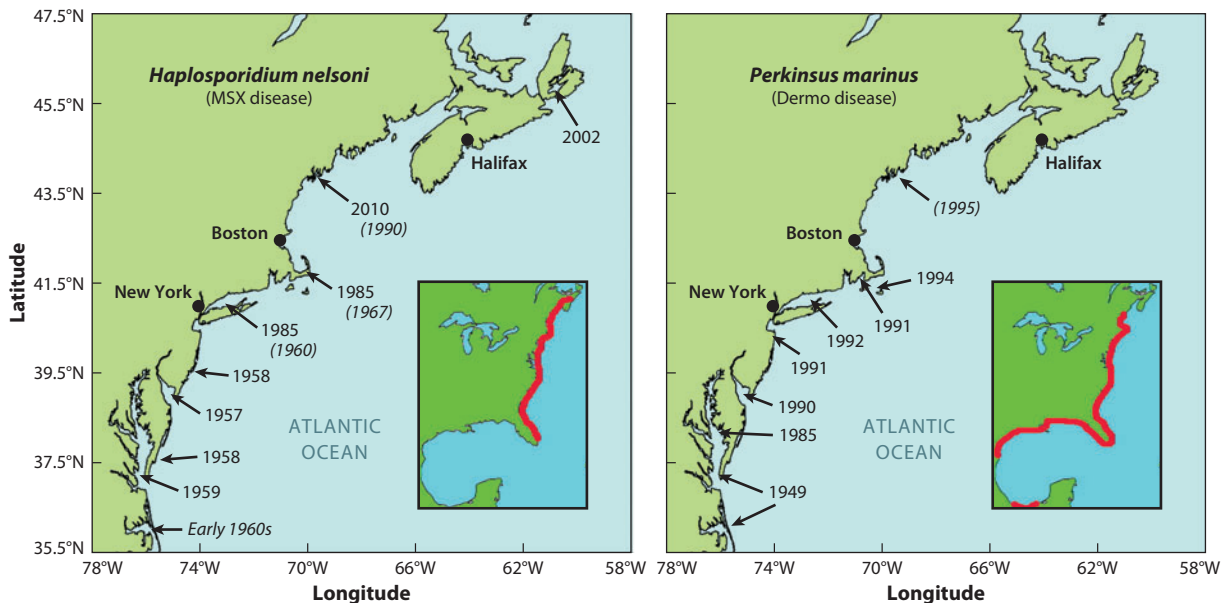


Figure 3

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 (Burrenson 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 (Burrenson & 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 (Soniati 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, Bureson & Ford 2004), and it has now been found in populations in Canada (Bureson & 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 (Bureson & 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; Césaires-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 ($\geq 18^{\circ}\text{C}$), 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 therapeutic 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 <i>Ichthyophonus</i> -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

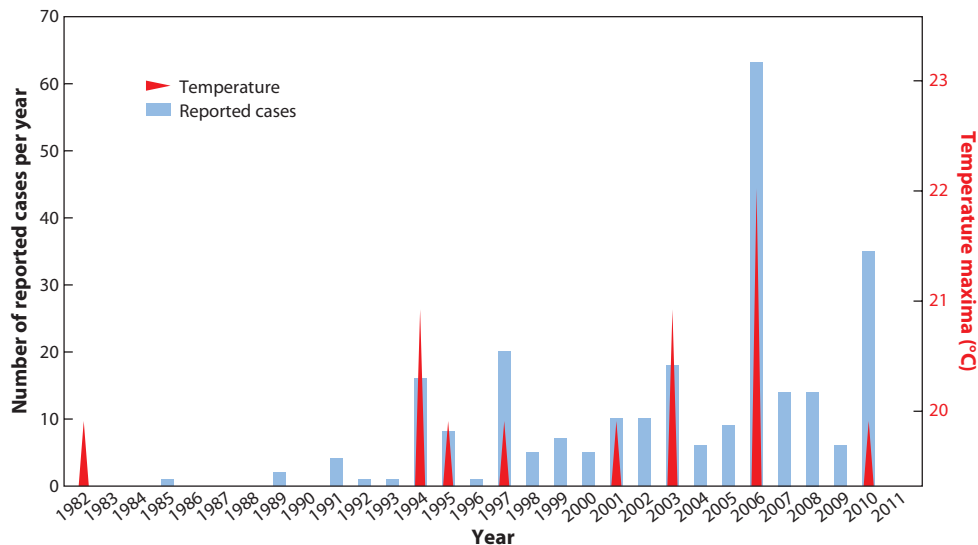


Figure 4

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. barveyi*, 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.

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LITERATURE CITED

- Aguilar A, Borrell A. 1994. Abnormally high polychlorinated biphenyl levels in striped dolphins (*Stenella coeruleoalba*) affected by the 1990–1992 Mediterranean epizootic. *Sci. Total Environ.* 154:237–47
- Aguilar A, Raga JA. 1993. The striped dolphin epizootic in the Mediterranean Sea. *Ambio* 22:524–28
- Albright R, Mason B, Miller M, Langdon C. 2010. Ocean acidification compromises recruitment success of the threatened Caribbean coral *Acropora palmata*. *Proc. Natl. Acad. Sci. USA* 107:20400–4
- Anderson PK, Cunningham AA, Patel NG, Morales FJ, Epstein PR, Daszak P. 2004. Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. *Trends Ecol. Evol.* 19:535–44
- Antonious A. 1999. *Halofolliculina corallasia*, a new coral-killing ciliate on Indo-Pacific reefs. *Coral Reefs* 18: 300

- Aronson RB, Precht WF. 2001. White-band disease and the changing face of Caribbean coral reefs. In *The Ecology and Etiology of Newly Emerging Marine Diseases*, Dev. Hydrobiol. 159, ed. JW Porter, pp. 23–35. Dordrecht: Kluwer Acad.
- Audemard C, Kator HI, Rhodes MW, Gallivan T, Erskine AJ, et al. 2011. High salinity relay as a postharvest processing strategy to reduce *Vibrio vulnificus* levels in Chesapeake Bay oysters (*Crassostrea virginica*). *J. Food Prot.* 74:1902–7
- Baker AC, Glynn PW, Riegl B. 2008. Climate change and coral reef bleaching: an ecological assessment of long-term impacts, recovery trends and future outlook. *Estuar. Coast. Shelf Sci.* 80:435–71
- Baker-Austin C, Stockley L, Rangdale R, Martinez-Urtaza J. 2010. Environmental occurrence and clinical impact of *Vibrio vulnificus* and *Vibrio parahaemolyticus*: a European perspective. *Environ. Microbiol. Rep.* 2:7–18
- Baker-Austin C, Trinanes JA, Taylor NGH, Hartnell R, Siltonen A, Martinez-Urtaza J. 2012. Emerging *Vibrio* risk at high latitudes in response to ocean warming. *Nat. Clim. Change* 3:73–77
- Bally M, Garrabou J. 2007. Thermodependent bacterial pathogens and mass mortalities in temperature benthic communities: a new case of emerging disease linked to climate change. *Glob. Change Biol.* 13:2078–88
- Barber BJ, Langan R, Howell TL. 1997. *Haplosporidium nelson* (MSX) epizootic in the Piscataqua River estuary (Maine/New Hampshire, U.S.A.). *J. Parasitol.* 83:148–50
- Barton A, Hales B, Waldbusser GG, Langdon C, Feely RA. 2012. The Pacific oyster, *Crassostrea gigas*, shows negative correlation to naturally elevated carbon dioxide levels: implications for near-term ocean acidification effects. *Limnol. Oceanogr.* 57:698–710
- Bastidas C, Bone D, Croquer A, Debrot D, Garcia E, et al. 2012. Massive hard coral loss after a severe bleaching event in 2010 at Los Roques, Venezuela. *Rev. Biol. Trop.* 60:29–37
- Beeden R, Maynard JA, Marshall PA, Heron SF, Willis BL. 2012. A framework for responding to coral disease outbreaks that facilitates adaptive management. *Environ. Manag.* 49:1–13
- Bender MA, Knutson TR, Tuleya RE, Sirutis JJ, Vecchi GA, et al. 2010. Modeled impact of anthropogenic warming on the frequency of intense Atlantic hurricanes. *Science* 327:454–58
- Benson SR, Croll DA, Marinovic BB, Chavez FP, Harvey JT. 2002. Changes in the cetacean assemblage of a coastal upwelling ecosystem during El Niño 1997–98 and La Niña 1999. *Prog. Oceanogr.* 54:279–91
- Böer S, Hauk G, Duty O, Luden K, Heinemeyer E-A, et al. 2012. Pathogenic *Vibrio* species in German coastal water of the North Sea and the Baltic Sea—a comparison. In *Pathogenic Vibrio spp. in Northern European Waters: International Symposium, 31 May–1 June 2012 in Koblenz*, pp. 36–42. Koblenz, Ger.: Bundesanstalt für Gewässerkunde
- Boettcher KJ, Geaghan KK, Maloy AP, Barber BJ. 2005. *Roseovarius crassostreae* sp. nov., a member of the *Roseobacter* clade and the apparent cause of juvenile oyster disease (JOD) in cultured Eastern oysters. *Int. J. Syst. Evol. Microbiol.* 55:1531–37
- Bowden TJ. 2008. Modulation of the immune system of fish by their environment. *Fish Shellfish Immunol.* 25:373–83
- Bower SM, Hervio D, Meyer GR. 1997. Infectivity of *Mikrocytos mackini*, the causative agent of Denman Island disease in Pacific oysters *Crassostrea gigas*, to various species of oysters. *Dis. Aquat. Org.* 29:111–16
- Braid BA, Moore JD, Robbins TT, Hedrick RP, Tjeerdema RS, Friedman CS. 2005. Health and survival of red abalone, *Haliotis rufescens*, under varying temperature, food supply, and exposure to the agent of withering syndrome. *J. Invertebr. Pathol.* 89:219–31
- Bruckner AW, Bruckner RJ. 1997. Outbreak of coral disease in Puerto Rico. *Coral Reefs* 16:260
- Bruckner AW, Hill R. 2009. Ten years of change to coral communities off Mona and Desecheo Islands, Puerto Rico, from disease and bleaching. *Dis. Aquat. Org.* 87:19–31
- Bruno JF, Selig ER. 2007. Regional decline of coral cover in the Indo-Pacific: timing, extent, and subregional comparisons. *PLoS ONE* 8:e711
- Bruno JF, Selig ER, Casey KS, Page CA, Willis BL. 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. *PLoS Biol.* 5:e124
- Burek KA, Gulland FMD, O'Hara TM. 2008. Effects of climate change on Arctic marine mammal health. *Ecol. Appl.* 18:S126–34
- Burge CA, Kim CJS, Lyles JM, Harvell CD. 2013. Special Issue Oceans and Humans Health: the ecology of marine opportunists. *Microb. Ecol.* 65:869–79

- Burreson EM, Ford SE. 2004. A review of recent information on the Haplosporidia, with special reference to *Haplosporidium nelsoni* (MSX disease). *Aquat. Living Resour.* 17:499–517
- Burreson EM, Ragone Calvo LM. 1996. Epizootiology of *Perkinsus marinus* disease of oysters in Chesapeake Bay, with emphasis on data since 1985. *J. Shellfish Res.* 15:17–34
- Burreson EM, Stokes NA, Friedman CS. 2000. Increased virulence in an introduced pathogen: *Haplosporidium nelsoni* (MSX) in the eastern oyster *Crassostrea virginica*. *J. Aquat. Anim. Health* 12:1–8
- Bushek D, Ford SE, Burt I. 2012. Long-term patterns of an estuarine pathogen along a salinity gradient. *J. Mar. Res.* 70:225–51
- Cáceres-Martínez J, Tinoco-Orta GD. 2001. Symbionts of cultured red abalone, *Haliotis rufescens* from Baja California, Mexico. *J. Shellfish Res.* 20:875–81
- Carpenter K, Abrar M, Aeby G, Aronson RB, Banks S, et al. 2008. One-third of reef-building corals face elevated extinction risk from climate change and local impacts. *Science* 321:560–63
- Castro KM, Cobb JS, Gómez-Chiarri M, Thlusty M. 2012. Epizootic shell disease in American lobsters *Homarus americanus* in southern New England: past, present and future. *Dis. Aquat. Org.* 100:149–58
- CDC (Cent. Dis. Control Prev.). 2005. *Vibrio* illnesses after Hurricane Katrina—multiple states, August–September 2005. *Morb. Mortal. Wkly. Rep.* 54:928–31
- CDC (Cent. Dis. Control Prev.). 2009a. *Vibrio parahaemolyticus*. <http://www.cdc.gov/nczved/divisions/dfbmd/diseases/vibriop/#common>
- CDC (Cent. Dis. Control Prev.). 2009b. *Vibrio vulnificus*. <http://www.cdc.gov/nczved/divisions/dfbmd/diseases/vibriov/#common>
- CDFG (Calif. Dep. Fish Game). 2005. *Abalone recovery and management plan*. Rep., CDFG, Sacramento. 363 pp.
- Chollet I, Müller-Karger FE, Heron SF, Skirving W, Mumby PJ. 2012. Seasonal and spatial heterogeneity of recent sea surface temperature trends in the Caribbean Sea and southeast Gulf of Mexico. *Mar. Pollut. Bull.* 64:956–65
- Cole RG, Babcock RC. 1996. Mass mortality of a dominant kelp (Laminariales) at Goat Island, north-eastern New Zealand. *Mar. Freshw. Res.* 47:907–11
- Cook T, Folli M, Klinck J, Ford S, Miller J. 1998. The relationship between increasing sea surface temperature and the northward spread of *Perkinsus marinus* (Dermo) disease epizootics in oysters. *Estuar. Coast. Shelf Sci.* 40:587–97
- Cooley SR, Lucey N, Kite-Powell H, Doney SC. 2012. Nutrition and income from molluscs today imply vulnerability to ocean acidification tomorrow. *Fish Fish.* 13:182–215
- Costanza R, Pérez-Maqueo O, Martinez ML, Sutton P, Anderson SJ, Mulder K. 2008. The value of coastal wetlands for hurricane protection. *Ambio* 37:241–48
- Cox PC. 1914. Investigations of a disease of the herring (*Clupea harengus*) in the Gulf of St. Lawrence, 1914. *Contrib. Can. Biol. Fish.* 1914:81–85
- Croquer A, Weil E. 2009a. Changes in Caribbean coral disease prevalence after the 2005 bleaching event. *Dis. Aquat. Org.* 87:33–43
- Croquer A, Weil E. 2009b. Local and geographic variability in distribution and prevalence of coral and octocoral diseases in the Caribbean II: genera-level analysis. *Dis. Aquat. Org.* 83:209–23
- Daniel GE. 1933. Studies on *Ichthyophonus hoferi*, a parasitic fungus of herring, *Clupea harengus*. I. The parasite as it is found in the herring. *Am. J. Hyg.* 17:262–76
- De’ath G, Fabricius K, Sweatman H, Puotinen M. 2012. The 27-year decline of coral cover on the Great Barrier Reef and its causes. *Proc. Natl. Acad. Sci. USA* 109:17995–99
- Dégremont L. 2011. Evidence of herpesvirus (OsHV-1) resistance in juvenile *Crassostrea gigas* selected for high resistance to the summer mortality phenomenon. *Aquaculture* 317:94–98
- Dionne M, Miller KM, Dodson JJ, Caron F, Bernatchez L. 2007. Clinal variation in MHC diversity with temperature: evidence for the role of host-pathogen interaction on local adaptation in Atlantic salmon. *Evolution* 61:2154–64
- Domingo M, Visa J, Pumarola M, Marco AJ, Ferrer L, et al. 1992. Pathological and immunocytochemical studies of morbillivirus infection in striped dolphins (*Stenella coeruleoalba*). *Vet. Patbol.* 29:1–10
- Doney SC, Fabry VJ, Feely RA, Kleypas JA. 2009. Ocean acidification: the other CO₂ problem. *Annu. Rev. Mar. Sci.* 1:169–92

- Doney SC, Ruckelshaus M, Duffy JE, Barry JP, Chan F, et al. 2012. Climate change impacts on marine ecosystems. *Annu. Rev. Mar. Sci.* 4:11–37
- Dungan ML, Miller TE, Thomson DA. 1982. Catastrophic decline of a top carnivore in the Gulf of California rocky intertidal zone. *Science* 216:989–91
- Eakin CM, Morgan JA, Heron SF, Smith TB, Liu G, et al. 2010. Caribbean corals in crisis: record thermal stress, bleaching, and mortality in 2005. *PLoS ONE* 5:e13969
- Elston RA, Ford S. 2011. Shellfish diseases and health management. In *Shellfish Aquaculture and the Environment*, ed. SE Shumway, pp. 359–94. Chichester, UK: Wiley & Sons
- Elston RA, Hasegawa H, Humphrey KL, Polyak IK, Hase CC. 2008. Re-emergence of *Vibrio tubiashii* in bivalve shellfish aquaculture: severity, environmental drivers, geographic extent and management. *Dis. Aquat. Org.* 82:119–34
- Fabry VJ, McClintock JB, Mathis JT, Grebeier JM. 2009. Ocean acidification at high latitudes: the bellwether. *Oceanography* 22(4):160–71
- Faruque SM, Nair GB. 2006. Epidemiology. See Thompson et al. 2006, pp. 385–98
- Feely RA, Sabine CL, Lee K, Berelson W, Kleypas J, et al. 2004. Impact of anthropogenic CO₂ on the CaCO₃ system in the oceans. *Science* 305:362–66
- Fish FF. 1934. A fungus disease in fishes of the Gulf of Maine. *Parasitology* 26:1–18
- Ford SE, Powell EN, Klinck JM, Hofmann EE. 1999. Modeling the MSX parasite in eastern oyster (*Crassostrea virginica*) populations. I. Model development, implementation and verification. *J. Shellfish Res.* 18:475–500
- Ford SE, Scarpa E, Bushek D. 2012. Spatial and temporal variability of disease refuges in an estuary: implications for the development of resistance. *J. Mar. Res.* 70:253–77
- Ford SE, Smolowitz R. 2007. Infection dynamics of an oyster parasite in its newly expanded range. *Mar. Biol.* 151:119–33
- Ford SE, Tripp MR. 1996. Disease and defense mechanisms. In *The Eastern Oyster: Crassostrea virginica*, ed. VS Kennedy, RLE Newell, AE Eble, pp. 581–660. College Park: Univ. Md. Sea Grant
- Friedman CS, Andree KB, Beauchamp K, Moore JD, Robbins TT, et al. 2000. “*Candidatus Xenohalotis californiensis*”, a newly described pathogen of abalone, *Haliotis* spp., along the west coast of North America. *Int. J. Syst. Evol. Microbiol.* 50:847–55
- Friedman CS, Biggs W, Shields JD, Hedrick RP. 2002. Transmission of withering syndrome in black abalone, *Haliotis cracherodii* Leach. *J. Shellfish Res.* 21:817–24
- Friedman CS, Scott BB, Strenge RE, McCormick TB. 2007. Oxytetracycline as a tool to manage and prevent losses of the endangered white abalone, *Haliotis sorenseni*, to withering syndrome. *J. Shellfish Res.* 26:887–85
- Friedman CS, Thomson M, Chun C, Haaker PL, Hedrick RP. 1997. Withering syndrome of the black abalone, *Haliotis cracherodii* (Leach): water temperature, food availability, and parasites as possible causes. *J. Shellfish Res.* 16:403–11
- Friedman CS, Trevelyan G, Mulder EP, Fields R. 2003. Development of an oral administration of oxytetracycline to control losses due to withering syndrome in cultured red abalone *Haliotis rufescens*. *Aquaculture* 224:1–23
- García-Esquivel Z, Montes-Magallón S, Gonzáles-Gómez MA. 2007. Effect of temperature and photoperiod on the growth, feed consumption, and biochemical content of juvenile green abalone, *Haliotis fulgens*, fed on a balanced diet. *Aquaculture* 262:129–41
- Garrett KA, Dobson ADM, Kroschel J, Natarajan B, Orlandini S, et al. 2013. The effects of climate variability and the color of weather time series on agricultural diseases and pests, and on decisions on their management. *Agric. For. Meteorol.* 170:216–27
- Gattuso JP, Hansson L, eds. 2011. *Ocean Acidification*. Oxford, UK: Oxford Univ. Press
- Gauger EJ, Gómez-Chiarri M. 2002. 16S ribosomal DNA sequencing confirms the synonymy of *Vibrio harveyi* and *V. carchariae*. *Dis. Aquat. Org.* 52:39–46
- Geraci JR, St. Aubin DJ, Barker IK, Webster RG, Hinshaw VS, et al. 1982. Mass mortality of harbor seals: pneumonia associated with influenza A virus. *Science* 215:1129–31
- Gilg O, Kovacs KM, Aars J, Fort J, Gauthier G, et al. 2012. Climate change and the ecology and evolution of Arctic vertebrates. In *Year in Ecology and Conservation Biology*, ed. RS Ostfeld, WH Schlesinger, pp. 166–90. New York: NY Acad. Sci.

- Gilman EL, Ellison J, Duke NC, Field C. 2008. Threats to mangroves from climate change and adaptation options: a review. *Aquat. Bot.* 89:237–50
- Gladfelter WB. 1982. White band disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. *Bull. Mar. Sci.* 32:639–43
- Grabowski JH, Peterson CH. 2007. Restoring oyster reefs to recover ecosystem services. In *Ecosystem Engineers: Plants to Protists*, ed. K Cuddington, JE Byers, WG Wilson, A Hastings, pp. 281–98. Amsterdam: Elsevier
- Greer A, Ng V, Fisman D. 2008. Climate change and infectious diseases in North America: the road ahead. *Can. Med. Assoc. J.* 178:715–22
- Gregg JL, Grady CA, Friedman CS, Hershberger PK. 2012. Inability to demonstrate fish-to-fish transmission of *Ichthyophonus* from laboratory-infected Pacific herring *Clupea pallasii* to naïve conspecifics. *Dis. Aquat. Org.* 99:139–44
- Grizel H, Mialhe E, Chagot D, Boulo V, Bachère E. 1988. Bonamiasis: a model study of diseases in marine molluscs. In *Disease Processes in Marine Bivalve Molluscs*, Am. Fish. Soc. Spec. Publ. 18, ed. WS Fisher, pp. 1–4. Bethesda, MD: Am. Fish. Soc.
- Guzmán HM, Cortés J. 1984. Mortandad de *Gorgonia flabellum* Linnaeus (Octocorallia: Gorgoniidae) en la costa Caribe de Costa Rica. *Rev. Biol. Trop.* 32:305–8
- Haapkylä J, Unsworth RKF, Flavell M, Bourne DG, Schaffelke B, Willis BL. 2011. Seasonal rainfall and runoff promote coral disease on an inshore reef. *PLoS ONE* 6:e16893
- Harding JM, Mann R, Southworth MJ, Wesson JA. 2010. Management of the Piankatank River, Virginia, in support of oyster (*Crassostrea virginica*, Gmelin 1791) fishery repletion. *J. Shellfish Res.* 29:867–88
- Harvell CD, Altizer S, Cattadori IM, Harrington L, Weil E. 2009. Climate change and wildlife diseases: When does the host matter the most? *Ecology* 90:912–20
- Harvell CD, Jordan-Dahlgren E, Merkel S, Rosenberg E, Raymundo L, et al. 2007. Coral disease, environmental drivers, and the balance between coral and microbial associates. *Oceanography* 20(1):172–195
- Harvell CD, Kim K, Burkholder JM, Colwell RR, Epstein PR, et al. 1999. Emerging marine diseases: climate links and anthropogenic factors. *Science* 285:1505–10
- Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, et al. 2002. Climate warming and disease risk for terrestrial and marine biota. *Science* 296:2158–62
- Haskin HH, Andrews JD. 1988. Uncertainties and speculations about the life cycle of the eastern oyster pathogen *Haplosporidium nelsoni* (MSX). In *Disease Processes in Marine Bivalve Molluscs*, Am. Fish. Soc. Spec. Publ. 18, ed. WS Fisher, pp. 5–22. Bethesda, MD: Am. Fish. Soc.
- Haskin HH, Stauber LA, Mackin JA. 1966. *Minchinia nelsoni* n. sp. (Haplosporida, Haplosporidiidae): causative agent of the Delaware Bay oyster epizootic. *Science* 153:1414–16
- Heron SF, Willis BL, Skirving WJ, Eakin CM, Page CA, Miller IR. 2010. Summer hot snaps and winter conditions: modelling white syndrome outbreaks on Great Barrier Reef corals. *PLoS ONE* 5:e12210
- Hershberger PK, van der Leeuw BK, Gregg JL, Grady CA, Lujan K, et al. 2010. Amplification and transport of an endemic fish disease by an invasive species. *Biol. Invasions* 12:3665–75
- Hobday AJ, Tegner MJ. 2000. *Status review of white abalone (Haliotis sorenseni) throughout its range in California and Mexico*. Tech. Memo. NOAA-TM-SWR-035, Natl. Mar. Fish. Serv., Natl. Ocean. Atmos. Adm., Long Beach, CA
- Hoegh-Guldberg O, Mumby PJ, Hooten AJ, Steneck RS, Greenfield P, et al. 2007. Coral reefs under rapid climate change and ocean acidification. *Science* 318:1737–42
- Hofmann EE, Ford SE, Powell EN, Klinck J. 2001. Modeling studies of the effect of climate variability on MSX disease in eastern oyster (*Crassostrea virginica*) populations. *Hydrobiologia* 460:195–212
- Hönisch B, Ridgwell A, Schmidt DN, Thomas E, Gibbs SH, et al. 2012. The geological record of ocean acidification. *Science* 335:1058–63
- Howard J, Babij E, Griffis RB, Helmuth A, Himes-Cornell P, et al. 2013. Oceans and marine resources in a changing climate. *Oceanogr. Mar. Biol.* 51:71–192
- Hoyer J, Engelmann E, Liehr RM, Distler A, Hahn H, Shimada T. 1995. Septic shock due to *Vibrio vulnificus* serogroup 04 wound infection acquired from the Baltic Sea. *Eur. J. Clin. Microbiol.* 14:1016–18
- Hughes TP. 1994. Catastrophes, phase shifts and large scale degradation of a Caribbean coral reef. *Science* 265:1547–49

- Huq A, Sack RB, Nizam A, Longini IM, Nair GB, et al. 2005. Critical factors influencing the occurrence of *Vibrio cholerae* in the environment of Bangladesh. *Appl. Environ. Microbiol.* 71:4645–54
- Iida T, Park KS, Honda T. 2006. *Vibrio parahaemolyticus*. See Thompson et al. 2006, pp. 340–48
- IPCC (Intergov. Panel Clim. Change). 2007. *Climate Change 2007: Synthesis Report: Contribution of Working Groups I, II and III to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*. Ed. RK Pachauri, A Reisinger. Geneva: IPCC
- Jensen SK, Aars J, Lydersen C, Kovacs KM, Asbakk K. 2010. The prevalence of *Toxoplasma gondii* in polar bears and their marine mammal prey: evidence for a marine transmission pathway? *Polar Biol.* 33:599–606
- Jones RJ, Bowyer J, Hoegh-Guldberg O, Blackall LL. 2004. Dynamics of a temperature-related coral disease outbreak. *Mar. Ecol. Prog. Ser.* 281:63–77
- Kaysner CA, Abeyta C Jr, Stott RF, Krane MH, Wekell MM. 1990. Enumeration of *Vibrio* species, including *V. cholerae*, from samples of an oyster growing area, Grays Harbor, Washington. *J. Food Prot.* 53:300–2
- Keller BD, Gleason DF, McLeod E, Woodley CM, Airame S, et al. 2009. Climate change, coral reef ecosystems, and management options for marine protected areas. *Environ. Manag.* 44:1069–88
- Kelly MT, Stroh EMD. 1988. Temporal relationship of *Vibrio parahaemolyticus* in patients and the environment. *J. Clin. Microbiol.* 26:1754–56
- Kocan RM, Hershberger PK, Sanders G, Winton J. 2009. Effects of temperature on disease progression and swimming stamina in *Ichthyophonus*-infected rainbow trout, *Oncorhynchus mykiss* (Walbaum). *J. Fish Dis.* 32:835–43
- Kocan RM, Hershberger PK, Winton J. 2004. Ichthyophoniasis: an emerging disease of Chinook salmon (*Oncorhynchus tshawytscha*) in the Yukon River. *J. Aquat. Anim. Health* 16:58–72
- Kocan RM, LaPatra S, Gregg J, Winton J, Hershberger PK. 2006. *Ichthyophonus*-induced cardiac damage: a mechanism for reduced swimming stamina in rainbow trout. *J. Fish Dis.* 29:521–27
- Kordas RL, Harley CDG, O'Connor MI. 2011. Community ecology in a warming world: the influence of temperature on interspecific interactions. *J. Exp. Mar. Biol. Ecol.* 400:218–26
- Kuiken T, Kennedy S, Barrett T, Van de Bildt MWG, Borgsteede FH, et al. 2006. The 2000 canine distemper epidemic in Caspian seals (*Phoca caspica*): pathology and analysis of contributory factors. *Vet. Pathol.* 43:321–38
- Kurath G, Winton J. 2011. Complex dynamics at the interface between wild and domestic viruses of finfish. *Curr. Opin. Virol.* 1:73–80
- Lafferty KD, Kuris AM. 1993. Mass mortality of abalone *Haliotis cracherodii* on the California Channel Islands: tests of epidemiologic hypotheses. *Mar. Ecol. Prog. Ser.* 96:239–48
- Lafferty KD, Porter JW, Ford SE. 2004. Are diseases increasing in the ocean? *Annu. Rev. Ecol. Evol. Syst.* 35:31–54
- Latshaw DJ. 1991. Nutrition: mechanisms of immunosuppression. *Vet. Immunol. Immunopathol.* 30:111–20
- Lauzon-Guay JS, Scheibling RE, Barbeau MA. 2009. Modelling phase shifts in a rocky subtidal ecosystem. *Mar. Ecol. Prog. Ser.* 375:25–39
- Lavigne DM, Schmitz OJ. 1990. Global warming and increasing population densities: a prescription for seal plagues. *Mar. Pollut. Bull.* 21:280–84
- Leet WS, Dewees CM, Klingbeil R, Larson E, eds. 2001. *California's living marine resources: a status report*. Rep., Calif. Dep. Fish Game, Sacramento
- Lejeusne CP, Chevaldonné, Pergent-Martini C, Boudouresque CF, Pérez T. 2010. Climate change effects on a miniature ocean: the highly diverse, highly impacted Mediterranean Sea. *Trends Ecol. Evol.* 25:250–60
- Lessios HA, Robertson DR, Cubit JD. 1984. Spread of *Diadema* mass mortality throughout the Caribbean. *Science* 226:335–37
- Lyons MM, Smolowitz R, Gómez-Chiarri M, Ward JE. 2007. Epizootiology of quahog parasite unknown (QPX) disease in northern quahogs (=hard clams) *Mercenaria mercenaria*. *J. Shellfish Res.* 26:371–81
- Mackin JG, Owen HM, Collier A. 1950. Preliminary note on the occurrence of a new protistan parasite, *Dermocystidium marinum* n. sp. in *Crassostrea virginica* (Gmelin). *Science* 111:328–29
- Mann R, Harding JM, Southworth MJ. 2009. Reconstructing pre-colonial oyster demographics in the Chesapeake Bay, USA. *Estuar. Coast. Shelf Sci.* 85:217–22
- Mann R, Powell EN. 2007. Why oyster restoration goals in the Chesapeake Bay are not and probably cannot be achieved. *J. Shellfish Res.* 26:905–17

- Martenot CE, Oden E, Travaille JP, Malas A, Houssin M. 2011. Detection of different variants of Ostreid Herpesvirus 1 in the Pacific oyster, *Crassostrea gigas* between 2008 and 2010. *Virus Res.* 160:25–31
- Martinez-Urtaza J, Bowers JC, Trinanes J, DePaola A. 2010. Climate anomalies and the increasing risk of *Vibrio parahaemolyticus* and *Vibrio vulnificus* illnesses. *Food Res. Int.* 43:1780–90
- Marty GD, Hulson PJ, Miller SE, Quinn TJ II, Moffitt ST, Merizon RA. 2010. Failure of population recovery in relation to disease in Pacific herring. *Dis. Aquat. Org.* 90:1–14
- Maynard JA, Anthony KRN, Harvell CD, Burgman MA, Beeden R, et al. 2011. Predicting outbreaks of a climate-driven coral disease in the Great Barrier Reef. *Coral Reefs* 30:485–95
- McClanahan TR, Weil E, Cortés J, Baird A, Ateweberhan M. 2009. Consequences of coral bleaching for sessile organisms. In *Coral Bleaching: Patterns, Processes, Causes and Consequences*, ed. M van Oppen, J Lough, pp. 121–38. Berlin: Springer-Verlag
- McVicar AH. 2011. *Ichthyophonus*. In *Fish Diseases and Disorders*, Vol. 3: *Viral, Bacterial, and Fungal Infections*, ed. PTK Woo, DW Bruno, pp. 721–47. Cambridge, MA: CAB Int. 2nd ed.
- Møllergaard S, Spanggaard B. 1997. An *Ichthyophonus hoferi* epizootic in herring in the North Sea, the Skagerrak, the Kattegat, and the Baltic Sea. *Dis. Aquat. Org.* 28:191–99
- Meyers TR, Short S, Lipson K. 1999. Isolation of the North American strain of viral hemorrhagic septicemia virus (VHSV) associated with epizootic mortality in two new host species of Alaskan marine fish. *Dis. Aquat. Org.* 38:81–86
- Miller J, Muller A, Rogers CS, Waara JR, Atkinson A, et al. 2009. Coral disease following massive bleaching in 2005 causes 60% decline in coral cover on reefs in the US Virgin Islands. *Coral Reefs* 28:925–37
- Moore JD, Finley CA, Robbins TT, Friedman CS. 2002. Withering syndrome and restoration of southern California abalone populations. *Calif. Coop. Fish. Investig. Rep.* 43:112–17
- Moore JD, Robbins TT, Friedman CS. 2000. Withering syndrome in farmed red abalone, *Haliotis rufescens*: thermal induction and association with a gastrointestinal Rickettsiales-like procaryote. *J. Aquat. Anim. Health* 12:26–34
- Moore JD, Robbins TT, Hedrick RP, Friedman CS. 2001. Transmission of the Rickettsiales-like procaryote “*Candidatus Xenohaliotis californiensis*” and its role in withering syndrome of California abalone *Haliotis* spp. *J. Shellfish Res.* 20:867–74
- Murray AG, O’Callaghan M, Jones B. 2003. A model of spatially evolving herpesvirus epidemics causing mass mortality in Australian pilchard *Sardinops sagax*. *Dis. Aquat. Org.* 54:1–14
- Muth M, Anderson D, Karns S, Murray B, Domanico J. 2000. *Economic impacts of requiring post-harvest treatment of oysters*. Rep., Cent. Econ. Res., Res. Triangle Inst., Res. Triangle Park, NC
- Negri AP, Marshall PA, Heyward AJ. 2007. Differing effects of thermal stress on coral fertilization and early embryogenesis of four Indo Pacific species. *Coral Reefs* 26:759–63
- Nicolas JL, Basuyaux O, Mazurie J, Thebault A. 2002. *Vibrio carchariae*, a pathogen of the abalone *Haliotis tuberculata*. *Dis. Aquat. Org.* 50:35–43
- Nye J, Link JS, Hare JA, Overholtz WJ. 2009. Changing spatial distributions of northwest Atlantic fish stocks in relation to temperature and stock size. *Mar. Ecol. Prog. Ser.* 393:111–29
- O’Hara TM, Holcomb D, Elzer P, Estepp J, Perry Q, et al. 2010. *Brucella* species survey in polar bears (*Ursus maritimus*) of northern Alaska. *J. Wildl. Dis.* 46:687–94
- Okamoto N, Nakase K, Sano T. 1987. Relationships between water temperature, fish size, infective dose and *Ichthyophonus* infection of rainbow trout. *Bull. Jpn. Soc. Sci. Fish.* 53:581–84
- Oliver JD. 2006. *Vibrio vulnificus*. See Thompson et al. 2006, pp. 349–66
- Page CA, Baker DA, Harvell CD, Golbuu Y, Raymundo L, et al. 2009. Influence of marine reserves on coral disease prevalence. *Dis. Aquat. Org.* 87:135–50
- Parmesan C, Yohe G. 2003. A globally coherent fingerprint of climate change impacts across natural systems. *Nature* 421:37–42
- Patterson KL, Porter JW, Ritchie KE, Polson SW, Mueller E, et al. 2002. The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acorpora palmata*. *Proc. Natl. Acad. Sci. USA* 99:8725–30
- Petes LE, Brown AJ, Knight CR. 2012. Impacts of upstream drought and water withdrawals on the health and survival of downstream estuarine oyster populations. *Ecol. Evol.* 2:1712–24

- Powell EN, Gauthier JD, Wilson EA, Nelson A, Fay RR, Brooks JM. 1992. Oyster disease and climate change: Are yearly changes in *Perkinsus marinus* parasitism in oysters (*Crassostrea virginica*) controlled by climatic cycles in the Gulf of Mexico? *Mar. Ecol.* 13:243–70
- Powell EN, Klinck JM. 2007. Is oyster shell a sustainable estuarine resource? *J. Shellfish Res.* 26:181–94
- Powell EN, Klinck JM, Ashton-Alcox K, Hofmann EE, Morson JM. 2012. The rise and fall of *Crassostrea virginica* oyster reefs: the role of disease and fishing in their demise and a vignette on their management. *J. Mar. Res.* 70:505–58
- Powles PM, Garnett DG, Ruggieri GD, Nigrelli RF. 1968. *Ichthyophonus* infection in yellowtail flounder (*Limanda ferruginea*) off Nova Scotia. *J. Fish. Res. Board Can.* 25:597–98
- Rahimian H, Thulin J. 1996. Epizootiology of *Ichthyophonus hoferi* in herring populations off the Swedish west coast. *Dis. Aquat. Org.* 27:187–95
- Raimondi PT, Wilson CM, Ambrose RF, Engle JM, Minchinton TE. 2002. Continued declines of black abalone along the coast of California: Are mass mortalities related to El Niño events? *Mar. Ecol. Prog. Ser.* 242:143–45
- Raymundo LJH, Halford AR, Maypa AP, Kerr AM. 2009. Functionally diverse reef-fish communities ameliorate coral disease. *Proc. Natl. Acad. Sci. USA* 106:17067–70
- Raymundo LJH, Harvell CD, Reynolds TL. 2003. Porites ulcerative white spot disease: description, prevalence, and host range of a new coral disease affecting Indo-Pacific reefs. *Dis. Aquat. Org.* 56:95–104
- Richardson LL, Goldberg WM, Carlton RG, Halas JC. 1998. Coral disease outbreak in the Florida Keys: plague type II. *Rev. Biol. Trop.* 46:187–89
- Rippey SR. 1994. Infectious diseases associated with molluscan shellfish consumption. *Clin. Microbiol. Rev.* 7:419–25
- Robblee MB, Barber TR, Carlson PR, Durako MJ Jr, Fourqurean JW, et al. 1991. Mass mortality of the tropical seagrass *Thalassia testudinum* in Florida Bay (USA). *Mar. Ecol. Prog. Ser.* 71:297–99
- Rogers CS, Muller EM. 2012. Bleaching, disease and recovery in the threatened scleractinian coral *Acropora palmata* in St. John, US Virgin Islands: 2003–2010. *Coral Reefs* 31:807–19
- Ruckelshaus M, Doney SC, Galindo HM, Barry JP, Chan F, et al. 2013. Securing marine ecosystem services in the face of climate change. *Mar. Policy* 40:154–59
- Ruetzler K, Santavy D, Antonius A. 1983. The black band disease of Atlantic reef corals. III. Distribution ecology and development. *Mar. Ecol.* 4:329–58
- Ruggieri GD, Nigrelli RF, Powles PM, Garnett DG. 1970. Epizootics in yellowtail flounder, *Limanda ferruginea* Storer, in the western North Atlantic caused by *Ichthyophonus*, an ubiquitous parasitic fungus. *Zoologica* 55:57–62
- Ruiz-Moreno D, Willis BL, Page AC, Weil E, Croquer A, et al. 2012. Global coral disease prevalence associated with sea temperature anomalies and local factors. *Dis. Aquat. Org.* 100:249–61
- Sato Y, Bourne DG, Willis BL. 2009. Dynamics of seasonal outbreaks of black band disease in an assemblage of *Montipora* species at Pelorus Island (Great Barrier Reef, Australia). *Proc. R. Soc. Lond. B* 276:2795–803
- Sato Y, Bourne DG, Willis BL. 2011. Effects of temperature and light on the progression of black band disease on the reef coral *Montipora hispida*. *Coral Reefs* 30:753–61
- Scattergood LW. 1948. *A report on the appearance of the fungus Ichthyosporidium hoferi in the herring of the northwestern Atlantic*. Spec. Sci. Rep., US Fish Wildl. Serv., Washington, DC. 33 pp.
- Selig ER, Bruno JF. 2010. A global analysis of the effectiveness of marine protected areas in preventing coral loss. *PLoS ONE* 5:e9278
- Shapiro K, Conrad PA, Mazet JAK, Wallender WW, Miller WA, Largier JL. 2010. Effect of estuarine wetland degradation on transport of *Toxoplasma gondii* surrogates from land to sea. *Appl. Environ. Microbiol.* 76:6821–28
- Sindermann CJ. 1956. *Diseases of fishes of the western North Atlantic. IV. Fungus disease and resultant mortalities of herring in the Gulf of Saint Lawrence in 1955*. Res. Bull. 25, Maine Dep. Sea Shore Fish., Augusta
- Soniati TM, Hofmann EE, Klinck JM, Powell EN. 2009. Differential modulation of eastern oyster (*Crassostrea virginica*) disease parasites by the El-Niño-Southern Oscillation and the North Atlantic Oscillation. *Int. J. Earth Sci.* 98:99–114
- Soniati TM, Klinck JM, Powell EN, Hofmann EE. 2005. Understanding the success and failure of oyster populations: climatic cycles and *Perkinsus marinus*. *J. Shellfish Res.* 24:83–93

- Sproston NG. 1944. *Ichthyosporidium boferi* (Plehn & Mulsow, 1911), an internal fungoid parasite of the mackerel. *J. Mar. Biol. Assoc. UK* 26:72–78
- Staudinger MD, Grimm NB, Staudt A, Carter SL, Chapin FS III, et al. 2012. *Impacts of climate change on biodiversity, ecosystems, and ecosystem services: technical input to the 2013 National Climate Assessment*. Coop. Rep. 2013 Natl. Clim. Assess., Washington, DC. 296 pp. <http://downloads.usgcrp.gov/NCA/Activities/Biodiversity-Ecosystems-and-Ecosystem-Services-Technical-Input.pdf>
- Stentiford GD, Neil DM, Peeler EJ, Shields JD, Small HJ, et al. 2012. Disease will limit future food supply from the global crustacean fishery and aquaculture sectors. *J. Invertebr. Pathol.* 110:141–57
- Sunila I, Karolus J, Volk J. 1999. A new epizootic of *Haplosporidium nelsoni* (MSX), a haplosporidian oyster parasite, in Long Island Sound, Connecticut. *J. Shellfish Res.* 18:169–74
- Sutherland KP, Porter JW, Truner JW, Thomas BJ, Loney EE, et al. 2010. Human sewage identified as likely source of white pox disease of the threatened Caribbean elkhorn coral, *Acropora palmata*. *Environ. Microbiol.* 12:1122–31
- Szmant AM, Gassman NJ. 1990. The effects of prolonged “bleaching” on the tissue biomass and reproduction of the reef coral *Montastraea annularis*. *Coral Reefs* 8:217–24
- Tantillo GM, Fontanarosa M, Di Pinto A, Musti M. 2004. Updated perspectives on emerging vibrios associated with human infections. *Lett. Appl. Microbiol.* 39:117–26
- Thompson FL, Austin B, Swings J, eds. 2006. *The Biology of Vibrios*. Washington, DC: ASM Press
- Tibbo SN, Graham TR. 1963. Biological changes in herring stocks following an epizootic. *J. Fish. Res. Board Can.* 20:435–49
- Tinajero MDC, Cáceres-Martínez J, Avilés JGG. 2002. Histopathological evaluation of the yellow abalone *Haliotis corrugata* and the blue abalone *Haliotis fulgens* from Baja California, Mexico. *J. Shellfish Res.* 21:825–30
- Tissot BN. 1995. Recruitment, growth, and survivorship of black abalone on Santa Cruz Island following mass mortality. *Bull. South. Calif. Acad. Sci.* 94:179–89
- Tissot BN. 2007. *Long-term population trends in the black abalone, Haliotis cracherodii, along the eastern Pacific coast*. Unpublished Rep. Off. Prot. Resour., Southwest Reg., Natl. Mar. Fish. Serv., Long Beach, CA. 43 pp.
- Travers M-A. 2008. *Interaction of the bacterium Vibrio harveyi with its host the abalone Haliotis tuberculata: physiological, cellular and molecular approaches*. PhD thesis. Univ. Bretagne Occidentale–Cent. Natl. Rech. Sci., Brest, France. 272 pp.
- Travers M-A, Basuyaux O, LeGoic N, Huchette S, Nicolas J-L, et al. 2009. Influence of temperature and spawning effort on *Haliotis tuberculata* mortalities caused by *Vibrio harveyi*: an example of emerging vibriosis linked to global warming. *Glob. Change Biol.* 15:1365–76
- Van Loveren H, Ross PS, Osterhaus ADME, Vos JG. 2000. Contaminant-induced immunosuppression and mass mortalities among harbor seals. *Toxicol. Lett.* 112:319–24
- Vezzulli L, Brettar I, Pezzati E, Reid PC, Colwell RR, et al. 2012. Long-term effects of ocean warming on the prokaryotic community: evidence from the vibrios. *ISME J.* 6:21–30
- Vilchis LI, Tegner MJ, Moore JD, Friedman CS, Riser KL, et al. 2005. Ocean warming effects on growth, reproduction, and survivorship of southern California abalone. *Ecol. Appl.* 15:469–80
- Villalba A, Reece KS, Camino Ordás M, Casas SM, Figueras A. 2004. Perkinsosis in molluscs: a review. *Aquat. Living Resour.* 17:411–32
- Voss JD, Richardson LL. 2006. Nutrient enrichment enhances black band disease progression in corals. *Coral Reefs* 25:569–76
- Walther G-R, Post E, Convey P, Menzel A, Parmesan C, et al. 2002. Ecological responses to recent climate change. *Nature* 416:389–95
- Weil E, Croquer A, Urreiztieta I. 2009a. Temporal variability and consequences of coral diseases and bleaching in La Parguera, Puerto Rico from 2003–2007. *Caribb. J. Sci.* 45:221–46
- Weil E, Croquer A, Urreiztieta I. 2009b. Yellow band disease compromises the reproductive output of the reef-building coral *Montastraea faveolata* (Anthozoa, Scleractinia). *Dis. Aquat. Org.* 87:45–55
- Weil E, Torres JL, Ashton M. 2005. Population characteristics of the sea urchin *Diadema antillarum* in La Parguera, Puerto Rico, 17 years after the mass mortality event. *Rev. Biol. Trop.* 53:219–31

- Wetchateng T, Friedman CS, Wight NA, Lee P-Y, Teng PH, et al. 2010. Withering syndrome in abalone *Haliotis diversicolor supertexta*. *Dis. Aquat. Org.* 90:69–76
- Willis BL, Page CA, Dinsdale EA. 2004. Coral disease on the Great Barrier Reef. In *Coral Health and Disease*, ed. E Rosenberg, Y Loya, pp. 69–104. Berlin: Springer
- Yan SM, Wu GA. 2011. Possible impact of global warming on the evolution of hemagglutinins from influenza A viruses. *Biomed. Environ. Sci.* 24:62–67
- Zuray S, Kocan R, Hershberger P. 2012. Synchronous cycling of ichthyophoniasis with Chinook salmon density revealed during the annual Yukon River spawning migration. *Trans. Am. Fish. Soc.* 141:615–23

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Errata

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