ARE DISEASES INCREASING IN THE OCEAN?[*]

Kevin D. Lafferty,[1] James W. Porter,[2] and Susan E. Ford[3]

[1]U.S. Geological Survey, Western Ecological Research Center, c/o Marine Science Institute, University of California, Santa Barbara, California 93106; email: Lafferty@lifesci.ucsb.edu
[2]Institute of Ecology, University of Georgia, Athens, Georgia 30602; email: jporter@uga.edu
[3]Haskin Shellfish Research Laboratory, Rutgers University, Port Norris, New Jersey 08349; email: susan@hsrl.rutgers.edu

■ **Abstract** Many factors (climate warming, pollution, harvesting, introduced species) can contribute to disease outbreaks in marine life. Concomitant increases in each of these makes it difficult to attribute recent changes in disease occurrence or severity to any one factor. For example, the increase in disease of Caribbean coral is postulated to be a result of climate change and introduction of terrestrial pathogens. Indirect evidence exists that (*a*) warming increased disease in turtles; (*b*) protection, pollution, and terrestrial pathogens increased mammal disease; (*c*) aquaculture increased disease in mollusks; and (*d*) release from overfished predators increased sea urchin disease. In contrast, fishing and pollution may have reduced disease in fishes. In other taxa (e.g., sea grasses, crustaceans, sharks), there is little evidence that disease has changed over time. The diversity of patterns suggests there are many ways that environmental change can interact with disease in the ocean.

INTRODUCTION

The perception of an ecological crisis in the oceans has led to research on signs of deterioration in ocean health. Recent mass mortalities in marine systems, including Caribbean sea urchins (Lessios 1988), phocine distemper virus (Heide-Jorgensen et al. 1992), pilchard mortalities (Jones et al. 1997), and especially coral bleaching (Hoegh-Guldberg 1999), have consequently received the attention and concern of marine ecologists (e.g., Goreau et al. 1998, Greenstein et al. 1998, Hayes & Goreau 1998, Hoegh-Guldberg 1999, Porter et al. 2001, Wilkinson 2002). The scale of these events and their impact on populations and associated communities have

---

[*]The U.S. Government has the right to retain a nonexclusive, royalty-free license in and to any copyright covering this paper.

been dramatic in some cases. Some species, such as black abalone in California (Lafferty & Kuris 1993), sun stars in the Sea of Cortez (Dungan et al. 1982), staghorn coral (Diaz-Soltero 1999), and long-spined sea urchin in the Caribbean (Lessios 1988), were reduced to such low population densities by infectious disease that recovery is in question. That these events seem unprecedented has led to the hypothesis that disease outbreaks in marine organisms have increased in recent years (Epstein 1996, Harvell et al. 1999, Hayes et al. 2001, Williams & Bunkley-Williams 1990). Here, we review the little systematic evidence available on changes in marine disease and outline factors that can increase (or decrease) disease in the ocean.

Coral diseases have received increasing attention as an explanation for the recent decline in coral reefs and this notable situation provides a fitting start for our review. Wilkinson (2002) estimates that 27% of reefs worldwide have already been lost, with an additional 16% at serious risk. Between 1996 and 2001, coral reefs of the Florida Keys lost 37% of their living coral (Porter et al. 2002), and some species, such as the massive, branching elkhorn coral (*Acropora palmata*), have declined by 91% (Patterson et al. 2002). Surveys (Connell 1997, Dustan 1999, Porter et al. 2002, Porter & Meier 1992) reveal that disease is a primary cause of this coral reef decline (Aronson & Precht 1998, Gladfelter 1982, Kim & Harvell 2004). Elevated sea surface temperature from El Niño events is a common explanation for coral bleaching (Hoegh-Guldberg 1999, Williams & Bunkley-Williams 1990), and such a rise in temperature could increase coral susceptibility to infectious diseases (Harvell et al. 2001). Diseases have had a major impact on the biodiversity of coral reefs (Chadwick-Furman 1996), eliminating rare species and severely reducing the abundance of common ones (Porter et al. 2001). For example, species within the branching acroporidae reproduce almost exclusively by fragmentation rather than by sexual reproduction (Aronson & Precht 2001, Knowlton 1992). Although this reproductive strategy works well after storms, it is ineffective after epizootics that destroy all the living tissue (Patterson et al. 2002). *Acropora palmata*, the most common coral in the Caribbean, experienced such rapid and widespread losses that it has been proposed for inclusion on the Endangered Species List (Diaz-Soltero 1999). The species susceptible to the greatest number of diseases are the following massive reef building corals: *Montastraea annularis* (nine diseases), *Colpophyllia natans* (eight diseases), *M. faveolata* (six diseases), *Acropora palmata* (six diseases), *M. frankski* (five diseases), *M. cavernosa* (five diseases), and *Diploria labyrinthiformis* (five diseases). Loss of massive reef builders has obvious large-scale effects on coral reef ecosystems.

In this paper, we assess whether there is direct or indirect evidence for a change in disease over time. We then consider difficulties in making simple predictions about stress and disease. We discuss how increasing anthropogenic factors, namely climate warming, pollution, changes in density (through fishing and conservation), and pathogen pollution, could affect disease in the ocean. We conclude with a case study of increasing disease in oysters.

EVIDENCE FOR CHANGE

Determining whether disease is changing over time is problematic because the lack of baseline data for most marine communities precludes a direct test (Ward & Lafferty 2004). In this section, we consider some of the empirical evidence as well as suggestive patterns from reviews of the literature.

Direct Observations of Disease

Systematically collected data for changes in disease prevalence or severity are rare compared with lists of recent, dramatic events. Proof that change has occurred in any complex system requires baseline data from before a disease event that can be compared with data collected during and postepizootic. If monitoring programs track disease, they can reveal trends in disease over time.

We reanalyzed data on sea urchins (from the Channel Islands in southern California) from Lafferty (2004) to track changes over 20 years in the percent of stations where bacterial disease was observed (Figure 1). Over the course of the study, urchin disease significantly increased with time ($R = 0.87$, $N = 20$, $P < 0.0001$). This is because disease was absent for the first decade of the study and,
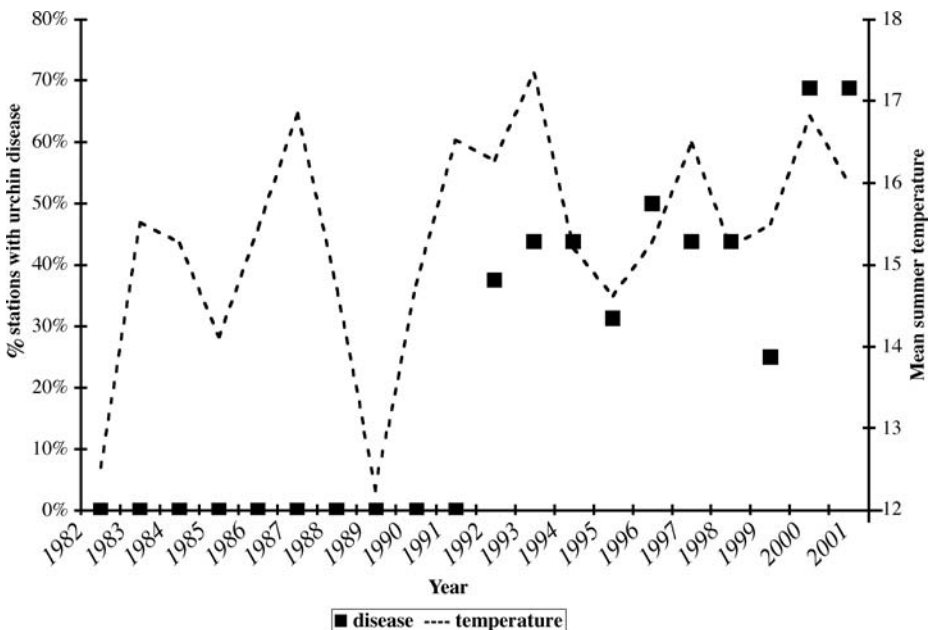
Figure 1 The percentage of 16 Channel Islands Kelp Forest monitoring stations with disease in sea urchins. Disease increased significantly over 20 years, but not evidently because of warming (Lafferty 2004).

after appearing dramatically in 1992, had a slight, but insignificant, increase (R = 0.5, N = 10, P = 0.14). Variation in the percent of stations with disease (following the arrival of the disease) was not significantly correlated with summer temperatures (R = 0.4, N = 10, P = 0.27). These results support an emerging pathogen scenario, but not one of increasing disease because of warming. As we describe below, fishing lobsters appears to be the main factor explaining disease in this system.

The Coral Reef Monitoring Project was established in the Florida Keys National Marine Sanctuary in 1996 (Aronson & Precht 1998, Gladfelter 1982, Porter et al. 2001). Between 1996 and 2003, there were significant increases in the number of stations containing diseased coral (Figure 2), the number of species with disease, and the number of different types of disease. These data show definitively that (a) coral disease increased throughout the Florida Keys over a six-year period and (b) these increases were not due to observational inadequacies. Although the short time series does not necessarily allow the detection of a long-term trend, the most dramatic increases in disease fell on the heels of the 1997–1998 El Niño event, suggesting a link between disease and climate. Between 1996 and 1998, the number of coral species with disease in the Florida Keys survey increased from 11 to 36 (85% of all species are now affected by disease, a 218% increase over 1996 values) (Porter et al. 2001).
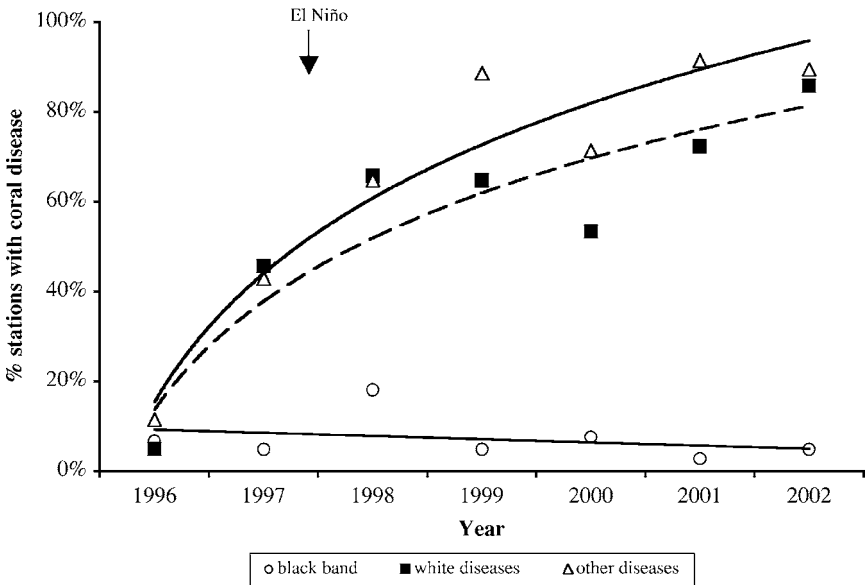

Figure 2   The percentage of the Florida Keys Coral Reef Monitoring Project stations with disease increased significantly between 1996 and 2002, with 1997–1998 El Niño noted. These data come from 105 randomly chosen stations that were censused annually by uniform survey techniques.

Patterns in the Literature

Because few marine studies systematically track disease over time, an indirect approach has been to determine if reports of disease in the scientific literature have increased over time. In one of the first efforts, Epstein (1996) and Epstein et al. (1998) plotted reports of various marine events over time. Many of these events were disease related, but they also included other mass mortalities (e.g., harmful algal blooms). Most increased in frequency from 1970 to the late 1990s. The authors suggest this corresponds to climatic changes, most notably higher frequencies of high sea-surface temperature. Subsequently, Harvell et al. (1999) published a table of examples of marine diseases over time as evidence consistent with the hypothesis that the rate of disease events had increased, but noted that the apparent increase could be an artifact of increased detection ability. For example, the advent of molecular techniques has improved diagnostics for viruses and other pathogens difficult to assay by traditional means. Harvell et al. (1999) also listed environmental correlates associated with each event, noting that high temperature was often a factor in marine disease, and suggested a role for pollution in diseases of marine mammals. They further recognized that many of the diseases appeared novel, resulting from host shifts, range shifts, or species introductions. Hayes et al. (2001) statistically analyzed the selected examples from the Harvell et al. (1999) and Epstein (1996) studies and found a significant increase over time in both the variety of disease organisms and the absolute number of episodes of disease (though it is questionable whether statistical analysis of selected examples is appropriate). Hayes et al. (2001) also noted that the timing of a climate regime shift in the mid-1970s corresponded with the increase in reports of disease. They further postulated that changes in atmospheric circulation resulted in an increased deposition of iron-rich dust that they hypothesized could favor the growth of opportunistic pathogens. Predictions of future increases in disease in the ocean owing to climate change lend new urgency to the desire to understand causes and trends of marine disease outbreaks (Harvell et al. 2002).

Combining trends among host taxa may obscure trends specific to a particular group. In an effort to determine if coral diseases have increased over time, we show reports for the number of coral disease agents described over time. Because this is a cumulative list, it will increase or stay the same from year to year. It is the shape of the increase that informs whether disease is increasing or decreasing. Figure 3 shows an exponential increase in the number of described coral diseases from 1965 to 2003 (Sutherland et al. 2004). Although it is impossible to determine if the exponential increase is due to an increase in observational intensity or to real increases in the number of diseases, many of the most recently described diseases, such as white pox (Patterson et al. 2002), have such clear manifestations that it is unlikely they were historically common. Fortunately, corals leave an excellent fossil record. This has allowed researchers to determine that the recent shift of dominant corals (from *Acropora* to *Agaricia*) on reefs owing to white band disease was unprecedented in the last 3000 years (Aronson et al. 2002).
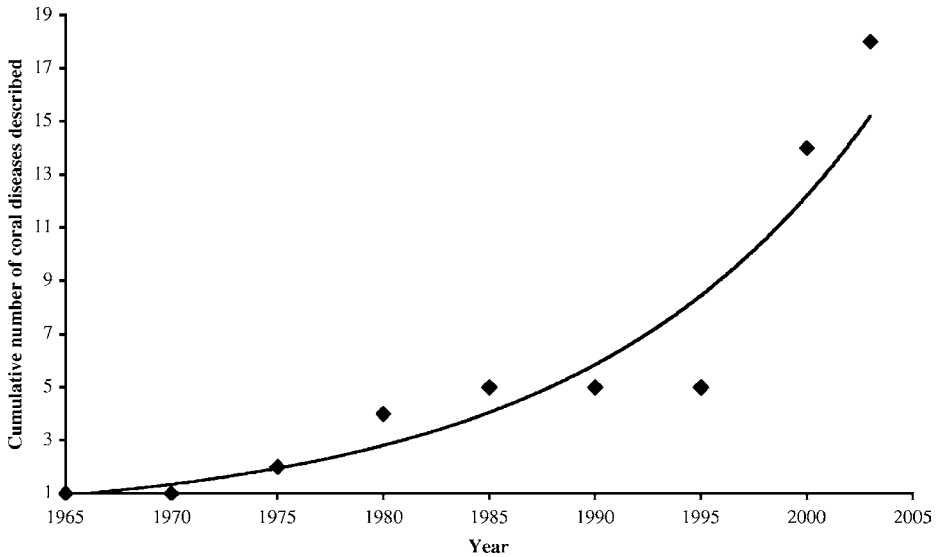
Figure 3 The cumulative increase in described coral diseases shows an exponential rise since 1965 (Sutherland et al. 2004).

Ward & Lafferty (2004) directly addressed the possibility that the increase in reports of disease could be a simple artifact of burgeoning publication rates. By systematically searching a literature database, they acquired a data set on disease reports over time that was more representative and comprehensive than previous studies. They found that the absolute number of studies of disease had increased in all taxa. However, they determined that this increase was, at least in part, a consequence of the overall growth of the scientific literature and was driven by the vast number of studies on fishes. To account for the underlying bias in publication rates, they normalized reports of disease as a proportion of all reports (i.e., reports on disease divided by all reports). To distinguish trends among taxa, they did this separately for nine marine taxonomic groups. Several patterns emerged when these normalized reports were analyzed for temporal trends between 1970 and 2001. The proportion of studies on disease increased significantly with time in turtles, corals, mammals, sea urchins, and mollusks. A closer inspection of the coral data indicated that reports of noninfectious coral bleaching increased significantly over the past three decades, but reports of infectious coral disease have not. However, reports of coral disease were high following the two large El Niño events (Figure 4). No significant trends were detected for sea grasses, decapods, or sharks/rays (though disease occurred in these groups). Surprisingly, the normalized reports of disease decreased in fishes. The finding that disease did not increase in all taxa indicates that increases were not exclusively the result of increased study or detection ability of disease by marine biologists. The finding that disease reports increased in most taxa is strong support for the hypothesis that there has been a real increase in some
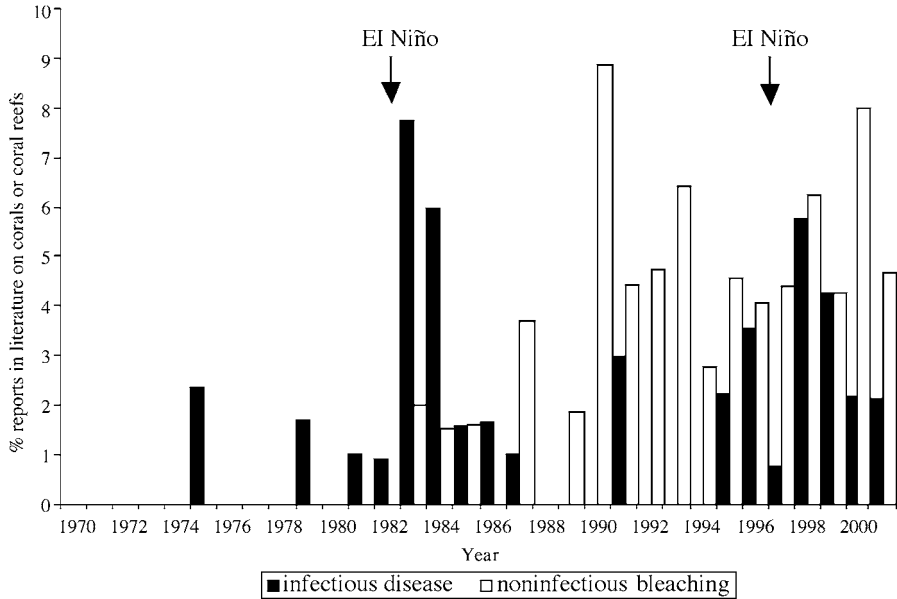
Figure 4   The percentage of the scientific literature on corals or coral reefs that was related to disease. Data are from Ward & Lafferty (2004). Over time, there is a significant overall increase in disease reports as well as a significant increase for bleaching. Infectious coral disease does not show a significant trend over time but instead shows a bimodal pattern coincident with the two large El Niño events of the last three decades.

diseases. Below, we consider what factors might be associated with increases or decreases in disease.

PROBLEMS WITH SIMPLE PREDICTIONS

Several anthropogenic stressors are changing in concert: Climate warming may lead to thermal stress; coastal development leads to habitat destruction and degradation; introduced species bring disease; fishing reduces once common species and favors others; runoff and discharge contain nutrients and pathogens along with fertilizers, pesticides, antibiotics, anthelminthics, and herbicides. This list of potential stressors is matched by a diverse set of infectious and noninfectious diseases, most of which are poorly understood. In this review, we focus primarily on infectious diseases, and such infectious diseases clearly have always been common in natural communities (Dobson et al. 1992, Kennedy et al. 1986). Moreover, although infectious diseases are usually perceived as negative, some play positive roles in promoting biodiversity and maintaining ecosystems (Lafferty 2003). A vast diversity of parasitic strategies naturally occur in the ocean, including typical parasites (e.g., intestinal tapeworms), pathogens (e.g., many viruses, fungi,

and protozoa), parasitoids (e.g., hyperiid amphipods), parasitic castrators (e.g., rhizocephalan barnacles), trophically transmitted parasites (e.g., larval acanthocephalans), and, by some considerations, micropredators (e.g., leeches) (Lafferty & Kuris 2002). Some (perhaps most) of these infectious agents have no detectable effect on host populations, whereas others cause mortality, reduce growth, lower fecundity, alter behavior, or lower social status. Furthermore, different parasites and pathogens might interact differently with environmental change. Given the diversity of interactions between environmental disturbance and infectious disease, we cannot overly generalize about whether diseases should increase in association with environmental degradation. Disease may increase or decrease, depending on the infectious agent and the factors affecting its host population (Lafferty 1997).

EFFECTS OF ANTHROPOGENIC CHANGE ON DISEASE

The foremost assumption related to the hypothesis that disease is increasing in marine environments is that increasing stressors, such as toxic chemicals (Khan 1990), pollutants (Harvell et al. 1999), and warming (Harvell et al. 2002), increase individual susceptibility to infectious diseases. Under this view, Porter & Tougas (2001) proposed a general coral disease model that integrates stress factors from climate change predictions and pollution. Global factors include predicted rise in ocean temperature (Hoegh-Guldberg 1999), storm frequency (Birkeland 1997), and oceanic carbon dioxide concentrations (Kleypas et al. 1999). Pollution factors include elevated nutrients (Lapointe et al. 2002), elevated toxicants (Glynn et al. 1984), and reduced water clarity (Cook et al. 2002). This model predicts that deteriorating environmental conditions will be expressed by increases in the number of places and number of species simultaneously experiencing epizootics. These predictions match current patterns of disease outbreaks in the Florida Keys (Porter et al. 2001).

Predictions become complicated, however, because some stressors can have a more negative impact on parasites than on hosts (Lafferty 1997). This will increase recovery rates of infected individuals and mitigate the population-level impacts of disease. Infected hosts might also experience differentially high mortality when under stress. This will remove parasites more rapidly from the host population than would occur without the stressor. Although this increases the impact of disease on infected individuals, it simultaneously decreases the spread of an epizootic through the host population. Such a relationship underscores the point that effects of stress and infectious disease at the population level cannot necessarily be predicted from their effects on individuals (Lafferty & Holt 2003).

Other predictions emerge if one considers how contact between individuals affects the dynamics of infectious disease. High host density increases contact rates between infected and uninfected individuals (Anderson & May 1986, Stiven 1964). For this reason, dense populations tend to have more parasites (Arneberg et al. 1998), meaning that some epizootics could be due to increasing host density,

not to outside stressors. In contrast, if stress impairs host vital rates (birth, death, growth), this will depress host population density, thus reducing the chance of an epizootic. Again, this is the opposite of the prediction that stress increases disease.

Perhaps the main benefit of explicitly listing these hypotheses is that it acknowledges how anthropogenic effects can decrease disease as well as increase disease. In addition, none of the above hypotheses are exclusive. Thus, a particular stress can have multiple effects on hosts and parasites, such as increasing host susceptibility to disease while impairing host vital rates. This can make it difficult to predict how a particular stressor will affect disease in a host population. For example, although stressed individuals should be more susceptible to infection if exposed, the stressor could also reduce contact rates between infected and uninfected individuals. Simulation models help resolve the opposing predictions stemming from these alternative effects (Lafferty & Holt 2003). Although many interactions between stress and disease are theoretically possible, stress seems most likely to reduce the impact of infectious diseases as long as transmission is closed (infections tend to be transmitted between members of the same population) and host specificity is high. Stress will generally increase the impact of noninfectious diseases or infectious diseases that are not host specific or that maintain themselves in large (often relatively resistant) reservoir populations (Lafferty & Holt 2003).

Climate Warming

Predictions for coastal marine areas include changes in air and sea temperatures, ocean currents, atmospheric storms, freshwater inputs from land, and sea levels. Of these, the most notable prediction is for widespread increases in average sea surface temperatures driven by elevated greenhouse gases (Hoegh-Guldberg 1999, Houghton et al. 1996, Kleypas et al. 1999). The ocean has already increased in temperature, on average, a third of a degree in the last half century (Levitus et al. 2000). This aspect of climate change should alter the geographic distribution of infectious disease by shifting host and parasite latitudinal ranges poleward (Marcogliese 2001a).

Higher temperatures may stress organisms, increasing their susceptibility to disease (Holmes 1996, Kim et al. 2000, Scott 1988), but an increase in susceptibility will depend on the relative sensitivity of hosts and parasites to temperature. Hosts and their parasites exhibit a peak performance at a thermal optimum (Harvell et al. 2002). If the optimum differs between host and parasite, the resulting gene-by-gene-by-environment interaction will either increase or decrease susceptibility to disease at a given temperature (Elliot et al. 2002). Tropical marine organisms are naturally much closer to their upper lethal temperature than to their lower lethal temperature, and this may put them at a disadvantage in fighting disease if temperature increases (Porter et al. 1989). For example, the optimal temperature of a fungal pathogen is higher than the optimal temperature of its sea fan host, placing the sea fan at risk to climate warming (Alker et al. 2001). One aspect of warming is universally stressful for marine invertebrates and fishes. As temperature rises,

oxygen levels decrease and metabolic rates increase, suggesting that warming could lead to respiratory stress.

Some parasites have higher growth rates (Chubb 1980) (and, subsequently, higher pathology), decreased generation times (Pojmanska et al. 1980), or higher reproductive output (Chubb 1979) at warmer temperatures, suggesting that the severity of disease could increase with temperature. This may be why withering syndrome in black abalone results in faster die-offs in warm water (Lafferty & Kuris 1993). Climate warming may also favor disease in sea turtles. Green turtle fibropapilloma tumors are hypothesized to grow more rapidly in warm water and the prevalence of this disease has increased since the 1980s (Herbst 1994). Reports of diseases of sea turtles have increased greatly over the last three decades (Ward & Lafferty 2004), consistent with the evidence that warming favors this disease.

Although several stressors can produce bleaching in corals, the vast majority of bleaching is caused by elevated water temperature (Brown 1997). This may explain why reports of noninfectious coral bleaching have significantly increased in the last three decades (Ward & Lafferty 2004). Several other coral diseases, such as aspergillosis (Alker et al. 2001), black band (Carlton & Richardson 1995, Edmunds 1991, Rutzler et al. 1983), white pox (Patterson et al. 2002), and dark spot disease (Gil-Agudelo & Garzon-Ferreira 2001), also grow more rapidly under elevated temperature. Because the growth rate and spread of many coral diseases increase during the late summer period, one of the likeliest outcomes of global warming is for the summer disease season to become (*a*) more severe, as summer temperature maxima increase; and (*b*) longer, as these elevated thermal regimes start earlier and persist later in the season. Also, winter warming may release some infectious diseases from the low-temperature control that provides hosts with a seasonal escape from disease (Harvell et al. 2002). Nevertheless, disease will not always increase with warming. For example, aptly named cold-water disease in salmonids disappears as water temperature increases (Holt et al. 1989).

Pollution

Toxic pollution affects both parasites and hosts, making it difficult to generalize broadly about its effects on disease (Lafferty 1997). In general, toxicants increase an individual's susceptibility to disease by impairing defenses. For example, the intensity and prevalence of parasitic gill ciliates and monogenes of fishes increase with a wide range of pollutants (Khan & Thulin 1991) because toxicants impair mucus production, a fish's main defense against gill parasites (Khan 1990). Similarly, marine mammals bioaccumulate lipophillic toxicants (O'Shea 1999) that can affect the mammalian immune system (Swart et al. 1994) by lowering killer cell activity, responses to T and B cell mitogens, and antibody responses (DeStewart et al. 1996). Such immunosuppression may increase pathological conditions in seals exposed to morbillivirus (Van Loveren et al. 2000), Phocine Distemper (Harder et al. 1992), leptospirosis, and calicivirus (Gilmartin et al. 1976).

This suggests that individuals exposed to infectious disease are more likely to become infected or to suffer more from disease when in toxic environments.

Most helminth parasites, however, decline in polluted areas (Lafferty 1997). This is because, in addition to impacting host defenses, toxicants increase parasite mortality rates, thereby reducing parasitic disease (Lafferty 1997). For example, selenium is more toxic to tapeworms than to fish hosts (Riggs et al. 1987). Also, free-swimming trematode larvae are sensitive to heavy metals (Siddall & Clers 1994), as are their mollusk first intermediate hosts. This is probably why reduced trematode diversity occurs at sites contaminated with heavy metals (Lefcort et al. 2002). Toxic pollution, therefore, should impede trematode transmission to vertebrates that serve as final hosts. Toxic pollution can also negatively affect parasites if infected hosts are differentially killed by pollution (Guth et al. 1977, Stadnichenko et al. 1995). For instance, cadmium kills amphipods infected with larval acanthocephalans more readily than it kills uninfected amphipods (Brown & Pascoe 1989).

Changes in Population Density

Anthropogenic change can affect the density of marine organisms in several ways, and such changes in host density affect infectious disease transmission. Toxicants can reduce host density by reducing host vital rates (Johnson 2001). Therefore, even if toxicants make uninfected individuals more susceptible to infectious disease, the contact between uninfected and infected individuals may be sufficiently low in a polluted environment that infectious disease cannot persist in the host population.

Habitat degradation and human disturbance may drive some species, particularly top predators, from coastal areas. Take, for instance, the assumption that a diverse and abundant trematode community in snails is impossible without a diverse and abundant final host community. This is because final hosts are the source of trematode stages infectious to snails, and final hosts vary in the type of adult trematode communities they harbor. Trematode infections in snails should be higher at locations where final host birds are abundant (Bustnes et al. 2000, Hoff 1941, Marcogliese 2001b, Robson & Williams 1970, Smith 2001). A salt marsh restoration project provided an opportunity to test the hypothesis that habitat degradation decreases parasitism of snails by trematodes (Huspeni & Lafferty 2004). Before restoration, snails at impacted sites had significantly fewer larval trematodes (lower prevalence and species richness) than did snails at control sites located in intact salt marsh habitat. After restoration, the impacted sites increased in trematode prevalence and species richness, eventually surpassing controls. This is because birds prefer areas with diverse and abundant prey populations. This example indicates how some parasites may actually be positive indicators of healthy ecosystems.

Fishing has dramatically reduced the abundance of many species (Jackson et al. 2001, Myers & Worm 2003). If a stock is fished to a density below the host density threshold for transmission, a fishery can fish out parasites (Dobson & May 1987). For instance, fishing substantially reduced the prevalence of a tapeworm in

whitefish (Amundsen & Kristoffersen 1990), apparently extirpated a swim bladder nematode from native trout in the Great Lakes (Black 1983), and dramatically reduced the prevalence of bucephalid trematodes in scallops (Sanders 1966). Fishing out a parasite is most likely when the parasite has a recruitment system that is relatively closed compared with the recruitment of its host (Kuris & Lafferty 1992). The significant decline in the normalized reports of disease in marine fish (Ward & Lafferty 2004) was based almost entirely on data from commercially fished species. This suggests the hypothesis that exploitation has reduced diseases in fishes by making transmission more difficult. In contrast, aquaculture intentionally increases species densities, and this should favor diseases (as detailed in our case study on oysters below).

Fishing and hunting can indirectly increase diseases in species at lower levels of the food web (Hochachka & Dhondt 2000, Jackson et al. 2001). At the California Channel Islands, extirpation of sea otters (and removal of Native American hunter-gatherers) facilitated an increase in black abalone populations to great abundance. Under these conditions, a previously unknown rickettsial disease caused a catastrophic collapse of black abalone populations (Lafferty & Kuris 1993). In this same area, lobsters and sea otters historically kept sea urchin populations at low levels, and kelp forests developed in a community-level trophic cascade (Tegner & Levin 1983). Where lobsters were fished (nearly everywhere but in marine reserves), urchin populations increased and often overgrazed kelps. An urchin-specific bacterial disease recently entered the area where urchin densities well exceeded the host-threshold density for transmission; epizootics followed, except in a marine reserve where lobsters were protected (Lafferty 2004). Similarly, in the 1980s, an unknown pathogen led to a 98% die-off of the long-spined sea urchin, *Diadema antillarum*, throughout the Caribbean (Lessios 1988). Fishing of sea urchin predators and the subsequent increase in sea urchin populations is common (Babcock et al. 1999, Pinnegar et al. 2000, Sala et al. 1998, Shears & Babcock 2002). This suggests that increased reports of disease in echinoderms (Ward & Lafferty 2004) may be more an indirect result of fishing top predators than of climate stress. Indeed, fishing, rather than climate change, may be the major cause of increased diseases in marine organisms at lower trophic levels (Jackson et al. 2001). Protection from fishing/hunting can also increase the density of exploited species and facilitate disease transmission. For example, protected pinniped populations have soared in some areas (Stewart & Yochem 2000). The combination of increased susceptibility owing to stressors and increased population density owing to successful marine mammal protection regulations may explain why reports of disease in marine mammals have increased (Ward & Lafferty 2004).

Eutrophication is a common consequence of coastal development. However, unlike toxic pollution (with which it is often associated), eutrophication may raise rates of parasitism because the associated increased productivity can increase the abundance of hosts and, subsequently, parasites. In addition, some coral pathogens can directly use nutrients to increase their growth and pathogenicity (Bruno et al.

2003). Parasites that increase under eutrophic conditions tend to be host generalists and have local recruitment, such as cestodes with short life cycles, or, like trematodes, have intermediate hosts, such as snails, that benefit from enrichment (Marcogliese 2001a).

Pathogen Pollution

Pathogen pollution, or the introduction of new disease-causing agents, is unequivocally increasing disease in the ocean and elsewhere. Global trade and travel are increasingly introducing species, particularly to estuarine habitats, where introduced species can make up the bulk of the fauna (Ruiz et al. 2000). Averaging across several taxa, introduced animals bring a mean of 16% of their parasite species when they invade (Torchin et al. 2003). Although most parasites of introduced species are left behind, a fraction of those that do invade can have severe consequences. This is best illustrated by the fact that when epizootics decimate formerly common species, the source of the disease is usually a new pathogen (Lafferty & Gerber 2002). Many successful introduced pathogens have broad host specificity and are more pathogenic in naive hosts than in their original, abundant (exotic) hosts (Gog et al. 2002, McCallum & Dobson 1995, Woodroffe 1999). When the Caspian stellate sturgeon invaded the Aral Sea, it brought a monogene that infected gills of the native spiny sturgeon, leading to mass mortalities of this naive host (Dogiel & Lutta 1937). Similarly, when European trout were introduced to North America, whirling disease spread from stocked trout to native trout, with severe consequences for the natives (Bergersen & Anderson 1997, Gilbert & Granath 2003). A similar type of pathogen pollution occurs where domestic animals create a large source of disease. Such disease reservoirs can cause rare species to decline (Lafferty & Gerber 2002). This can theoretically occur in marine systems when mariculture operations maintain a continual source of disease transmission to closely related native species. One possible example is in the rapidly expanding shrimp farming industry in which several viruses of marine penaeid shrimp have spread through farms and wild populations (Lightner 1996). Other, better-documented cases are sea lice, *Lepeophtheirus salmonis*, and the bacterial agent of furunculosis, *Aeromonas salmonicida*, which can escape from salmon farms into wild stocks.

Unusual contact events between typically segregated species have the potential to spread disease to new host species. Canine distemper virus (CDV) and related morbilliviruses, i.e., phocine distemper virus (PDV), have recently emerged in a number of marine hosts. This may be a viral host shift from domestic dogs to marine mammals. For example, a 1955 mass mortality of Antarctic crab-eater seals was attributed to CDV introduced by sled dogs (Bengston & Boveng 1991). In 1987–1988, PDV emerged in European harbor seals (Osterhaus & Vedder 1988), perhaps from contact with infected harp seals or terrestrial canids (Heide-Jorgensen & Harkonen 1992), causing ~60% mortality in most regions (Heide-Jorgensen & Harkonen 1992, Osterhaus & Vedder 1988).

Another aspect of pathogen pollution involves infective agents, "pollutogens," that have a source outside the ecosystem. For instance, *Aspergillus sydowii* is a common terrestrial fungus that may enter the marine environment via local sediment runoff (Smith et al. 1996) or by long-distance transport in African dust (Shinn et al. 2000). In the marine environment, this is a widespread pathogen of sea fans throughout the Caribbean (Kim & Harvell 2002, Kim et al. 2000). Pollutogens may reproduce within a host and elicit defensive responses but have little or no infectious dynamic within the host population (Lafferty & Kuris 2004). Because they do not require contact between hosts for transmission, pollutogens should increase with host stress. Two diseases of California sea otters are good examples of pollutogens; valley fever is caused by a fungus that enters the marine environment from eroded soil and toxoplasmosis is caused by a protozoan that enters the ocean along with feces from domestic cats (Kreuder et al. 2003).

Gathering evidence suggests that pollutogens may be related to coral decline (Richmond 1993). Within the Caribbean, populations of the most common reef-building coral, *Acropora palmata*, are being decimated by white pox disease and other causes, with losses of living cover in the Florida Keys averaging 85% or greater (Miller et al. 2002, Patterson et al. 2002). *Serratia marcescens* is the cause of white pox disease (Patterson et al. 2002). This is a common gram-negative bacterium classified as a fecal coliform of humans and other animals. Concurrent studies also show that human sewage markers (e.g., human enteric viruses) are prevalent among near-shore corals and environments of the Florida Keys (Griffin et al. 2000, X). Santavy et al. (2001) have further demonstrated that the incidence of white pox disease on *A. palmata* is significantly greater on coral reefs near Key West than on reefs in the Dry Tortugas. They speculate that this correlation is due to the superior water quality in the Dry Tortugas (Boyer & Jones 2002). These studies strongly suggest that pollutogens affect coral health.

Pathogen pollution is likely to increase with expanding coastal human populations and the increased precipitation predicted from climate change models. This is also an impact for which management can provide some relief, such as by tighter controls on species introductions and growing pressure by coastal communities to improve sewage run off. Concerns for the effects of pollutogens on sea otters and coral reefs have led to public pressure for better sewage management in the United States.

OYSTERS: A CASE STUDY

Oysters, in particular, offer an attractive model for gauging whether diseases are increasing in the marine environment. They are sessile, and mortalities are relatively easy to observe because shells remain in situ after death. They are probably the most valuable commercial molluscan species worldwide and consequently have drawn the attention of local and national governments for centuries. Production figures date to the 1800s in both Europe and the United States (MacKenzie et al.

1997), at which time oysters also became the object of numerous descriptive and scientific studies (Brooks 1880, Ingersoll 1881, Nelson 1889).

Some of the earliest accounts of oyster fisheries mention mortalities large enough to have reduced harvestable supplies (Hoek 1879, cited in Dijkema 1997; Ingersoll 1881; LaFont 1874). In addition to predation, siltation, and freshwater runoff (Goulletquer & Heral 1997, Nelson 1889), disease was sometimes listed as a cause of the deaths, but without being attributed to a specific agent (Strand & Vølstad 1997). Shell disease, first described in 1878 (Alderman & Jones 1971), was probably the first scientifically recognized oyster disease caused by an infectious agent (Table 1). Pathogens were suspected in epizootics in Canada (1915–1916) and Europe (1920–1921) but never identified. Since the mid-1900s, a series of oyster diseases and disease-causing organisms has been described, many causing epizootic mortalities and ruinous declines in oyster production in the United States and Western Europe (Table 1).

Several factors underlie the recent increase in reported oyster (and other molluscan) disease outbreaks. Climate change has been implicated in the northward expansion of dermo and possibly MSX diseases of oysters in the United States (Cook et al. 1998, Hofmann et al. 2001). Parasites have been introduced into new areas through increased shipment of host oysters for fisheries and aquaculture, and increased shipping may have introduced parasites in ballast water or in oysters attached to ships' hulls (Bustnes et al. 2000, Elston et al. 1986, Farley 1992). Newly introduced animals may be susceptible to local pathogens (Ford et al. 2002, Maes & Paillard 1992). Sometimes, diseases appear to spread rapidly around the globe (Table 1), either because of transport of oysters or the spread of recognition among growers. Molluscan aquaculture, which has increased markedly over the past few decades, is also the source of disease outbreaks caused by culture conditions themselves. The high densities under which animals are grown and the high temperatures sustained in hatcheries favor the proliferation and transmission of opportunistic pathogens (Elston & Wilkinson 1985, LeDeuff et al. 1996).

Although normalized reports of disease in mollusks have increased over time (Ward & Lafferty 2004), perceived increases in disease could be an artifact of improvements in detection (Harvell et al. 1999). The level of scientific observation has grown significantly since the oyster epizootics of the 1950s–1970s. Many governments now require health inspections of mollusks before importation. They also conduct pathological surveys of wild and cultured populations. Both efforts uncover previously undescribed parasites and diseases, although anecdotal and documentary evidence of earlier unexplained mortalities suggest that many of the diseases were not new when first described. Finally, increased funding for shellfish disease research in many countries has resulted in (*a*) the creation by universities and other research agencies of positions in shellfish pathology and (*b*) the training of students to fill those positions—where they expand the potential for discovering, documenting, and investigating diseases and disease agents. Although some oyster disease outbreaks are clearly new, others may be long-standing conditions, noted and accepted for years by shellfish harvesters, but they become newly discovered

TABLE 1 Diseases of oysters over time, their causative agents (where known), host species, areas reported, and year first reported[a]

Disease/Agent	Host(s)	Region	Reported
Shell disease/*Ostracoblabe implexa* (fungus)	*Ostrea edulis, Crassostrea angulata*	Europe	1878[b]
Malpeque disease/Unknown	*C. virginica*	Eastern Canada	1915
Australian Winter disease/ *Mikrocytos mackini* (protozoan)	*Saccostrea commercialis*	Australia	1924[b]
Dermo disease/*Perkinsus marinus* (protozoan)	*C. virginica, C. gigas, C. ariakensis*	Gulf Coast and eastern United States	1940s[b] (1900s)
Summer mortality/Bacteria and viruses?	*C. gigas*[c]	Japan, western United States, France	1940s to 1980s
MSX disease/*Haplosporidium nelsoni* (protozoan)	*C. virginica, C. gigas*	Eastern and western United States, Asia[d]	1957
SSO disease/*H. costale* (protozoan)	*C. virginica*	Eastern United States	1959[b]
Marteiliosis/*Marteilia refringens* (protozoan)	*O. edulis, O. angasi, Tiostrea chilensis*	Western Europe	1967[b]
QX disease/*Marteilia sydneyi* (protozoan)	*Saccostrea commercialis*	Australia	1969[b]
Denman Island disease/ *Mikrocytos mackini* (protozoan)	*C. gigas, C. virginica, O. edulis, Ostreola conchaphila*	Western Canada	1960[b]
Herpes disease/Herpes-like virus(es)	Numerous bivalves[c]	Worldwide	1970s to 1990s
Juvenile Oyster disease/Bacteria?	*Crassostrea virginica*[c]	Eastern United States	1988
Bonamiosis/*Bonamia ostrea* (protozoan)	Five *Ostrea* spp., *O. conchaphila, Tiostrea chilensis, C. ariakensis*?	Western Europe, northeastern and northwestern United States	1979[b] (eastern United States, 1960s)
Bonamiosis/*Bonamia exositosa*	*Tiostrea chilensis*	New Zealand	1986

[a]For Supplemental References to Table 1: Follow the Supplemental Material link from the Annual Reviews home page at http://www.annualreviews.org.

[b]Probably present earlier.

[c]In culture/hatcheries.

[d]Introduced from Asia to United States.

diseases when investigated. Despite this potential for bias, the evidence suggests that diseases have increased in oysters over time.

CONCLUSIONS

A high economic and social value is placed on the abundance of marine species, making increases in disease a concern for society. Unfortunately, disease appears to be increasing in several marine taxa. Climate warming may be responsible for some of the more notable examples. Increasing temperature facilitates the spread of warm-water parasites or weakens the defenses of those marine organisms that are already near the upper end of their thermal tolerance. The resulting stress should especially increase noninfectious and generalist diseases and parasites. This appears to be leading to unprecedented bleaching events in corals. Infectious coral diseases may be related to El Niño events, which seem likely to increase in frequency with climate warming. Because corals create habitat for whole communities, an increase in coral disease could lead to dramatic changes in tropical near-shore communities. These effects are alarming, and the effects of climate warming on coral disease should generate considerable concern. Other aspects of global change can increase disease in the ocean. Increasing populations, such as seen in many protected marine mammals, or as an indirect result of fishing top predators (such as for the urchin disease example mentioned above), or of eutrophication, provide increased opportunities for disease transmission. Increases in introduced species, aquaculture, and contacts between terrestrial and marine species also leads to the emergence of new pathogens. Terrestrial runoff is increasingly polluted, raising the chance that terrestrial pathogens may affect marine species, even if marine hosts are dead ends for the "pollutogen." Several of these factors that increase disease are indications of how much humans stress marine ecosystems.

Contrary to most views on disease in the ocean, some diseases will decline with environmental degradation. Some parasites are more sensitive to toxic pollution than are their hosts. Perhaps more importantly, fishing may result in widescale losses of infectious disease. Given the vast scale of fisheries, parasite loss may be the hidden, but dominant, effect of anthropogenic change on disease in the ocean. Although few will mourn the loss of fish diseases, their disappearance should be alarming if they indicate broadscale overfishing.

Scientists increasingly realize that disease is an important aspect of ecology and that environmental degradation and disease interact in a complex manner. Roughly half of the studies of parasite ecology now concern how changes in the environment affect parasites (Lafferty 2003). Two thirds of these environmental studies focus on emerging diseases or increases in infectious disease associated with environmental change, whereas the other third find that changes in the environment reduce infectious disease. However, few studies acknowledge that environmental change can either increase or decrease infectious disease, depending on the disease and the type of change. Progress in understanding and managing diseases in the ocean will require an acknowledgment of the complex interactions between disease and

the environment. It will also require a dedication to systematic, long-term studies that track changes in disease over time across several taxa and habitats.

ACKNOWLEDGMENTS

The *Annual Review of Ecology, Evolution, and Systematics* is online at
http://ecolsys.annualreviews.org

LITERATURE CITED

Alderman DJ, Jones EBG. 1971. Shell disease of oysters. *Fish. Invest. London Ser. I* 26:1–19

Alker AP, Smith GW, Kim K. 2001. Characterization of *Aspergillus sydowii* (Thom et Church), a fungal pathogen of Caribbean sea fan corals. *Hydrobiologia* 460:105–11

Amundsen PA, Kristoffersen R. 1990. Infection of whitefish (*Coregonus lavaretus* L. s.l.) by *Triaenophorus crassus* Forel (Cestoda: Pseudophyllidea): a case study in parasite control. *Can. J. Zool.* 68:1187–92

Anderson RM, May RM. 1986. The invasion, persistence and spread of infectious diseases within animal and plant communities. *Philos. Trans. R. Ser. B* 314:533–70

Arneberg P, Skorping A, Grenfell B, Read A. 1998. Host densities as determinants of abundance in parasite communities. *Philos. R. Soc. London Ser. B* 265:1283–89

Aronson RB, Macintyre IG, Precht WF, Murdoch TJT, Wapnick CM. 2002. The expanding scale of species turnover events on coral reefs in Belize. *Ecol. Monogr.* 72:233–49

Aronson RB, Precht WF. 1998. Extrinsic control of species replacement on a Holocene reef in Belize: the role of coral disease. *Coral Reefs* 17:223–30

Aronson RB, Precht WF. 2001. White-band diseases and the changing face of Caribbean coral reef. *Hydrobiologia* 460:25–38

Babcock RC, Kelly S, Shears NT, Walker JW, Willis TJ. 1999. Changes in community structure in temperate marine reserves. *Mar. Ecol. Prog. Ser.* 189:125–34

Bengston JL, Boveng P. 1991. Antibodies to canine distemper virus in Antarctic seals. *Mar. Mammal Sci.* 7:85–87

Bergersen EP, Anderson DE. 1997. The distribution and spread of *Myxobolus cerebralis* in the United States. *Fisheries* 22:6–7

Birkeland C. 1997. *Life and Death of Coral Reefs.* New York: Chapman & Hall

Black GA. 1983. Taxonomy of a swimbladder nematode, *Cystidicola stigmatura* (Leidy), and evidence of its decline in the Great Lakes. *Can. J. Fish. Aquat. Sci.* 40:643–47

Boyer JN, Jones RD. 2002. A view from the bridge: external and internal forces affecting the ambient water quality of the Florida Keys National Marine Sanctuary. See Porter & Porter 2002, pp. 609–28

Brooks WK. 1880. Development of the American oyster (*Ostrea virginica* L.). *Stud. Biol. Lab., Johns Hopkins Univ.* 4:1–81

Brown AF, Pascoe D. 1989. Parasitism and host sensitivity to cadmium: an acanthocephalan infection of the freshwater amphipod *Gammarus pulex*. *J. Appl. Ecol.* 26:473–87

Brown BE. 1997. Coral bleaching: causes and consequences. *Coral Reefs* 16:129–38

Bruno JF, Petes LE, Harvell CD, Hettinger A. 2003. Nutrient enrichment can increase the severity of coral diseases. 6:1056–61

Bustnes JO, Galaktionov KV, Irwin SWB. 2000. Potential threats to littoral biodiversity: Is increased parasitism a consequence of human activity? *Oikos* 90:189–90

Carlton RG, Richardson LL. 1995. Oxygen and sulfide dynamics in a horizontally migrating cyanobacterial mat: black band disease of corals. *FEMS Microbiol. Ecol.* 18:155–62

Chadwick-Furman NE. 1996. Reef coral diversity and global change. *Global Change Biol.* 2:559–68

Chubb JC. 1979. Seasonal occurrences of helminths in freshwater fishes. Part II. Trematoda. *Adv. Parasitol.* 17:141–313

Chubb JC. 1980. Seasonal occurrence of helminths in freshwater fishes. Part III. Larval Cestoda and Nematoda. *Adv. Parasitol.* 18:1–120

Connell JH. 1997. Disturbance and recovery of coral assemblages. *Coral Reefs* 16:101–13

Cook CB, Mueller EM, Ferrier MD, Annis E. 2002. The influence of nearshore waters on corals of the Florida Reef Tract. See Porter & Porter 2002, pp. 770–83

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

DeStewart RL, Ross PS, Voss JG, Osterhaus ADME. 1996. Impaired immunity in harbour seals (*Phoca vitulina*) fed environmentally contaminated herring. *Vet. Q.* 18:S127–28

Diaz-Soltero H. 1999. Endangered and threatened species: a revision of candidate species list under the Endangered Species Act. *Fed. Regist.* 64:33466–68

Dijkema R. 1997. Molluscan fisheries and culture in the Netherlands. See MacKenzie et al. 1997, pp. 115–36

Dobson AP, Hudson PJ, Lyles AM. 1992. Macroparasites: worms and others. In *Natural Enemies. The Population Biology of Predators, Parasites and Diseases*, ed. MJ Crawley, pp. 329–48. Oxford, UK: Blackwell

Dobson AP, May RM. 1987. The effects of parasites on fish populations—theoretical aspects. *Int. J. Parasitol.* 17:363–70

Dogiel VA, Lutta A. 1937. Mortality among spiny sturgeon of the Aral Sea in 1936. *Rybn Khoz* 12:26–27

Dungan M, Miller T, Thomson D. 1982. Catastrophic decline of a top carnivore in the Gulf of California rocky intertidal zone. *Science* 216:989–91

Dustan P. 1999. Coral Reefs under stress: sources of mortality in the Florida Keys. *Nat. Resour. Forum* 23:147–55

Edmunds PJ. 1991. Extent and effect of black band disease on a Caribbean reef. *Coral Reefs* 10:161–65

Elliot SL, Blanford S, Thomas MB. 2002. Host-pathogen interactions in a varying environment: temperature, behavioural fever and fitness. *Philos. R. Soc. London Ser. B* 269:1599–607

Elston RA, Farley CA, Kent ML. 1986. Occurrence and significance of bonamiasis in European flat oysters *Ostrea edulis* in North America. *Dis. Aquat. Organ.* 2:49–54

Elston RA, Wilkinson MT. 1985. Pathology, management and diagnosis of oyster velar virus disease (OVVD). *Aquaculture* 48:189–210

Epstein P. 1996. Emergent stressors and public health implications in large marine ecosystems: an overview. In *The Northeast Shelf Ecosystem: Assessment, Sustainability, and Management*, ed. T Smayda, pp. 417–38. Cambridge, MA: Blackwell

Epstein PR, Sherman B, Spanger-Siegfried E, Langston A, Prasad S, McKay B. 1998. *Marine Ecosystems: Emerging Diseases as*

Indicators of Change. Boston, MA: Cent. Health Glob. Environ., Harvard Med. Sch. 85 pp.

Farley CA. 1992. Dispersal of pathogens, parasites, pests, predators and competitors. In *Dispersal of Living Organisms into Aquatic Ecosystems*, ed. A Rosenfield, R Mann, pp. 139–54. College Park, MD: Maryland Sea Grant

Ford SE, Kraeuter JN, Barber RD, Mathis G. 2002. Aquaculture associated factors in QPX disease of hard clams: density and seed source. *Aquaculture* 208:23–38

Gil-Agudelo DL, Garzon-Ferreira J. 2001. Spatial and seasonal variation of Dark Spots Disease in coral communities of the Santa Marta area (Colombian Caribbean). *Bull. Mar. Sci.* 69:619–29

Gilbert MA, Granath W. 2003. Whirling Disease of salmonid fish: life cycle, biology and disease. *J. Parasitol.* 89:658–67

Gilmartin WG, DeLong RL, Smith AW, Sweeney JC, Lappe BWD, et al. 1976. Premature parturition of the California sea lion. *J. Wildlife Dis.* 12:104–15

Gladfelter WB. 1982. White-Band Disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. *Bull. Mar. Sci.* 32:639–43

Glynn PW, Howard LS, Corcoran E, Freay AD. 1984. The occurrence and toxicity of herbicides in reef building corals. *Mar. Pollut. Bull.* 15:370–74

Gog J, Woodroffe R, Swinton J. 2002. Disease in endangered metapopulations: the importance of alternative hosts. *Philos. R. Soc. London Ser. B* 269:671–76

Goreau TJ, Cervino J, Goreau M, Hayes R, Hayes M, et al. 1998. Rapid spread of diseases in Caribbean coral reefs. *Rev. Biol. Trop.* 46:157–71

Goulletquer P, Heral M. 1997. Marine molluscan production trends in France: from fisheries to aquaculture. See MacKenzie et al. 1997, pp. 137–64

Greenstein BJ, Curran HA, Pandolfi JM. 1998. Shifting ecological baselines and the demise of *Acropora cervicornis* in the western North

Atlantic and Caribbean Province: a Pleistocene perspective. *Coral Reefs* 17:249–61

Griffin DW, Gibson CJ, Lipp EK, Riley K, Paul JH, Rose JB. 2000. Detection of viral pathogens by reverse transcriptase PCR and of microbial indicators by standard methods in the canals of the Florida Keys. *Appl. Environ. Microbiol.* 66:876

Guth DJ, Blankespoor HD, Cairns J. 1977. Potentiation of zinc stress caused by parasitic infection of snails. *Hydrobiologia* 55:225–29

Harder TC, Willhus T, Leibold W, Liess B. 1992. Investigations on the course and outcome of phocine distemper virus infection in harbor seals (*Phoca vitulina*) exposed to polychlorinated biphenyls. *J. Vet. Med. Ser. B* 39:19–31

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 D, Kim K, Quirolo C, Weir J, Smith G. 2001. Coral bleaching and disease: contributors to 1998 mass mortality in *Briareum asbestinum* (Octocorallia, Gorgonacea). *Hydrobiologia* 460:97–104

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

Hayes ML, Bonaventura J, Mitchell TP, Prospero JM, Shinn EA, et al. 2001. How are climate and marine biological outbreaks functionally linked? *Hydrobiolgia* 460:213–20

Hayes RL, Goreau NI. 1998. The significance of emerging diseases in the tropical coral reef ecosystem. *Rev. Biol. Trop.* 46:173–85

Heide-Jorgensen M-P, Harkonen T, Dietz R, Thompson PM. 1992. Retrospective of the 1988 European seal epizootic. *Dis. Aquat. Organ.* 13:37–62

Heide-Jorgensen MP, Harkonen T. 1992. Epizootiology of the seal disease in the Eastern North Sea. *J. Appl. Ecol.* 29:99–107

Herbst LH. 1994. Fibropapillomatosis of marine turtles. *Annu. Rev. Fish Dis.* 4:389–425

Hochachka WM, Dhondt A. 2000. Density-dependent decline of host abundance resulting from a new infectious disease. *Proc. Natl. Acad. Sci. USA* 97:5303–6

Hoegh-Guldberg O. 1999. Climate change, coral bleaching and the future of the world's coral reefs. *Mar. Freshw. Res.* 50:839–66

Hoff CC. 1941. A case of correlation between infection of snail hosts with *Cryptocotyle lingua* and the habits of gulls. *J. Parasitol.* 27:539

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

Holmes JC. 1996. Parasites as threats to biodiversity in shrinking ecosystems. *Biodivers. Conserv.* 5:975–83

Holt RA, Amandi A, Rohovec JS, Fryer JL. 1989. Relation of water temperature to bacterial coldwater disease in coho salmon, chinook salmon, and rainbow trout. *J. Aquat. Anim. Health* 1:94–101

Houghton JT, Filho LGM, Callandar BA, Harris N, Kattenberg A, Maskell K, eds. 1996. *Climate Models—Projections of Future Climate*. New York: Cambridge Univ. Press

Huspeni TC, Lafferty KD. 2004. Using larval trematodes that parasitize snails to evaluate a salt-marsh restoration project. *Ecol. Appl.* 14(3):795–804

Ingersoll E. 1881. *The Oyster Industry*. Washington, DC: US Dep. Interior. 251 pp.

Jackson JBC, Kirby MX, Berger WH, Bjorndal KA, Botsford LW, et al. 2001. Historical overfishing and the recent collapse of coastal ecosystems. *Science* 293:629–38

Johnson LL. 2001. *An analysis in support of sediment quality thresholds for polycyclic aromatic hydrocarbons to protect estuarine fish. Rep. NMFS-NWFSC-47*, US Dep. Commer., NOAA

Jones JB, Hyatt AD, Hine PM, Whittington RJ, Griffin DA, Bax NJ. 1997. Special topic review: Australasian pilchard mortalities. *World J. Microb. Biot.* 13:383–92

Kennedy CR, Bush AO, Aho JM. 1986. Patterns in helminth communities: Why are birds and fish different? *Parasitology* 93:205–15

Khan RA. 1990. Parasitism in marine fish after chronic exposure to petroleum hydrocarbons in the laboratory and to the Exxon Valdez oil spill. *Bull. Environ. Contam. Toxicol.* 44:759–63

Khan RA, Thulin J. 1991. Influence of pollution on parasites of aquatic animals. *Adv. Parasitol.* 30:201–38

Kim K, Harvell CD. 2002. Aspergillosis of sea fan corals: disease dynamics in the Florida Keys. In *The Everglades, Florida Bay, and the Coral Reefs of the Florida Keys: An Ecosystem Sourcebook*, ed. JW Porter, KG Porter, pp. 824–913. Boca Raton, FL: CRC Press

Kim K, Harvell CD. 2004. The rise and fall of a 6 year coral-fungal epizootic. *Am. Nat.* In press

Kim K, Harvell CD, Kim PD, Smith GW, Merkel SM. 2000. Fungal disease resistance of Caribbean sea fan corals (*Gorgonia* spp.). *Mar. Biol.* 136:259–67

Kleypas JA, Buddemeier RW, Archer D, Gattuso JP, Langdon C, Opdyke BN. 1999. Geochemical consequences of increased atmospheric carbon dioxide on coral reefs. *Science* 284:118–20

Knowlton N. 1992. Thresholds and multiple stable states in coral reef community dynamics. *Am. Zool.* 32:674–82

Kreuder C, Miller MA, Jessup DA, Lowenstein LJ, Harris MD, et al. 2003. Patterns of mortality in southern sea otters (*Enhydra lutris nereis*) from 1998–2001. *J. Wildlife Dis.* 39:495–509

Kuris AM, Lafferty KD. 1992. Modelling crustacean fisheries: effects of parasites on management strategies. *Can. J. Fish. Aquat. Sci.* 49:327–36

Lafferty KD. 1997. Environmental parasitology: What can parasites tell us about human impacts on the environment? *Parasitol. Today* 13:251–55

Lafferty KD. 2003. Is disease increasing or decreasing, and does it impact or maintain biodiversity? *J. Parasitol.* 89:S101–5

Lafferty KD. 2004. Fishing for lobsters indirectly increases epidemics in sea urchins. *Ecol. Appl.* In press

Lafferty KD, Gerber L. 2002. Good medicine for conservation biology: the intersection of epidemiology and conservation theory. *Conserv. Biol.* 16:593–604

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

Lafferty KD, Kuris AM. 1993. Mass mortality of abalone *Haliotis cracherodii* on the California Channel Islands: tests of epidemiological hypotheses. *Mar. Ecol. Prog. Ser.* 96: 239–48

Lafferty KD, Kuris AM. 2002. Trophic strategies, animal diversity and body size. *Trends. Ecol. Evol.* 17:507–13

Lafferty KD, Kuris AM. 2004. Parasitism and environmental disturbances. In *Parasites and Ecosystems*, ed. F Thomas, JF Guégan, F Renaud. Oxford: Oxford Univ. Press. In press

LaFont A. 1874. *Note sur les Huitrières du Bassin d'Archacon.* Bordeaux: Chez Feret, Libraire. 52 pp.

Lapointe BE, Matzie WR, Barille PJ. 2002. Biotic phase shifts in Florida Bay and back reef communities in the Florida Keys: linkages with historical freshwater flows and nitrogen loading from Everglades runoff. In *The Everglades, Florida Bay, and the Coral Reefs of the Florida Keys*, ed. JW Porter, KG Porter, pp. 629–53. Boca Raton, FL: CRC Press

LeDeuff RM, Renault T, Gerard A. 1996. Effects of temperature on herpes-like virus detection among hatchery-reared larval Pacific oyster *Crassostrea gigas. Dis. Aquat. Organ.* 24:149–57

Lefcort H, Aguon MQ, Bond KA, Chapman KR, Chaquette R, et al. 2002. Indirect effects of heavy metals on parasites may cause shifts in snail species compositions. *Arch. Environ. Contam. Toxicol.* 43:34–41

Lessios HA. 1988. Mass mortality of *Diadema antillarum* in the Caribbean: What have we learned? *Annu. Rev. Ecol. Syst.* 19:371–93

Levitus S, Antonov JI, Boyer TP, Stephans C. 2000. Warming of the world ocean. *Science* 287:2225–29

Lightner DV. 1996. Epizootiology, distribution and the impact on international trade of two penaeid shrimp viruses in the Americas. *Rev. Sci. Tech. Off. Int. Epizoot.* 15:579–601

Lipp EK, Jarrell JL, Griffin DW, Lukasik J, Jacukiewicz J, Rose JB. 2002. Preliminary evidence for human fecal contamination in corals of the Florida Keys, USA. *Mar. Pollut. Bull.* 44:666–70

MacKenzie CLJ, Burrell VGJ, Rosenfield A, Hobart WL, eds. 1997. *The History, Present Condition, and Future of the Molluscan Fisheries of North and Central America and Europe.* Vol. 129. Washington, DC: US Dep. Commer. 240 pp.

Maes P, Paillard C. 1992. Effect du *Vibrio* P1, pathogene de *Ruditapes philippinarum*, sur d'autres espèces de bivalves. *Les Molluscques Marins, Biologies et Aquaculture. IFREMER, Actes de Colloques* 14:141–48

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

Marcogliese DJ. 2001b. Pursuing parasites up the food chain: implications of food web structure and function on parasite communities in aquatic systems. *Acta Parasitol.* 46: 82–93

McCallum HI, Dobson AP. 1995. Detecting disease and parasite threats to endangered species and ecosystems. *Trends Ecol. Evol.* 10:190–94

Miller MW, Bourque AS, Bohnsack JA. 2002. An analysis of the loss of acroporid corals at Looe Key, Florida, USA: 1983–2000. *Coral Reefs* 21:179–82

Myers RA, Worm B. 2003. Rapid worldwide depletion of predatory fish communities. *Nature* 423:280–83

Nelson J. 1889. *Oyster interests of New Jersey. Rep. Spec. Bull. E*, NJ Agric. Coll. Exp. Stn., New Brunswick

O'Shea T. 1999. Environmental contaminants and marine mammals. In *Biology of Marine*

Mammals, ed. SA Rommel, pp. 485–564. Washington, DC: Smithsonian Inst. Press

Osterhaus ADME, Vedder EJ. 1988. Identification of virus causing recent seal deaths. *Nature* 335:20

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, *Acropora palmata*. *Proc. Natl. Acad. Sci. USA* 99:8725–30

Pinnegar JK, Polunin NVC, Francour P, Badalamenti F, Chemello R, et al. 2000. Trophic cascades in benthic marine ecosystems: lessons for fisheries and protected-area management. *Environ. Conserv.* 27:179–200

Pojmanska T, Grabda-Kazubska B, Kazubski SL, Machalska J, Niewiadomska K. 1980. Parasite fauna of five fish species from the Konin lakes complex, artificially heated with thermal effluents, and from Goplo Lake. *Acta Parasitol.* 27:319–57

Porter JW, Dustan P, Jaap WC, Patterson KL, Kosmynin V, et al. 2001. Patterns of spread of coral disease in the Florida Keys. *Hydrobiologia* 460:1–24

Porter JW, Fitt WK, Spero HJ, Rogers CS, White MW. 1989. Bleaching in reef corals—physiological and stable isotopic responses. *Proc. Natl. Acad. Sci. USA* 86:9342–46

Porter JW, Kosmynin V, Patterson K, Porter KG, Jaap WC, et al. 2002. Detection of coral reef change by the Florida Keys Coral Reef Monitoring Project. See Porter & Porter 2002, pp. 749–69

Porter JW, Meier OW. 1992. Quantification of loss and change in Floridian reef coral populations. *Am. Zool.* 32:625–40

Porter JW, Porter KG, eds. 2002. *The Everglades, Florida Bay, and Coral Reefs of the Florida Keys.* Boca Raton, FL: CRC Press

Porter JW, Tougas JI. 2001. Reef ecosystems: threats to their biodiversity. *Encyclop. Biodiv.* 5:73–95

Richmond RH. 1993. Coral reefs—present problems and future concerns resulting from anthropogenic disturbance. *Am. Zool.* 33:524–36

Riggs MR, Lemly AD, Esch GW. 1987. The growth, biomass, and fecundity of *Bothriocephalus acheilognathi* in a North Carolina cooling reservoir. *J. Parasitol.* 73:893–900

Robson EM, Williams IC. 1970. Relationships of some species of digenea with the marine prosobranch *Littorina littorea.* Part 1. The occurrence of larval digenea in *Littorina littorea* on the North Yorkshire coast. *J. Helminthol.* 44:153–68

Ruiz GM, Fofonoff P, Carlton JT, Wonham MJ, Hines AH. 2000. Invasions of coastal marine communities in North America: apparent patterns, processes, and biases. *Annu. Rev. Ecol. Syst.* 31:481–531

Rutzler K, Santavy DL, Antonius A. 1983. The Black Band Disease of Atlantic reef corals. III. Distribution, ecology and development. *PSZNI Mar. Ecol.* 4:329–58

Sala E, Boudouresque CF, Harmelin-Vivien M. 1998. Fishing, trophic cascades, and the structure of algal assemblages: evaluation of an old but untested paradigm. *Oikos* 82:425–39

Sanders MJ. 1966. Parasitic castration of scallop *Pecten alba* (Tate) by a bucephalid trematode. *Nature* 212:307

Santavy DL, Mueller E, Peters EC, MacLaughlin L, Porter JW, et al. 2001. Quantitative assessment of coral diseases in the Florida Keys: strategy and methodology. *Hydrobiologia* 460:39–52

Scott ME. 1988. The impact of infection and disease on animal populations: implications for conservation biology. *Conserv. Biol.* 2:40–56

Shears NT, Babcock RC. 2002. Marine reserves demonstrate top-down control of community structure on temperate reefs. *Oecologia* 132:131–42

Shinn EA, Smith GW, Prospero JM, Betzer P, Hayes ML, et al. 2000. African dust and the demise of Caribbean coral reefs. *Geophys. Res. Lett.* 27:3029–32

Siddall R, Clers SD. 1994. Effect of sewage sludge on the miracidium and cercaria of *Zoogonoides viviparus* (Trematoda: Digenea). *Helminthologia* 31:143–53

Smith GW, Ives LD, Nagelkerken IA, Ritchie

KB. 1996. Caribbean sea-fan mortalities. *Nature* 383:487

Smith NF. 2001. Spatial heterogeneity in recruitment of larval trematodes to snail intermediate hosts. *Oecologia* 127:115–22

Stadnichenko AP, Ivanenko LD, Gorchenko IS, Grabinskaya OV, Osadchuk LA, Sergeichuk SA. 1995. The effect of different concentrations of nickel sulphate on the horn snail (Mollusca: Bulinidae) infected with the trematode *Cotylurus cornutus* (Strigeidae). *Parazitologiya* 29:112–16

Stewart BS, Yochem PK. 2000. Community ecology of California Channel Islands pinnipeds. In *Fifth California Islands Symposium*, ed. DR Brown, KL Mitchell, HW Chang, pp. 413–20. Santa Barbara, CA: Miner. Manag. Serv.

Stiven AE. 1964. Experimental studies on the host parasite system hydra and *Hydramoeba hydroxena* (Entz.). II. The components of a single epidemic. *Ecol. Monogr.* 34:119–42

Strand O, Vølstad JH. 1997. Molluscan fisheries and culture of Norway. See MacKenzie et al. 1997, pp. 7–24

Sutherland KP, Porter JW, Torres C. 2004. Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. *Mar. Ecol. Prog. Ser.* 266:273–302

Swart RL, Ross PS, Vedder LJ, Timmerman HH, Heisterkamp S, et al. 1994. Impairment of immune function in harbor seals (*Phoca vitulina*) feeding on fish from polluted waters. *Ambio* 23:155–59

Tegner MJ, Levin LA. 1983. Spiny lobsters and sea urchins: analysis of a predator-prey interaction. *J. Exp. Mar. Biol. Ecol.* 73:125–50

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

Van Loveren H, Ross PS, Osterhaus A, Vos JG. 2000. Contaminant induced immunosuppression and mass mortalities among harbor seals. *Toxicol. Lett.* 112:319–24

Ward JR, Lafferty KD. 2004. The elusive baseline of marine disease: Are diseases in ocean ecosystems increasing? *PLoS Biol.* 2:542–47

Wilkinson C, ed. 2002. *Status of Coral Reefs of the World: 2002*. Townsville: Aust. Inst. Mar. Sci.

Williams J, Ernest H, Bunkley-Williams L. 1990. The world-wide coral reef bleaching cycle and related sources of coral mortality. *Atoll Res. Bull.* 335:1–71

Woodroffe R. 1999. Managing threats to wild mammals. *Anim. Conserv.* 2:185–93

$\overset{A}{R}$ *Annual Review of Ecology, Evolution, and Systematics*
Volume 35, 2004

CONTENTS