



REVIEW: MARINE ECOLOGY

Emerging Marine Diseases—Climate Links and Anthropogenic Factors

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Mass mortalities due to disease outbreaks have recently affected major taxa in the oceans. For closely monitored groups like corals and marine mammals, reports of the frequency of epidemics and the number of new diseases have increased recently. A dramatic global increase in the severity of coral bleaching in 1997–98 is coincident with high El Niño temperatures. Such climate-mediated, physiological stresses may compromise host resistance and increase frequency of opportunistic diseases. Where documented, new diseases typically have emerged through host or range shifts of known pathogens. Both climate and human activities may have also accelerated global transport of species, bringing together pathogens and previously unexposed host populations.

The oceans harbor enormous biodiversity by terrestrial terms (1), much of which is still poorly described taxonomically. Even less well known are the dynamics of intermittent, ephemeral, threshold phenomena such as disease outbreaks. Despite decades of intense study of the biological agents structuring natural communities, the ecological and evolutionary impact of diseases in the ocean remains unknown, even when these diseases affect economically and ecologically important species. The paucity of baseline and epidemiological information on normal disease levels in the ocean challenges our ability to assess the novelty of a recent spate of disease outbreaks and to determine the relative importance of increased

pathogen transmission versus decreased host resistance in facilitating the outbreaks. Our objectives here are to review the prevalence of diseases of marine taxa to evaluate whether it can be concluded that there has been a recent increase. We also assess the contributing roles of human activity and global climate, and evaluate the role of the oceans as incubators and conveyors of human disease agents.

Is There an Increase in Diseases in the Ocean?

In the past few decades, there has been a worldwide increase in the reports of diseases affecting marine organisms (2, 3) (Table 1). In the Caribbean, mass mortalities among plants, invertebrates, and vertebrates have resulted in dramatic shifts in community structure. Recent outbreaks of coralline algae lethal orange disease and a coralline fungal disease have affected Indo-Pacific communities on unprecedented scales. In the North Atlantic, frequency of mass mortalities of marine mammals appears to be increasing, particularly along heavily polluted coastal areas, suggesting human activity as a factor in disease dynamics. Ecologically and economically important species from temperate oceans, such as seagrasses, oysters, and sea urchins, have also been affected by large-scale epidemics. Although the frequencies of such accounts are compelling, whether they are indeed “new” or are simply artifacts of improved detection requires further evaluation.

Several criteria have been proposed (4) to distinguish new diseases affecting humans. Criteria applicable to disease of nonhuman hosts include novelty of disease symptoms and rapid increases in disease prevalence and virulence. These criteria require either the availability of historical baseline data or stan-

dard epidemiological measures of disease level (that is, prevalence, incidence, virulence). Although the increasing numbers of unusual mass mortalities are suggestive, the lack of additional information for most marine taxa greatly challenges our ability to assess disease novelty. For a few taxa, however, the available data on the novelty of disease symptoms (5) and/or host shifts of a known pathogen present convincing evidence of new diseases.

New symptoms. Marked by two large-scale epidemics with significant community level impacts, the Caribbean basin has emerged as a disease hot spot. The virtual eradication of *Diadema antillarum* (dominant sea urchin) in the 1980s was one of the first well-studied marine epidemics (6), although the pathogen is yet to be identified. In some locations, loss of this keystone herbivore contributed to phase shifts from coral- to algae-dominated reefs (7). Other dominants, like the staghorn and elkhorn and corals, *Acropora* spp., also were virtually eradicated at many localities in the 1980s (8) by an unknown agent from which they have yet to recover. Also during the late 1980s at least 4000 ha of turtle grass, *Thalassia testudinum*, died in Florida Bay (U.S.A.); an additional 23,000 ha were severely affected (9). Diseases affecting benthic marine species such as corals and seagrasses will have disproportionate impacts by altering habitat and ecosystem function. In spite of the impact, little progress has been made in identifying the causative agents for marine diseases or in applying standard epidemiological methods to assess impact or mode of transmission. Of the dozen or so coral diseases currently described for the Caribbean region, the identity of the causative agent is known only for three (10); nonetheless, the severity and novelty of many of the disease symptoms suggest that the diseases are indeed new. Three additional lines of evidence support this view. First, monitoring of coral diseases in the Florida Keys indicates that there has been an increase in the number of new diseases (11) (Fig. 1). Second, because corals are long-lived and many of the diseases are highly virulent (10), current levels of disease prevalence, if they had occurred in previous decades, would have been detected. Finally, evidence from the fossil record indicates that shifts in com-

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munity structure due to disease are not commonplace on these coral reefs. The rapid replacement of the coral *Acropora cervicornis* with *Agaricia* in Belize (12) with *Porites* in the Bahamas (13), taken as a "signature" of epidemics, was absent from geologic cores representing several thousand years of reef development. These results suggest that the current *Agaricia* and *Porites* replacements were unique in the recent ecological history of the Caribbean coral fauna.

In addition to diseases, there has been an apparent increase in the frequency of reports of toxic algal blooms in the last decade. Cetacean, pinniped, and fish populations have been affected, often severely, by algal toxins and/or viral epidemics (3, 14–16). Many toxic blooms in the ocean have been attributed to dinoflagellates, and more than 85 toxic species have been identified (17). Harmful algal blooms appear to have increased globally in the past several decades (2, 14, 18). The toxic dinoflagellate *Pfiesteria piscicida* was originally isolated from an outbreak at an aquaculture facility and has been described as the causative agent of massive fish kills along the Atlantic Coast of the United States (19).

Host shifts. It appears that most new diseases are not caused by new micro-organisms, but rather by known agents infecting new or previously unrecognized hosts. Evidence for this is persuasive in studies of morbilliviral diseases of marine mammals, which indicate that some severe outbreaks have been caused by introduction from terrestrial or other aquatic mammalian reservoir species. For instance, canine distemper virus (CDV) was thought to be introduced into crab-eating seals in Antarctica by contacts with infected sled dogs used during an antarctic expedition (20). Similarly, CDV isolated from Lake Baikal seals (*Phoca sibirica*) was genetically identical to CDV present in domestic dogs in Siberia (21) suggesting that the seal die-off was caused by direct or indirect contacts with domestic dogs. A closely related morbillivirus—phocine distemper virus (PDV)—that previously had not been recognized, was identified as the cause of another mass mortality that occurred in the late 1980s among harbor seals (*Phoca vitulina*) and grey seals (*Halichoris gryphus*) inhabiting the coastal waters of northwestern Europe (22). Soon after, infections with two other

newly recognized morbilliviruses, dolphin morbillivirus (DMV) and porpoise morbillivirus (PMV), were shown to be the cause of mass mortalities and disease outbreaks among dolphins, porpoises, and other cetacean species all over the world (23). PDV was thought to be transmitted to the previously unexposed seals of northwestern Europe by infected harp seals, which migrated toward Europe in response to food shortages due to overfishing around Greenland in the late 1980s (24). Serological studies have shown also that morbilliviruses like DMV and PMV are ubiquitous among cetaceans and are probably transmitted periodically between species (25). A recent survey conducted among terrestrial and aquatic carnivores of Alaska showed that both CDV and PDV are endemic in these populations (26). Recently, DMV- and PMV-like viruses were found in the highly endangered Mediterranean monk seals, which had died either during a mass mortality off the coast of Mauritania or as individually dispersed animals found in Greek waters (27) (Fig. 2). In addition, influenza viruses that had spilled over from aquatic or migratory avian reservoirs have caused

Table 1. Mass mortalities (>10% mortality within populations, where enumerated) among natural populations of selected marine species. Environmental correlates: T, temperature; ND, no data; sal, salinity; turb, turbidity; hur, hurricane.

Start date	Host species	Outbreak location	Pathogen identity	Estimated mortality (%)	Environmental correlates	Ref.
1938	Sponges	North Caribbean	Fungus?	70–95	ND	86
1931	<i>Zostera</i> (seagrasses)	North America, Europe	Slime mold	Extensive	High T	87
1946	<i>Crassostrea</i> (oyster)	Gulf Coast, U.S.A.	<i>Perkinsus marinus</i>	Extensive	High T, sal	88
1954	<i>Clupea</i> (herring)	Gulf St. Lawrence	<i>Ichthyophonus hoferi</i>	50	ND	89
1955	<i>Lobodon</i> (seal)	Antarctica	Virus	Extensive	ND	90
1974	<i>Ostrea</i> (flat oyster)	Northwestern Spain	<i>Marteilia refringens</i>	Extensive	ND	19
1975	<i>Heliaster</i> (starfish)	Western U.S.A.	?	<100	High T	91
1980	<i>Strongylocentrotus</i> (urchin)	Northwestern Atlantic	Amoeba?	>50	ND	92
1980	<i>Ostrea</i> (oyster)	Netherlands	<i>Bonamia ostreae</i>	Extensive	ND	93
1981	<i>Acropora</i> (coral)	Caribbeanwide	Bacteria?	>100	ND	8
1982	<i>Gorgonia</i> (coral)	Central America	?	Extensive	High T	29
1982–6	<i>Haliotis</i> (abalone)	Australia	<i>Perkinsus</i> sp.	Extensive	High T	94
1983	Corals	Caribbeanwide	Microbial consortium	?	Seasonal	95
1983	<i>Patinopecten</i> (scallop)	Western Canada	<i>Perkinsus qugwadi</i>	Extensive	ND	96
1983	<i>Diadema</i> (urchin)	Caribbeanwide	Bacteria?	>95	High T	6
1985	<i>Haliotis</i> (abalone)	Northeastern Pacific	?	>95	High T	97
1986–90	<i>Ruditapes</i> (clam)	Portugal	<i>Perkinsus atlanticus</i>	Extensive	ND	98
1987	<i>Thalassia</i> (seagrass)	Florida, U.S.A.	Slime mold	<95	High T, sal	9
1988	<i>Argopecten</i> (scallop)	North Caribbean	Protozoan	Extensive	ND	99
1988	<i>Phoca</i> (seals)	Northwestern Europe	Virus	~70	Pollution	22
1988	<i>Phocoena</i> (porpoise)	Northeastern Ireland	Virus	?	Pollution	100
1989	<i>Argopecten</i> (scallop)	Eastern Canada	<i>Perkinsus</i> sp.?	Extensive	ND	101
1989	<i>Phoca</i> (seals)	Lake Baikal	Virus	>10	ND	102
1990	<i>Stenella</i> (dolphin)	Western Mediterranean	Virus	>20	Pollution	103
1991	<i>Clupea</i> (herring)	Western Sweden	<i>Ichthyophonus hoferi</i>	>10	Low T	104
1992	<i>Ecklonia</i> (kelp)	Northeastern New Zealand	?	40–100	High turb	105
1993	Coralline algae	South Pacific	Bacteria?	Extensive	ND	106
1995	<i>Strongylocentrotus</i> (urchin)	Norway	Nematode?	~90	ND	107
1995	<i>Gorgonia</i> (corals)	Caribbeanwide	Fungus	Extensive	ND	108
1995	<i>Dichocoenia</i> and others (coral)	Florida, U.S.A.	Bacteria	<38	Seasonal	109
1996	<i>Diploria</i> and others (coral)	Puerto Rico	Bacteria	Extensive	Seasonal, hur	110
1997	<i>Porolithon</i> (algae)	Samoa	Fungus	Extensive	ND	111
1997	<i>Sardinops</i> (pilchard)	Southern Australia	Virus?	Extensive	ND	112
1997	<i>Monachus</i> (seal)	West Africa	Virus/toxin	>75	ND	16

mortality among seals and whales (28). An unusual case of a host shift in a marine invertebrate is the aspergillosis of Caribbean sea fan corals (29). The pathogen, identified as *Aspergillus sydowii* (30), is typically a soil-borne fungus that is known to cause opportunistic infections of terrestrial species (31). In sea fans (*Gorgonia* spp.), monitoring studies show that the fungus can rapidly erode the coral (Fig. 3) and, in some cases, cause death. Its emergence as a marine pathogen suggests the ineffectiveness of the land-sea boundary as a barrier to disease transmission.

Conditions Favoring Disease Outbreaks

A disease outbreak is favored by changing environmental conditions that either increase prevalence and virulence of existing disease or facilitate new disease (32). Two conditions—climate variability and human activity—appear to have played roles in epidemics by undermining host resistance and facilitating pathogen transmission.

Role of climate variability. Climate-induced changes in the environment affect health and productivity of marine ecosystems over extended spatial and temporal scales. The current trend toward a warming climate could result in modifications to the basic biological properties of many marine popula-

tions, thereby making them more susceptible to disease. For example, a mid-1980s epidemic among northern European harbor seals was preceded by increased temperatures, which promoted higher than normal densities of these seals on land and thus provided an ideal setting for transmission of disease (33). The El Niño Southern Oscillation (ENSO) is one of the more visible climate variations that has had large-scale effects on marine ecosystems. During the past 5000 years, ENSO events have typically occurred at a frequency of one to two per decade (34) but, since the mid-1970s, have occurred more often and persisted longer (35). The impact of these climatological events on marine species is clearly evident among corals, which are known to bleach (expulsion of the symbiotic algae) in response to a range of environmental stresses (36). The coral bleaching of 1998 was the most geographically extensive and severe in recorded history (37), causing significant mortality worldwide (38). The stress for many of these coral reef systems seems to be the result of long-term exposure to unusually high water temperatures resulting from a prolonged ENSO event (39). Although reported only as bleaching-related mortality, demise of some corals is likely to have been accelerated by opportunistic infections (40). Given that a bacterium may be contributing to bleaching in at least one coral pathosystem (41), additional research is needed to fully evaluate the interaction between bleaching and disease.

In addition, ENSO events have been implicated in interannual variation in Dermo, a disease of the Eastern oyster (*Crassostrea virginica*), caused by the protozoan parasite *Per-*

kinsus marinus (42). Throughout the Gulf of Mexico, where Dermo is endemic, *P. marinus* infection intensity closely follows the ENSO cycle. Gulfwide *P. marinus* infection intensity and prevalence drop during El Niño events and rise during La Niña events. La Niña events tend to produce warm, dry conditions in the Gulf of Mexico, which can trigger *P. marinus* outbreaks; El Niño events produce cold wet conditions, which reduce prevalence and intensity. The apparent relation between *P. marinus* infection in Gulf of Mexico oyster populations and ENSO suggests that epidemics may be predictable from climate models. Because *P. marinus* controls oyster populations in the Gulf, the status of ENSO events needs to be considered when setting management strategies for oyster populations.

Recent ENSOs also affected species ranges and composition of marine communities (43), which in turn, produced cascading changes through all trophic levels over large spatial scales (44). In particular, warming oceans have had a number of consequences for disease dynamics. The almost 25-year trend of warming winter temperatures (45) on the east coast of the United States may have facilitated the spread of both Dermo (Fig. 4) and MSX (multinucleated spore unknown), an oyster disease caused by *Haplosporidium nelsoni* (46, 47). Throughout the 1980s, diseases spread and intensified in oyster populations throughout Chesapeake Bay. In the early 1990s, Dermo became epidemic in Delaware Bay and by 1995 occurred in Maine. In the summer of 1998, MSX was epidemic in oyster populations of Long Island, New York, resulting in extensive mortality. The northward expansion of these shellfish diseases has been attributed to environmental changes that favor the parasites (44, 45). For MSX, warmer winters decreased parasite mortality, resulting in oysters retaining heavy infections. A warming trend produces an environment that is likely to favor northward range expansion of *P. marinus* into new, susceptible host populations (46).

Direct role of human activity. Human activity has greatly enhanced global transport of marine species (48) including pathogens. Human-facilitated epidemics are most common in aquaculture (49, 50) and, in fact, it has been suggested (49) that most mass mortalities of bivalve mollusks have resulted from transfer of infectious stocks. Because of obvious economic concerns, spread of shrimp viral diseases has been generally well documented. The infectious hypodermal and hematopoietic necrosis virus, which appears to have its origins in the Indo-Pacific, now occurs throughout the world causing catastrophic epidemics in aquaculture facilities. Moreover, its host range appears to include a wild species of shrimp and its spread was partially responsible for halting Mexican commercial

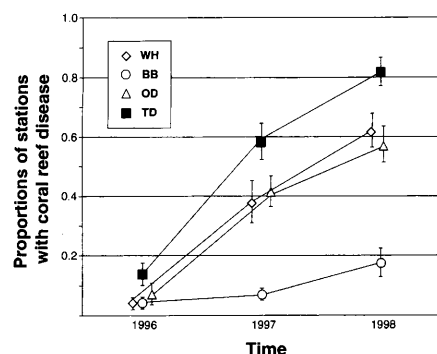


Fig. 1. The proportion of reef stations in the Florida Keys National Marine Sanctuary with coral disease (85). Disease became significantly more widespread (F = Wald's chi-square divided by degrees of freedom for year effects) for black band (BB, open circles; F = 9.28, P < 0.0002), white diseases (WH, open diamonds; F = 33.48, P < 0.0001), other diseases (OD, open triangles; F = 21.10, P < 0.0001), and total diseases (TD, closed squares; F = 42.33, P < 0.0001; df = 2,78) from 1996 to 1998. Whereas only 26 of 160 stations (16%) were diseased in 1996, 131 (82%) were in 1998. Further, there has also been an increase in the number of species affected. Whereas only 11 species exhibited signs of disease in 1996 (27% of all species in the survey), by 1998, this number had risen to 35 species (85% of all species). Over the same period, living coral cover on the deep fore-reef (17 to 18 m depth) of Carysfort Reef has declined from 13.3% to 5.3% (a 60% reduction of living coral cover on this reef during the survey).



Fig. 2. Mass mortality of monk seals due to morbillivirus or algal toxin in Mauritania, 1997 (Photo by K. van der Meulen, Seal Rehabilitation and Research Center, Pieterburen, Netherlands).

fishery for a few years (51). A large-scale epidemic of herpesvirus-infected Australasian pilchard (*Sardinops sagax*) spread at about 30 km/day from Anxious Bay, South Australia, to cover a total of about 5000 km of Australian coastline from March to September 1995. Evidence suggests that the virus may have been introduced with frozen pilchards imported to feed sea-caged southern bluefin tuna in South Australia (52). A second large-scale epidemic started in October 1998 in Spenser Gulf, Australia, where frozen imported pilchard feed also has been used (53).

Habitat degradation and pollutant inputs, often brought about by human activity, can facilitate disease outbreaks (54). Work on aquatic mammals indicates that pollutants, for example, organochlorides, have immunotoxic properties, impairing natural killer cell activity, as well as a series of mitogen- and antigen-induced T cell responses (55). Because most coastal waters are typically affected by suites of anthropogenic pollutants and inputs, it often is difficult to identify any one specific cause of deteriorating health or disease outbreak. Recent mass mortality off the coast of Mauritania among Mediterranean monk seals, thought to have resulted from the transmission of DMV from dolphins that had died in the same area (56), may have been facilitated or caused by a toxic algal bloom (15, 57). In addition to directly affecting marine hosts, some infections can compromise the host immune system, which is then capable of serving as a reservoir for other infectious agents (58) including many "new" viruses, some of which are pathogenic to humans and domestic animals (28, 59). In contrast, we know little about how habitat degradation facilitates disease emergence, particularly among invertebrates. To date, much of what is known comes from a limited number of correlative studies that show increased prevalence of coral diseases (60) and increased parasite burden in oysters (61) in more degraded sites. Silt in run-off has been a leading cause of coral mortality worldwide. In one case, the emergence of a new disease,

aspergillosis of sea fan corals, has been associated with transmission of disease in terrestrial run-off (29, 30). A better understanding of the origins of emergent disease and invertebrate immunity (62) is needed before we can evaluate the role of changing environments in host-pathogen interactions. Studies of invertebrate resistance to disease will not only provide important insights for management of commercial and natural populations, but also will yield molecules and compounds with biomedical applications (63) (Fig. 4).

Oceans as Incubators and Conveyors of Human Diseases

Many potentially pathogenic organisms, including *Aeromonas*, *Clostridium*, *Klebsiella*, *Legionella*, *Listeria*, *Pseudomonas*, and *Vibrio*, are naturally active in estuaries and oceans (64); some can persist in dormant, unculturable, but viable states (65). Human activity has also added to the pathogen load in the oceans, primarily through sewage discharges, although storm waters also carry human and animal wastes (66). Other non-point sources of pollution are important. For example, recent studies showed that materials in septic tanks and injection wells moved rapidly seaward (67) and into offshore marine waters within a day (68). Once in coastal waters, pathogens can persist (64, 69) and infect humans through recreational exposure or consumption of contaminated fish or shellfish (70). In urban centers where rapid growth continues, these problems are expected to increase (71), and the added burden on wastewater treatment systems is likely to increase the probability of serious human fecal contamination of drinking water by a variety of pathogens including hepatitis A, Cocksackie, and Norwalk-like viruses (72). Cholera provides a good example of how human health threats from the ocean are affected by climate (73). The causative agent of cholera (*Vibrio cholerae*) is associated with marine plankton. Annual epidemics that occur in Bangladesh have been correlated with sea surface temperature and sea surface height. Clinical data underlying incidence and severity of epidem-

ics have been linked to water temperature, turbidity, and plankton blooms, notably zooplankton. By remote sensing, monitoring of sea surface temperature, turbidity, chlorophyll, and sea surface height, it has been possible to determine which environmental parameters strongly correlate with epidemics. Because nutrients enter riverine and coastal systems during heavy rainfall, often triggering plankton blooms, ENSO-related events also play a role in cholera outbreaks (74). ENSO-related increases in rainfall are likely also to intensify microbial and toxic contaminant inputs from terrestrial sources (75) and to promote the emergence of novel infections among marine species and humans (76).

New Tools in the Study of Marine Diseases

Fundamental to the understanding of infectious disease is the identification, isolation, and characterization of the causative agent, enabling development of specific diagnostic methods for epidemiological surveys and host resistance. Identification of marine pathogens has been problematic because of ineffective culturing techniques. Recent advances in molecular biology provide approaches in the identification of species, strains, and life-cycle stages of microbial pathogens (77). Species-specific DNA probes from ribosomal sequences provide accurate and rapid diagnostic tools for the evaluation of environmental samples. When used in combination with the polymerase chain reaction (PCR), these probes allow detection and identification of an increasing number of etiological agents. Selecting the NTS region between the 5S and SSU ribosomal RNA genes as the target nucleotide sequence, a semiquantitative PCR-based assay was developed for the diagnosis of *P. marinus* in oyster tissues (78, 79). PCR-based assays are more specific than most current methodologies, and can be designed to be strain-specific, species-specific, or

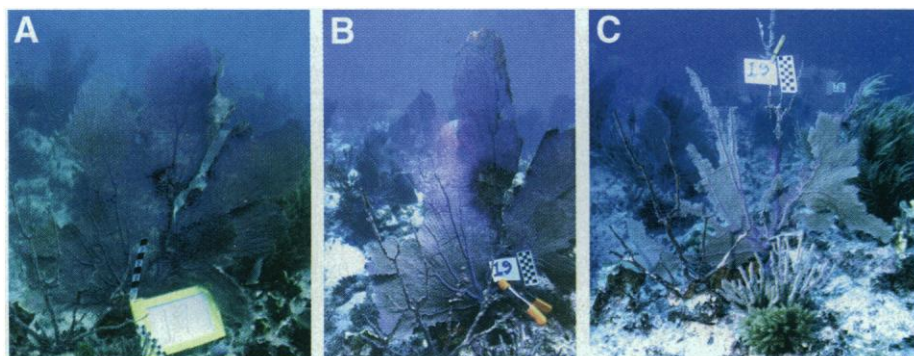


Fig. 3. Time series of damage caused by *Aspergillus sydowii* on a monitored sea fan: (A) 16 June 1996, (B) 8 August 1996, (C) 10 November 1997 (113) (Photos by Craig Quirolo, Reef Relief).

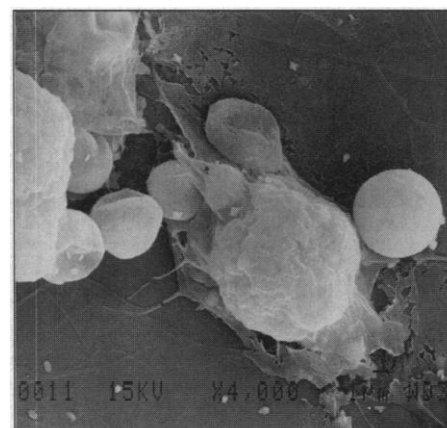


Fig. 4. Phagocytosis of *Perkinsus marinus* by eastern oyster (*Crassostrea virginica*) hemocytes (J. D. Gauthier and G. R. Vasta). *Perkinsus marinus* is one of few marine protistan pathogens in clonal culture.

genus-specific (68, 77). The recent application of "real-time" PCR to field diagnostics of microbial pathogens reveals the potential of this approach for the rapid and reliable diagnosis of diseases in the marine environment (80).

A critical need in the study of marine diseases is epidemiologically structured historical records and more quantitative analyses of marine epidemics. Epidemiological studies of marine disease have lagged far behind work in terrestrial communities, where analyses have revealed links between host immunity, modes of transmission, and disease virulence (32, 81). Epidemiological studies of marine organisms have been limited to a few selected host species (33, 46, 82) and although these studies illustrate the utility of modeling approaches, broader usage has been impeded by limited data sets for most natural populations, and complexities of strong environmental signals operating in open marine ecosystems. For some taxa, like corals (83), monitoring data are currently being compiled to elucidate disease processes; however, greater effort is needed if we are to gain a broader understanding of disease dynamics in the ocean. For instance, corals seem already to be acting as indicator species of a heightened disease load and are tractable for examining the frequencies of temperature stress and disease emergence in tropical oceans because they are sessile (which allows us to pinpoint impacted locations), secrete large and permanent skeletons (which can record the passage of disease, even after the coral is dead), and they are modular (which allows signs of chronic infections to endure without killing the whole colony, unlike small unitary species).

Conclusion

By the measures cited, it can be concluded that reports of diseases in the ocean are on the rise. We have illustrated this point using species that are important economically and ecologically like shellfish, corals, and marine mammals. Epidemics must also be affecting less apparent species, many of which may be disappearing without notice (84). Most new diseases occur by host shifts and not by the emergence of new microorganisms. Contributing to the emergence of new diseases would be a long-term warming trend, coupled with extreme ENSO events and human activities that have modified marine communities. The inability even to identify most causative agents and the lack of standard epidemiological data for diseased populations limit our ability to examine host-pathogen interactions, to analyze changes in disease dynamics, and to assess the impact of diseases on host populations and associated communities in the world oceans. Given this prospect, there is an urgent need for interdisciplinary studies of marine diseases, focusing on the develop-

ment of better molecular and computational tools and on understanding mechanisms of disease resistance in marine organisms.

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- A human disease is classified as new when its symptoms are distinct from those previously known, it becomes virulent, it becomes newly detectable, or it affects different segments of the host population [R. Levins et al., *Am. Sci.* **82**, 52 (1994)].
- In the disease literature, the term "signs," rather than "symptoms," is suggested as more appropriate for nonhuman subjects; we use the term symptom (as well as "epidemic," "epidemiological," and so forth) more broadly to include both human and nonhuman hosts.
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