

# 9 Diseases and the Conservation of Marine Biodiversity

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Natural populations, both terrestrial and marine, appear to be under increasing threats from emerging disease (Daszak et al. 2000; Harvell et al. 1999) (Table 9.1). Well-publicized events such as the widespread mortality of 18,000 harbor seals (*Phoca vitulina*) in Europe in 1988 (Heide-Jorgensen et al. 1992), the sudden death of 400 Florida manatees (*Trichechus manatus*) in 1996 (Bossart et al. 1998), and recent mass mortalities affecting coralline algae (Littler and L09ittler 1995), kelp (Cole and Babcock 1996), seagrasses (Roblee et al. 1991), corals (Cerrano et al. 2000; Richardson et al. 1998), crustaceans and mollusks (Alstatt et al. 1996; Ford et al. 1999; Moyer et al. 1993), and fishes (Jones et al. 1997; Rahimian and Thulin 1996) have raised serious concerns about the state of the marine environment. In spite of this, there has been little progress in understanding the role of diseases in the sea. In this chapter we review the emerging role of diseases in the marine realm. In doing so, we will summarize what is currently known about the role of pathogens<sup>1</sup> in marine ecosystems, highlighting causal links between disease outbreaks and recent changes in the environment. This is not meant to be an exhaustive review of diseases of marine organisms, but rather an overview of general patterns and principles. In this review, we have relied on studies of diseases affecting a small subset of species that are commercially or ecologically important because so few data are available on diseases of most marine species.

## Diseases in the Sea

Although the importance of pathogens in terrestrial ecosystems has long been recognized (Grenfell and Dobson 1995), the role of diseases in most marine communities is comparatively unknown (Harvell et al. 1999; Peters 1993; Richardson et al. 1998). This paucity of information is surprising, given that the sea is a “microbial soup” supporting an immeasurable abundance and diversity of potential parasites. Although diseases clearly can have significant impacts on marine species and communities (Harvell et al. 1999), we lack a basic understanding of the full range of roles diseases play in the marine realm. Foremost, we lack information on disease processes such as the dynamics of host population regulation, the factors that promote disease emergence and outbreak, and the mechanisms of pathogen transmission. Exceptions to this dearth of information are a few ecologically or economically important marine taxa, for which some data are available. In the following sections, we discuss these examples of marine diseases to highlight what we currently know about roles that pathogens play in marine ecosystems.

## Population Regulation

Viruses are the most abundant plankton in the sea, typically numbering in the tens of billions per liter (Furhman 1999). Not surprisingly, these virioplankton

**TABLE 9.1. Mass Mortalities among Natural Populations of Selected Marine Species**

<i>Start Date</i>	<i>Host Species</i>	<i>Outbreak Location</i>	<i>Pathogen Identity</i>	<i>Estimated Mortality (percent)</i>	<i>Environ Correlates</i>	<i>Ref.</i>
1938	Sponges	N Caribbean	fungus?	70–95	ND	1
1931	<i>Zostrea</i> (seagrasses)	N America, Europe	slime mold	extensive	high T	2
1946	<i>Crassostrea</i> (oyster)	Gulf Coast, USA	<i>Perkinsus marinus</i>	extensive	high T, sal	3
1954	<i>Clupea</i> (herring)	Gulf St. Lawrence	<i>Ichthyophonus hoferi</i>	50	ND	4
1955	<i>Lobodon</i> (seal)	Antarctica	virus	extensive	ND	5
1960	<i>Halichoerus</i> (seal)	St. Kilda, Scotland	<i>Pneumococcus</i> sp.	50	ND	6
1975	<i>Heliaster</i> (starfish)	W USA	?	<100	high T	7
1980	<i>Strongylocentrotus</i> (urchin)	NW Atlantic	amoeba?	>50	ND	8
1980	<i>Ostrea</i> (oyster)	Netherlands	<i>Bonamia ostreae</i>	extensive	ND	9
1981	<i>Acropora</i> (coral)	Caribbean-wide	bacterium?	<100	ND	10
1982	<i>Gorgonia</i> (coral)	Central America	?	extensive	high T	11
1982–6	<i>Haliotis</i> (abalone)	Australia	<i>Perkinsus</i> sp.	extensive	high T	12
1983	Scleractinian Corals	Caribbean-wide	microbial consortium	seasonal		13
1983	<i>Patinopecten</i> (scallop)	W Canada	<i>Perkinsus qugwadi</i>	extensive	ND	14
1983	<i>Diadema</i> (urchin)	Caribbean-wide	bacterium?	98	high T	15
1985	<i>Haliotis</i> (abalone)	NE Pacific	?	>95	high T	16
1987	<i>Thalassia</i> (seagrass)	Florida, USA	slime mold	<95	high T, sal	17
1988	<i>Argopecten</i> (scallop)	N Caribbean	protozoan	extensive	ND	18
1988	<i>Phoca</i> (seals)	NW Europe	virus	~70	pollution	19
1988	<i>Phocoena</i> (porpoise)	NE Ireland	virus	?	pollution	20
1989	<i>Argopecten</i> (scallop)	E Canada	<i>Perkinsus karlssoni</i>	extensive	ND	21
1989	<i>Phoca</i> (seals)	Lake Baikal	virus	>10	ND	22
1990	<i>Stenella</i> (dolphin)	W Mediterranean	virus	>20	pollution	23

*(continued)*

can play a major role in regulating population density and diversity of their hosts, which include bacterio- and phytoplankton. By some accounts, as much as 50 percent of all bacteria and cyanobacteria in the open ocean are infected by viruses. As a result, viral pathogens can significantly affect primary production in the sea (reviewed in Furhman 1999; Wommack and Collwell 2000). Suttle et al. (1990) found that viruses can produce as significant an impact on phytoplankton abundance as grazing and nutrient limitation and can reduce primary productivity by as

much as 78 percent. Given the importance of phytoplankton in virtually all marine food webs, the importance of diseases cannot be underestimated.

### Catastrophic Population Declines and Community Shifts

Perhaps more visible examples of pathogen impacts are the episodic and often unpredictable disease outbreaks that severely diminish host population densities (see Table 9.1). When these outbreaks affect ecologically important species, the reduction in host

TABLE 9.1. (continued)

Start Date	Host Species	Outbreak Location	Pathogen Identity	Estimated Mortality (percent)	Environ Correlates	Ref.
1991	<i>Clupea</i> (herring)	W Sweden	<i>Ichthyophonus hoferi</i>	>10	low T	24
1992	<i>Ecklonia</i> (kelp)	NE New Zealand	?	40-100	high Turb	25
1993	Coralline Algae	S Pacific	bacterium?	extensive	ND	26
1995	<i>Strongylocentrotus</i> (urchin)	Norway	nematode?	~90	ND	27
1995	<i>Gorgonia</i> (corals)	Caribbean-Wide	fungus	extensive	ND	28
1995	<i>Dichocoenia</i> and others (coral)	Florida, USA	bacterium	<38	seasonal	29
1996	<i>Diploria</i> and others (coral)	Puerto Rico	bacterium	extensive	seasonal, hur	30
1997	<i>Porolithon</i> (algae)	Samoa	fungus	extensive	ND	31
1997	<i>Sardinops</i> (pilchard)	S Australia	virus?	extensive	ND	32
1997	<i>Monachus</i> (seal)	W Africa	virus/toxin	>75	ND	33
1999	Gorgonian Corals	NW Mediterranean	protozoan/fungi?	>20	high T	34
2000	<i>Phoca</i> (seal)	Caspian Sea	virus	extensive	ND	35
2002	<i>Phoca</i> (seal)	N Europe	virus	>10	ND	36
2003	Krill	NW US	<i>Collinia</i> sp. (ciliate)	?	ND	37

1. Galtsoff et al. (1939), Smith (1939); 2. Muehlstein et al. (1988), Rasmussen (1977); 3. Soniat (1996); 4. Sinderman (1956); 5. Bengston et al. (1991); 6. Gallacher and Waters (1964); 7. Dungan et al. (1982); 8. Miller and Colodey (1983), Scheibling and Stephenson (1984), Jones and Scheibling (1985); 9. van Banning (1991); 10. Antonius (1981), Gladfelter (1982), Peters (1993); 11. Guzmán and Cortéz (1984); 12. Lester et al. (1990); 13. Rützler et al. (1983), Carlton and Richardson (1995); 14. Blackburn et al. (1998), Bower et al. (1998); 15. Lessios et al. (1984b); 16. Lafferty and Kuris (1993); 17. Roblee et al. (1991); 18. Moyer et al. (1993); 19. Kennedy (1998), Osterhaus and Vedder (1988); 20. Barrett et al. (1993); 21. McGladdery et al. (1991); 22. Osterhaus et al. (1989); 23. Domingo et al. (1990), Barrett et al. (1993); 24. Rahimian and Thulin (1996); 25. Cole and Babcock (1996); 26. Littler and Littler (1995); 27. Skadsheim et al. (1995); 28. Nagelkerken et al. (1997), Nagelkerken et al. (1996); 29. Richardson et al. 1998), 30. Bruckner and Bruckner (1997); 31. Littler and Littler (1998); 32. Hyatt et al. (1997), Whittington et al. (1997); 33. Hernández et al. (1998), Osterhaus et al. (1998); 34. Cerrano et al. (2000); 35. Kennedy et al. (2000); 36. Jensen et al. (2002); 37. Gómez-Gutiérrez et al. (2003)

For each reported outbreak, location, pathogen identity, if known, and environmental correlates are shown: T = temperature, ND= no data, sal = salinity, turb = turbidity, hur = hurricane.

densities can result in dramatic changes in community structure. Thus disease can play a role in effecting whole community shifts (Scheffer et al. 2001). For instance, sea urchins are a dominant grazer in many temperate benthic communities and can turn extensive kelp beds into much less productive "barrens" (Harrold and Pearse 1987). However, outbreaks of disease can decimate the urchin populations and allow reestablishment of kelp beds (Lafferty and Kushner 2000). Over a period of years, the urchins increase in density and return kelp beds to barrens, in what ap-

pears to be a relatively predictable cycle of community phase shifts (Hagen 1995).

In some cases, particularly virulent disease outbreaks can drive host populations below a threshold from which they cannot recover, or can do so only at a very slow rate. A particularly well documented example of this is the Caribbean-wide die-off of the long-spined sea urchin (*Diadema antillarum*) in the early 1980s (Lessios et al. 1984a). During this epidemic, which was caused by a pathogen of unknown identity, the entire Caribbean population of *D. antillarum* was

reduced by approximately 98 percent, providing a spectacular example of the transmission potential of a novel, virulent marine pathogen. Within a year, the pathogen, apparently dispersed by major surface currents, affected approximately 3.5 million km<sup>2</sup>. Even after a decade, Lessios (1995) found little evidence of recovery by this sea urchin, which remained at less than 3.5 percent of its pre-epidemic levels. A barrier to recovery could be the extremely low population densities that limit fertilization success (the Allee effect) and thus larval supply in this broadcast spawning species (Levitan and McGovern, Chapter 4). The demise of *Diadema*, an important herbivore, had profound consequences for Caribbean reefs, especially those reefs already impacted by overfishing, by facilitating a shift from communities dominated by corals to those dominated by macroalgae (Hughes 1994).

Another example of a population driven below a recovery point is the once strikingly abundant black abalone (*Haliotis cracherodii*) in southern California. Populations started experiencing site-specific mass mortalities related to withered foot syndrome that were initially thought to be a result of warm water events, such as El Niño/Southern Oscillation (ENSO) (Davis et al. 1992). However, subsequent analysis of the spatiotemporal pattern in the die-offs indicated an infectious process (Lafferty and Kuris 1993), which was later found to be associated with a rickettsia (Gardner et al. 1995). Little to no recovery has been observed to date, perhaps because abalone, like urchins, are relatively sessile, yet must be in close proximity for successful spawning. Disease-mediated reduction of an ecologically dominant host species to a point where recovery is uncertain appears to be the case for a number of other marine species, including the Caribbean staghorn coral (*Acropora cervicornis*; Gladfelter 1982), California sea star (*Heliaster kubiniji*; Dungan et al. 1982), and sea fan corals (*Gorgonia* spp.; Garzón-Ferreira and Zea 1992; Guzmán and Cortéz 1984).

### Extinction

The most extreme case of disease impact is extinction. Although there are some prominent examples of dis-

ease-mediated extinction of terrestrial species—amphibians from chytrid fungus (Daszak et al. 1999) and Hawaiian birds from avian malaria (Warner 1968), for instance—the extent to which diseases pose extinction threats in the sea is currently unknown. There is on record, however, a “disease-related” extinction of the gastropod limpet, *Lottia alveus*. When the eelgrass *Zostera marina*, the only known habitat for the limpet, suffered catastrophic disease-related declines along the North Atlantic coast during the early 1930s, the limpet disappeared. Carlton et al. (1991) suggested that the extinction of the limpet was attributed to its high degree of habitat specialization. Although more examples are not at hand, this issue of disease-related extinctions should be considered seriously. Because the rates of disease impact are so high in obvious indicator species like corals and marine mammals, it is plausible that less well monitored and less obvious taxa could be disappearing unnoticed.

### Are Diseases Increasing?

Fundamental to addressing this question is determining whether the growing number of reports of mass mortalities, new diseases, and new pathogens is evidence of a real increase or an artifact of heightened effort and improved diagnostics. In a recent report, Hayes et al. (2001) proposed that increased deposition of pathogen-laden and iron-rich “African dust” in the Caribbean was an important factor in marine disease outbreaks during the last several decades. As evidence, they present compilations of disease reports by Epstein (1996) and Harvell et al. (1999) as time series data, showing increased reporting of disease outbreaks affecting marine species. However, these compilations cannot be taken as an appropriate test of their hypothesis without considering potential sources of reporting biases. For instance, Harvell et al. (1999) only list outbreaks affecting selected marine species. Moreover, in the absence of longer-term baseline or historical data, it is simply not possible to gauge the significance of recent disease outbreaks against “normal” levels.

The increased attention to diseases of marine mammals by pathologists following a number of large, well-publicized die-offs, coupled with the development of improved techniques for detecting pathogens, further confuses the interpretation of increased reports of diseases in marine mammals. Although novel viral, bacterial, fungal, and protozoal pathogens now are reported regularly in marine mammals, it is likely that some of these pathogens previously existed but only recently were identified in marine mammals. Forty years ago, most of the diseases reported in marine mammals were caused by macroparasites detectable by unaided human eyes (Kinne 1985). Twenty years ago, a number of novel bacteria were cultured as monitoring programs improved and pathologists examined fresh material (Dunn 1990). Over the last 15 years, most novel viruses (Mamaev et al. 1995; Osterhaus et al. 1998, 2000), protozoa (Cole et al. 2000; LaPointe et al. 1998; Lindsay et al. 2000), and biotoxins (Scholin et al. 2000), were identified, primarily due to the advent of newly developed molecular techniques. For example, domoic acid intoxication was first reported as the cause of a die-off of California sea lions (*Zalophus californianus*) in 1998 (Scholin et al. 2000), shortly after the development of rapid diagnostic tests for this biotoxin. However, sea lions and fur seals exhibiting symptoms similar to those caused by domoic acid intoxication had been observed for at least 20 years previous to this.

Investigations of carcasses of stranded marine mammals and those hunted by humans suggest an increase in the prevalence of neoplasia (i.e., tumors) over the past 30 years. In 1974, there were only seven reports in the literature of neoplasia in marine mammals (Mawdesley-Thomas 1974). By 1983, a survey of stranded animals in California revealed a 2.5 percent prevalence of neoplasia in 1,500 animals (Howard et al. 1983). Over the last decade, prevalence of neoplasia in sexually mature stranded California sea lions was 18 percent (Gulland et al. 1996b), while 50 percent of beluga whales (*Delphinapterus leucas*) stranded in the St. Lawrence estuary had one or more tumors (Martineau et al. 1994). However, these surveys had

different sampling biases and were performed on different species in different years.

Long-term data on single species, collected by standardized sampling methods, are needed to address the question of whether diseases are increasing. One such dataset could be that of green turtle fibropapillomas (GTFP). GTFP is a pandemic tumorous condition affecting the endangered green turtle (*Chelonia mydas*) (Williams et al. 1994). This disease appears to be associated with a virus of unknown identity (Herbst 1994), which can cause tumors up to 30 centimeters in length (George 1997). Since the 1980s, prevalence has increased dramatically, affecting as much as 92 percent of the green turtle populations at some sites (Herbst 1994). This is an impressive example because the tumors are large and very apparent and therefore unlikely to have been overlooked in earlier work, and the dataset was collected from one species over time. Fibropapillomatosis has also been documented recently in the olive Ridley turtle (*Lepidochelys olivacea*) (Aguirre et al. 1999), and the loggerhead turtle (*Caretta caretta*) (Lackovich et al. 1999), suggesting an increase in the prevalence of this condition.

In addition to monitoring data, other novel approaches to establishing baseline conditions are possible. For instance, using the fossil record as a baseline for coral assemblages, Aronson and Precht (1997) and Greenstein et al. (1998) demonstrated that the demise of the staghorn coral (*Acropora cervicornis*) in the mid-1980s was unique to recent (Holocene–Pleistocene) geologic history.

### Role of Climate Change and Human Activity

An alternative tack in assessing the growing importance of diseases in the sea might be to ask the following questions: (1) What factors increase the likelihood of disease outbreaks? and (2) How have these factors changed over time? Based on the observation that many of the diseases we are seeing now have emerged because of relatively recent climate- and human-mediated changes in the environment, several

studies have concluded that, indeed, there has been an increase in diseases affecting both marine and terrestrial species (Daszak et al. 2000). There are at least two ways in which climate change and human activity can increase the emergence of new diseases: by increasing the rate of contact between novel pathogens and naive (i.e., susceptible) hosts, and by altering the environment in favor of the pathogen.

### Creating Novel Host–Pathogen Interactions

There is increasing evidence that many of the recent disease outbreaks have resulted from the introduction of known pathogens to naive host populations, often facilitated by changes in climate or human activity. For example, *Perkinsus marinus*, an endoparasitic protozoan that causes Dermo disease in the eastern oyster (*Crassostrea virginica*), appears to have extended its range northward in response to a trend of warming winter water temperatures on the eastern coast of the United States (Cook et al. 1998). Although the protozoan is considered a “southern,” warm-water parasite, common along Florida’s Gulf coast, increasing winter water temperatures along the northeast coast reduced its overwintering mortality and allowed subsequent development of epidemics over a 500 kilometer range north of the Chesapeake Bay. *P. marinus* has since been detected as far north as Maine (Cook et al. 1998).

Climate-mediated changes in host–pathogen interaction can be quite complex, particularly when pathogens and parasites are vectorborne. For instance, in association with the 1997–98 ENSO event, the junction of the warmer Kuroshio and the cooler Oyashio currents moved 3,000 kilometers northward from Kyushu to Hokkaido, Japan. This shift increased water temperatures in Hokkaido, resulting in a bloom of krill, which are intermediate hosts of nematode parasites. Steller sea lions (*Eumetopias jubatus*), which only occur in Hokkaido, became parasitized as did cetaceans that migrated to feed on the krill. Fish feeding on the krill also harbored nematodes, which were consumed by new hosts, humans (Ishikura et al. 1998).

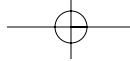
The emergence of new diseases following host

shifts (i.e., known pathogens affecting previously unaffected hosts) has also been accelerated by human activity. Increased contact between marine mammals and humans or domestic animals appears to have enhanced transmission of disease to marine mammals. For instance, canine distemper virus was thought to have been introduced into crab-eater seals (*Lobodon carcinophagus*) in Antarctica by contacts with infected sled dogs used for an expedition (Bengston et al. 1991). Recent outbreaks of canine distemper have occurred in Baikal (*Phoca sibirica*) and Caspian (*P. caspica*) seals, although the source of infection is unclear (Kennedy et al. 2000; Mamaev et al. 1995). Other examples include detection of human influenza virus in seals, although the virus also could have been acquired from birds (Hinshaw et al. 1984). More than 400 mostly immature harbor seals died along the New England coast between December 1979 and October 1980 of acute pneumonia associated with influenza virus A (Geraci et al. 1982). Influenza B virus was recently isolated from harbor seals in the Netherlands, four to five years after the serotype was prevalent in an outbreak affecting humans (Osterhaus et al. 2000).

### Shifting the Host–Pathogen Balance

In addition to creating new host–pathogen interactions, climate change and human activity have altered or degraded marine habitats, increasing stress-mediated susceptibility to diseases. Parasitologists have long known that climate determines the distribution of species, while weather influences the timing of disease outbreaks (Dobson and Carper 1993). In the marine realm, water temperatures appear to play an important role in disease dynamics. In particular, ENSO events are linked to outbreaks of diseases affecting a range of marine taxa. Throughout the Gulf of Mexico, infection intensity and prevalence of Dermo, an endemic disease of oysters, drop during the cool and wet El Niño events and rise during warm and dry La Niña events (Powell et al. 1996).

On coral reefs, bleaching<sup>2</sup> coincides with ocean warming during ENSO events (Hoegh-Guldberg 1999). Coral bleaching in 1998 was the most geographically



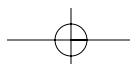
extensive and severe in recorded history, causing significant coral mortality worldwide (Baird and Marshall 1998; Strong et al. 1998; Wilkinson et al. 1999). Evidence suggests that some bleaching-related mortality can be exacerbated by increased susceptibility of corals to opportunistic diseases. During the 1997–98 ENSO event, mass bleaching and subsequent mortality in the gorgonian *Briareum asbestinum* were associated with an infectious cyanobacterium (Harvell et al. 2001). Recently, an extensive mass mortality of gorgonian corals (60–100 percent mortality) in the Ligurian Sea (northwest Mediterranean) was coincident with sudden increases in water temperature down to 50 m, which apparently increased coral susceptibility to a variety of opportunistic pathogens (Cerrano et al. 2000). Because events as severe as the 1998 ENSO are expected to become commonplace within 20 years, catastrophic or significant loss of coral reefs on a global scale is possible (Hoegh-Guldberg 1999). We predict that opportunistic diseases are likely to accelerate and exacerbate this decline (Harvell et al. 2002).

Because most marine pathogens are not readily cultured, it has not been possible to determine the direct link between temperature and disease. An exception is *Aspergillus sydowii*, a fungal pathogen of sea fan corals (Geiser et al. 1998; Smith et al. 1996). In a recent study, Alker et al. (2001) showed that the optimum temperature for *A. sydowii* growth is 30°C, the temperature at which many corals begin to bleach. The higher temperature optimum of the pathogen provides a clear mechanism for temperature-related epidemics.

ENSO events also have dramatic effects on Pacific pinniped populations. Changes in upwelling and currents cause changes in invertebrate and fish populations that have devastating effects on pinnipeds feeding in upwelling regions (Trillmich and Ono 1991). These severe food shortages cause such high neonatal mortality in California sea lions, South American sea lions (*Otaria flavescens*), and South American fur seals (*Arctocephalus australis*) that whole cohorts from El Niño years could be missing. Yearling animals might survive but can show reduced growth rates and in-

creased susceptibility to infectious diseases. Leptospirosis is a bacterial disease of California sea lions that recurs at approximately four-year intervals, generally after El Niño years (Gulland et al. 1996a; Vedros et al. 1971). It is caused by *Leptospira pomona*, an organism that can cause disease in a wide range of mammals, including humans, and that is extremely sensitive to changes in environmental temperature, salinity, and pH (Faine 1993). Although the method of transmission between sea lions is unknown, it is likely to be direct, as this bacterium, shed in urine, cannot survive long in the marine environment. Changes in sea lion immune status due to poor nutrition during El Niño years might contribute to disease outbreaks.

Indirect effects of human activities on infectious diseases can occur through degradation of the marine environment, primarily by the deposition of pollutants and poorly treated sewage into the seas. A variety of chemical contaminants, especially polychlorinated biphenyls (PCBs), DDTs, and organometals, can bioaccumulate up the food chain and are found in tissues of marine mammals (O'Shea 1999). The effects of these contaminants on endocrine function, immune competence, and carcinogenesis are well documented in laboratory rodents. Adrenal hyperplasia with associated pathology in Baltic seals has been attributed to exposure to high levels of PCBs (Bergman and Olsson 1986). Both cellular and humoral immunity were reduced in harbor seals fed herring from the Baltic Sea compared to immune responses in seals fed less contaminated Atlantic fish (Ross et al. 1995). Severity of phocine distemper in experimentally infected harbor seals was greater in animals fed diets with higher levels of PCBs (Harder et al. 1992). Contaminants have also been associated with high prevalence of *Leptospira* and calicivirus infections in California sea lions demonstrating premature parturition (Gilmartin et al. 1976). Whether the contaminants had a direct effect on parturition or an indirect one by reducing resistance to the pathogens is unclear in this case. Beluga whales from the St. Lawrence estuary have higher prevalence of infectious disease and neoplasia than belugas from the Arctic, where contaminant levels are



lower, suggesting that contaminant suppression of the immune response is important in causing high disease prevalence in these whales (Martineau et al. 1994). Similarly, experimental exposure of oysters to tributyl tin (TBT) increased infection intensity and mortality (Fisher et al. 1999). As Lafferty and Kuris (1999) point out, however, while many of the stressors that result from increasing human impacts to the aquatic environment are likely to increase disease, some are likely to reduce disease if they lower host population densities, introduce species to regions that lack pathogens, or create conditions that are less suitable for parasites than for their hosts (particularly likely for parasites with complex life cycles). Indeed, because pollutants can impact parasites as well as their hosts, the nature of the relationship between disease and pollution can vary depending on the circumstances. For example, digenetic trematode flatworms and acanthocephalans tend to be negatively affected by a broad range of pollutants; ciliates and nematodes tend to be positively associated with pollutants; eutrophication and thermal effluent tend to favor most parasites; industrial effluent and heavy metals tend to reduce most parasites; and crude oil leads to an increase in some parasites and a decrease in others (Lafferty 1997). Taken together, these examples reinforce the notion that the dynamics of diseases are complex, requiring the consideration of interactions among multiple stressors (e.g., Lenihan et al. 1999).

In addition to increasing contaminant loads, human activity is directly adding to the list of potential pathogens in the sea. A number of fecal bacterial isolates from coastal marine mammals possess multiple antibiotic resistance (Johnson et al. 1998). As these animals have not been previously treated with antibiotics, this probably results from exposure to bacteria of human origin. The detection of *Toxoplasma gondii*, a protozoan that only produces infectious oocysts in the felid definitive host, in cases of encephalitis in harbor seals (Van Pelt and Dieterich 1973), California sea lions (Migaki et al. 1977), sea ot-

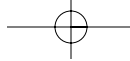
ters (*Enhydra lutris*; Thomas and Cole 1996), West Indian manatees (*Trichechus manatus*; Buegelt and Bonde 1983), and bottle-nosed dolphins (*Tursiops truncatus*; Inskeep et al. 1990) has raised concern over the potential contamination of coastal waters by cat litter. *Giardia lamblia* has recently been detected in feces of seals from both coasts in Canada (Measures and Olson 1999; Olson et al. 1997) but it is still unclear whether this is a common pathogen of marine mammals or one resulting from exposure to terrestrial animal or human feces.

### Diseases as a Threat to Marine Biodiversity

Like other disturbance events (Connell 1978), disease outbreaks have the potential to affect diversity by occasionally removing ecological dominants or resetting succession. For example, disease outbreaks affecting sea urchins in kelp forests (Hagen 1995; Harrold and Pearse 1987; Scheibling et al. 1999) and crown-of-thorns starfish (*Acanthaster planci*) on the Great Barrier Reef (Zan et al. 1990) probably enhance diversity in those communities. However, there is growing concern that the increase in both the frequency and the impact of disease outbreaks (Harvell et al. 1999) is posing a threat to marine biodiversity.

The extent to which increasing diseases will lead to biodiversity losses is difficult to predict. For instance, although eradication of species is possible, current understanding of disease processes suggests that direct, disease-mediated extinction in the sea, as on land, is likely to be rare. This is probably because of a general property of directly transmitted diseases, namely density-dependence of pathogen transmission, which leads to the subsidence of an outbreak when some threshold host-density is reached (Anderson and May 1986; Areneberg et al. 1998). Indeed, of the 133 known marine extinctions (see Carlton et al. 1999; Dulvy et al. 2003; Vermeij 1993), only one, *Lottia alveus*, was associated, albeit indirectly, with disease (Carlton et al. 1991). There is some concern, however, that density dependence can be relaxed; for instance,





by the existence of reservoirs, either biotic or environmental, that allow pathogens to persist even when host species are rare. Because the sea is likely to be more hospitable to pathogens than terrestrial environments, we predict that reservoirs play a greater role in marine disease dynamics. The *Diadema* epidemic reduced urchin populations throughout the Caribbean by more than 98 percent in spite of more than three orders of magnitude variation in host densities (Lessios 1988). More recent outbreaks affecting Caribbean gorgonian corals have also been severe and density independent (Kim and Harvell 2004).

## Conclusions

There is a growing body of literature calling for a better understanding of diseases in conservation efforts (e.g., Daszak et al. 2000; Deem et al. 2001; Lafferty and Gerber 2002; McCallum and Dobson 1995). For example, although endangered species might be protected from disease outbreaks by small population sizes, they also might be put at risk for disease by captive breeding programs or low genetic diversity. Minimizing such risks requires increased vigilance in screening for disease before translocating individuals and in preventing contact with novel diseases and reservoir species. Control of diseases in wild terrestrial populations involves techniques such as vaccination, culling, and quarantine. However, with few exceptions (e.g., Kuris and Lafferty 1992), these traditional terrestrial techniques are unlikely to be effective in open marine environments. Thus marine conservationists are facing likely increases in disease impacts, with only a basic understanding of disease dynamics and without traditional tools for management. We propose three priorities for conservation of marine species and communities under disease stress.

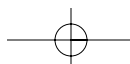
### Priority 1: Long-Term Monitoring

Although some commercially fished species have been adequately monitored, for the most part, the majority of marine taxa have been ignored except during sig-

nificant epidemics. Even when epidemics are monitored, few efforts have monitored host species after the epidemic to assess how affected species and communities recover (e.g., Lessios 1995). The absence of long-term data on disease dynamics and recovery of natural populations severely limits our understanding of the role and importance of diseases in the sea. Moreover, questions such as whether there has been an increase in the frequency and impact of disease outbreaks are largely intractable in the absence of such data. Much of what we currently know comes from studies of epidemics, but long-term monitoring should also reveal the role of endemic diseases in regulating host populations and diversity. Similarly, we know little about the potential for disease-mediated extinction—a threat that is likely to be detectable only through monitoring.

### Priority 2: Better Understanding of Disease Dynamics

The lack of process-oriented information on diseases of most marine organisms should be a concern for conservation biologists. Pathogen identity, transmissibility, dispersal mechanisms, host specificity, factors affecting disease virulence and host susceptibility, and other basic features of a disease are all important data for devising conservation measures. However, in many cases, even the identity of the causative pathogen has not been established (see Table 9.1). Molecular techniques have afforded significant advances in the identification of viral pathogens of marine mammals (e.g., Kennedy 1998) and should be equally useful in other groups. Furthermore, the development of molecular diagnostics should provide the means for early detection of disease outbreaks, as well as for determining host specificity, and even pathogen origin (i.e., phylogeography). Where significant insights have been gained (e.g., in diseases of marine mammals), collaboration among scientists in a broad range of disciplines has occurred. We believe that a multidisciplinary approach, involving microbiologists, pathologists, ecologists, and epidemiolo-



gists, is critical to a fuller understanding of marine pathogens and diseases.

### Priority 3: Consideration of Diseases in Marine Reserves

Marine reserves are an important tool for conservation and potentially can offset losses due to disease by providing habitats with lower levels of anthropogenic stresses and reduced pathogen load. Current criteria for siting marine reserves implicitly include disease; that is, avoiding sites prone to human and natural catastrophes (Roberts et al. 2003). However, an explicit consideration of disease parameters, such as potential sources of pathogens, modes of disease transmission, dispersal potential of infective agents, and occurrences of reservoir species, is clearly needed to predict how marine reserves will affect disease dynamics (see Lafferty and Gerber 2002). For instance, although reserves can facilitate outbreaks of directly transmitted diseases by building up host densities, an increase in species richness within reserves (Halpern and Warner 2002) can reduce the impact of vectored diseases by reducing the role of the most competent disease reservoir (LoGiudice et al. 2003). Given the openness of marine systems, and thus our inability to control access to reserves by pollutants and pathogens (or vectors and reservoir species), effectively incorporating disease concerns is expected to be a considerable challenge for siting and designing marine reserves.

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### Notes

1. Infectious diseases are those transmitted by pathogens, which can be divided on epidemiological grounds into micro- and macroparasites (Anderson and May 1979; May and Anderson 1979). Microparasites, commonly referred to as pathogens, include viruses, bacteria, and fungi and are characterized by their ability to reproduce directly within individual hosts, their small size, and the relatively short duration of infection. Microparasites can be controlled by acquired immunity in the host. Macroparasites, commonly referred to as parasites (e.g., various nematodes, cestodes, and arthropods), produce infective stages that pass between hosts for transmission. Macroparasites are less likely to be controlled by immune responses and are long lived and often visible to the naked eye.

2. Although bleaching, which refers to the disassociation of corals and their zooxanthellae endosymbionts, is not an infectious disease, recent reports suggest that at least for one coral species, bleaching results from an infection by the bacterium *Vibrio* (Kushmaro et al. 1996, 1997).

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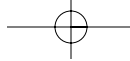
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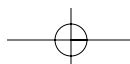
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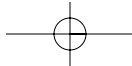
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