

The rising tide of ocean diseases: unsolved problems and research priorities

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New studies have detected a rising number of reports of diseases in marine organisms such as corals, molluscs, turtles, mammals, and echinoderms over the past three decades. Despite the increasing disease load, microbiological, molecular, and theoretical tools for managing disease in the world's oceans are underdeveloped. Review of the new developments in the study of these diseases identifies five major unsolved problems and priorities for future research: (1) detecting origins and reservoirs for marine diseases and tracing the flow of some new pathogens from land to sea; (2) documenting the longevity and host range of infectious stages; (3) evaluating the effect of greater taxonomic diversity of marine relative to terrestrial hosts and pathogens; (4) pinpointing the facilitating role of anthropogenic agents as incubators and conveyors of marine pathogens; (5) adapting epidemiological models to analysis of marine disease.

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Infectious diseases have recently caused substantial community- and ecosystem-wide impacts in marine communities. A long-spined sea urchin disease virtually eradicated urchins from the Caribbean and facilitated a coral to algal shift on many reefs (Hughes *et al.* 1994). Coral diseases, such as white band (Figure 1), white plague, white pox, and aspergilliosis (Figure 2), have caused major changes in Caribbean reef community structure (Aronson and Precht 2001; Weil and Smith 2003; Kim and Harvell 2004). Populations of marine mammals such as seals, otters, and sea lions (Figure 3) have been heavily impacted by diseases (Kim *et al.* 2004), yet the community and ecosystem consequences of these mass mortalities is unknown. These acute and chronic disease events have

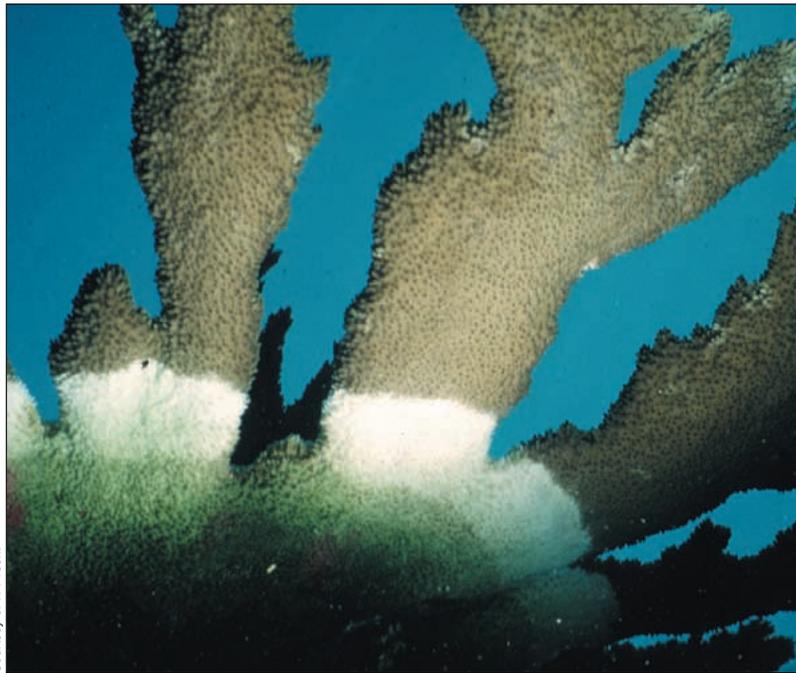
caused serious economic losses in terms of declining fisheries revenue and ecosystem damage. Recent examples of economically destructive infectious diseases include coral diseases in the Caribbean, abalone disease in California, herpes and leptospirosis in California sea lions, lobster disease and salmon virus in Maine, and oyster protozoans in Maryland and Texas.

A 2-year effort by the Marine Disease Working Group at the National Center for Ecological Analysis and Synthesis (MDWG-NCEAS) to evaluate key issues in marine diseases focused on three problems: (1) whether disease impacts are increasing in the ocean; (2) whether current modeling and management approaches for terrestrial organisms are adequate for marine situations; and (3) developing case studies of new statistical and modeling approaches to manage marine organisms that are under disease threat. Both the impacts and prevalence of marine diseases were unusually high in recent decades, but lack of adequate baseline data makes this challenging to quantify (Harvell *et al.* 1999, 2002). Because of the logistical difficulties in conducting marine research and monitoring, many marine mortality events probably go undetected or are poorly understood. Often, the specific cause of the disease outbreak, whether an infectious agent (such as viruses, bacteria, fungi, protozoans, and macroparasites such as helminthes and nematodes) or non-infectious agent, has not even been identified. In addition, activities that scientists predict will increase disease occurrence are on the rise (Lafferty *et al.* in press). For example, ocean aquaculture increased two-fold from 1996–1998 (Pew Oceans Commission 2003); the quantities of ballast water from shipping has increased; the rate of new invertebrate introductions to the marine environment has risen; many marine fisheries are collapsing; and global temperatures

In a nutshell:

- An increasing incidence of disease has been detected in marine organisms such as corals, molluscs, marine mammals, turtles, and echinoderms, over the past three decades
- Spread rates appear faster in some marine epidemics than in terrestrial ones
- Controls applied for terrestrial outbreaks (quarantine, culling, vaccination) are not yet used in the ocean
- Some marine pathogens originate on land, then flow downstream into oceans
- Marine disease can alter populations and cause major changes in marine communities

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Courtesy of W. Precht

Figure 1. Caribbean elkhorn coral (*Acropora palmate*) infected with white band disease.

are expected to increase. Each of these activities has the potential to substantially accelerate the transmission rate of new pathogens and some will also make the hosts more susceptible.

Quantitative analysis of the scientific literature of the past three decades indicates a large increase in the number of reports about marine diseases in corals, turtles, molluscs, marine mammals, and echinoderms (Ward and Lafferty 2004; Figure 4). Lack of baseline data for marine organism diseases makes quantifying the magnitude of this increase difficult, but Ward and Lafferty's (2004) comprehensive, carefully designed quantitative study is the best estimate so far. They used literature reports as a proxy for actual events, and tested the reliability with known disease events, such as rabies outbreaks. Other groups of marine organisms, including crustaceans, seagrasses, and marine fishes, showed no proportionate increase in disease reports. Fish disease actually decreased over 30 years in agreement with expectations based on declining stocks. This literature analysis provides a quantitative basis for earlier inferences that impacts from disease are higher in the oceans of today than in the past, with the important caveat that only some taxa show a clear increase.

The MDWG concludes that the perceived burgeoning of marine disease outbreaks is real for some marine taxa, but is undetectable or not present in others (Harvell *et al.* 1999; Hayes *et al.* 2001). Corals have clearly experienced increased impacts from bleaching and disease over the past 30 years (Ward and Lafferty 2004; Figure 4). In Florida, coral cover is being lost at an escalating rate (Porter *et al.* 2001; Patterson *et al.* 2004), apparently linked to diseases such as white band (Figure 1) and aspergillosis (Figure 2), and to declining water quality.

Globally, coral disease is widespread, with many new syndromes being discovered, such as those affecting plating corals of Palau (Figure 5). Thus far, the Caribbean appears to be a hot spot for disease syndromes and outbreaks (Weil and Smith 2003).

Another reliable baseline also suggests that the current high levels of damage from coral disease are unusual. *Acropora cervicornis* was the dominant coral on many Caribbean reefs until it was virtually eradicated by disease. The signature of this species loss, which has not occurred in the previous 3800 years, is readily detectable in recent cores (Aronson and Precht 2001).

Epidemiological models for the analysis of human disease were developed early in the 20th century (Kermack and McKendrick 1927) and were more recently adapted by Anderson and May (1979) for the study of human disease and terrestrial wildlife. Examples of new management decisions or approaches originating from modeling studies, include the decision to vaccinate dogs but

not lions for distemper to slow a distemper epidemic among Serengeti lions (Packer *et al.* 1999), the damping of red grouse cycles by controlling trichostrongylid worms (Hudson *et al.* 1998), and mapping the spread of rabies in raccoons (Smith *et al.* 2002). Adapting these models to the analysis of marine diseases requires an appreciation of the fundamental differences between marine and terrestrial systems and outbreaks, in particular the greater host and pathogen diversity in the ocean, the fact that transmission dynamics and spread rates may be higher in marine systems than in terrestrial systems, and that hosts in the ocean are predominantly invertebrates with short-lived, localized immunity dynamics. Management strategies, such as population viability assessments and decisions about the size and networking of marine reserves, may be dependent on the role of disease (Lafferty and Gerber 2002; McCallum *et al.* unpublished).

■ Problems and priorities

To better address the lack of information and tools for marine disease, we highlight five unsolved problems and priorities for future work.

The origins and spread of most marine diseases are poorly known

Important diseases of marine organisms can originate from aquaculture. Recent examples include infectious salmon anemia (ISA), which spread from farmed to wild populations and from Norway to Canada and Maine over several years (Ritchie *et al.* 2001; Murray *et al.* 2002); white spot syndrome virus, which affects wild and cultured penaeid



Figure 2. (a) Caribbean seafans (*Gorgonia ventalina*) infected with *Aspergillus* and (b) Sea fan skeletons killed by aspergillosis.

shrimp and spread to the Americas from Asia in 1995 (Jory and Dixon 1999); and Taura syndrome virus of white shrimp (*Penaeus vannamei*), which began to spread to North America from Ecuador in 1992 (Overstreet *et al.* 1999). There are currently no accurate estimates of the magnitude of the spillover problem for other possible pathogens, but aquaculture is a likely source of new pathogens entering wild populations in the ocean.

Ballast water is also an important potential source of pathogens that has yet to be investigated. Given the hundreds of new invertebrates and unknown numbers of microorganisms and potential pathogens introduced in ballast water, this is an urgent priority for future research.

Data on pathogen spread in the ocean are limited, but the few cases in which information is available indicate that disease may spread at least as rapidly as any terrestrial epidemic (McCallum *et al.* 2003). For example, a herpes epidemic in pilchards spread along the Australian coastline at approximately 10 000 km per year (Murray *et al.* 2001), a bacterial infection in long-spined urchins in the Caribbean in the mid 1980s spread at 3000–4800 km per year (Lessios 1988), and morbillivirus infection in seals spread at 3000 km per year (Heide-Jorgensen *et al.* 1992). In terrestrial environments, only the epizootics of myxomatosis and calicivirus in Australian rabbits and the virus-vector West Nile virus in North American birds have rates of spread in excess of 1000 km per year. Although the data are still too limited to conclude whether spread rates are really different in marine and terrestrial ecosystems, it is clear that they are extremely rapid in the ocean and in some cases may well exceed those on land.

The most likely explanation for this relatively rapid spread is the lack of barriers to dispersal in some parts of the ocean and the potential for long-term survival of pathogens outside the host. Whether these rates are the rule is uncertain; despite the impression that marine systems are quite open, studies of gene flow indicate that some marine systems are functionally more closed than

they appear (McCallum *et al.* 2003). The findings suggest that pathogens may pose a particularly severe problem in the ocean.

Not only is spread rate in the ocean rarely measured, information on the modes of marine disease transmission is also lacking. Many terrestrial epidemics propagate via flying insect vectors, but vectored transmission in the ocean is poorly documented. One of the few known examples is a coral predator, the fireworm, that transmits *Vibrio shiloi*, the bacterium responsible for infectious coral bleaching (Figure 6; Sussman *et al.* 2003). Long-range dispersal of some marine parasites with complex life cycles occurs where migratory sea birds are the definitive hosts. Pathogen interchange between terrestrial and marine environments seems to be predominantly from land to ocean (usually via rivers), although little is known about the actual rates of pathogen exchange. For example, the



Figure 3. Sea lion with aborted pup. The probable cause was beta herpes virus.

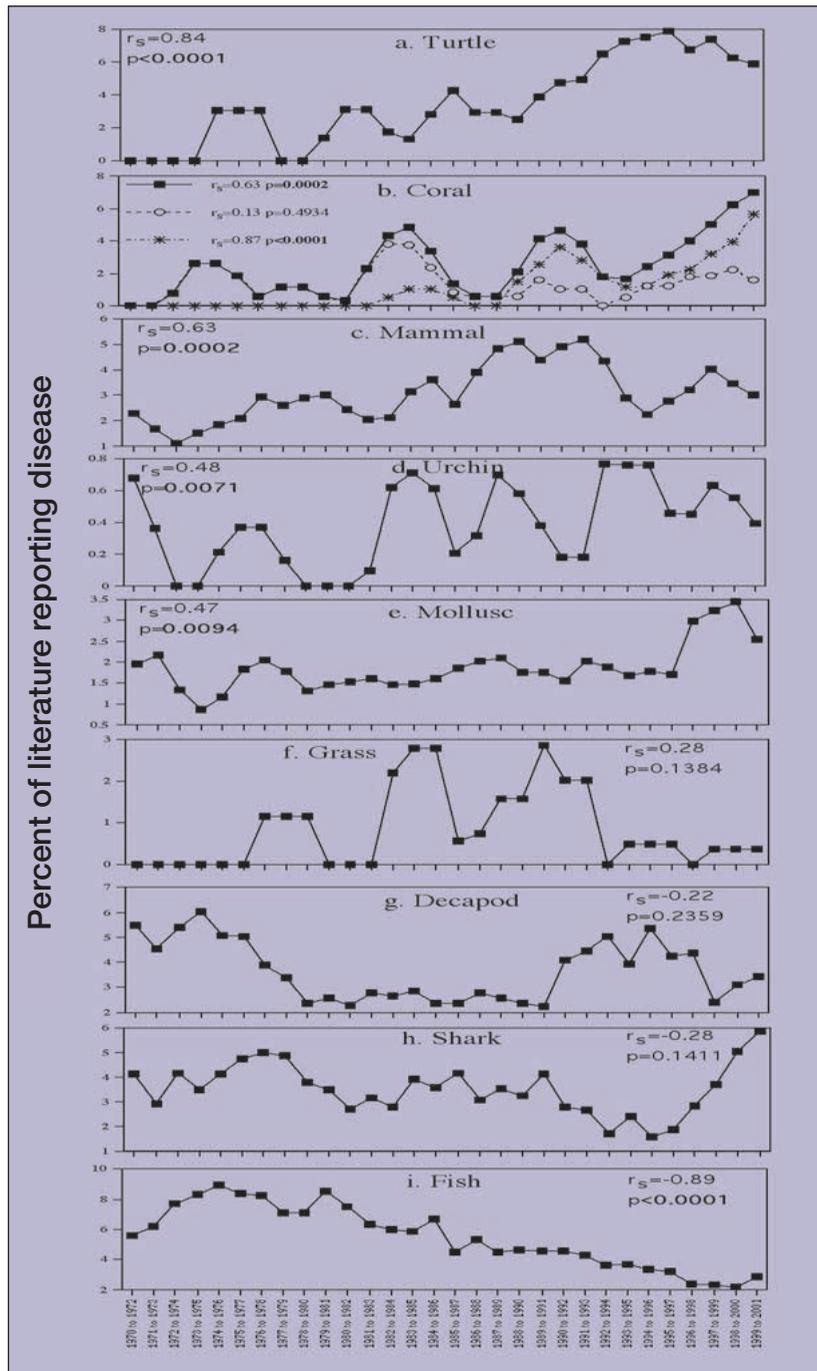


Figure 4. Thirty years of literature reports about marine diseases (from Ward and Lafferty 2004). Data for each taxon are the proportion of papers reporting disease studies for each group, normalized for total literature in each group, and plotted as 3-year running means. In each of the five taxa shown, the proportion of the literature about disease increased from 1970 to 2001. Diseases of other taxa reported in Ward and Lafferty (2004), such as fish and crustaceans, did not increase significantly over the same time interval.

parasite *Toxoplasma gondii*, which infects otters in Southern California, is considered an emergent disease from land because only domestic cats are known to shed infective oocysts. Otters sampled near freshwater runoff are three times more likely to be seropositive for *T gondii* than otters that live distant from freshwater runoff (Miller

et al. 2002; Cole et al. 2002). Developing evidence suggests that two coral diseases, aspergillosis and serratosiis, also originated in terrestrial ecosystems (Patterson et al. 2002; Garrison et al. 2003). Seal distemper in Lake Baikal appears to have originated from dogs, while the origins of a different strain of marine phocine distemper is still not verified (Harvell et al. 1999). Although not all marine diseases necessarily originate on land, it is important to understand that some do and others, like acanthocephalan infestations in sea otters, are affected by their host food base, which is sensitive to terrestrial influences.

The immediate research priority to ameliorate this problem is to develop molecular and microbiological diagnostics that can identify and track particular pathogen types, to trace origins and the spread of marine pathogens.

Infectious stages and host range

It is possible that some microorganisms can persist longer in marine than terrestrial conditions. Although most terrestrial bacteria do not survive for long in the marine environment, there are exceptions. Enteric bacteria are well adapted for increased salinities, but are susceptible to bacteriophage and bacteriovores like *Bdellovibrio* species. Bacteria that do not form spores can remain dormant in saltwater sediments for long periods and are resuspended when sediments are disturbed (Heidelberg et al. 2002). Spore-forming bacteria and fungi can remain viable for thousands of years and can be transported over tremendous distances (Moir and Smith 1990). Viruses of terrestrial origin may make up a substantial proportion of the microbial plankton (Griffin et al. 1999; Wommack et al. 1999). The survival of terrestrial microbes in estuarine environments and along shorelines can have a great impact. For example, runoff from land containing non-marine species (particularly human-associated species such as members of the Enterobacteriaceae) has necessitated the closure of beaches for extended periods

of time (days to weeks), even after the primary input has subsided (Haile et al. 1999). Viable spores of normal soil fungi, including *Aspergillus sydowii*, which infects gorgonian corals (Figure 2) have been found in open oceanic waters and trenches.

An environmental reservoir is a habitat in which a

pathogen survives in a viable form without its primary host. These reservoirs may be biotic, such as a secondary host in which there is no pathogenic relationship, or an alternate abiotic habitat in which the pathogen can either persist or multiply. A pathogen with an environmental reservoir has the potential to kill every member of a host species, because it is not limited by host density. Examples of such abiotic reservoirs are brackish water, estuarine sediment (for cholera), and soil. For aspergillosis in corals, airborne dust has been suggested as a reservoir (Garrison *et al.* 2003). Crevices in reefs appear to be reservoirs for black band disease of corals (Kuta and Richardson 2002), and some of the normal gut microbiota in humans or seagulls are possibly pathogenic to certain coral species (Patterson *et al.* 2002).

In addition to having environmental reservoirs, many marine pathogens have wide host ranges, allowing them to be unusually destructive. For example, coral disease outbreaks caused by generalist pathogens include black band disease, reported on 42 species from 21 genera, white plague types I and II, collectively known to infect 22 species and 16 genera (Weil and Smith 2003; Green and Bruckner 2000), and *Porites* pox, which affects at least 10 species of Philippine *Porites* (Raymundo *et al.* 2002). From a conservation perspective, pathogens with a wide host range are of particular concern (Lafferty and Gerber 2002); those infecting large host populations are responsible for virtually all recent disease outbreaks in endangered species (see Dobson and Foufopoulos 2001).

This problem could be mitigated by developing rapid response capability to identify, study, and manage disease outbreaks as they occur.

Taxonomic diversity of hosts and pathogens

Of the 34 animal phyla, only nine occur on land (Schubel and Butman 2000), making the potential for diverse host–parasite interactions in marine environments greater than in terrestrial ones. In addition to the greater diversity of host phyla, more classes of organisms are involved in parasitic relationships in marine environments (McCallum *et al.* unpublished) and hosts in the ocean include groups such as coral that have no terrestrial counterpart. Among animals, modular, clonal life forms, like corals and sponges are more common in marine environments. As hosts, modular and other clonal species may permit a build-up of more virulent disease strains, because their genetic homogeneity, coupled with the rel-



Figure 5. Recently detected coral diseases: Palauan *Pachyceris* infected with unknown red band infection.

atively rapid evolution of pathogens compared to hosts, should facilitate epidemic spread of relatively virulent pathogens. This occurs among clonal terrestrial plants and where genetic variation has been restricted, such as in farmed animals and plants. The unusual colonial biology and short-lived immunity dynamics of these hosts requires modifications to the modeling framework developed by Anderson and May (1979).

Microbial diversity is also higher in marine environments, although numbers are severely underestimated because many marine microorganisms cannot be cultured. In addition, new bacterial pathogens, such as coral white plague (*Aurantimonas*), are being discovered in entirely new genera (Denner *et al.* 2003).

Research priorities here should include the development of better molecular diagnostics.

Anthropogenic agents and pathogens

The interaction of disease-resistance mechanisms with environmental stressors is reviewed briefly for corals in Mullen *et al.* (2004), and for other marine animals in Kim *et al.* (2004). Chemical contaminants, especially polychlorinated biphenyls (PCBs), DDTs, and organometals, can bioaccumulate up the food chain and are found in the tissues of marine mammals. 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. Both cellular and humoral immunity were reduced in harbor seals fed herring from the Baltic sea

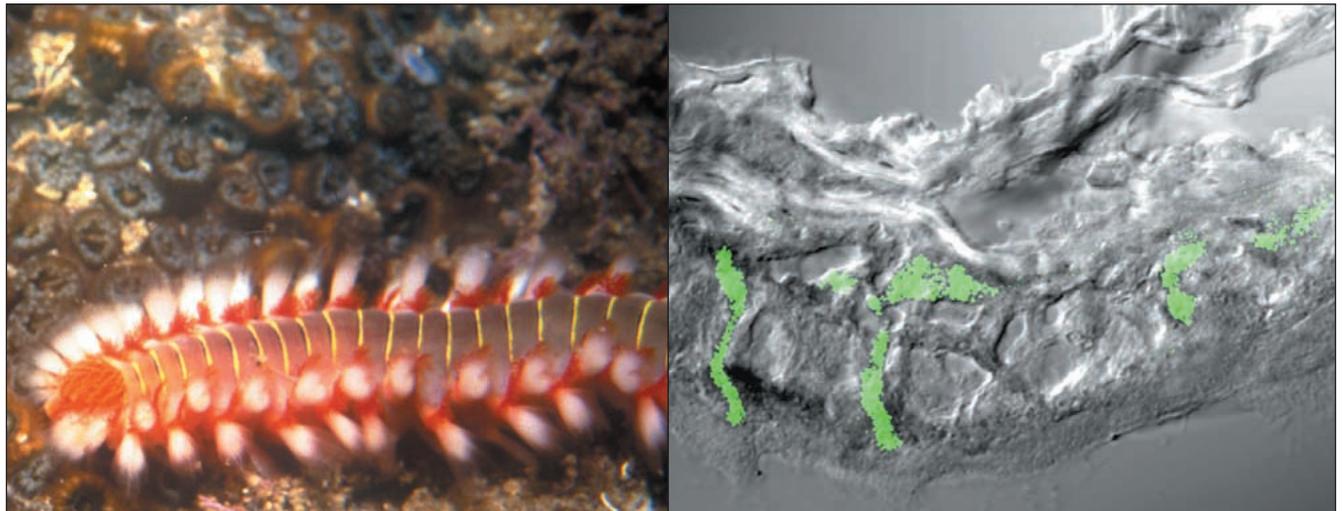


Figure 6. (a) Vectors of disease in the ocean. *Hermodice carunculata* feeding on *Oculina* (Sussman *et al.* 2003). (b) Photomicrograph of *Hermodice* gut stained with FISH probe specific for *Vibrio shiloi*, the pathogen causing coral bleaching (Sussman *et al.* 2003).

compared to immune responses in seals fed less contaminated Atlantic fish. 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 prevalences of *Leptospira* and calicivirus infections in California sea lions demonstrating premature parturition (Gilmartin *et al.* 1976). Experimental exposure of oysters to tributyl tin increased infection intensity and mortality.

Eutrophication due to runoff of nutrient or organic materials may also cause an increase in the abundance of diseased organisms. The best examples are for non-infectious microorganisms, such as harmful algal blooms. *Pfiesteria* outbreaks in North Carolina estuaries correlate with increasing eutrophication of these environments. The 2002 “blackwater” event in Florida Bay, which killed 70% of the scleractinian corals and all of the clionid sponges in long-term benthic transects on coral reefs within the Bay, occurred during a red tide event (*Karenia brevis*), driven by elevated nutrient conditions in the bay (Porter *pers comm*).

Coral reefs, especially the elkhorn coral, *Acropora palmata* (Figure 1), grow in oligotrophic waters, and it is hypothesized that newly elevated nutrient conditions in the Florida Keys may have contributed to the outbreak of a fecal enteric microbe that traditionally grows under such conditions (Patterson *et al.* 2002). Disease severity caused by seafan aspergilliosis (Figure 2) sometimes correlates with lower water quality conditions, including elevated nitrogen concentrations and turbidity (Kim and Harvell 2002), but aspergilliosis also occurs at pristine sites which are low in anthropogenic inputs. Aspergillotic lesions grow at a greater rate when nitrate levels are elevated (Bruno *et al.* 2003). Kuta and Richardson (2002) detected significantly higher nitrite concentrations at sites with black band disease (BBD) than at control sites without

BBD in the Florida Keys, but the authors emphasize how difficult the relationship between disease and water quality has been to detect on coral reefs.

A better understanding of environmental facilitators of disease and host immunity should be another research priority.

Epidemiological models

Epidemiological models developed for the study of human disease have not yet been successfully applied to ocean wildlife or management in the ocean. Adapting such models to marine organisms requires understanding fundamental differences between marine and terrestrial systems and outbreaks, including the fact that transmission dynamics and spread rates may be greater in the relatively more open ocean. Hosts in the ocean are predominantly invertebrates with short-lived immunity, so human-based models with lifetime immunity may be inappropriate. Different environmental facilitators may be predictive of marine epizootics than those on land. Initial disease modeling in marine protected areas suggests some very different patterns than on land (McCallum *et al.* unpublished).

A further research priority is to develop forecasting models for outbreaks with environmental or climate sensitivity. These could be modeled after crop disease and coral bleaching forecasts.

Conclusions

Measures to prevent and manage marine diseases could be as varied as the organisms and pathogens involved. Remediation strategies for infectious diseases in humans and wildlife include vaccination, antibiotic therapy, quarantine, culling, and the development of resistant transgenics. Each of these are currently ineffective (vaccina-

tion, quarantine, culling, antibiotics) or prohibitively expensive (transgenics) in marine ecosystems. The most practical immediate remediation for many marine communities is to reduce pathogen inputs (especially from land) and synergistic stressors, such as warm temperatures and eutrophic waters. Reducing such inputs requires knowing the source of new marine pathogens – research should focus on identifying sources and reducing inputs, while at the same time developing control measures. Immunological strategies, such as vaccination or breeding of resistant strains, may be effective for marine vertebrates, as long as the specific pathogen is known. Marine invertebrate hosts also have cellular and antimicrobial responses to pathogens that could perhaps be enhanced through breeding or engineering. Implementing these approaches may be a decade away, although some breeding programs have already been successful in oysters.

Consideration of what is known about current marine diseases and what sorts of approaches have been useful on land, for both humans and wildlife, suggest the following five research priorities: (1) Develop molecular and microbiological diagnostics and capability to identify and track particular pathogen types to trace origins and spread of marine pathogens; (2) Develop rapid response capability to identify, monitor, and manage disease outbreaks as they occur; (3) Document longevity and host range of infectious stages; (4) Pinpoint the facilitating role of environment in disease outbreaks; and (5) Develop forecasting models for outbreaks that are sensitive to environmental or climatic factors.

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