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Sea Sickness: The Upsurge in Marine Diseases

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Most visitors to salt marshes along the southern California coast will spot Caspian terns, plovers, and sandpipers feasting on snails, crabs, and killifish at low tide. Only Kevin Lafferty and a few like-minded colleagues look at the same scene and envision packets of parasites and pathogens on the move. Yet calculations by Lafferty and Armand Kuris show the biomass of trematode parasites alone—flatworms such as flukes—contained within the visible creatures may exceed that of the birds in a healthy estuary.

"Parasites and pathogens are everywhere, and that's a normal state of nature," says Lafferty, a US Geological Survey marine ecologist at the University of California–Santa Barbara. "Ecologists have been slow to truly recognize this because there's not a tradition of looking inside organisms. Yet parasitism is the most popular lifestyle among animals."

Nowhere is that truer than in the oceans, where both host and parasite diversity exceed that on land. Marine parasites (including disease-causing pathogens) are not just weighty and numerous, they also play powerful roles in orchestrating the makeup, diversity, and health of natural marine communities. In the marshes that Lafferty studies, for instance, trematodes manipulate the behavior and reproductive success of their multiple hosts: The worms castrate the snails they infect and use them to produce hordes of free-swimming trematode larvae; when the larvae burrow into the tissues of killifish, they form cysts in the brain that cause the fish to flash on their sides at the water's surface, where they are much more likely to be eaten by birds, in whose guts the worms complete their life cycle. Parasites also influence the physical habitat. Trematodes prevent infected cockles from burrowing in the mud, leaving shells exposed as hard surfaces where sessile organisms can attach. Just offshore, periodic bacterial disease outbreaks depress populations of kelp-grazing sea urchins and allow kelp forests to rebound.

"I think the general statement that parasites are embedded in and dominate food webs is true everywhere," Lafferty says. "They're important because they're regulators. They tend to knock back common species, and that provides opportunities for biodiversity."

Increasingly, however, human activities are disturbing marine ecosystems and changing the dynamics of parasitism and disease in the oceans. Lafferty and Jessica Ward, of Cornell University, have found evidence that disease outbreaks are becoming more common in several key groups of marine animals, including mammals, turtles, corals, mollusks, and urchins, and many of these diseases are linked to human impacts on the oceans. Paradoxically, the most alarming finding of the study, Lafferty says, is a decline in reports of disease outbreaks in fishes. He attributes this to overharvesting, which may have left many fish populations too sparse for infectious diseases to be transmitted between individuals.

"We've all seen increasing signs that the world's oceans are sick, and in some cases dying," says Andrew Dobson, of Princeton University. "These signs vary from increased disease outbreaks in marine mammals and corals to the sudden disappearance of once-common species. These things are occurring because humans are increasingly treating the oceans as an all-purpose toilet and garbage dump. By putting all this extra stuff in the oceans, we're creating problems not only for species that live in the oceans but ultimately for ourselves."

Stresses that can alter the emergence, spread, and impacts of diseases in the oceans include discharges of human sewage and agricultural runoff, windborne dust and pollution, introduction of exotic species, destruction of coastal habitat, harvesting of fish and shellfish, and rising global temperatures. These stresses interact in complex ways with pathogen distribution and virulence, host resistance, and other aspects of disease dynamics that researchers are just beginning to explore.

Sewage and pathogen pollution

A major source of emerging diseases on land and in the sea is "pathogen pollution," the introduction of novel pathogens to a community. Ships taking on and discharging ballast water in coastal areas worldwide are undoubtedly spreading microbes and invertebrate parasites to new regions, but little effort has been made to document such introductions. A much more noticeable impact is coming from sewage, freshwater runoff, and windborne contaminants that bring land-based pathogens into contact with ocean creatures.

California sea otters, hunted to near extinction for their fur in the 1800s, have been federally protected for almost 30 years, but their rebound has been slowed by a high death rate. Nearly 40 percent of otter deaths are caused by disease, including some new to the oceans. One of the greatest challenges facing otters, says University of California–Davis parasitologist Patricia Conrad, is a protozoan parasite, *Toxoplasma gondii*, found in domestic cat feces; *T. gondii* can cause brain lesions, tremors, and seizures in otters. (The parasite infects humans and many other animals but can reproduce only in cats.) Toxoplasmosis is responsible for 17 percent of otter deaths and renders other otters more vulnerable to shark attack. Conrad has found antibodies indicating *T. gondii* exposure in 52 percent of dead otters and 38 percent of live ones. The infection risk triples for otters living near heavy freshwater outflows, which presumably carry cat feces washed from lawns, streets, and discarded kitty litter. Other assaults from the land facing sea otters include the brain parasite *Sarcocystis neurona*, carried in opossum feces, and valley fever caused by spores of the fungus *Coccidioides immitis* transported in wind-blown dust and eroded soil.

In the Florida Keys, nearly 90 percent of the massive elkhorn coral—the most common reef-building coral in the Caribbean—has been lost since the mid-1990s, largely to a bacterial disease called white pox. The known pox pathogen is *Serratia marcescens*, a fecal gut bacterium of humans and animals. Marine ecologist Kathryn Sutherland, of Rollins College in Winter Park, Florida, and microbiologist Erin Lipp, of the University of Georgia, screened water and sewage samples with molecular techniques and found that although the bacterium is rare in marine environments, it is common in human sewage and in nearshore waters contaminated by leaks from septic systems and injection wells. Using DNA fingerprinting techniques, they have matched one strain of the bacterium isolated from coral lesions to an isolate from human sewage, but they are still hunting down a definitive source for the known coral-killing strain.

This elkhorn coral bears the white scars of a disease called white pox. Nearly 90 percent of the common reef-building elkhorn coral in the Florida Keys has been lost in the past decade, largely to white pox. The known pathogen is a fecal gut bacterium of humans and animals, Serratia marcescens*, believed to enter the ocean in sewage. Photograph: James W. Porter.*

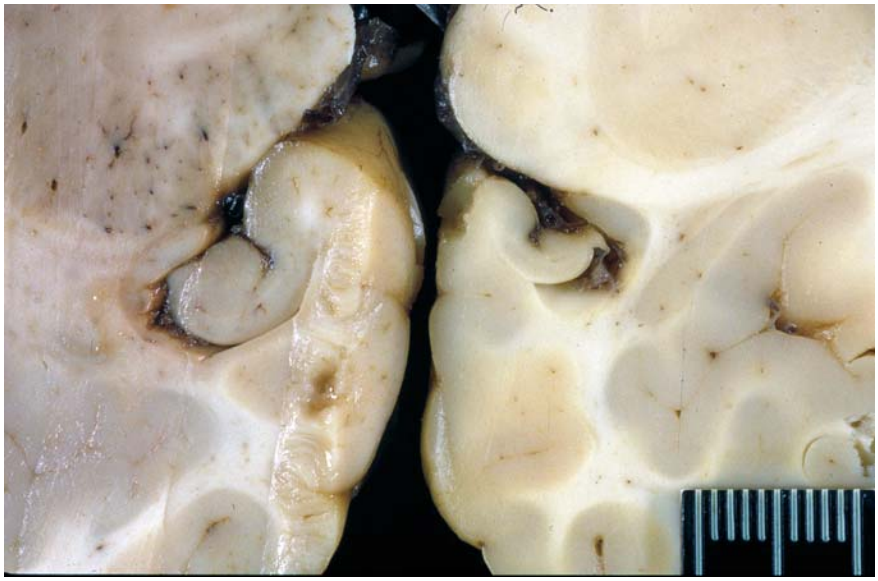
David Kline, of the Smithsonian Tropical Research Institute in Panama, points out that less than 10 percent of the sewage in Central America and the Caribbean receives any treatment at all before being dumped into the ocean. Sewage is "turning our oceans into a giant petri dish that supports the rapid growth of bacteria that can kill corals," Kline says. His focus is not on novel pathogens in the sewage but instead on its role in spurring the normally beneficial bacteria on reefs to burgeon out of control and cause coral disease and death. Healthy corals live in a bacterial soup, coated with mucus or slime containing a distinct bacterial community whose growth is normally tightly regulated by the corals. Kline cultured bacteria from coral mucus and found that in high numbers they can kill their host. To find out what could spur such growth, he set up an experimental seawater system and tested individual runoff contaminants on live corals. Surprisingly, it was not the usual fertilizer nutrients, nitrate or phosphate, but instead simple sugars (dissolved organic carbon)—a component seldom measured in water quality tests—that allowed bacteria to overcome the coral's tight controls, grow aggressively, and cause disease. Not only do the sugars in runoff fuel the bacteria directly, but the nutrients also encourage the growth of algae.

"It's a positive feedback loop," Kline says. "The bacterial disease kills coral and makes more room for algae to grow, and the algae make and release glucose during photosynthesis, spurring more bacterial growth and perhaps altering the pathogenicity of some of them."

Toxic algal blooms

The frequency of harmful algal blooms that produce toxins damaging to human and animal health appears to be increasing worldwide, and the suspected culprits include nutrient-laden runoff, eutrophication, overharvesting of algae-grazing fish, and perhaps climate warming. Several unusual die-offs of marine mammals have been linked to exposure to algal biotoxins: humpback whales to saxitoxin, Hawaiian monk seals to ciguatoxin, California sea lions to domoic acid, and bottlenose dolphins and Florida manatees to brevetoxin.

Red tides—blooms of the dinoflagellate *Karenia brevis*—are becoming increasingly common along the Florida coast, and the brevetoxins they produce helped make 2005 the second deadliest year on record for endangered manatees, according to Gregory Bossart, a marine mammal veterinarian and pathologist at the Harbor Branch Oceanographic Institution in Fort Pierce, Florida. Manatees can literally be "gassed to death" by brevetoxin that has been aerosolized by wind and wave action, or poisoned while grazing on sea grasses, even weeks after the algal bloom has dissipated, Bossart notes. He attributes 17 percent of annual manatee deaths to red tides.



Domoic acid, a toxin released by blooms of diatoms, damages the brains of sea lions, eventually causing epilepsy, seizures, and death. The shrunken hippocampal region of a sea lion that died of domoic acid poisoning is shown in the brain section at right, compared to a healthy sea lion brain on the left. Photograph: The Marine Mammal Center.

Humans likewise can suffer neurotoxic shellfish poisoning from eating contaminated seafood or respiratory distress from inhaling brevetoxins. Bossart and his colleagues recently correlated frequent red tides off Florida's western coast with a 54 percent increase in emergency room admissions for pneumonia, asthma attacks, and other respiratory illnesses. He also suspects that chronic, repeated brevetoxin exposure can suppress the immune systems of manatees—and perhaps humans—making them more susceptible to infectious diseases. As a sentinel species, Bossart says, the manatee is "Florida's 2000-pound canary."

A sea lion dubbed Chippy was rescued after he trekked inland from the California coast and climbed onto the hood of a California Highway Patrol cruiser. Domoic acid released by algal blooms has killed more than 1000 sea lions since 1998 and caused chronic brain damage and miscarriages in others. Sick animals often become stranded or confused, wandering into farm fields and urban areas. Photograph: The Marine Mammal Center.

Off the California coast, deaths of sea lions and other marine mammals due to domoic acid poisoning are increasing, along with the frequency of blooms of diatoms in the genus *Pseudonitzschia*.

"Over the years, we've treated more than 10,000 seals and sea lions," recounts veterinarian Frances Gulland of the Marine Mammal Center in Sausalito. "But on Memorial Day weekend 1998, we saw something we'd never seen before—over 70 big fat adult female sea lions stranded along the beaches of Monterey Bay having convulsions and seizures. Over half died within hours."

The sea lions had eaten anchovies and sardines that grazed on the toxin-producing algae. Since 1998, there have

been repeated poisonings of sea lions by domoic acid, and more than 1000 animals have died.

Most recently, Gulland and her colleagues have learned that even sea lions that are not killed can suffer miscarriages and chronic, irreversible brain damage from repeated exposure to lower levels of domoic acid. As their brains decay, animals often chew their tails obsessively or become stranded or confused. Sick sea lions have wandered into farm fields and airports, and even onto the hood of a parked highway patrol cruiser. The toxin also crosses the placenta and damages the fetus, causing pregnant females to abort. "We've now found domoic acid in aborted sea lion fetuses," she says.

"These aren't just abstract concerns for ocean health," Gulland points out. "These sea lions that are washing up along the coast are getting poisoned from a diet they share with us." Indeed, domoic acid's toxicity was first noted 20 years ago when people who ate contaminated mussels suffered what came to be called "amnesic shellfish poisoning." "We now call Caesar salad [with its anchovy-based dressing] 'seizure salad,'" she quips.

A sea lion with urogenital carcinoma lingers in a pen at the Marine Mammal Center in Sausalito, California. Urogenital cancers, which are thought to require an interaction between a relatively benign genital herpes infection and exposure to organic pollutants, are found in 18 percent of adult sea lions that die on California beaches. Photograph: The Marine Mammal Center.

Pollutant–pathogen synergy

The accumulation of persistent organic pollutants, heavy metals, and other land-derived contaminants in the marine food chain can also alter interactions between parasites and hosts in complex ways. Particularly for predatory fish and marine mammals at the top of the food chain, pollutants may weaken disease resistance.

Among the sea lions that strand and die on California beaches, 18 percent of adults have urogenital carcinomas, an extremely high cancer prevalence for a wild mammal, Gulland says. The general adult population also has a relatively high incidence of a sexually transmitted herpes virus infection: 22 percent among females and 43 percent among males. Virtually all of the animals with carcinomas also have herpes infections, and their blubber contains much higher concentrations of organic pollutants (PCBs and DDT) than that of animals without cancers. Gulland believes that development of these cancers requires an interaction between herpes infection, pollutant exposure, and probably genetic factors.

"Herpes in sea lions is relatively benign," notes Dobson, who is collaborating with Gulland and others to model the interaction. "Unfortunately, if you're also exposed to relatively common organic pollutants, then this benign pathogen can become much more damaging, causing very aggressive carcinomas."

He modeled the expected dynamics of the sea lion population with herpes infection alone, with pollution alone, and with the two together. The results are counterintuitive: Pollutant-exposed females that get infected with herpes develop aggressive carcinomas, die more quickly, and have less chance of passing on the infection. "Ironically, this is working to wash the disease [herpes] out of the system," Dobson concludes. "We know very little about synergisms between pollutants and other benign pathogens, but we think this might be the first well-documented example of many similar phenomena."

Dobson and Bernd Sures, of the University of Karlsruhe, Germany, have also examined an interaction, however, that illustrates how some parasites "play major beneficial roles in ecosystems" by helping to protect their hosts from toxicants. In particular, parasites such as acanthocephalid worms that feed on substances in the guts of fish may literally suck lead, cadmium, and other heavy metals out of their hosts, building up much higher concentrations of toxicants than the hosts. Modeling indicates that "fish can stand a much more polluted environment if they're infected by worms," Dobson notes. "So the worms are performing a significant and unexpected ecosystem service that we'll lose if eutrophication or other factors knock out parasites."

Fish farm spillover

Global transport of infected fish, shrimp, and shellfish for aquaculture and the spillover of parasites from fish farms to wild stocks also alter the dynamics of disease in coastal waters. Sea lice, for example (actually a crustacean, *Lepeophtheirus salmonis*), are emerging pathogens of wild juvenile salmon in the Pacific Northwest. Canada's British Columbia coast hosts 131 salmon farms holding

Healthy corals (top) are coated with mucus or slime containing distinct bacterial communities whose growth the corals tightly regulate. David Kline, of the Smithsonian Tropical Research Institute in Panama, has found that dissolved organic carbon in sewage runoff spurs these bacteria to grow aggressively, degrading the host corals and supporting the growth of algae (bottom). Photographs: © 2005 David Kline.

60 million captive Atlantic salmon, and lice infection levels are higher closer to the pens. Yet the link between the farms and lice infections in wild fish remains controversial.

Mathematical biologist Mark Lewis and doctoral student Martin Krkosek, of the University of Alberta, and colleague John Volpe, of the University of Victoria, used field experiments and models to document the transfer and spreading pattern of sea lice from a fish farm to 12,000 juvenile wild chum and pink salmon as they approached, passed, and migrated into the sea 60 kilometers beyond the farm. Near the farm, Krkosek says, the rate of sea lice infections of the wild fish was 73 times higher than the rate from ambient levels, and infections continued to exceed ambient levels for 30 kilometers of the migration route.

The researchers are still working to calculate direct louse-induced mortality and examine how it interacts with other sources of mortality to affect the wild salmon population. But their data are already having an impact on farm management decisions. At least one major fish farming company has agreed to move its adult salmon pens to a site further away from a major wild salmon migration route, Lewis says.

Marine disease dynamics

Understanding the changing dynamics of disease in the oceans is vital for managing fisheries, siting and managing marine reserves, protecting native species, and monitoring the health of marine ecosystems. Yet little baseline information exists on the origins, mechanisms, rates of spread, or frequency of disease in the oceans. In the past it has been difficult even to isolate the causative agent in disease outbreaks, although molecular biology is providing a powerful array of new diagnostic tools. Few studies have systematically tracked diseases over time, much less documented population- or community-level impacts in the sea. This is a situation, Dobson says, that calls for models.

"When you have little information, the most powerful things you have are models that allow you to explore 'what if' scenarios and what types of phenomena could create the patterns you're seeing," he says. "We use models as a kind of macroscope to try to see the bigger picture of what's going on, to understand the patterns we're seeing, and to try to point the finger at what is causing a particular problem."

Epidemiological models developed to help understanding and control of disease in humans and terrestrial wildlife populations, however, are ill suited to analyzing disease in marine systems. Ecologists have come up with a list of fundamental differences between land and sea that they believe must be considered in developing a new generation of models for use in understanding and managing marine disease:

The ocean harbors greater host and pathogen diversity. Only 9 of the 34 phyla of animals on Earth are found on land, and the greater diversity of life forms, body plans, and life histories in the oceans offers a greater potential for novel host–parasite relationships. Also, more classes of organisms have adopted parasitic lifestyles in the oceans.

The ocean has more "modular colonial" animal hosts. Colonies of genetically uniform animals such as corals, sponges, and bryozoans are unique to the oceans and may be more vulnerable to virulent disease epidemics. Since

relatively short-lived invertebrates are the predominant hosts in the ocean, epidemiological models based on humans or other animals with lifetime immunity to a disease after exposure may not apply.

Potential rates of disease spread are much faster in the ocean. The ocean is generally a more open system with fewer barriers to long-distance dispersal, and it offers more potential for pathogens to survive long periods outside a host or in secondary hosts.

The record for documented spread of a disease was set by a herpes virus epidemic in pilchards in 1995 that spread along the southern coast of Western Australia, against the prevailing currents, at more than 10,000 kilometers a year. The origin of that epidemic remains in dispute, but the chief suspect is frozen pilchard imported from Thailand to feed penned bluefin tuna.

"That one is so fast, it's hard to explain using almost any of the standard epidemiological models," says Hamish McCallum of the University of Queensland. "The virus is so infectious it has proved impossible to maintain the pilchards in culture, so it's hard to do lab investigations on the disease." Despite several other examples of rapid pathogen spread in the ocean, he says, "it's surprisingly frustrating when you try to find general patterns because remarkably few cases have been documented."

McCallum has used models to try to gain insight into the impact that marine protected areas might have on disease dynamics, since the goal of most reserves is to increase population densities of exploited species, and denser populations can sustain more parasites and pathogens. "We know that fish farms have enormous disease problems that do spill over into the environment, so are reserves anything like that?" he asks. Unlike fish farms, however, a reserve would only enable fish densities—and those of native pathogens—to return to natural preharvesting levels. "I think the bottom line is that concern for pathogens is not an argument against reserves. It's most unlikely a reserve is going to cause an old disease to reemerge, although we need to be wary of highly virulent exotic pathogens getting into reserves," McCallum says.

Lafferty says the return of natural parasite and pathogen levels in protected areas should be viewed as a good thing: "I would hope to see an increase in native parasites in reserves. I think it would be a mark of their success. Pristine marshes have twice the abundance of parasites as degraded marshes."

The greater concern is what human pressures are doing to disease dynamics in the oceans at large. "The oceans aren't as safe as they were when we all grew up," Dobson says. "We used to see the ocean as a source of healthy food, healthy recreation. But if you're going to have a beach full of sick marine mammals, do you really want to go there for vacation? And you may think twice about sushi for lunch. The only way to deal with it is proper upstream legislation to reduce dumping of substances that are pouring into the world's oceans."

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