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Marine Infectious Disease Ecology

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Abstract

To put marine disease impacts in context requires a broad perspective on the roles infectious agents have in the ocean. Parasites infect most marine vertebrate and invertebrate species, and parasites and predators can have comparable biomass density, suggesting they play comparable parts as consumers in marine food webs. Although some parasites might increase with disturbance, most probably decline as food webs unravel. There are several ways to adapt epidemiological theory to the marine environment. In particular, because the ocean represents a three-dimensional moving habitat for hosts and parasites, models should open up the spatial scales at which infective stages and host larvae travel. In addition to open recruitment and dimensionality, marine parasites are subject to fishing, filter feeders, dose-dependent infection, environmental forcing, and death-based transmission. Adding such considerations to marine disease models will make it easier to predict which infectious diseases will increase or decrease in a changing ocean.

INTRODUCTION

The Smithsonian Environmental Research Center recently posted the first academic job announcement for a marine disease ecologist. This marine disease ecologist will face a challenge when a stranger asks what they do. The safe answer is "marine biologist," which prompts questions about dolphins and scuba diving. A more mundane answer is "ecologist," which leads to discussions about recycling and solar energy. The answer "parasitologist," however, risks making the acquaintance itch. But given the increased interest in ocean health, the description "marine disease ecologist" might become a more familiar answer, helping to explain stranded sea lions, melting sea stars, and withered abalone to an increasingly concerned public.

Thus far, marine disease has been less a career focus than a job hazard for marine ecologists. I overheard two elder marine ecologists, Ernesto Weil and Jim Porter, compare notes about sites where they were again seeing the long-spined sea urchin *Diadema antillarum* (**Figure 1*a***). These urchins were once common throughout the Caribbean, and Weil and Porter recalled how they had cursed every time razor-sharp spines punctured them back in the 1970s. But during 1983–1984, the uncountable black urchins dropped their spines and died within days (**Figure 1*b***). In just a few weeks, urchin populations across the Caribbean crashed by 95% (Lessios et al. 1984). With no herbivorous urchins and few herbivorous fishes, algae began to dominate reefs and outcompete corals; sedimentation and diseases followed until Caribbean reefs were devastated (Hughes 1994). This was one of the most important mass mortality events documented, but it happened fast and marine microbiology was in its infancy. Thus, no diagnosis was possible, and tissues were not archived for future work because there were no marine disease ecologists to do this.

For other infectious diseases, the culprit (an infectious disease agent generically called a parasite) is far too obvious. The literature considers approximately 25 viruses, 33 bacteria, 23 protists, and 21 metazoans to cause notable marine diseases in plants, corals, molluscs, crustaceans, echinoderms, fishes, turtles, and mammals (**Table 1**). A case in point is salmon farming, an industry so big it has its own Nasdaq Index. Salmon lice (parasitic copepods) build up in farm pens, slowing salmon growth, killing young fish, and requiring expensive pesticides to which sea lice are becoming resistant. Sea lice damage costs the industry US $500 million/year (Duchene 2016).

Infectious disease is only now a mainstream topic in marine ecology. For example, although a few previous marine ecology texts had mentioned disease or parasites, the revised *Marine*

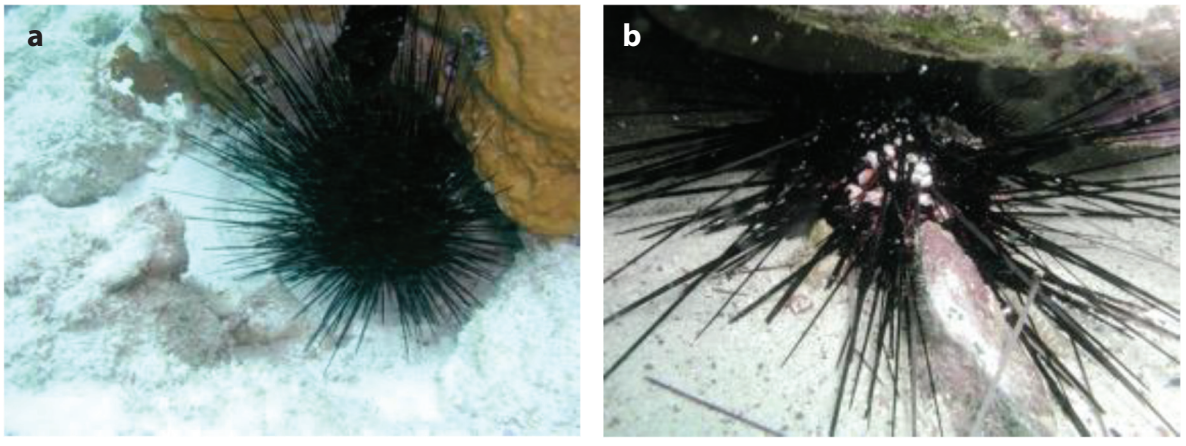

Figure 1

(*a*) Healthy *Diadema antillarum*; (*b*) dying *Diadema antillarum* (photo by K. Lafferty).

Table 1 Examples of marine diseases from around the world that have notable economic or ecological impacts[a]

Host	Disease agent	Region	Impact
Plants			
	Bacteria		
Laminaria spp.	Various	Pacific	Fishery/aquaculture
	Protists		
Zostera marina	*Labyrinthula zosterae*	North America and Europe	Ecological
Brown algae	*Eurychasma dicksonii*	North America and Europe	Ecological
Corals and sea fans			
	Viruses		
Scleractinians	Herpesvirus (white plague)	Worldwide	Ecological
	Bacteria		
Scleractinians	*Vibrio* spp. (bleaching)	Worldwide	Ecological
	Cyanobacteria consortium (black band)	Worldwide	
	Aurantimonas coralicida (white plague)	Caribbean	
	Unknown (yellow band)	Caribbean	
Acropora palmata	*Serratia marcescens* (white pox)	Caribbean	Ecological
Acropora spp.	*Vibrio* spp. (white band)	Caribbean	Ecological
	Protists		
Sea fans	*Aspergillus sydowii*	Caribbean	Ecological
Scleractinians	*Philaster* spp. and other ciliates (brown band)	Pacific	Ecological
Molluscs			
	Viruses		
Abalone (*Haliotis laevigata* and *Haliotis rubra*)	Abalone ganglioneuritis virus (Victoria)	Australia (Victoria)	Fishery/aquaculture
Oysters (*Crassostrea angulata*)	*Crassostrea angulata* iridovirus	France	Fishery/aquaculture
Oysters (*Crassostrea gigas*)	Ostreid herpesvirus 1 (including microvariants)	Europe, Asia, New Zealand, Australia, western United States (California)	Fishery/aquaculture
	Bacteria		
Abalone (various)	Withering syndrome *Rickettsia*-like organism (known as WS-RLO)	North America, Asia, Europe, Iceland	Fishery/aquaculture
Abalone (*Haliotis tuberculata*)	*Vibrio harveyi*	France	Fishery/aquaculture
Oysters (*Crassostrea gigas*)	*Vibrio tubiashii*	North America, Europe	Fishery/aquaculture
	Nocardia crassostreae	United States, Japan, Europe	
Clams (various)	*Vibrio tapetis*	Europe	
	Protists		
Molluscs (various)	*Perkinsus olseni*	Temperate and tropical regions	Fishery/aquaculture
Oysters (*Crassostrea virginica*)	*Haplosporidium costale*	Eastern United States	Fishery/aquaculture
Oysters (*Ostrea edulis*)	*Bonamia exitiosa*	Southern Ocean, Europe	Fishery/aquaculture
	Bonamia ostreae	North America, Europe	

(*Continued*)

Table 1 (*Continued*)

Host	Disease agent	Region	Impact
Oysters (various)	*Mikrocytos mackini*	Western Canada, western United States	Fishery/aquaculture
	Haplosporidium nelsoni	Eastern and western United States, Japan	
Bivalves (various)	*Marteilia refringens*	North Africa, Europe	Fishery/aquaculture
	Perkinsus marinus	Western Atlantic, Gulf of Mexico, western Mexico	
Clams (various)	QPX (quahog parasite unknown)	North America, Europe	Fishery/aquaculture
Scallop (*Pecten maximus*)	*Perkinsus quagwadi*	Western Canada	Fishery/aquaculture
	Metazoans		
Abalone (various)	*Terebrasabella heterouncinata*	Western United States (California)	Fishery/aquaculture
Crustaceans			
	Viruses		
Shrimp (penaeid)	*Baculovirus penaei*	Southeast Atlantic, Gulf of Mexico, Caribbean, Pacific	Fishery/aquaculture
	Infectious hypodermal and hematopoietic necrosis virus	Americas, Asia, Africa, Western Indo-Pacific, India, Australia	
	Aparavirus spp.	Americas, Middle East, Southeast Asia	
	Penaeus monodon–type baculovirus	Tropical regions	
	Whispovirus spp.	Asia, India, Middle East, Mediterranean, Americas	
	Okavirus spp.	Asia, Sri Lanka, Australia, Mexico	
Lobsters (panulirids)	*Panulirus argus* virus 1	Western Atlantic	Fishery/aquaculture
	Bacteria		
Lobsters (homarids)	*Aerococcus viridans* var. *homari*	North Atlantic	Fishery/aquaculture
Shrimp (penaeid)	Necrotic hepatopancreatitis bacterium	Americas	Fishery/aquaculture
	Vibrio parahaemolyticus	Asia	
	Protists		
Crabs (various)	*Hematodinium* spp.	Arctic Sea, Bering Sea, North Pacific, North Atlantic	Fishery/aquaculture
	Metazoans		
Crabs (various)	Rhizocephalan barnacles	Worldwide	Fishery/aquaculture
Crabs (Dungeness)	*Carcinonemertes errans*	North America	Fishery/aquaculture
Crabs (king)	*Carcinonemertes regicides*, *Ovicides paralithodis*	United States (Alaska)	Fishery/aquaculture
Echinoderms			
	Viruses		
Sea stars	Sea star–associated densovirus	Northeast Pacific	Ecological
	Bacteria		
Purple sea urchins	*Vibrio* spp.?	Eastern Pacific	Ecological

(*Continued*)

Table 1 (*Continued*)

Host	Disease agent	Region	Impact
	Protists		
Green sea urchins	*Paramoeba invadens*	Northwest Atlantic	Fishery/aquaculture
	Metazoans		
Green sea urchins	*Echinomermella matsi*	Norway	Fishery/aquaculture
Fishes			
	Viruses		
Fishes (marine)	Nodaviruses	Worldwide	Fishery/aquaculture
Fishes (herring)	Hemorrhagic septicemia virus	Holarctic	Fishery/aquaculture
Fishes (various)	Aquabirnaviruses	Worldwide	Fishery/aquaculture
Red sea breams	Red sea bream iridovirus	Japan, Mediterranean	Fishery/aquaculture
Pilchards	Pilchard herpesvirus	Australia	Fishery/aquaculture
Salmon	Infectious salmon anemia virus	North Atlantic, Chile	Fishery/aquaculture
	Infectious hematopoietic necrosis virus	United States, Europe, Japan	
	Bacteria		
Fishes (marine)	*Amyloodinium ocellatum*	Tropical and subtropical regions worldwide	Fishery/aquaculture
	Vibrio spp.	Worldwide	
	Mycobacterium spp.	Worldwide	
	Streptococcus spp.	Worldwide	
	Listonella anguillarum	Worldwide	
	Moritella viscosa	North Atlantic	
	Photobacterium damselae	United States, Japan, Europe	
	Tenacibaculum maritimum	United States, Japan, Europe	
	Chlamydia-like bacteria	Worldwide	
Flounders	*Edwardsiella tarda*	Japan	Fishery/aquaculture
Salmon	*Renibacterium salmoninarum*	Worldwide	Fishery/Aquaculture
	Vibrio salmonicida	Holarctic	
	Aeromonas salmonicida	Holarctic	
Yellowtail	*Nocardia seriolae*	Japan	Fishery/aquaculture
	Lactococcus garvieae	Japan	
	Protists		
Fishes (marine)	*Ichthyophonus hoferi*	Holarctic	Fishery/aquaculture
	Cryptocaryon irritans	Tropical and subtropical regions	
	Amyloodinium ocellatum	Worldwide	
	Metazoans		
Fishes (marine)	*Kudoa* spp.	Worldwide	Fishery/aquaculture
	Enteromyxum spp.	Mediterranean, Japan	
	Anisakid nematodes	Worldwide	
	Cryptocotyle lingua	Worldwide	
	Monogenea (various)	Worldwide	
	Lepeophtheirus spp., *Caligus* spp.	Worldwide	

(*Continued*)

Table 1 (*Continued*)

Host	Disease agent	Region	Impact
Salmon	*Parvicapsula* spp.	Norway	Fishery/aquaculture
	Eubothrium spp.	Europe	
Turtles			
	Viruses		
Sea turtles	Fibropapilloma herpesvirus	Worldwide	Ecological
	Metazoans		
Sea turtles	Spirorchid trematodes	Worldwide	Ecological
	Anisakid nematodes		
Mammals			
	Viruses		
Pinnipeds, toothed whales	Herpesvirus	Worldwide	Ecological
	Influenza virus		
	Morbillivirus (distemper)		
	Poxvirus		
Seals	Coronavirus	Eastern Pacific	Ecological
	Bacteria		
Toothed whales, pinnipeds, otters	*Nocardia* spp.	Worldwide	Ecological
Bottlenose dolphins	*Lacazia loboi*	Atlantic	Ecological
	Protists		
Toothed whales, pinnipeds, sea otters	*Toxoplasma gondii* (from cats)	Worldwide	Ecological
	Sarcosystis neurona (from opossums)	Worldwide	
	Coccidioides spp.	Eastern Pacific	
Pinnipeds	*Leptospira* spp.	Eastern Pacific	Ecological
	Metazoans		
Pinnipeds, toothed whales	Metastrongylid lungworms	Worldwide	Ecological
Pinnipeds	*Uncinaria* spp.	Worldwide	Ecological
Sea otters	*Profillicolis altmani* (from shorebirds)	Eastern Pacific	Ecological
Pinnipeds, toothed whales	*Aspergillus* spp.	Worldwide	Ecological
Whales	*Nasitrema* spp.	Worldwide	Ecological
Sea lions	*Zalophotrema hepaticum*	Eastern Pacific	Ecological

[a]This infectious disease list has been expanded from Lafferty et al. (2015b) to include plants, corals, and sea fans.

Community Ecology and Conservation textbook (Bertness et al. 2014) has a chapter dedicated to marine infectious diseases (Lafferty & Harvell 2014). Furthermore, a marine disease book in is the works (Behringer et al. 2018). There is a growing appreciation that viruses and fungi affect phytoplankton population dynamics (Brussaard 2004), although this falls outside this review's focus on how infectious disease agents affect marine animals. The word "disease" in marine disease implies a focus on economic or ecological impacts (see **Table 1** for a list of global marine infectious diseases). The word "marine" in marine disease implies that epidemiology is different in the ocean than on land, with greater parasite phylogenetic diversity in the oceans compared with land

and the tendency toward different trophic strategies (McCallum et al. 2004). For instance, among marine parasites there are more parasitic castrators than parasitoids and also different transmission modes, such as fewer parasites transmitted by biting vectors. For these and other reasons discussed in the section titled Marine Infectious-Disease Theory, marine disease ecology needs its own theoretical framework (McCallum et al. 2004). This review starts with five case studies of marine infectious diseases in California sea lions, sea otters, cultured red abalone, wild black abalone, and sea stars. It then discusses new research on parasites in marine food webs and ends by reviewing new modeling approaches for marine infectious diseases.

CALIFORNIA CASE STUDIES

Marine biologists stalk the California coast, and they notice when marine life gets sick. Dying fish, invertebrates, or marine mammals generate hypotheses that the system is stressed or unbalanced. In the past, the implied stressor was often pollution. Now the implied stressor is usually climate change. However, as these five case studies suggest, determining what causes marine diseases, and whether marine parasites are a problem, is not as simple as it might seem.

The California sea lion (*Zalophus californianus*) is the state's most common marine mammal and, therefore, often seen dead or stranded on the shore. As a group, marine mammals have seen a proportional rise in infectious disease reports at the same time many species have recovered due to protection (Ward & Lafferty 2004). More marine disease reports could reflect density-dependent population regulation, especially given that marine mammals have few predators. An alternative explanation is that changing ocean conditions are stressful for marine mammals, and this subjects them to more disease.

Just as not all marine parasites cause disease, not all marine diseases are caused by parasites. One dramatic and visible noninfectious disease associated with changing ocean conditions in California is domoic acid toxicity. Domoic acid is a neurotoxin produced by a red-tide-associated dinoflagellate called *Pseudo-nitzschia australis* that blooms when nutrients are high (Heisler et al. 2008). Filter feeders, such as the northern anchovy, bioaccumulate domoic acid, and the toxin works its way up the food chain to mammals, in which it causes confusion, seizures, and death (Gulland et al. 2002).

Domoic acid kills many sea lions, but leptospirosis (a disease caused by bacteria in the genus *Leptospira*) kills three times as many (Greig et al. 2005). Stranded animals with leptospirosis can be treated with rehydration, but untreated animals often die from kidney failure (Gulland et al. 2002). Since 1970, major leptospirosis outbreaks have occurred every 3–5 years between July and December (Greig et al. 2005). Asymptomatic carriers help maintain long-term leptospirosis persistence (Prager et al. 2013), and epidemic cycling seems driven by interactions between birth rates and herd immunity rather than by environmental forcing (Lloyd-Smith et al. 2007). However, despite domoic acid and leptospirosis, most sea lion strandings stem from malnutrition, which could indicate overcrowding, competition with fisheries, or disruption to food webs (Greig et al. 2005). Therefore, when evaluating infectious-disease impacts, it is helpful to put infectious diseases in context with other mortality sources.

Another California marine mammal subject to infectious disease is the southern sea otter (*Enhydra lutris nereis*). Sea otters were almost extirpated by the fur trade, but they have expanded their range from Big Sur following protection (Lafferty & Tinker 2014). Normally, sea otters eat grazing invertebrates and thereby help maintain kelp forests (Estes & Duggins 1995), but sea otters can shift to feeding on sand crabs (*Emerita analoga* and *Blepharipoda occidentalis*) when kelp forests become too crowded (Tinker et al. 2008). Sand crabs host larval acanthocephalan parasites that use shorebirds as final hosts. Because sea otters are the wrong final host, the worms can

accidentally perforate the otter's intestine, causing peritonitis, which appears to be responsible for 14% of sea otter strandings (Thomas & Cole 1996), and strandings with peritonitis are associated with the higher annual mortality rates that are thought to impede species recovery (Lafferty & Gerber 2002). Also slowing recovery are the 3–8% of strandings associated with brain lesions caused by the cat parasite, *Toxoplasma gondii*. Because stranded sea otters with toxoplasmosis were most common near river mouths (Miller et al. 2002), veterinarians suggested that sea otters were sentinels of a dirty ocean (Jessup et al. 2004), blaming domestic and feral cats and urging pet owners to stop flushing pet waste into the sewer. To support this contention, researchers devised a study to compare sea otter infection and mortality rates at two locations: the populated Monterey Bay and the rural Big Sur coast (Tinker 2013). Counter to expectations, *T. gondii* was not a substantial mortality source in the study (Miller et al. 2013), and the rural Big Sur otters were many times more likely to become infected with *T. gondii* (Burgess et al. 2013), perhaps due to Big Sur having more bobcats and mountain lions that, like domestic cats, carry the parasite (Lafferty 2015). Ironically, both parasites that impact sea otters are more prevalent where other wildlife is common (Lafferty 2015, Smith 2007), casting doubt on whether these sea otter diseases indicate a degraded environment.

After sea otters were hunted to near extinction, abalone and other prey species increased, creating opportunities for high-value commercial and recreational fisheries (Tegner 1989). Red abalone (*Haliotis rufescens*) was a prized delicacy in California, defining California coastal cuisine the way that Maine lobster defines New England seafood. Up through the 1970s, divers would hold abalone barbecues and line their driveways with the shells. But as stocks dwindled due to overfishing, fisheries closed, creating markets for cultured abalone. Aquaculture keeps stocks at high density and also moves stocks from place to place, subjecting cultured species to disease (Lafferty et al. 2015b). In the early 1990s, a sabellid polychaete, *Terebrasabella heterouncinata*, infested California abalone farms after an abalone farmer introduced infested South African abalone to California (Kuris & Culver 1999). South African gastropods are adapted to these polychaetes, which induce the shell to grow around them. In contrast, the worms stunted and disfigured California abalone, reducing their value by two-thirds. Eventually, the California Department of Fish and Game (now Fish and Wildlife) began to eradicate the worm from farms and the wild by cleaning up the stock, screening outflow, and halting the dumping of shell debris into the intertidal zone. Unfortunately, the outflow from one farm in Central California spread the infestation to wild abalone and other snails (Culver & Kuris 2004). This could have spread a generalist parasite throughout the state's rocky intertidal and kelp forest habitats were it not for a volunteer effort to cull gastropods from around the farm, eventually eradicating the worm in the wild (Culver & Kuris 2000). This appears to be the only time a marine parasite has been eradicated from the wild through management.

In comparison to the red abalone, the black abalone (*Haliotis cracherodii*) was small and tough. Californians did not eat it, although it made good bait. Only when red abalone were overfished did black abalone become an export to the Asian market. That fishery focused on subtidal individuals, leaving intertidal areas so dense that black abalone were sometimes stacked on one another. The black abalone was familiar to any marine biologist who ran a transect through the intertidal zone. I recall one field trip to Santa Cruz Island to show students a black abalone aggregation. Instead, they saw abalone dying by the hundreds; on the next visit, the abalone were gone. Black abalone populations crashed in series around the Channel Islands, Southern California, and then north of Point Conception (Altstatt et al. 1996, Lafferty & Kuris 1993). They have yet to return. Abalone die-offs were associated with the withering syndrome–*Rickettsia*-like organism (WS-RLO) (Friedman et al. 2002), and they appeared to have an environmental driver because infected abalone die much faster when the temperature is warm (Lafferty & Kuris 1993). Infectious-disease models

do not predict mass mortality events like what happened to black abalone. To understand why black abalone populations collapsed, Ben-Horin et al. (2013) put healthy red abalone in aquaria and varied the temperature mean and variance, finding that susceptibility increased with temperature variance but not with the mean, and that mortality increased with mean temperature but not with variance. Because black abalone live in the intertidal zone, they experience higher temperature variation than subtidal abalone species. This high variance means that susceptibility to WS-RLO in the intertidal zone is high, which leads to high infection prevalence. With almost all black abalone infected at a site, a subsequent increase in mean temperature can trigger mass mortality. In contrast, infection prevalence tends to be lower for subtidal abalone species (Cruz-Flores et al. 2016), which act as an infection reservoir, making it hard for black abalone to recover. Carolyn Friedman (Friedman et al. 2014a) at the University of Washington was interested in whether the few surviving black abalone had developed resistance, so she designed experiments to compare susceptibility and mortality in southern black abalone that had survived mass mortality events with northern black abalone that were presumably naive to the pathogen. During the survivorship experiments, black abalone exposed to WS-RLOs from farmed red abalone survived far longer than expected. Puzzled, Friedman looked for pathology in the experimental animals and found what appeared to be a new WS-RLO. On closer inspection, these unusual RLOs were found to be infected by a phage (Friedman & Crosson 2012). The phage appeared to alter the WS-RLO pathology, helping infected abalone live longer (Friedman et al. 2014a). The helpful phage has now been found associated with WS-RLOs in several farmed and wild abalone species in various countries (Cruz-Flores et al. 2016). Whereas abalone culture might have had a role in spreading the WS-RLO, it might now have a role in spreading the phage that limits the WS-RLO pathology, and this might allow black abalone to recover (Lafferty & Ben-Horin 2013).

A different intertidal infectious disease has emerged, this time in California's diverse sea star communities. Every so often, there have been sea star mortality events that seemed to correspond with warmer water events (Bates et al. 2009). After a warm June in 2013, several sea star species started to die en masse, from Alaska, USA, to Baja California, Mexico. To investigate this unprecedented mass mortality, marine biologists interfaced with microbiologists by coordinating observations, sending samples, and running experiments. Ian Hewson (Hewson et al. 2014) at Cornell University used molecular genetics to isolate and characterize a densovirus associated with the pathology (including in archived tissues dating back seven decades) and found the first-ever-reported virus for a sea star, which was also only the second reported for an echinoderm. Mortality rates were higher with warm temperatures, suggesting a link with climate (Eisenlord et al. 2016). The die-off was termed a marine emergency due to its unprecedented scope and potential indirect effects on marine communities. Specifically, Paine (1966) had shown that ochre stars prevent mussels from dominating the intertidal zone, and this classic paper is often used to show how predators maintain biodiversity. Ironically, if one looks beyond the primary space holders, diversity is far higher in mussel beds than in the adjacent areas prowled by sea stars, so how sea star wasting disease alters biodiversity depends on one's perspective (Lafferty & Suchanek 2016). Given that the densovirus is not new to California, it is hard to say whether this dramatic and unprecedented marine disease is an emergency or simply how nature regulates sea star populations. The good news is that juvenile sea stars are showing up at many places where most adults had died (Menge et al. 2016). However, one sea star species, *Pycnopodia helianthoides*, is now difficult to find anywhere, prompting environmentalists to propose they be listed as a US species of special concern (the first step toward the designation of endangered).

Each marine disease outbreak described above hints at an unbalanced system. However, climate disruption and pollution can increase or decrease parasites, depending on their details (Lafferty

1997), and it can be difficult to separate natural from anthropogenic triggers. In these case studies, leptospirosis seems like a common endemic pathogen that might help regulate growing sea lion populations, although whether it interacts with pollution-induced immunosuppression is an open question. Sea otters experience parasites that spill over from other wildlife, and exposure to shorebird acanthocephalans might be higher where sea otters have saturated kelp forest resources and sought out alternative habitats where sand crabs are more common. It seems plausible that domestic cats contribute to *T. gondii* exposure but there is, so far, little evidence that this impairs sea otter recovery more than spillover from wild cats. However, species introductions, such as the abalone sabellid worm, lead to new infectious diseases with unpredictable effects that are best prevented with biosecurity protocols and quick action when biosecurity fails. The origins of the abalone WS-RLO are unknown, although it and its phage might have coexisted somewhere with a host that, like the sabellid-infested South African abalone, was introduced to California. Perhaps, initially, only the WS-RLO spread through the naive California abalone, but later the phage arrived, reducing disease-induced death and giving California abalone a second chance. Finally, as dramatic as the recent sea star mass mortalities have been, we do not know if the densovirus regulates sea star populations or whether environmental change triggered an unnatural mass mortality. Nor do we agree how sea star mass mortalities will affect marine diversity in California. Understanding how parasites fit into marine food webs is the first step toward separating the natural parts that parasites play from the problems diseases cause in stressed host populations.

PARASITES AND MARINE FOOD WEBS

Ecologists liken food webs to ecological maps that help trace energy flows through ecosystems. As such, food webs help illustrate bottom-up versus top-down control, indirect effects such as trophic cascades, keystone predation, resource competition, resource specialization, and system stability. Publications on food webs often ignore parasites. For all the empirical food webs that ecologists have studied, in 1997 almost none contained a single parasite species. That is when two Canadian parasitologists, David Cone and David Marcogliese (Marcogliese & Cone 1997), became frustrated with the extent that ecologists ignored their field. Marcogliese & Cone (1997) argued that parasites were consumers just like predators and were probably just as diverse. They tempted ecologists to think about parasites by showing how parasites could help reveal who eats whom. They also pointed out that sometimes parasites might affect food-web properties, including predator–prey interactions. In Canadian fashion, they politely titled their review, "Food webs: a plea for parasites" (Marcogliese & Cone 1997).

One response to the Canadians' plea was an estuarine food web with parasites (Lafferty et al. 2006). In addition to building a food web from scratch, this meant capturing each species in the food web and dissecting it for parasites. Many parasites were linked to free-living estuarine species, changing the ecological map in significant ways. For example, parasites altered food-web structural properties, such as connectance, because most links in the web included parasites. Furthermore, as Marcogliese and Cone had suggested, trophically transmitted parasites revealed several unobserved predator–prey interactions. A particular link type that emerged was termed concomitant predation, whereby a predator eats the parasite within its prey. Such accidental predator–parasite links were the most common links in the food web, and many parasites used these links to help complete their life cycles. Parasites have been put into other temperate estuarine webs (Dunne et al. 2013), and we are now expanding them to coral atoll sand flats, sandy beaches, and kelp forests (J.P. McLaughlin, in preparation; D.N. Morton, in preparation). Adding parasites to a food web is like adding streets to a highway map: You would be lost without them.

Although parasites alter ecological maps, their small body sizes might render them insignificant when it comes to energy flow through the system. To compare parasites with free-living species in the same energetic currency, Kuris et al. (2008) measured biomass density for each free-living and parasitic species, finding that parasites contributed unexpected biomass and biomass production. Most notably, larval trematodes were so abundant that they rivaled top-predator biomass. Applying standard metabolic scaling theory leads to the finding that parasites are less abundant than free-living species in these estuaries after accounting for body size. But after controlling for trophic level, all species, parasitic or not, fit better onto a single log–log abundance–body size plot with the theoretically expected −3/4 slope (Hechinger et al. 2011). Adding parasites to food webs revealed a new universal law: Trophic level, body size, and metabolic rate explain animal abundance. In other words, parasites are every bit as abundant as comparable free-living species.

Although we are most concerned by the diseases parasites cause, they can also affect food webs if they alter predator–prey interactions. In estuarine food webs, the most common parasite (*Euhaplorchis californiensis*) uses the most common fish (*Fundulus parvipinnis*) as an intermediate host. The parasitic worm completes its life cycle when a bird eats an infected fish. But the worm is not patient. Infected fish have more conspicuous behaviors, and birds eat them 10 to 20 times more often than they eat uninfected fish (Lafferty & Morris 1996). These behavioral alterations help the parasite complete its life cycle, but they also alter the food web by strengthening the feeding link between birds and *F. parvipinnis*. Simple food web models predict that some predators might even depend on parasites such as *E. californiensis* to make prey easier to catch (Lafferty 1992).

Food-web effects are most noticeable when parasites impact important hosts. Some hosts are foundational species, and coral diseases reduce fish and invertebrate habitat (Alvarez-Filip et al. 2009), whereas eelgrass wasting disease removes fish and bird habitat (Hughes et al. 2002). However, diseases that reduce predator hosts can release prey species. For instance, sea urchin mass mortalities favor the macroalgae that urchins eat (Behrens & Lafferty 2004, Hughes 1994, Lauzon-Guay & Scheibling 2010), and sea star wasting disease might favor mussel beds (Lafferty & Suchanek 2016). Furthermore, diseases that remove a dominant space holder can reduce competition, as seen in the rocky intertidal zone after the black abalone mass mortality (Miner et al. 2006). Finally, diseases that diminish prey populations can compete with predators for food, as shown by lower penguin breeding success after a pilchard mass mortality (Dann 2013). These examples show how disease impacts go beyond host health by extending through food webs to cause indirect effects.

It makes sense that parasites can affect marine food webs, but it is even more obvious that marine food webs affect parasites. One example is bacterial epizootics in sea urchins along the California coast, which are four times lower inside marine protected areas (Lafferty 2004). Simply put, fishing reduces lobster density, and this indirectly increases sea urchin population density, which probably increases contact rates between infectious and healthy sea urchins, sparking epizootics. But this pattern does not generalize because adding just one species to a food web can reverse the disease dynamics. Specifically, when comparing sea urchin parasites inside and outside marine reserves in the Galápagos Islands, small crabs, which shelter on urchins and eat their parasitic snails, reverse the predicted positive association between fishing and urchin parasitism (**Figure 2**) (Sonnenholzner et al. 2011). Food-web details matter to parasites.

Although we often think parasites are problems, it is obvious that parasites depend on rich and functioning food webs. In computer simulations, parasites evolve to specialize on dependable host species; only generalist parasites can risk attacking ephemeral hosts over the long term (Strona & Lafferty 2016). As also shown with global data on fish parasites, adaptation to past conditions makes parasites robust to host extinction, but changing which hosts are most vulnerable to extinction (as might happen under anthropogenic change) makes parasites far more likely to suffer extinction

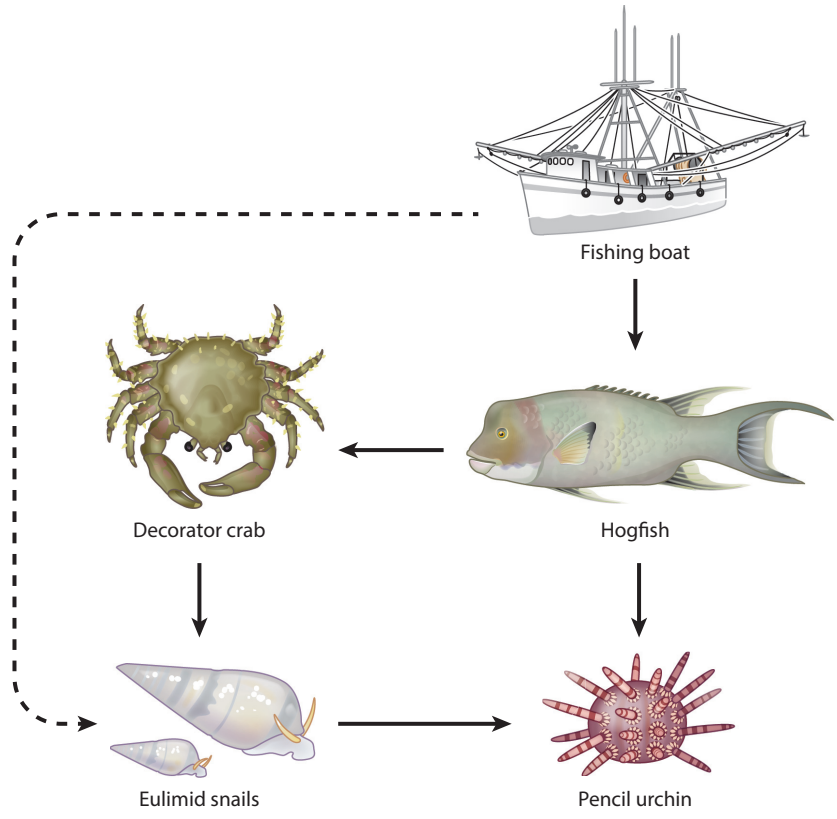

Figure 2

A Galápagos food web with fishing, showing how fishing has a net indirect negative effect (*dashed line*) on eulimid parasites of the pencil urchin due to the small crabs that eat the parasites. **Figure 2** based on concepts from Sonnenholzner et al. (2011).

(Strona & Lafferty 2016), leading to recent calls for parasite conservation (Dougherty et al. 2016). Parasite communities evolve to be robust but are sensitive to change.

If parasites are sensitive to host-species losses, then, ironically, their presence might indicate rich and functioning ecosystems. For instance, in California salt marshes, several trematode parasites use birds as final hosts and snails as first-intermediate hosts. Not surprisingly, at sites with many birds, more snails are infected, and at sites with many bird species, there are more trematode species in the snails (Hechinger & Lafferty 2005). For this reason, more trematodes in snails occurred inside a nature reserve compared with in an adjacent degraded area until 6 years after that degraded area was restored (Huspeni & Lafferty 2004). Land managers were not amused by being congratulated that they had spent several million dollars to restore parasites, but they understood the praise when it was explained that the parasites returned only because the restoration had succeeded in bringing back habitat for the invertebrates, fishes, and birds that the trematodes needed to complete their life cycles. A similar story played out in the Yucatán where a hurricane wiped out a diverse trematode community in snails that only recovered years after the invertebrate, fish, and bird populations rebounded (Aguirre-Macedo et al. 2011). Low parasite richness suggests a system in trouble. In other words, do not wait for the canary to die; you should leave the coal mine when the canary loses its parasites.

MARINE INFECTIOUS-DISEASE THEORY

Now that parasites are being put into marine food webs, modelers are developing new theoretical tools to predict their roles. Marine ecology has a strong theoretical underpinning, with a tradition in general consumer–resource modeling. Familiar examples are the Lotka–Volterra coupled differential equations that model interactions between prey and predator populations over time, and the logistic population growth equation that models how a single consumer species responds to a limited resource. Marine ecologists use these and other simple models, including single-species fisheries models that illustrate concepts such as the maximum sustainable yield (MSY). Because most ecological theory has been developed with terrestrial species in mind, models for marine infectious diseases require some rethinking to accommodate fishing, aquatic disease transmission, and oceanographic drivers (McCallum et al. 2004). One of the biggest challenges is to decipher the complex life cycles and transmission pathways that many infectious agents have (many are still unknown), and then represent this complexity with simple equations. But there are several general aspects to model building that apply across life-cycle types. Next, I discuss how to build models to better capture marine-disease dynamics.

Fishing was perhaps the first marine twist added to host–parasite models. It is as easy as adding an additional mortality term (f) to the host population (**Figure 3**). Dobson & May (1987) considered the case in which a fishery targeted a host population. Because transmission in their model increased with host density, fishing could drive the parasite to extinction. This and other fisheries models

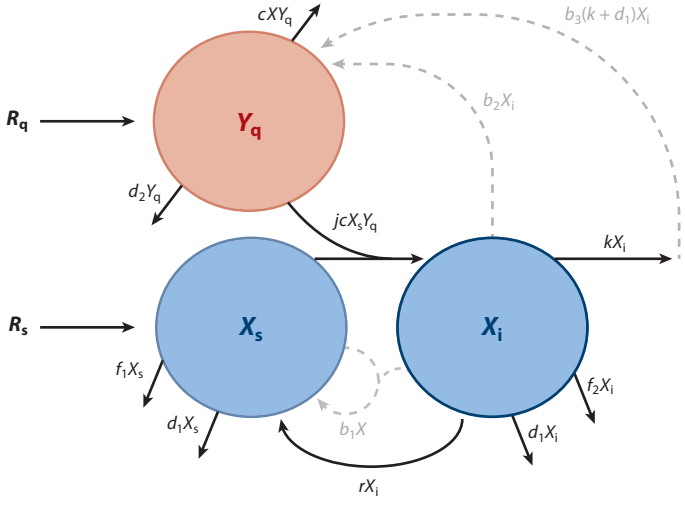

Figure 3

Flow diagram for a marine disease model for host species X (after Lafferty et al. 2015a). Here, X_s is the susceptible host density, which can transition to the infected class, X_i, through contact (c) with infective stages Y_q (which one could subsume using a separation of timescales). This simplified version assumes exposed hosts become infected rapidly and, although infected hosts can recover, there is no resistant class. The model allows host (R_s) and parasite (R_q) larval recruitment from outside the system, whereas b represents the local production of hosts (b_1), infective stages from the infected class (b_2), and infected stages following host death (b_3). The system's openness depends on its spatial scale and can be modeled by increasing R relative to b. Environmental variables might affect production rates (b), background death rates (d), additive mortality for infected hosts (k), and dose-dependent susceptibility (j). Fishing effects (f) can differ between infected and uninfected hosts. This general model does not specify functions, and its structure can be modified in many ways. For instance, a macroparasite model would merge the susceptible and infected host classes and add a parasite class.

assume that intraspecific competition limits a fishery species' per capita growth rate. Fishing reduces intraspecific competition, generates yield, and reduces stock size. One can find the MSY that optimizes the fishing rate that balances yield, stock size, and per capita production (in simple models, this is half the stock's carrying capacity, although modern fisheries models suggest a more conservative 30%). Fishing too little leaves too many fish to compete, whereas fishing too much leaves too few fish to resupply the population. Dobson & May (1987) took the classic MSY model and split the fished stock into infected and uninfected classes. They found that a parasitic pathogen decreases fishery yield if it suppresses the carrying capacity. Managing the fishery according to the MSY could lead to stock collapse if the parasite were pathogenic. However, a fishery could extirpate a pathogen from the stock if the stock density at the MSY was lower than the host-threshold density for transmission. In many ways, these insights about fishing might also apply to generic predators that eat hosts and reduce their density. In essence, parasites, predators, and fisheries compete for fish. A key assumption in this model is that fish-to-fish transmission increases with fish density. However, for fish that school, social interactions might determine contact rates more than host density does. For instance, experiments with guppies showed that epidemics do not depend on fish density: What matters is how social the initial carrier fish is (Johnson et al. 2011). Therefore, host behavior and parasite life cycles can alter predictions from simple models.

After disease and overfishing led managers to close the commercial red abalone fishery, Ben-Horin et al. (2016) wanted to know whether fishing infected abalone could have conservation benefits. Infected abalone could be sold because they pose no health risks to humans and have a market price similar to that of uninfected abalone. A model using published parameters for parasite transmission and fisheries practices showed how stock size and fishery yield might respond to targeting infected individuals. Although divers cannot diagnose whether an abalone is infected, managers can tag and then swab each abalone on a reef to test for infection back in the laboratory and inform divers which tagged abalone are infected. This way, the fishery could manage which individual abalone were fished. Although this is a cumbersome way to manage a fishery, an abalone's landed value is so high that individual-targeted fishing could be profitable. The models showed that targeting infected abalone could lead to a net increase in abalone density, resulting in a win-win for conservation and fishing if the fishery took more than 20% of the infected individuals. Furthermore, the more infected individuals that are taken, the less precise the targeting needs to be (i.e., the conservation outcome was robust to fishing some uninfected abalone). However, this model assumes close dynamics (see below), which makes it easier to fish out a parasite. At this date, the targeted fishing strategy has not been permitted.

A meta-analysis found that fishing can affect parasites, but not according to the assumptions in simple models (Wood & Lafferty 2015). Most surprisingly, parasites with direct life cycles did not decrease under fishing, suggesting that parasite transmission does not strongly increase with host density. Instead, fishing reduces parasites by removing the large, older individuals that often support the most parasite diversity. Furthermore, after controlling for host body size, parasites with complex life cycles declined if their hosts were fished, consistent with the prediction that such parasites are not robust to host removal (Rudolf & Lafferty 2011). In contrast, unfished hosts had more parasites where fishing occurred, perhaps because fishing removed predators that had kept these hosts in check, leaving parasites to fill the vacuum. To predict how fishing will affect parasites requires understanding how parasite life cycles integrate into marine food webs.

In addition to life cycles and food webs, interactions between fisheries and parasites depend on host and parasite dispersal. Kuris & Lafferty (1992) modeled how crustacean fisheries impacted by parasites could have different outcomes depending on host and parasite dispersal by considering birth rate to be either constant (open recruitment by dispersing larvae) or closed (local per capita offspring production). In addition to the closed-parasite, closed-host scenario modeled by Dobson

& May (1987), they considered open-parasite with open-host, closed-parasite with open-host, and open-parasite with closed-host scenarios, finding that open parasite recruitment makes it difficult for a fishery to manage the parasite population and sets up conditions under which the parasite can extirpate the host. Open host populations were the least affected by parasites or fisheries. Therefore, recruitment dynamics influence theoretical predictions for how to manage a fishery.

Many marine species, including parasites, have open recruitment at the scale at which most marine ecologists measure them. Perhaps the most open host–parasite systems are those that occur when opportunistic infective stages flow from land to sea (e.g., aspergillosis in sea fans, yellow band disease in corals, toxoplasmosis in marine mammals). Open recruitment in marine systems is driven, in part, by water motion that can transport passive particles. Because most marine species have planktonic larvae, offspring settling at a particular location are often not the offspring produced by adults at that location. This differs from terrestrial systems in which offspring often live near their parents. Open recruitment can be modeled by adding a constant recruitment term (R) to the host or parasite (**Figure 3**). Gaines & Lafferty (1995) used this approach to consider open recruitment in marine populations, including in host–parasite models. They noted how open and closed recruitment vary by species and scale, with systems being more closed when modeled at larger spatial scales. Recent marine disease models for the abalone WS-RLO (McCallum et al. 2005), coral disease (Sokolow et al. 2009), and oysters (Bidegain et al. 2017) have considered open and closed recruitment. For instance, Bidegain et al. (2016a) included a complex model in which infective stages come from an open pool and a local pool, depending on water motion and the inferred spatial scale. On land, the apple does not fall far from the tree, but in the ocean, the spore can drift kilometers from the kelp.

Open recruitment can disconnect infection from its source, as documented with the WS-RLO in captive abalone. For instance, in the above-mentioned abalone-infection laboratory experiments at the University of California, Santa Barbara, uninfected abalone became infected while in the campus seawater system (Ben-Horin et al. 2013). The laboratory's seawater is sourced from an ocean intake at Campus Point. Nobody had seen an abalone near Campus Point for decades, so it seemed odd that the WS-RLO's infective stages were entering from the pipe. However, a few kilometers up the coast there were thousands of infected red abalone at a commercial abalone farm. It seemed possible that the WS-RLO infections on campus were coming from the abalone farm because the WS-RLO can remain infectious in seawater for more than a week (Friedman et al. 2002), long enough to travel that distance under prevailing currents. Applying a polymerase chain reaction diagnostic for the WS-RLO (Friedman et al. 2014b) to seawater collected at the spot where the farm discharged its effluent into the ocean, and again at various distances between the farm and campus, showed that the discharge and nearby waters were loaded with WS-RLO DNA, which dissipated but was still present down the coast by the university (Lafferty & Ben-Horin 2013). Such spillover from aquaculture to wild stocks can lead to what economists call negative externalities (Lafferty et al. 2015b), which means making a profit while creating problems for someone else.

Open recruitment for an infectious disease implies a free-living infectious stage. Free-living infectious stages, such as eggs, spores, or cysts are the rule for parasites and pathogens, but most terrestrial disease modelers ignore these stages because they do not move far on land. However, as emphasized above, the aqueous environment is benign and facilitates planktonic transport, leading to open recruitment. A free-living infectious stage combined with open recruitment can cause the per capita infection risk to decline with host density (Buck et al. 2017), an important effect that marine disease models should capture. Unfortunately, adding a state variable for an infective stage makes host–parasite models more complicated, although one can use a separation of timescales

to subsume the free-living stage into the infected and uninfected host equations, resulting in a saturating (type II) functional response (Lafferty et al. 2015a).

In addition to having different spatial scales, the land and sea have different dimensionalities. It often makes sense to measure terrestrial systems in units per area, implying that ecology on land is nearly two-dimensional. This flat-earth perspective applies to infection risk as well. For instance, hookworm infection rates near a human village depend on how many infected larvae there are per square meter of surface soil. Water volume adds a third dimension to pelagic marine ecosystems, and this probably applies to some free-living infectious stages. Modeling infective-stage density should be most interesting when the volume changes relative to area. Volume change is most important in intertidal systems where the tides alter depth over the substrate. For instance, trematode cercariae shed from estuarine snails will be at higher density (and, therefore, more likely to contact a benthic host) if shed at low tide than at high tide. Furthermore, whereas oysters infected with Dermo (the dinoflagellate-like *Perkinsus marinus*) are benthic, Dermo spores are planktonic and their density can change with water depth (Ford et al. 2001). Specialized volumetric models can consider how water motion and hydrology move infective stages (Bidegain et al. 2017). Bidegain et al. (2016a) propose a marine disease model that includes how infectious stages can be diluted by the associated water volume into which they are shed, noting how outcomes depend on both water volume and the extent that transmission stages move in three dimensions, with volume acting as a reservoir, conduit, sink, or all three.

The most novel dimensionality in the ocean is the added planar habitat at the water's surface where some infective stages might float. Floating infective stages might explain how floating sea otters become infected with the terrestrial pathogen *T. gondii*, calling into question a long-standing hypothesis that otters become exposed by ingesting oocysts that survive being eaten by filter-feeding bivalves (Lindsay et al. 2004).

This is not to suggest that filter feeders are unimportant for marine parasite transmission. Some parasites exploit filter-feeding hosts (Ben-Horin et al. 2015), but for other parasites, filter feeding is an important mortality source (Bidegain et al. 2016b), increasing infective-stage mortality (d_2) (**Figure 3**). Filter feeding implies that host contact removes infectious stages (a loss term sometimes missing from models that can be important when infective stages are long lived). This can be added to models by specifying host contact (cXY_q) as a loss term for the infective stage (**Figure 3**). Areas with higher filter-feeder densities should have higher rates of infective stage loss, which should reduce host–parasite contact rates. For example, in Indonesia, where seagrass beds are present, bacterial pathogens of humans and marine animals are less abundant in seawater (and coral diseases decline by half) compared with paired sites without seagrass (Lamb et al. 2017). Although the mechanism is unknown, seagrass beds support rich and abundant filter-feeding communities, and these might process the water, digesting bacteria that would otherwise infect humans and marine animals. Therefore, one way to manage marine diseases might be to facilitate filter-feeding species (Burge et al. 2016). For instance, mussels will eat salmon lice larvae (Molloy et al. 2011), suggesting that mussel–salmon polyculture could save the farmed salmon industry millions of dollars.

Even when filter feeders are competent hosts, Bidegain et al. (2016b) notes that overfiltration can reduce transmission when infection success is dose dependent. Dose-dependent transmission arises because all free-living species have evolved defenses to infective stages, including cell types that scavenge foreign particles (Loker 1994). Such innate defenses might be swamped at high exposures (Chu & Volety 1997). Swamping is known for some human infectious diseases, such as cholera (Levine et al. 1979), and oyster examples include herpesvirus (Schikorski et al. 2011), MSX (multinucleated sphere unknown) (Haskin et al. 1966), and Dermo (Mackin et al. 1950). Bidegain et al. (2016a) modeled dose dependence by including an exposure rate below which the filter feeder can inactivate infective particles; once the exposure rate exceeds this inactivation rate, individuals

can become infected and the parasite can spread. When the host removes infective stages in considerable quantity, dose-dependent infection, along with parasite-induced host mortality, can create alternative disease states in host populations. In the first state, dense host populations keep the per capita infective stage exposure rate below the critical dose that overrides host defenses, thus preventing local epizootics from emerging. In the second state, low host density fails to filter out infective stages below the dose-dependent threshold, leading to host infection, mortality, and further reduction in host density until the system resets to a lower host density equilibrium and an enzootic parasite (Bidegain et al. 2016a). These alternative disease states might help explain why it is difficult to restore oyster reefs to high densities when Dermo is present (Powell et al. 2012).

It is also difficult to restore oyster reefs where salinity is high (Bushek et al. 2012), an indication that marine parasite transmission can be sensitive to the environment. Environmental forcing affects host–parasite dynamics, but with some differences between land and ocean. Most obviously, the oceans lack the fire and drought that might affect parasites on land. The ocean also has substantial changes in pressure and light with depth (with unknown effects on parasites). Under some circumstances, hypoxia can lead to physiological stress that might affect host–parasite dynamics. But for all the potential environmental drivers, ecologists are most familiar with temperature. Temperature affects mosquito death and biting rates and the *Plasmodium* parasite development rate, leading to nonlinear climate constraints on malaria transmission (Mordecai et al. 2013). In the ocean, most parasite life cycles include a free-living transmission stage and at least one ectothermic host, subjecting parasites to environmental temperature. Furthermore, temperature could alter host feeding rates and increase or decrease host immune responses (Nowakowski et al. 2016), which could drive exposure and susceptibility at the same time that temperature increases stress by reducing oxygen concentration. Host stress that increases susceptibility will benefit parasites, whereas host stress that decreases survivorship will hurt host and parasite populations alike (Lafferty & Holt 2003).

Corals develop under stable environmental conditions, but have declined with environmental change and associated diseases (Lafferty et al. 2004). In particular, corals can become more susceptible to infectious disease when temperatures rise enough to induce sublethal bleaching (Brandt & McManus 2009). Because sublethal bleaching in sensitive species tends to occur with cumulative exposure to temperatures above the maximum summertime mean (van Hooidonk & Huber 2009), it might be possible to predict disease risk. Specifically, the National Oceanic and Atmospheric Administration's Coral Reef Watch considers that four degree-heating weeks (when the sum of the number of degrees is greater than the maximum summer mean per week for the 12-week summer period) are likely to increase the sublethal bleaching risk (Maynard et al. 2015). These predictions are testable and, should they correspond to disease in the field, could help forecast disease outbreaks and estimate how disease might change with climate. Although managers cannot control temperature, they might control other external factors that affect disease. For instance, divers (Lamb et al. 2014) and anglers (Lamb et al. 2016) can subject corals to brown band disease, caused by a parasitic ciliate, because anchoring, gear entanglement, and accidental contact increase injuries that the ciliates can exploit. Therefore, excluding humans could mitigate the increased infection risk when temperature increases. Coral disease also increases with sedimentation (Pollock et al. 2014), which overrides benefits derived from restricting fishing on coral reefs (Lamb et al. 2015). Therefore, managers might prioritize policy and engineering to reduce sedimentation or demarcate marine-protected areas in sites not subject to sedimentation. By adding environmental forcing to models, managers might better understand how humans are cooking, smothering, and loving coral reefs to death.

Perhaps the most important reason to understand environmental forcing is in the extent that it can help trigger mass mortalities. Mass mortalities are rare, but they suggest an unbalanced

system (Harvell et al. 1999). Not all mass mortalities are parasite driven. Abrupt hypoxia, starvation, warming, cooling, or freshwater runoff can kill marine organisms en masse. As explained by experiments with black abalone (Ben-Horin et al. 2013), nonlinear relationships between environmental drivers and the death of infected hosts can create benign conditions in which parasites can build up to high prevalence, followed by mass mortality when the system crosses environmental thresholds that trigger simultaneous death. Introduced parasites might also cause mass mortalities if hosts are susceptible but naive to the parasite and there is an infection reservoir. For example, abalone herpesvirus associated with land-based aquaculture led to rapid death in wild Australian abalone (Hooper et al. 2007), and phocine distemper virus in North Sea harbor seals was probably introduced with migrating harp seals (Heide-Jorgensen & Harkonen 1992). In such cases, evolved resistance (Friedman et al. 2014a) or acquired immunity (Swinton et al. 1998) might allow host populations to recover from mass mortalities.

Mass mortalities should be more likely if dead hosts release infective stages, resulting in positive feedback that leaves few survivors. This is rare on land because most typical parasites die when their host dies. This death penalty puts limits on parasite virulence. An obvious exception is the bacterial pathogen anthrax, which releases spores when its host dies and, therefore, acts more like a parasitoid than a pathogen (Turner et al. 2014). The recent Ebola virus outbreak in West Africa pointed toward transmission from cadavers to living humans, with viable virus being present in a corpse for up to a week (Prescott et al. 2015). Plague, cholera, typhoid, tuberculosis, and smallpox can also involve limited transmission from dead bodies (Morgan 2004). However, transmission after death is not common on land because the terrestrial environment differs so much from the within-host environment that terrestrial infective stages need specialized adaptations. In the sea, the environment can be similar to host tissues in many regards, so there are fewer barriers when transitioning from inside to outside a host. This might be why some common marine pathogens, such as infectious salmon anemia virus (Vågsholm et al. 1994), white spot viral disease in shrimp (Soto & Lotz 2001), black band bacterial disease in corals (Richardson 2004), and Dermo in oysters (Bushek et al. 2002), release infective stages after the host dies.

For Dermo, spore release is higher from a dead oyster than from a live oyster, making dead and dying oysters the primary transmission sources (Bushek et al. 2002). Modeling transmission through dead hosts can be done by considering that the death of the infected host releases infective stages [$b_3(k+d_1)$] (**Figure 3**) or by creating a dead-host class that releases infective stages as it decays (Bidegain et al. 2016a). This death-transmission mode opens up new ways to control parasite spread. For instance, harvesting older oysters before they die can theoretically reduce Dermo outbreaks in fished and aquaculture oyster populations (T. Ben-Horin, C. Burge, R. Carnegie, M. Groner & D. Bushek, personal communication).

CONCLUSIONS

Future marine disease ecologists can borrow some theory developed by epidemiologists, but they should adapt that theory to marine environments. That means considering fishing, open recruitment, dimensionality, filtering, dose-dependent infection, environmental forcing, and death-based transmission, all within complex marine food webs. These new theoretical perspectives make it easier to integrate marine disease into marine ecology. With such theory, marine disease ecologists can make better testable predictions about when, where, and how marine parasites will spread, and they might even be able to forecast and manage some disease events. In addition to building a theoretical foundation, marine disease ecologists can benefit from veterinary, ecological, and environmental perspectives to diagnose diseases; determine how disease affects individual hosts; and find treatments. Imagine that a veterinarian, ecologist, and environmentalist walk into a bar

with their young colleague who has just been hired as the first marine disease ecologist. In the bar is an aquarium with several fish, one of which is in distress at the surface. The veterinarian gives a diagnosis and recommends a treatment. The ecologist suggests the tank is too crowded with fish. The environmentalist worries the temperature is too high. The marine disease ecologist thinks this advice over and then orders the distressed fish for lunch, thereby sparing the other fish in the tank from infection, after which the colleagues raise a toast to marine infectious disease ecology.

DISCLOSURE STATEMENT

The author is not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review. Any use of trade, product, or firm names in this publication is for descriptive purposes only and does not imply endorsement by the US government.

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