Infectious Diseases Affect Marine Fisheries and Aquaculture Economics

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Abstract

Seafood is a growing part of the economy, but its economic value is diminished by marine diseases. Infectious diseases are common in the ocean, and here we tabulate 67 examples that can reduce commercial species' growth and survivorship or decrease seafood quality. These impacts seem most problematic in the stressful and crowded conditions of aquaculture, which increasingly dominates seafood production as wild fishery production plateaus. For instance, marine diseases of farmed oysters, shrimp, abalone, and various fishes, particularly Atlantic salmon, cost billions of dollars each year. In comparison, it is often difficult to accurately estimate disease impacts on wild populations, especially those of pelagic and subtidal species. Farmed species often receive infectious diseases from wild species and can, in turn, export infectious agents to wild species. However, the impact of disease export on wild fisheries is controversial because there are few quantitative data demonstrating that wild species near farms suffer more from infectious diseases than those in other areas. The movement of exotic infectious agents to new areas continues to be the greatest concern.

INTRODUCTION

An average person eats approximately half his or her weight in seafood each year. The 156 million metric tons (mmt) of total seafood from 2011 landings had a farm production value exceeding US$215 billion (FAO 2012), accounting for ~0.3% of the world economy. However, the seafood profits that go to wild fisheries are US$50 billion less than they could be, because it takes more effort to maintain yield (Willman et al. 2009). With fisheries unable to keep up with the growing demand for seafood, aquaculture has expanded and now exceeds fisheries production (FAO 2012). A larger role for aquaculture has led to more seafood production, competition between wild and farmed fish production, and increased trade. One major cost to aquaculture is marine infectious diseases.

In this review, we begin by considering the infectious diseases known or suspected to have economic impacts, and then ask what economic costs they impose and why. We next discuss the impacts of disease on fisheries and aquaculture. Throughout, we review ways to mitigate the economic losses to infectious disease. We illustrate these points with five case studies for which information exists on economics and disease management: Dermo in oysters, various diseases in abalone, white spot disease in shrimp, betanodaviruses in groupers, and several diseases in salmon.

WHAT MARINE DISEASES HAVE ECONOMIC CONSEQUENCES?

Marine diseases are a natural part of ocean ecosystems, and many have economic consequences for fisheries or aquaculture. Of the 67 examples in **Table 1**, 25% are viruses, 34% are bacteria, 19% are protists, and 18% are metazoans; in terms of the hosts, 49% of the infectious agents affect fishes, 21% affect crustaceans, 28% affect molluscs, and 1% affect echinoderms. Most examples come from temperate waters, with a higher percentage from the Northern Hemisphere than from the Southern Hemisphere. Suffice it to say, many infectious agents cause significant marine diseases, and they infect a range of fished and cultured species.

Nonseafood marine species can also have economic value that can be diminished by disease. For example, tropical aquarium fishes are marketed through the pet trade, and corals, shells, and sea stars are sold as decorative pieces and souvenirs. Nontraded species can also have economic value: Mangroves, seagrasses, and coral reefs provide valuable ecosystem services, such as supporting fisheries and protecting shorelines from erosion. Iconic marine species such as whales, dolphins, corals, sea stars, and sea otters support tourism, but people also value their existence irrespective of any commercial interest. Such "indirect use benefits" and "existence values," and the consequent losses from diseases, are difficult to measure because of the absence of market prices or marginal harvest costs (Costanza et al. 1998, Peterson et al. 2003). Thus, although the infectious diseases of many marine species can have large economic impacts, we focus on seafood species.

ECONOMICS 101 FOR MARINE DISEASES

Economists use market data to estimate seafood's direct use value. Although the local economy that a fishery or aquaculture industry supports is important to workers and residents—some estimates place the indirect seafood economy at three times the direct economic value (Dyck & Sumaila 2010)—the wages received by workers are also a cost to the vessel owner or processing plant and therefore do not represent a fishery's true economic value. For example, money spent on processing infected fish fillets or vaccinating farmed salmon leads to employment and trade but

Table 1 Representative marine disease agents with known or potential economic significance

Host	Disease agent	Region	Impact
Molluscs			
Viruses			
Abalone (*Haliotis laevigata* and *Haliotis rubra*)	Abalone ganglioneuritis virus (Victoria)	Australia (Victoria)	Mortality
Oysters (*Crassostrea angulata*)	*Crassostrea angulata* iridovirus	France	Mortality
Oysters (*Crassostrea gigas*)	Ostreid herpesvirus 1 (including μVar)	Europe, Asia, New Zealand, Australia western United States (California)	Mortality
Bacteria			
Abalone (various)	Withering syndrome rickettsia-like organism (WS-RLO)	North America, Asia, Europe, Iceland, South America	Mortality
Abalone (*Haliotis tuberculata*)	*Vibrio harveyi*	France	Mortality
Oysters (*Crassostrea gigas*)	*Vibrio tubiashii*	North America, Europe	Mortality
Oysters (*Crassostrea gigas*)	*Nocardia crassostreae*	United States, Japan, Europe	Mortality
Clams (various)	*Vibrio tapetis*	Europe	Growth
Protists			
Molluscs (various)	*Perkinsus olseni*	Temperate and tropical regions	Mortality
Oysters (*Crassostrea virginica*)	*Haplosporidium costale*	Eastern United States	Mortality
Oysters (*Ostrea edulis*)	*Bonamia exitiosa*	Southern Ocean, Europe	Mortality
Oysters (*Ostrea edulis*)	*Bonamia ostreae*	North America, Europe	Mortality
Bivalves (various)	*Marteilia refringens*	North Africa, Europe	Mortality
Oysters (various)	*Mikrocytos mackini*	Western Canada and United States	Mortality
Bivalves (various)	*Perkinsus marinus*	Western Atlantic and Gulf of Mexico, western Mexico	Mortality
Oysters (various)	*Haplosporidium nelsoni*	Eastern and western North America, Japan	Mortality
Scallops (*Pecten maximus*)	*Perkinsus qugwadi*	Western Canada	Mortality
Metazoan			
Abalone (various)	*Terebrasabella heterouncinata*	Western United States (California), South Africa (farms)	Growth, mortality
Unknown			
Clams (various)	QPX (quahog parasite unknown)	North America, Europe	Growth
Crustaceans			
Viruses			
Shrimp (penaeid)	*Baculovirus penaei*	Southeast Atlantic, Gulf of Mexico, Caribbean, Pacific	Growth, mortality
Shrimp (penaeid)	Infectious hypodermal and hematopoietic necrosis virus	Americas, Asia, Africa, Western Indo-Pacific, India, Australia	Mortality
Shrimp (penaeid)	*Aparavirus* spp.	Americas, Middle East, Southeast Asia	Mortality
Shrimp (penaeid)	*Penaeus monodon*–type baculovirus	Tropical regions	Growth, mortality
Shrimp (penaeid)	*Whispovirus* spp.	Asia, India, Middle East, Mediterranean, Americas	Mortality
Shrimp (penaeid)	*Okavirus* spp.	Asia, Sri Lanka, Australia, Mexico	Mortality
Lobsters (panulirid)	*Panulirus argus* virus 1	Western Atlantic	Mortality

(*Continued*)

Table 1 (*Continued*)

Host	Disease agent	Region	Impact
Bacteria			
Lobsters (homarid)	*Aerococcus viridans* var. *homari*	North Atlantic	Mortality
Shrimp (penaeid)	Necrotic hepatopancreatitis bacterium	Americas	Mortality
Shrimp (penaeid)	*Vibrio parahaemolyticus*	Asia	Mortality
Protist			
Crabs (various)	*Hematodinium* spp.	Arctic Sea, Bering Sea, North Pacific, North Atlantic	Marketability, mortality
Metazoans			
Crabs (various)	Rhizocephalan barnacles	Worldwide	Marketability, recruitment
Crabs (Dungeness)	*Carcinonemertes errans*	North America	Recruitment
Crabs (king)	*Carcinonemertes regicides*, *Ovicides paralithodis*	Alaska	Recruitment
Echinoderm			
Protist			
Green urchins	*Paramoeba invadens*	Northwest Atlantic	Mortality
Fishes			
Viruses			
Fishes (marine)	Nodaviruses	Worldwide	Mortality
Fishes (herring)	Hemorrhagic septicemia virus	Holarctic	Mortality
Red sea breams	Red sea bream iridovirus	Japan, Mediterranean	Mortality
Pilchards	Pilchard herpesvirus	Australia	Mortality
Salmon	Infectious salmon anemia virus	North Atlantic, Chile	Mortality
Salmon	Infectious hematopoietic necrosis virus	United States, Europe, Japan	Mortality
Fishes (various)	Aquabirnaviruses	Worldwide	Mortality
Bacteria			
Fishes (marine)	*Amyloodinium ocellatum*	Tropical and subtropical regions worldwide	Mortality
Flounders	*Edwardsiella tarda*	Japan	Mortality, health
Fishes (marine)	*Vibrio* spp.	Worldwide	Mortality
Fishes (marine)	*Mycobacterium* spp.	Worldwide	Mortality
Fishes (marine)	*Streptococcus* spp.	Worldwide	Mortality
Fishes (marine)	*Listonella anguillarum*	Worldwide	Mortality
Fishes (marine)	*Moritella viscosa*	North Atlantic	Morbidity
Fishes (marine)	*Photobacterium damselae*	United States, Japan, Europe	Mortality
Fishes (marine)	*Tenacibaculum maritimum*	United States, Japan, Europe	Mortality
Salmon	*Renibacterium salmoninarum*	Worldwide	Mortality
Salmon	*Vibrio salmonicida*	Holarctic	Mortality
Salmon	*Aeromonas salmonicida*	Holarctic	Mortality
Yellowtail	*Nocardia seriolae*	Japan	Mortality
Fishes (marine)	*Chlamydia*-like bacteria	Worldwide	Growth
Yellowtail	*Lactococcus garvieae*	Japan	Mortality

(*Continued*)

Table 1 (*Continued*)

Host	Disease agent	Region	Impact
Protists			
Fishes (marine)	*Ichthyophonus hoferi*	Holarctic	Mortality
Fishes (marine)	*Cryptocaryon irritans*	Tropical and subtropical regions	Mortality
Fishes (marine)	*Amyloodinium ocellatum*	Worldwide	Mortality
Metazoans			
Fishes (marine)	*Kudoa* spp.	Worldwide	Marketability
Salmon	*Parvicapsula* spp.	Norway	Mortality
Fishes (marine)	*Enteromyxum* spp.	Mediterranean, Japan	Mortality
Fishes (marine)	Anisakid nematodes	Worldwide	Marketability
Fishes (marine)	*Cryptocotyle lingua*	Worldwide	Growth, mortality
Fishes (marine)	Monogenea (various)	Worldwide	Growth, mortality
Salmon	*Eubothrium* spp.	Europe	Growth
Fishes (marine)	*Lepeophtheirus* spp., *Caligus* spp.	Worldwide	Growth, mortality

diminishes profits. Specifically, the net economic benefit is the value to society from seafood harvest or production less the seafood harvest or production cost.

Because fish stocks are exploited over time, a fishery has its highest economic value when it maximizes the discounted value of present and future net benefits. At some point, the cost of catching another fish outweighs its value. For instance, increasing the harvest could decrease a stock's growth rate, thereby increasing future harvesting costs. Infectious diseases could change the optimal strategy: If a healthy fish is going to die before it spawns, one might benefit from harvesting it (Conrad & Rondeau 2014).

Marine diseases can impact a species' economic value in two ways. The first comes from the reduction in potential catch resulting from decreased biological productivity, which can arise from increased mortality, slower growth, immune defenses invested by hosts, and host responses to infection that harm both the hosts and the infectious agents. The second comes from a bad taste or appearance or from risks (or assumed risks) to human health. Not surprisingly, consumers avoid seafood if they can see or taste parasites. In some extreme cases, diseased animals, like crabs with bitter crab disease, lose all commercial value. Sorting out diseased crabs from the catch increases harvesting costs and thus reduces the total catch's net economic worth. Several infectious agents reduce the market value of infected muscle tissue, including sealworm larvae in the flesh of cod, pollock, and herring (McClelland 2002); visible myxozoans and postmortem myoliquefaction associated with *Kudoa thyrsites* (Moran et al. 1999); the ichthyosporean genus *Ichthyophonus* (White et al. 2013); and the hyperparasite *Urosporidium spisuli* in nematodes parasitizing surfclams (Perkins et al. 1975). Dealing with these infectious agents increases processing costs and thus reduces seafood's net value.

Negative externalities interest economists and challenge management. A negative externality occurs when a firm or individual impairs the productivity or welfare of others, such as when an aquaculture farm exports an infectious disease that then impacts a fished stock. Negative externalities are difficult to manage because neither the creator of the externality nor decision makers face external costs, and thus they have no direct incentives to take corrective measures. Therefore, controlling or managing a marine disease with an external effect might include coordinating individuals, firms, and government agencies.

FISHERIES AND INFECTIOUS DISEASES

The most obvious way to reduce marine diseases is to increase the harvest. Fishing reduces host density and can break the transmission of host-specific, density-dependent infectious diseases (Amundsen & Kristoffersen 1990, Dobson & May 1987, Kuris & Lafferty 1992, Ward & Lafferty 2004, Wood et al. 2010). This does not mean that overfishing is in a fishery's best interest; rather, as a fishery collapses, host-specific infectious diseases should, in theory, become a relatively minor problem.

Nonetheless, some fishing activities can increase infectious diseases. One example would be a fishery restoration effort that unknowingly outplanted infected abalone (Friedman & Finley 2003). Another example concerns fisheries that release infected individuals. Even a small amount of crabmeat infected with bitter crab disease will contaminate an entire can; crabbers can distinguish most healthy from infected crabs and, to avoid contaminating the catch, often throw infected crabs overboard while moving to a new fishing ground (Taylor & Kahn 1995), spreading the dinoflagellate that causes the disease. Similarly, Australian pilchards imported for bait might have introduced a herpesvirus linked to mass pilchard mortalities (Whittington et al. 1997). Fisheries should therefore assume that stocks carry infectious agents and be cautious about moving them.

Fisheries can also indirectly increase infectious diseases in nontarget species. An example is the California spiny lobster fishery: This fishery is profitable and sustainable, but lobster fishing can indirectly increase lobster prey, such as purple and red sea urchins, leading to compensatory increases in epizootics that reduce sea urchin populations, including commercially valuable species (Lafferty 2004). Here, the lobster fishery might benefit the red sea urchin fishery by reducing predation on urchins, but it also fosters epizootics, so the net effect on the urchin fishery is unclear. Whether their effects are indirect or direct, fisheries have commensurate impacts on infectious disease dynamics through their influence on host population dynamics.

AQUACULTURE AND INFECTIOUS DISEASES

Wild stocks support many infectious agents, which can enter aquaculture farms through water intake, feed, or infected broodstock. As a result, most infectious diseases in farmed hosts come from wild hosts in the surrounding waters (Kurath & Winton 2011, McVicar 1997, Saksida et al. 2014) (**Figure 1**). Regardless of the disease source, new aquaculture species can be hit by series of infectious diseases over time. For instance, Japanese yellowtail (*Seriola quinqueradiata*) culture became industrialized around 1961; vibriosis became a problem in 1963, nocardiosis and ichthyphoniasis in 1967, pseudotuberculosis in 1969, streptococcosis in 1974, lymphocystis in 1975, and so on (Egusa 1983). Aquaculture's history is one of victories over diseases followed by new challenges.

Infectious diseases that might not normally affect wild hosts can become problematic in aquaculture. Farmed stock is often not coevolved with local infectious agents, and this can lead to unpredictable outcomes. For example, local *Streptococcus iniae* (bacterial) strains tend to be more pathogenic to imported, naive fishes than to local wild fishes (Colorni et al. 2002). Another factor that makes aquaculture susceptible to disease outbreaks is the high stocking density of monocultures, which increases host contact rates and stress and reduces water quality (Kent 2000). These conditions are perfect for opportunistic bacterial infections by ubiquitous marine pseudomonads and vibrios (especially *Vibrio anguillarum*) (Sinderman 1984). By contrast, infectious agents with complex life cycles should do worse in simplified aquaculture settings than in the wild, where intermediate hosts are present.

To stay profitable, farms must invest in prevention and treatment. Preventing opportunistic diseases requires good water quality and moderate stocking density (Sinderman 1984). Farms can

		Export from wild to farmed species		
		No	**Maybe**	**Yes**
Export from farmed to wild species	**Yes**	Abalone ganglioneuritis virus Aquabirnaviruses *Terebrasabella heterouncinata* (California)	Nodaviruses	*Aeromonas salmonicida* *Amyloodinium ocellatum* *Bonamia exitiosa* *Haplosporidium costale* *Haplosporidium nelsoni* *Perkinsus marinus* QPX (quahog parasite unknown) Sea lice (salmon) *Vibrio tapetis* *Whispovirus* spp. Withering syndrome rickettsia-like organism (WS-RLO)
	Maybe	*Cryptocaryon irritans*	*Aparavirus* spp. *Baculovirus penaei* *Chlamydia*-like bacteria *Crassostrea angulata* iridovirus *Enteromyxum* spp. Infectious hematopoietic necrosis virus Infectious salmon anemia virus *Lactococcus garvieae* *Listonella anguillarum* *Marteilia refringens* *Mikrocytos mackini* Monogenea (various) *Moritella viscosa* Necrotic hepatopancreatitis bacterium *Nocardia seriolae* *Okavirus* spp. Ostreid herpesvirus 1 µVar *Parvicapsula* spp. *Penaeus monodon*–type baculovirus (nucleopolyhedrovirus) *Perkinsus olseni* *Photobacterium damselae* Red sea bream iridovirus *Tenacibaculum maritimum* *Vibrio harveyi* *Vibrio parahaemolyticus*	*Ichthyophonus hoferi* *Mycobacterium* spp. *Perkinsus qugwadi*
	No	Anisakid nematodes *Cryptocotyle lingua* *Eubothrium* spp. *Hematodinium* spp. Hemorrhagic septicemia virus *Kudoa* spp. *Nocardia crassostreae* *Renibacterium salmoninarum* *Vibrio salmonicida* *Vibrio tubiashii*	*Caligus* spp. Sea lice (cod)	*Terebrasabella heterouncinata* (South Africa)

Figure 1

Infectious disease interactions between wild and farmed stocks.

also treat stocks with pesticides and antibiotics. However, few therapeutants are registered for use in aquaculture in many countries owing to the expense of obtaining governmental approval combined with the small markets and potential health risks for consumers (Friedman et al. 2003, Morrison & Saksida 2013). Fortunately, vaccines for viruses and bacteria can be effective for finfish, which possess an adaptive immune response, and immunostimulants have shown some promise in invertebrates (Smith et al. 2003). For example, furunculosis and vibriosis were devastating to salmon cage culture in the 1980s and 1990s (McVicar 1997), but after vaccine use, these conditions became almost nonexistent, reducing the need for antibiotics (Lillehaug et al. 2003). Aquaculturists also minimize disease impacts by using frozen or dried feed that limits foodborne disease transmission, separating wild-caught foundational stock from cultured offspring, and growing disease-resistant genetic stocks. With good management, the added mortality from opportunistic infections can be minor in aquaculture, and the costs of reduced food conversion and growth associated with chronic infections then become the important concerns (Scholz 1999). In addition, the routine cost of using pesticides, vaccines, and therapeutics, for either prophylaxis or treatment, is substantial (Asche & Bjørndal 2011, Dixon 2012). As such, aquaculturists must balance the costs of prevention and treatment against those of decreased production and value.

WHEN MARINE DISEASES MOVE FROM AQUACULTURE TO WILD SPECIES

One promise of aquaculture was that it would relieve fishing pressure on declining wild stocks and help to restore ocean ecosystems. This has happened to some extent, but aquaculture has also led to the escape of exotic species, eutrophication, habitat destruction, the conversion of scarce protein feed to luxury commodities, and the spread of marine diseases (Diana 2009). In **Figure 1**'s tabulation of marine diseases of economic importance, 45 of 57 infectious agents found in aquaculture are or might be exported from farms to wild species. Whether disease export affects wild species depends on the quantity, location, and nature of the exported infectious agent combined with host susceptibility, resistance, and tolerance.

Infectious agents can build up on farms with poor management, but good management can also foster infectious agents if farms manage for host tolerance to infection. In California, abalone farms can maintain red abalone infected with withering syndrome rickettsia-like organism (WS-RLO) in locations with cool temperatures. Although bacterial loads and transmission are low at these temperatures (Braid et al. 2005, Friedman & Finley 2003), tolerant red abalone might become sources of WS-RLO for wild (and endangered) black abalone if farms discharge effluent into abalone habitats (Lafferty & Ben-Horin 2013).

Where farming fosters disease transmission, infective stages should be higher in farm outflow than in farm inflow, making aquaculture a net exporter of infectious disease to wild stocks. Currents, outflow flushing, and infective-stage longevity determine the dispersal kernel of the disease plume. For example, a strong DNA signal of WS-RLO from an abalone farm discharge pipe quickly dissipates within less than one to a few kilometers of the coastline (Lafferty & Ben-Horin 2013).

Whether farm effluent containing an infectious agent affects wild stocks depends on many factors. The extent to which an effluent plume reaches wild species that are sensitive to infection is the most obvious concern. As discussed below, siting salmon pens in areas where wild smolts return to the ocean is controversial for this reason. However, wild stocks are often adapted to their infectious agents, and the population-level consequences of increased exposure to most native pathogens should be mild (Jackson et al. 2013), although increased exposure to exotic or introduced pathogens is obviously a danger to wild stocks. Regardless, the economic impacts are difficult to document, as exemplified by sea lice on salmon, because there is not enough baseline

information on infectious diseases in wild stocks to assess the net effect of disease export from aquaculture (Kent et al. 1998). Siting aquaculture away from the habitats of susceptible hosts and developing more rigorous best practices are obvious ways to reduce potential externalities from aquaculture, but this can carry significant economic costs to aquaculture.

The stakes increase when aquaculture releases infectious agents that have evolved in culture (Kurath & Winton 2011). In farms, susceptible hosts are always available, minimizing a key cost to the parasite of harming the host. A possible example is infectious salmon anemia, a viral disease of farmed Atlantic salmon that is thought to be nonpathogenic in wild fish but is highly pathogenic in farms (Raynard et al. 2007). On the one hand, adaptation to the water quality, host type, and treatment regime within a farm should select for infectious agents that become maladapted to the wild; on the other hand, high culture densities could create a bridge that allows exotic and maladapted infectious agents time to adapt to the farm environment and eventually establish themselves in the wild. Alternatively, culture-adapted infectious agents might cause suboptimal pathologies in naive wild hosts, leading to greater impacts than is seen in coevolved host-parasite relationships. Either way, this is a large knowledge gap, and the outcome is difficult to predict.

Impacts on wild species are easier to document when aquaculture moves farmed species and their infectious agents. One such infectious agent is *Bonamia ostreae*, which was introduced in Europe in the late 1970s through infected flat oyster spat from California, spread throughout Europe as oysters were transferred to different growing areas, and spilled over into wild oysters (Friedman et al. 1989). The spread of these kinds of novel diseases has led to regulations on moving cultured species between different areas; most often, new species are certified free of specific infectious agents before import. For instance, limiting translocations to egg stages keeps most infectious agents from spreading (Kent & Kieser 2003). Of the infectious agents that slip through the screening process, few will persist in wild stocks (Kennedy et al. 1991). For those that do, however, the local stocks will often lack an evolutionary history with the infectious agent, leading to substantial disease (Johansen et al. 2011, Johnsen & Jensen 1991). Although disease spread from aquaculture has declined with better management, aquaculture still contributes to outbreaks of novel infectious agents (Raynard et al. 2007), such as *Haplosporidium nelsoni* (introduced by Pacific oysters from Japan to eastern oysters in the United States) (Burreson et al. 2000) and the sabellid worm *Terebrasabella heterouncinata* (introduced from South African abalone to Californian abalone) (Kuris & Culver 1999).

CASE STUDIES

Dermo in Oysters

The oyster culture industry has moved oyster diseases around the world, and several of these diseases have led to substantial mortality and economic impacts (Sinderman 1984). Most of the cost of oyster diseases falls on aquaculture because the 4.5 mmt produced via aquaculture (valued at US$3.7 billion) towers over the current 200,000 mt per year of wild harvest (FAO 2012).

Perkinsus marinus is a parasite that causes Dermo disease in adult eastern oysters (*Crassostrea virginica*). This apicomplexan protozoan, which is related to dinoflagellates, has a swimming zoospore. Under experimental conditions, zoospores are capable of initiating infections; however, they are not as infective as vegetative stages (Chu 1996, Perkins 1988), and their role in transmission under natural conditions is unknown. The parasites are acquired through feeding and often colonize digestive epithelia before entering the hemolymph and becoming systemic. The parasite is usually found intracellularly in hemocytes (Bushek et al. 1997, Mackin 1951), where it proliferates. The parasite is released into the water with host feces or when the host dies and decomposes (Bushek

et al. 2002). Prevalence varies in time and space, but it is not unusual for most oysters to be infected (**Figure 2**).

Dermo was first identified in the late 1940s in the northwestern Gulf of Mexico (Ray & Chandler 1955) and shortly thereafter was recorded as far north as Chesapeake Bay. Beginning around 1990, the parasite's range expanded into the Gulf of Maine (Ford & Chintala 2006). Warm winters and droughts (Powell et al. 1996), along with El Niño–Southern Oscillation cycles in the Gulf of Mexico and Northern Atlantic Oscillation cycles in the Mid-Atlantic (Soniat et al. 2009), are implicated in triggering Dermo epizootics.

During epizootics, Dermo can reduce oysters' carrying capacity fivefold (Powell et al. 2009). In the Mid-Atlantic region, Dermo has absorbed at least two-thirds of the surplus production available to the fishery (Bushek et al. 2012, Powell et al. 2011), severely limiting the sustainable harvest throughout this region (Mann & Powell 2007). In Delaware Bay, with roughly 2,000 mt landed, the total potential ex-vessel economic loss to Dermo equates to approximately US$6 million per year. Dermo is likely to be a long-term cost for oyster growers because resistance has been slow to evolve owing to susceptible oysters persisting in low-salinity refuges (Powell et al. 2012b).

Dermo impacts more than the oyster industry. Oysters build reefs that attract more oysters and reef-associated species that both support fisheries and contribute ecosystem services such as shoreline wave barriers and natural filtration. Dermo-infected reefs recede, and this is abetted by overfishing of the diseased stock (Powell et al. 2012a). Thus, the restriction on stock production imposed by Dermo can explain much of the oyster reef loss in the Gulf of Mexico and Mid-Atlantic (Beck et al. 2011). Only one program is testing the management of a Dermo-infected population under the dual requirement of a sustainable stock and habitat (Soniat et al. 2012).

Dermo also complicates those oyster restoration efforts that focus on creating unfished oyster sanctuaries (Paynter et al. 2010). Most sanctuaries are in the lower-salinity (<12‰) reaches of estuaries (Paynter et al. 2010) because these areas have higher survival rates from disease. This strategy protects susceptible genotypes (Ford et al. 2012) whose offspring recruit down-estuary, where they become infected with Dermo, helping the disease persist in the system (Munroe et al. 2012). If the evolution of resistant genotypes is a long-term fisheries management goal, one would instead fish upper-estuary sites to select against susceptible genotypes and protect resistant survivors in the lower estuaries, but implementing this strategy is not an easy sell (Carlsson et al. 2008, Munroe et al. 2013).

Current and future research on Dermo is focusing on the development of disease-resistant genotypes in oysters and the identification of loci contributing to genetic resistance (Abbe et al. 2010, He et al. 2012, Powell et al. 2012b), the development and testing of new management practices to retain carbonate balance in fished populations burdened by the disease (Soniat et al. 2012), and the application of classic transmission dynamics models to investigate the influence of natural and anthropogenic changes on the dynamics of the disease process. The approach in most of these studies is to develop new ways to sustain oyster culture both in aquaculture and in the fishery that is consistent with the continued infection of 50% or more of the adult oysters in most East and Gulf Coast populations by *P. marinus* (Kim & Powell 2007).

Withering Syndrome Rickettsia-Like Organism, Sabellid Worm, and Herpesvirus in Abalone

Wild abalone fisheries are a high-value, high-cost, shallow-water endeavor, and these characteristics make abalone stocks vulnerable to overharvesting and poaching under open-access management. Several stocks that supported diving-based fisheries have collapsed (Karpov et al. 2000). Annual production from wild abalone fisheries now stands at approximately 100,000 mt

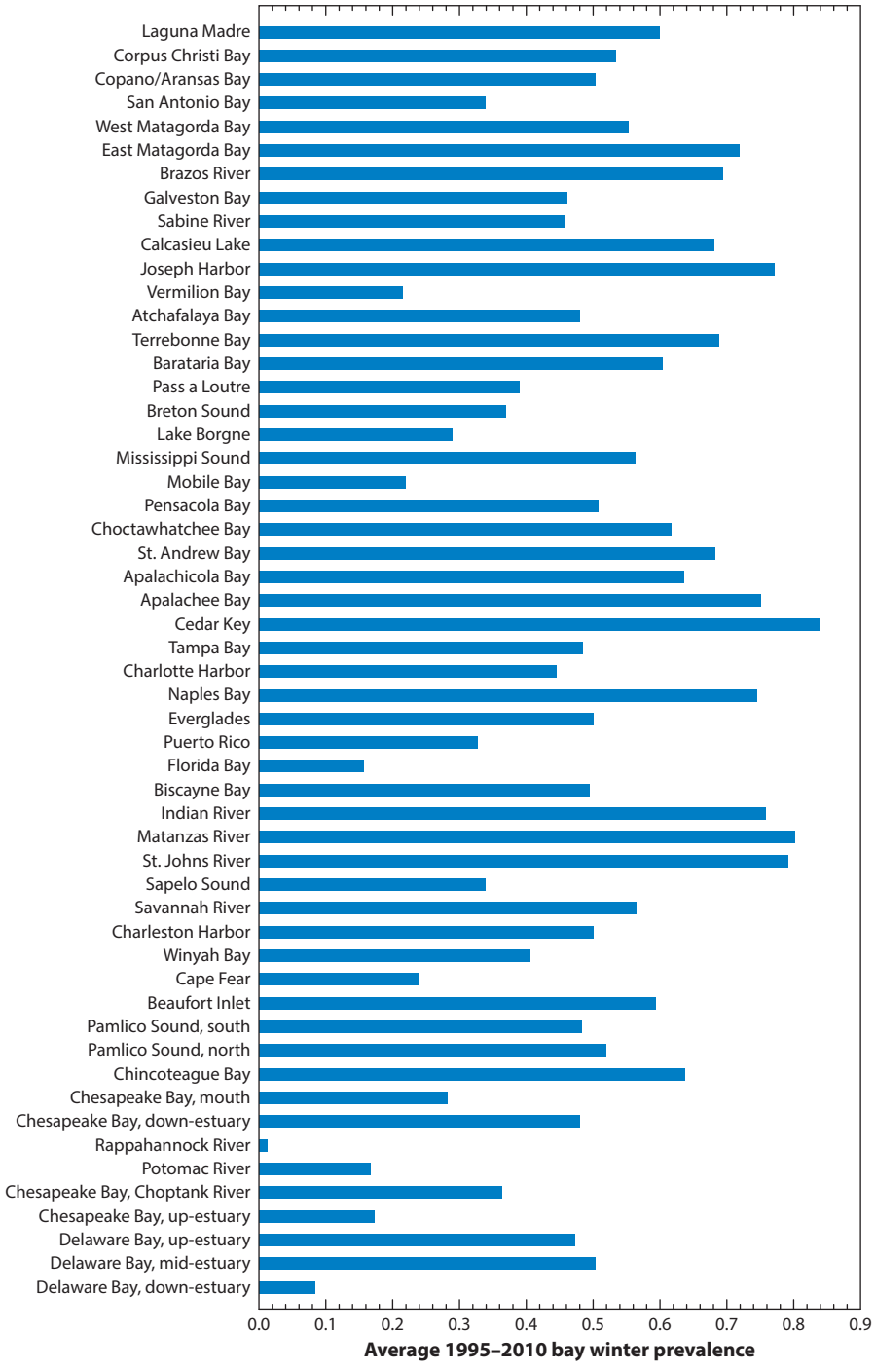

Figure 2

Average Dermo prevalence for bays sampled in the winter, when prevalences are normally near yearly lows, by the NOAA Status and Trends Mussel Watch program from 1995 to 2010. For details of the sampling program, see Kim & Powell (2007).

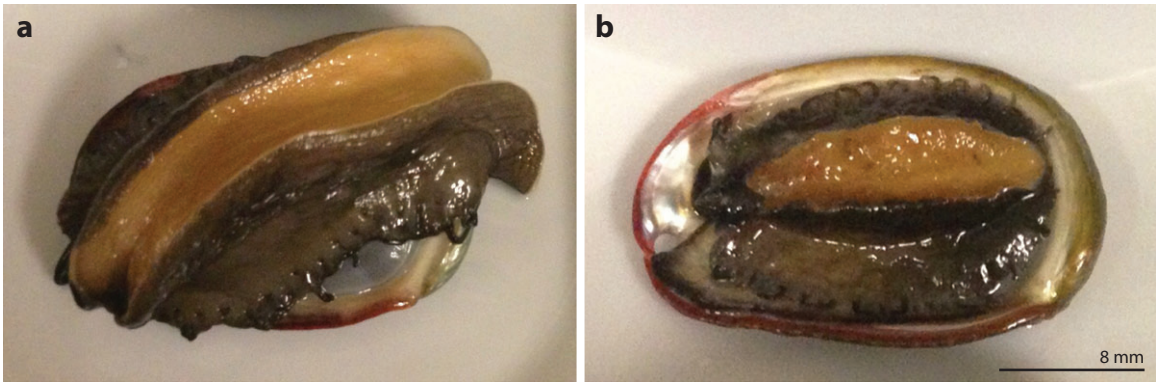

Figure 3

Red abalone (*Haliotis rufescens*) (*a*) without and (*b*) with withering syndrome. Note that the abalone with withering syndrome has an atrophied pedal muscle and is lethargic, whereas the uninfected abalone is actively trying to right itself. Photographs courtesy of L. Crosson.

(FAO 2012). Although abalone grow slowly, farmed abalone production (400,000 mt) has a high farm-gate value worth approximately US$1 billion (FAO 2012), despite production losses caused by several novel infectious agents.

In the mid-1980s, WS-RLO caused a fatal withering syndrome in various California abalone species (Crosson et al. 2014). WS-RLO is an intracellular bacterium that spreads from abalone to abalone when bacteria are passed with abalone feces and then consumed by an abalone during feeding. At warm temperatures, the bacteria damage the digestive gland so that the animal starves, catabolizing proteins from a withering foot before dying (**Figure 3**). All wild fisheries were closed as abalone populations crashed because of both overfishing and WS-RLO. With no commercial fishery in California, prices for cultured red abalone (*Haliotis rufescens*) increased. Although WS-RLO infected cultured abalone, farms could keep red abalone alive to market size if they were located in cooler-water sites or—for one farm that participated in the US Department of Agriculture's Investigational New Animal Drug program for approximately one year—by applying the antibiotic oxytetracycline (Friedman et al. 2003, Moore et al. 2011). However, antibiotic use was discontinued because abalone were declared safe to sell only one year after the oxytetracycline treatment (Friedman et al. 2007). By 2008, abalone culture in California had risen to ~227 mt (US$8–9 million) (FAO 2012). It remains unclear whether WS-RLO discharged from abalone farms is an externality that prevents endangered black abalone from recruiting (Lafferty & Ben-Horin 2013). A newly found phage hyperparasite of WS-RLO increases tolerance to WS-RLO in abalone farms and might signal a reprieve for wild populations as well (Friedman et al. 2014).

In the early 1990s, California abalone farms became infested with a sabellid polychaete, *Terebrasabella heterouncinata* (Kuris & Culver 1999) that an abalone farmer unintentionally introduced along with abalone from South Africa. These polychaetes settle on the growing margin of gastropod shells and induce the shell to grow around them; the worms then brood larvae that can settle on nearby hosts. On the shells of their coevolved, wild South African hosts, the worms appear benign, but they slow growth and disfigure Californian abalone. Most abalone in farms became infested, and abalone with heavy sabellid infestations had ~30–40% the value of uninfested animals. In response, the California Department of Fish and Game established policies to eradicate the worm from farms and the wild, including cleaning up the stock, screening outflow, and halting shell-debris dumping into the intertidal zone; however, because the outflow from one farm was

situated over a rich rocky intertidal area, the discharged sabellids spread to wild abalone and other gastropods (Culver & Kuris 2004). Volunteers eventually eradicated the worm by culling potential hosts (Culver & Kuris 2000).

Abalone viral ganglioneuritis (AVG) appeared in several abalone farms in Victoria, Australia, in December 2005. This virus is transmitted horizontally (individual to individual); vertical transmission (parent to offspring) is not verified. Because the host appears to have had no evolutionary history with the virus, the disease killed 90% of the stock, but the potential for virus latency in survivors is a concern. Then, in May 2006, AVG appeared in wild *Haliotis rubra* and *Haliotis laevigata* stocks next to an infected abalone farm. By August 2006, AVG had spread 5 km to the west and 12 km to the east (Hooper et al. 2007). Over the next five years, it spread 280 km along the Victoria coast, mostly to the east (Mayfield et al. 2011), into an area with 71 individual transferable quota (ITQ) holders in the well-regulated abalone dive fishery. The AVG outbreak was devastating, leading regulators to reduce the total allowable catch from 296 mt to 16 mt—a 280-mt drop valued at approximately US$10 million (Mayfield et al. 2011). ITQ licenses—the price of which is the best estimate of the net present value of a fishery—that were trading for US$5–6 million before the epizootic lost almost all their value (Conrad & Rondeau 2014). To help prevent another AVG outbreak, regulators imposed greater management restrictions on abalone aquaculture elsewhere in Australia (Jones & Fletcher 2012).

The spread of AVG illustrates the close biological and economic interdependence of wild fisheries and aquaculture, where farm decisions lead to risks and damage in the wild and vice versa. In practical terms, this means that to make efficient economic use of the resource, aquaculture management must account not only for the farm-level costs and benefits of prevention and remedial actions but also for the benefits of those actions for wild fisheries. If a disease in the wild responds to host density, then setting efficient catch levels also requires accounting for the impact of different wild stock levels on disease prevalence and the risks to farms. Here, and elsewhere, rapid responses to disease outbreaks can be critical. Recognition that a new disease is spreading leaves some time to modify harvesting policies, salvage the still-healthy stocks, and perhaps halt the spread of the disease. Under simplified scenarios, the optimal response to the impending arrival of a disease can be a drastic increase in the allowable catch (Conrad & Rondeau 2014). Yet even in well-managed fisheries, such as the Australian abalone fishery, it can be difficult to shift management policies in response to impending doom.

Successful management of wild and farmed abalone will require resolving several unanswered questions. For example, how long does WS-RLO survive in seawater? What is the biological relevance of quantitative polymerase chain reaction (qPCR) data on pathogen DNA abundance for abalone? What is the relative risk of infection for an abalone throughout its range? Is an abalone more at risk near a wild or farmed population or near a developed area of coastline? Will the phage infecting WS-RLO be an efficient "natural treatment" for withering syndrome? Will AVG reemerge? What conditions are needed for the emergence of AVG? What is the rate of evolution of abalone pathogens, and how does their evolution impact host-pathogen relationships? The sources of WS-RLO and AVG are still unknown, and understanding their origin will help elucidate disease introduction mechanisms.

White Spot Disease in Shrimp

Penaeid shrimp are the world's most valuable seafood. By the 1980s, declining wild fisheries could not keep up with the increased demand from wealthy countries, leading to increased aquaculture production, particularly in Thailand and Ecuador (Flegel et al. 2008, Lightner 2011). In 2011, farms produced 3.5 mmt of shrimp worth US$16 billion, and fisheries captured 3 mmt of wild

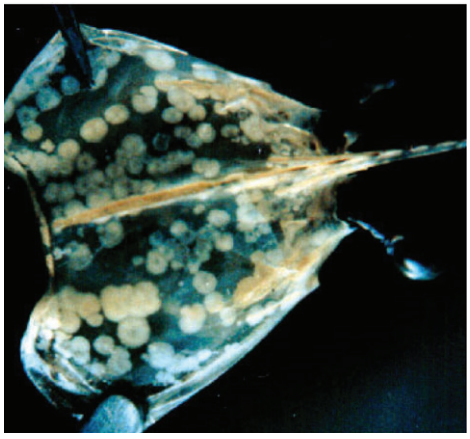

Figure 4

Carapace of a prawn with white spot disease. Photograph courtesy of D.V. Lightner.

shrimp (FAO 2012). Shrimp fisheries use trawlers, whereas penaeid shrimp aquaculture requires inexpensive coastal land. These contrasting economic models mean that developed nations tend to fish (e.g., along the Gulf Coast of the United States), whereas developing countries (especially in Asia and Latin America) do more shrimp farming. Shrimp farms have been hit by several infectious diseases, including white spot syndrome, yellow-head virus, hepatopancreatic parvovirus, monodon baculovirus, Taura syndrome virus, infectious hypodermal and hematopoietic virus (Flegel 2006), and, more recently, infectious myonecrosis and early mortality syndrome (D.V. Lightner, personal communication).

The agent of white spot syndrome is *Whispovirus*, a new genus of double-stranded DNA viruses in the family Nimaviridae (Sánchez-Mártinez et al. 2007). This directly transmitted virus infects all decapods tested to date, including crabs, crayfish, lobsters, and shrimp (Lightner 2011). Penaeid shrimp captured for broodstock are the main source of infections on farms, where the virus then spreads under high culture density (Lotz & Soto 2002) and warm temperatures (Jiravanichpaisal et al. 2004). During an outbreak on a commercial farm, white spot syndrome virus (WSSV) (**Figure 4**) can kill all shrimp within a week (Lightner 2011).

White spot syndrome outbreaks began in 1992 in Southeast Asia, where they devastated a thriving industry (Flegel 2006). In the 1990s, a shrimp pond could be farmed for only approximately seven years before disease outbreaks made the pond unprofitable, after which it was often abandoned and new ponds dug from mangrove forests, perpetuating a cycle of coastal environmental damage and economic loss (Dierberg & Kiattisimkul 1996). Losses from the 1992 and 1993 outbreaks throughout Asia were valued at ~US$6 billion (Lightner 2011). In 1999, the virus had catastrophic impacts to the farming of *Litopenaeus vannamei* and *Litopenaeus stylirostris* in the Americas (Lightner 2011). Ecuador declared a state of emergency when production plummeted by 65%, representing almost US$1 billion in lost exports (McClennon 2004). Starting in 2002, disease outbreaks in Iran reduced exports by two-thirds (Salehi 2010). During the worst years, the disease claimed 40% of global shrimp production.

The outbreaks in the 1990s and early 2000s changed shrimp farm management (Lightner 2011). In particular, using closed systems and culturing shrimp larvae from resistant stocks protected against disease. Furthermore, farms manage shrimp virus diseases by culturing domesticated specific-pathogen-free or specific-pathogen-resistant (SPR) stocks, reducing stress,

and emphasizing biosecurity (Lightner 2011). As a result of these biosecurity practices, white spot syndrome is a far smaller problem now.

It might appear that exports of WSSV into the wild would have environmental impacts on wild crustaceans. After all, the virus enters the ocean through farm effluent and shrimp escapes, and it infects many crustacean species. Interestingly, there are few reports of economic effects caused by viral release from aquaculture. One reason for this might be that the virus kills stressed shrimp in farm conditions, which do not often apply to wild shrimp. The most compelling evidence for an economic impact of viral export from shrimp farms comes from another agent, infectious hypodermal and hematopoietic virus: After aquaculture presumably introduced it in 1987, this virus infected 60–100% of the wild *Penaeus stylirostris* in the Gulf of Mexico and affected the fishery for a decade (Morales-Covarrubias et al. 1999). However, instead of being a net negative externality for shrimp fisheries, it seems possible that, by impairing aquaculture, shrimp viruses reduce the extent to which shrimp farms compete with wild fisheries, shifting the balance of global trade.

This example illustrates another interdependence between fishing and aquaculture. If consumers consider products from two sources (e.g., fished or farmed) to be equivalent, then disease outbreaks that reduce the supply from one source can benefit the other source because of an increase in the market price. Therefore, limiting the economic analysis of marine diseases (or of any other economic problem, for that matter) to the sale value of products can be misleading. The true value of seafood is not the price at which it is sold but rather the maximum amount that consumers will pay minus the delivery cost (including user costs in wild fisheries). An overall drop in the farmed shrimp supply that drives up the price benefits capture fisheries and penalizes consumers by an identical amount. The real economic losses come from the diseased shrimp that are neither produced nor eaten, not the overall change in the value of shrimp sold.

Because there are no treatments for white spot syndrome, the focus of current research is on prevention through clean aquaculture protocols, new diagnostic methods, and breeding SPR strains. Diagnostics research focuses on sensitive multiplex tests to detect multiple viral pathogens such as WSSV, yellow-head virus, and a shrimp densovirus (Panichareon et al. 2011). The development of SPR strains amounts to breeding the survivors of experimental outbreaks. But these survivors are also locally adapted to particular environmental settings, and most have been subject to multiple diseases (i.e., WSSV, Taura syndrome virus, and infectious hypodermal and hematopoietic virus), making it hard to focus selection on WSSV (Cuéllar-Anjel et al. 2012). Furthermore, it is important to distinguish whether SPR strains are tolerant or resistant because tolerant strains can act as carriers (Cuéllar-Anjel et al. 2012). Nonetheless, there are WSSV-resistant stocks of *Fenneropenaeus chinensis* (Zhiqin et al. 2005), *Penaeus monodon* (Dutta et al. 2013), and *Litopenaeus vannamei* (Huang et al. 2011).

Betanodaviruses in Groupers

Tropical groupers (*Epinephelus* spp.) and sea bass (*Dicentrarchus* spp.) support valuable localized sport, commercial, and artisanal fisheries (Seng 1998). Their high price (particularly on the live market) combined with declining wild stocks has led to an increase in net-pen culture. To stock the pens, young groupers are either caught from nearby coral reefs or raised in a hatchery.

Farmed and wild groupers are susceptible to diseases caused by generalist betanodaviruses that probably have both vertical and horizontal transmission (Munday et al. 2002). Viruses from farmed groupers have complex genetics that are similar in viruses from Asia and Europe (Kara et al. 2014) and in several reassortant strains (Panzarin et al. 2012). Reassortment is highest in viral isolates

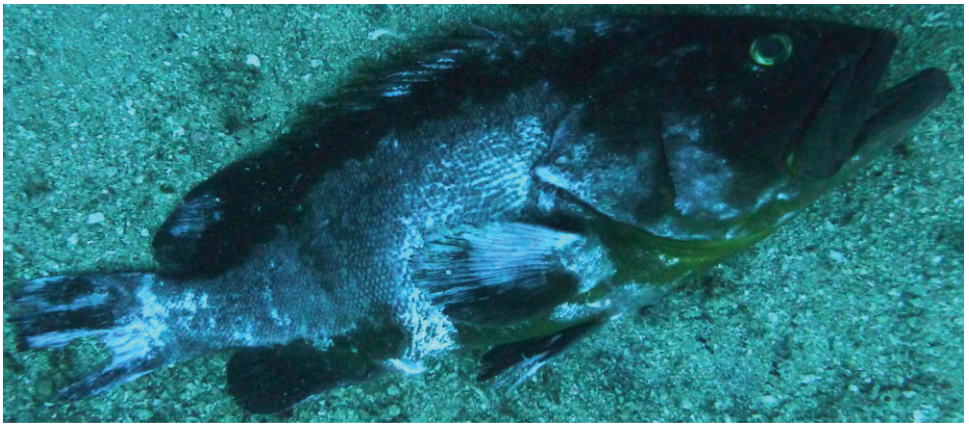

Figure 5

A living but moribund endangered dusky grouper in Italy infected with betanodavirus. The grouper has lost control of its swimming, causing it to crash into the bottom, which has led to severe trauma in the skin and fins. Photograph reproduced from Vendramin et al. (2013).

from farmed fish, suggesting that farms create environments for viral evolution (Panzarin et al. 2012).

Most infected adult fish are resistant to these viruses but sometimes show bizarre disease symptoms (**Figure 5**). For instance, in the Gulf of Annaba, Algeria, baffled divers saw several groupers with opaque eyes, skin lesions, unbalanced swimming, and a hyperinflated swim bladder, with some fish floating on the surface; these fish, which later stranded on a nearby beach, were infected with betanodavirus (Kara et al. 2014).

The increasing number of reports of simultaneous but distant die-offs has led to the hypothesis that death in infected adult fish results from the stress of spawning combined with warm temperature events (Kara et al. 2014). Optimal temperatures for viral growth vary by strain owing to local adaptation (Iwamoto et al. 2005), and the movement of locally adapted and benign strains into novel culture settings could lead to unusual pathologies. Betanodaviruses are now a major threat to aquaculture in the Mediterranean (Vendramin et al. 2013). Here, the main economic impact is their constraint on hatchery production (Le Breton et al. 1997) because fry and juveniles cannot tolerate infection (Breuil et al. 1991).

Viral exchange between farms and wild fish seems probable and was proposed to explain a mass mortality of wild dusky grouper 15–20 km from sea bass farms (Vendramin et al. 2013). If this hypothesis is correct, then grouper farming could impede the recovery of this endangered species (Marino & Azzuro 2001). As is the case for most marine diseases, whether farms are net virus exporters or simply victims of exposure from wild fishes is difficult to determine (Vendramin et al. 2013) but worthy of investigation.

Infectious Hematopoietic Necrosis Virus, *Kudoa*, and Sea Lice in Salmon

After years of decline resulting from river damming and overfishing, wild Pacific salmon stocks have stabilized, with landings totaling approximately 1 mmt per year. By contrast, the production of farmed Atlantic salmon (*Salmo salar*) has increased. In 2011, salmon farming delivered 1.7 mmt, with a farm-gate value of US$9.7 billion (FAO 2012).

Infectious hematopoietic necrosis virus. Infectious hematopoietic necrosis virus (IHNv) is a rhabdovirus endemic in the Pacific Northwest. This virus has efficient transmission in water, with acute disease occurring within days of exposure for susceptible species. In both fresh water (for wild and hatchery fish) and salt water (for farmed Atlantic salmon), young infected fish often die from hemorrhaging and septicemia. The disease severity depends on the pathogen genotype, host species, and stock, with the most susceptible being Atlantic salmon (Traxler et al. 1993). Sea-caged Atlantic salmon suffered several IHNv outbreaks in British Columbia and Washington State in 1992–1996 (St-Hilaire et al. 2002), 2001–2003 (Saksida 2006), and 2012, with mortalities ranging from 18% to 78%. Chinook salmon are less susceptible to the disease and have been identified as virus carriers when held near Atlantic salmon in net pens (St-Hilaire et al. 2001). The virus has also become established in Japan, Taiwan, and Europe, possibly from moving infected fish and fish eggs (Hostnik et al. 2002). Sockeye is the most susceptible of the Pacific salmon species, with a 48–85% death rate (Meyers et al. 2003).

IHNv has cost the salmon farming industry in several ways. In addition to lost production, carcass disposal has been an economic burden to farms. Many small farms went bankrupt in British Columbia and were absorbed into large, foreign-owned companies. During the 2001–2003 outbreaks on 36 farms, culling was imposed to halt the spread, and the cumulative mortality on these farms reached 58%; more than 12 million Atlantic salmon, valued in the tens of millions of dollars, were killed or culled (Saksida 2006). After this outbreak, most farms installed disinfection systems at costs of up to US$4 million (B. Milligan & G. Murphy, personal communication). To avoid vertical transmission, all farms also now screen broodstock for IHNv at an annual cost of more than US$200,000. Some farms reduce risk by vaccinating their Atlantic salmon against IHNv at an annual cost of US$4 million (D. Morrison, personal communication). Companies in areas with low infection risk chose not to vaccinate their fish after an economic assessment predicted that the high vaccination cost would outweigh the expected benefits. However, in 2012, an outbreak of an IHNv strain identical to an isolate from wild fish spread among unvaccinated farms (K. Garver, personal communication). The decision by some farms to forgo vaccination is a classic market failure in economics (Stiglitz 1988): Immunization at one farm is a public good that, when ignored, leads to lower-than-optimal protection against disease among farms. Achieving optimal protection might require public policies that impose vaccination or subsidize its implementation.

There has been one documented IHNv outbreak among wild sockeye salmon in the marine environment (Traxler et al. 1997), which is the most valuable wild salmon fishery (FAO 2012), with an annual Alaskan harvest of more than 100,000 mt (Alsk. Dep. Fish Game 2014). Although this outbreak is worrisome, the main cost to the sockeye fishery is the extra expense that IHNv management and prevention impose on stock enhancement. For example, using UV sterilization and modular facilities to help prevent outbreaks cost one hatchery US$300,000 (C. Cherry, personal communication). Hatcheries bear this cost because the alternative is to destroy their stock if they detect IHNv.

***Kudoa*.** *Kudoa* is a common genus in the phylum Myxozoa that infects marine fishes. Based on our knowledge of other myxozoans, *Kudoa thyrsites* probably requires an annelid host in the marine environment to complete its life cycle. Fish are likely infected by direct exposure or ingestion of a free-floating actinosporean stage in the water. Most *Kudoa* species do not kill their hosts, but several species infect commercially important fishes like yellowtail, mahimahi, Atlantic mackerel, and Pacific hake, causing visible pseudocysts throughout the muscle, postharvest myoliquefaction (**Figure 6**), or both (Moran et al. 1999). These infections undermine the fishes' value and, in particular, impede some fisheries for Pacific hake, the most abundant commercial fish species off the US West Coast (Kabata & Whitaker 1981).

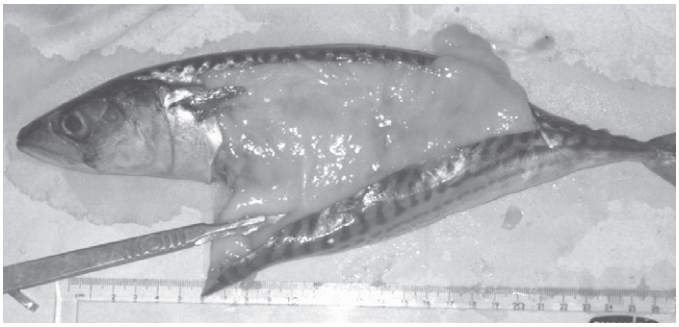

Figure 6

A mackerel with myoliquefaction caused by infection with *Kudoa thyrsites.* Photograph courtesy of C. Whipps.

K. thyrsites was first documented in pen-reared Atlantic salmon industry in British Columbia in the early 1990s (Whitaker & Kent 1991), and by 2000 it was costing the industries more than US$7 million per year, based on a 0.5% claim rate charged back to the producers for spoiled fish (Dodd 2001). The infection in farmed salmon continues to be a major economic problem, and one large company's 2011 annual report estimated that total costs related to discards and claims as a result of soft flesh caused by *K. thyrsites* amounted to NOK 68 million, and that the average price in Canada was 11% lower than in 2010 owing to the high presence of *Kudoa* (Mar. Harvest 2011). An even bigger problem for the industry is how consumers view farmed salmon quality. A few mushy *Kudoa*-infected fish can have a long-lasting impression, driving down demand and price.

Sea lice. Small caligoid copepod crustaceans in the genera *Caligus* and *Lepeophtheirus* are called sea lice. Free-living nauplius larvae hatch from eggs of parasitic females. The nauplius molts into a free-living copepodid that does not feed until it enters the chalmus stage and uses its second antenna to attach to a fish. After feeding and molting, the chalmus transforms first into a parasitic preadult and then into an adult that can move about on the fish host. Sea lice feed on fish skin, mucus, and blood, causing lesions. The resulting impact on infected fish varies by parasite species, parasite life stage, fish species, fish age, and fish health, but disease severity increases with sea lice intensity. At low intensities, infected fish can keep up with their energetic demands by feeding more, but this increases feed costs for penned salmon by 5% (Sinnott 1998). At high intensities, as is often seen on farms, damage to the epithelium and blood loss leads to stress, secondary infections, reduced growth, diminished flesh quality, and, in extreme cases, death (Pike & Wadsworth 2000).

Salmon aquaculture operations from Norway to Chile, Canada, the United Kingdom, and Ireland manage for sea lice. Best practices include separating age classes, all-in–all-out production cycles with fallowing, and regular sea lice monitoring (Saksida et al. 2011). Farms also purchase therapeutants to control sea lice (Revie et al. 2002), which can raise production costs by 5–20% (Rae 2002). Overall, sea lice increase farmed salmon costs by US$0.15–0.30/kg, which translates to a global annual cost exceeding US$400 million (Costello 2009).

The potential export of sea lice to wild stocks is controversial and leads to negative views of salmon farming. Some analyses have suggested that the increased sea lice intensity on smolts that migrate near fish farms is not great enough to reduce survival (Jackson et al. 2013). However, models have predicted that sea lice from salmon farms could extirpate wild stocks in the Broughton Archipelago, British Columbia (Krkošek et al. 2007). This strong claim was contested by the salmon farming industry and has not been borne out over time. A subsequent analysis of the

links between farms and sea lice on wild fish suggested that lice from farms do not reduce wild salmon production, that salmon might benefit from eating sea lice, and that aquaculture does not need to protect wild stocks from sea lice (Marty et al. 2010). However, an independent analysis suggested that salmon production was lower near salmon farms (Krkošek et al. 2011). The actual effect is important because a model of wild pink and chum salmon populations in the Broughton Archipelago predicted that a lice-induced mortality between 20% and 30% could lead to sharp stock declines unless countered by reduced harvest rates or the closure of the commercial fishery (Liu et al. 2011).

Although salmon farms have a private interest in controlling sea lice in net pens, little incentive exists for farms to reduce hypothetical (and difficult-to-prove) impacts on wild stocks. Regulators have therefore established sea lice intensity thresholds above which fish must be treated (Saksida et al. 2011), and Norway has set aside farming-free fjords and set management goals to reduce sea lice intensity on wild fish to 10 lice. Meeting this goal will be difficult because there are far more farmed salmon than wild salmon in Norway (Finstad & Bjørn 2011).

A key research need for sea lice (and other marine diseases) is to collect baseline data on pathogen prevalence and distributions in wild species before establishing an aquaculture enterprise (Saksida et al. 2014). This would help regulators assess whether impacts to wild stocks are within natural variation or are increased by spillover from aquaculture. Other useful data for understanding the dynamics and risks of transmission of various marine diseases include the survival time of an infectious agent in the water, its host range, and other required hosts in the life cycle.

CONCLUSIONS

Marine diseases are common in the ocean and can reduce seafood's economic value by decreasing meat quality, increasing the marginal costs of harvest and processing, and diminishing biological productivity. Fishing can inadvertently reduce infectious disease prevalence in fished stocks. By contrast, farming favors infectious diseases and therefore requires investment in disease management (Sinderman 1984). Because disease management has costs that weigh against profits, the economic effects of infectious diseases are more obvious to farmers than to fishers.

Aquaculturists try to control transfers and introductions. They also reduce stress-activated disease by maintaining water quality and limiting crowding. For finfish, prophylactic therapeutants and immunization can reduce viral and bacterial diseases. If, despite these investments in prevention, a disease emerges, the farm should first diagnose the problem and then, if it makes economic sense, hope to treat it. In worst-case scenarios, regulatory actions might require that farms cull stock to prevent spread to other farms or to the wild.

The extent to which aquaculture exports infectious agents that in turn infect wild stock is controversial, drawing critiques from conservation biologists and attention from regulators. Water flow connects farmed species to wild infectious agents, which can then build up under dense farming conditions, escape from farms, and transmit back to wild hosts. Documenting the extent to which aquaculture magnifies wild infectious agents has proven difficult given the sparse baseline information on their background prevalence, but impacts from amplified native diseases should often be local. Local effects are of greatest concern in areas that are critical habitats for susceptible endangered species. Clearer are the negative impacts of infectious agents accidently moved around by aquaculture, such as when new infectious agents arrive with imported stock. The imported stock might have coevolved tolerance to its infectious agents, but the release of a new infectious agent into the wild might lead to new disease in naive wild stocks. Aquaculture's role in facilitating the evolution of new infectious strains is more speculative. The argument is that farms present a novel evolutionary environment for infectious agents (e.g., IHNv), which might lead to greater

selection for virulence, crossing among parasite strains, or adaptation to farm conditions that leads to unusual pathologies in the wild. Such impacts are currently hypothetical, but new molecular tools will make it easier to identify novel genotypes of infectious agents associated with aquaculture.

Scientific studies could fill key knowledge gaps and mitigate the economic consequences of marine diseases in several ways. Given the continual onslaught of existing and new infectious agents, the aquaculture industry needs continued research into diagnostics, treatment, and epizootiology. More focus on economics specifically could help to identify best practices and strategies for maximizing the economic value of seafood in the face of disease pressures. However, a quantitative understanding of host–infectious agent dynamics is essential to the economic assessment of these systems. Perhaps because of the complexity of the ocean ecosystem, reliable population models are rare. There is therefore a substantial need for basic research into the ecology and evolution of marine diseases.

Resolving the controversial issues surrounding the export of marine diseases from aquaculture will require understanding the background rates of disease in potentially affected wild species as well as testing hypotheses about environmental impacts. Finally, marine diseases are part of a dynamic human-natural system in which there are apt to be important sociological, behavioral, legal, and political issues that interact with the biology we review here. Given the staggering economic cost of marine diseases, research is a clear public good.

The most obvious economic impact of marine disease is the cost that aquaculture expends to manage infectious agents on farms. In addition, infectious diseases are moving targets that can rapidly destabilize markets, which might dissuade investment. Less obvious, and more controversial, is the export of infectious agents from aquaculture to wild stock, a negative externality that might affect wild fisheries and threaten biodiversity. However, the impact of infectious diseases on farms might also indirectly help wild fisheries that already struggle with declining catch per effort and competition with aquaculture. Regardless, consumers bear the brunt of marine diseases in the form of reduced availability and higher seafood prices.

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The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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Errata

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