Non-native Bacterial and Viral Pathogens in Ballast Water: Potential for Impacts to ESA-listed Species under NOAA's Jurisdiction

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Contents

Cite as: Cohen, A.N. 2010. *Non-native Bacterial and Viral Pathogens in Ballast Water: Potential for Impacts to ESA-listed Species under NOAA's Jurisdiction*. A report prepared for the National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Endangered Species Division, Silver Spring, MD. Center for Research on Aquatic Bioinvasions (CRAB), Richmond, CA.

Introduction

NOAA is investigating the potential impacts of ballast water discharges on listed species protected under the federal Endangered Species Act (ESA), in order to understand what regulatory measures are needed to protect listed species. To assist those investigations, this report reviews the current level of understanding of the potential for non-native bacteria or viruses that are pathogenic to ESA-listed species (including proposed and candidate species) that fall under NOAA's jurisdiction to be carried in ballast water and released into waters where the listed species occur, and to infect and impact those species. To prepare this report I reviewed the scientific literature, interviewed experts, and consulted other relevant information sources.

The review discusses the presence of relevant non-native bacterial or viral pathogens in source regions, the potential for such pathogens to be taken up in and transported in ballast water, the volumes of ballast water transferred between source regions and relevant U.S. waters, the capacity of pathogens to survive ballast transport and release, the potential for listed species to be exposed to these pathogens, and the potential impacts to listed species if exposed, insofar as these quantities, capacities or probabilities are known or can be reasonably estimated.

In this report, "non-native" refers to species or distinct strains that are not native to the waters that they are released into, and includes the release of non-U.S. pathogens, strains or genotypes of pathogens into U.S. waters as well as the transfer of such pathogens from one part of the U.S. into another part where they are not native. The review thus considers ballast water's potential role both in transferring pathogens into U.S. waters from outside the U.S., and in transferring pathogens into new regions of the U.S. from regions of the U.S. where they are present as either native or non-native pathogens.

ESA-listed species that fall under NOAA's jurisdiction

As of May 13, 2010, there were 136 species or subspecies listed as endangered, threatened, proposed or candidate species under the ESA that fall under NOAA's jurisdiction.[1] Nine of these—5 cetacean, 3 pinniped and one fish species—are not found in U.S. waters (Appendix 1) and are not considered further in this report.[2] The 127 taxa

[1] The listed species and population groups considered in this report are based on the endangered and threatened marine species listed on the NOAA Fisheries/Office of Protected Resources web page at http://www.nmfs.noaa.gov/pr/species/esa, accessed on 5/13/2010. Changes to NOAA's posted list that were made since that date have not been included.

[2] Species, subspecies or population groups that do not occur in U.S. waters cannot be directly affected by the transport of pathogens into U.S. waters in ballast water. However, indirect impacts may be possible. For example, a bacterium or virus introduced into southern California waters might then spread into Mexican waters in the Gulf of California where the endangered Vaquita porpoise (*Phocoena sinus*) and

that occur in U.S. waters consist of 11 cetacean, 5 pinniped, 6 sea turtle, 18 fish, 2 mollusk, 84 coral and 1 plant species or subspecies (Appendix 2).

Some of these are further subdivided into various types of population groups, designated as Distinct Population Segments (DPS), Evolutionary Significant Units (ESU), runs, populations or portions of ranges, with one or several such groups listed rather than the whole species or subspecies. In the remainder of this report I refer to entities listed as any of these various types of population groups, or listed as species or as subspecies, as "ESA species", to distinguish them from species in the taxonomic sense. Not included in these are the 33 entities that NOAA lists as Species of Concern, which are defined as species, subspecies or population groups about which NOAA has concerns regarding status and threats, but for which there is insufficient information to support listing under the ESA.

In all there are 153 listed ESA species that occur partially or wholly in U.S. waters (Appendix 2). These are classified into the four categories of listing status defined in Table 1. The breakdown by major taxonomic group and listing status is shown in Table 2. Eighty-four (55%) of the 153 listed ESA species are corals, including 82 candidate species listed in response to a petition submitted in October 2009. Forty-one fish form the second-largest group of species (27% of the total), with 28 of these consisting of different runs of five Pacific Coast salmonid species.

Table 1. Listing status definitions. In this table, "species" refers to a species, subspecies or population, and corresponds to the term "ESA species" as used in this report.

Status	Definition
Endangered	A species that is in danger of extinction throughout all or a significant portion of its range.
Threatened	A species that is likely to become endangered within the foreseeable future throughout all or a significant portion of its range.
Proposed	A species found to warrant listing as threatened or endangered and officially proposed as such. NMFS generally has one year to determine whether to list a proposed species as threatened or endangered.
Candidate	A species undergoing a status review following a determination in response to a petition or on NMFS' initiative that listing may be warranted, but which is not yet the subject of a proposed rule.

Totoaba fish (*Totoaba macdonaldi*) reside. Also, once an exotic organism becomes established in a U.S. port area, that port then becomes a potential source area for introducing the organism to other parts of the world via ballast water or by other transport mechanisms. Because these indirect pathways involve at least one additional step beyond the pathways affecting ESA-listed species found in U.S. waters, they are that much less likely, and are not specifically considered further in this report.

Table 2. ESA species under NOAA's jurisdiction that occur in U.S. waters, by taxonomic group and listing status. Source: NOAA 2010a.

Taxonomic Group	Endangered	Threatened	Proposed	Candidate	Total
Cetaceans	10	0	0	1	11
Pinnipeds	2	2	0	2	6
Turtles	5	3	0	0	8
Fish	9	24	4	4	41
Mollusks	2	0	0	0	2
Corals	0	2	0	82	84
Plants	0	1	0	0	1
Total	28	32	4	89	153

From their habitat descriptions, ESA species can be classified as occupying marine, marine and estuarine, or marine, estuarine and freshwater habitats (Table 3). None of the listed species under NMFS' jurisdiction that occur in U.S. waters are found only in estuarine or fresh waters; however, 108 (71%) are exclusively marine. Considered by habitat occurrence, all of the 153 ESA species occur in marine waters at some times or for some of their life stages, 45 (29% of the total) occur in estuarine waters at some times, and 36 (24% of the total) occur in fresh waters at some times (Table 4). These patterns of habitat occurrence will affect a species' exposure to pathogens released in ballast water which, depending on shipping routes and ballast management practices, may be variously derived from or released into marine, estuarine or fresh waters.

Table 3. ESA species under NOAA's jurisdiction that occur in U.S. waters, by taxonomic group and habitat classification. Sources: CBD 2009; NOAA 2010a,b.

Taxonomic Group	Marine	Marine & Estuarine	Marine, Estuarine & Freshwater	Total
Cetaceans	10	0	1	11
Pinnipeds	2	2	0	6
Turtles	2	6	0	8
Fish	4	2	35	41
Mollusks	2	0	0	2
Corals	84	0	0	84
Plants	0	1	0	1
Total	108	9	36	153

Table 4. ESA species under NOAA's jurisdiction that occur in U.S. waters, by taxonomic group and habitat occurrence. Sources: CBD 2009; NOAA 2010a,b.

Taxonomic Group	Marine	Estuarine	Freshwater
Cetaceans	11	1	1
Pinnipeds	6	0	0
Turtles	8	6	0
Fish	41	37	35
Mollusks	2	0	0
Corals	84	0	0
Plants	1	1	0
Total	153	45	36

ESA species can also be classified by the coastal region of the U.S. in which they are found (Table 5, Appendix 3). Cetacean and sea turtle ESA species are found in all regions. Pinniped ESA species occur mainly in Alaska and on the West Coast, with one species in Hawaii. Most ESA fish species (33 (80%) of the ESA fish species) are found on the West Coast (primarily various runs of salmonids and some rockfish species), with a few ESA fish species occurring in Alaska, on the East Coast and in the Gulf of Mexico, but none in the more tropical waters of the Caribbean, Hawaii and the Pacific Islands. The coral ESA species mainly occur in the Pacific Islands (71 species (85%)), with a smaller number (7-10 species per region) in Hawaii and in the Caribbean, Gulf of Mexico and East Coast regions; none occur on the West Coast or, of course, in Alaska. The two ESA mollusk species (both abalone) are found only on the West Coast, and the sole plant species (a seagrass) is found only in Florida on the East Coast. These different regions are exposed to different volumes of ballast water discharge from different source regions, which affects the risk to the different species groups of encountering ballast water-introduced pathogens (see the section on **Ballast water pathways** below).

Table 5. ESA species under NOAA's jurisdiction that occur in U.S. waters, by taxonomic group and region. The Caribbean region includes Puerto Rico, the U.S. Virgin Islands and Navassa Island; the Hawaii region includes the Hawaiian Islands and Midway Island: the Pacific Islands region includes Guam, the Northern Mariana Islands, American Samoa, Johnston Atoll, Wake, Howland, Baker and Jarvis islands, Palmyra Atoll and Kingman Reef. Sources: CBD 2009; NOAA 2010a,b.

Taxonomic Group	East Coast	Gulf of Mexico	Caribbean	Alaska	West Coast	Hawaii	Pacific Islands
Cetaceans	6	4	4	7	7	5	4
Pinnipeds	0	0	0	4	2	1	0
Turtles	5	5	6	2	6	5	4
Fish	5	3	0	2	33	0	0
Mollusks	0	0	0	0	2	0	0
Corals	9	7	9	0	0	10	71
Plants	1	0	0	0	0	0	0
Total	26	19	19	15	50	21	79

Impacts of pathogens on naive hosts

Parasites or pathogens often have a greater impact on populations that lack prior exposure to them (referred to as naive host populations). This is a well-known effect in human epidemic disease (*e.g.* Tauxe *et al.* 1995), and is often cited as a dominant factor in the decimation of aboriginal populations soon after European contact and the introduction of novel diseases (Crosby 1972, 1986; Cook 1978; Cronon 1983). There are many documented examples among plants and animals, including marine mammals (Kennedy 1998; Australia DAFF 2000), fish (Hoffman 1970; Bauer & Hoffman 1976; Combes & Le Brun 1990; Kennedy 1994; Nielsen 1999; Mo 1994; Ashworth 1994; Walker & Winton 2010) and mollusks (Culver & Kuris 1999; Burreson *et al.* 2000).

The enhanced pathogenic impact generally occurs because the naive host lacks defenses, such as an effective immunological response, although there may also be behavioral or other adaptations in the host that reduce the incidence or severity pathogenic impact where there has been a long association with a pathogen. In some cases, there may also be a greater impact on naive hosts because some pathogens evolve to become less damaging to their host populations over time, which may enhance the potential for effective transmission and long-term persistence (Fenner & Myers 1977; Anderson & May 1982).

The implication for this review is that bacteria or viruses introduced into a new region could have substantial impact on naive hosts encountered there, even if they have little or no pathogenic effect on related host organisms in their native regions.

Antibiotic resistance

Distinct from concerns about the known pathogenicity of imported bacteria, or the introduction of novel genotypes that may prove to be pathogenic to naive hosts, is the potential for ballast water to introduce antibiotic-resistant forms. Some recent studies have documented the common presence of antibiotic-resistant bacteria in ships' ballast tanks (Thomson *et al.* 2003; Goodrich 2006; Thomson 2009). Bacteria can carry antibiotic-resistant genes either on chromosomes, or on heritable, non-chromosomal bodies called plasmids. In the latter case two bacteria considered to have the same genotype (*i.e.* identical chromosomal DNA) may nonetheless have differing resistance to antibiotics. Plasmids can also be transferred between bacteria by a process called horizontal gene transfer, with some plasmids (conjugative plasmids) being more likely to transfer than others. Thus, even if bacteria introduced in ballast water have the same genotype as bacteria that were already present in the receiving waters, they could still pose a risk if the introduced bacteria have greater antibiotic resistance, Furthermore, the mixing of bacteria from the world's harbors via ballast transport increases the risk that bacteria with plasmids promoting antibiotic resistance will come into contact with and transfer these plasmids to pathogenic bacteria (F. Dobbs, pers. comm.). In a review of emerging cetacean diseases, Van Bressem *et al.* (2008) have expressed concern about "the world-wide dissemination of...antibiotics-resistant marine bacteria through water ballast."

Transport of bacteria, viruses and other pathogenic microbes in ballast water

Although most of the studies on organisms transported in ballast water have focused on multi-cellular organisms (primarily zooplankton) or on phytoplankton (primarily diatoms and dinoflagellates), there is nonetheless good documentation of a large number and diversity of other microbial organisms, including bacteria and viruses, being collected from ballast tanks at the ends of voyages (*e.g.* see Table 1 in Drake *et al.* 2001; and Drake *et al.* 2007). For example, Galil & Hulsmann (1997; see also Hulsmann & Galil 2001) identified 198 living protozoan species in 82 heterotrophic genera collected from water and sediments in the ballast tanks of 17 cargo vessels arriving at Israeli ports in 1996, including flagellates (Euglenozoa, Cryptomonadea, Prymnesiomonadea, Choanoflagellata, Heterokonta, Dinoflagellata and others), ciliates (Karyorelictida, Spirotrichea, Litostomatea, Phyllopharyngea, Oligohymenphorea) and pseudopodial organisms (Gymnamoebea, Heterolobosea, Filosea, Granuloreticulosea, Heliozoa, Labyrinthulea and others). Most nanoflagellates, heliozoans and other organisms smaller than 5-10 µm were not identified.

The transport of toxic dinoflagellates in ballast tanks has received particular attention. Approximately 60 marine dinoflagellates are known to produce substances that are toxic and sometimes fatal to humans or animals (Doblin & Dobbs 2006). Some of these form cysts that can remain dormant and viable for months or even years, and such cysts have been found in the sediments in ballast tanks at up to 22,500 per gram of wet

sediment (Hallegraeff & Bolch 1992; Hamer *et al.* 2000, 2001). There is good evidence that some toxic dinoflagellates have been introduced into new regions of the world in ballast discharges (Hallegraeff *et al.* 1995; McMinn *et al.* 1997; Hallegraeff 1998; Lilly *et al.* 2001; Dobbs & Rogerson 2005; Drake *et al.* 2007). *Pfiesteria piscicida* and *P. shumwayae*, dinoflagellates that have been implicated in numerous fish kills, were found in one of four vessels sampled on arrival in Chesapeake Bay or the Great Lakes (Doblin *et al.* 2004; Drake *et al.* 2005) and in about 10% of the tanks sampled for residual ballast water on NOBOB vessels (vessels that declare "no ballast on board" but that typically have relatively small volumes of ballast water remaining in their tanks) arriving at ports in the Great Lakes in 2001-2002 (Johengen *et al.* 2005). Aguirre-Macedo *et al.* (2008) found bacteria that are known coral pathogens in ballast discharges near a coral reef in the Gulf of Mexico.

A series of studies carried out on vessels arriving in Chesapeake Bay and the Great Lakes have enumerated the number of bacteria and virus-like particles (VLPs) in ballast water, in residual water at the bottoms of ballast tanks after they are emptied, in the pore water of sediments collected from the bottoms of ballast tanks after they are emptied, and in biofilms (organic matrices that form on submerged surfaces) on the sides of ballast tanks (Table 6). These studies have been carried out in bulk carriers and coal carriers (colliers) arriving in Chesapeake Bay, and in NOBOB vessels arriving at ports in the Great Lakes. They include vessels arriving from both foreign and domestic ports, and ballast tanks that both had and had not undergone ballast water exchange at sea.

These studies reported mean concentrations in ballast water of 8×10^8 to 3×10^9 bacteria/liter and 7×10^9 to 3×10^{11} VLPs/liter (Table 6). These are reasonably comparable to normal concentrations in Chesapeake Bay of 2-12 $\times 10^9$ bacteria/liter and 3-140 $\times 10^9$ VLPs/liter, a range of concentrations in the summer in Lake Erie of 1.5-5.5 $\times 10^{11}$ VLPs/liter, typical concentrations in lakes and oceans of 10^9 bacteria/liter and 10^9–10^{12} VLPs/liter, and concentrations of bacteria and viruses in coastal waters of 10^6–10^{11}/liter (Wommack & Colwell 2000; Drake *et al.* 2001; Dobbs & Rogerson 2005; Wilhelm *et al.* 2006). The concentrations of bacteria and VLPs in residual water in recently emptied ballast tanks were similar to the concentrations reported for filled ballast tanks (Drake *et al.* 2007; Table 6), but the concentrations in residual water in NOBOB vessels were generally orders of magnitude higher, especially for bacteria (Johengen *et al.* 2005; Table 6). Sediment pore water in recently emptied ballast tanks generally contained 80-320 times higher concentrations of bacteria and 4-160 times higher concentrations of VLPs than was found in ballast water, depending on the data sets compared (Drake *et al.* 2007; Table 6). Ballast tank biofilms generally contained 2-8 times higher concentration of bacteria and 2-90 times higher concentration of VLPs than found in ballast water (Drake *et al.* 2005, 2007; Table 6). The ranges reported for pore water in NOBOB vessels suggest similarly elevated concentrations (compared to ballast water) for VLPs but perhaps not for bacteria (Johengen *et al.* 2005; Table 6). Ballast tank biofilms generally contained 2-8 times higher concentrations of bacteria and 2-90 times higher concentrations of VLPs than found in ballast water (Drake *et al.* 2005, 2007; Table 6).

Table 6. Concentrations of bacteria and viruses in ballast tanks. n = number of tanks sampled.

Types of Voyage and Vessel Samples	Bacteria		VLPs	
	n	#/liter	n	#/liter
In Ballast Water				
Bulk carriers to Chesapeake Bay (Ruiz *et al.* 2000)	11	8.3×10^8	7	7.4×10^9
Bulk carriers from foreign ports to Chesapeake Bay in 1996-2000 (Drake *et al.* 2001)	18	1.8×10^9	12	1.4×10^{10}
Bulk carriers from foreign ports to Chesapeake Bay, surface samples only (Drake *et al.* 2005)	18	1.7×10^9	12	1.3×10^{10}
Massachusetts to Chesapeake Bay in 2002 (Drake *et al.* 2005)	4	3.2×10^9	4	3×10^{11}
Unexchanged ballast water (MEPC 2003)	11	8.3×10^8	7	7.4×10^9
Coal carriers or foreign ports to Chesapeake Bay in 1996-2001 (Drake *et al.* 2007)	53	8×10^8	31	1.4×10^{10}
One cargo vessel sampled at 5 Great Lakes' ports in July 2003 (Wilhelm *et al.* 2006)	5	1.4×10^9	5	3.3×10^{11}
In Residual Ballast Tank Water				
NOBOB voyages to ports in the Great Lakes in 2000-2002 (Johengen *et al.* 2005)	75	10^8–10^{12}	75	10^{10}–10^{12}
Bulk carriers from foreign or domestic ports to Chesapeake Bay in 2003 (Drake *et al.* 2007)	13	4.4×10^8	13	6.2×10^{10}
In Pore Water of Ballast Tank Sediments				
NOBOB voyages to ports in the Great Lakes in 2000-2002 (Johengen *et al.* 2005)	73	10^7–10^{11}	73	10^{10}–10^{14}
Bulk carriers from foreign or domestic ports to Chesapeake Bay in 2003 (Drake *et al.* 2007)	12	2.6×10^{10}	12	1.2×10^{12}
In Ballast Tank Biofilms				
Bulk and coal carriers from foreign or domestic ports to Chesapeake Bay or Great Lakes in 2002-03 (Drake *et al.* 2005, 2007)	3	6.6×10^9	5	6.3×10^{11}

In general, these studies revealed a wide range in variation in bacterial and VLP concentrations in ballast water, and did not reveal significant differences in concentrations between exchanged and unexchanged tanks (although exchange presumably alters the composition of the organisms; Drake *et al.* 2002). Bacterial concentrations declined during voyages in tanks that did not undergo mid-ocean exchanges, and VLP concentrations declined in tanks with or without exchange (Drake *et al.* 2002, 2007). Ballast tank biofilms generally contained 2-8 times higher concentration of bacteria and 2-90 times higher concentration of VLPs than found in ballast water (Drake *et al.* 2005, 2007; Table 6). Although bacterial and viral concentrations were often found to be considerably greater in residual water, pore water or biofilms than in ballast water, due to the much larger volume of ballast water carried by bulk carriers travelling in ballast the estimated total number of bacteria and viruses carried in the ballast water on a vessel is much greater, by ≈1 to >3 orders of magnitude, than is carried in residual or pore water or biofilms (Drake *et al.* 2005, 2007).

This is presumably also true for most or all other vessel types when travelling in ballast, and these differences are probably even greater for ballast discharges, since in a typical discharge much of the sediment and most of the biofilm probably remains in the ballast tank (Drake *et al.* 2007).

Theoretically, ballast tanks could serve as incubators resulting in increases in certain microbes over the course of a voyage: in darkened tanks phytoplankton should cease photosynthesizing and die, in turn resulting in the starvation and death of zooplankton; the decomposition of these organisms could promote bacterial growth, leading to increased viral replication (Drake *et al.* 2002). On the other hand some bacteria have antiviral properties, so that increased bacteria growth could lead to declines in virus concentrations, while declines in bacteria could enhance viral survival (Girones *et al.* 1989). General declines in phytoplankton and zooplankton populations in ballast tanks have been observed during voyages (*e.g.* Gollasch *et al.* 2000), but overall increases in bacterial and viral concentrations have not (Drake *et al.* 2002). Nevertheless, the relationships are complex and it is possible that some species of bacteria or viruses may increase in ballast tanks over voyages even while overall numbers decline. In this regard it is worth considering that many proposed or recently adopted ballast water discharge standards would require removing or killing phytoplankton and zooplankton in ballast tanks at greater rates than they decline naturally due to cessation of photosynthesis, without requiring concomitant reductions in bacteria or viruses, including the discharge standards in the IMO Convention (a proposed international treaty, developed by the International Maritime Organization, which is awaiting ratification by enough member states to go into effect), standards adopted by some states under Section 401 of the Clean Water Act and/or state authority (Illinois, Indiana, Minnesota, New York (Interim Standard), Ohio, Pennsylvania (Interim Standard) and Wisconsin), and the standards proposed for near-term implementation by the U.S. Coast Guard (Notice of Proposed Rulemaking Phase I) (Alpert *et al.* 2010; Lee *et al.* 2010). A substantial increase in the number of dead phytoplankton and zooplankton sinking to the bottom of ballast tanks could enhance bacterial and viral populations.

Based on sampling data, shipping data and temperature tolerances, Drake *et al.* (2007) estimate that each year around 10^{20} bacteria and viruses are released in ballast discharges at the Port of Hampton Roads and survive in Chesapeake Bay. Although there is little information available on actual or possible bacterial or viral introductions in coastal waters (see discussion below), Ruiz *et al.* (2000) and Drake *et al.* (2001) concluded—based on the ballast tank concentrations, biological capacities (including asexual reproduction, high intrinsic growth rate, and dormant resting stages) and environmental tolerances of many bacteria and viruses—that invasions may nonetheless be relatively common, though unrecognized.

Bacteria or viruses could potentially be transported along with ballast water in a variety of media or modes, some of which have been mentioned. They may be carried in the ballast water itself, including residual water in nominally empty tanks, which may be fresh, brackish or salt; in sediments in the bottom of the tank, either in pore water or associated with sediment particles, in biofilms on the walls of the tanks; or carried in or

on other organisms. The fraction surviving the voyage will depend on the particular bacteria or virus, the specific mode of travel, the length of the voyage, the temperature of the water, and other factors. Appendix 4 shows some typical data on survival times and rates for some pathogenic fish viruses in few different media, illustrating some of these differences. Studies on the persistence in aquatic environments of human or animal bacterial and viral pathogens have established the potential for longer survival of the pathogen at lower temperatures (Gerba 2007; Sinclair *et al.* 2008), if sediment or organic material is present (Gerba & Schaiberger 1975; Gerba & McLeod 1976; Lipson & Stotzky 1984; Clarke *et al.* 1998; Gerba 2007), and in biofilms (Alam *et al.* 2007), and the same principles can be seen in the data on fish viruses (Appendix 4). For example, survival is longer at lower temperatures for VHSV in sea water (Parry & Dixon 1997; Mori *et al.* 2002; see also Hawley & Garver 2008) and fresh water (Mori *et al.* 2002), for IHNV in sea water (Toranzo & Hetrick 1982), for ISAV in sea water (MacLeod *et al.* 2003), and for SAV in both fresh water and sea water (Graham *et al.* 2007); and several viruses are stable for long periods in mud or clay (Ahne 1982; Yoshinaka *et al.* 2000) or in enriched water (VHSV–Kocan *et al.* 2001). There is also evidence that some fish viruses survive longer in sterilized water (KHV in fresh water–Shimizu *et al.* 2006; ISAV in sea water at 15°C–MacLeod *et al.* 2003; SAV in sea water–Graham *et al.* 2007), which may be due to the elimination of bacteria with antiviral properties (Toranzo & Hetrick 1982; Girones *et al.* 1989; Shimizu *et al.* 2006). Different viruses or different strains of viruses can have substantially different abilities to survive in water or mud (*e.g.* IPNV vs. VHSV, PFRV & SVCV–Ahne 1982; different freshwater European VHSV isolates–Parry & Dixon 1997; but smaller differences between European and North American marine VHSV isolates–Parry & Dixon 1997; Winton *et al.* 1991). Differences in genotypes may account for some of the large differences in survival in different studies under nominally similar test conditions (*e.g.* the VHSV data in Appendix 4).

Bacterial or viral pathogens may also be carried in or on their hosts in ballast water. Among the groups of organisms that include NOAA ESA species—cetaceans, pinnipeds, sea turtles, fish, two mollusks, corals and one plant—transport in the host is most likely for pathogens of fish, some of which can infect fish species or life stages (larval or juvenile) that are small enough to survive passage through and transport in a ships' ballast water system. Mollusks and corals are transported in ballast tanks almost entirely as larvae (the exceptions being the possible but probably rare transport of small, post-larval specimens on small floating algae or debris; and transport of the few mollusk species that are planktonic as adults which, being only distantly related to the abalone ESA species, are less likely to host pathogens that can infect them). Since most invertebrate pathogens are thought to be hosted by juvenile or adult rather than larval life stages, the transport and introduction of mollusk or coral pathogens in mollusk or coral specimens carried in ballast tanks is unlikely. It might be possible for a pathogen capable of infecting the one ESA plant species to be carried in a small piece of floating plant material carried in ballast water.

Some bacterial pathogens have been found on a variety of taxonomically diverse alternative hosts, which could include small organisms that are commonly transported in ballast waters. In addition, some bacterial or viral pathogens may occur routinely or

incidentally on the outer surfaces of invertebrates, which may serve as mechanical vectors[3]. For example, it has been shown that the bacterium *Vibrio cholerae* attaches to the chitinous body parts of copepods and other crustaceans (Huq *et al.* 2001; Lipp *et al.* 2002), is found in association with other planktonic invertebrates including larval stages (Martinelli Filho *et al.* 2010), and attaches in abundance to the mucilaginous outer sheaths of some phytoplankton (Huq *et al.* 2001; Lipp *et al.* 2002). Copepods and phytoplankton are common and often abundant in ballast water (*e.g.* Carlton & Geller 1993; Subba Rao *et al.* 1994; Ruiz & Hines 1997; Cohen 1998; MEPC 2003), and associating with these organisms is thought to facilitate *V. cholerae*'s transport in ballast water and possibly its capacity to subsequently infect a host (Colwell 1996; Lipp *et al.* 2002).[4] Although there is generally less information on such associations by animal pathogens, several ectoparasites of fish are known to transmit viral fish pathogens, including fish lice (isopods), copepods and leeches (Ahne 1985; Mulcahy *et al.* 1990; Nylund *et al.* 1994; PAHW 2007; Overstreet *et al.* 2009), and nematode and trematode parasites of sea lions as well as turtle leeches are suspected of transmitting pathogenic viruses between their hosts (Van Bressom *et al.* 1999; Greenblatt *et al.* 2004). Recently, the fish pathogen VHSV was found in benthic amphipods in the Great Lakes (M. Faisal pers. comm.; see also Kipp & Ricciardi 2006 regarding possible survival of VHSV in invertebrates in the bottom of culture ponds). Carried in or on invertebrates or algae (whether serving as hosts or as mechanical vectors), some bacteria and viruses may survive and remain infective for long periods of time, while also being buffered against changes in water chemistry and other environmental conditions (for example, see Huq *et al.* 2001; Lipp *et al.* 2002). These circumstances would presumably facilitate transport in ballast water, and by concentrating the pathogen may possibly increase the chance of infecting a host after discharge from a ballast tank.

I found four examples—involving one bacterium and three viruses—that may represent introductions of these microbes by ballast water. *Vibrio cholerae* is the bacterium that causes human cholera. The world experienced six pandemics of cholera between 1817 and the 1920s. In 1961, after a nearly 40-year hiatus, a seventh pandemic began in Indonesia. It spread through Southeast Asia and to the Indian subcontinent by the mid-1960s, reached Africa in 1970, and spread through Africa and the Middle East in the 1980s (Colwell 1996; Den Enden Erwin 2004). In 1990-1991 cholera broke out on the coast of Peru and spread epidemically throughout Latin America. The same strain of *V. cholerae* that caused the Latin American epidemic was discovered by USFDA in fish and oysters in Mobile Bay, Alabama in 1991 and 1992 (DePaola *et al.* 1992; CDC 1993). The USFDA subsequently found the same strain in the ballast water (and on some ships also in the bilge, fire main or sewage water) of 5 out of 16 ships arriving in

[3] A mechanical vector, also called a paratenic host or a transport host, is an organism that can convey a pathogen to a new host but is not essential to the development of the pathogen.

[4] By attaching to invertebrates or phytoplankton, microbial pathogens may be able to achieve concentrations on these individual substrata that approach or exceed an infective dose, with a greater likelihood of infecting a host than if they were dispersed though the water column. There can be as many as 10^4–10^6 *Vibrio cholerae* cells attached to a single copepod, while an infectious dose, based on human volunteer studies, is only 10^3 cells (Colwell 1996; Lipp *et al.* 2002). Thus Lipp *et al.* (2002) report that in areas without proper sanitation, filtering drinking water through a cloth, which removes copepods but not individual *V. cholerae* cells, can reduce the incidence of cholera infection by approximately 50%.

the U.S. from ports in Latin America (McCarthy *et al.* 1992; McCarthy & Khambaty 1994). Researchers reported an "alarming number" of culturable *V. cholerae* in these samples, estimated at 10^9/liter or greater (McCarthy & Khambaty 1994). They concluded that the South American toxicogenic strain of *V. cholerae* was introduced into U.S. Gulf Coast waters by cargo vessels, probably in ballast water, and that cargo vessels may also have initially introduced the strain into South America from Asia. Subsequent reviewers have generally supported the conclusion that ballast water introduced the toxicogenic strain of *V. cholerae* into Gulf Coast waters (Drake *et al.* 2001, 2007; Dobbs & Rogerson 2005; Tibbetts 2007); but are not in agreement regarding its initial arrival in South America, with opinions ranging from statements that it was introduced in ballast water (Epstein *et al.* 1993; Ditchfield 1993; Epstein 1995; Tibbetts 2007; Aguirre-Macedo *et al.* 2008; this was also reported to be the view of the Pan American Health Organization—Anderson 1991) to statements that it was not (Colwell 1996; Lipp *et al.* 2002). My view is that the data supporting ballast water introduction into Gulf waters are compelling, but that ballast water remains one possible vector among several for the initial introduction to South America, with the available data being inadequate to resolve this.

Infectious Salmon Anemia Virus (ISAV) was first isolated from farmed Atlantic salmon in Norway in 1984, and has since been reported elsewhere in Europe, in the Northwest Atlantic and in Chile (PAHW 2007). ISAV is relatively stable outside its host and may remain infective in water for weeks or more (MacLeod *et al.* 2003; PAHW 2007). Murray *et al.* (2002) investigated the regional pattern of ISAV spread to Atlantic salmon farms in Scotland and the Orkney and Shetland islands in 1998-1999, across >850 km of coastline, and concluded that the virus' spread to several of the farms was not due to either transfers of fish or to diffusive spread by currents, wild fish, fish parasites or seabirds, but rather was associated with the movement of well boats, which transport fish, equipment and supplies to or from salmon farms. Excluding a few sites where infection was clearly caused by transfers of fish from known infected sites, there was a highly significant relationship between well boat visits and infection status (r^2=0.66, p=0.000004). Multivariate analysis showed that site infection was related to the number of harvest visits and not to fish transfer visits or visits for general work or to deliver supplies. In harvest visits, well boats arrived from a fish processing center carrying ballast that was loaded near the center, discharged the ballast near the salmon farm, loaded fish from the farm, and returned to the processing center. At the processing center, salmon were held in net pens before slaughter, and the processing plant discharged effluent from that was not fully disinfected. Thus, a well boat leaving on a harvest visit would ballast with water that could contain ISAV from either the penned salmon or from the plant effluent, which would then be transported to and released near a salmon farm. An alternative explanation to ballast water is that some infected fish or fish detritus could remain in a boats' wells or in its pumps or pipes after salmon are delivered to the harvest station, which could then be discharged by some mechanism while loading salmon at the next farm. Thus, this is a possible and perhaps probable case of the successful transfer of a pathogenic fish virus in ballast water, presumably with an initially high concentration of the virus, to infect other sites across distances of up to several hundred kilometers. Given the relative stability of this virus in sea water,

especially when water temperatures are low (MacLeod *et al.* 2003), this may indicate a potential for considerably longer-distance introductions in ballast water, especially if vessels load ballast near infected fish processing plants or fish farms.

Another possible example of a virus introduced in ballast water is the recent discovery of Viral Hemorrhagic Septicemia Virus (VHSV) in the Great Lakes (Lumsden 2005; Elsayed *et al.* 2006), which has resulted in several large fish kills involving a variety species (Lumsden 2005; Groocock *et al.* 2007; CFSPH 2010). Different researchers and agencies have suggested, argued or concluded that VHSV arrived in the Great Lakes either in ballast water (Whelan 2007; Michigan Sea Grant 2007; Michigan DNR 2009; M. Faisal, pers. comm.; G. Whelan pers. comm.), in migrating fish (M. Bain pers. comm.), in ballast water or migrating fish (Elsayed *et al.* 2006; Kipp & Ricciardi 2006; Wisconsin DNR 2007; NPS 2008; New York Invasive Species Clearinghouse 2008), in migrating fish or birds (J. Casselman pers. comm.) or in migrating fish, ballast water or frozen bait (Illinois - Indiana Sea Grant 2007). VHSV occurs in four distinct genotypes, with Types I, II and III found in Europe and Type IV reported from western North America and Asia (Nishizawa *et al.* 2002; Hedrick *et al.* 2003; Einer-Jensen *et al.* 2004; Snow *et al.* 2004; PAHW 2007). After the discovery of and sequencing of VHSV from the Great Lakes, Type IV was subdivided into Type IVa, occurring on both sides of the North Pacific, and Type IVb, from the Great Lakes (Elsayed *et al.* 2006). Further genetic analysis revealed that the VHSV that had been found in a few fish on the northwestern Atlantic coast (New Brunswick and Nova Scotia) were also apparently Type IVb (Olivier 2002; Gagne *et al.* 2007; Winton *et al.* 2008), suggesting that the Atlantic Coast is the likely source for the introduction of VHSV into the Great Lakes (Elsayed *et al.* 2006; Kipp & Ricciardi 2006; Winton *et al.* 2008). Because the genetic diversity of the VHSV found in the Great Lakes is extremely limited (Winton & Batts 2007; Winton *et al.* 2008), some researchers have argued that it was probably introduced very recently, most likely sometime in the five years before the first record of VHSV in the Great Lakes in 2003 (Winton *et al.* 2008; Michigan DNR 2009; J. Winton, pers. comm.). Other evidence supporting a very recent introduction includes VHSV's high degree of virulence to a wide variety of fish species in the Great Lakes (Lumsden 2005; APHIS 2006; Groocock *et al.* 2007; PAHW 2007; Getchell 2007; CFSPH 2010) suggesting the sudden exposure of a novel pathogen to a naive community of fish (Winton & Batts 2007; Winton *et al.* 2008; J. Winton, pers. comm.), the absence of records of VHS prior to 2003 (Elsayed *et al.* 2006; Winton *et al.* 2008), and its apparent progressive spread from the lower Great Lakes to the upper lakes (Table 7; M. Faisal, pers. comm.). However, other researchers have argued, based on recent documentation of VHSV's wide distribution in the Great Lakes, that it had been present and widespread in the Great Lakes for a significantly longer period, that the lower-to-upper lake progression of discovery is an artifact of where and when sampling was conducted, and that the sudden occurrence of mass mortalities in different species must have a cause other than recent introduction (Bain *et al.* 2010; M. Bain, pers. comm.).

Table 7. Spread of records of VHSV in the Great Lakes. Sources: Bain *et al.* 2010; G. Whelan, pers. comm.

Year	Extension of Range of Records
2003	Lake St. Claire
2005	Downstream: to Lake Ontario (Bay of Quinte)
2006	Downstream: to St. Lawrence River. Upstream: to Lake Huron (Thunder Bay and Swan River).
2007	Upstream: to Lake Michigan (Sturgeon Bay and Algoma)
2008	Upstream: to Lake Michigan (Milwaukee Harbor)
2009	Upstream: to Lake Superior (Paradise and Apostle Islands)

Questions about the source and timing of introduction bear on assessments of the probable vector. The predominant fish species migrating from coastal waters into the Great Lakes is the American eel, *Anguilla rostrata*, and VHSV has been found in the European eel, *A. anguilla* (Castric *et al.* 1992). However, the number of eels migrating into the Great Lakes has declined precipitously in recent decades (Casselman 2003; J. Casselman pers. comm.). If migrating fish are the vector, and the introduction is recent, why did VHSV arrive now when it hadn't arrived during the preceding decades and centuries of eel migration into Lake Ontario? If transport in the water or sediment in ballast tanks is the vector, the virus could probably survive the short voyage from the Atlantic Coast to the Great Lakes well enough (Parry & Dixon 1997; Mori *et al.* 2002; CFSPH 2007; Appendix 4), but delivering enough of a virus in ballast discharges to produce sufficient concentrations in the receiving waters to infect a susceptible fish host would be very challenging. On the other hand, transport of the virus in small fish[5] or invertebrates[6] within ballast tanks might be considerably more likely to deliver an infective dose (*e.g.* Colwell 1996; see discussion above). Despite the interest and importance of determining when and how VHSV arrived in the Great Lakes, the question does not yet appear to have been investigated thoroughly; my sense from these inquiries is that a more complete compilation and analysis of the available data could help resolve this. In the meantime, ballast water remains one of several mechanisms that might be responsible for the introduction of VHSV.

One last example of a possible ballast water introduction of a virus is the discovery of an apparently marine cyanophage in Lake Erie in 2002-2003, primarily in the lake's western basin (Wilhelm *et al.* 2006). A virus capable of lysing a marine cyanobacterium, *Synechococcus* sp. strain WH7803, was collected at numerous sites in the lake even though this cyanobacterium cannot persist in freshwater culture medium representative

[5] For example VHSV is common in gobies (Groocock *et al.* 2007) and has been found in 3-spined stickleback on both North American coasts (Kent *et al.* 1998; Hedrick *et al.* 2006; Gagne *et al.* 2007); both of these small fish are among the most common types of fish reported from ballast tanks (Wonham *et al.* 2000).

[6] Invertebrates serving as alternative hosts or as mechanical vectors have not been reported for VHSV, but as discussed earlier there is evidence for this with other marine pathogens including fish viruses (Ahne 1985; Mulcahy *et al.* 1990; Nylund *et al.* 1994), and VHSV has been found in invertebrates in the Great Lakes (M. Faisal pers. comm.).

of Lake Erie and neither it nor any other host has been found in the lake. Furthermore, the Lake Erie cyanophage isolates amplified on *Synechococcus* sp. strain WH7803 did not lyse other marine or freshwater cyanobacteria that they were tested on. The gene sequences from these isolates clustered with other marine virus sequences. Wilhelm *et al.* (2006) suggest that these findings would be explained by the introduction of the cyanophage from the marine environment in ballast water discharges.

Ballast water pathways

The U.S. Coast Guard and the National Ballast Information Clearinghouse (NBIC) assemble nationwide information on the transport and discharge of ballast water from the Ballast Water Reporting Forms filled out by vessels arriving in U.S. ports. The National Invasive Species Act of 1996 (PL 104-332) required vessels to submit these forms starting in 1997. NBIC was established in 1999, and from 1999-2004 collected data that included all commercial vessels that arrived at U.S. ports from outside of the 200-mile Exclusive Economic Zone (EEZ). After June 2004 the data have included all commercial vessels that arrive at a port or place in the U.S. NBIC uses these data to estimate the rate of ballast water exchange and the release of foreign ballast water, and provides periodic reports to the U.S. Congress (NBIC 2008).

The most recent report available from NBIC covers ballast water records for 2004-2005. Tables 8-10 show the estimated annual number of foreign vessels arriving in U.S. waters, the volume of foreign and domestic ballast water discharged in U.S. waters, and the source regions for foreign ballast water discharged in U.S. waters.[7] "Foreign" and "domestic" have particular idiosyncratic meanings in the NBIC reports and in these tables. "Foreign" refers to vessels arriving in U.S. waters that have traveled in waters outside the U.S. and Canadian EEZs since leaving their last ports, and to the ballast water carried by such vessels. It thus includes vessels traveling between one U.S. Coast (East Coast or Gulf of Mexico) and the other (West Coast or Alaska), or between the U.S. mainland and U.S. island states or territories. It does *not* include vessels traveling along one coast between U.S. ports or between U.S. and Canadian ports while remaining within the combined EEZs. These vessels are classified as "domestic" which refers to vessels, and the ballast water of vessels, that have not traveled outside the combined U.S. and Canadian EEZs since leaving their last ports. Thus the terms foreign and domestic, in the NBIC reports and in this report when referring to vessel or ballast data derived from NBIC, do not imply anything about the nationality, ownership or registration of a vessel.

[7] To develop these estimates I adjusted the NBIC figures by dividing both the number of vessel arrivals and the ballast water discharge volumes by the rates of reporting given in Tables 3 and 4 of Miller *et al.* 2007. No rate of domestic reporting is provided for the Guam region in that report so I used the lowest rate reported for any other discharge region (the Caribbean), since Guam had the lowest rate of foreign reporting of all the regions. Rates of reporting were particularly low for domestic vessels in 2004 because domestic vessels were not required to submit Ballast Water Reporting Forms until after June of that year.

Table 8. Average Annual Number of Foreign Vessels Arriving in U.S. Waters, by Ballast Water Treatment and Discharge Region. Based on 2004-2005 data from Miller *et al.* 2007 (Tables 5a & 5b), adjusted for reporting rates (Miller *et al.* 2007, Table 3). "Pacific Islands" includes Guam and American Samoa. Does not include the Great Lakes, inland waterways and unknown discharge regions.

Discharge Region	Number of Foreign Vessels				
	No Discharge	No Exchange	Some Exchange	Unknown Exchange	Total
East Coast	14,711	2,130	1,183	103	18,126
Gulf of Mexico	11,557	1,029	1,965	68	14,618
Caribbean	4,652	355	391	36	5,433
Alaska	146	60	138	3	346
West Coast	6,197	94	1,417	21	7,728
Hawaii	983	55	47	1	1,085
Pacific Islands	703	44	22	0	768
Total	38,947	3,765	5,160	231	48,103

Table 9. Average Annual Ballast Water Discharge in U.S. Waters, by Ballast Water Treatment and Discharge Region. Based on 2004-2005 data from Miller *et al.* 2007 (Tables 6 & 8), adjusted for reporting rates (Miller *et al.* 2007, Tables 3 & 4a). "Pacific Islands" includes Guam and American Samoa. Does not include the Great Lakes, inland waterways and unknown discharge regions.

Discharge Region	Source	Reported Ballast Water Discharge (1,000 metric tons)			
		No Exchange	Some Exchange	Unknown Exchange	Total
East Coast	Foreign	2,100	4,781	202	7,083
	Domestic	9,968	977	648	11,593
Gulf of Mexico	Foreign	4,148	13,174	403	17,726
	Domestic	49,754	4,392	3,542	57,689
Caribbean	Foreign	2,090	4,608	283	6,982
	Domestic	1,980	117	79	2,177
Alaska	Foreign	1,812	1,595	95	3,502
	Domestic	7,902	893	4	8,799
West Coast	Foreign	638	13,703	225	14,565
	Domestic	10,035	2,810	179	13,024
Hawaii	Foreign	220	211	16	447
	Domestic	45	45	9	100
Pacific Islands	Foreign	152	149	0	301
	Domestic	6	3	0	9
Total	Foreign	11,160	38,222	1,224	50,606
	Domestic	79,691	9,238	4,461	93,390

Table 10. Average Annual Foreign Ballast Water Discharge in U.S. Waters, by Ballast Water Source and Discharge Region. Based on 2004-2005 data from Miller *et al.* 2007 (estimated from Figures 17a & 17b), adjusted for reporting rates (Miller *et al.* 2007, Table 3). "Pacific Islands" includes Guam and American Samoa. Does not include the Great Lakes, inland waterways and unknown discharge regions, or 283,000 MT of discharge from unknown sources.

	Sources of Reported Foreign Ballast Water Discharged (1,000 metric tons)										
Discharge Region	**Northwest Atlantic Ocean**	**Southwest Atlantic Ocean**	**Northeast Atlantic Ocean**	**Mediterranean & Black Seas**	**Southeast Atlantic Ocean**	**Indian Ocean**	**Northwest Pacific Ocean**	**Southwest Pacific Ocean**	**Northeast Pacific Ocean**	**Southeast Pacific Ocean**	**Total**
E. Coast	2,296	70	2,609	661	52	285	696	14	278	52	7,014
GOM	9,927	72	3,453	1,870	108	388	432		1,151	180	17,582
Caribbean	6,797	17		17			17		167	17	6,931
Alaska	17						3,207		260		3,485
W. Coast	112	56	56	56		168	12,580	56	1,426	56	14,565
Hawaii	62					14	42	39	286	3	446
Pacific Is.						2	86	209	4		301
Total	19,111	214	6,118	2,604	160	858	17,060	317	3,572	308	50,324

Note that these estimates are based on data that are compiled from self-reporting by vessels, with little or no independent verification by the U.S. Coast Guard or other entities.[8] Studies have shown that vessel self-reporting tends to substantially overstate the fraction of ballast discharge that is exchanged and/or to overstate the completeness of the exchanges that are conducted (Lockwood 1999; Harkless 2003; Lyles 2004; see also Cohen & Foster 2000, footnote 163). Some aspects of the data suggest that in addition the total amount of discharge may be understated. These annual estimates should therefore be considered to have significant uncertainty associated with them, and to probably understate the risk of transporting and releasing coastal organisms into new locations in ballast water.

However, accepting these data as the best currently available, and combining them with information on the distributions of ESA species (Table 5, Appendix 3) and related

[8] The sole independent verification conducted by federal agencies consists of the U.S. Coast Guard boarding and sampling the salinity in some ballast tanks on some unknown but apparently small number of vessels arriving at coastal ports. If the salinity is too low, the vessel is assumed to not have conducted an exchange of ballast water that meets federal regulations and could be subject to fines or other penalties. However, salinity measurements are only effective at assessing the conduct of mid-ocean exchange in the relatively small number of vessels that load fresh or very low salinity ballast water. For most vessels, salinity measurements are not capable of determining whether a vessel has conducted a ballast exchange that meets regulations. The absence of reliable independent testing, the low legal penalties for inaccurate reporting or intentional falsification of reports, and the paucity of enforcement actions undertaken for inaccurate or false reporting increase the incentives for vessels to under-report discharges and overstate exchanges.

species, allows some comparison of the risks and source areas of risk for different groups of ESA Species. For example, ESA coral species occur in two general regions, the tropical Pacific (Hawaii and the Pacific Islands, with a total of 75 ESA species, four of which are restricted to the Hawaiian Islands and six of which are found in both Hawaii and the Pacific Islands), and the tropical/subtropical Atlantic (a total of nine ESA species found in the Caribbean, the Gulf of Mexico and in the East Coast region in southern Florida; all of these occur in both the Caribbean and Florida, with seven also found in U.S. waters in the Gulf of Mexico and one in southern (non-U.S.) waters in the Gulf of Mexico). In the Hawaii and Pacific Island regions, relatively little foreign ballast water is discharged into U.S. waters (about 0.75 million metric tons (MT) annually), though this is concentrated in a relatively small number of ports. Most of this is derived from the Indo-West Pacific region, with only 62,000 MT coming from the Atlantic. In contrast, the three Atlantic U.S. regions receive nearly 32 million MT of foreign ballast water, of which 1.8 million MT comes from the Indo-West Pacific region. Although this discharge is spread over a larger number or ports and a larger area of coast, including East Coast areas that are too far north to support corals,[9] it nonetheless appears that, based on ballast volumes, there is greater opportunity for coral diseases to be introduced in ballast water from the Indo-West Pacific into U.S. waters in the Atlantic than from the Atlantic into U.S. waters in the Pacific.

Similarly, the sole ESA plant species, Johnson's seagrass (*Halophila johnsonii*), ranges only from Biscayne Bay, Florida (near Miami) to Sebastian Inlet, Florida, about 200 miles north (NOAA 2010b). Vessels arriving in the Miami area discharge about 1.3 million MT of foreign ballast water each year (based on data in Miller *et al.* 2007). Of the foreign ballast water discharged in the East Coast region, 47% is derived from the Eastern Atlantic (mainly the coast of Europe), 14% from the Indo-West Pacific region and 5% from the Eastern Pacific (Table 10), suggesting ample opportunity to introduce seagrass diseases from other coastal regions of the world. Ballast water source data specific to vessels arriving the Port of Miami could be developed from the NBIC database.

The two ESA mollusk species, white abalone (*Haliotis sorenseni*) and black abalone (*H. cracherodii*), are more-or-less restricted to southern California and Baja California (NOAA 2010b).[10] Within this range, ports in the Los Angeles and San Diego areas receive about 3 million MT of foreign ballast water, or about one-fifth of the foreign ballast water discharged to U.S. West Coast waters. They also receive about 40% of the domestic ballast water discharged to the U.S. West Coast (based on data in Miller *et al.* 2007). About 86% of the foreign ballast water discharged to the U.S. West Coast is derived from the Asian Pacific coast, which is home to several species of *Haliotis* that are not found on the U.S. West Coast (Geiger 1999). This suggests that there is a

[9] Of the 7 million MT of foreign ballast water discharged by vessels arriving in East Coast waters each year, 1.3 million MT is discharged by vessels arriving at ports in the Miami region (based on data in Miller *et al.* 2007), within or near the range of ESA coral species.

[10] Black abalone are rare north of San Francisco, with a few unconfirmed records in Oregon (NOAA 2010b).

significant risk of introducing abalone diseases into the U.S. range of these two ESA-listed abalone species via ballast water, especially from Asia.

Another major group of ESA species with limited distributions are the pinnipeds, with five ESA species in Alaska and/or on the West Coast and one ESA species in Hawaii (the Hawaiian monk seal). Both Alaska and the West Coast receive substantial discharges of foreign ballast water (3.5 million MT/yr and 14.6 million MT/yr, respectively), primarily from northern Asia (92% and 86%, respectively) (Table 10). There are two species of otariid pinnipeds and five species of phocid pinnipeds that occur in northern Asia (Nagasawa 1999; Trukhin 2009), all which are found in Alaska and three of which occur on the U.S. West Coast (Orr & Helm 1989; NOAA 2010b). However, there are species, subspecies or DPS in Alaska or on the U.S. West Coast that aren't found in Asia, and five of the Asian species, subspecies or DPS aren't found on the U.S. West Coast (Orr & Helm 1989; Stanley *et al.* 1996; NOAA 2010b). There thus appears to be a significant chance of ballast water transporting pinniped pathogens or strains of pathogens from Asia and releasing them into waters with naive hosts, especially on the West Coast and possibly in Alaska. Hawaii, on the other hand, receives much less foreign ballast water (less than 0.5 million MT/yr), and so there is less opportunity for ballast water to deliver pinniped pathogens. However, since most of that ballast water comes from the northeastern Pacific (64%), which has four species and two subspecies of phocid seals, there may still be a significant risk to the Hawaiian monk seal.

Bacterial and viral threats to ESA-listed species

In general, our knowledge of the diseases of marine and estuarine organisms is quite limited. Much of what we do know is derived from disease outbreaks in animals kept in captivity or used in aquaculture, with less known in many cases about the occurrence of disease in wild populations. Even when diseases are well documented in wild populations, their etiology often remains unknown, and may be viral, bacterial, fungal, protozoan or otherwise in nature. In some cases where pathogenic microbes have been identified in association with disease outbreaks, it is unclear whether the identified pathogens are the primary cause of the morbidity or mortality or represent opportunistic, secondary infections. The life histories of most microbial pathogens are often largely unknown (even for some human pathogens[11]), and the evolutionary history, biogeography of different genotypes, and the varying virulence of different genotypes to different host species is often complicated and poorly worked out.

Below is a summary of bacterial and viral pathogens known to have affected ESA-listed species or considered to be threats to them, and some bacteria and viruses that have

[11] For example, until recently *Vibrio cholerae* was considered to be only a human pathogen with merely incidental occurrence in the environment outside of its host. It wasn't until the late 1970s or 1980s that it was recognized that it has a normal and widespread occurrence as an autochthonous inhabitant of riverine and coastal waters (Lipp *et al.* 2002).

produced disease or mortality in related species. This is not intended to be a complete or exhaustive survey, but rather provides some examples of the types of pathogens that could potentially be transported in ballast water and impact listed species.

Cetacean Pathogens. Most of the cetacean ESA species that occur in U.S. waters have broad distributions and are, as individuals, wide-ranging (NOAA 2010b), which is generally true of whales. This means that species or strains of cetacean pathogens are also likely to be relatively wide-spread, and therefore provide less opportunity for ballast water to introduce a novel pathogen to a naive host population.

Although no ESA-listed cetacean is considered by NOAA to be threatened by disease (NOAA 2010b), bacterial and viral diseases have affected other cetaceans. Bacteria that have been implicated in cetacean diseases and mortalities include species of *Pseudomonas*, *Erysipelothrix*, *Klebsiella* and *Brucella*, as well as *Edwardsiella* and *Salmonella* species which may most often affect animals that are already debilitated or stressed from other causes (Moeller 2002; Van Bressem *et al.* 2008, 2009). Pathogens belonging to at least nine virus families have been detected in cetaceans, with species of morbillivirus, poxvirus and papillomavirus having demonstrated a potential to affect population abundance by raising mortality rates or lowering reproductive success (Van Bressem *et al.* 1999, 2008). In general, inshore and estuarine cetaceans may be at greater risk from bacterial or viral disease due to stressful alterations in habitat and environmental conditions, including contamination, vessel interactions, etc. (Van Bressem *et al.* 2009).

Emerging pathogens of major concern for marine mammals are the morbilliviruses, which were discovered in pinnipeds and cetaceans in the late 1980s (morbilliviruses are discussed in further detail in the section on *Pinniped Pathogens*). Morbillivirus was associated with mass mortalities of striped dolphins in the Mediterranean in 1990-1991 and 2006-2007, of bottlenose dolphins along the U.S. east coast in 1982 and 1987-1988 and in the Gulf of Mexico in 1993-1994, and of long-finned pilot whales in the Mediterranean in 2006-2007 (Barrett *et al.* 1992, 1995; Lipscomb *et al.* 1994, 1996; Kennedy 1998; Moeller 2002; Fernandez *et al.* 2008; Van Bressem *et al.* 2008), including the loss of possibly >50% of the bottlenose dolphins in inshore waters between New Jersey and Florida in 1987-88 (Lipscomb *et al.* 1994; Van Bressem *et al.* 1999, 2008). Kennedy (1998) concluded that morbillivirus mass mortalities “probably resulted from transfer of virus to immunologically-naive populations.” Others have noted that the transmission of morbilliviruses under natural conditions is usually restricted to a single mammalian order (Visser *et al.* 1993c; Osterhaus *et al.* 1995). Evidence of morbillivirus infection was found in several other dolphin and porpoise species in the Atlantic and Mediterranean, and in short-finned pilot whales in the Atlantic and a fin whale and a minke whale in the Mediterranean (Duignan 1995a,b; Barrett *et al.* 1995; Gaydos *et al.* 2004; Wohlsein *et al.* 2007). These were caused by two distinct viruses or virus strains, named Dolphin Morbillivirus (DMV) and Porpoise Morbillivirus (PMV) (Visser *et al.* 1993b,c; Osterhaus *et al.* 1995; De Swart *et al.* 1995; Moeller 2002; Van de Bildt *et al.* 2005), with a possible third strain identified in a pilot whale (Pilot Whale

Morbillivirus, or PWMV–Taubenberger *et al.* 2000; Van Bressem *et al.* 2008). Van Bressem *et al.* (1999) concluded that "cetacean morbillivirus induces a serious disease with a high mortality rate [that] may have long-term effects on the dynamics of cetacean populations." Although initially considered to be diseases of the Atlantic Ocean and Mediterranean Sea, cetacean morbillivirus has been detected in other parts of the world, including common dolphins from a mass stranding in southern California and a stranded pygmy sperm whale in Taiwan (Reiderson *et al.* 1998; Van Bressom *et al.* 1998, 2001; Yang *et al.* 2006).

Gaydos *et al.* (2004) assessed the disease threat to the southern resident killer whale population, an ESA species. They listed seven bacterial and three viral or suspected viral pathogens of captive killer whales and a two other bacterial and one other viral pathogen reported in wild killer whales. One of these, consisting of one or more unidentified species of *Brucella* bacteria from the northeastern Atlantic Ocean, was considered to be capable of reducing fecundity in killer whale populations but of low virulence and epizootic potential. Gaydos *et al.* (2004) also considered four additional bacterial and three viral pathogens found in other odontocete species. Both herpesviruses and morbillivirus scored high on virulence and epizootic potential, but only morbillivirus was judged to have a high capacity to affect fecundity.

Pinniped Pathogens. Except for the Hawaiian monk seal (*Monachus schauinslandi*), no ESA-listed pinniped is considered by NOAA to be threatened by disease (NOAA 2010b). NOAA (2010b) lists exposure to disease from human interactions and disease outbreaks as threats to the Hawaiian monk seal. Much of the concern appears to be with the transmission of diseases from terrestrial hosts (leptospirosis, toxoplasmosis, West Nile virus, etc.), but there are also concerns about transmission from northern elephant seals that occasionally visit the islands: three were sighted between 1978 and 2006 (NMFS 2007).

Bacteria causing pinniped disease include *Pseudomonas*, *Brucella*, *Edwardsiella* and *Salmonella* species, as well as *Klebsiella* and *Leptospira* in California sea lions and northern fur seals (Moeller 2002; Cameron *et al.* 2008; Zuerner *et al.* 2009; Jang *et al.* 2010). A number of serious viral diseases of pinnipeds have been reported including Sea Lion Hepatitis Virus, Seal Herpesvirus (in harbor seals in Europe and in a California Sea Lion), San Miguel Sea Lion Virus (in sea lions and seals) and Seal Pox (primarily in California and South American sea lions and harbor seals) (Moeller 2002). The reported distributions of many of these pinniped diseases are very restricted; if the distributions of the pathogen strains causing these diseases are similarly restricted. There may be a significant potential for ballast water to carry and release pathogens to waters with naive, ESA-listed hosts. As noted earlier, there is some ballast water carried from the northeastern Pacific region (home to eight otariid or phocid pinniped species, two subspecies and two DPS's that are not found in Hawaii[12]) to Hawaii, home of the endangered Hawaiian monk seal; and a great deal of ballast water is carried from Asia (home to four otariid or phocid pinniped species, one subspecies and one DPS that are

[12] Except for rare visits by northern elephant seals, as noted.

not found on the U.S. West Coast) to the U.S. West Coast[13], home to the Eastern DPS of the Steller Sea Lion and a population of the Guadalupe Fur Seal, both ESA threatened species. Ballast water transfers of pinniped pathogens into new regions may be facilitated by the common occurrence of some pinniped species in harbor areas (*e.g.* harbor seals, California sea lions), close to areas where ballast water is discharged. For example, for the past twenty years hundreds of California sea lions have occupied floating docks at the northern end of the Port of San Francisco for much of the year (Wikipedia 2010).

As with cetaceans, morbillivirus is an emerging disease concern for pinniped species. Until the late 1980s morbilliviruses were known only from terrestrial animals, where they are the cause of several major diseases including measles in humans, canine distemper in dogs and other carnivores, and rinderpest and peste-des-petits-ruminants virus in ruminants (Barrett *et al.* 1992; Visser *et al.* 1993c). In 1988 more than 23,000 harbor seals and grey seals died in an epidemic in northwestern Europe, which killed 50-80% of the harbor seal population in some areas (Osterhaus & Vedder 1988; Barrett *et al.* 1992; Visser *et al.* 1993a,c; De Swart *et al.* 1995; Kennedy 1998; Pomeroy *et al.* 2005; Harkonen *et al.* 2006). The epidemic was caused by a previously unknown morbillivirus that was named Phocine Distemper Virus (PDV). Other pinniped species on both sides of the North Atlantic were subsequently found to have been infected by PDV, including harp seal, hooded seal, ringed seal and walrus (Barrett *et al.* 1995; Nielsen *et al.* 2000; Harkonen *et al.* 2006), and a second northern European epidemic in 2002 killed over 30,000 harbor seals (Pomeroy *et al.* 2005; Harkonen *et al.* 2006). Meanwhile, two or three distinct cetacean morbilliviruses were discovered in dolphins, porpoises and whales starting in 1988, as discussed earlier; and a morbillivirus-caused mortality of thousands of freshwater Siberian seals in Lake Baikal in 1987-1998 which initially was thought to be related to the morbillivirus outbreak in harbor seals in northwestern Europe, instead turned out to be due to Canine Distemper Virus (CDV), probably acquired by contact with domestic dogs or other terrestrial carnivores (Visser *et al.* 1990, 1993c; Barrett *et al.* 1992, 1995; De Swart *et al.* 1995; Mamaev *et al.* 1996; Kennedy 1998; Kennedy *et al.* 2000; Harkonen *et al.* 2006). CDV was apparently also responsible for the death of 10,000 Caspian seals in 2000 (Kennedy *et al.* 2000; Fujii *et al.* 2006).

Several surveys did not find any evidence of exposure to PDV in Pacific Ocean pinnipeds in Alaska (harbor seal, spotted seal, bearded seal, ribbon seal, ringed seal, Steller sea lion and walrus sampled between 1976 and 1999–Osterhaus *et al.* 1988; Calle *et al.* 2002, 2008; Burek *et al.* 2005; Zarnke *et al.* 2006[14]) or in British Columbia and the U.S. West Coast (harbor seals sampled between 1992 and 2000–Duignan *et al.* 1995c; Ham-Lamme *et al.* 1999; Hester *et al.* 2004). PDV was believed to be absent from pinnipeds in Pacific waters (Kennedy 1998; Harkonen *et al.* 2006; Calle *et al.*

[13] 12.6 million metric tons/year, which is 86% of the ballast water discharged on the U.S. West Coast (Table 10).

[14] Zarnke *et al.* (2006) did record 2 samples as positive for PDV out of 160 harbor seal samples, but considered these to likely be false positives since the antibody titers barely exceeded the minimum threshold.

2002, 2008; Goldstein *et al.* 2009), and Pacific Ocean pinnipeds were thought to be highly vulnerable to a PDV epidemic due to lack of genetic or acquired immunity (Duignan *et al.* 1994, 1995c; Ham-Lamme *et al.* 1999; Calle *et al.* 2002, 2008)[15]. Recently, Goldstein *et al.* (2009) reported serologic and RNA evidence of PDV in 41 live-captured or dead-collected Alaskan sea otter, including a 300 nucleotide fragment that was identical to an isolate from the 2002 PDV outbreak in European harbor seals and differed from an isolate from the 1998 PDV outbreak at two nucleotide positions. Goldstein *et al.* (2009) concluded that PDV had been introduced to the Pacific Ocean at least after 2000 and probably after the 2002 epidemic, and had likely been introduced by migrating Arctic or sub-Arctic seals, possibly facilitated by reductions in sea ice in 2004 and 2005. However, Goldstein *et al.* and the other cited authors were apparently unaware of studies published in Japanese journals reporting a high incidence of PDV antibodies in Kuril harbor seals, spotted seals and Steller sea lions on Hokkaido in 1996-1997 (Ohashi & Kai 2000, cited by Fujii *et al.* 2006), and declining levels of PDV antibodies in Kuril harbor seals on Hokkaido in 1998-2005 (Fujii *et al.* 2006). Fujii *et al.* (2006) interpreted these data as indicating an epidemic occurrence of PDV in Japanese pinnipeds before 1998 and sporadic occurrence since. In any event, PDV is apparently now present in both Alaskan and Asian waters, with some potential (as noted above) for transport in ballast water to U.S. West Coast or Hawaiian waters where ESA pinniped species reside that have apparently had no prior exposure to PDV.

Based on *in vitro* tests of blood cells, Osterhaus *et al.* (1992) concluded that Mediterranean monk seals (*Monachus monachus*) were susceptible to some marine mammal morbilliviruses (PDV from seals and PMV from porpoises, but not DMV from dolphins). Visser *et al.* (1993c) suggested that morbillivirus infection posed a serious threat to both Mediterranean and Hawaiian monk seals. In 1997 a disease outbreak killed off 70% of the West African population of Mediterranean monk seals—there were only two populations of Mediterranean monk seals, consisting of about 500 animals in the Mediterranean and about 270 animals in West Africa (Osterhaus *et al.* 1997, 1998; Dhermain 2003). Virus collected from several of the dead seals was identified as a new strain, Monk Seal Morbillivirus (MSMV), which is closely related to DMV (Osterhaus *et al.* 1997). Some researchers, however, concluded that the die-off was due to feeding on fish with accumulated dinoflagellate toxin from a red tide, rather than the morbillivirus (Dhermain 2003).

Sea Turtle Pathogens. Although a number of potentially pathogenic, lesion- or disease-associated bacteria (including *Vibrio alginolyticus*, *Aeromonas hydrophila*, *Flavobacterium*, *Mycobacterium*, *Pseudomonas* and *Salmonella enteridis*–Glazebrook & Campbell 1990a,b) and viruses (including Lung-Eye-Trachea Virus, Gray Patch Virus, Loggerhead Genital-Respiratory Herpesvirus and Loggerhead Orocutaneous Herpesvirus–Curry *et al.* 1999, 2000; Stacy *et al.* 2007) have been

[15] "Further studies also must be done to evaluate the potential risk to the harbor seals along the Pacific coast... The only thing protecting Pacific harbor seals at present is the barrier formed by the Arctic...it might only take a single [introduction] event to produce an epizootic as devastating as the recent one in Europe" (Duignan *et al.* 1995c).

reported from captive or wild sea turtles, the main disease concern has been with fibropapillomatosis, which occurs most commonly in green turtles. For example, NOAA (2010b,c) lists fibropapillomatosis in green turtles as the only turtle disease threatening any ESA species, although it also mentions that diseases that affect reef corals indirectly threaten hawksbill turtles. The IUCN (2002) found that fibropapillomatosis threatened 14 out of 34 green turtle populations around the world, including the Florida population. Fibropapillomatosis is characterized by tumors, which are sometimes very large or numerous, both externally on the skin, eyes, oral cavity and carapace (fibropapillomas) and internally on organs (fibromas). These tumors, though considered benign, can interfere with vision, swallowing, breathing and organ function, are a primary cause of strandings in Hawaii and elsewhere, and often result in immunosuppression and secondary bacterial infections (Herbst 1994; Quackenbush *et al.* 1998; Work *et al.* 2003; NOAA 2010c; USGS 2010).

Fibropapillomatosis was first observed in green turtles in the Florida Keys in 1938, and in Hawaii in 1958 (or possibly as early as the 1940s–Williams & Williams 1996), and has become progressively more common and reported more widely (Quackenbush *et al.* 1998; IUCN 2002). It has now been found throughout the greater Caribbean region and in California, Hawaii, Australia, Asia and Africa (Herbst 1994; IUCN 2002). It is most common in green, loggerhead and olive ridley turtles (Herbst 1994; Quackenbush *et al.* 1998; Greenblatt *et al.* 2005), but may occur, at least occasionally, in all sea turtle species (Williams & Williams 2006: questioned by Casey 2006). Since fibropapillomas are easily observed, and sea turtles are highly visible animals that have long been exploited commercially, the lack of earlier records of fibropapillomas suggests that fibropapillomatosis was rare or absent in these regions in the past.

Fibropapillomatosis is generally thought to be caused by a virus that is usually referred to as Fibropapilloma-associated Turtle Herpesvirus (FPTHV).[16] Experiments showed that fibropapillomatosis is transmitted by a filterable agent such as a virus (Herbst 1994), and FPTHV was detected in every fibropapilloma and fibroma tested (Greenblatt *et al.* 2004) at concentrations of at least one copy of viral DNA per cell (Quackenbush *et al.* 1998; Greenblatt *et al.* 2004), which was found to be 2.5-4.5 logs higher than in tumor-free skin from the same animal (Quackenbush *et al.* 2001). However, all efforts to culture FPTHV have been unsuccessful, so the accepted criteria for establishing that a particular microbe is the cause of a disease (known as Koch's postulates) have not been satisfied (Herbst 1994; Work *et al.* 2003, 2009), and there remains some uncertainty whether FPTHV is the cause of fibropapillomatosis.

Quackenbush *et al.* (1998) found three distinct but closely related FPTHV genotypes associated with fibropapillomas in different regions and species, with one genotype in green turtles in Hawaii, another in green and loggerhead turtles in Florida, and a third in olive ridley turtles (probably from the west coast of Central America, though the collection site was not stated). Further work confirmed three genotypes in Australia/Hawaii, Mexico/Costa Rica and Florida/Caribbean regions (Quackenbush *et al.* 2001). Analysis of a larger portion of the genome suggested four or possibly five

[16] It has also been called Chelonid Fibropapilloma-associated Herpesvirus (CFPHV) (Work *et al.* 2009).

genotypes, separated primarily by geography rather than host species, with separate genotypes in the Caribbean (one or two genotypes in green turtles), the American Pacific Coast (green and olive ridley turtles), Hawaii (green turtles) and Australian (green and loggerhead) (Greenblatt *et al.* 2005). On the basis of these global-scale geographic variations in gene sequences, Greenblatt *et al.* (2005) concluded that FPTHV was established in these different regions long before fibropapillomatosis was recognized in the 1930s-1950s.

The regional variation in gene sequence suggest that introductions of FPTHV genotypes between these regions could have impacts on sea turtle populations that are naive to the introduced genotype. Experiments with a different herpesvirus of sea turtles that has been cultured successfully, Lung-Eye-Trachea Virus, found that it remained infective in artificial or natural seawater for over 5 days at 15-23°C (Curry *et al.* 1999, 2000), suggesting that FPTHV might also be stable enough in ballast water to survive relatively short voyages between certain genotype regions, such as between Hawaii and San Diego. Greenblatt *et al.* (2004) examined an array of green turtle parasites and found high loads of FPTHV in marine leeches (*Ozobranchus* spp.), with some sample concentrations indicating nearly 10^7 copies of FPTHV DNA per leech, suggesting that these may serve as mechanical vectors for the transmission of FPTHV. Transport of FPTHV in leeches in ballast water might thus be a more likely mechanisms for successful ballast water introduction than FPTHV in water.

The regional separation of sea turtle species and breeding populations suggests that other microbial pathogens of sea turtles might also be candidates for ballast water introduction, but these have been received less attention and little is known of regarding the geographic distribution of their genotypes.

Fish Pathogens. Probably more is known about the bacterial and viral pathogens of fish than about the pathogens of other types of ESA species. Nonetheless, NOAA doesn't consider any ESA fish species to be threatened by disease (NOAA 2004, 2005, 2010b). NOAA does note that disease transmission resulting from hatchery introductions may significantly impact West Coast salmonid species (NOAA 2010d); and that while West Coast salmonids are affected by bacterial diseases (Bacterial Kidney Disease, columnaris, furunculosis, redmouth disease) and viral diseases (Infectious Hematopoietic Necrosis and Erythrocytic Inclusion Body Syndrome) that they have co-evolved with, there could be greater impacts from introduced pathogens not historically present in a particular watershed (NOAA 1996, 1998, 2004). Similarly, NOAA notes concerns that Atlantic sturgeon could be affected by non-native sturgeon pathogens introduced through aquaculture operations. I briefly review here some of the better-known bacterial and viral fish pathogens, their occurrence in ESA fish species or related species, their geographic distribution and where available, the geographic distribution of recognized genotypes and factors related to their potential transmission in ballast water. Each of the pathogens discussed here has produced substantial and sometimes large-scale mortality in some fish species, often in aquaculture situations but sometimes in

the wild. For reference, the orders, families and genera of ESA fish species are listed in Table 11.

Table 11. Orders, families and genera of ESA fish species.

Order	Family	Genus	ESA Species
Salmoniformes	Salmonidae	Salmo	Atlantic salmon
		Oncorhynchus	9 runs of Pacific Coast Chinook salmon
		Oncorhynchus	2 runs of Pacific Coast Chum salmon
		Oncorhynchus	4 runs of Pacific Coast Coho salmon
		Oncorhynchus	2 runs of Pacific Coast Sockeye salmon
		Oncorhynchus	11 runs of Pacific Coast Steelhead Trout
Acipenseriformes	Acipenseridae	Acipenser	4 sturgeon species
Scopaeniformes	Sebastidae	Sebastes	3 Pacific Coast rockfish species
Pristiformes	Pristidae	Pristis	2 sawtooth species
Gadiformes	Lotidae	Brosme	Cusk
Clupeiformes	Clupeidae	Clupea	Pacific Herring, Southeast Alaska DPS
Osmeriformes	Osmeridae	Thaleichthys	Pacific Eulachon, Southern DPS

Aeromonas salmonicida subsp. *salmonicida* is a bacterium that causes the disease furunculosis in salmonid species. It has been isolated from Atlantic, chinook, coho, chum, sockeye salmon, rainbow trout[17], and 10 other salmonid species, including both farmed and wild fish. It has been reported from other freshwater and a few marine species including two gadiforms (Atlantic cod, coalfish), but serious disease problems have been limited to salmon (Kent *et al.* 1998; Raynard *et al.* 2007). Bakke & Harris (1998) noted that the original spread of the disease was "strongly suggestive of an introduced pathogen," probably initially by transfers of live salmonids in aquaculture, with secondary spread by escapes from fish farms and migrations of wild fish. It is now present wherever salmonids occur. Some strains of this bacteria have multiple resistance to antibiotics, indicating that they probably originated on fish farms. The bacterium can persist in the water for some time; it tends to attach to organic matter in sediment, where it may persist for at least 18 months (Enger 1997; Raynard *et al.* 2007, citing Husevag 1994).

Aeromonas salmonicida–atypical strains refers to a non-*salmonicida* strains of this bacteria, involving at least two apparent subspecies and perhaps more, that are responsible for diseases in a variety of fish species in northern Europe, North America, Japan, Australia and South Africa. Atypical *A. salmonicida* have been isolated from salmonids (Atlantic, coho, chum and sockeye salmon, rainbow trout, and other salmonids), scorpaeniforms (black rockfish (*Sebastes schlegeli*), greenling, sablefish

[17] Rainbow trout and steelhead are the same species, *Oncorhynchus mykiss*; steelhead are the sea-run form.

and lingcod), gadiforms (Atlantic cod, tom cod, haddock) and Pacific herring (Kent *et al.* 1998; Raynard *et al.* 2007).

Listonella (Vibrio) anguillarum may be the most common pathogenic fish bacterium in the world. It causes a vibriosis in fish that has been recognized since 1817. *L. anguillarum* occurs in a wide variety of fish, and is also associated with disease in mollusks and crustaceans. Susceptible species include salmonids (Atlantic salmon and other salmonids) and a gadiform fish (Atlantic cod) (Raynard *et al.* 2007). *L. anguillarum* occurs in several serotypes, which vary among fish species with some serotypes having an apparently narrow host range and others having broad host ranges, and there are genetic differences within serotypes which are sometimes differentiated geographically (Toranzo *et al.* 2005; Raynard *et al.* 2007). Transmission of the disease includes transmission through contact and through food, and the bacterium can reportedly survive for a long time outside its host (Hoff 1989; Raynard *et al.* 2007).

Vibrio salmonicida causes a vibriosis, sometimes called Hitra disease, in farmed Atlantic salmon. It has also been isolated from farmed Atlantic cod (a gadiform fish) and halibut in Europe and Atlantic Canada, but has not been reported from wild fish or from outside the North Atlantic (Raynard *et al.* 2007).

Moritella viscosa (=*Vibrio viscosus*) is associated with winter ulcer or winter lesions disease in Atlantic salmon, rainbow trout, Atlantic cod (a gadiform fish) and other species in northern Europe. Two distinct genetic subgroups relate appear to be related to origins in Norway and Iceland (Raynard *et al.* 2007).

Renibacterium salmoninarum causes Bacterial Kidney Disease (BKD)[18], which was first described in Atlantic salmon in Scotland in the 1930s. It was subsequently reported in salmonids in the United States including chinook, coho and sockeye salmon and steelhead trout, and is now known from a variety of fish in fresh and salt waters in Europe, North and South America and Asia (NOAA 1996; Raynard *et al.* 2007). The disease is primarily known from farmed fish, but the bacterium is common in wild salmonids in Alaska and the U.S. West Coast (NOAA 1996; Raynard *et al.* 2007). Among ESA-listed species, NOAA (1996, citing Foott *et al.* 1994) reported that steelhead may be able to tolerate *R. salmoninarum* infection better than chinook or coho salmon, but Raynard *et al.* (2007) stated that chinook and sockeye salmon are considered more susceptible than Atlantic salmon or rainbow trout. Experiments have shown that transmission of *R. salmoninarum* is possible in Pacific herring and in sablefish, a scorpaeniform fish (Traxler & Bell 1988; Bell *et al.* 1990; Raynard *et al.* 2007), and it has been found in Pacific hake, a gadiform (Kent *et al.* 1998; Raynard *et al.* 2007). Larval fish can carry *R. salmoninarum* (Raynard *et al.* 2007). Although *R. salmoninarum* isolates are generally similar worldwide, some genetic differentiation has been found (Raynard *et al.* 2007).

Piscirickettsia salmonis was first isolated in 1989, and has been found in Atlantic, chinook, coho and other salmon, in rainbow trout, and in sea bass species in Europe,

[18] BKD has also been called Dee disease, white boil disease and kidney disease (Raynard *et al.* 2007).

Chile and British Columbia (Lannan & Fryer 1994; Raynard *et al.* 2007). *P. salmonis* occurs in at least two genotypes, one that was found in all three regions that the disease has been observed and one that was found in Chile (Fryer & Mauel 1997). Isolates from Ireland may represent additional genotypes (Reid *et al.* 2004). Most rickettsiae are transmitted through alternative hosts, which could be the case for *P. salmonis*; however, it's possible that it may be directly transmitted, as it survives in sea water at 5-15°C for at least 14 days (Lannan & Fryer 1994; Fryer & Mauel 1997). It can also be transmitted to larval fish (Raynard *et al.* 2007).

Epizootic Hematopoietic Necrosis Virus (EHNV) is a ranavirus that was isolated in 1986. It is only known from Australia. It has been found in rainbow trout and redfin perch, and several species have been experimentally infected including Atlantic salmon (by injection) and Mountain galaxias, an osmeriform fish (by immersion) (Langdon 1989; PAHW 2007; OIE 2009). It is weakly infective in rainbow trout but often fatal (OIE 2009). EHNV can survive in water for months (Langdon 1989).

Infectious Hematopoietic Necrosis Virus (IHNV) is a rhabdovirus that causes a disease found primarily in salmonids, including both farmed and wild fish. Naturally infected fish include Atlantic, chinook, coho, chum, sockeye and other salmon, rainbow trout, and other salmonid species, as well as Pacific herring (PAHW 2007). White sturgeon larvae and other fish have been infected experimentally by immersion (PAHW 2007). IHNV was first described in sockeye salmon on the U.S. West Coast in the 1950s. It was reported in Japan in 1971 and later on Russia's Pacific Coast (Rudakova *et al.* 2007), probably introduced with imported salmon eggs (Nishizawa *et al.* 2006). It was found from Europe in 1987, thought to have resulted from a single importation of salmon eggs (Enzmann *et al.* 2005). IHNV has also been reported in the Caribbean (Dominican Republic). Two genotypes have been found on the North American West Coast separated geographically, with one ranging from Alaska to Oregon and the other in southern Oregon and California (PAHW 2007), and different strains of IHNV produce different rates of mortality in their hosts (Garver *et al.* 2006). It remains infective for up to 2 weeks in sea water, up to 7 weeks in fresh water at 10-15°C, and up to 9 weeks when adsorbed to clay or other substances (Appendix 4). IHNV was also found to be common in ectoparasites of sockeye salmon, including copepods and leeches (and persisted for at least 16 days in leeches), and may prove to be transmitted by them (Mulcahy *et al.* 1990).

Infectious Pancreatic Necrosis Virus (IPNV) causes an often fatal disease of salmonid species that was first described in 1941, and the virus was isolated in 1972 (Raynard *et al.* 2007). It has been found in Atlantic salmon, rainbow trout and other salmonids, and in various other fish (Raynard *et al.* 2007). It has been spread primarily through transfers of live fish and eggs, and is now found in Europe and the Middle East, the U.S. Atlantic region and New Zealand (Raynard *et al.* 2007). In addition to farmed and wild fish, IPNV has been isolated from water, sediment, birds, scallops, shrimp and other shellfish, which may also contribute to its spread (Cutrin *et al.* 2001). IPNV can accumulate in sediments and shellfish at levels more than two orders of magnitude

greater than those in sea water (Raynard *et al.* 2007). Isolates from the same location often differ in genotype and virulence (Cutrin *et al.* 2001; Shivappa *et al.* 2004).

Infectious Salmon Anemia Virus (ISAV) was first reported in farmed Atlantic salmon in Norway in 1984 (PAHW 2007). It has been isolated from Atlantic and coho salmon and rainbow and brown trout, and in two gadiform and one clupeiform species as well as other fish. In addition, chum salmon have been experimentally infected by injection and another clupeiform, the Atlantic herring *Clupea harengus*, was infected by immersion (Nylund *et al.* 2002; PAHW 2007; Raynard *et al.* 2007). ISAV has been found in northern Europe, in southeastern Canada and northeastern U.S., and in Chile (Murray *et al.* 2002; Cipriano 2002; Raynard *et al.* 2007). There are distinct European and North American genotypes that predate aquaculture, with the Chilean isolate being closely related to the North American (Devold *et al.* 2006; Cipriano 2002; Nylund *et al.* 2003; Raynard *et al.* 2007). The European genotype has been further subdivided into several distinct subgroups (Raynard *et al.* 2007). ISAV can survive in sea water for weeks or longer (MacLeod *et al.* 2003; Appendix 4). It might also be carried in sea lice (Nylund *et al.* 1994).

Koi Herpesvirus (KHV) infects primarily common carp (including varieties such as koi). It has been reported in sturgeon, however the test that was used to determine this cannot distinguish between the mere presence of the virus and its replication in the host fish (PAHW 2007). The disease was first reported in Israel and Germany in 1998 and has spread throughout Europe and to Asia, South Africa and the U.S., mainly through the koi trade (PAHW 2007; OIE 2009). Isolates from different regions of the world are nearly identical, though comparisons of the complete genome revealed some geographic separation into strains (PAHW 2007; Aoki *et al.* 2007; OIE 2009). KHV survives for at most a few days in freshwater or freshwater sediment (Shimizu *et al.* 2006; Appendix 4).

Viral Hemorrhagic Septicemia Virus (VHSV) is a wide-ranging pathogen that has been found in at least 70 different fish species (Skall *et al.* 2005; PAHW 2007). It was first recognized as a disease of farmed rainbow trout in Europe in the 1930s, and the virus was isolated in the 1960s (PAHW 2007). Until 1979, when it was isolated from a wild Atlantic cod, it was considered to be a virus that infected only freshwater fish (Snow *et al.* 2004). In 1988 it was found in chinook and coho salmon in the western U.S., the first records outside of Europe (Brunson *et al.* 1989; Hopper 1989; Winton *et al.* 1991). Over time it was isolated from an increasing number of marine and estuarine fish in Europe, the North Pacific and, in 2000, eastern Canada (Meier *et al.* 1994; Meyers and Winton 1995; Meyers *et al.* 1999; King *et al.* 2001; Brudeseth & Evensen 2002; Hedrick *et al.* 2003; Einer-Jensen *et al.* 2004; Skall *et al.* 2005; Gagne *et al.* 2007) and came to be seen as a virus of marine origin that had recently invaded fresh waters in Europe (Meyers & Winton 1995; Dixon 1999; Einer-Jensen *et al.* 2004; Snow *et al.* 2004). In 2003 VHSV appeared in the Great Lakes (Elsayed *et al.* 2006).

VHSV occurs in four geographically-separated genotypes, with Types I, II and III in Europe and Type IV in North America and Japan (Einer-Jensen *et al.* 2004; Snow *et al.* 2004; PAHW 2007). Type I occurs in five genetic subgroups: one (Type Ib) occurs

mainly in marine fish in the Baltic Sea and North Seas (the latter possibly being migrants from the Baltic), and the other four occur primarily in farmed rainbow trout in different parts of continental Europe (Einer-Jensen *et al.* 2004; Snow *et al.* 2004; Skall *et al.* 2005). These are thought to have been derived from the disease in marine fish, most likely via the practice of feeding marine fish to farmed trout, and to have evolved recently and rapidly in farmed fish (Meyers & Winton 1995; Einer-Jensen *et al.* 2004). Type II occurs in Baltic Sea marine fish associated with the deep water Eastern Gotland Basin, and Type III occurs in North Sea marine fish and has caused substantial mortalities in farmed turbot in the U.K (Einer-Jensen *et al.* 2004; Snow *et al.* 2004). In Europe, disease impacts are mostly restricted to farmed trout and farmed turbot (Einer-Jensen *et al.* 2004). Type IV occurs in two subgroups, IVa in a variety of marine and salmonid fish in western North America and in farmed flounder and some wild marine fish in Japan and Korea (Meyers & Winton 1995; Nishizawa *et al.* 2002; Hedrick *et al.* 2003; Kim & Park 2004; PAHW 2007) and IVb in coastal fish in eastern Canada and a wide variety of freshwater fish in the Great Lakes (Elsayed *et al.* 2006; Gagne *et al.* 2007; Winton *et al.* 2008). VHSV Type IV appears to be nearly avirulent to salmonid species (Meyers & Winton 1995), but there have been mass mortalities in a variety of marine fish in western North America and freshwater fish in the Great Lakes (Meyers & Winton 1995; Meyers *et al.* 1999; Lumsden 2005; Groocock *et al.* 2007; CFSPH 2010).

VHSV has been found in Atlantic, chinook and coho salmon, rainbow trout and other salmonids; two scorpaeniform fish, the black rockfish *Sebastes inermis* and the sablefish; many species of gadiform fish including one species of Lotidae (fourbeard rockling *Enchelyopus cimbrius*); several clupeiform fish including Pacific herring; and several osmeriform fish including Pacific eulachon (Brunson *et al.* 1989; Hopper 1989; Meyers *et al.* 1994; Meyers & Winton 1995; Smail 2000; King *et al.* 2001; Hedrick *et al.* 2003; PAHW 2007), and has caused mass mortalities in rainbow trout (VHSV Type I), Pacific herring (VHSV Type IV) and a variety of other marine and freshwater fish (Meier *et al.* 1994; Meyers & Winton 1995; Meyers *et al.* 1999; Skall *et al.* 2005; Lumsden 2005; Groocock *et al.* 2007; PAHW 2007; Kim *et al.* 2009; CFSPH 2010).

There is a great deal of variation in the data on the survival of VHSV in water (Appendix 4), perhaps due to varying capabilities in different genotypes (Winton *et al.* 1991). However, several studies suggest that VHSV can sometimes survive weeks to months in fresh water, and days to weeks in sea water, especially in colder water (Appendix 4). It is not known whether VHSV is ever transmitted through invertebrates serving as alternate hosts or mechanical vectors, but it was recently found to be associated with benthic amphipods in the Great Lakes (M. Faisal pers. comm.).

Clearly there are a variety of serious fish diseases which have either been shown to affect ESA fish species or that may have the potential to do so, based on natural or experimental infection of related species (Table 12). Some of these have been reported to be persistent (capable of surviving for weeks or longer) in fresh water, sea water or sediment, especially at lower temperatures. Many are capable of infecting small fish or larval fish, which may potentially be transported in ballast water. Several also have a

reported association with invertebrates such as leeches, copepods, isopods or amphipods, which similarly have a potential for transport in ballast water.

Table 12. Occurrence of selected bacterial and viral pathogens in orders of ESA fish species. "X" indicates the pathogen occurs in at least one species in the indicated order; bold font indicates that it occurs in an ESA species; square brackets indicate that the records are for experimentally induced infections. The report of KHV in sturgeon might be due to the mere presence of the virus or contamination of the sample, rather than infection.

Pathogen	**Salmoniformes (salmon & trout)**	**Acipenseriformes (sturgeon)**	**Scorpaeniformes (rockfish)**	**Pristiformes (sawfish)**	**Gadiformes (cusk)**	**Clupeiformes (herring)**	**Osmeriformes (smelt)**
Aeromonas salmonicida subsp. *salmonicida*	**X**				X		
Aeromonas salmonicida–atypical strains	**X**		X		X	**X**	
Listonella anguillarum	**X**				X		
Vibrio salmonicida	**X**				X		
Moritella viscosa	**X**				X		
Renibacterium salmoninarum	**X**		[X]		X	[**X**]	
Piscirickettsia salmonis	**X**						
EHNV	**X**						[X]
IHNV	**X**	[X]				**X**	
IPNV	**X**						
ISAV	**X**				X	X	
KHV		?					
VHSV	**X**		X		X	**X**	**X**

Abalone Pathogens. Among mollusk species, disease outbreaks due to harmful pathogens are probably best documented for oysters, and some of these are believe to have involved introductions of pathogens into new regions, including *Haplosporidium nelsoni* (the cause of MSX disease) in California, eastern North America and Europe (Ford & Haskin 1982; Andrews 1984; Burreson *et al.* 2000; Renault *et al.* 2000; Goulletquer *et al.* 2002; Burreson & Ford 2004), *Bonamia ostreae* in Europe (Van Banning 1987; Cigarria & Elston 1997; Goulletquer *et al.* 2002) and *Perkinsus marinus* (the cause of Dermo disease) in the northeastern U.S. (Ford 1996). These pathogen introductions are generally assumed to have occurred as a result of the transport of oysters, but Burreson & Ford (2004) suggest that *H. nelsoni* spores might have been introduced to western or eastern North America in ballast water, and the same reasoning would apply to the other haplosporidian species. Another group of microbes that have a large impact on mollusks—toxic red tide dinoflagellates that render clams

and mussels unfit for consumption—are believed to have been introduced to some parts of the world via transport in ballast tanks (Hallegraeff & Bolch 1992; Hallegraeff *et al.* 1995; McMinn *et al.* 1997; Hallegraeff 1998; Hamer *et al.* 2000, 2001; Lilly *et al.* 2001).

The molluscan ESA species consist of two endangered abalones, the black abalone *Haliotis cracherodii* and the white abalone *H. sorenseni*, which are largely restricted to California and Baja California (NOAA 2010b). One introduced parasite and one probably introduced bacterial disease have had major impacts on farmed or wild abalone in California in recent decades. The sabellid worm shell parasite, *Terebrasabella heterouncinata*, attacked farmed red abalone, *Haliotis rufescens*, after its introduction in South African abalone, *H. midae*, imported by an abalone farm in the late 1980s (Culver & Kuris 2002; Cohen 2002; Bower 2006). All California abalone farms were affected, some went out of business, and the parasite spread to native gastropods in at least one location necessitating an eradication effort (Culver & Kuris 2000, 2002; Cohen 2002). Although farmed red abalone were the primary species affected, the parasite is capable of infecting most or all California abalone species (Kuris & Culver 1999).

Since 1985, black abalone populations have declined and gone locally extinct in many locations in part due to a wasting disease known as withering syndrome[19], caused by a *Rickettsia*-like bacterium, *Xenohaliotis californiensis*[20] (Lafferty & Kuris 1993; Friedman *et al.* 2000; Smith *et al.* 2003; Bower 2009; NOAA 2010b). At most locations the disease reduced populations by 80% or more, with the greatest reductions in warmer waters (Richards & Davis 1993; Altstatt *et al.* 1996; Moore *et al.* 2002; Raimondi *et al.* 2002; Bower 2009; NOAA 2010b; Smith *et al.* 2003). NOAA (2010b) lists withering syndrome as a primary factor in the decline of black abalone, and it is the reason that the IUCN lists black abalone as Critically Endangered (Smith *et al.* 2003). Other abalone can also be affected, including white abalone (Moore *et al.* 2000; Moore *et al.* 2002; Burton *et al.* 2007; OIE 2009). Several aspects of the disease, including its recent appearance and its progressive spread from an initial occurrence in southern California (Lafferty & Kuris 1993; Altstatt *et al.* 1996; Bergen and Raimondi 2001; Raimondi *et al.* 2002) strongly suggest that it resulted from an introduced pathogen (Smith *et al.* 2003), but if so the mechanism of introduction in unknown. All that is known of *X. californiensis'* potential to survive in water is that it does so long enough to infect other abalone in water-borne transmission studies (OIE 2009). Though all terrestrial rickettsiae are transmitted through alternative arthropod hosts, this may be due to the challenge of bacterial survival and transmission in a terrestrial environment and may not apply to marine rickettsiae (Fryer & Mauel 1997; Moore *et al.* 2002). *X. californiensis* was recently found in China and Thailand in native abalone that exhibited no sign of the disease (OIE 2009). If *X. californiensis* was introduced to southern California, Asia could thus have been the source region, and large quantities of Asian ballast water are discharged to southern California ports (see discussion above under **Ballast water pathways**).

[19] Also called withering disease, foot withering syndrome and abalone wasting disease.
[20] Also sometimes referred to as "*Candidatus* Xenohaliotis californiensis", the use of the term *Candidatus* indicating that the organism is a bacterium that cannot be maintained in a bacteriology culture collection.

Bacterial and viral pathogens of abalone are known from other parts of the world. The bacterium *Vibrio harveyi* has infected abalone species (*Haliotis* spp.) in France, Japan and Australia, causing a vibriosis that results in tissue necrosis and death; *Vibrio carchariae*[21] is the likely cause of some abalone mass mortalities in Japan and Europe; *Vibrio fluvialis* II causes blister disease with high mortalities in China (though *V. fluvialis* is widespread in estuaries around the world); *Vibrio parahaemolyticus* is associated with mass mortalities of abalone in China; *Vibrio alginolyticus* causes mass mortalities in larval and post-larval abalone in Baja California and China; and *Vibrio splendidus* I is associated with abalone disease outbreaks in Australia (Anguiano-Beltran *et al.* 1998; Cai *et al.* 2006; Raynard *et al.* 2007; Bower 2007a, 2010). A *Flavobacterium*-like bacterium is associated with abalone disease outbreaks in Australia (Raynard *et al.* 2007) and *Klebsiella oxytoca* is associated with mass mortalities of abalone in China (Bower 2010). Abalone Viral Ganglioneritis (AVG)[22] is caused by Abalone Herpes-like Virus (AbHV) in China, Taiwan and Australia (Chang *et al.* 2005; Bower 2007b; OIE 2009), and amyotrophia, a fatal wasting disease found in Japan, is believed to be caused by a virus (Bower 2001). Clearly there are a number of serious bacterial and viral diseases of abalone that are present in regions of the world that send substantial quantities of ballast water to California, which could potentially pose a threat to the two species of ESA-listed abalone that reside there.

Coral Pathogens. Coral diseases are a growing threat to coral species around the world. An increasing number of diseases and affected species have been reported since the mid-1990s, along with increases in the range, incidence, prevalence and mortality rates of these diseases (Bruckner 2009; CBD 2009; NOAA 2010e). There are over 30 named coral diseases in the Caribbean and at least seven major diseases and about 30 additional "conditions" characterizing compromised health in scleratinian corals in the Indo Pacific (Bruckner 2009). However, these diseases are still poorly understood: they are often difficult to distinguish, their etiology is frequently unknown, and their development may be complexly related to a variety of factors including pollution, sedimentation, ultraviolet radiation and water temperature. There has been a rapid emergence, wide distribution and high level of virulence of coral diseases in the Caribbean region since the 1970s, producing most of the world's records of coral disease even though most of the corals are in the Indo-Pacific, so that the Caribbean has been considered the world's main "hot spot" for coral disease (Bruckner 2009). This would suggest a greater risk of introducing coral disease from the Atlantic into the Pacific than in the reverse direction; on the other hand, for ballast water discharged into U.S. waters, the greatest transfer of ballast water is from the Pacific into the Atlantic (as discussed earlier under **Ballast water pathways**).[23]

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[21] *V. carchariae* may possibly be a junior synonym of (that is, the same species as) *V. harveyi* (Raynard *et al.* 2007; Bower 2010).

[22] Also reported as "crack-shell disease".

[23] Of course, corals in U.S. waters can be put at risk by coral diseases introduced into nearby non-U.S. waters, and it is not known whether the *total* volume of ballast water transported between tropical regions is greater in the direction from the Indo-Pacific to the Atlantic or from the Atlantic to the Indo-Pacific. The U.S. discharge data collected by the U.S. Coast Guard is insufficient for addressing this.

NOAA (2010b) lists disease as the main cause of the decline of elkhorn coral *Acropora palmata* and staghorn coral *Acropora cervicornis*, the two threatened ESA species, and the listing petition for the 82 additional Candidate species (CBD 2009) argues that "the increase in disease outbreaks and the rapid emergence of new diseases in recent years represents an ever-growing threat to all species of corals." The information provided in NOAA's species descriptions and in the listing petition indicate substantial documented disease impacts on at least 4 Caribbean ESA species; notable susceptibility to disease in all 9 Caribbean ESA species, in 3 of 9 Hawaiian ESA species[24], and in 44 of 66 Indo-Pacific ESA species; resistance to disease in 5 Indo-Pacific ESA species; and hampered ability to recover from disease in 3 Caribbean, 1 Hawaiian and 26 Indo-Pacific ESA species (Table 13).[25] Both the incidence of many coral diseases and/or their impacts appear to increase with water temperatures (Rosenberg & Ben-Haim 2002; Patterson *et al.* 2002; Cervino *et al.* 2004, 2008; Bruno *et al.* 2007; Hall-Spencer *et al.* 2007; CBD 2009; NOAA 2010f) and with coral bleaching (which weakens corals and may reduce their ability to resist or recover from disease, and is itself related to water temperatures) (Brant & McManus 2009; Croquer & Weil 2009; CBD 2009). Although disease impacts are high at many disturbed sites, disease has also spread to reefs in unpopulated areas (Bruckner 2009; NOAA 2010e).

The causative agents of most coral diseases are unknown (Cervino *et al.* 2004; Barash *et al.* 2005; Bruckner 2009; Sussman 2009). However, some of the most important coral diseases appear to be caused by one or more bacteria or cyanobacteria. In some cases (*e.g.* some of the "band" diseases), the disease is characterized by a microbial consortium consisting of a bacteria-dominated community, many or most of whose members are different from the microbe species found in the surrounding water, in healthy coral tissue or on dead coral skeletons (Frias-Lopez *et al.* 2002). These microbes are formed into a microbial mat (the "band") that migrates over the coral surface at rates that may exceed several millimeters per day, destroying coral tissue by exposing it to the anoxic, sulfide-rich basal region of the mat, and leaving dead coral skeleton behind. In such cases of polymicrobial disease, where the disease properties emerge synergistically from the community of microbes, it can be extremely difficult to determine which members of the community (which can include hundreds of species) are the actual "cause" of the disease (Frias-Lopez *et al.* 2004; Cooney *et al.* 2002; Barneah *et al.* 2007; NOAA 2010f). Over the past decade there has been considerable work on the bacterial communities of corals and the role of bacteria in coral disease, but very little work on viruses associated with corals (Rosenberg *et al.* 2007). I briefly review some of the more important coral diseases that are thought to be caused by bacteria.

[24] Five of the nine Hawaiian ESA coral species are also found in other parts of the Indo-Pacific region (see Appendix 3).

[25] Note that these data may be incomplete in various ways. Many of the species descriptions did not mention disease, which could mean that there are no disease impacts or threats to the described species, or that there's no information, or the information may not have been reported. Some species may be so rare and so immediately threatened by other stressors that disease is not currently important, even though it might become very important if the other stressors were controlled. On the other hand, some statements about susceptibility may be speculative, based on the susceptibility of other species in the genus or on the described species' geographic or habitat range.

Table 13. Impacts from, susceptibility to, and recovery from disease for ESA coral species, based on NOAA's online species descriptions and the listing petition for 82 candidate species. Indicates the predominant reported condition for each genus in each region. Source: CBD 2009; NOAA 2010b; summarized from Appendix 5.

Genus	Number of Species	Documented Population Impacts	Generally Susceptible	Generally Less Susceptible	Generally Slow to Recover
CARRIBEAN SPECIES					
Acropora	2	X	X		X
Agaricia	1		X		X
Montastraea	3	X	X		
Dendrogyra	1		X		
Dichocoenia	1		X		
Mycetophyllia	1	X	X		
HAWAIIAN SPECIES (SOME ALSO OCCUR IN THE INDO-PACIFIC REGION)					
Acropora	1		X		X
Montipora	3				
Leptoseris	1		X		
Cyphastrea	2				
Porites	1		X		
Psammocora	1				
INDO-PACIFIC SPECIES					
Acropora	21		X		X
Anacropora	2		X		X
Astreopora	1		X		X
Isopora	2		X		X
Montipora	5		X		
Leptoseris	1		X		
Pachyseris	1				
Pavona	5				
Barabattoia	1		X		
Caulastrea	1				
Acanthastrea	4		X		
Pocillopora	2		X		
Seriatopora	1		X		
Galaxea	1				
Alveopora	3			X	
Porites	3		X		
Turbinaria	4		X		
Euphyllia	3				
Physogyra	1				
Pectinia	1				
Heliopora	1				
Millepora	2			X	

Black Band Disease was first reported in Belize and Florida in 1972, and subsequently spread throughout the western Atlantic (UNEP 2010). It was reported in the Indo-Pacific in 1985, in the Red Sea in 1988, and on the Great Barrier Reef in Australia in 1994 (Rosenberg & Ben-Haim 2002; Sussman 2009; UNEP 2010). It affects a wide variety of species including 26 scleratinean corals, 1 hydrozoan and 6 gorgonians in the western Atlantic, and 49 species in 19 genera in the Indo-Pacific and Red Sea, most commonly on acroporid and faviid corals (Bruckner 2009). Black band disease results from a microbial mat that moves in a narrow band across coral colonies at rates of 3-10 mm/day. This mat has been described as having three functionally dominant components: gliding cyanobacteria dominated by *Phormidium corallyticum*[26], which provide the structure and movement; sulfide-oxidizing bacteria dominated by *Beggiatoa* spp.; and at the base of the mat, sulfate-reducing bacteria dominated by *Desulfovibrio* spp. (Ducklow & Mitchell 1979; Ruetzler & Santavy 1983; NOAA 2010f). More recent research, however, has suggested a different picture, in which some of the initial identifications may be wrong and the actual dominant microbes are a more complicated mix of cyanobacteria and other bacterial forms, along with large numbers of gram negative bacteria amounting in all to over 500 species of bacteria, which moreover may vary in composition or dominant species in different locations (Cooney *et al.* 2002; Frias-Lopez *et al.* 2002, 2003, 2004; Rasoulouniriana *et al.* 2009; Sussman 2009), or may change in composition over time as some corals develop resistance to some agents (Sussman 2009). Sussman (2009) considered the question of whether Black Band Disease is endemic in its widespread locations or introduced to some, and concluded that there is as yet insufficient genetic information to determine this.

Red Band Disease is also believed to be caused by a cyanobacterial mat (Bruckner 2009; UNEP 2010). It occurs in two forms (or perhaps as two different diseases). Type I (RBD-I) was first reported in the 1980s in Belize and has since been found in five genera of corals and sea fans (*Gorgonia*) throughout the Caribbean (Bruckner 2009; UNEP 2010). Type II (RBD-II) has only been found in a single location in the Bahamas, in six genera of corals (Bruckner 2009; UNEP 2010).

Yellow Band Disease[27], also known as Yellow Blotch Disease, was first reported in the Florida Keys in 1994, and has now been found throughout the western Atlantic where it affects corals in the *Montastraea annularis* complex and *M. faveolata* (Dona *et al.* 2008; UNEP 2010; NOAA 2010e,f). It has also occurred at various sites in the southwestern Pacific and the Philippines on *Diploastrea heliopora*, *Herpolitha* spp. and *Fungia* spp. corals since at least 2005 (Cervino *et al.* 2008; Dona *et al.* 2008). Tissue loss from yellow band disease is slower than in black band disease, progressing at a rate of around 0.2-0.3 millimeters per day (Cervino *et al.* 2004; Rosenberg *et al.* 2007; NOAA 2010f). Recent studies suggest that yellow band disease is caused collectively by strains of four or five species of *Vibrio* bacteria: *Vibrio harveyi*, *V. proteolyticus, V. rotiferianus* and *V. alginolyticus*, plus an unidentified *Vibrio* that could be either a new species or a subspecies of *V. alginolyticus* (Cervino *et al.* 2004, 2008). When all four

[26] Recently renamed *Geitlerinema* sp. (Sussman 2009).
[27] A distinct coral disease, also called Yellow Band Disease, has been reported in Turbinaria, Porites, Cyphastrea and Acropora species in the Arabian Gulf (Bruckner 2009).

Vibrio species together were applied to healthy coral, Yellow Band lesions appeared; when the *Vibrio*s were applied singly or in twos or threes, some paling but no lesions appeared (Cervino *et al.* 2004, 2008). The bacteria primarily attack the symbiotic zooxanthellae algae in coral tissues, and only secondarily affect the coral tissues themselves (Cervino *et al.* 2004, 2008; Dona *et al.* 2008). Cervino *et al.* (2008) suggest that Yellow Band Disease may have been introduced into the Pacific by unknown means.[28] Since the *Vibrio* strains that appear to be the cause of Yellow Band Disease may be closely related to strains that have caused mortalities in the Pacific oyster *Crassostrea gigas* and other invertebrates (Cervino *et al.* 2008), introduction with transfers of live shellfish in aquaculture is one possible mechanism. *Vibrio* species are also transported in ballast water, sometimes in large numbers (McCarthy & Khambaty 1994; Ruiz *et al.* 2000; Drake *et al.* 2005), perhaps primarily attached to the chitinous shells of copepods and other crustaceans that are common in ballast water (Huq *et al.* 2001; Lipp *et al.* 2002), so transport in ballast water may be another possible mechanism. A marine worm serves as a reservoir and vector of another *Vibrio* that is a coral pathogen, *V. shiloi* (Sussman *et al.* 2003), and this suggests another possible medium for transport in ballast water.

White Plague Type II was discovered in sea fans in the Florida Keys in 1995, ultimately affecting dozens of coral species in the western Atlantic (Richardson *et al.* 1998; Denner *et al.* 2003; Cervino *et al.* 2004; Bruckner 2009). The disease progresses over the coral surface at a very rapid rate of up to 20 mm per day (NOAA 2010f). The disease is caused by a bacterium that was initially described as "*Sphingomonas*-like" or "a possibly novel species of *Sphingomonas*," but was eventually determined to be a member of a new genus, *Aurantimonas coralicida* (Denner *et al.* 2003). Aguirre-Macedo *et al.* (2008) reported finding "*Sphingomonas* White Plague Type II" in ballast water discharged near a coral reef in the Gulf of Mexico, but since this identification was based on morphology and did not reference Denner *et al.* 's (2003) description of *A. coralicida*, it's not clear to me whether Aguirre-Macedo *et al.* 's *Sphingomonas* is in fact the bacterium that causes White Plague Type II.

Two other types of White Plague have also been described in the Caribbean, Type I which was first reported in Florida in 1975, and Type III which was first reported in 1999 (Bruckner 2009; UNEP 2010; NOAA 2010f). The causative agents of these other White Plague types are not known.

White Plague-like Disease was found in the Gulf of Aqaba in the Red Sea in 2002, primarily affecting the major reef-building genera *Favia* and *Goniastrea* (Barash *et al.* 2005; Rosenberg *et al.* 2007). The disease is caused by the bacterium *Thalassomonas loyana* (Thompson *et al.* 2006) in combination with an unidentified extracellular

[28] "The data provided here shows that the YBD of the Indo-Pacific and Caribbean are identical at the morphological and cellular levels and seem to be caused by a consortium of *Vibrio* species. To speculate how part of this bacterial consortium was found in distant and separated geographic locations such as the Pacific and Caribbean, is beyond the scope of this study; however, it certainly is an interesting topic that involves ongoing research" (Cervino *et al.* 2008, at page 11).

virulence factor contained in a 0.2 µm filtrate of water collected near a disease coral (Barash *et al.* 2005).

White Pox Disease, also known as Patchy Necrosis Disease, Necrotic Patch Syndrome and Acroporid Serratiosis, was discovered in the Florida Keys in 1996 on the ESA species elkhorn coral (*Acropora palmata*), the only species it is known to affect (Patterson *et al.* 2002; NOAA 2010e). It has since been observed throughout the Caribbean (Patterson *et al.* 2002; Bruckner 2009). White Pox is cased by a common mammalian intestinal bacterium *Serratia marcescens*, which can also live independently in soil or water (Patterson *et al.* 2002; Sutherland & Ritchie 2005; Rosenberg *et al.* 2007), and has also been found in ballast water discharged near a coral reef in the Gulf of Mexico (Aguirre-Macedo *et al.* 2008). It is pathogenic to a variety of organisms including mammals, birds, fishes, insects and plants (Sutherland & Ritchie 2005). It may have been released to marine waters in sewage discharges (Sutherland & Ritchie 2005; NOAA 2010e,f).

Bacterial Bleaching was first observed in the Mediterranean in *Oculina patagonica* in 1995, and shown to be caused by the bacterium *Vibrio shiloi* (Kushmaro *et al.* 1996, 2001; Ben-Haim *et al.* 1999). Bacterial bleaching was then reported in *Pocillopora damicornis* in Zanzibar in the Indian Ocean and in the Red Sea in 2001, where it is caused by *Vibrio coralliilyticus* (Ben-Haim & Rosenberg 2002; Ben-Haim *et al.* 2003a). A recent study shows that acute coral tissue loss classified as White Syndrome at three Pacific Ocean locations is also caused by *V. coralliilyticus* (Sussman *et al.* 2008). Sussman (2009) considered the question of whether coral-pathogenic strains of *V. coralliilyticus* are endemic throughout the Indo-Pacific or whether they may have been recently introduced to parts of this range, and concluded that there is as yet insufficient genetic information to determine this. If they are introduced, a possible mechanism could be shellfish transfers for aquaculture, since *V. coralliilyticus* has been reported in larval oysters in England (Ben Haim *et al.* 2003b; Sussman 2009). On the other hand, the records of *Vibrio* species in ballast water (McCarthy & Khambaty 1994; Ruiz *et al.* 2000; Drake *et al.* 2005), the propensity for *Vibrio* species to attach in high numbers to copepods and other invertebrates that are abundant in ballast water (Huq *et al.* 2001; Lipp *et al.* 2002), and the occurrence of at least *Vibrio* species in small invertebrates that may act as both disease reservoirs and as vectors for transport in ballast water (*V. shiloi* in the marine fireworm *Hermodice carunculata*—Sussman *et al.* 2003), suggest that ballast water is also a possible transport mechanism.

In recent decades there has been a confusing proliferation of coral diseases and syndromes involving the loss of coral tissue and the destruction or ejection or symbiotic zooxanthellae, which have had devastating effects of coral populations and coral reef structures, initially concentrated in the Caribbean region but increasingly reported from other parts of the world. Many of these are caused or are suspected of being caused by bacterial agents (Table 14). In five cases causation has been demonstrated by fulfillment of Koch's postulates (White Pox and White Plague Type II in the Caribbean, White Plague-like disease in the Red Sea, and bacterial bleaching in the Mediterranean

Sea and in the Indo-Pacific region). In other cases, where a microbial consortium or a non-culturable bacterium may be the cause of disease, fulfilling Koch's postulates may be infeasible or impossible, and other approaches are being developed to determine etiology (Sussman *et al.* 2008; Sussman 2009). It is generally accepted that Yellow Band Disease is caused by a consortium of *Vibrio* bacteria, and that Black Band Disease is caused by members of a highly complex microbial consortium, though which members is unclear. For several other diseases there is an association between disease lesions and concentrations of certain bacterial species (Table 14), suggesting that these bacteria contribute to the disease, although they could also be opportunistic attackers of coral tissue that has been weakened by disease. *Vibrio harveyi*, or bacteria

Table 14. Coral diseases with known or suspected bacterial agents.

Disease	Location	Coral Hosts	Agent(s)	Reference
DISEASES CAUSED BY BACTERIAL AGENT(S)				
White Pox	Caribbean	*Acropora palmata*	*Serratia marcescens*	Patterson *et al.* 2002
White Plague Type II	Caribbean	many species	*Aurantimonas coralicida*	Denner *et al.* 2003
White-Plague-like disease	Red Sea	*Favia* spp., *Goniastre*a spp.	*Thalassomonas loyana*	Thompson *et al.* 2006
Bacterial Bleaching	Mediterranean Sea	*Oculina patagonica*	*Vibrio shiloi*	Kushmaro *et al.* 1996
Bacterial Bleaching; White Syndrome (part)	Indo-Pacific	several species	*Vibrio coralliilyticus*	Ben Haim *et al.* 2003a; Sussman *et al.* 2008
DISEASES PROBABLY CAUSED BY BACTERIAL AGENT(S)				
Black Band Disease	Caribbean, Indo-Pacific	many species	bacterial consortium	Frias-Lopez *et al.* 2003, 2004
Yellow Band Disease	Caribbean, Pacific	several species	*Vibrio* consortium	Cervino *et al.* 2008
DISEASES THAT MAY BE CAUSED, FACILITATED OR ENHANCED BY BACTERIAL AGENT(S)				
White Band Disease Type II	Caribbean	*Acropora* spp.	*Vibrio harveyi* complex	Gil-Agudelo *et al.* 2006
Rapid Tissue Necrosis; White Syndrome (part)	Indo-Pacific	*Pocillopora damicornis*	*Vibrio harveyi* complex	Luna *et al.* 2007, 2010
Dark Spots Disease	Caribbean	several species	*Vibrio harveyi* complex	Gil-Agudelo *et al.* 2007
Red Band Disease	Caribbean	several species	unknown bacteria	Richardson 1992
Atramentous Necrosis	Great Barrier Reef	*Montipora aequituberculata*	unknown bacteria	Sussman 2009, citing Bourne 2005

very closely related to *V. harveyi*, are implicated in two or three of these[29], and other *Vibrio* species are the cause of three additional diseases. Whether viruses are involved in any coral disease is not known, and little studied (Rosenberg *et al.* 2007).

Our very limited knowledge regarding the pathogens that cause coral disease—for most coral diseases these have not even been identified—makes it difficult to assess the potential for ballast water transport. The distribution of coral diseases suggests the possibility that some disease introductions may have already occurred (Cervino *et al.* 2008; Sussman 2009), whether by ballast water or other means. Clearly bacteria, particularly including *Vibrio* species, are transported in ballast water in substantial numbers and have multiple mechanisms for transport in ballast tanks. Whether coral diseases caused by microbial consortiums are capable of being transported to a new region without a coral host—and whether one or all members of a consortium must be introduced in order to initiate disease—is unknown.

Seagrass Pathogens. There is only one ESA seagrass species, Johnson's seagrass *Halophila johnsonii*, which is found only in southeastern Florida from Sebastian Inlet to Biscayne Bay (Kenworthy 1997). Disease is not considered to be a threat to this species (Kenworthy 1997; NMFS 2002; Kenworthy *et al.* 2007; NOAA 2010b). However, male flowers are not known in this species nor have any seeds or seedlings been found, so it is only known to reproduce vegetatively (Kenworthy 1997; Kenworthy *et al.* 2007).The species has low genetic diversity and a high degree of clonality (Freshwater & York 1999; Freshwater 2004; Kenworthy *et al.* 2007), which could make it especially vulnerable to an introduced pathogen.

The literature on seagrass disease is dominated by papers on eelgrass wasting disease, which struck *Zostera marina* on the U.S. Atlantic coast in the 1930s, eliminating over 95% of the population (Short *et al.* 1988). It also appeared in Europe and on the U.S. Pacific Coast, showed up in New Zealand in the 1960s, and in Atlantic North America again in the 1980s (Short *et al.* 1987, 1988; Steele *et al.* 2005). Eelgrass wasting disease is caused by a marine slime mold, *Labyrinthula zosterae* (Short *et al.* 1987; Muehlstein *et al.* 1988, 1991). A die-off of *Thalassia testudinum* in Florida Bay in the late 1980s, with the complete loss of *T. testudinum* from 10,000 acres and substantial reductions over another 57,000 acres, is also thought to have been caused by a *Labyrinthula* species (Robblee *et al.* 1991; Steele *et al.* 2005). The marine fungus *Lindra thalassiae* causes 'Thalassia disease' on *T. testudinum* (and also "raisin disease" on the brown alga *Sargassum*) (Orpurt *et al.* 1964; Kohlmeyer 1971; Ross *et al.* 2008). There appear to be no reports of any seagrass diseases caused by bacteria or viruses, although of course these might exist undetected. With no information about such diseases, if they exist, there is little that can be said about the potential for ballast transport. However, it is worth noting that there is some potential for ballast water transport of seagrass diseases within their hosts if small pieces of seagrass are entrained in ballast waters.

[29] It is possible that White Band Disease Type II of the Caribbean is the same as some occurrences of the disease that has been called either Rapid Tissue Necrosis or White Syndrome in the Indo-Pacific region.

Conclusions

When naive animal or plant populations are exposed to novel, introduced pathogens, the virulence and impacts of resulting disease can greatly exceed those found in the pathogen's natural host. Our knowledge of the diseases of marine and estuarine organisms is still quite limited, and often predominantly based on species that are maintained in captivity or used in aquaculture. Nevertheless, it is clear that there are many bacterial and viral pathogen strains present in other parts of the world which, if they were introduced into the U.S. ranges of NOAA ESA species and infected those species, could cause them grave harm. As discussed in this report, there are many serious bacterial and viral diseases of cetaceans, pinnipeds, sea turtles, fish and abalone; several devastating bacterial diseases of corals, though no known viral diseases; and apparently no known bacterial or viral diseases of sea grasses (Table 15). Some of the more notable examples are the morbillivirus infections of cetaceans and pinnipeds, emerging diseases whose distributions suggest they are spreading to new regions of the world; fibropapillomatosis of sea turtles, which is believed to be caused by a virus; viral hemorrhagic septicemia of fish, recently introduced into the Great Lakes (possibly in ballast water) where it has caused mass mortalities in numerous fish species; abalone withering syndrome, a primary factor in the decline of the endangered black abalone, which is caused by a bacterium that appears to have been introduced into the black abalone's range; and several bacterial diseases of coral, caused in some cases by single species of bacteria and in others by bacterial consortia, and whose recent emergence and spread may in part be due to introductions.

It is also clear that bacteria and viruses are routinely present in ballast water in substantial numbers, and that in some cases these include pathogens that can infect the types of animals that comprise NOAA's ESA species. It is less clear whether these pathogens are transported and discharged in ballast water in numbers that could reasonably be expected to result in infections in ESA species. While some bacterial or viral pathogens have substantial capacity to survive outside of their hosts for extended periods in fresh water or sea water, others rapidly decline. However, there are other modes of transport within ballast tanks that would allow more sustained transport of bacterial or viral pathogens. Some pathogens are more stable in sediments or biofilms, which are typically present in ballast tanks. At least some, and possibly many, potential pathogens of ESA species have alternative hosts or mechanical vectors that are small enough to be successfully transported in ballast water, and some main hosts (*e.g.* of fish pathogens) may also be small enough to travel in ballast tanks. Examples would include the fish virus VHSV which infects (among many other species) gobies and stickleback, which are small fish that are commonly transported in ballast water; and the various species of *Vibrio* bacteria that cause disease in sea turtles, fish, abalone or corals (Table 15), which are commonly transported in ballast water in concentrated numbers attached to the chitinous shells of copepods or in the mucilaginous sheaths of

Table 15. Examples of known or suspected bacterial and viral pathogens of ESA species groups.

Taxonomic Group	Bacterial Pathogens	Viral Pathogens
Cetaceans	*Pseudomonas*, *Erysipelothrix*, *Klebsiella*, *Brucella*, *Edwardsiella*, *Salmonella*	Dolphin Morbillivirus (DMV), Porpoise Morbillivirus (PMV), Pilot Whale Morbillivirus (PWMV), poxvirus, papilloma virus
Pinnipeds	*Pseudomonas*, *Brucella*, *Edwardsiella*, *Salmonella*, *Klebsiella*, *Leptospira*	Phocine Distemper Virus (PDV), Monk Seal Morbillivirus (MSMV), Sea Lion Hepatitis Virus, Seal Herpesvirus, San Miguel Sea Lion Virus, Seal Pox
Turtles	*Vibrio alginolyticus*, *Aeromonas hydrophila*, *Flavobacterium*, *Mycobacterium*, *Pseudomonas*, *Salmonella enteridis*	Fibropapilloma-associated Turtle Herpesvirus (FPTHV), Lung-Eye-Trachea Virus, Gray Patch Virus, Loggerhead Genital-Respiratory Herpesvirus, Loggerhead Orocutaneous Herpesvirus
Fish	*Aeromonas salmonicida* subsp. *salmonicida* & atypical strains, *Listonella anguillarum*, *Vibrio salmonicida*, *Moritella viscosa*, *Renibacterium salmoninarum*, *Piscirickettsia salmonis*	Viral Hemorrhagic Septicemia Virus (VHSV), Epizootic Hematopoietic Necrosis Virus (EHNV), Infectious Hematopoietic Necrosis Virus (IHNV), Infectious Pancreatic Necrosis Virus (IPNV), Infectious Salmon Anemia Virus (ISAV), Koi Herpesvirus (KHV)
Abalone	*Xenohaliotis californiensis*, *Vibrio harveyi, Vibrio carchariae*, *Vibrio fluvialis* II, *Vibrio parahaemolyticus, Vibrio alginolyticus*, *Vibrio splendidus* I, *Flavobacterium*-like bacterium, *Klebsiella oxytoca*	Abalone Viral Ganglioneritis (AVG), possibly the amyotrophia pathogen
Corals	*Vibrio shiloi*, *Vibrio coralliilyticus*, *Vibrio harveyi*, *Vibrio proteolyticus, Vibrio rotiferianus*, *Vibrio alginolyticus, Aurantimonas coralicida*, *Thalassomonas loyana*, *Serratia marcescens*, and the bacteria that cause Black Band, Red Band and Atramentous Necrosis Disease	None known.
Seagrasses	None known.	None known.

phytoplankton, which are themselves often abundant in ballast water. Transported in ballast water in these alternative hosts or vectors, pathogens would be more likely to reach potential hosts in sufficient concentrations to deliver infective doses. As described in this report, there are a few known outbreaks of human or animal disease that may have resulted from the transport of bacterial or viral pathogens in ballast water.

References

Aguirre-Macedo, M.L., V.M. Vidal-Martinez, J.A.Herrera-Silveira, D.S. Valdés-Lozano, M. Herrera-Rodríguez and M.A. Olvera-Novoa. 2008. Ballast water as a vector of coral pathogens in the Gulf of Mexico: The case of the Cayo Arcas coral reef. Marine Pollution Bulletin 56(9): 1570-1577.

Ahne, W. 1982. Comparative studies on the stability of four fish-pathogenic viruses (VHSV, PFR, SVCV, IPNV). Zentralbl. Veterinarmed. [B] 29: 457–476.

Ahne, W. 1985. *Argulus foljaceus* L. and *Piscicola geometra* L. as mechanical vectors of spring viraemia of carp virus (SVCV). Journal of Fish Diseases 8: 241-242.

Alam, A., M. Sultana, G.B. Nair, A.K. Siddique, N.A. Hasan, R.B. Sack, D.A. Sack, K.U. Ahmed, A. Sadique, H. Watanabe, C.J. Grim, A. Huq and R.R. Colwell. 2007. Viable but nonculturable Vibrio cholerae O1 in biofilms in the aquatic environment and their role in cholera transmission. PNAS 104(45): 17801-17806.

Alpert, R., R. Everett, J. Lishman and D. Smith. 2010. Availability and Efficacy of Ballast Water Treatment Technology: Background and Issue Paper. U.S. Environmental Protection Agency, Washington D.C.

Altstatt, J.M., Ambrose, R.F., Engle, J.M., Haaker, P.L., Lafferty, K.D. and P.T. Raimondi. 1996. Recent declines of black abalone *Haliotis cracherodii* on the mainland coast of central Califor*nia. Marine Ecology Progress Series 1*42: 185-192.

Anderson, C. 1991. Cholera epidemic traced to risk miscalculation. Nature 354: 255.

Anderson, R.M. and R.M. May. 1982. Coevolution of hosts and parasites. Parasitology 85: 411-426.

Andrews, J.D. 1984. Epizootiology of diseases of oysters (*Crassostrea virginica*), and parasites of associated organisms in eastern North America. Helgoland Marine Research 37(1-4): 149-166.

Anguiano-Beltrán, C., R. Searcy-Bernal and M.L. Lizárraga-Partida. 1998. Pathogenic effects of *Vibrio alginolyticus* on larvae and poslarvae of the red abalone *Haliotis rufescens*. Diseases of Aquatic Organisms 33: 119-122.

Aoki ,T., Hirono, I., Kurokawa, K., Fukuda, H., Nahary, R., Eldar, A., Davison, A.J., Waltzek, T.B., Bercovier, H. AND R.P. Hedrick. 2007. Genome sequences of three koi herpesvirus isolates representing the expanding distribution of an emerging disease threatening koi and common carp worldwide. Journal of Virology 81(10): 5058-5065.

APHIS. 2006. Viral Hemorrhagic Septicemia in the Great Lakes, July 2006 Emerging Disease Notice. United States Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, Centers for Epidemiology and Animal Health, Fort Collins, CO.

Ashworth, S.T. 1994. Possible regulation in the Anguillicola crassus host-parasite system. Pages 4141-150 in: Parasitic Diseases of Fish, A.W. Pike and J.W. Lewis (eds.), Samara Press, Dyfed, Great Britain.

Australia DAFF. 2000. Importation of Animals of the Suborder Pinnipedia into Australian Zoos. Final Import Risk Analysis Report. Australia Department of Agriculture, Fisheries and Forestry, Canberra

Bain, M.B., E.R. Cornwell, K.M. Hope, G.E. Eckerlin, R.N. Casey, G.H. Groocock *et al.* 2010. Distribution of an invasive aquatic pathogen (Viral Hemorrhagic Septicemia Virus) in the Great Lakes and its relationship to shipping. PLoS ONE 5(4) e10156. doi:10.1371/journal.pone.0010156.

Bakke, T.A. and P.D. Harris. 1998. Diseases and parasites in wild Atlantic salmon (*Salmo salar*) populations. Canadian Journal of Fisheries and Aquatic Sciences 55(Supplement 1): 247-266.

Barash, Y., R. Sulam, Y. Loya and E. Rosenberg. 2005. Bacterial strain BA-3 and a filterable factor cause a white plague-like disease in corals from the Eilat coral reef. Aquatic Microbial Ecology 40: 183-189.

Barneah, O., E. Ben-Dov, E. Kramarsky-Winterand A. Kushmaro. 2007. Environmental Microbiology 9(8): 1995-2006.

Barrett, T., M. Blixenkrone-Moller, M. Domingo, T. Harder, P. Have, B. Liess, C. Orvell, A.D.M.E. Osterhaus, J. Plana and V. Svansson. 1992. Round table on morbilliviruses in marine mammals. Veterinary Microbiology 33(1-4): 287-295.

Bauer, O.N. and G.L. Hoffman. 1976. Helminth range extension by translocation of fish. Pages 163-172 in: Wildlife Diseases, L.A. Page (ed.) Plenum Press, New York.

Bell, G.R., R,W, Hoffmann and L.L. Brown. 1990. Pathology of experimental infections of the sablefish, *Anoploma fimbria* (Pallas), with *Renibacterium salmoninarum*, the agent of bacterial kidney disease in salmonids. Journal of Fish Diseases13: 355-367.

Ben-Haim, Y. and E. Rosenberg. 2002. A novel *Vibrio* sp. pathogen of the coral *Pocillopora damicornis*. Marine Biology 141: 47-55.

Ben-Haim, Y., Banim, E., Kushmaro, A., Loya, Y. and E. Rosenberg. 1999. Inhibition of photosynthesis and bleaching of zooxanthellae by the coral pathogen *Vibrio shiloi*. Environmental Microbiology 1(3): 223-229.

Ben-Haim, Y., M. Zicherman-Keren and E. Rosenberg. 2003a. Temperature-Regulated bleaching and lysis of the coral *Pocillopora damicornis* by the novel pathogen *Vibrio coralliilyticus*. Applied and Environmental Microbiology 69(7): 4236-4242.

Ben-Haim, Y., F.L. Thompson, C.C. Thompson, M.C. Cnockaert, B. Hoste, J. Swings and E. Rosenberg. 2003b. *Vibrio coralliilyticus* sp. nov., a temperature-dependent pathogen of the coral *Pocillopora damicornis*. International Journal of Systematics and Evolutionary Microbiology 53: 309-315.

Bergen, L. and P. Raimondi. 2001. PISCO Update: Withering Syndrome in Black Abalone. Ecosystem Observations (Monterey Bay National Marine Sanctuary). [Online] http://www.piscoweb.org/publications/scientific-publications/scientific-articles/withering-syndrom-in-black-abalone

Bourne, D.G. 2005. Microbial assessment of a disease outbreak on coral from Magnetic Island (Great Barrier Reef, Australia). Coral Reefs 24: 304-312.

Bower, S.M. 2006. Synopsis of Infectious Diseases and Parasites of Commercially Exploited Shellfish: Sabellid Polychaete Infestation Disease in Abalone. Department of Fisheries and Oceans, Canada. [Online] http://www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/pages/sabelab-eng.htm

Bower, S.M. 2007a. Synopsis of Infectious Diseases and Parasites of Commercially Exploited Shellfish: Blister Disease of Cultured Abalone. Department of Fisheries and Oceans, Canada. [Online] http://www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/pages/blistdab-eng.htm

Bower, S.M. 2007b. Synopsis of Infectious Diseases and Parasites of Commercially Exploited Shellfish: Abalone Viral Mortality. Department of Fisheries and Oceans, Canada. [Online] http://www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/pages/viralmortab-eng.htm

Bower, S.M. 2009. Synopsis of Infectious Diseases and Parasites of Commercially Exploited Shellfish: Withering Syndrome of Abalone. Department of Fisheries and Oceans, Canada. [Online] http://www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/pages/fwsab-eng.htm

Bower, S.M. 2010. Synopsis of Infectious Diseases and Parasites of Commercially Exploited Shellfish: Bacterial Diseases of Abalone. Department of Fisheries and Oceans, Canada. [Online] http://www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/pages/vibrioab-eng.htm

Brandt, M.E. and J.W. McManus. 2009. Disease incidence is related to bleaching extent in reefbuilding corals. Ecology 90: 2859-2869.

Bruckner, A. 2009. The global perspective of incidence and prevalence of coral diseases. Pages 90-121 in: Coral Health and Disease in the Pacific: Vision for Action, S.B. Galloway, A.W. Bruckner and C.M. Woodley (eds.), NOAA Technical Memorandum NOS NCCOS 97 and CRCP 7, National Oceanic and Atmospheric Administration, Silver Spring, MD.

Brudeseth B.E. and O. Evensen. 2002. Occurrence of viral haemorrhagic septicaemia virus (VHSV) in wild marine fish species in the coastal regions of Norway. Diseases of Aquatic Organisms 52: 21–28.

Bruno J.F., E.R. Selig, K.S. Casey, C.A. Page, B.L. Willis, C.D. Harvell, H. Sweatman, and A.M. Melendy. 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biology 5(6): 1220-1227.

Brunson, R., K. True and J. Yancey. 1989. VHS virus isolated at Makah Natlonal Fish Hatchery. American Fisheries Society Fish Health Newsletter 17(2): 3-4.

Burek, K.A., F.M.D. Gulland, G. Sheffield, K.B. Beckmen, E. Keyes, T.R. Spraker, A.W. Smith, D.E. Skilling, J.F. Evermann, J.L. Stott, J.T. Saliki and A.W. Trites. 2005. Infectious disease and the decline of Steller sea lions (*Eumetopias jubatus*) in Alaska, USA: insights from serologic data. Journal of Wildlife Diseases 41(3): 512-524.

Burreson, E. M. and S. E. Ford. 2004. A review of recent information on the Haplosporidia, with special reference to *Haplosporidium nelsoni* (MSX disease). Aquatic Living Resources 17: 499-517.

Burreson, E.M., N.A. Stokes and C.S. Friedman. 2000. Increased virulence in an introduced pathogen: *Haplosporidium nelsoni* (MSX) in the eastern oyster *Crassostrea virginica*. Journal of Aquatic Animal Health 12(1): 1-8.

Burton, R.S., T. B. McCormick, J.D. Moore and C.S. Friedman. 2007. Restoration of Endangered White Abalone, Haliotis sorenseni: Resource Assessment, Genetics, Disease and Culture of Captive Abalone. California Sea Grant College, Project Number R/F-196 Final Report, University of California, San Diego, CA.

Cai, J., H. Han, Z. Song, C. Li and J. Zhou. 2006. Isolation and characterization of pathogenic *Vibrio alginolyticus* from diseased postlarval abalone, *Haliotis diversicolor supertexta* (Lischke). Aquaculture Research 37(12): 1222-1226.

Calle P.P., Seagars, D.J., McClave, C., Senne, D., House, C. and J.A.House. 2002. Viral and bacterial serology of free-ranging Pacific walrus. Journal of Wildlife Diseases 38: 93-100.

Calle, P.P., D.J. Seagars, C. McClave, D. Senne, C. House and J.A. House. 2008. Viral and bacterial serology of six free-ranging bearded seals *Erignathus barbatus*. Diseases of Aquatic Organisms 81: 77–80.

Cameron, C.E., R.L. Zuerner, S. Raverty, K.M. Colegrove, S.A. Norman, D.M. Lambourn, S.J. Jeffries and F.M. Gulland. 2008. Detection of Pathogenic *Leptospira* Bacteria in Pinniped Populations via PCR and Identification of a Source of Transmission for Zoonotic Leptospirosis in the Marine Environment. Journal of Clinical Microbiology 46(5): 1728-1733.

Carlton, J.T. and J.B. Geller. 1993. Ecological roulette: The global transport of nonindigenous marine organisms. Science 261: 78-82.

Casey, J.W. 2006. Author's reply. Journal of Virology 80(9): 4633-4644.

Casselman, J.M. 2003. Dynamics of resources of the American eel, *Anguilla rostrata*: declining abundance in the 1990s. Pages 255-274 in: Eel Biology, K. Aida, K. Tsukamoto and K. Yamauchi (eds.), Springer-Verlag, Tokyo.

Castric, J., Jeffroy, J., Bearzotti, M. and P. de Kinkelin. 1992. Isolation of viral hemorrhagic septicaemia virus (VHSV) from wild elvers *Anguilla anguilla*. Bulletin of the European Association of Fish Pathologists 12(1): 21-23.

CBD. 2009. Petition to List 83 Coral Species under the Endangered Species Act. Submitted to the Secretary of Commerce, Oct. 30. 2009. Center for Biological Diversity, San Francisco, CA.

CDC. 1993. Isolation of Vibrio cholerae O1 from Oysters - Mobile Bay, 1991-1992. Centers for Disease Control, Morbidity and Mortality Weekly Report 42: 91-93.

Cervino, J.M., R.L. Hayes, S.W. Polson, S.C. Polson, T.J. Goreau, R.J. Martinez and G.W. Smith. 2004. Relationship of *Vibrio* species infection and elevated temperatures to yellow blotch/band disease in Caribbean corals. Applied and Environmental Microbiology 70: 6855–6864.

Cervino, J.M., F.L. Thompson, B. Gomez-Gil, E.A. Lorence, T.J. Goreau, R.L. Hayes, K.B. Winiarski-Cervino, G.W. Smith, K. Hughen and E. Bartels. 2008. The *Vibrio* core group induces yellow band disease in Caribbean and Indo-Pacific reef-building corals. Journal of Applied Microbiology 105(5): 1658-1671.

Dona, A.R., J.M. Cervino, V. Karachun, E.A. Lorence, E. Bartels, K. Hughen, G.W. Smith and T.J. Goreau. 2008. Coral Yellow Band Disease; current status in the Caribbean, and links to new Indo-Pacific outbreaks. Pages 236-240 in: Proceedings of the 11th International Coral Reef Symposium, Ft. Lauderdale, Florida, 7-11 July 2008.

CFSPH. 2007. Viral Hemorrhagic Septicemia. Center for Food Security and Public Health, Institute for International Cooperation in Animal Biologics and College of Veterinary Medicine, Iowa State University, Ames, Iowa.

CFSPH. 2010. VHS – Viral Hemmorrhagic Septicemia. Center for Food Security and Public Health, U.S. Department of Agriculture, Animal and Plant Health Inspection Service and Iowa State University. [Online] http://www.focusonfishhealth.org/index.php

Chang, P.H., S.T. Kuo, S.H. Lai, H.S. Yang, Y.Y. Ting, C.L. Hsu and H.C. Chen. 2005. Herpes-like virus infection causing mortality of cultured abalone *Haliotis diversicolor supertexta* in Taiwan. Diseases of Aquatic Organisms 65: 23-27.

Cigarria, J. and R. Elston. 1997. Independent introduction of *Bonamia ostreae,* a parasite of *Ostrea edulis,* to Spain. Diseases of Aquatic Organisms 29: 157-158.

Cipriano, R.C. 2002. Infectious Salmon Anemia Virus. Fish Disease Leaflet #85, United States Geological Survey, National Fish Health Research Laboratory, Kearneysville, WV.

Clark, K.J., A.B. Sarr, P.G. Grant, T.D. Phillips and G.N. Woode. 1998. In vitro studies on the use of clay, clay minerals and charcoal to absorb bovine rotavirus and bovine coronavirus. Veterinary Microbiology 63: 137-146.

Cohen, A.N. 1998. Ships' Ballast Water and the Introduction of Exotic Organisms into the San Francisco Estuary: Current Status of the Problem and Options for Management. A report for CALFED and the California Urban Water Agencies. San Francisco Estuary Institute, Richmond CA.

Cohen, A.N. 2002. The Release of Pest Species by Marine Aquaculture: Lessons from a South African Parasite Introduced into California Waters. Pages 9-13 in: Biological Invasions in Aquatic Ecosystems: Impacts on Restoration and Potential for Control, Proceedings of a Workshop, April 25, 1998, Sacramento, California, A.N. Cohen and S.K. Webb (eds.), San Francisco Estuary Institute, Oakland, CA.

Cook, S.F. 1978. Historical demography. Pages 91-98 in The Handbook of North American Indians, Volume 8 (ed Heizer, R. F.), Smithsonian Institution, Washington, D.C.

Cooney, R., Pantos, O., Le Tissier, M., Barer, M., O'Donnell, A. and J. Bythell.2002. Characterization of the bacterial consortium associated with black band disease in coral using molecular microbiological techniques. Environmental Microbiology 4(7): 401-413.

Combes, C. and N. Le Brun. 1990. Invasions by parasites in continetal Europe. Pages 285-296 in: Biological Invasions in Europe and the Mediterranean Basin, F. di Castri, A.J. Hansen and M. Debussche (eds.), Monographiae Biologicae 65, Kluwer Academic Publishers, Dordrecht.

Cronon, W. 1983. Changes in the Land: Indians, Colonists and the Ecology of New England. Hill and Wang, New York.

Croquer, A. and E. Weil. 2009. Changes in Caribbean coral disease prevalence after the 2005 bleaching event. *Diseases of Aquatic Organisms* 87(1-2): 33-43

Crosby, A.W. 1972. The Columbian Exchange: Biological and Cultural Consequences of 1492. Greenwood Press, Westport, CT.

Crosby, A.W. 1986. Ecological Imperialism: The Biological Expansion of Europe, 900-1900. Cambridge University Press, Cambridge.

Culver, C.S. and A.M. Kuris. 2000. The apparent eradication of a locally established introduced marine pest. Biological Invasions 2(3): 245-253.

Culver, C.S. and A.M. Kuris. 2002. The Introduction of the South African Worm: Biology, History, and Implications for Management. Pages 3-7 in: Biological Invasions in Aquatic Ecosystems: Impacts on Restoration and Potential for Control, Proceedings of a Workshop, April 25, 1998, Sacramento, California, A.N. Cohen and S.K. Webb (eds.), San Francisco Estuary Institute, Oakland, CA.Colwell, R.R. 1996. Global climate and infectious disease: the cholera paradigm. Science 274(5295): 2025-2031.

Curry, S.S., D.R. Brown, E.R. Jacobson and P.A. Klein. 1999. Persistent infectivity of chelonian herpes viruses after exposure to artificial seawater. Page 236 in: Proceedings of the Nineteenth Annual Symposium on Sea Turtle Biology and Conservation, H.J. Kalb and T. Wibbels (eds.), NOAA Technical Memorandum NMFS-SEFSC-443, National Marine Fisheries Service, Miami Laboratory Sea Turtle Program, Miami, FL.

Curry, S.S., D.R. Brown, J.M. Gaskin, E.R. Jacobson, L.M. Ehrhart, S. Blahak, L.H. Herbst and P.A. Klein. 2000. Persistent infectivity of a disease-associated herpesvirus in green turtles after exposure to seawater. Journal of Wildlife Diseases 36(4): 792-797.

Cutrin, J.M., Olveira, J.G., Barja, J.L. and C.P. Dopazo. 2000. Diversity of infectious pancreatic necrosis virus strains isolated from fish, shellfish and other reservoirs in Northwestern Spain. Applied and Environmental Microbiology 66(2): 839-843.

Den Enden Erwin, V. 2004. Illustrated Lecture Notes on Tropical Medicine, Edition 2004. Institute of Tropical Medicine, Antwerp. [Online] http://www.itg.be/evde

Denner E.B.M., G.W. Smith, H.J. Busse, P. Schumann, T. Narzt, S.W. Polson, W. Lubitz and L.L. Richardson. 2003. *Aurantimonas coralicida* gen. nov., sp. nov., the causative agent of white plague type II on Caribbean scleractinian corals. International Journal of Systematic and Evolutionary Microbiology 53: 1115-1122.

DePaola, A., Capers, G.M., Motes, M.L., Olsvik, O., Fields, P.I., Wells, J., Wachsmuth, I.K., Cebula, T.A., Koch, W.H., Khambaty, F., Payne, W.L. and B.A. Wentz. 1992. Isolation of Latin American epidemic strain of Vibrio cholerae O1 from US Gulf Coast. Lancet 339: 624.

De Swart, R.L, T.C. harder, P.S. Ross, H.W. Vos and A.D.M.E. Osterhaus. 1995. Morbilliviruses and morbillivirus diseases of marine mammals. Infectious Agents and Disease 4(3): 125-130.

Devold, M., Karlsen, M. and A. Nylund. 2006. Sequence analysis of the Fusion protein gene from infectious salmon anaemia virus (ISAV) isolates: Evidence of recombination and reassortment. Journal of General Virololgy 87: 2031-2040

Dhermain, F. 2003. Significance of strandings–pathology of cetaceans. Pages 1-59 in: Cetacean Strandings in the Mediterranean Sea, MedNature 2, Regional Activity Centre for Specially Protected Areas (RAC/SPA), Tunis.

Ditchfield, J. 1993. Cholera, plankton blooms, and ballast water. Global Biodiversity (Canadian Museum of Nature) 3(3): 17-18.

Dixon, P.F. 1999. VHSV came from the marine environment: clues from the literature, or just red herrings? *Bulletin of the European Association of Fish Pathology* 19: 60-65.

Dobbs, F.C. and A. Rogerson. 2005. Ridding ships' ballast water of microorganisms. Environmental Science & Technology 39(12): 259A-264A.

Doblin, M.A. and F.C. Dobbs. 2006. Setting a size-exclusion limit to remove or inactivate toxic dinoflagellate cysts in ships' ballast water. Marine Pollution Bulletin 52: 259–263.

Doblin, M.A., Drake, L.A., Coyne, K.J., Rublee, P.A. and F.C. Dobbs. 2004. *Pfiesteria* species identified in ships' ballast water and residuals: a possible vector for introductions to coastal areas. Pages 317-319 in: Harmful Algae, K.A. Steidinger, J.H. Landsberg, C.R. Tomas and G.A. Vargo (eds.), Florida Fish and Wildlife Conservation Commission, Florida Institute of Oceanography, and Intergovernmental Oceanographic Commission of UNESCO.

Drake, L.A., K.H. Choi, G.M. Ruiz and F.C. Dobbs. 2001. Global redistribution of bacterioplankton and virioplankton communities. Biological Invasions 3: 193–199.

Drake, L.A., Meyer, A.E., Forsberg, R.L., Baier, R.E., Doblin, M.A., Heinemann, S., Johnson, W.P., Koch, M., Rublee, P.A. and F.C. Dobbs. 2005. Potential invasion of microorganisms and pathogens via 'interior hull fouling': biofilms inside ballast-water tanks. Biological Invasions 7: 969–982.

Drake, L.A., M.A. Doblin and F.C. Dobbs. 2007. Potential microbial bioinvasions via ships' ballast water, sediment, and biofilm. Marine Pollution Bulletin 55: 333-341.

Ducklow, H. and R. Mitchell. 1979. Observations on naturally and artificially diseased tropical corals: A scanning electron microscope study. Microbial Ecology 5(3): 215-223.

Duignan, P.J., J.T. Saliki, D.J. St. Aubin, J.A. House and J.R. Geraci. 1994. Neutralizing antibodies to phocine distemper virus in Atlantic walrus (*Odobenus rosmarus rosmarus*) from Arctic Canada. Journal of Wildlife Diseases 30: 90-94.

Duignan, P.J., C. House, J.R. Geraci, G. Early, H.G. Copland, M.T. Walsh, Gregory D. Bossart, C. Cray, S. Sadove, D.J. St. Aubin and M. Moore. 1995a. Morbillivirus infection in two species of pilot whale (Globicephala sp.) from the western Atlantic. Marine Mammal Science 11(2): 150-162.

Duignan, P.J., C. House, J.R. Geraci, N. Duffy, B.K. Rima, M.T. Walsh, G. Early, D.J. St. Aubin, S. Sadoveg, H. Koopman and H. Rhinehart. 1995b. Morbillivirus infection in cetaceans of the Western Atlantic. Veterinary Microbiology 44(2-4): 241-249.

Duignan, P.J., Saliki, J.T., St Aubin, D.J., Early, G., Sadove, S., House, J.A., Kovacs, K. and J.R. Geraci. 1995c. Epizootiology of morbillivirus infection in North America harbor seals (*Phoca vitulina*) and grey seals (*Halichoerus grypus*). Journal of Wildlife Diseases 31(4): 491-501.

Einer-Jensen, K., P. Ahrens, R. Forsberg and N. Lorenzen. 2004. Evolution of the fish rhabdovirus viral haemorrhagic septicaemia virus. *Journal of General Virology* 85: 1167-1179.

Elsayed, E., M. Faisal, M. Thomas, G. Whelan, W. Batts and J. Winton. 2006. Isolation of viral haemorrhagic septicaemia virus from muskellunge, *Esox masquinongy* (Mitchell), in Lake St. Clair, Michigan, USA reveals a new sublineage of the North American genotype. Journal of Fish Diseases 29: 611-619.

Enger, O. 1997. Survival and inactivation of Aeromonas salmonicida outside the host – a most superficial way of life. Pages 159-177 in: Furunculosis: Multidisciplinary Fish Disease Research, Bernoth, E.M., A.E. Ellis, P.J. Midtlyng, G. Olivier and P. Smith (eds.), Academic Press London.

Enzmann, P.J., Kurath, G., Fichtner, D. and S.M. Bergmann. 2005. Infectious hematopoietic necrosis virus: monophyletic origin of European isolates from North American Genogroup M. Diseases of Aquatic Organisms 66: 187-195.

Epstein, P.R. 1995. Emerging diseases and ecosystem instability: new threats to public health. Am J Public Health 85(2): 168-172.

Epstein, P.R., Ford, T.E. and R.R. Colwell. 1993. Marine ecosystems. Lancet 342: 1216-19.

Fenner, F. and K. Myers. 1977. Myxoma virus and myxomatosis in retrospect: the first quarter century of a new disease. Pages 539-570 in: Viruses and Environment, E. Kurstak and K. Maramorosch (eds.), Academic Press, New York.

Fernandez, A. F. Esperón, P. Herraéz, A. Espinosa de los Monteros, C. Clavel, A. Bernabé, J. Manuel Sánchez-Vizcaino, P. Verborgh, R. DeStephanis, F. Toledano and A. Bayón. 2008. Morbillivirus and Pilot Whale Deaths, Mediterranean Sea. Emerging Infectious Diseases 14(5): 792-794.

Ford, S.E. 1996. Range extension by the oyster parasite *Perkinsus marinus* into the northeastern United States: response to climate change? Journal of Shellfish Research 15: 45-56.

Ford, S.E. and H.H. Haskin. 1982. History and epizootiology of *Haplosporidium nelsoni* (MSX), an oyster pathogen in Delaware Bay, 1957-1980. Journal of Invertebrate Pathology 40(1): 118-141.

Freshwater, D.W. and R. York. 1999. Determination of genetic diversity in the proposed endangered threatened species *Halophila johnsonii* Eiseman. Center for Marine Science Research, University of North Carolina, Wilmington, NC.

Freshwater, D.W. 2004. Analyses of genotypic and phylogenetic relationships of *Halophila johnsonii* and preliminary testing of AFLPs for detecting genets of *Halophila johnsonii*, Center for Marine Science, University of North Carolina, Wilmington, NC.

Frias-Lopez, J., A. Zerkle, G. Bonheyo and B. Fouke. 2002. Partitioning of bacterial communities between seawater and healthy, black band diseased, and dead coral surfaces. Applied and Environmental Microbiology 68(5): 2214-2228.

Frias-Lopez, J., G.T. Bonheyo, Q. Jin, and B.W. Fouke. 2003. Cyanobacteria Associated with Coral Black Band Disease in Caribbean and Indo-Pacific Reefs. Applied and Environmental Microbiology 69(4): 2409-2413.

Frias-Lopez, J., J.S. Klaus, G.T. Bonheyo and B.W. Fouke. 2004. Bacterial Community Associated with Black Band Disease in Corals. Applied and Environmental Microbiology 70(10): 5955-5962.

Friedman, C.S., K.B. Andree, K.A. Beauchamp, J.D. Moore, T.T. Robbins, J.D. Shields, and R.P. Hedrick. 2000. '*Candidatus Xenohaliotis californiensis*', a newly described pathogen of abalone, *Haliotis* spp., along the west coast of North America. International Journal of Systematic Evolution and Microbiology 50: 847-855.

Fryer, J.L. and M.J. Mauel. 1997. The Rickettsia: an Emerging Group of Pathogens in Fish. Emerging Infectious Diseases 3(2): 137-144.

Fujii, K., Sato, H., Kakumoto, C., Kobayashi, M., Saito, S., Kariya, T., Watanabe, Y., Sakoda, Y., Kai, C., Kida, H. and M. Suzuki. 2006. Seroepidemiological survey of morbillivirus infection in Kuril harbor seals (*Phoca vitulina stejnegeri*) of Hokkaido, Japan. Japanese Journal of Veterinary Research, 54(2-3): 109-117.

Gagne, N., MacKinnon, A.M., Boston, L., Souter, B., Cook-Versloot, M., Griffiths, S., and G. Olivier. 2007. Isolation of viral haemorrhagic septicaemia virus from mummichog, stickleback, striped bass and brown trout in eastern Canada. Journal of Fish Diseases 30: 213-223.

Galil, B.S. and N. Hulsmann. 1997. Protist transport via ballast water – biological classification of ballast tanks by food web interactions. European Journal of Protistology 33: 244-253.

Garver, K.A, Batts, W.N. and G. Kurath 2006. Virulence comparisons of infectious hematopoietic necrosis virus U and M genogroups in sockeye salmon and rainbow trout. Journal of Aquatic Animal Health 18: 232-243.

Gaydos, J.K., K.C. Balcomb, III, R.W. Osborne and L. Dierauf. 2004. Evaluating potential infectious disease threats for southern resident killer whales, Orcinus orca: a model for endangered species. Biological Conservation 117: 253-262.

Geiger, D.L. 1999. Distribution and biogeography of the recent Haliotidae (Gastropoda: Vetigastropoda) world-wide. *Bolletino Malacologico* 35: 57-120.

Gerba, C.P. 2007. Virus Occurrence and Survival in the Environmental Waters. Human Viruses in Water, Bosch, A. (ed.), Perspectives in Medical Virology 17: 91-108.

Gerba C.P. and J.S. McLeod. 1976. Effect of sediments on the survival of Escherichia coli in marine waters. Applied Environmental Microbiology 32(1): 114-120.

Gerba C.P. and G.E. Schaiberger. 1975. Effect of particulates on virus survival in seawater. *Journal Water Pollution Control Federation* 47(1): 93-103.

Getchell, R. 2007. VHSV in the Great lakes. Slide presentation at Cool Water Egg Disinfection Workshop, August 9, 2007, University of Wisconsin Extension/Northern Aquaculture Demonstration Facility. http://www.uwsp.edu/cls/aquaculture/docs/projects/VHS%20in%20the%20Great%20Lakes.pdf.

Gil-Agudelo, D.L., G.W. Smith and E. Weil. 2006. The white band disease type II pathogen in Puerto Rico. International Journal of Tropical Biology 54 (Suppl. 3): 59-67.

Gil-Agudelo, D.L., Fonseca, D.P., Weil. E., Garzon-Ferreira, J. and G.W. Smith. 2007. Bacterial communities associated with the mucopolysaccharide layers of three coral species affected and unaffected with dark spots disease. Canadian Journal of Microbiology 53: 465-471.

Girones, R., J.T. Jofre and A. Bosch. 1989. Isolation of marine bacteria with antiviral properties. Canadian Journal of Microbiology 35: 1015-1021.

Glazebrook, J.S. and R.S.F. Campbell. 1990. A survey of the diseases of marine turtles in northern Australia. I. Farmed turtles. Diseases of Aquatic Organisms 9: 83-95.

Glazebrook, J.S. and R.S.F. Campbell. 1990. A survey of the diseases of marine turtles in northern Australia. II. Oceanarium-reared and wild turtles. Diseases of Aquatic Organisms 9: 97-104.

Goldstein, T., J.A.K. Mazet, V.A. Gill, A.M. Doroff, K.A. Burek, and J.A. Hammond. 2009. Phocine Distemper Virus in Northern Sea Otters in the Pacific Ocean, Alaska, USA. Emerging Infectious Diseases 15(6): 925-927.

Gollasch, S., J. Lenz, M. Dammer and H.G. Andres. 2000. Survival of tropical ballast water organisms during a cruise from the Indian Ocean to the North Sea. Journal of Plankton Research 22(5): 923-937.

Goodrich, A.L. 2006. Characterization of b-lactam resistant, pandemic serotypes of *Vibrio cholerae* isolated from ships' ballast tanks and coastal waters. M.S. thesis in Ocean and Earth Sciences, Old Dominion University, Norfolk, VA.

Goulletquer, P., Bachelet, G., Sauriau, P.G. and P. Noel. 2002. Open Atlantic coast of Europe - a century of introduced species into French waters. Pages 276-290 in: Invasive Aquatic Species of Europe Distribution Impacts and Management, E. Leppakoski *et al.* (eds.), Kluwer Academic Publishers, The Netherlands.

Graham, D.A., Staples, C., Wilson, C.J., Jewhurst, H., Cherry, K., Gordon, A. and H. M. Rowley. 2007. Biophysical properties of salmonid alphaviruses: influence of temperature and pH on virus survival. Journal of Fish Diseases 30(9): 533-543.

Greenblatt, R. J., T. M. Work, G. H. Balazs, C. A. Sutton, R. N. Casey, and J. W. Casey. 2004. The *Ozobranchus* leech is a candidate mechanical vector for the fibropapilloma-associated turtle herpesvirus found latently infecting skin tumors on Hawaiian green turtles (*Chelonia mydas*). Virology 321:101-110.

Greenblatt, R.J., S.L. Quackenbush, R.N. Casey, J. Rovnak, G.H. Balazs, T.M. Work, J.W. Casey and C.A. Sutton. 2005. Genomic variation of the fibropapilloma-associated marine turtle herpesvirus across seven geographic areas and three host species. Journal of Virology 79(2): 1125-1132.

Groocock, G.H., Getchell, R.G., Wooster, G.A., Britt, K.L., Batts, W.N., Winton, J.R., Casey, R.N., Casey, J.W. and P.R. Bowser. 2007. Detection of viral hemorrhagic septicemia in round gobies in New York State (USA) waters of Lake Ontario and the St. Lawrence River. *Diseases of Aquatic Organisms* 76: 187-192.

Groocock, G.H., R.G. Getchell, G.A. Wooster, K.L. Britt, W.N. Batts, J.R. Winton, R.N. Casey, J.W. Casey and P.R. Bowser. 2007. Detection of viral hemorrhagic septicemia in round gobies in New York State (USA) waters of Lake Ontario and the St. Lawrence River. Diseases of Aquatic Organisms 76: 187-192.

Hallegraeff, G.M. 1998. Transport of toxic dinoflagellates via ships' ballast water: bioeconomic risk assessment and efficacy of possible ballast water management strategies. Marine Ecology Progress Series 168: 297-309.

Hall-Spencer, J.M., J. Pike and C.B. Munn. 2007. Diseases affect cold-water corals too: *Eunicella verrucosa* (Cnidaria: Gorgonacea) necrosis in SW England. Diseases of Aquatic Organisms 76: 87-97.

Hallegraeff, G.M. and C.J. Bolch. 1992. Transport of diatom and dinoflagellate resting spores in ships' ballast water: implications for plankton biogeography and aquaculture. Journal of Plankton Research 14(8): 1067-1084.

Hallegraeff, G.M., M.A. McCausland and R.K. Brown. 1995. Early warning of toxic dinoflagellate blooms of Gymnodinium catenatum in southern Tasmanian waters. Journal of Plankton Research 17: 1163-1176.

Hamer, J.P., T.A. McCollin and I.A.N. Lucas. 2000. Dinoflagellate cysts in ballast tank sediments: between tank variability. Marine Pollution Bulletin 40(9): 731-733.

Hamer, J.P., I.A.N. Lucas and T.A. McCollin. 2001. Harmful dinoflagellate resting cysts in ships' ballast tank sediments: potential for introduction into English and Welsh waters. Phycologia 40(3): 246–255.

Ham-Lammé, K. ., D.P. King, B.C. Taylor, C. House, D.A. Jessup, S. Jeffries, P.K Yochem, F.M.D. Gulland, D.A. Ferrick and J.L. Stott. 1999. The application of immuno-assays for serological detection of morbillivirus exposure in free ranging harbor seals (*Phoca vitulina*) and sea otters (*Enhydra lutris*) from the western coast of the United States. Marine Mammal Science 15(2): 601-608.

Harkless, K. 2003. How accurate is ballast water reporting? Aquatic Invaders 14(1): 2-7.

Harkonen, T., R. Dietz, P. Reijnders, J. Teilmann, K. Harding, A. Hall, S. Brasseur, U. Siebert, S.J. Goodman, P.D. Jepson, T.D. Rasmussen and P. Thompson. 2006. A review of the 1988 and 2002 phocine distemper virus epidemics in European harbour seals. Diseases of Aquatic Organisms 68: 115-130.

Hawley, L.M. and K.A. Garver. 2008. Stability of viral hemorrhagic septicemia virus (VHSV) in freshwater and seawater at various temperatures. Diseases of Aquatic Organisms 82(3): 171-178.

Hedrick, R.P., W.N. Batts, S. Yun, G.S. Traxler, J. Kaufman and J.R. Winton. 2003. Host and geographic range extensions of the North American strain of viral hemorrhagic septicemia virus. *Diseases of Aquatic Organisms* 55: 211-220.

Herbst, L.H. 1994. Fibropapillomatosis of marine turtles. Annual Review of Fish Diseases 4: 389-425.

Hester, M., S. Allen2, D. Adams and H. Nevins. 2004. Draft Pinniped Long-Term Monitoring Program, San Francisco Area Network of Parks. National Park Service, Natural Resource Program Center, Fort Collins, CO.

Hoff, K.A. 1989. Survival of *Vibrio anguillarum* and *Vibrio salmonicida* at different salinities. Applied Environmental Microbiology 55: 1775-1786.

Hoffman, G.L. 1970. Intercontinental and transcontinental dissemination and transfaunation of fish parasites with emphasis on whirling disease (*Myxosoma cerebralis*). Pages 69-81 in: A Symposium on Diseases of Fishes and Shellfishes, S.F. Snieszko (ed.), American Fisheries Society, Washington, D.C.

Hopper, K. 1989. The isolation of VHSV from chinook salmon at Glenwood Springs, Orcas Island, Washington. *American Fisheries Society Fish Health Newsletter* 17(2): 1.

Huq, A., R.B. Sack and R.R. Colwell. 2001. Cholera and global ecosystems. Pages 327-347 in: Ecosystem Change and Public Health: A Global Perspective, J.L. Aron and J. Patz (eds.), Johns Hopkins University Press, Baltimore, MD.

Hulsmann, N. and B.S. Galil. 2001. The effects of freshwater flushing on marine heterotrophic protists - implications for ballast water management. Marine Pollution Bulletin 42(11): 1082-1086.

Husevåg, B. 1004. Survival of *Aeromonas salmonicida* and *Vibrio salmonicida* in marine fish farm environments. Ph.D. dissertation, Department of Microbiology, University of Bergen, Norway.

Illinois–Indiana Sea Grant. 2007. VHS in Lake Michigan: Questions and Answers. Illinois–Indiana Sea Grant College Program, University of Illinois, Urbana, IL.

IUCN. 2002. Marine Turtle Specialist Group Review Draft, 2002 IUCN Red List Status Assessment: Green Turtle (*Chelonia mydas*). Marine Turtle Specialist Group, Species Survival Commission, Red List Programme, The World Conservation Union (IUCN).

Jang, S. L. Wheeler, R.B. Carey, B. Jensen, C.M. Crandall, K.N. Schrader, D. Jessup, K, Colegrove and F.M.D. Gulland. 2010. Pleuritis and suppurative pneumonia associated with a hypermucoviscosity phenotype of Klebsiella pneumoniae in California sea lions (*Zalophus californianus*). Veterinary Microbiology 141: 174-177.

Johengen, T.J., Reid, D., Fahnenstiel, G., MacIsaac, H., Dobbs, F.C., Doblin, M.A. Jenkins, P.T. 2005. Assessment of transoceanic NOBOB vessels and low-salinity ballast water as vectors for nonindigenous species introductions to the Great Lakes. Final report for the project to the Great Lakes Protection Fund, the National Oceanic and Atmospheric Administration, the U.S. Environmental Protection, and the U.S. Coast Guard.

Kennedy, S. 1998. Morbillivirus infections in aquatic mammals. Journal of Comparative Pathology 119: 201-225.

Kennedy, S. Kuiken, T. Jepson, P.D. Deaville, R. Forsyth, M. Barret, T. Van de Bildt, MW.G. Osterhaus, A.D.M.E. Eybatov, T. Duck, C. Kydyrmanov, I. Mitrofanov and S. Wilson. 2000. Mass die-off of Caspian Seals caused by Canine Distemper Virus. Emerging Infectious Diseases 6(6): 637-639.

Kent, M.L., Traxler, G.S., Kieser, D., Richard, J., Dawe, S.C., Shaw, R.W., Prosperi-Porta, G., Ketcheson, J. and T. Evelyn. 1988. Survey of salmonid pathogens in ocean-caught fishes in British Canada. Journal of Aquatic Animal Health 10: 211-219.

Kent, M.L., Traxler, G.S., Kieser, D., Richard, J. at. al. 1998. Survey of salmonid pathogens in ocean-caught fishes in British Columbia, Canada. Journal of Aquatic Animal Health 10: 211-219.

Kenworthy, W.J. 1997. An Updated Biological Status Review and Summary of the Proceedings of a Workshop to Review the Biological Status of the Seagrass, *Halophila johnsonii* Eiseman. Office of Protected Resources, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Silver Spring, MD.

Kenworthy, W.J., S. Norton, S. harter and J.B. Landry. 2007. 5-Year Review–Johnson's Seagrass (*Halophila johnsonii* Eiseman). National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Silver Spring, MD.

Kim, S.U. and S.I. Park. 2004. Detection of viral hemorrhagic septicemia virus (VHSV) in wild marine fishes in the coastal region of Korea. Journal of Fish Pathology 17(1): 1-10.

Kim, W.S. S.R. Kim, D. Kim, J.O. Kim, M.A. Park, S.I. Kitamura, H.Y. Kim, D.H. Kim, H.J. Han, S.J. Jung and M.J. Oh. 2009. An outbreak of VHSV (viral hemorrhagic septicemia virus) infection in farmed olive flounder *Paralichthys olivaceus* in Korea. Aquaculture 296(1-2): 165-168.

King, J.A., M. Snow, D.A. Smail and R.S. Raynard. 2001. Distribution of viral haemorrhagic septicaemia virus in wild fish species of the North Sea, north east Atlantic Ocean and Irish Sea. Diseases of Aquatic Organisms 47: 81-86.

Kipp, R.M. and A. Ricciardi. 2006. NOAA – Viral Hemorrhagic Septicemia (VHS) Fact Sheet. GLANSIS.

Kocan R.M., P.K. Hershberger, N.E. Elder and J.R. Winton. 2001. Survival of the North American strain of viral hemorrhagic septicemia virus (VHSV) in filtered seawater and seawater containing ovarian fluid, crude oil and serum-enriched culture medium. Diseases of Aquatic Organisms 44: 75–78.

Kohlmeyer, J. 1971. Fungi from the Sargasso Sea. Marine Biology 8(4): 344-350.

Kuris A.M. and C.S. Culver. 1999. An introduced sabellid polychaete pest infesting cultured abalones and its potential spread to other California gastropods. Invertebrate Biology 118: 391-403.

Kushmaro, A., Loya, Y., Fine, M. and E. Rosenberg. 1996. Bacterial infection and coral bleaching. Nature 380: 396.

Kushmaro, A., Banin, E., Loya, Y., Stackebrandt E. and E. Rosenberg. 2001. *Vibrio shiloi* sp. nov., the causative agent of bleaching of the coral *Oculina patagonica*. International Journal of Systematic and Evolutionary Microbiology 51(4): 1383-1388.

Lafferty, K.D. and A.M. Kuris. 1993. Mass mortality of abalone *Haliotis cracherodii* in the California Channel Islands: tests of epidemiological hypothesis. Marine Ecology Progress Series 96: 239-248.

Langdon, J.S. 1989. Experimental transmission and pathogenicity of epizootic haematopoietic necrosis virus (EHNV) in redfin perch, *Perca fluviatilis* L., and 11 other teleosts. Journal of Fish Diseases 12: 295-310.

Lannan, C.N. and J.L. Fryer. 1994. Extracellular survival of *Piscirickettsia salmonis*. Journal of Fish Diseases 17: 545-548.

Lee II, H., D.A. Reusser, M. Frazier and G. Ruiz. 2010. Density Matters: Review of Approaches to Setting Organism-Based Ballast Water Discharge Standards. U.S. Environmental Protection Agency, Office of Research and Development, National Health and Environmental Effects Research Laboratory, Western Ecology Division (EPA/600/R-10/031).

Lilly, E.L., D.M. Kulis, P. Gentien and D.M. Anderson. 2001. Paralytic shellfish poisoning toxins in France linked to a human-introduced strain of *Alexandrium catenella* from the western Pacific: evidence from DNA and toxin analysis. J. Plankton Res. 24:443–452.

Lipp, E.K., A. Huq and R.R. Colwell. 2002. Effects of global climate on infectious disease: the cholera model. Clinical Microbiology Reviews 15(4): 757–770.

Lipscomb, T.P., F.Y Schulman, D. Moffett and S. Kennedy. 1994. Morbilliviral disease in Atlantic bottlenose dolphins (Tursiops truncatus) from the 1987-1988 epizootic. Journal of Wildlife Diseases 30(4): 567-571.

Lipscomb, T.P., S. Kennedy, D. Moffett, A. Krafft, B.A. Klaunberg, J.H. Lichy, G.T. Regan, G.A.J. Worthy and J.K. Taubenberger. 1996. Morbilliviral epizootic in bottlenose dolphins of the Gulf of Mexico. J. Vet. Diagn. Invest. 8: 283-290.

Lipson, S.M. and G. Stotzky. 1984. Effect of proteins on reovirus adsorption to clay minerals. Applied and Environmental Microbiology 48: 525-530.

Lockwood, P. 1999. Presentation to the Pacific Coast Ballast Water Group, June 17, 1999, at the Port of Oakland, Oakland, CA.

Lumsden, J.S. 2005. A mortality event in freshwater drum (*Aplodinotus grunniens*) from Lake Ontario, associated with viral hemorrhagic septicemia virus (VHSV), type IV. Wildlife Health Centre Newsletter (Canadian Cooperative Wildlife Health Centre) 11(1): 10.

Luna, G.M., F. Biavasco and R. Danovaro. 2007. Bacteria associated with the rapid tissue necrosis of stony corals. Environmental Microbiology 9(7): 1851-1857.

Luna, G.M., Bongiorni, L., Gili, C., Biavasco, F. and R. Danovaro. 2010. *Vibrio harveyi* as a causative agent of the White Syndrome in tropical stony corals. Environmental Microbiology Reports 2(1): 120-127.

Lyles, K. 2004. How accurate is ballast water reporting? Presentation at the 2nd International Conference & Exhibition on Ballast Water Management, 19-21 May, 2004, Singapore. Institute of Environmental Science and Engineering, Singapore.

MacLeod, L.A., Raynard, R.S., Murray, A.G. and Gregory, A. 2003. Survival of infectious salmon anaemia virus (ISAV) in aquatic environments. Abstract from the 11th EAFP International Conference: Diseases of Fish and Shellfish, Malta.

Mamaev, L.V., Visser, I.K.G. Belikov, S.I., Denikina, N.N., Harder, T., Goatley, L., Rima, B., Edginton, B., Osterhaus, A.D.M.E. and T. Barrett. 1996. Canine distemper virus in Lake Baikal seals (Phoca sibirica). Veterinary Record 138: 437-439.

Martinelli Filho, J.E., R.M. Lopes, I.N.C. Rivera and R.R. Colwell. 2010. Vibrio cholerae O1 detection in estuarine and coastal zooplankton. Journal of Plankton Research doi:10.1093/plankt/fbq093.

McCarthy, S.A., Khambaty, F.M., 1994. International dissemination of epidemic *Vibrio cholerae* by cargo ship ballast and other nonpotable waters. Applied and Environmental Microbiology 60: 2597–2601.

McCarthy, S.A., J.L. Gaines , R.M. McPhearson and A.M. Guarino. 1992. Toxigenic *Vibrio cholerae* O1 and cargo ships entering Gulf of Mexico. Lancet 339: 624–625.

McMinn, A., G.M. Hallegraeff, P. Thomson, A.V. Jenkinson and H. Heijnis. 1997. Cyst and radionucleotide evidence for the recent introduction of the toxic dinoflagellate *Gymnodinium catenatum* into Tasmanian waters. Marine Ecology Progress Series 161: 165-172.

Meier, W., M. Schmitt and T. Wahli. 1994. Viral hemorrhagic septicemia (VHS) of nonsalmonids. Annual Review of Fish Diseases 4: 359-373.

MEPC. 2003. Harmful Aquatic Organisms in Ballast Water: Comments on Draft regulation E-2: Concentrations of Organisms Delivered in Ships' Ballast Water in the Absence of any Treatment: Establishing a Baseline for Consideration of Treatment Efficacy. MEPC 49/2/21, 49th Session, Agenda Item 2, 23 May 2003. Marine Environment Protection Committee, International Maritime Organization, London.

Meyers, T.R., Short, S. and K. Lipson. 1999. Isolation of the North American strain of Viral Hemorrhagic Septicemia Virus (VHSV) associated with epizootic mortality in two new host species of Alaskan marine species. Diseases of Aquatic Organisms 38: 81-86.

Meyers, T.R., Short, S., Lipson, K., Batts, W.N., Winton, J.R. Wilcock, J. and E. Brown. 1994. Association of viral hemorrhagic septicemia virus with epizootic hemorrhages of the skin in Pacific herring *Clupea harengus pallasi* from Prince William Sound and Kodiak Island, Alaska, USA. Diseases of Aquatic Organisms 19: 27-37.

Meyers, T.R. and J.R. Winton. 1995. Viral hemorrhagic septicemia virus in North America. *Annual Review of Fish Diseases* 5: 3-24.

Michigan DNR. 2009. Viral Hemorrhagic Septicemia (VHS) Briefing Paper. Michigan Department of Natural Resources – Fisheries Division.

Michigan Sea Grant. 2007. Viral Hemorrhagic Septicemia (VHS) in the Great Lakes. Michigan Sea Grant College Program, University of Michigan, Ann Arbor, MI and Michigan State University, East Lansing, MI.

Miller, A.W., K. Lion, M.S. Minton and G.M. Ruiz. 2007. Status and Trends of Ballast Water Management in the United States. Third Biennial Report of the National Ballast Information Clearinghouse. Smithsonian Environmental Research Center, Edgewater, MD.

Mo, T.A. 1994. Status of *Gyrodactylus salaris* problems and research in Norway. Pages 43-56 in: Parasitic Diseases of Fish, A.W. Pike and J.W. Lewis (eds.), Samara Press, Dyfed, Great Britain.

Moeller, R.B. 2002. Diseases of Marine Mammals. Syllabus from the C. L. Davis Foundation. [Online] http://www.cldavis.org/cgi-bin/download.cgi?pid=178

Moore, J.D. and T.T. Robbins. 2000. Withering syndrome in farmed red abalone *Haliotis rufescens*: thermal induction and association with a gastro-intestinal rickettsiales-like prokaryote. Journal of Aquatic Animal Health 12: 26-34.

Moore, J.D., C.A. Finley, T.T. Robbins and C.S. Friedman. 2002. Withering syndrome and restoration of southern California abalone populations. CalCOFI Reports 43: 112-117.

Mori, K., Iida, H., Nishizawa, T., Arimoto, M., Nakajima, K. and Muroga, K. 2002. Properties of viral haemorrhagic septicaemia virus (VHSV) isolated from Japanese flounder (*Paralichthys olivaceus*). Fish Pathology 37: 169-174.

Muehlstein, L.K., D. Porter and F.T. Short. 1988. *Labyrinthula* sp., a marine slime mold producing the symptoms of wasting disease in eelgrass, *Zostera marina*. Marine Biology 99: 465-472.

Muehlstein, L.K., D. Porter and F.T. Short. 1991. *Labyrinthula zosterae* sp. nov., the causative agent of wasting disease of eelgrass, *Zostera marina*. Mycologia 83: 180-191.

Mulcahy, D., Klaybor, D. and W.N. Batts. 1990. Isolation of infectious hematopoietic necrosis virus from a leech (*Piscicola salmositica*) and a copepod (*Salmincola* sp.), ectoparasites of sockeye salmon *Oncorhynchus nerka*. Diseases of Aquatic Organisms 8: 29-34.

Murray, A.G., R.J. Smith and R.M. Stagg. 2002. Shipping and the Spread of Infectious Salmon Anemia in Scottish Aquaculture. Emerging Infectious Diseases 8(1): 1-5.

Nagasawa, K. 1999. Parasites of Pinnipeds (Mammalia: Carnivora). In: Japan: Checklist and Bibliography. Bulletin of the National Research Institute of Far Seas Fisheries 36: 27-32.

NBIC 2008. National Ballast Information Clearinghouse Online Database. Electronic publication, Smithsonian Environmental Research Center and United States Coast Guard. Available from http://invasions.si.edu/nbic/search.html; searched 5/3/2010.

New York Invasive Species Clearinghouse. 2008. Viral Hemorrhagic Septicemia (VHS). [Online]

http://nyis.info/pathogens/ViralHemorrhagicSepticemia.aspx

Nielsen, O., R.E.A. Stewart, L. Measures, P. Duignan and C. House. 2000. A morbillivirus antibody survey of Atlantic walrus, narwhal and beluga in Canada. Journal of Wildlife Diseases, 36(3): 508-517.

Nishizawa, T., Iida, H., Takano, R., Isshiki, T.K., Nakajima, K. and K. Muroga. 2002. Genetic relatedness among Japanese, American and European isolates of viral hemorrhagic septicemia virus (VHSV) based on partial G and P genes. Diseases of Aquatic Organisms 48: 143-148.

Nishizawa, T, Kinoshita, S., Kim, W.S., Higashi, S. and M. Yoshimizu. 2006. Nucleotide diversity of Japanese isolates of infectious hematopoietic necrosis virus (IHNV) based on the glycoprotein gene. Diseases of Aquatic Organisms 71: 267-272.

NMFS. 2002. Recovery Plan for Johnson's Seagrass (*Halophila johnsonii*). Prepared by the Johnson's Seagrass Recovery Team for the National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Silver Spring, MD.

NMFS. 2007. Recovery Plan for the Hawaiian Monk Seal (*Monachus schauinslandi*)–Revision. National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Honolulu, HI.

NOAA. 2004. Endangered and Threatened Species: Proposed Listing Determinations for 27 ESUs of West Coast Salmonids. National Oceanic and Atmospheric Administration, Washington, DC. Federal Register 69 (113): 33102-33179.

NOAA. 2005. Endangered and Threatened Species: Final Listing Determinations for 16 ESUs of West Coast Salmon, and Final 4(d) Protective Regulations for Threatened Salmonid ESUs. National Oceanic and Atmospheric Administration, Washington, DC. Federal Register 70 (123): 37160-37204.

NOAA. 2010a. Endangered and Threatened Species Under NMFS' Jurisdiction (Last Updated March 18, 2010). National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Office of Protected Resources, Washington, DC.

NOAA. 2010b. Species descriptions. National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Office of Protected Resources, Washington, DC. [Online] http://www.nmfs.noaa.gov/pr/species/esa

NOAA. 2010c. Threats to Marine Turtles. National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Office of Protected Resources, Washington, DC. [Online] http://www.nmfs.noaa.gov/pr/species/turtles/threats.htm

NOAA. 2010d. Pacific Salmonids: Major Threats and Impacts. National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Office of Protected Resources, Washington, DC. [Online] http://www.nmfs.noaa.gov/pr/species/fish/salmon.htm

NOAA. 2010e. Coral Reef Diseases. National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Office of Protected Resources, Washington, DC. [Online] http://www.nmfs.noaa.gov/habitat/ead/disease.htm

NOAA. 2010f. Coral Health and Monitoring Program. National Oceanic and Atmospheric Administration, Atlantic Oceanographic and Meteorological Laboratory, Ocean Chemistry Division. [Online] http://www.coral.noaa.gov/

NPS. 2008. Emergency Prevention and Response Plan For Viral Hemorrhagic Septicemia. National Park Service, Lake Superior Basin National Parks; and the Grand Portage Band of Lake Superior Chippewa, Natural Resources Management.

Nylund, A.S., T. Hovland, K. Hodneland, F. Nilsen and P. Lovik. 1994. Mechanisms for transmission of infectious salmon anemia (ISA). Diseases of Aquatic Organisms 19: 95-100.

Nylund, A., Devold, M., Mullins, J. and H. Plarre. 2002. Herring (*Clupea harengus*): A host for infectious salmon anemia virus (ISAV). Bull. Eur. Ass. Pathology 22(5):.311-318.

Nylund, A., Devold, M., Plarre, H., Isdal, E. and M. Arseth. 2003. Emergence and maintenance of infectious salmon anaemia virus (ISAV) in Europe: A new hypothesis. Diseases of Aquatic Organisms 56: 11-24.

Ohashi, K. and C. Kai. 2000. Morbillivirus infections in wildlife of Japan. Journal of the Veterinary Medicine 53: 834-838. [in Japanese]

OIE. 2009. Manual of Diagnostic Tests for Aquatic Animals 2009. Office International des Epizooties, World Organization for Animal Health. [Online] http://www.oie.int/eng/normes/fmanual/A_summry.htm?e1d11

Olivier, G. 2002. Disease interactions between wild and cultured fish - perspectives from the American Northeast (Atlantic Provinces). Bulletin of the European Association of Fish Pathologists 22: 103-109.

Orpurt, P.A.; Meyers, S.P.; Boral, L.L.; Sims, J. 1964. Thalassiomycetes V. a New Species of Lindra from Turtle Grass, Thalassia Testudinum König. Bulletin of Marine Science 14(3): 405-417.

Orr, R.T. and R.C. Helm. 1989. Marine Mammals of California. California Natural History Guides, University of California Press, Berkeley, CA.

Osterhaus, A.D.M.E. and E.J. Vedder. 1988. Identification of virus causing recent seal deaths. Nature 335: 20.

Osterhaus A.D.M.E., Groen, J., DeVries, P., Uytdehaag, F.G.C.M., Klingeborn, B. and R. Zarnke. 1988. Canine distemper virus in seals. Nature 335: 403-404.

Osterhaus, A.D.M.E. Groen, J. Spijkers, H.E.M. Broeders, H.W.J. UytdeHaag, F.G.C.M. De Vries, P. Teppema, J.S. Visser, I.K.G. Van de Bildt, M.W.G and E.J. Vedder. 1990. Mass mortality in seals caused by a newly discovered virus-like morbillivirus. Veterinary Microbiology 23(1-4): 343-350.

Osterhaus, A.D.M.E. Visser, I.K.G. Swart, R.L. de Van Bressem, M.F. Van de Bildt M.W.G. Barrett, T. Raga, J.A. 1992. Morbillivirus threat to Mediterranean monk seals? Veterinary Record 130:141-142.

Osterhaus, A.D.M.E., R.L. de Swart, H.W. Vos, P.S. Ross, M..H. Kenter and T. Barrett. 1995. Morbillivirus infections of aquatic mammals: newly identified members of the genus. Veterinary Microbiology 44(2-4):219-27.

Osterhaus, A.D.M.E. Groen, J. Niesters, H. Van de Bildt, M. Martina, B. Vedder, L. Vos, J. Van Egmond, H. Abou-Sidi, B. Barham and M.E. Ould. 1997. Morbillivirus in monk seal mass mortality. Nature 388: 838-839.

Osterhaus, A.D.M.E., M. van de Bildt. L. Vedder, B. Martina, H. Niesters, J. Vos, H. van Egmond, D. Liem, R. Baumann, E. Androukaki, S. Kotomatas, A Komnenou. B. Abou Sidie, A. Bent Jiddoue and M.E.O. Barhame. 1998. Monk seal mortality: virus or toxin? Vaccine 16(9-10): 979-981.

Overstreet, R.M., J. Jovonovich and H. Ma. 2009. Parasitic crustaceans as vectors of viruses, with an emphasis on three penaeid viruses. Integrative and Comparative Biology 49(2): 127-141.

PAHW. 2007. Possible Vector Species and Live Stages of Susceptible Species not Transmitting Disease as Regards Certain Fish Diseases: Scientific Opinion of the Panel on Animal Health and Welfare. The EFSA (European Food Safety Authority) Journal 584: 1-163.

Parry L. and P.F. Dixon. 1997. Stability of nine viral haemorrhagic septicaemia virus (VHSV) isolates in seawater. Bull. Eur. Assoc. Fish Pathol. 17: 31–36.

Patterson, K.L., Porter, J.W., Ritchie, K.B., Polson, S.W., Mueller, E., Peters, E.C., Santavy, D.L. and G.W. Smith. 2002. The etiology of white pox, a lethal disease of the caribbean elkhorn coral, *Acropora palmata*. Proceedings of the National Academy of Sciences 99(13): 8725-8730.

Pomeroy, P.P., J.A. Hammond, A.J. Hall, M. Lonergan, C.D. Duck, V.J. Smith and H. Thompson. 2005. Morbillivirus neutralising antibodies in Scottish grey seals *Halichoerus grypus*: assessing the effects of the 1988 and 2002 PDV epizootics. Marine Ecology Progress Series 287: 241-250.

Quackenbush, S.L., T.M. Work, G.H. Balazs, R.N. Casey, J. Rovnak, A. Chaves, L. duToit, J.D. Baines, C.R. Parrish, P.R. Bowser and J.W. Casey. 1998. Three closely related herpesviruses are associated with fibropapillomatosis in marine turtles. Virology 246: 392-399.

Quackenbush, S.L., R.N. Casey, R.J. Murcek, T.A. Paul, T.M. Work, C.J. Limpus, A. Chaves, L. Dutoit, J.V. Perez, A.A. Aguirre, T.R. Spraker, J.A. Horrocks, L.A. Vermeer, G.H. Balazs and J.V. Casey. 2001. Quantitative analysis of herpesvirus sequences from normal tissue and fibropapillomas of marine turtles with real-time PCR. Virology 1287(1): 105-111.

Raimondi, P.T., C.M. Wilson, R.F. Ambrose, J.M. Engle and T.E. Minchinton. 2002. Continued declines of black abalone along the coast of California : Are mass mortalities related to El Niño events? *Marine Ecology Progress Series* 242: 143-152.

Raynard, R., T. Wahli, I. Vatsos and S. Mortensen. 2007. Review of disease interactions and pathogen exchange between farmed and wild finfish and shellfish in Europe. Produced for the European Commission Framework Program. Veterinærmedisinsk Oppdragssenter AS.

Reid, H.I., A.A. Griffen and T.H. Birkbeck. 2004. Isolates of *Piscirickettsia salmonis* from Scotland and Ireland show evidence of clonal diversity. Applied Environmental Microbiology 70: 4393-4397.

Reidarson, T.H., McBain, J., House, C., King, D.P., Stott, J.L., Krafft, A., Taubenberger, J.K., Heyning, J. and T.P. Lipscomb. 1998. Morbillivirus infection in stranded common dolphins from the Pacific Ocean. Journal of Wildlife Diseases 34: 771-776.

Renault, T., Stokes, N.A., Chollet, B., Cochennec, N., Berthe, F., Gérard, A. and E.M. Burreson. 2000. Haplosporidiosis in the Pacific oyster *Crassostrea gigas* from the French Atlantic coast. Diseases of Aquatic Organisms 42: 207-214.

Richards, D.V. and G.E. Davis. 1993. Early warnings of modern population collapse in black abalone, *Haliotis cracherodii* Leach, 1814, at the California Channel Islands. Journal of Shellfish Research 12(2): 189-194.

Richardson, L.L. 1992. Red band disease: a new cyanobacterial infestation of corals. Pages 153-160 in: Diving for Science–1992, L.B. Calhoun (ed.), Proceedings of the Twelfth Annual Scientifc Diving Symposium of the American Academy of Underwater Sciences, September 24-27, 1992, University of North Carolina, Wilmington, NC.

Richardson, L.L., W.M. Goldberg, K.G. Kuta, R.B. Aronson, G.W. Smith, K.B. Ritchie, J.C. Halas, J.S. Feingold and S.L. Miller. 1998. Florida's mystery coral-killer identified. Nature 392: 557-558.

Robblee, M.B., Barber, T.R., Carlson, P.R., Jr., Durako, M.J., Fourqurean, J.W., Muehlstein, L.K., Porter, D., Yarbro, L.A., Zieman, R.T. and J.C. Zieman. 1991. Mass mortality of the tropical seagrass *Thalassia testudinum* in Florida Bay (USA). Marine Ecology Progress Series 71(3): 297-299.

Rosenberg, E. and Y. Ben-Haim. 2002. Microbial diseases of corals and global warming. Environmental Microbiology 4(6): 318-326.

Rosenberg, E., C.A. Kellogg and F. Rohwer. 2007. Coral Microbiology. Oceanography 20(2): 146-154.

Ross, C., M.P. Puglisi and V.J. Paul. 2008. Antifungal defenses of seagrasses from the Indian River Lagoon, Florida. Aquatic Botany 88: 134-141.

Rudakova, S.L., Kurath, G. and E.V. Bochkova. 2007. Occurrence and genetic typing of infectious hematopoietic necrosis virus in Kamchatka, Russia. Diseases of Aquatic Organisms 75: 1-11.

Ruetzler, K. and D.L. Santavy. 1983. The black band disease of Atlantic reef corals. I. Description of the cyanophyte pathogen. Marine Ecology 4: 301-319.

Ruiz, G.M. and A.H. Hines. 1997. The Risk of Nonindigenous Species Invasion in Prince William Sound Associated with Oil Tanker Traffic and Ballast Water Management: Pilot Study. Report to Regional Citizen's Advisory Committee of Prince William Sound, Valdez AK.

Ruiz, G.M., K. Rawlings , F.C. Dobbs, L.A. Drake, T. Mullady, A. Huq and R.R. Colwell. 2000. Global spread of microorganisms by ships. Nature 408: 49-50.

Shimizu, T., Yoshida, N., Kasai, H. and Yoshimizu M. 2006. Survival of koi herpesvirus (KHV) in environmental water. Fish Pathology 41: 153-157.

Shivappa, R.B, Song, H., Yao, K., Aas-Eng, A., Evensen, O. and V.N. Vakharia. 2004. Molecular characterization of Sp serotype strains of infectious pancreatic necrosis virus exhibiting differences in virulence. Diseases of Aquatic Organisms 61: 23-32.

Short, F.T., L.K. Muelstein and D. Porter. 1987. Eelgrass Wasting Disease: cause and recurrence of a marine epidemic. Biological Bulletin 173: 557-562.

Short, F.T., B.W. Ibelings and C. den Hartog. 1988. Comparison of a current eelgrass disease to the wasting disease in the 1930's. Aquatic Botany 30: 295-304.

Sinclair R., S.A. Boone, D. Greenberg, P. Keim and C.P. Gerba. 2008. Persistence of Category A Select Agents in the Environment, Applied and Environmental Microbiology 74(3): 555-563.

Skall, H.F., N.J. Olesen and S. Mellergaard. 2005. Viral hemorrhagic septicaemia virus in marine fish and its implications for fish farming – a review. Journal of Fish Diseases 28: 509-529.

Smail, D.A. 2000. Isolation and identification of viral haemorrhagic septicaemia (VHS) viruses from cod *Gadus morhua* with the ulcus syndrome and from haddock *Melanogrammus aeglefinus* having skin haemorrhages in the North Sea. *Diseases of Aquatic Organisms* 41: 231-235.

Smith, G., C. Stamm, C. and F. Petrovic. 2003. Haliotis cracherodii. In: IUCN Red List of Threatened Species. Version 2010.3. [Online] http://www.iucnredlist.org/apps/redlist/details/41880/0

Snow, M., Bain, N., Black, J., Taupin, V., Cunningham, C.O., King, J.A., Skall, H.F., Raynard, R.S., 2004. Genetic population structure of marine viral haemorrhagic septicaemia virus (VHSV). *Diseases of Aquatic Organisms* 61: 11–21.

Stacy B.A., Wellehan J.F., Foley A.M., Coberley S.S., Herbst L.H., Manire C.A., Garner M.M., Brookins M.D., Childress A.L. and E.R. Jacobson. 2007. Two herpesviruses associated with disease in wild Atlantic loggerhead sea turtles (*Caretta caretta*). Veterinary Microbiology 126(1-3): 63-73.

Stanley, H.F., S. Casey, J.M. Carnahan, S. Goodman, J. Harwood and R.K. Wayne. 1996. Worldwide patterns of mitochondrial DNA differentiation in the harbor seal (*Phoca vitulina*). Molecular Biology and Evolution 13: 368-382.

Steele, L., M. Caldwell, A. Boettcher and T. Arnold. 2005. Seagrass-pathogen interactions: 'pseudo-induction' of turtlegrass phenolics near wasting disease lesions. Marine Ecology 303: 123-131.

Subba Rao, D.V., Sprules, W.G., Locke, A. and J.T. Carlton. 2004. Exotic phytoplankton from ships' ballast waters: risk of potential spread to mariculture sites on Canada's east coast. Canadian Data Report on Fisheries and Aquatic Sciences No. 937. Department of Fisheries and Oceans, Bedford Institute of Oceanography, Darmouth, Nova Scotia.

Sussman, M. 2009. Coral disease pathogens of the Indo-Pacific Ocean. Ph.D. dissertation, James Cook University, School of Marine and Tropical Biology, Toensville, Australia.

Sussman, M., Y. Loya, M. Fine and E. Rosenberg. 2003. The marine fireworm *Hermodice carunculata* is a winter reservoir and spring-summer vector for the coral-bleaching pathogen *Vibrio shiloi.* Environmental Microbiology 5: 250-255.

Sussman, M., B.L. Willis, S. Victor and D.G. Bourne. 2008. Coral pathogens identified for White Syndrome (WS) epizootics in the Indo-Pacific. PLoS One 3(6): e2393.

Sutherland, K.P. and K.B. Ritchie. 2004. White pox disease of the Caribbean Elkhorn coral, *Acropora palmata*. Pages 289-297 in: Coral Health and Disease, E. Rosenberg and Y. Loya (eds.), Springer-Verlag, Berlin.

Taubenberger, J.K., M.M. Tsai, T.J. Atkin, T.G. Fanning, A.E. Krafft, R.B. Moeller, S.E. Kodsi, M.G. Mense and T.P. Lipscomb. 2000. Molecular genetic evidence of a novel morbillivirus in a long-finned pilot whale (*Globicephalus melas*). Emerging Infectious Diseases 6(1): 42-45.

Tauxe, R.V., E.D. Mintz, and R.E. Quick. 1995. Epidemic cholera in the new world: translating field epidemiology into new prevention strategies. Emerging Infectious Diseases 1: 141-146.

Thomson, F.K. 2009. Characterization of antibiotic resistance in *Vibrio cholerae* isolated from ships' ballast and other environmental sources. Ph.D. Dissertation in Oceanography, Old Dominion University, Norfolk, VA.

Thomson, F.K., S.A. Heinemann and F.C. Dobbs. 2003. Patterns of antibiotic resistance in cholera bacteria isolated from ships' ballast water. Page 118 in: Proceedings of the Third International Conference on Marine Bioinvasions, March 16-19, 2003, La Jolla, CA.

Thompson, F.L., Y. Barash, T. Sawabe, G. Sharon, J. Swings and E. Rosenberg. 2006. *Thalassomonas loyana* sp. nov., a causative agent of the white plague-like disease of corals on the Eilat coral reef. International Journal of Systematic and Evolutionary Microbiology 56: 365-368.

Tibbetts, J. 1997. Exotic invasion. Environmental Health Perspectives 105(6): 590-593.

Toranzo, A.E. and Hetrick, F.M. 1982. Comparative stability of two salmonid viruses and poliovirus in fresh, estuarine and marine waters. Journal of Fish Diseases 5: 223-231.

Toranzo, A. E., B. Magarinos and J.L. Romalde. 2005. A review of the main bacterial fish diseases in mariculture systems. Aquaculture 246: 37-61.

Traxler, G.S. and G.R. Bell. 1998. Pathogens associated with impounded Pacific herring *Clupea harengus pallasi*, with emphasis on viral erythrocytic necrosis (VEN) and atypical *Aeromonas salmonicida*. Diseases of Aquatic Organisms 5: 93-100.

Trukhin, A.M. 2009. Current status of pinnipeds in the Sea of Okhotsk. Pages 82-89 in: Proceedings of the Fourth Workshop on the Okhotsk Sea and Adjacent Areas. PICES Scientific Report No. 36.

UNEP. 2010. Field Guide to Western Atlantic Coral Diseases and Other Causes of Coral Mortality. United Nations Environmental Programme, World Conservation Monitoring Centre, Marine and Coastal Programme, Cambridge, U.K. [Online] http://www.unep-wcmc.org/GIS/coraldis/cd/index.htm

USGS. 2010. Marine Turtles. U.S. Geological Survey, Honolulu Field Station. [Online] http://www.nwhc.usgs.gov/hfs/Turtles.htm

Van Banning, P. 1987. Further results of the *Bonamia ostreae* challenge tests in Dutch oyster culture. Aquaculture 67(1-2): 191-194.

Van Bressem, M.F., K. Van Waerebeek, M. Fleming and T. Barrett. 1998. Serological evidence of morbillivirus infection in small cetaceans from the Southeast Pacific. Veterinary Microbiology 59(2-3): 89-98.

Van Bressem, M.F., K. Van Waerebeek and J.A. Raga. 1999. A review of virus infections of cetaceans and the potential impact of morbilliviruses, poxviruses, and papillomaviruses on host population dynamics. Diseases of Aquatic Organisms 38: 53-65.

Van Bressom, M., K.V. Waerebeek, P.D. Jepson, J.A. Raga, P.J. Duignan *et al.* 2001. An insight into the epidemiology of dolphin morbillivirus worldwide. Veterinary Microbiology 81(4): 287-304.

Van Bressem, M.F., Raga, J.A., di Guardo, G., Jepson, P., Duignan, P., Siebert, U., Barrett, T., Santos, M.C.d.O, Moreno, I., Siciliano, S., Aguilar, A. and K. Van Waerebeek. 2008. Emerging and recurring diseases in cetaceans worldwide and the role of environmental stressors. Paper SC/60/DW5 presented to the International Whaling Commission Scientific Committee, May 2008. http://iwcoffice.org/_documents/sci_com/SC60docs/SC-60-DW5.pdf

Van Bressem, M.F., J.A. Raga, G. Di Guardo, P.D. Jepson, P.J. Duignan, U. Siebert, T. Barrett, M.C. de Oliveira Santos, I.B Moreno, S. Siciliano, A. Aguilar and K. Van Waerebeek. 2009. Emerging infectious diseases in cetaceans worldwide and the possible role of environmental stressors. Diseases Of Aquatic Organisms 86(2): 143-157.

Van de Bildt, M.W.G, T. Kuiken and A.D.M.E. Osterhaus. 2005. Cetacean morbilliviruses are phylogenetically divergent. Archives of Virology 150(3): 577-583.

Visser, I.K.G., V.P. Kumarev, C. Örvell, P. de Vries, H.W.J. Broeders, M.W.G. van de Bildt, J. Groen, J.S. Teppema, M.C. Burger, F.G.C.M. UytdeHaag and A.D.M.E. Osterhaus. 1990. Comparison of two morbilliviruses isolated from seals during outbreaks of distemper in North West Europe and Siberia. Archives of Virology 111(3-4): 149-164.

Visser, I.K., van Bressem, M.F., van de Bildt, M.W., Groen, J., Orvell, C., Raga J.A. and A.D. Osterhaus. 1993a. Prevalence of morbilliviruses among pinniped and cetacean species. Review of Science and Technology 12(1): 197-202.

Visser, I.K.G. Van Bressem, M.F. Swart, R.L. de Van de Bildt, M.W.G. Vos, H.W. Van der Heijden, R.W.J. Saliki J.T., Örvell, C. Kitching, P. Kuiken, T., Barrett, T. and A.D.M.E. Osterhaus. 1993b. Characterization of morbilliviruses isolated from dolphins and porpoises in Europe. Journal of General Virology 74: 631-641.

Visser, I.K.G., M.F. Van Bressem, T. Barrett and A.D.M.E. Osterhaus. 1993c. Morbillivirus infections in aquatic mammals. Veterinary Research 24: 169-178.

Walker, P.J. and J.R. Winton. 2010. Emerging viral diseases of fish and shrimp. Veterinary Research 41:51 doi: 10.1051/vetres/2010022.

Whelan, G.E. 2007. VHS—The Viral Invader. ANS Update (Aquatic Invasions News from the Great Lakes Commission) 13(1): 1.

Wikipedia. 2010. History of Pier 39 Sea Lions. [Online] http://en.wikipedia.org/wiki/Pier_39

Wilhelm, S.W., M.J. Carberry, M.L. Eldridge, L. Poorvin, M.A. Saxton and M.A. Doblin. 2006. Marine and Freshwater Cyanophages in a Laurentian Great Lake: Evidence from Infectivity Assays and Molecular Analyses of g20 Genes. Applied and Environmental Microbiology 72(7): 4957-4963.

Williams, E.H., Jr. and L. Bunkley-Williams. 1996. Fibropapillomas in Hawaiian sea turtles. Bishop Museum Occasional Papers 46: 46-49.

Williams, E.H., Jr. and L. Bunkley-Williams. 2006. Early fibropapillomas in Hawaii and occurrences in all sea turtle species: the panzootic, associated leeches wide-ranging on sea turtles, and species of study leeches should be identified. Journal of Virology 80(9): 4643.

Winton, J.R., Batts, W., Deering, R., Brunson, R., Hopper, K. and T. Nishizawa. 1991. Characteristics of the first North American isolates of viral hemorrhagic septicemia virus. Pages 43-50 in: Second International Symposium on Viruses of Lower Vertebrates, Oregon State University, Corvallis, OR.

Winton, J.R. and W. Batts. 2007. Viral Hemorrhagic Septicemia Virus in the Great Lakes. Slide presentation at Cool Water Egg Disinfection Workshop, August 9, 2007, University of Wisconsin Extension/Northern Aquaculture Demonstration Facility. http://www.uwsp.edu/cls/aquaculture/docs/projects/VHSv%20in%20the%20Great%20Lakes.pdf.

Winton, J., G. Kurath and W. Batts. 2008. Molecular Epidemiology of Viral Hemorrhagic Septicemia Virus in the Great Lakes *Region*. U.S. Geological Survey, Western Fisheries Research Center, Seattle, WA.

Wisconsin DNR. 2007. VHS Virus – Viral Hemorrhagic Septicemia. Wisconsin Department of Natural Resources, Madison, WI.

Wohlsein, P. C. Puff, M. Kreutzer, U. Siebert and W. Baumgärtner. 2007. Distemper in a Dolphin. Emerging Infectious Diseases 13(12): 1959-1961.

Wommack, K.E. and R.R. Colwell. 2000. Virioplankton: Viruses in Aquatic Ecosystems. Microbiol. Mol. Biol. Rev. 64(1):69-114.

Wonham, M.J., J.T. Carlton, G.M. Ruiz and L.D. Smith. 2000. Fish and ships: relating dispersal frequency to success in biological invasions. Marine Biology 136: 1111-1121.

Work, T., G. Balazs, M. Wolcott and R. Morris. 2003. Bacteraemia in free-ranging Hawaiian green turtles *Chelonia mydas* with fibropapillomatosis. Diseases of Aquatic Organisms 53: 41-46.

Work, T.M., J. Dagenais, G.H. Balazs, J. Schumacher, T.D. Lewis, J.C. Leong, R.N. Casey and J.W. Casey. 2009. In vitro biology of fibropapilloma-associated turtle herpesvirus and host cells in Hawaiian green turtles (*Chelonia mydas*). Journal of General Virology 90: 1943-1950.

Yang, W.C., V.F. Pang, C.R. Jeng, L.S. Chou and L.L. Chueh. 2006. Morbilliviral infection in a pygmy sperm whale (*Kogia breviceps*) from Taiwanese waters. Veterinary Microbiology 116: 69-76.

Yoshinaka, T., Yoshimizu, M. and Ezura, Y. 2000. Adsorption and infectivity of infectious hematopoietic necrosis virus (IHNV) with various solids. Journal of Aquatic Animal Health 12: 64-68.

Zarnke, RL, J.T. Saliki, A.P. Macmillan, S.D. Brew, C.E. Dawson, J.M. Ver Hoef, K.J. Frost and R.J. Small. 2006. Serologic survey for Brucella spp., phocid herpesvirus-1, phocid herpesvirus-2, and phocine distemper virus in harbor seals from Alaska, 1976–1999. Journal of Wildlife Disease 42: 290-300.

Zuerne, R.L., C.E. Cameron, S. Raverty, J. Robinson, K.M. Colegrove, S.A. Norman, D. Lambourn, S. Jeffries, D.P. Alt and F. Gulland. 2009. Geographical dissemination of *Leptospira interrogans* serovar Pomona during seasonal migration of California sea lions. Veterinary Microbiology 137: 105-110.

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Appendix 1. ESA-listed species under NMFS' jurisdiction that do not occur in U.S. waters

Habitat: M = Marine, E = Estuarine, F = Freshwater

Sources: NOAA 2010a,b.

Family	Common Name	Scientific Name	Listing Status	Habitat	Range
CETACEANS					
Balaenidae	Southern Right Whale	*Eubalaena australis*	Endangered	M	Southern hemisphere
Eschrichtiidae	Gray Whale, Western North Pacific DPS	*Eschrichtius robustus*	Endangered	ME	Western Pacific
Lipotidae	Chinese River Dolphin/Baiji	*Lipotes vexillifer*	Endangered	F	Yangtze River, China
Phocoenidae	Gulf of California Harbor Porpoise/Vaquita	*Phocoena sinus*	Endangered	ME	Gulf of California
Platanistidae	Indus River Dolphin	*Planista minor*	Endangered	EF	Indus River, Pakistan
PINNIPEDS					
Phocidae	Mediterranean Monk Seal	*Monachus monachus*	Endangered	M	Mediterranean Sea & East Atlantic Ocean
Phocidae	Saimaa Seal	*Phoca hispida saimensis*	Endangered	F	Lake Saimaa, Finland
Phocidae	Spotted Seal, Southern DPS	*Phoca largha*	Proposed	M	Northwestern Pacific
FISH					
Sciaenidae	Totoaba	*Totoaba macdonaldi*	Endangered	ME	Gulf of California

Appendix 2. ESA-listed species under NMFS' jurisdiction that occur in U.S. waters

Habitat: M = Marine, E = Estuarine, F = Freshwater

Sources: CBD 2009; NOAA 2010a,b.

Family	Common Name	Scientific Name	Listing Status	Habitat	Range
CETACEANS					
Balaenidae	Bowhead Whale	*Balaena mysticetus*	Endangered	M	Arctic
Balaenidae	North Atlantic Right Whale	*Eubalaena glacialis*	Endangered	M	North Atlantic
Balaenidae	North Pacific Right Whale	*Eubalaena japonica*	Endangered	M	North Pacific
Balaenopteridae	Blue Whale	*Balaenoptera musculus*	Endangered	M	worldwide
Balaenopteridae	Fin Whale	*Balaenoptera physalus*	Endangered	M	Atlantic, Pacific & Southern Oceans
Balaenopteridae	Humpback Whale	*Megaptera novaeangliae*	Endangered	M	worldwide
Balaenopteridae	Sei Whale	*Balaenoptera borealis*	Endangered	M	worldwide
Monodontidae	Beluga Whale, Cook Inlet DPS	*Delphinapterus leucas*	Endangered	MEF	Pacific Coast
Physeteridae	Sperm Whale	*Physeter macrocephalus*	Endangered	M	worldwide
Delphinidae	Killer Whale, Southern Resident DPS	*Orcinus orca*	Endangered	M	Pacific Coast
Delphinidae	False Killer Whale, Insular Hawaiian DPS	*Pseudorca crassidens*	Candidate	M	Hawaii
PINNIPEDS					
Otariidae	Guadalupe Fur Seal	*Arctocephalus townsendi*	Threatened	M	Pacific Coast
Otariidae	Stellar Sea Lion, Eastern DPS	*Eumetopias jubatus*	Threatened	M	Pacific Coast
Otariidae	Stellar Sea Lion, Western DPS	*Eumetopias jubatus*	Endangered	M	Western Pacific including western Aleutians
Phocidae	Hawaiian Monk Seal	*Monachus schauinslandi*	Endangered	M	Hawaii
Phocidae	Ringed Seal	*Phoca hispida*	Candidate	M	circumpolar north of 35°
Phocidae	Bearded Seal	*Erignathus barbatus*	Candidate	M	Arctic

Family	Common Name	Scientific Name	Listing Status	Habitat	Range
SEA TURTLES					
Cheloniidae	Green Turtle, Florida & Mexico's Pacific coast breeding colonies	*Chelonia mydas*	Endangered	ME	Atlantic, Gulf & Pacific Coasts
Cheloniidae	Green Turtle, all other areas	*Chelonia mydas*	Threatened	ME	worldwide in tropical/temperate waters
Cheloniidae	Hawksbill Turtle	*Eretmochelys imbricata*	Endangered	ME	worldwide in tropical waters
Cheloniidae	Kemp's Ridley Turtle	*Lepidochelys kempii*	Endangered	M	Atlantic & Gulf Coasts
Cheloniidae	Loggerhead Turtle	*Caretta caretta*	Threatened*	ME	worldwide in tropical/temperate waters
Cheloniidae	Olive Ridley Turtle, Mexico's Pacific coast breeding colonies	*Lepidochelys olivacea*	Endangered	ME	Pacific Coast
Cheloniidae	Olive Ridley Turtle, all other areas	*Lepidochelys olivacea*	Threatened	ME	worldwide in tropical waters
Dermochelyidae	Leatherback Turtle	*Dermochelys coriacea*	Endangered	M	worldwide in tropical/temperate waters
FISH					
Salmonidae	Atlantic Salmon, Gulf of Maine DPS	*Salmo salar*	Endangered	MEF	Atlantic Coast
Salmonidae	Chinook Salmon, California coastal ESU	*Oncorhynchus tshawytscha*	Threatened	MEF	Pacific Coast
Salmonidae	Chinook Salmon, Central Valley spring-run	*Oncorhynchus tshawytscha*	Threatened	MEF	Pacific Coast
Salmonidae	Chinook Salmon, Sacramento River winter-run	*Oncorhynchus tshawytscha*	Endangered	MEF	Pacific Coast
Salmonidae	Chinook Salmon, Lower Columbia River	*Oncorhynchus tshawytscha*	Threatened	MEF	Pacific Coast
Salmonidae	Chinook Salmon, Upper Columbia River spring-run	*Oncorhynchus tshawytscha*	Endangered	MEF	Pacific Coast
Salmonidae	Chinook Salmon, Upper Willamette River	*Oncorhynchus tshawytscha*	Threatened	MEF	Pacific Coast
Salmonidae	Chinook Salmon, Snake River fall-run	*Oncorhynchus tshawytscha*	Threatened	MEF	Pacific Coast
Salmonidae	Chinook Salmon, Snake River spring/summer-run	*Oncorhynchus tshawytscha*	Threatened	MEF	Pacific Coast

Family	Common Name	Scientific Name	Listing Status	Habitat	Range
FISH - continued					
Salmonidae	Chinook Salmon, Puget Sound	*Oncorhynchus tshawytscha*	Threatened	MEF	Pacific Coast
Salmonidae	Chum Salmon, Columbia River	*Oncorhynchus keta*	Threatened	MEF	Pacific Coast
Salmonidae	Chum Salmon, Hood Canal summer-run	*Oncorhynchus keta*	Threatened	MEF	Pacific Coast
Salmonidae	Coho Salmon, Central California coast	*Oncorhynchus kisutch*	Endangered	MEF	Pacific Coast
Salmonidae	Coho Salmon, Southern Oregon & Northern California coasts	*Oncorhynchus kisutch*	Threatened	MEF	Pacific Coast
Salmonidae	Coho Salmon, Oregon Coast	*Oncorhynchus kisutch*	Threatened	MEF	Pacific Coast
Salmonidae	Coho Salmon, Lower Columbia River	*Oncorhynchus kisutch*	Threatened	MEF	Pacific Coast
Salmonidae	Sockeye Salmon, Ozette Lake	*Oncorhynchus nerka*	Threatened	MEF	Pacific Coast
Salmonidae	Sockeye Salmon, Snake River	*Oncorhynchus nerka*	Endangered	MEF	Pacific Coast
Salmonidae	Steelhead Trout, Southern California	*Oncorhynchus mykiss*	Endangered	MEF	Pacific Coast
Salmonidae	Steelhead Trout, South-Central California coast	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast
Salmonidae	Steelhead Trout, Central California coast	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast
Salmonidae	Steelhead Trout, California Central Valley	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast
Salmonidae	Steelhead Trout, Northern California	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast
Salmonidae	Steelhead Trout, Lower Columbia River	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast
Salmonidae	Steelhead Trout, Middle Columbia River	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast
Salmonidae	Steelhead Trout, Upper Columbia River	*Oncorhynchus mykiss*	Endangered	MEF	Pacific Coast
Salmonidae	Steelhead Trout, Upper Willamette River	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast
Salmonidae	Steelhead Trout, Snake River basin	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast

Family	Common Name	Scientific Name	Listing Status	Habitat	Range
FISH - continued					
Salmonidae	Steelhead Trout, Puget Sound	*Oncorhynchus mykiss*	Threatened	MEF	Pacific Coast
Acipenseridae	Atlantic Sturgeon	*Acipenser oxyrinchus oxyrinchus*	Candidate	MEF	Atlantic Coast
Acipenseridae	Shortnose Sturgeon	*Acipenser brevirostrum*	Endangered	MEF	Atlantic Coast
Acipenseridae	Gulf Sturgeon	*Acipenser oxyrinchus desotoi*	Threatened	MEF	Gulf Coast
Acipenseridae	Green Sturgeon, southern DPS	*Acipenser medirostris*	Threatened	MEF	Pacific Coast
Sebastidae	Bocaccio, Puget Sound/Georgia Basin DPS	*Sebastes paucispinis*	Proposed	M	Pacific Coast
Sebastidae	Canary Rockfish, Puget Sound/Georgia Basin DPS	*Sebastes pinniger*	Proposed	M	Pacific Coast
Sebastidae	Yelloweye Rockfish, Puget Sound/Georgia Basin DPS	*Sebastes ruberrimus*	Proposed	M	Pacific Coast
Pristidae	Smalltooth Sawfish, United States DPS	*Pristis pectinata*	Endangered	ME	Atlantic & Gulf Coast
Pristidae	Largetooth Sawfish	*Pristis perotetti*	Candidate	MEF	Gulf Coast, Central & South America
Lotidae	Cusk	*Brosme brosme*	Candidate	M	Atlantic Coast
Clupeidae	Pacific Herring, Southeast Alaska DPS	*Clupea pallasi*	Candidate	ME	Pacific Coast
Osmeridae	Pacific Eulachon/Smelt, Southern DPS	*Thaleichthys pacificus*	Proposed	MEF	Pacific Coast
MOLLUSKS					
Haliotidae	Black Abalone	*Haliotis cracherodii*	Endangered	M	Pacific Coast
Haliotidae	White Abalone	*Haliotis sorenseni*	Endangered	M	Pacific Coast
CORALS					
Acroporidae	Elkhorn Coral	*Acropora palmata*	Threatened	M	Caribbean
Acroporidae	Staghorn Coral	*Acropora cervicornis*	Threatened	M	Caribbean
Acroporidae	Fuzzy Table Coral	*Acropora paniculata*	Candidate	M	Hawaii
Acroporidae	Hawaiian Reef Coral	*Montipora dilatata*	Candidate	M	Hawaii
Acroporidae	Blue Rice Coral	*Montipora flabellata*	Candidate	M	Hawaii
Acroporidae	Sandpaper Rice Coral	*Montipora patula*	Candidate	M	Hawaii
Acroporidae		*Acropora aculeus*	Candidate	M	Indo-Pacific

Family	Common Name	Scientific Name	Listing Status	Habitat	Range
CORALS - continued					
Acroporidae		*Acropora acuminata*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora aspera*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora dendrum*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora donei*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora globiceps*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora horrida*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora jacquelineae*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora listeri*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora lokani*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora microclados*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora palmerae*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora pharaonis*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora polystoma*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora retusa*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora rudis*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora speciosa*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora striata*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora tenella*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora vaughani*	Candidate	M	Indo-Pacific
Acroporidae		*Acropora verweyi*	Candidate	M	Indo-Pacific
Acroporidae		*Anacropora puertogalerae*	Candidate	M	Indo-Pacific
Acroporidae		*Anacropora spinosa*	Candidate	M	Indo-Pacific
Acroporidae		*Astreopora cucullata*	Candidate	M	Indo-Pacific
Acroporidae		*Isopora crateriformis*	Candidate	M	Indo-Pacific
Acroporidae		*Isopora cuneata*	Candidate	M	Indo-Pacific
Acroporidae		*Montipora angulata*	Candidate	M	Indo-Pacific
Acroporidae		*Montipora australiensis*	Candidate	M	Indo-Pacific
Acroporidae		*Montipora calcarea*	Candidate	M	Indo-Pacific
Acroporidae		*Montipora caliculata*	Candidate	M	Indo-Pacific
Acroporidae		*Montipora lobulata*	Candidate	M	Indo-Pacific
Agaricidae	Lamarck's Sheet Coral	*Agaricia lamarcki*	Candidate	M	Caribbean

Family	Common Name	Scientific Name	Listing Status	Habitat	Range
CORALS - continued					
Agaricidae		*Leptoseris incrustans*	Candidate	M	Hawaii
Agaricidae		*Leptoseris yabei*	Candidate	M	Indo-Pacific
Agaricidae		*Pachyseris rugosa*	Candidate	M	Indo-Pacific
Agaricidae		*Pavona bipartita*	Candidate	M	Indo-Pacific
Agaricidae		*Pavona cactus*	Candidate	M	Indo-Pacific
Agaricidae		*Pavona decussata*	Candidate	M	Indo-Pacific
Agaricidae		*Pavona diffluens*	Candidate	M	Indo-Pacific
Agaricidae		*Pavona venosa*	Candidate	M	Indo-Pacific
Faviidae	Boulder Star Coral	*Montastraea annularis*	Candidate	M	Caribbean
Faviidae	Mountainous Star Coral	*Montastraea faveolata*	Candidate	M	Caribbean
Faviidae		*Montastraea franksi*	Candidate	M	Caribbean
Faviidae	Agassiz's Coral	*Cyphastrea agassizi*	Candidate	M	Hawaii
Faviidae	Ocellated Coral	*Cyphastrea ocellina*	Candidate	M	Hawaii
Faviidae		*Barabattoia laddi*	Candidate	M	Indo-Pacific
Faviidae		*Caulastrea echinulata*	Candidate	M	Indo-Pacific
Meandrinidae		*Dendrogyra cylindrus*	Candidate	M	Caribbean
Meandrinidae	Elliptical Star Coral	*Dichocoenia stokesii*	Candidate	M	Caribbean
Mussidae		*Mycetophyllia ferox*	Candidate	M	Caribbean
Mussidae		*Acanthastrea brevis*	Candidate	M	Indo-Pacific
Mussidae		*Acanthastrea hemprichii*	Candidate	M	Indo-Pacific
Mussidae		*Acanthastrea ishigakiensis*	Candidate	M	Indo-Pacific
Mussidae		*Acanthastrea regularis*	Candidate	M	Indo-Pacific
Mussidae		*Pocillopora danae*	Candidate	M	Indo-Pacific
Mussidae		*Pocillopora elegans*	Candidate	M	Indo-Pacific
Mussidae		*Seriatopora aculeata*	Candidate	M	Indo-Pacific
Oculinidae		*Galaxea astreata*	Candidate	M	Indo-Pacific
Poritidae		*Porites pukoensis*	Candidate	M	Hawaii
Poritidae		*Alveopora allingi*	Candidate	M	Indo-Pacific
Poritidae		*Alveopora fenestrata*	Candidate	M	Indo-Pacific

Family	Common Name	Scientific Name	Listing Status	Habitat	Range
CORALS - continued					
Poritidae		*Alveopora verrilliana*	Candidate	M	Indo-Pacific
Poritidae		*Porites horizontalata*	Candidate	M	Indo-Pacific
Poritidae		*Porites napopora*	Candidate	M	Indo-Pacific
Poritidae		*Porites nigrescens*	Candidate	M	Indo-Pacific
Siderastreidae	Stellar Coral	*Psammocora stellata*	Candidate	M	Hawaii
Dendrophyllidae		*Turbinaria mesenterina*	Candidate	M	Indo-Pacific
Dendrophyllidae		*Turbinaria peltata*	Candidate	M	Indo-Pacific
Dendrophyllidae		*Turbinaria reniformis*	Candidate	M	Indo-Pacific
Dendrophyllidae		*Turbinaria stellula*	Candidate	M	Indo-Pacific
Euphyllidae		*Euphyllia cristata*	Candidate	M	Indo-Pacific
Euphyllidae		*Euphyllia paraancora*	Candidate	M	Indo-Pacific
Euphyllidae		*Euphyllia paradivisa*	Candidate	M	Indo-Pacific
Euphyllidae		*Physogyra lichtensteini*	Candidate	M	Indo-Pacific
Pectinidae		*Pectinia alcicornis*	Candidate	M	Indo-Pacific
Helioporidae		*Heliopora coerulea*	Candidate	M	Indo-Pacific
Milleporidae		*Millepora foveolata*	Candidate	M	Indo-Pacific
Milleporidae		*Millepora tuberosa*	Candidate	M	Indo-Pacific
PLANTS					
Hydrocharitaceae	Johnson's Seagrass	*Halophila johnsonii*	Threatened	ME	Atlantic Coast

* Seven populations of Loggerhead Turtle have been proposed for listing as endangered (Mediterranean Sea, North Indian Ocean, North Pacific Ocean, Northeast Atlantic Ocean, Northwest Atlantic Ocean, South Pacific Ocean and Southeast Indo-Pacific Ocean populations).

Appendix 3. ESA-listed species under NMFS' jurisdiction that occur in U.S. waters, by coastal regions

Regions: **EC = East Coast** **GM = Gulf of Mexico** **CR = Caribbean**
AK = Alaska **WC = West Coast** **HI = Hawaii** **PI = Pacific Islands**

Caribbean region includes Puerto Rico, the U.S. Virgin Islands and Navassa Island.
Hawaii region includes the Hawaiian Islands and Midway Island.
Pacific Islands region include Guam, the Northern Mariana Islands, American Samoa, Johnston Atoll, Wake Island, Howland Island, Baker Island, Palmyra Atoll, Kingman Reef and Jarvis Island.

Sources: CBD 2009; NOAA 2010b.

Species	Atlantic Ocean			Pacific Ocean			
	EC	GM	CR	AK	WC	HI	PI
CETACEANS							
Bowhead Whale				X			
North Atlantic Right Whale	X						
North Pacific Right Whale				X	X		
Blue Whale	X	X	X	X	X	X	X
Fin Whale	X	X	X	X	X	X	X
Humpback Whale	X	X	X	X	X	X	X
Sei Whale	X				X		
Beluga Whale, Cook Inlet DPS				X			
Sperm Whale	X	X	X	X	X	X	X
Killer Whale, Southern Resident DPS					X		
False Killer Whale, Insular Hawaiian DPS						X	
PINNIPEDS							
Guadalupe Fur Seal					X		
Stellar Sea Lion, Eastern DPS				X	X		
Stellar Sea Lion, Western DPS				X			
Hawaiian Monk Seal						X	
Ringed Seal				X			
Bearded Seal				X			
TURTLES							
Green Turtle, Florida & Mexico Pacific coast breeding colonies	X	X	X	X	X		
Green Turtle, all other areas						X	X
Hawksbill Turtle	X	X	X		X	X	X
Kemp's Ridley Turtle	X	X	X				
Loggerhead Turtle	X	X	X		X	X	X
Olive Ridley Turtle, Mexico Pacific coast breeding colonies					X		
Olive Ridley Turtle, all other areas			X		X	X	
Leatherback Turtle	X	X	X	X	X	X	X

Species	Atlantic Ocean			Pacific Ocean			
	EC	GM	CR	AK	WC	HI	PI
FISH							
Atlantic Salmon, Gulf of Maine DPS	X						
Chinook Salmon, California coastal ESU					X		
Chinook Salmon, Central Valley spring-run					X		
Chinook Salmon, Sacramento River winter-run					X		
Chinook Salmon, Lower Columbia River					X		
Chinook Salmon, Upper Columbia River spring-run					X		
Chinook Salmon, Upper Willamette River					X		
Chinook Salmon, Snake River fall-run					X		
Chinook Salmon, Snake River spring/summer-run					X		
Chinook Salmon, Puget Sound					X		
Chum Salmon, Columbia River					X		
Chum Salmon, Hood Canal summer-run					X		
Coho Salmon, Central California coast					X		
Coho Salmon, Southern Oregon & Northern California coasts					X		
Coho Salmon, Oregon Coast					X		
Coho Salmon, Lower Columbia River					X		
Sockeye Salmon, Ozette Lake					X		
Sockeye Salmon, Snake River					X		
Steelhead Trout, Southern California					X		
Steelhead Trout, South-Central California coast					X		
Steelhead Trout, Central California coast					X		
Steelhead Trout, California Central Valley					X		
Steelhead Trout, Northern California					X		
Steelhead Trout, Lower Columbia River					X		
Steelhead Trout, Middle Columbia River					X		
Steelhead Trout, Upper Columbia River					X		
Steelhead Trout, Upper Willamette River					X		
Steelhead Trout, Snake River basin					X		
Steelhead Trout, Puget Sound					X		
Atlantic Sturgeon	X						
Shortnose Sturgeon	X						
Gulf Sturgeon		X					
Green Sturgeon, southern DPS				X	X		
Bocaccio, Puget Sound/Georgia Basin DPS					X		
Canary Rockfish, Puget Sound/Georgia Basin DPS					X		
Yelloweye Rockfish, Puget Sound/Georgia Basin DPS					X		
Smalltooth Sawfish, United States DPS	X	X					
Largetooth Sawfish		X					

Species	Atlantic Ocean			Pacific Ocean			
	EC	GM	CR	AK	WC	HI	PI
FISH - continued							
Cusk	X						
Pacific Herring, Southeast Alaska DPS				X			
Pacific Eulachon/Smelt, Southern DPS					X		
MOLLUSKS							
Black Abalone					X		
White Abalone					X		
CORALS							
Acropora cervicornis	X		X				
Acropora palmata	X		X				
Agaricia lamarcki	X	X	X				
Montastraea annularis	X	X	X				
Montastraea faveolata	X	X	X				
Montastraea franksi	X	X	X				
Dendrogyra cylindrus	X	X	X				
Dichocoenia stokesii	X	X	X				
Mycetophyllia ferox	X	X	X				
Acropora paniculata						X	X
Montipora dilatata						X	
Montipora flabellata						X	
Montipora patula						X	X
Leptoseris incrustans						X	X
Cyphastrea agassizi						X	X
Cyphastrea ocellina						X	X
Porites pukoensis						X	
Psammocora stellata						X	
Acropora aculeus							X
Acropora acuminata							X
Acropora aspera							X
Acropora dendrum							X
Acropora donei							X
Acropora globiceps							X
Acropora horrida							X
Acropora jacquelineae							X
Acropora listeri							X
Acropora lokani							X
Acropora microclados							X
Acropora palmerae							X

Species	Atlantic Ocean			Pacific Ocean			
	EC	GM	CR	AK	WC	HI	PI
CORALS - continued							
Acropora pharaonis							X
Acropora polystoma							X
Acropora retusa							X
Acropora rudis							X
Acropora speciosa							X
Acropora striata							X
Acropora tenella							X
Acropora vaughani							X
Acropora verweyi							X
Anacropora puertogalerae							X
Anacropora spinosa							X
Astreopora cucullata							X
Isopora crateriformis							X
Isopora cuneata							X
Montipora angulata							X
Montipora australiensis							X
Montipora calcarea							X
Montipora caliculata							X
Montipora lobulata							X
Leptoseris yabei							X
Pachyseris rugosa							X
Pavona bipartita							X
Pavona cactus							X
Pavona decussata							X
Pavona diffluens							X
Pavona venosa							X
Barabattoia laddi							X
Caulastrea echinulata							X
Acanthastrea brevis							X
Acanthastrea hemprichii							X
Acanthastrea ishigakiensis							X
Acanthastrea regularis							X
Pocillopora danae							X
Pocillopora elegans							X
Seriatopora aculeata							X
Galaxea astreata							X
Alveopora allingi							X
Alveopora fenestrata							X

Species	Atlantic Ocean			Pacific Ocean			
	EC	GM	CR	AK	WC	HI	PI
CORALS - continued							
Alveopora verrilliana						X	X
Porites horizontalata							X
Porites napopora							X
Porites nigrescens							X
Turbinaria mesenterina							X
Turbinaria peltata							X
Turbinaria reniformis							X
Turbinaria stellula							X
Euphyllia cristata							X
Euphyllia paraancora							X
Euphyllia paradivisa							X
Physogyra lichtensteini							X
Pectinia alcicornis							X
Heliopora coerulea							X
Millepora foveolata							X
Millepora tuberosa							X
PLANTS							
Johnson's Seagrass	X						

Appendix 4. Some data on the survival of fish viruses in water and mud

Virus	Temperature	Period	End Status	Source
Viral Hemorrhagic Septicemia Virus VHS				
fresh water	4°C	5 d	survival	Kipp & Ricciardi 2001
fresh water	4°C	months	0.1% survival	PAHW 2007
fresh water	4°C	up to 28-35 d	survival	Parry & Dixon 1997
fresh water [1]	12°C	1 h	2% survival	Winton *et al.* 1991
fresh water [2]	12°C	1 h	0.5% survival	Winton *et al.* 1991
fresh water	15°C	13 d	0.1% survival	Hawley & Garver 2008
fresh water	20°C	≈28 d	0.1% survival	PAHW 2007
fresh water	20°C	60 d	survival	Mori *et al.* 2002
fresh water	25°C	40 d	survival	Mori *et al.* 2002
fresh water-filtered	4°C	up to >1 y	infective	Hawley & Garver 2008
tap water	10°C	49 d	survival	Ahne 1982
sea water	4°C	7-21 d	survival	Parry & Dixon 1997
sea water	4°C	up to 25 d	survival	Mori *et al.* 2002
sea water	4°C	10 mon	infective	CFSPH 2007
3% NaCl solution [1]	12°C	1 h	50% survival	Winton *et al.* 1991
3% NaCl solution [2]	12°C	1 h	10% survival	Winton *et al.* 1991
sea water	15°C	4 d	0.1% survival	Hawley & Garver 2008
sea water	15°C	a few days	survival	Mori *et al.* 2002
sea water	15-20°C	<7 d	survival	Parry & Dixon 1997
sea water–sterile [3]	15°C	0.5 d	50% survival	Kocan *et al.* 2001
sea water–sterile [3]	15°C	1.5 d	10% survival	Kocan *et al.* 2001
sea water–sterile [4]	15°C	60 d	survival	Mori *et al.* 2002
sea water–sterile [4]	20°C	32 d	survival	Mori *et al.* 2002
sea water–enriched [5]	15°C	4 d	20% survival	Kocan *et al.* 2001
sea water–enriched [6]	15°C	4 d	≈100% survival	Kocan *et al.* 2001
sea water–enriched [7]	15°C	15 d	≈100% survival	Kocan *et al.* 2001
sea water–enriched [7]	15°C	36 d	55% survival	Kocan *et al.* 2001
mud	4°C	10 d	stable	Ahne 1982

[1] European (F1) isolate
[2] Northwestern Pacific (Makah) isolate
[3] 5 µm-filtered and UV
[4] 0.22 µm filtered
[5] 5 µm-filtered and UV, with 0.1% ovarian fluid added
[6] 5 µm-filtered and UV, with 1% ovarian fluid added
[7] 5 µm-filtered and UV, with culture medium added
[8] 0.45 µm-filtered

Virus	Temperature	Period	End Status	Source
Infectious Haematopietic Necrosis Virus (IHNV)				
fresh water	4°C	140 d	0.1% survival	Pietsch *et al.* 1977
fresh water	15°C	25 d	0.1% survival	Toranzo & Hetrick 1982
fresh water–lake	10°C	49 d	survival	Wedemayer *et al.* 1978
fresh water–river	15°C	1 d	1% survival	LaPatra *et al.* 2001
fresh water	20°C	14 d	0.1% survival	Toranzo & Hetrick 1982
distilled water	10°C	14 d	survival	Wedemayer *et al.* 1978
sea water	15°C	14 d	0.1% survival	Toranzo & Hetrick 1982
sea water	15°C	4 d	0.01% survival	Pietsch *et al.* 1977
sea water-artificial	15°C	3 d	0.01% survival	Pietsch *et al.* 1977
sea water	15°C	3 d	survival	Kamei *et al.* 1987
sea water	20°C	12 d	0.1% survival	Toranzo & Hetrick 1982
sea water-artificial	25°C	2 h	survival	LaPatra *et al.* 2001
adsorbed to clay	?	up to 63 d	infective	Yoshinaka *et al.* 2000
Koi Herpesvirus (KHV)				
fresh water	23-35°C	<21 h	infective	Perelberg *et al.* 2003
fresh water	15-25°C	3 d	≤0.1% survival	Shimizu *et al.* 2006
fresh water–sterile [8]	15-25°C	>7 d	≈1% survival	Shimizu *et al.* 2006
sediment	15-25°C	3 d	≤0.1% survival	Shimizu *et al.* 2006
Infectious Salmon Anemia Virus (ISAV)				
fresh water–sterile	4°C	>126 d	detectable	MacLeod *et al.* 2003
fresh water–sterile	15°C	>126 d	detectable	MacLeod *et al.* 2003
sea water	4°C	105 d	detectable	MacLeod *et al.* 2003
sea water	6°C	>20 h	infective	Nylund *et al.* 1994
sea water	15°C	28 d	detectable	MacLeod *et al.* 2003
sea water–sterile	4°C	>105 d	infective	MacLeod *et al.* 2003
sea water–sterile	15°C	>105 d	infective	MacLeod *et al.* 2003
Pike Fry Rhabdovirus (PFRV)				
tap water	10°C	70 d	survival	Ahne 1982
mud	4°C	42 d	stable	Ahne 1982
Infectious Pancreatic Necrosis Virus (IPNV)				
tap water	10°C	>231 d	survival	Ahne 1982
mud	4°C	>210 d	stable	Ahne 1982
Spring Viraemia of Carp Virus (SVCV)				
tap water	10°C	42 d	survival	Ahne 1982
mud	4°C	42 d	stable	Ahne 1982

[1] European (F1) isolate
[2] Northwestern Pacific (Makah) isolate
[3] 5 µm-filtered and UV
[4] 0.22 µm filtered
[5] 5 µm-filtered and UV, with 0.1% ovarian fluid added
[6] 5 µm-filtered and UV, with 1% ovarian fluid added
[7] 5 µm-filtered and UV, with culture medium added
[8] 0.45 µm-filtered or autoclaved at 121°C for 15 min.

References for Appendix 4

Ahne, W. 1982. Comparative studies on the stability of four fish-pathogenic viruses (VHSV, PFR, SVCV, IPNV). Zentralbl. Veterinarmed. [B] 29: 457–476.

CFSPH. 2007. Viral Hemorrhagic Septicemia. Center for Food Security and Public Health, Institute for International Cooperation in Animal Biologics and College of Veterinary Medicine, Iowa State University, Ames, Iowa.

Hawley, L.M. and K.A. Garver. 2008. Stability of viral hemorrhagic septicemia virus (VHSV) in freshwater and seawater at various temperatures. Diseases of Aquatic Organisms 82(3): 171-178.

Kamei, Y., Yoshmizu, M., Ezura, Y. and T. Kimura 1987. Effects of estuarine and marine waters on the infectivities of infectious hematopoietic necrosis virus (IHNV) and infectious pancreatic necrosis virus (IPNV). Bull. Fac. Fish., Hokkaido University 38: 271- 285.

Kipp, R.M. and A. Ricciardi. 2006. NOAA – Viral Hemorrhagic Septicemia (VHS) Fact Sheet. GLANSIS.

Kocan R.M., P.K. Hershberger, N.E. Elder and J.R. Winton. 2001. Survival of the North American strain of viral hemorrhagic septicemia virus (VHSV) in filtered seawater and seawater containing ovarian fluid, crude oil and serum-enriched culture medium. Diseases of Aquatic Organisms 44: 75–78.

LaPatra, S.E., Batts, W.N., Overturf, K., Jones, G.R., Shewmaker, WD. and Winton, J.R. 2001. Negligible risk associated with the movement of processed rainbow trout, *Oncorhynchus mykiss* (Walbaum), from an infectious haematopoietic necrosis virus (IHNV) endemic area. Journal of Fish Diseases 24: 399-408.

MacLeod, L.A., Raynard, R.S., Murray, A.G. and Gregory, A. 2003. Survival of infectious salmon anaemia virus (ISAV) in aquatic environments. Abstract from the 11th EAFP International Conference: Diseases of Fish and Shellfish, Malta.

Mori, K., Iida, H., Nishizawa, T., Arimoto, M., Nakajima, K. and Muroga, K. 2002. Properties of viral haemorrhagic septicaemia virus (VHSV) isolated from Japanese flounder (*Paralichthys olivaceus*). Fish Pathology 37: 169-174.

Nylund, A.S., T. Hovland, K. Hodneland, F. Nilsen and P. Lovik. 1994. Mechanisms for transmission of infectious salmon anemia (ISA). Diseases of Aquatic Organisms 19: 95-100.

PAHW. 2007. Possible Vector Species and Live Stages of Susceptible Species not Transmitting Disease as Regards Certain Fish Diseases: Scientific Opinion of the Panel on Animal Health and Welfare. The EFSA (European Food Safety Authority) Journal 584: 1-163.

Parry L. and P.F. Dixon. 1997. Stability of nine viral haemorrhagic septicaemia virus (VHSV) isolates in seawater. Bull. Eur. Assoc. Fish Pathol. 17: 31–36.

Perelberg, A., Smirnov, M., Hutoran, M., Diamant, A., Bejerano, Y. and M. Kotler. 2003. Epidemiological description of a new viral disease afflicting cultured *Cyprinus carpio* in Israel. Israeli Journal of Aquaculture 55: 5-12.

Pietsch, J.P., Amend, D.F. and Miller, C. 1977. Survival of infectious hematopoietic necrosis virus held under various conditions. J. Fish. Res. Board Can. 34: 1360-1364.

Shimizu, T., Yoshida, N., Kasai, H. and Yoshimizu M. 2006. Survival of koi herpesvirus (KHV) in environmental water. Fish Pathology 41: 153-157.

Toranzo, A.E. and Hetrick, F.M. 1982. Comparative stability of two salmonid viruses and poliovirus in

fresh, estuarine and marine waters. Journal of Fish Diseases 5: 223-231.

Wedemeyer, G.A., Nelson, N.C. and Smith, C.A. 1978. Survival of the salmonid viruses infectious hematopoietic necrosis (IHNV) and infectious pancreatic necrosis (IPNV) in ozonated, chlorinated, and untreated water. J. Fish. Res. Board Canada 35: 875-879.

Winton, J.R., Batts, W., Deering, R., Brunson, R., Hopper, K. and T. Nishizawa. 1991. Characteristics of the first North American isolates of viral hemorrhagic septicemia virus. Pages 43-50 in: Second International Symposium on Viruses of Lower Vertebrates, Oregon State University, Corvallis, OR.

Yoshinaka, T., Yoshimizu, M. and Ezura, Y. 2000. Adsorption and infectivity of infectious hematopoietic necrosis virus (IHNV) with various solids. Journal of Aquatic Animal Health 12: 64-68.

Appendix 5. NOAA and Listing Petition comments on disease impacts and threats to ESA-listed coral species

Comments on the 2 Threatened species (*Acropora cervicornis* and *A. palmata*) are summarized from NOAA's online species descriptions (NOAA 2010b); comments on the 82 Candidate species are summarized from the Listing Petition (CBD 2009).

Species	Comments on disease impacts and threats
CARIBBEAN SPECIES	
Acropora cervicornis	Disease is the main cause of decline; low genetic diversity due to asexual reproduction makes recovery from disease difficult.
Acropora palmata	Disease is the main cause of decline; low genetic diversity due to asexual reproduction makes recovery from disease difficult.
Agaricia lamarcki	Threatened by white plague & black band disease; ability to resist and recover from disease is hampered by overlapping colonies.
Montastraea annularis	Threatened by infectious diseases.
Montastraea faveolata	Develops white plague infections after bleaching events.
Montastraea franksi	Threatened by infectious diseases, but more resistant than other *Montastraea* species.
Dendrogyra cylindrus	Especially sensitive to white plague.
Dichocoenia stokesii	Highly susceptible to white plague, which is a major threat; also susceptible to black band disease.
Mycetophyllia ferox	Localized declines and partial recovery from white plague outbreaks in 1970s-1980s; later outbreaks have been increasingly virulent with significant mortality; also susceptible to black band disease.
HAWAIIAN SPECIES (SOME SPECIES ALSO OCCUR IN THE INDO-PACIFIC REGION)	
Acropora paniculata	Especially susceptible to disease and slow to recover.
Montipora dilatata	Disease not mentioned.
Montipora flabellata	Disease not mentioned.
Montipora patula	Disease not mentioned.
Leptoseris incrustans	Susceptible to disease.
Cyphastrea agassizi	Disease not mentioned.
Cyphastrea ocellina	Disease not mentioned.
Porites pukoensis	More susceptible to disease than many corals.
Psammocora stellata	Disease not mentioned.

Species	Comments on disease impacts and threats
INDO-PACIFIC SPECIES	
Acropora aculeus	Especially susceptible to disease and slow to recover.
Acropora acuminata	Especially susceptible to disease and slow to recover.
Acropora aspera	Especially susceptible to disease and slow to recover.
Acropora dendrum	Especially susceptible to disease and slow to recover.
Acropora donei	Especially susceptible to disease and slow to recover.
Acropora globiceps	Especially susceptible to disease and slow to recover.
Acropora horrida	Especially susceptible to disease and slow to recover.
Acropora jacquelineae	Especially susceptible to disease and slow to recover.
Acropora listeri	Especially susceptible to disease and slow to recover.
Acropora lokani	Especially susceptible to disease and slow to recover.
Acropora microclados	Especially susceptible to disease and slow to recover.
Acropora palmerae	Especially susceptible to disease and slow to recover.
Acropora pharaonis	Especially susceptible to disease and slow to recover.
Acropora polystoma	Especially susceptible to disease and slow to recover.
Acropora retusa	Especially susceptible to disease and slow to recover.
Acropora rudis	Especially susceptible to disease and slow to recover.
Acropora speciosa	Especially susceptible to disease and slow to recover.
Acropora striata	Especially susceptible to disease and slow to recover.
Acropora tenella	Especially susceptible to disease and slow to recover.
Acropora vaughani	Especially susceptible to disease and slow to recover.
Acropora verweyi	Especially susceptible to disease and slow to recover.
Anacropora puertogalerae	Especially susceptible to disease and slow to recover.
Anacropora spinosa	Especially susceptible to disease and slow to recover.
Astreopora cucullata	Low tolerance or resistance to disease and slow to recover.
Isopora crateriformis	Low tolerance or resistance to disease, and slow to recover due to limited reproduction and dispersal.
Isopora cuneata	Low tolerance or resistance to disease, and slow to recover due to limited reproduction and dispersal.
Montipora angulata	Susceptible to disease, but broad distribution and depth range could provide some resilience at the population level.
Montipora australiensis	Susceptible to disease.
Montipora calcarea	Susceptible to disease.
Montipora caliculata	Susceptible to disease.
Montipora lobulata	Susceptible to disease.
Leptoseris yabei	Susceptible to disease.
Pachyseris rugosa	Disease not mentioned.

Species	Comments on disease impacts and threats
INDO-PACIFIC SPECIES - continued	
Pavona bipartita	Disease not mentioned.
Pavona cactus	Disease not mentioned.
Pavona decussata	Disease not mentioned.
Pavona diffluens	Disease not mentioned.
Pavona venosa	Susceptible to disease.
Barabattoia laddi	Susceptible to disease due to restricted depth range.
Caulastrea echinulata	Disease not mentioned.
Acanthastrea brevis	Disease not mentioned.
Acanthastrea hemprichii	Especially susceptible to disease.
Acanthastrea ishigakiensis	Especially susceptible to disease due to restricted depth range.
Acanthastrea regularis	Especially susceptible to disease due to restricted depth range.
Pocillopora danae	Especially susceptible to disease.
Pocillopora elegans	Especially susceptible to disease.
Seriatopora aculeata	Especially susceptible to disease.
Galaxea astreata	Disease not mentioned.
Alveopora allingi	This genus is thought to be relatively unsusceptible to disease.
Alveopora fenestrata	This genus is thought to be relatively unsusceptible to disease.
Alveopora verrilliana	This genus is thought to be relatively unsusceptible to disease.
Porites horizontalata	This genus is more susceptible to disease than most corals.
Porites napopora	This genus is more susceptible to disease than most corals.
Porites nigrescens	This genus is more susceptible to disease than most corals.
Turbinaria mesenterina	Disease not mentioned.
Turbinaria peltata	Disease not mentioned.
Turbinaria reniformis	Susceptible to disease due to restricted depth range.
Turbinaria stellula	Susceptible to disease due to restricted depth range and distribution.
Euphyllia cristata	Disease not mentioned.
Euphyllia paraancora	Disease not mentioned.
Euphyllia paradivisa	Disease not mentioned.
Physogyra lichtensteini	Disease not mentioned.
Pectinia alcicornis	Disease not mentioned.
Heliopora coerulea	Disease not mentioned.
Millepora foveolata	In Fiji, the genus appears to be resistant to disease.
Millepora tuberosa	In Fiji, the genus appears to be resistant to disease.