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# Evaluating potential infectious disease threats for southern resident killer whales, *Orcinus orca*: a model for endangered species

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**Abstract**

Infectious diseases have the potential to play a role in the decline of threatened wildlife populations, as well as negatively affect their long-term viability, but determining which infectious agents present risks can be difficult. The southern resident killer whale, *Orcinus orca*, population is endangered and little is known about infectious diseases in this species. Using available reference literature, we identified 15 infectious agents (bacteria, viruses, and fungi) reported in free-ranging and captive killer whales, as well as 28 additional infectious agents reported in free-ranging and captive odontocete species sympatric to southern resident killer whales. Infectious agents were scored as having a high, medium, or low ability to affect fecundity or reproductive success, to cause disease in individual animals, and to cause epizootics. Marine *Brucella* spp., cetacean poxvirus, cetacean morbilliviruses, and herpesviruses were identified as high priority pathogens that warrant further study. Using identified pathogens to develop a standardized necropsy and disease testing protocol for southern resident killer whales and sympatric odontocetes will improve future efforts to better understand the impacts of priority and non-priority infectious agents on southern resident killer whales. This model can be used to evaluate potential infectious disease risks in other threatened wildlife populations.


*Keywords:* Endangered species; Infectious diseases; Killer whale; *Orcinus orca*; Wildlife diseases

## 1. Introduction

Infectious diseases have the potential to negatively impact wild animal populations (Scott, 1988; Gulland, 1995; Deem et al., 2001), which has important implications for conservation (Young, 1994). Infectious diseases may play a role in the decline of threatened and endangered wildlife populations, as well as negatively affect population recovery (McCallum, 1994; Gaydos and Corn, 2001). Determining which infectious agents present a risk to a population can be difficult as these populations often are understudied from a wildlife health perspective, and potential disease threats may be unknown. The "southern resident" killer whale population, found frequently within the inland waters of Washington (USA) and British Columbia (Canada) but known to range as far south as Monterrey, California (USA), has declined 20% since 1996 (Krahn et al., 2002). This population of killer whales that primarily eat fish is comprised of individuals from three pods (J, K, and L) (Ford et al., 1998). It has been listed as endangered by the Canadian Committee for the Status on Endangered Wildlife in Canada (Baird, 2001). The US National Oceanic and Atmospheric Administration–National Marine Fisheries Service recently reviewed the status of the population under the US Endangered Species Act (Krahn et al., 2002). They concluded that the southern resident population was not considered a discrete population segment (DPS) as defined by the US Endangered Species Act; however, if

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re-evaluation of the current global species taxon redefines the southern resident population as a DPS, the southern resident population would likely be listed as endangered.

As with many endangered and threatened wildlife populations, information about the role that infectious diseases may play in the decline of this population is not available, nor is information regarding infectious diseases that may threaten the long-term viability of this small population. Consequently, the US Biological Review Team for Southern Resident Killer Whales (Krahn et al., 2002) determined that due to lack of information, it was "not possible to estimate the probability that disease poses a major risk to the Southern Resident killer whale group." Despite the lack of knowledge, Baird (2001) notes that diseases "should be taken into account in conservation planning and population viability analysis" for the southern resident killer whale population.

We identify infectious disease threats for this population, and then evaluate them for their potential to be involved in the population's decline or to threaten the long-term viability of this small population. Identified infectious diseases serve as a baseline for developing a standardized necropsy and disease testing protocol for stranded southern resident killer whales and sympatric odontocetes. Additionally, we identify specific high priority infectious diseases that warrant further study. This model may be useful in identification and evaluation of disease threats for other endangered or threatened populations.

## 2. Methods

### 2.1. Identification of pathogens in killer whales and sympatric odontocete species

Using available databases including Aquatic Science and Fisheries Abstracts, BIOSIS Previews, Current Contents, Fish and Wildlife Reference Service, and MEDLINEplus, as well as reference literature, we identified infectious agents (bacteria, viruses, and fungi) reported in both free-ranging and captive killer whales. Also identified were infectious agents not reported in killer whales, but reported in free-ranging and captive odontocete species sympatric to southern resident killer whales. While southern resident killer whales encounter Dall's (*Phocoenoides dalli*) and harbor porpoises (*Phocoena phocoena*) most frequently within their "core" range, throughout their entire range individuals from this population also encounter bottlenose dolphins (*Tursiops truncatus*), false killer whales (*Pseudorca crassidens*), northern right whale dolphins (*Lissodelphis borealis*), Pacific white-sided dolphins (*Lagenorhynchus obliquidens*), Risso's dolphins (*Grampus griseus*), short-beaked common dolphins (*Delphinus delphis*), short-finned pilot whales (*Globicephala macrorhynchus*), and striped dolphins (*Stenella coeruleoalba*) (Osborne et al., 1988). We assumed that if a species was susceptible to an infectious disease within the range where the disease was identified, the members of that species that are truly sympatric to southern resident killer whales also would be susceptible to the disease. Because probability of exposure is important in evaluating risk, the geographic location where the pathogen was reported was noted and taken into consideration when evaluating infectious agents reported in free-ranging species.

### 2.2. Evaluating possible pathogen involvement in the population decline of southern resident killer whales

Using distinct physical markings and photographic identification, the Center for Whale Research (Friday Harbor, Washington, USA) has conducted an annual census of the southern resident killer whale population since 1974. Photographs and observations permit aging individual animals, cataloging them into known or assumed genealogical relationships, and observing animals as individuals (Olesiuk et al., 1990). These data and analysis of these data performed by the US Biological Review Team for Southern Resident Killer Whales (Krahn et al., 2002) were evaluated for mortality trends that could be related to an epizootic caused by an infectious disease. Identified diseases also were evaluated and scored as a high, medium, or low ability to affect fecundity or reproductive success in killer whales.

### 2.3. Evaluating the potential of pathogens to negatively affect future population size and viability

Based on what was reported in the literature reviewed and what is known about the transmissibility of the pathogen, identified pathogens also were scored as having a low, medium, or high ability to cause disease in individual animals (virulence in individuals) and to cause disease at the population level (epizootic potential). Pathogens for which virulence in individuals and epizootic potential were unknown were scored as "unknown." What was known about pathogen transmissibility was used to classify the likelihood of the pathogen having a high epizootic potential. Where pathogens were reported from different species, from different locations, or described with differing virulence or epizootic potential, each reference was listed and scored accordingly. All infectious agents that were assigned as having a high epizootic potential and described in the literature as being able to cause mortality were considered to have the potential to impact the long-term viability of the southern resident population.

## 3. Results

### 3.1. Pathogens identified

We identified 43 pathogens reported in captive and free-ranging killer whales or sympatric odontocete species, including 20 bacteria, eight viruses, and 15 fungi. Of these, three infectious agents were reported in free-ranging killer whales (Table 1), 13 from captive killer whales (Table 2), and 11 and 16 from free-ranging (Table 3) and captive (Table 4) odontocete species, respectively that are sympatric to southern resident killer whales. The only pathogen reported to cause mortality in a member of the southern resident population is the bacteria *Edwardsiella tarda*, which was responsible for the death of J18 in 2000 (Ford et al., 2000). Details of the pathologic findings are not given in the reference, however based on what is known about the transmissibility and nature of the pathogen, it was scored low for epizootic potential as well as for ability to reduce fecundity of affect reproductive success. Three pathogens identified (*Edwardsiella tarda*—Table 1, porpoise morbillivirus—Table 3, and *Coccidiodes immitus*—Table 3) were reported from the northeast Pacific Ocean.

### 3.2. Pathogens potentially involved in population decline

Since the annual census of southern resident killer whales was started in 1974, 78 animals have died. When evaluated by the number of deaths per year, death counts were the highest in 1998 and 2000 (7 per year), however no obvious epizootic patterns were noted (Fig. 1). Of the 78 deaths, 26% ($n=20$) were infants (0–3 years old), 14% ($n=11$) were juveniles (4–11 years old), 4% ($n=3$) were adolescents (12–17 years old), and 56% ($n=44$) were adults (>18 years old) (Fig. 2). When mortality was evaluated by age in years at death, only calves dying in their first year were over-represented ($n=11$), representing 69% of all infant mortalities (11/16) since 1978 (Fig. 3). Supporting this, survival estimates calculated by the US Biological Review Team for Southern Resident Killer Whales (Krahn et al., 2002) found that calves dying in their first year and old males had the lowest survival rates. Because some diseases may preferentially affect calves born to first time (nulliparous) or second time (premiparous) mothers, life history data for mothers of southern resident killer whale calves dying in the first year of life were evaluated by birth order to see if calves born to nulliparous,

Table 1
Infectious diseases reported in free-ranging killer whales

| Pathogen | Reference | Virulence in individuals | Epizootic potential | Ability to reduce fecundity | Ocean |
|---|---|---|---|---|---|
| **Bacteria** | | | | | |
| *Brucella* spp. | Jepson et al. (1997) | Low | Low | High | Northeastern Atlantic |
| *Edwardsiella tarda* | Ford et al. (2000) | High | Low | Low | Northeastern Pacific |
| **Viruses** | | | | | |
| Cetacean pox virus (Orthopoxvirus) | Van Bressem et al. (1999) | Low—Adults<br>High—Neonates | Low | Low | Not Reported |

Table 2
Infectious diseases reported in captive killer whales

| Pathogen | Reference | Virulence in individuals | Epizootic potential | Ability to reduce fecundity |
|---|---|---|---|---|
| **Bacteria** | | | | |
| *Burkholderia pseudomallei* | Hicks et al. (2000) | High | Low | Low |
| *Clostridium perfringens* | Walsh et al. (1994) | High | Low | Low |
| *Erysipelothrix rhusiopathiae* | Bossart et al. (1988) | Unknown | Unknown, likely low | Unknown, likely low |
| *Nocardia asteroides* | Sweeney et al. (1976) | High | Low | Low |
| *Nocardia otitidiscaviarum* | Dunn et al. (2001) | High | Low | Low |
| *Salmonella* sp. | Ridgway (1979) | High | Low | Low |
| *Streptococcus* sp., beta-hemolytic | Greenwood and Taylor (1985) | High | Low | Low |
| **Viruses** | | | | |
| Hepatitis-B like virus | Bossart et al. (1990) | Low | Low | Low |
| Influenza (suspected; no virus isolated) | Ridgway (1979) | High | Low | Low |
| Cutaneous papilloma virus | Bossart et al. (1996) | Low | Low | Low |
| **Fungi** | | | | |
| *Aspergillus fumigatus* | Reidarson et al. (1999) | High | Low | Low |
| *Candida alibicans* | Greenwood and Taylor (1985); Ridgway (1979); Sweeney et al. (1976) | High | Low | Low |
| *Saksenaea vasiformis* | Reidarson et al. (1999) | Unknown | Unknown, likely low | Unknown, likely low |

Table 3
Infectious diseases of free-ranging sympatric odontocete species not reported in free-ranging or captive killer whales

| Pathogen | Species | Reference | Virulence in individuals | Epizootic potential | Ability to reduce fecundity | Ocean |
|---|---|---|---|---|---|---|
| **Bacteria** | | | | | | |
| *Actinomyces bovis* | Bottlenose dolphin | Sweeney et al. (1976) | High | Low | Low | Gulf of Mexico |
| *Helicobacter* sp. | Common dolphin | Harper et al. (2000) | Unknown, likely low | Unknown, likely low | Unknown, likely low | Northwestern Atlantic |
| *Vibrio alginolyticus* | Bottlenose dolphin | Buck and Spotte (1986) | Unknown | Unknown, likely low | Unknown, likely low | |
| | Striped dolphins | Buck and Spotte (1986) | Unknown | Unknown, likely low | Unknown, likely low | Northwestern Atlantic |
| *Vibrio parahaemolyticus* | Bottlenose dolphin | Buck and Spotte (1986) | Unknown | Unknown, likely low | Unknown, likely low | Not Reported |
| **Viruses** | | | | | | |
| Herpesviruses | Bottlenose dolphin | Blanchard (et al. (2001) | High | High | Low | Western Atlantic |
| | Harbor porpoise | Kennedy et al. (1992b) | High | High | Low | Western Atlantic |
| | Harbor porpoise | Lipscomb et al. (1996b) | Low | Low | Low | Western Atlantic |
| Morbillivirus, Dolphin | Bottlenose dolphin | Lipscomb at al. (1994a,b); Taubenberger et al. (1996); Van Bressem et al. (2001) | High | High | High | Western Atlantic; Gulf of Mexico |
| | Striped dolphins | Domingo et al. (1992) | High | High | High | Mediterranean Sea |
| | Short-finned pilot whales | Duignan (1995) | High | High | High | Western Atlantic |
| | Harbor porpoise | Van Bressem et al. (2001) | High | High | High | Northeastern Atlantic; North Sea |
| | Striped dolphin | Van Bressem et al. (2001) | High | High | High | Mediterranean Sea |
| | Risso's dolphin | Van Bressem et al. (2001) | High | High | High | Mediterranean Sea |
| | Common dolphin | Reidarson et al. (1998b) | High | High | High | Northeastern Pacific |
| Morbillivirus, Porpoise | Harbor porpoise | McCullough et al. (1991); Visser et al. (1993) | High | High | High | Northeastern Atlantic; North Sea |
| | Bottlenose dolphin | Taubenberger et al. (1996) | High | High | High | Western Atlantic |
| | Common porpoise | Kennedy et al. (1992a) | High | High | High | Eastern Atlantic |
| **Fungi** | | | | | | |
| *Coccidioides immitis* | Bottlenose dolphin | Reidarson et al. (1998a) | High | Low | Low | Northeastern Pacific |
| *Cryptococcosis neoformans* | Striped dolphin | Gales et al. (1985) | High | Low | Low | South Pacific |
| *Loboa loboi* | Bottlenose dolphin | Caldwell et al. (1975) | Low | Low | Low | Western Atlantic |
| *Rhizopus* sp. | Harbor porpoise | Wunschmann et al. (1999) | High | Low | Low | Baltic Sea |

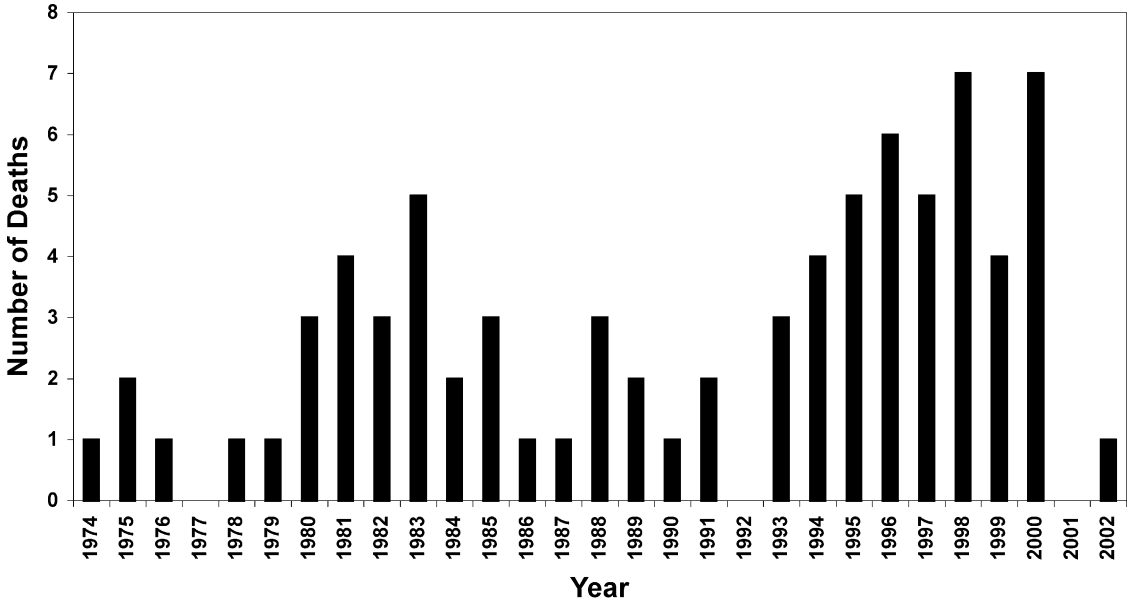


Fig. 1. Southern resident killer whale mortality data by year (1976–2002; $n = 78$ mortalities).

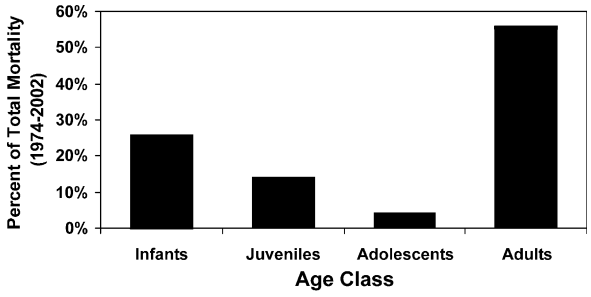


Fig. 2. Southern resident killer whale mortality data by age class (1976–2002; $n = 78$ mortalities).

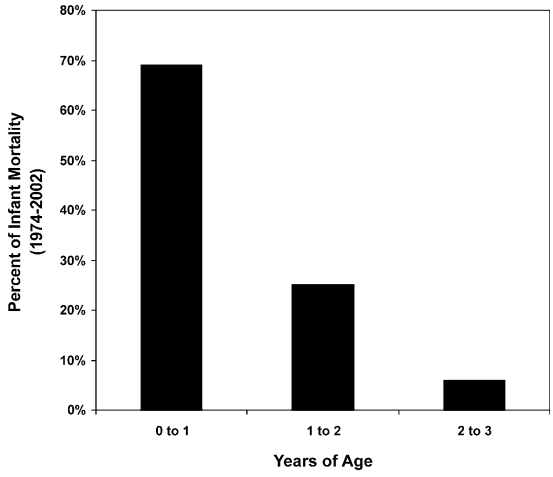


Fig. 3. Southern resident killer whale infant mortality data by age (1976–2002; $n = 16$ mortalities).

premiparous, or multiparous females were over-represented. No apparent trends were noted.

Of the 43 potential infectious disease agents identified, cetacean poxvirus was the only pathogen scored as having a high potential to cause neonatal mortality. Marine *Brucella* was the only pathogen scored as having a high ability to reduce fecundity or affect reproductive success.

### 3.3. Pathogens that may threaten the long-term viability of the population

No pathogen known to infect free-ranging or captive killer whales was scored as having a high potential to cause epizootics; however, such pathogens were identified in sympatric odontocete species and may have the potential to threaten the long-term viability of the small southern resident population. Specifically, morbilliviruses (dolphin and porpoise) and herpesviruses have high potentials to cause epizootics in several species of sympatric odontocete species and may have the potential to negatively affect the long-term viability of the southern resident killer whale population.

## 4. Discussion

Finding only 16 infectious agents reported from free-ranging and captive killer whales exemplifies the severe lack of knowledge about infectious diseases in this species. In comparison, a health protocol for translocation of free ranging elk, *Cervus elaphus*, identified 190 infectious agents and ectoparasites reported in the species (Corn and Nettles, 2001). Complete and standardized postmortem examination of all dead animals from threatened or endangered populations serves as an excellent first step in gathering information about

Table 4
Infectious diseases of captive sympatric odontocete species not reported in free-ranging odontocete species nor in free-ranging or captive killer whales

| Pathogen | Species | Reference | Virulence in individuals | Epizootic potential | Ability to reduce fecundity |
|---|---|---|---|---|---|
| **Bacteria** | | | | | |
| *Nocardia braziliensis* | Bottlenose dolphin | Sweeney et al. (1976) | Unknown | Unknown, likely low | Unknown, likely low |
| *Nocardia caviae* | Bottlenose dolphin | Sweeney et al. (1976) | Unknown | Unknown, likely low | Unknown, likely low |
| *Nocardia paraguayensis* | Bottlenose dolphin | Jasmin et al. (1972) | Low | Low | Low |
| *Pasteurella hemolyticum* | Bottlenose dolphin | Sweeney and Ridgway (1975) | High | Low | Low |
| *Pasteurella multocida* | Bottlenose dolphin | | | | |
| | Common dolphin | Sweeney and Ridgway (1975) | High | Low | Low |
| *Pseudomonas pseudomallei* | Bottlenose dolphin | Sweeney (1986) | High | Low | Low |
| *Staphlococcus aureus* | Bottlenose dolphin | Ketterer and Rosenfeld (1974) | High | Low | Low |
| **Viruses** | | | | | |
| Caliciviruses | Bottlenose dolphin | Smith (1983) | Low | Low | Low |
| **Fungi** | | | | | |
| *Apophysomyces elegans* | Bottlenose dolphin | | | | |
| | Pacific white-sided dolphin | Reidarson et al. (1999) | Unknown | Unknown, likely low | Unknown, likely low |
| *Aspergillus flavis* | Bottlenose dolphin | Sweeney et al. (1976) | Unknown | Unknown, likely low | Unknown, likely low |
| *Blastomyces dermatitidus* | Bottlenose dolphin | Cates et al. (1986) | High | Low | Low |
| *Cladophialophora bantiana* | Harbor porpoise | Reidarson et al. (1999) | Unknown | Unknown, likely low | Unknown, likely low |
| *Histoplasma capsulatum* | Bottlenose dolphin | Jensen et al. (1998) | High | Low | Low |
| *Mucor* sp. | Bottlenose dolphin | Sweeney et al. (1976) | Unknown | Unknown, likely low | Unknown, likely low |
| *Sporothrix schenckii* | Pacific white-sided dolphin | Migaki et al. (1978) | High | Low | Low |
| *Trichophyton* sp. | Bottlenose dolphin | Hoshina and Sigiura in Sweeney and Ridgway (1975) | Low | Low | Low |

infectious diseases in the population. Unfortunately, of the 78 southern resident killer whales that have died since 1974 (Krahn et al., 2002), carcasses have been recovered from only eight known southern residents (one neonate each from J, K, and L pods, J-4, J-18, L-14, L-51, and L-60) (Osborne, 1999; and unpublished data). Complete postmortem examinations were not performed on all recovered carcasses, and necropsy findings are scattered and difficult to find for the animals that did receive postmortem examinations. Our method of literature review for infectious diseases reported in free-ranging and captive killer whales, as well as in sympatric odontocete species, coupled with qualitative scoring of each agent for virulence in individuals, epizootic potential, and ability to reduce fecundity was the best method available for getting baseline information and evaluating the role infectious diseases may play in the decline of this population or in negatively affecting its future viability.

In long-lived species like killer whales, infectious diseases that affect fecundity or reproductive success could significantly impact the population's size and viability. Marine *Brucella* spp. are Gram negative bacteria closely related to better known terrestrial pathogens in the genus *Brucella* (Cloeckaert et al., 2001). Infection by *Brucella* has been documented to cause abortion in captive bottlenose dolphins (Miller et al., 1999); however, the clinical and pathologic significance of infection by these organisms in other marine mammals, including *Orcinus orca*, is not well understood. It is not known if marine *Brucella* infection occurs in southern resident killer whales, although antibodies to *Brucella* spp. have been identified in a free-ranging killer whale (Jepson et al., 1997) and were detected in a female transient killer whale stranded within the range of southern resident killer whale population (CA 189; January 2002, Dungeness Spit, Washington; pers. comm., S. Raverty). It is possible that killer whales within the southern resident population could be infected with marine *Brucella*, and the potential exists for this pathogen to cause abortion and decreased fecundity in this population. More information is needed on the pathogenesis of marine *Brucella* in killer whales as is more information on the prevalence of this pathogen in southern resident killer whales. When the opportunity arises, all captive and free-ranging killer whales should be screened for antibodies to these bacteria, the placenta and tissues from all aborted killer whale fetuses should be cultured for the bacteria, and attempts should be made to isolate the bacteria or amplify *Brucella* nucleic acid from all stranded killer whales.

The southern resident killer whale population (Olesiuk et al., 1990; Krahn et al., 2002), like many wild animal populations (Nettles, 1992), experience highest mortality in the first year age class. Reasons for southern resident calf mortality in year one could include poor mothering, infectious or non-infectious diseases, infanticide, or other causes. Cetacean poxviruses

typically cause cutaneous lesions referred to as "tattoo" or "ring" skin lesions in odontocetes (Van Bressem et al., 1999). Reported clinical and epidemiological data do not suggest poxvirus infection causes high mortality in adult cetaceans (Geraci et al., 1979); however it has been suggested that poxvirus infection could cause neonatal and calf mortality in immunologically naïve cetaceans (Van Bressem et al., 1999). Cetacean poxvirus has been documented to cause cutaneous lesions in killer whales (Van Bressem et al., 1999), but never neonatal calf mortality. It is not know if poxvirus occurs within the southern resident population. More information is needed about this virus and its occurrence in the southern resident population before its role in causing neonatal mortality can be determined. Attempts should be made to isolate the virus from "tattoo" or "ring" lesions found on dead southern resident killer whales and sympatric odontocetes, serum collected should be screened for antibodies to poxviruses, and poxvirus infection should be considered as a possible cause of death in all dead killer whale neonates and calves.

Due to the small size of the southern resident killer whale population and the gregarious social nature of these animals (Dalheim and Heyning, 1999), introduction of a highly virulent and transmissible pathogen has the potential to catastrophically affect the long-term viability of the population. Porpoise and dolphin morbilliviruses are antigenically and genetically similar (Barrett et al., 1993) and are now generally considered strains of the same viral species, cetacean morbillivirus (Kennedy-Stoskopf, 2001). Although precise mortality rates are unknown for epizootics caused by these viruses, they have caused large-scale epizootics in bottlenose dolphins in the Western Atlantic (Lipscomb et al., 1994a) and the Gulf of Mexico (Lipscomb et al., 1996a), and in striped dolphins in the Mediterranean Sea (Forcada et al., 1994). Antibodies to morbilliviruses have been found in free-ranging common dolphins in the northeastern Pacific Ocean (Reidarson et al., 1998b), suggesting that these viruses may occur within the range of the southern resident killer whale population. It is not known if killer whales are susceptible to infection by cetacean morbillivirus. Cetacean morbillivirus infection may be enzootic among short-finned and long-finned (*Globicephala melas*) pilot whales in the western Atlantic Ocean (Duignan et al., 1995). Southern resident killer whales are sympatric with short-finned pilot whales. Cetacean morbilliviruses have a history of being highly virulent in some odontocete species, antibodies have been found in free-ranging common dolphins in the northeast Pacific Ocean, and short-finned pilot whales, a possible reservoir host for these viruses, could potentially transmit these viruses to southern resident killer whales. For these reasons, morbilliviruses represent a potential threat to the long-term viability of the southern resident killer whale population. If cetacean morbillivirus occurs or is introduced into the range of southern resident killer whales and if morbilliviruses prove to be highly virulent in killer whales, an epizootic within the population could have catastrophic consequences and could threaten to the long-term viability of this population. Continued surveillance for antibodies to cetacean morbilliviruses in southern resident killer whales and sympatric odontocetes needs to continue, and morbillivirus infection must be considered a differential diagnosis in all dead southern resident killer whales.

Historically, herpesviruses were thought to cause cutaneous (Van Bressem and Van Waerbeek, 1996) and mucosal (Lipscomb et al., 1996b) lesions in odontocetes. More recently they have been documented to also cause severe encephalitis in harbor porpoises (Kennedy et al., 1992b; Blanchard et al., 2001) in the eastern Atlantic Ocean, and systemic disease in bottlenose dolphins (Blanchard et al., 2001) in the western Atlantic Ocean. Very little is known about the epidemiology of these viruses. Reports of herpesviruses causing fatal disease may represent recrudescence of latent infections, infection in immunologically naïve hosts, or atypical infections in aberrant hosts. Similar viruses have not been identified in the northeast Pacific Ocean, and there is no evidence that killer whales would be susceptible to infection by these viruses if they were present. However, because herpesviruses have been reported to cause severe disease in two species of free-ranging odontocetes and because of the infectious nature of these viruses, they may have the ability to cause large-scale mortality in the southern resident killer whale population. Surveillance for herpesviruses in odontocetes from the northeast Pacific Ocean needs to be started and continuing research on the epidemiology of these pathogens is warranted.

There are potential flaws in assuming that southern resident killer whales are equally susceptible to infection by pathogens known to cause epizootics in sympatric odontocete species. It is not known if pathogens that cause epizootics or severe disease in individual sympatric odontocete species will similarly affect killer whales. Conversely, we cannot be certain that infectious agents in sympatric odontocete species classified as less virulent might not be more virulent in killer whales. High total polychlorinated biphenyls (PCB) concentrations in tissues of southern resident killer whales make them among the most contaminated cetaceans in the world (Ross et al., 2000). Although age may be a confounding factor, it has been suggested that there is an association between cetacean exposure to PCBs and mortality due to infectious diseases (O'Hara and O'Shea, 2001). For example, Jepson et al. (1999) found that harbor porpoises that died from infectious diseases had higher chlorobiphenyl concentrations than those that died from physical trauma. Also, it has been suggested that

the high mortality seen in a morbillivirus epizootic in striped dolphins in the Mediterranean Sea may have been related to high PCB concentrations (Domingo et al., 1992; Aguilar and Borrell, 1994). If high PCB levels in southern resident killer whales increase host susceptibility to infectious agents, pathogens that are not documented to cause severe disease in other odontocete species may be more virulent in this population of killer whales.

Compared to some wildlife species, information about infectious diseases in free-ranging killer whales is sparse, making identification of potentially important pathogens difficult. Despite this, we were able to use the best available existing information about infectious diseases in killer whales and sympatric odontocete species to develop a list of potentially important infectious diseases for the endangered southern resident killer whale population. Although surveillance may identify previously undocumented pathogens, identified diseases will serve as a baseline for a standardized necropsy protocol and disease testing for stranded southern resident killer whales and sympatric odontocetes. Additionally, evaluation of identified infectious diseases targets four specific high priority infectious agents (marine *Brucella*, cetacean poxvirus, cetacean morbillivirus, and herpesviruses) that warrant further study. Infectious disease risk assessments needs to be carried out for all threatened or endangered wildlife populations, even if there is limited information about infectious diseases in the species of concern.

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