In April of 1999 the Committee on the Status of Endangered Wildlife in Canada ("COSEWIC") listed Resident populations of killer whales as threatened, indicating that the Residents are likely to become endangered if nothing is done to reverse the threats facing the populations. However, COSEWIC currently has no legal mandate, and unless the proposed Species at Risk Act ("SARA") is passed, the listing will only serve an advisory role.

4. International Law

The Convention on International Trade in Endangered Species of Wild Fauna and Flora ("CITIES") lists killer whales as an Appendix II species, because commercial products made from killer whales may be indistinguishable from commercial products made from critically endangered whales. As such, international trade of killer whales or parts thereof by any countries that are parties to CITES requires export permits from the country of origin. However, other then requiring documentation of trade between countries, Appendix II listing provides no substantive protection for killer whales, and there are no special rules to protect subpopulations of the killer whale. Furthermore, threats from capture and trade are not pressing issues facing the Southern Residents. Thus, the CITIES regulations are not adequate to preserve this population.

Killer whales are considered "small cetaceans" by the International Whaling Commission ("IWC"), and there is currently considerable disagreement within the Commission as to whether small cetaceans are covered by the Convention. However, in 1980, in response to a large Russian take of killer whales in the Antarctic in the 1979/80 season, the IWC added a new sentence to Schedule paragraph 9(d), officially including killer whales in their moratorium on factory ship whaling (IWC, 1981). Other IWC management measures (e.g., the Southern Ocean Sanctuary, moratorium on commercial whaling, etc.) do not apply to killer whales.

E. OTHER FACTORS

1. Increased Levels of Toxic Chemicals

Southern Residents are contaminated with persistent organochlorines, including DDT and its metabolites, PCBs (polychlorinated biphenyls), dioxins (polychlorinated dibenzo-p-dioxins), and furans (polychlorinated dibenzofurans). Ross et al., (2000) and Jarman et al. (1996) provide the only documentation to date of the extent of organochlorine contamination in the Southern Residents, and are discussed below.

a. Contamination Levels of Southern Resident Killer Whales

Ross et al. (2000) analyzed blubber samples from Northern Residents (N = 26), Southern Residents (N = 6), and Transients (N=15). They analyzed these samples for PCB, dioxin, and furan congeners. In all three populations, total PCB concentrations were high relative to concentrations of PCBs in marine mammals measured elsewhere (Kamrin and Ringer, 1994; Eisler and Belisle, 1996). Southern Resident males were less contaminated than Transient males, but more contaminated than Northern Resident males. Southern Resident females were more contaminated than Northern Resident females, and had nearly the same level of contamination as

the Transient females.

Table 5. Summary of PCB concentrations in killer whales recently sampled near the coast of British Columbia (Ross et al., 2000).

Mean total PCB concentrations mg/kg lipid weight (N)		Population
Males	Females	
146.3 ±32.7 (4)	55.4 ± 19.3 (2)	Southern Residents
37.4 ± 6.1 (8)	9.3 ± 2.8 (9)	Northern Residents
251.2 ± 54.7 (5)	58.8 ± 20.6 (5)	Transients

Lower concentrations of dioxins (1050 ? 258 ng/kg lipid weight) and furans (55 ? 6 ng/kg lipid weight) were observed in the Ross et al. (2000) study. There were no significant differences in dioxin and furan concentrations between the three populations. These results are consistent with observations of pinnipeds and cetaceans elsewhere (Jarman et al., 1996; Muir et al., 1996; Kannan et al., 1989). The relatively low levels of dioxins and furans may be the result of relatively low exposures, or an ability of these animals to metabolize and excrete certain dioxin congeners and structurally related compounds (Tanabe et al., 1988) including some of the PCB congeners. Of the dioxin-like organochlorine compounds known to contaminate Southern Resident killer whales, PCBs appear to constitute the overwhelming majority (Ross et al., 2000).

Jarman et al. (1996), the only other study in which whales from these populations were analyzed for persistent chemicals, corroborates the results in Ross et al. (2000). Jarman et al. (1996) examined the blubber of six killer whales found dead between 1986 and 1989, five of which were recovered within the range of the Southern Resident population, and reported that the geometric mean concentration of total PCBs in blubber of killer whales sampled was 24.2 mg/kg lipid weight.[5] Jarman et al. (1996) also reported concentrations of a wide range of organic pollutants. Notable concentrations of DDT and its metabolites were reported, with a geometric mean total DDT in blubber samples of 35.2 mg/kg DDT lipid weight.[6] Of the mean total DDT concentration, 87.5 percent was *pp'*-DDE, a toxic and persistent metabolite of DDT.

b. Notes On Interpreting Data

Because it is impossible to conduct controlled toxicological studies on killer whales, there are no direct assessments of DDT, PCB, dioxin, and furan toxicities in this species. However, concentrations of PCBs and dioxins in Southern Resident killer whales can be interpreted by comparing concentrations reported by Ross et al. (2000) and Jarman et al. (1996) with concentrations in other mammals in which toxic effects have been observed, or by comparing total dioxin equivalents (TEQs) in Southern Residents to TEQ thresholds.[7] This

[5] Geometric mean calculations tend to be lower than arithmetic mean calculations.

[6] Jarman et al. (1996) reported an average of 91 percent lipid in killer whale tissues. Average organochlorine concentrations (mg/kg) reported as wet weight were divided by 0.91 to estimate lipid weight concentrations.

[7] Certain PCB, furan, and dioxin congeners exhibit toxicological similarities to 2,3,7,8 – tetrachlorinated dibenzo-*p*-dioxin (2,3,7,8 – TCDD). These compounds are called 'dioxin-like' congeners, and are believed to act through the same mechanism in causing some toxic effects. Scientists have derived a system of toxic equivalency factors (TEFs) which relate the potency of each individual 'dioxin-like' congener to that of 2,3,7,8 – TCDD, the most potent of these chemicals. The TEQ is the sum of all the individual dioxin-like congener concentrations multiplied by their respective TEFs, and can be compared to other TEQs known to cause adverse effects, or to

discussion focuses on DDTs and PCBs because of their relatively high concentrations in the Southern Residents, while dioxins and furans are considered only as additive with PCBs, through calculation of TEQs

Many studies have been conducted to characterize the role of organochlorine contaminants in the morbidity and mortality of marine mammals (O'Shea, 1999). Most studies have been of wild mammals or have been conducted using wild foods, exposing test animals to a mixture of contaminants, which can confound interpretation of a specific compound as the direct "cause" of an illness or death. In much of the available research, as in Ross et al. (2000) and Jarman et al. (1996), animals were exposed to substantially higher concentrations of PCBs and DDT and its metabolites relative to concentrations of other chemicals. Therefore, much of the published literature is useful for understanding how PCBs and DDTs may be affecting the Southern Residents.

Metabolism of many toxic PCB congeners appears to be poor in cetaceans relative to other mammals (Tenabe et al., 1988; 1994; Kannan et al., 1989), although studies in both pinnipeds (seals and sea lions) and cetaceans indicate that these taxa metabolize some highly toxic dioxin-like organochlorines more readily than other mammals (Kannan et al., 1989; Ross et al., 2000). Pinnipeds metabolize some of the more toxic PCB congeners more rapidly than cetaceans, and there appears to be some variation among different cetacean species in the ability to metabolize coplanar PCBs (Duinker et al., 1989; Kannan, 1989). Because their metabolic capacities are similar, and because controlled toxicity studies in cetaceans are rare, toxicity studies of pinnipeds provide the best model for understanding toxicity of organochlorine mixtures in cetaceans.

Because of many confounding factors in toxicant effects studies of wild populations, scientists are careful to avoid conclusions of direct causality between organochlorine contamination and mortality or illness in the wild. However, given direct observations of the effects of organochlorines in laboratory studies described below, it is also inappropriate to conclude that organochlorines play no role in killer whale survival. Moreover, killer whales have to survive many stressors simultaneously, including reproductive effort, migration, storms, and local reductions in prey abundance; contaminant exposures must be interpreted in the context of life in the wild. In his review, Geraci (1999) makes this observation regarding immunotoxicity:

> "Starvation and malnutrition can affect a marine mammal's susceptibility to disease by more than one mechanism (Suskind, 1977; Seth and Beotra, 1986). For example, the associated weakness and stress might result in immunosuppression and increased likelihood of secondary infection. The utilization of blubber may lead to the release of fat soluble toxins into the blood stream, with possible consequences to immune function."

Organochlorine concentrations in Southern Resident killer whales exceed known adverse effects thresholds. Their susceptibility to toxicant effects must be considered in light of

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concentrations of just 2,3,7,8 – TCDD known to cause effects. For a complete explanation of this method, see Safe (1994).

reductions in the availability of food and other stressors in their environment.

c. Exposure Pathways of Southern Resident Killer Whales

Ingestion is the primary route of exposure of wildlife to organochlorine contaminants because concentrations of organochlorines in prey are so large relative to concentrations in other media (air or water) to which wildlife are exposed. Complete resolution of the sources and pathways of exposure of Southern Resident killer whales to PCBs and dioxins is not possible with existing information. The high trophic level, known sources of contamination in their habitat, and the fairly long life span of killer whales likely combine to result in the high exposures observed in this population.

Below, available data on Southern Resident killer whale diets and environment are reviewed to aid understanding of how the individuals sampled in the Ross et al. (2000) study could have become so contaminated, and why those individuals are likely not anomalous, but in fact are representative of the Southern Resident population.

The available data indicate that Southern Resident killer whales are 5^{th} or 6^{th} level consumers in Puget Sound and Strait of Juan de Fuca food webs. Specific data describing the Southern Resident killer whale diet are rare. Existing information shows that killer whales feed on both pelagic and epibenthic fish species, and that Pacific salmon, particularly chinook (*Oncorhynchus tshawytscha*) are a primary prey item for Southern Residents. Ford et al. (1998), collected remnants from prey capture events by Southern Resident killer whales and analyzed these "leftovers" (i.e., food particles left behind after a predation event) to identify prey species. Although there are some limitations to this study and biases acknowledged by the authors, the data showed that 96% of observed prey capture events were of salmonids, and of these, 62 percent were chinook salmon. Although chinook were the most common prey, they were not the most numerically abundant of the available fish, which suggests that killer whales seek this species when foraging. In another study, Heimlich-Boran (1988) observed Resident whales in Washington State and southern British Columbia feeding along major routes of salmon migration. These studies do not quantify chinook in the diet of Southern Residents, but illustrate that chinook and other salmonids play an important role in the Southern Resident diet.

Other observations include stomach content information from whales found stranded on beaches and autopsied (Ford et al., 1998). The majority of the killer whales identified as Resident were found to have remains of chinook and other salmon species. Other ingested items included salmon lures and halibut hooks. Also, in two separate stomachs (25 percent of the beached-whale sample) were multiple non-salmon, epibenthic species, including four species of sole, flounder, and several sculpins.

Observations of sole and halibut hooks in the guts of killer whales are important because they indicate that killer whales forage on epibenthic fish assemblages. Southern Residents have regularly been observed diving to greater than 100m depth by Baird et al. (1998). Other data for killer whales in Washington State describe a very diverse diet in killer whales (Scheffer and Slipp 1948), including salmon, birds and other marine mammals, suggesting that this species is opportunistic and responsive to changing availability of prey. These authors stated that killer

whales in Puget Sound have been observed feeding in near shore waters of estuaries, possibly an important source of contaminants to the Southern Resident population. Scheffer and Slipp's (1948) observations were made in the 1930s and 1940s at various locations within Puget Sound. They did not distinguish between fish-eating Residents and mammal-eating Transients.

Killer whale experts have emphasized the role of chinook salmon in the diet of killer whales (Osborne, 1999; Ford et al., 1998). It is plausible that killer whales target chinook because they are, on the average, the largest of the Pacific salmon, and they congregate in groups in the pelagic zone. Salmon may be preferred over epibenthic species because diving to the benthic zone is energetically more costly than capture of salmon within the top 40m of the water column. However, the availability of chinook and other salmon is variable – depending on migrations and annual stock abundance. The degree to which Southern Residents must use other species for food may be dependent on water temperature and seasonal availability. Observations of epibenthic fish remains and halibut hooks in killer whale stomachs suggest that, even if salmon are a primary prey, killer whales may rely on a large variety of fish stocks. Species observed to be killer whale prey are relatively long-lived, feed on or near the bottom, or are themselves predators. These characteristics of killer whale prey contribute to higher exposures to bioaccumulative contaminants among killer whales.

A survey of near shore marine food webs in Washington by Simenstad et al. (1979) identified 16 food webs in Puget Sound and the Strait of Juan de Fuca. Of the discrete food webs that exist in nearshore habitats of the study region, the killer whales belong to the 2 neritic food webs, and the shallow sublittoral food web. These food webs are complex, with 38 and 41 nodes, respectively, and link the killer whales to near shore materials, including contaminated sediments. Simenstad et al. (1979) describe killer whales as third or fourth level consumers in diagrams of both neritic and shallow sublittoral food webs. Juvenile salmon are included in this description, but adult chinook and other salmon are considered to be outside the "nearshore" food webs.

Groot and Margolis (1991) summarized the literature on chinook food habits in the region inhabited by Southern Resident killer whales. Because the data were derived from the commercial fishery, larger chinook are emphasized. In the Strait of Georgia, small fish, particularly herring, constituted 28 to 63 percent of the diet of chinook. In a separate study, 79 percent of chinook stomach contents were fish in the Strait of Georgia. Pelagic invertebrates were also important to adult chinook. In the waters of British Columbia, herring, sand lance and pilchards were most important to chinook diets, with herring dominating stomach contents in many studies. Coho salmon, also prey of killer whales, begin to eat fish as smolts, and use fish and marine invertebrates during the early part of their marine life stage. In the Strait of Georgia, coho also use herring, sand lance, and other fish, and pelagic invertebrates. Overall, coho and chinook salmon have very similar diets, but the coho use a greater proportion of invertebrates than chinook (Groot and Margolis, 1991). Thus, coho and chinook are at roughly equivalent trophic levels. Their predation on fish makes them at least third, and possibly fourth level consumers, making Southern Resident killer whales fourth and possibly fifth level consumers.

d. Sources of Contaminants

Because the prey of Southern Resident killer whales consists of wide-ranging species such as chinook and coho salmon, and killer whales are themselves wide ranging, it is not possible to pinpoint a single source of persistent organochlorines to the killer whale diet. Several factors can be considered to understand the likely pathways of contaminants to the Southern Resident killer whales.

i. Organochlorine sources in Puget Sound

According to the Environmental Protection Agency, there are 16 Superfund sites in the Puget Sound Basin at which PCBs are a contaminant of concern, and at least 7 of those include marine or freshwater sediment contamination. See Appendix D. Many are also sources of DDTs, heavy metals, and other pesticides. Most of these were the site of industrial operations conducted over 20 to 70 years of the 20th century, and most of these sources are either now contained, or undergoing containment. Several important point sources of organochlorines exist in Puget Sound, including Elliott Bay, Commencement Bay, Port Hadlock, the Whidbey Island Naval Air Station (Ault Field), The Puget Sound Naval Shipyard Complex, and the Keyport Undersea Warfare Engineering Station.

Even though the most concentrated areas of contamination at these sites may be addressed through the Superfund process, available data suggest that ecological processes have contributed to the dispersal of these contaminants throughout Puget Sound. See Appendix C.

ii. Contamination of the killer whale food web

The proximity of Southern Resident killer whales to Puget Sound hazardous materials sites likely explains their high organochlorine exposures relative to the Northern Resident killer whales (Ross et al., 2000), which have a diet similar to that of Southern Resident killer whales (Ford et al., 1998). Data summarized in Appendix C shows that marine organisms (mussels and sole) collected from highly contaminated urban waterways such as Commencement Bay and Elliott Bay are very contaminated, but also that PCBs are dispersed fairly broadly into rural bays such as Useless Bay at the southern end of Whidbey Island (Ylitalo et al., 1999).

When contaminant concentrations in samples from various points across the entire range of Puget Sound, the Strait of Juan de Fuca, and southern Georgia Strait are averaged, both DDT and PCB contamination of the food web seem substantial. Average PCB concentrations in fish from this region ranged from 0.39 to 10.07 mg/kg lipid weight, and DDTs ranged from 0.34 to 2.82 mg/kg lipid weight. In contrast, Kawano et al. (1986) reported PCBs in salmon from the Pacific Ocean and the Bering Sea at 0.090 mg/kg lipid weight, and total DDTs at 0.076 mg/kg lipid. Puget Sound chinook analyzed by O'Neill et al. (1995, 1998) had an average concentration 28 times higher than this chum salmon from the Pacific, and the average coho from Puget Sound was 19 times more contaminated than open ocean chum. The maximum PCB concentration in Puget Sound chinook was more than 80 times higher than the mean reported by Kawano et al. (1986). The relative concentrations of DDT in Puget Sound salmon were also

elevated, with the average DDT concentrations in coho and chinook 11 and 26 times more than Kawano et al.'s (1986) chum, respectively.

Some of the killer whale prey reported by Ford et al. (1998) do not migrate great distances, and will transfer contaminants to killer whales according to contaminant levels in their local habitats (e.g., Puget Sound harbor seals of Hong et al., 1996). While chinook and coho may migrate to open ocean habitats to mature, there is evidence of "resident" chinook stocks in inside waters of Washington. In mark-recapture studies reviewed by Groot and Margolis (1991), there was limited dispersal of chinook tagged in inside waters of British Columbia and Washington State. The existence of a chinook stock restricted to the inside waters of Washington State could explain why chinook consistently have higher PCB concentrations than coho (there is no evidence of a restricted stock of coho in inside waters), even after lipid content is accounted for (O'Neill et al. 1995), and could explain why chinook from Puget Sound are much more contaminated than open ocean chum reported by Kawano et al. (1986). A chinook stock with limited migrations outside of Puget Sound and the Straits of Juan de Fuca and Georgia would undoubtedly constitute an important part of the Southern Resident killer whale diet, and, if O'Neill et al.'s (1995, 1998) data are representative of such a stock, could explain the very high levels of persistent organochlorines in Southern Resident killer whales.

iii. Global atmospheric transport

Transfer of persistent organochlorines to marine environments via the atmosphere is also substantial (Iwata et al., 1993). On the basis of the Henry's Law constants of individual congeners, Iwata et al. (1993) estimated the potential of long range transport of PCBs and DDTs. They concluded that marine environments in the northern latitudes are a sink for PCBs released to the global atmosphere. DDTs are less likely to be transported across long distances, and tend to be absorbed into marine waters close to the point of release.

e. Bioaccumulation of Persistent Organic Contaminants

Increasing concentrations of organic contaminants at higher trophic levels is well established (e.g., Eisler, 1986; Mamontov et al., 1997). DDTs, PCBs, dioxins and furans are readily absorbed following ingestion (e.g., studies reviewed by Smith, 1991; O'Connor, 1984), and only some of the congeners are metabolized and excreted, while others remain in the body. In longer lived species, the effect is cumulative as the animals build up the contaminants to which they have been exposed over the years. Whales live a long time and, with the exception of breeding females, their body burdens generally increase with age (Ross et al., 2000). Bioaccumulated contaminants can be mobilized in time of stress such as when reductions in food supplies require that the animals draw on their fat reserves for energy, or during reproduction.

The chronic, low-level inputs of atmospheric sources and the slow distribution of PCBs and DDTs from point sources in Puget Sound are the most likely sources of persistent organochlorines in the diet of Southern Resident killer whales. The length and complexity of the food webs in Southern Resident killer whale habitats, and the long-term exposure of individuals explain how low levels of persistent contaminants in abiotic media become dangerously high in top predators.

f. Organochlorine Toxicity

Numerous adverse health effects have been observed in many mammal species following exposure to PCBs, dioxins, and furans. The severity and types of effects observed depend on the age and sex of the animal, the species, and the route and duration of exposure. Safe (1994) summarized the range of effects that have been observed in laboratory studies using commercial mixtures (i.e., PCB mixtures used in commercial applications, such as Aroclors):

> "acute lethality, hepatomegaly [enlarged liver cells], fatty liver and other indications of hepatotoxicity, porphyria, body weight loss, dermal toxicity, thymic atrophy, immunosuppressive effects, reproductive and developmental toxicity, carcinogenesis, other genotoxic responses, modulation of diverse endocrine-derived pathways, and neurotoxicity."

In general, the effects of chronic ingestion of DDT and its metabolites are similar. A summary of DDT effects in mammals (Smith, 1991) includes increased risk of mortality and liver tumors, alteration of metabolic and other enzyme systems, neurological effects (e.g., tremors), estrogenicity, reproductive effects such as failure to reach estrus and poor survivorship of young, and cancer promotion.

i. Immunotoxicity

Brouwer et al. (1989) conducted a two-year controlled laboratory study in which harbor seals (*Phoca vitulina*) were exposed to fish from a DDT- and PCB-contaminated source (the Wadden Sea, Netherlands) and compared to seals fed fish from relatively uncontaminated waters. Seals were exposed to 1.5 mg/d PCBs and 0.4 mg/d pp'-DDE (treatment group), or 0.22 mg/d PCBs and 0.13 mg/d pp'-DDE (control). Comparison of biochemical indicators between the two groups showed that chronic exposures of the seals to this mixture of organochlorine compounds resulted in reductions in plasma proteins and thyroid hormone levels. Brouwer et al. (1989) interpreted these biochemical alterations to be sufficient to cause reproductive impairment and immunotoxicity in these seals. In a series of studies (summarized by Ross et al., 1996a) harbor seals were fed Baltic Sea fish containing high levels of total DDTs (2,155 µg/kg lipid weight), total PCBs (4,398 µg/kg lipid weight), and some dioxins and furans for 2 years. This level of exposure resulted in dose-related reductions in T-cell function, natural killer cell activity, and vitamin A levels, all indicators of immunotoxicity.

Ross et al. (2000) calculated TEQs in the average middle-aged adult Southern Resident killer whales and compared them to TEQs known to cause suppression of immune function in pinnipeds.[8] According to Ross et al.'s (2000) analysis, all of the male Southern Resident killer whales analyzed in their study, and most of the females, currently have body burdens which exceeded immunological effects thresholds established for seals (DeSwart et al., 1996; Ross et al., 1996b).

Examples of high organochlorine concentrations in marine mammals stricken by viral

[8] These effects-threshold TEQs were derived by Ross et al. (2000) using toxicity thresholds derived from laboratory studies. See Ross et al. (2000) for the derivation of this TEQ threshold for pinnipeds.

epizootics are common in the literature (Geraci et al., 1999). An epizootic of morbillivirus killing 18,000 harbor seals (*Phoca vitulina*) in northern Europe in April of 1988 partly occurred in areas of high contamination in the Wadden and North Seas. Seals that were found dead after this disease outbreak had concentrations of 0.96 – 7.99 mg/kg wet weight total DDT in blubber and 5 – 46 mg/kg PCBs (Hall et al., 1992), levels lower than the average in Southern Resident killer whales. Striped dolphins (*Stenella coeruleoaba*), which succumbed to an epizootic in the Mediterranean Sea in 1991, contained high levels of PCBs (100 – 500 mg/kg lipid; Aguilar and Borell, 1994). PCB and DDT concentrations in Baikal seals (*Phoca sibirica*) are among the highest in pinnipeds anywhere in the world (3.5- 64 and 4.9-160 mg/kg lipid, respectively; Nakata et al., 1995; Kucklick et al., 1994). This population suffered a reduction of several thousands due to a viral epizootic in 1987 (Grachev et al., 1989). In St. Lawrence beluga whales (*Delphinapterus leucas*), the frequent occurrence of infections due to relatively mild infectious bacteria has been interpreted to be the result of immunosuppression due to chronic organochlorine exposures in this population (DeGuise et al., 1995). These whales had an average DDT concentration of 81.1 mg/kg lipid in blubber, and average total PCB concentration of 78.9 mg/kg lipid, somewhat higher, but within an order of magnitude, of mean concentrations in Southern Resident killer whales.

ii. Reproductive Toxicity

In another study of Wadden Sea harbor seals, Reijnders (1986) did not detect alteration of two blood hormones (oestradiol-17ß, progesterone), but did document significantly lower reproductive success in seals chronically exposed to PCBs and metabolites of DDT. Concentrations of contaminants in the food of the seals were not reported by Reijnders (1986), but estimated doses were reported as 1.5 mg/d PCBs and 0.4 mg/d pp'-DDE in seals with poor reproduction, and 0.22 mg/d PCBs, and 0.13 mg/d pp'-DDE in control seals. Reijnders (1986) noted that hormone profiles of the affected group indicated that the reproductive failure occurred around the implantation stage. This finding is significant because it corroborated observations of the process of reproductive failure in PCB-dosed mink, suggesting that the same toxicological mechanism is in effect in both species.

Impaired reproduction has been observed in studies of sea lions (*Zalophus californianus*) in environments contaminated with PCBs and dioxins (summarized by Kamrin and Ringer, 1996). These studies, while lacking the control of laboratory experiments, inform assessment of risk to Southern Resident killer whales. In two separate studies, sea lions giving birth prematurely on the west coast of the United States had significantly higher concentrations of PCBs in blubber (mean = 112 ppm wet weight, or about 133 ppm lipid weight) than in females bearing young normally (mean = 17 ppm wet weight or about 20 ppm lipid weight; DeLong *et al.* 1973). DeLong et al. (1973) observed concentrations of total DDTs 8 times higher in blubber of sea lions bearing young prematurely (mean = 824 ppm wet weight or about 980 ppm lipid weight) than in sea lions bearing young normally (mean =103 ppm wet weight or about 121 ppm lipid weight). Gilmartin *et al*. (1976) observed similarly high total DDTs (mean = 651 ppm wet weight) and PCBs (mean = 57.2 ppm wet weight) in female sea lions bearing young prematurely. While sea lions in both of these studies had very high total DDTs, their PCB levels in blubber are comparable to those observed in Southern Resident killer whales.

Other studies attempting to correlate PCB exposures with reproductive effects in wild pinnipeds are difficult to interpret due to low sample sizes and confounding variables.

iii. Endocrine Disruption

The role of organochlorines as endocrine disrupters in mammals is complex. The evidence for alteration of endocrine systems in marine mammals is mixed, showing that some endocrine pathways are clearly affected by organochlorines administered through food (Brouwer et al., 1989) while other endocrine biomarkers are unaffected by the same chemical mixtures (Reijnders, 1986). One additional study of endocrine effects in marine mammals is worth noting. Subramanian et al. (1987) documented a significant negative correlation between concentrations of DDE (7.61 – 16.5 mg/kg wet weight) in the blubber of Dall's porpoises (*Phocoenoides dalli*) and testosterone in the blood of these specimens. Also, a negative correlation between PCBs in blubber (5.62 – 17.8 mg/kg wet weight) and reduced testosterone was non-significant but "apparent," according to the authors, from a scatter plot. A second hormone (aldosterone) functionally not directly connected to reproduction but to regulation of blood sodium, showed no correlation at all with organochlorine exposure. DDT and PCB concentrations in blubber of Southern Resident killer whales exceed concentrations in blubber of affected porpoises in the Subramanian et al. (1987) study.

iv. Effects on Early Life Stage Development

In mammals, fats stored by the female directly support the life of a developing fetus and are also transferred to the young following birth via lactation. Because organochlorine contaminants are stored in fats, juveniles in a contaminated population begin to be exposed to organochlorines very early in their development, probably shortly after conception. In Southern Resident killer whales, adult females have the lowest body burdens of fat-soluble organic contaminants of any segment of the population (Ross et al., 2000). This is because during the processes of fetal development and lactation, breeding females impart substantial loads of organic contaminants to their young, from 20 to 90 percent of the mother's organochlorine load in pinnipeds and cetaceans studied (Nakata et al., 1998). Because the milk of cetaceans and pinnipeds is very high in fat, the majority of the dose received by juveniles prior to weaning is thought to be through lactation (Nakata et al., 1995).

Early life stage exposure to DDT and metabolites, PCBs, dioxins and furans is an important risk factor for this population. In rats, perinatal exposures to the mix of organochlorines present in Baltic Sea fish (discussed above) resulted in more severe immunotoxicity than exposures in previously unexposed adults (Ross et al., 1996a and supporting studies). Only after dosing ceased did the juvenile rats begin to recover from the immunotoxicity. Perinatal exposures of humans to PCBs has been linked to defects in cognitive functioning (Jacobson et al., 1990), and perinatal exposures in non-human primates has been linked to long-term behavioral dysfunction (Schantz et al., 1991). In addition, estrogenicity or other endocrine-like reactions of these chemicals can affect the "organization" of developing embryos (Guillette et al., 1995). Embryos are particularly susceptible to the action of hormones and hormone-like compounds because many embryonic cells have receptors for hormones even before the embryo itself is synthesizing hormones. Thus the developing embryo will be

responsive to the presence of hormones and hormone-like compounds to which it is exposed *in utero*, via the mother's bloodstream. Because hormones and hormone-like materials affect the organization of the developing organism, the effects are permanent. For example, female guinea pigs neonatally dosed with testosterone exhibited altered (masculine) mating behavior as adults (Phoenix et al., 1959). On the basis of the guinea pigs' behavior and physiology, the authors of this study concluded that the prenatal exposures affected both neural and genital tissues. *In utero* and early life stage (*post-partum*) exposure of Southern Resident killer whales to organochlorines which are immunotoxic, neurotoxic, and estrogenic or otherwise endocrine-disrupting increases the susceptibility of the Southern Resident population to lasting effects of organochlorine toxicity.

v. Other Possible Toxic Effects

On the basis of studies in other mammals, additional adverse health effects of DDT and metabolites, PCBs, dioxins, and furans are possible in killer whales, and even likely in individuals with high exposure. Exposure to mono-*ortho* and di-*ortho* PCB (non dioxin-like) congeners and metabolites may result in effects not mediated by the same biochemical pathways as 2,3,7,8-TCDD, and therefore not predicted by TEQs. Such effects include neurobehavioral, neurochemical, carcinogenic, and endocrinological changes (Ahlborg et al., 1992). Because these types of effects are difficult to observe in wild populations, there is no way to account for such effects in Southern Resident killer whales with available information.

Direct assessments of DDT, PCB, dioxin, and furan effects in many species of mammals (as well as fish and birds) have proven these organochlorines to be potent agents of numerous adverse health effects (Eisler and Belisle, 1996; Eisler, 1986; Smith, 1991). For example, Beland et al. (1993) and DeGuise et al. (1995) documented high incidences of tumors, including malignant neoplasms, in St. Lawrence beluga whales contaminated with several organochlorine types, including DDT and metabolites (3.36 – 389 mg/kg lipid weight in blubber), PCBs, (8.3 – 412 mg/kg lipid weight in blubber) and lower levels of dioxins and furans (Muir et al. 1996). From a population estimated at 500 animals, 18 collected post-mortem had tumors, a rate of 3.6 percent. The possibility that such effects occur in Southern Resident killer whales is relevant to its risk of extinction: an animal fighting an infection or the development of a tumor, one that has neurobehavioral abnormalities, liver disease or an altered endocrine system, or some combination of these effects, will be less fit for survival in the wild.

g. Summary

Contaminant concentrations measured in Southern Resident killer whales are likely sufficient to result in adverse health effects in these animals. Total dioxin equivalents (TEQs) in Southern Resident killer whales exceed TEQ thresholds of immunotoxicity derived for pinnipeds. Concentrations of DDTs and PCBs in Southern Resident killer whales exceed those found in pinnipeds and cetaceans stricken by morbillivirus in Europe and central Asia. Exposure of early life stages to organochlorines may be enhancing susceptibility to immunotoxicity, as well as causing permanent physiological alterations via endocrine disruption during development, and possibly altering neurological functioning. Reproductive effects may be occurring in the population via reductions in testosterone in males, and implantation failure in

females, both of which have been observed in similarly exposed pinnipeds. Poor survivorship of young after birth is also possible given existing body burdens in Southern Resident killer whales relative to affected pinnipeds in southern California. Concentrations of organochlorines in Southern Resident killer whales are within the range of those in St. Lawrence beluga whales, 3.6 percent of which have cancerous and other tumors.

Because Southern Resident killer whales occupy a high trophic level, live for a relatively long time, and regularly forage in Puget Sound which is the location of numerous sites contaminated with PCBs and DDTs, and because these contaminants do not readily degrade, exposures in this population can be expected to continue to be high for many years to come. The presence of other environmental stressors such as changing weather patterns, reductions in prey abundances, and human encroachment on habitats is likely to exacerbate the role of contaminants in the decline of this population.

2. Risks of Rarity

It is clear that the effective population of the Southern Residents is quite small. The low number of individuals within the population makes the Southern Residents particularly susceptible to stochastic perturbations. There are four types of stochastic perturbations that small populations may be subject: demographic stochasticity, environmental stochasticity, genetic stochasticity, and natural catastrophes.[9]

Demographic stochasticity refers to accidental variations in birth rate, death rate, and the ratio of the sexes. Environmental stochasticity refers to fluctuations in weather, in food supply, and in the population levels of predators, competitors, parasites, and disease organisms that may affect the killer whale population. Genetic stochasticity refers to the loss of specific alleles through the processes of genetic drift, and the increased expression of the genetic load of the population. All of these stochastic effects create survival risks for populations. Indeed, these stochastic factors, combined with the effects of natural catastrophes, can interact in a dire feedback cycle by which a small population spirals to extinction.

In general, when the effective population of a species falls below 500 individuals, the population faces an overall net-loss of genetic variability through the loss of rare alleles, known as genetic drift. In populations below this size, the gains of genetic diversity brought on through mutation are outpaced by the loss brought on by genetic drift. As the population continues to decline, the rate of loss tends to increase, because smaller populations have smaller rates of mutation. Overall, this effect leads to a loss of long-term genetic adaptability within the population. (Franklin, 1980).

Further genetic risks occur when a population declines to 50 individuals. At this point, the population becomes susceptible to inbreeding depression, i.e., the increased expression of deleterious alleles. For populations with a large genetic load, inbreeding can be particularly devastating. However, a population that historically has low population numbers will likely have a low genetic load (otherwise the relatively small population would not have survived over time),

[9] *See* Mark L. Shaffer, Determining Minimum Viable Population Sizes: A Case Study of the Grizzly Bear (*Ursus arctos L.*), (1978) (unpublished Ph.D. dissertation, Duke University).