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# Cumulative Effects Assessment for Northern and Southern Resident Killer Whale Populations in the Northeast Pacific

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## Foreword

This series documents the scientific basis for the evaluation of aquatic resources and ecosystems in Canada. As such, it addresses the issues of the day in the time frames required and the documents it contains are not intended as definitive statements on the subjects addressed but rather as progress reports on ongoing investigations.

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# ABSTRACT

The Northern and Southern Resident Killer Whale populations (NRKW and SRKW) that inhabit the waters of the Canadian Pacific coast are listed as Threatened (NRKW) and Endangered (SRKW) under the Species at Risk Act (SARA). The SARA recovery plan developed for these populations identified the assessment of the cumulative effects of anthropogenic threats impacting these populations as a high priority. To address this, a cumulative effects assessment framework was developed and applied comprising two components: a Pathways of Effects (PoE) conceptual model and a subsequent Population Viability Analysis (PVA) model. The PoE model summarises the current understanding of each priority threat (prey availability, disturbance and contaminants) and describes the structure of the threats in the assessment, including threat interactions and potential impacts to population parameters (fecundity and mortality). The PoE model forms the basis for the subsequent PVA model, which utilises the most recent available threat data to quantify the way threats impact population parameters and, together with demographic data, explore patterns of population growth and decline in different threat scenarios. The impacts of individual and cumulative threat scenarios on modelled SRKW and NRKW populations were compared to the observed population trajectories (2000-2017) in order to define a model that best captured the real world dynamics of the two populations. Of the various individual and combined threat models tested, the cumulative threats model, which incorporated all priority threats (Chinook salmon abundance, vessel noise/presence, vessel strike, and PCB contamination), predicted demographic rates closest to that observed for both populations. Population dynamics predicted by the model closely followed the observed demographics for NRKW and though it was the closest model to the observed population size for SRKW, it did not include the observed values within the bounds of uncertainty. However, when historical Chinook salmon model data were included in the model prediction, rather than a randomly chosen Chinook salmon index value, the fit improved for SRKW and the uncertainty bounds of both models included the observed values, suggesting that the cumulative model is a useful representation of the system.

The findings of this cumulative effects assessment highlight the importance of considering threats collectively. Specifically, within the cumulative effects PVA assessment, Chinook salmon abundance and its interactions with vessel noise/presence and PCBs strongly influenced modelled killer whale population dynamics. The cumulative effects PVA model was also used to project population trajectories for NRKW and SKRW into the future. The model outputs indicate that the mean modelled NRKW population trajectory increased to the carrying capacity set in the model within 25 years. In contrast, the mean modelled SRKW population trajectory declined, with a 26% probability of population extinction (defined in the model as only one sex remaining), and in those projections, extinction was estimated to occur after 86 (± 11) years. The cumulative effects assessment framework developed, that combines a PoE with a PVA model, is a novel approach that explicitly identifies and quantifies threat linkage pathways, and associated uncertainties. The framework is a potentially useful tool for managers and scientists and has been refined and tested with the latest threat information for these populations but could also be applied to other populations and species. It is cautioned that as model outputs are only as good as the model inputs, changes in exposure to natural and anthropogenic threats can affect the model's accuracy. An iterative approach should be used so that model inputs and structure are regularly reviewed and updated to include new information about existing threats and the addition of new threats as knowledge is increased on these populations.

# 1 INTRODUCTION

## 1.1 BACKGROUND

Three genetically and acoustically distinct killer whale (*Orcinus orca*) ecotypes inhabit the waters of the Northeast Pacific coast of North America: offshore, Bigg's (or transient), and residents (Ford et al. 1998). The resident fish-eating ecotype is further divided into the Northern and Southern Resident Killer Whale (NRKW and SRKW) and the Southern Alaskan Resident Killer Whale populations (SARKW) (Ford et al. 2000; Matkin et al. 1999; 2014). Though all populations of Resident Killer Whales are fish-eating cetaceans, feeding primarily on Chinook (*Oncorhynchus tshawytscha*) and Chum salmon (*O. keta*), and overlap to some extent in habitat and diet, they do not interact with one another socially and are distinct in terms of their culture, acoustics, and genetics (DFO 2017a).

The NRKW and SRKW populations were listed as Threatened (NRKW) and Endangered (SRKW) in Schedule 1 under the Species at Risk Act (SARA) in 2003. The NRKW range includes the coastal waters from Glacier Bay (Alaska, USA) to Gray's Harbor (Washington State, USA), and the SRKW range extends from southeastern Alaska to central California (Ford et al. 2000, 2006).

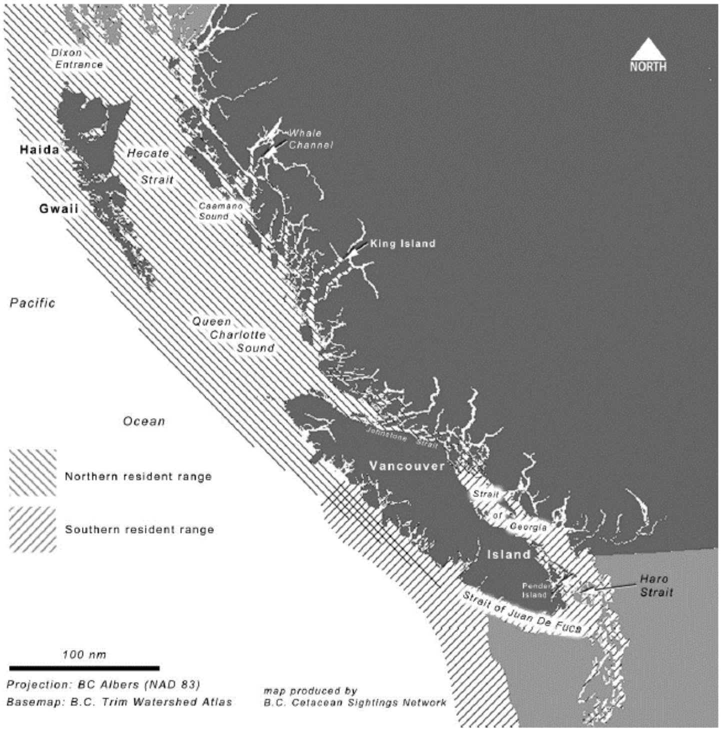


*Figure 1 - Overlapping ranges of Northern Resident and Southern Resident Killer whales centered in Canadian waters (after DFO 2018a).*

In summer, the movements and habitat use by Resident Killer Whale populations often reflects the timing and locations of inbound Pacific salmon migrations. The SRKW population tends to concentrate in the waters of southern Vancouver Island and northern Washington State while a portion of the NRKW population are often found frequenting Johnstone Strait and Queen Charlotte Strait (Figure 1; DFO 2018a). The SRKW range has a higher overlap with major coastal population centres (Vancouver and Seattle) than the NRKW population and consequently, is more likely to be exposed to, and potentially impacted by anthropogenic threats such as contaminants and vessel related threats (noise and physical disturbance) (Krahn et al. 2004; Wiles 2004; Figure 1).

### 1.1.1 Population Trends

Long-term photo-identification census surveys for both populations were initiated by Michael Bigg in the 1970s and continue to the present day (DFO Cetacean Research Program; Center for Whale Research, CWR). The SRKW census (begun in 1976) is considered to be more accurate than the NRKW census (begun in 1973), as not all members of the Northern population are seen each year (DFO 2018a).

Population trends based on the census data indicate that the SRKW population has experienced an overall negative population growth rate (-0.002; 1979-2017), but experienced particularly sharp declines between 1995 and 2001 (Figure 2). Since then, the population has shown little recovery, having 77 members in December 2017. In contrast, the NRKW population has experienced a steady increase over the census period (population growth rate = 0.02; 1979-2017), except for a decline between 1997 and 2001 (Figure 2). The population has since increased from 219 members in 2004, to 308 members in 2017 (41% increase).

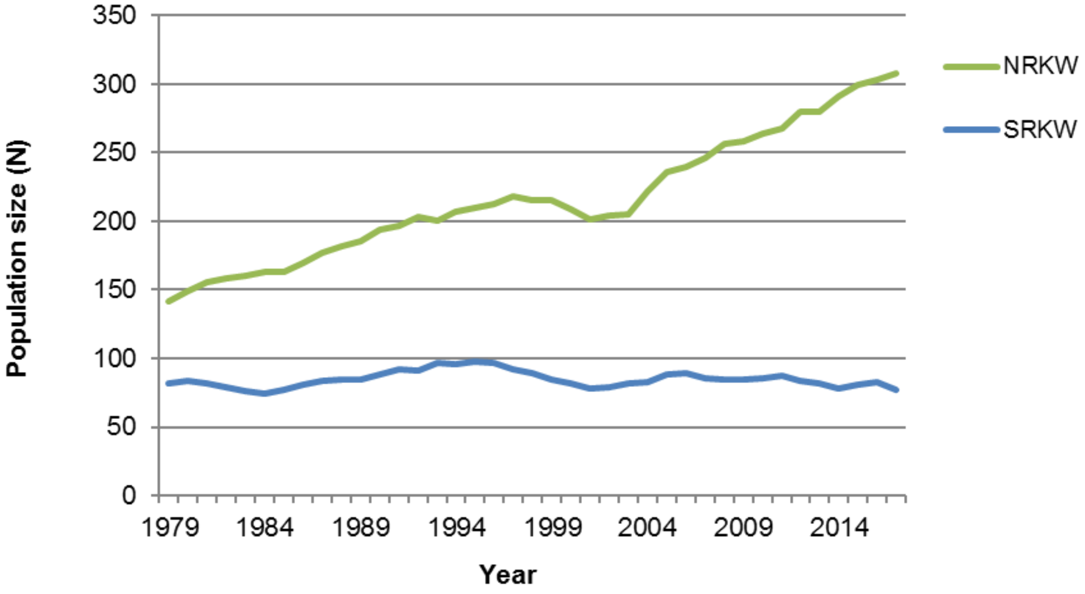


*Figure 2 – Resident killer whale population time series (data shown 1979-2017).*

### 1.1.2 Goal of the Assessment

The Southern and Northern Resident populations were listed as Endangered and Threatened, respectively, under SARA in 2003. Under SARA, the federal government has a commitment to prevent wildlife species from being extirpated or becoming extinct, to provide for the recovery of wildlife species that are Extirpated, Endangered or Threatened as a result of human activity and to manage species of Special Concern to prevent them from becoming Endangered or Threatened. The Minister of Fisheries and Oceans Canada (DFO) is the competent minister for the recovery of aquatic species at risk.

The three primary stressors (from hereon referred to as threats) to NRKW and SRKW identified by the DFO Species at Risk Program (COSEWIC 2009; DFO 2011; DFO 2017b) are:

1. Reduced prey availability,
2. Acoustic and physical disturbance, and
3. Environmental contaminants

There is a legal requirement to assess cumulative effects within the Canadian SARA action plan for NRKW and SRKW (DFO 2017b).Three of the 98 Recovery Measures (RMs) in the action plan relate (directly or indirectly) to cumulative effects (RM 6, 11, 17), and the focus of this assessment is to address RM 11 (Table 1).

*Table 1 - SARA Recovery Measures for Resident Killer Whales related to cumulative effects.*

| # | **Recovery Measure** | **Priority** |
|---|---|---|
| 6 | Take into account both the seasonal (acute) as well as the cumulative (chronic) effects of poor returns for Chinook and other important prey species on Resident Killer Whales when managing fisheries. | High |
| 11 | Assess cumulative effects of potential anthropogenic impacts on Resident Killer Whales using an appropriate impact assessment framework for aquatic species. | High |
| 17 | Review and assess project impacts on Resident Killer Whales and their habitat, and provide advice on avoidance and mitigation measures as required. | High |

## 1.2 WHY A CUMULATIVE EFFECTS ASSESSMENT IS REQUIRED

DFO's Species at Risk Program has requested that the Science Branch provide an assessment of the cumulative effects of the three primary anthropogenic threats on NRKW and SRKW populations. To date, most research on threats to killer whales has studied these threats in isolation, for instance focusing solely on prey availability or acoustic disturbance. Assessment of cumulative effects, however, involves examining the combined, incremental impacts that threats from multiple human activities can have on individuals, populations, communities and ecosystems through space and time. Cumulative effects assessments evaluate the effects of multiple threats by converting impacts into a single currency or metric, thereby allowing for comparisons among threats and their combined long-term impact.

This study provides an opportunity to incorporate the best available scientific information into a single assessment that includes all three threats, the interactions between these threats, and the resulting long-term impacts on the population. Previous cumulative effects assessments (CEAs) fall into three categories: risk assessment, statistical analysis, and population viability analysis (Lawson and Lesage 2012; O et al. 2015; Williams et al. 2017; Lacy et al. 2017). An example of risk assessment is the general framework to evaluate the relative additive cumulative risk for a range of activities and ecosystem components that was developed by O et al. (2015). Statistical models have been used to evaluate the impact of single threats on mortality and fecundity of Resident Killer Whales (Ward et al. 2009; Vélez-Espino et al. 2014b). Williams et al. (2017) developed a cumulative population viability analysis (PVA) model to quantify factors limiting the recovery of the St. Lawrence Estuary Beluga population and Lacy et al. (2017) evaluated the cumulative effects of anthropogenic threats on SRKW using a PVA.

A DFO framework has been developed to quantify and cumulate risks of impacts for marine mammal populations, the Cumulative Effects Risk Assessment Framework (CERAF) (Lawson and Lesage 2012; DFO 2017c; Figure 3). While the current work does not explicitly utilise the CERAF, for comparison, the current work fits within the CERAF steps (Lawson and Lesage 2012; DFO 2017c). The scoping and relative risk phases (Box A, B, C) were conducted previously, either explicitly or implicitly in the various recovery documents that identified the most important threats to SRKW and NRKW (Figure 3). The focus of the current work corresponds to the last step in the CERAF (Box D), i.e., taking the highest risk threats and assessing them together in a viability analysis to investigate the cumulative effects on the long-term persistence of the population.

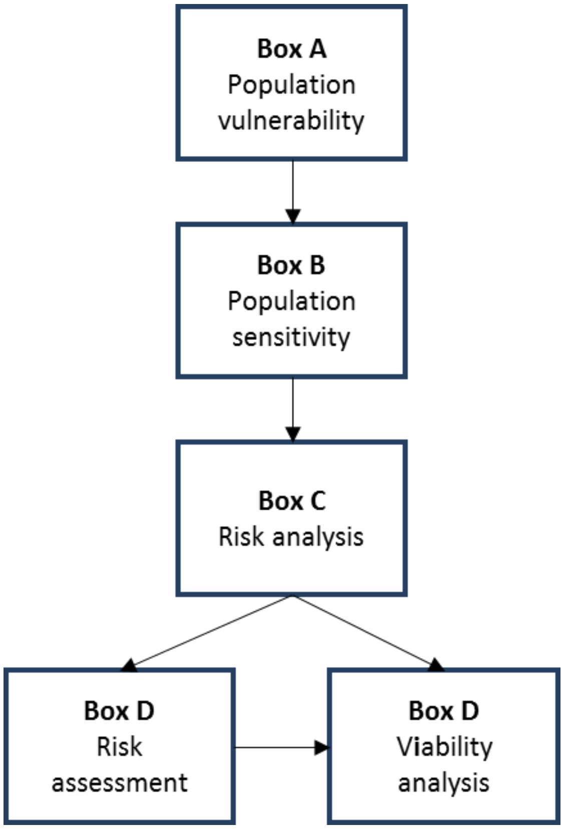


*Figure 3 - Structure of the Cumulative Ecological Risk Assessment Framework (CERAF), adapted from DFO 2017c.*

The PVA method was selected as the most appropriate assessment to use to address the recovery measure (RM 11) as it incorporates the required threats and has been applied to a number of species, including SRKW. Relatively minor modifications were necessary to adapt the PVA to also assess NRKW. In addition, the software used to carry out the PVA (Vortex) is open access and available for use (Lacy and Pollak 2014).

## 1.3 CUMULATIVE EFFECTS ASSESSMENT FOR RESIDENT KILLER WHALES

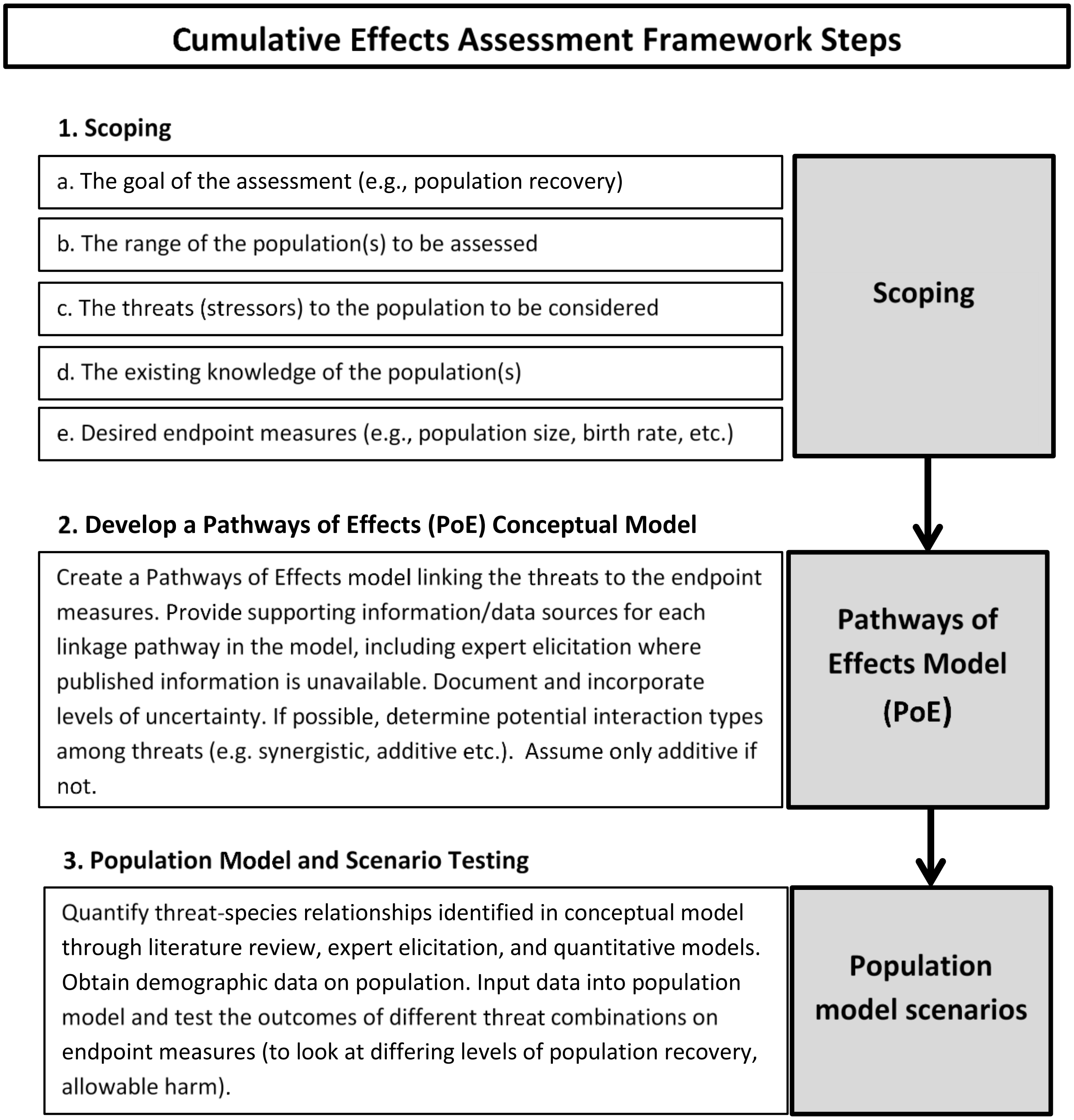
The current cumulative effects assessment consists of two phases. First, a Pathways of Effects (PoE) conceptual model describes the impacts of threats on the mortality and fecundity of the species. As threats can interact over space and time, altering their respective intensities and consequent effects on individuals and populations, this study will also assess potential interactions between threats to more accurately represent the natural system. The outputs of the PoE conceptual model were used to design and refine the PVA model in the next phase.

In the second phase, impacts are parameterised (e.g., effect size for each threat and its impact on vital rates) and a quantitative PVA is conducted to assess the cumulative effects, building upon the methods and results of previous work (Taylor and Plater 2001; Ward et al. 2009; Vélez-Espino et al. 2014b; Williams et al. 2017; Lacy et al. 2017; DFO 2018a). Existing literature and data are used to parameterise the impact of each threat on killer whale vital rates and previously published relationships are updated with recent data and re-analysed. These quantitative values and relationships specific to each population (SRKW and NRKW) are used to define the inputs to a population model describing the combined impact on population persistence through time. The model structure builds upon an existing PVA model developed for SRKW by Lacy et al. (2017). To capture the unique population structure and threat exposure, a PVA model is run for each population separately (SRKW and NRKW).

An overview of the steps used in the current work to assess cumulative effects on NRKW and SRKW is outlined in Figure 4.

### 1.3.1 Assumptions for this Assessment

- The mechanisms and consequences of threats on individuals are assumed to be the same for both Resident Killer Whale populations, whereas the level of exposure to threats is assumed to be population-specific.
- Impacts from threats to population vital rates (mortality and fecundity), based on the best available information at the time of the assessment, are assumed to be accurately described in the Pathways of Effects model
- The analysis assumes impacts only from the focal threats examined (reduced prey availability, disturbance, and contaminants), and does not consider other threats and the effects of broader impacts such as changing climate conditions and increasing human populations.
- The way that impacts are parameterised in the PVA model is assumed to represent the impacts of the entire threat (e.g. impacts of PCBs represents the Contaminants threat). Specific limitations and assumptions for each threat are described in detail in section 5.3.
- Information used to quantify threat impacts to vital rates was assumed to represent threat conditions throughout the range and throughout the year, despite primarily being obtained in the Salish Sea area in the summer/fall period.
- The assessment assumes no spatial or temporal variation at the sub-population level, even though variation between matrilines could affect their exposure to threats.
- Projections of the RKW population dynamics into the future assume that current threat levels remain the same. It is also assumed that that no threat mitigation measures and management actions are taken.
- The population model chosen to be used for predictions is assumed to be an effective surrogate for the real population dynamics for SRKW and NRKW populations.



**1. Scoping**

a. The goal of the assessment (e.g., population recovery)

b. The range of the population(s) to be assessed

c. The threats (stressors) to the population to be considered

d. The existing knowledge of the population(s)

e. Desired endpoint measures (e.g., population size, birth rate, etc.)

**2. Develop a Pathways of Effects (PoE) Conceptual Model**

Create a Pathways of Effects model linking the threats to the endpoint measures. Provide supporting information/data sources for each linkage pathway in the model, including expert elicitation where published information is unavailable. Document and incorporate levels of uncertainty. If possible, determine potential interaction types among threats (e.g. synergistic, additive etc.). Assume only additive if not.

**3. Population Model and Scenario Testing**

Quantify threat-species relationships identified in conceptual model through literature review, expert elicitation, and quantitative models. Obtain demographic data on population. Input data into population model and test the outcomes of different threat combinations on endpoint measures (to look at differing levels of population recovery, allowable harm).

*Figure 4 – Steps in the current cumulative effects assessment framework.*

## 1.4 OBJECTIVES

The current working paper has four major objectives:

1. Develop a Pathways of Effects (PoE) conceptual model to visually represent threat-impact pathways, limited to the primary threats identified by the Species at Risk Program (DF0 2017b). Provide supporting text to accompany the PoE model diagram to describe and justify the linkage pathways presented and explain how threats act on population parameters based on evidence in the literature and elsewhere. The PoE conceptual model will be generic to include both SRKW and NRKW populations, as the mechanisms of impact

are expected to be similar in both populations but the levels of exposure to each threat are different.

2. Quantify threat linkage pathways identified in the PoE model by determining the best available and most recent data or information from data mining, literature review and expert elicitation. This information will be used to develop and parameterise a quantitative PVA model.
3. Assess the cumulative effects acting on Resident Killer Whales by running single and cumulative PVA model scenarios to evaluate and compare the effects of each scenario to the observed population dynamics.
4. Identify uncertainties in data and methods and highlight knowledge gaps

### 1.5 SCOPE OF THIS STUDY

The aim of the current assessment is to evaluate the cumulative effects of anthropogenic threats on Resident Killer Whales. The study is limited to considering the primary threats identified in the (SARA action plan for NRKW and SRKW (DFO 2017b, 2018a). The effects of low probability but high impact events, such as catastrophic oil spills, are not included in the current assessment. Future changes in anthropogenic activities are not included or assessed. Potential mitigation measures and management actions will not be evaluated, but this assessment can be used as a tool to evaluate future changes and mitigation measures once the cumulative effects model has been reviewed.

## 2 PATHWAYS OF EFFECTS CONCEPTUAL MODEL

### 2.1 DEFINITION, STRUCTURE AND OUTPUTS

PoE models are widely used conceptual modelling tools that can guide assessments by providing a science-based foundation for decision-making (Government of Canada 2012). They can be useful for scoping different types of cumulative effects assessments (activity, threat, species, and area) and they help identify the threats and clarify links between human activities and potential impacts on aquatic ecosystem components. The Government of Canada has developed national guidelines for the format of these models (Government of Canada 2012). PoEs can range from small scale, simple impact links, suitable for a species-specific habitat, to more complex, large scale networks, suitable for a bioregion (Government of Canada 2012). PoEs have typically been used to describe activities, such as aquaculture, but can also be used to illustrate the linkage pathways between anthropogenic activities, threats and population parameters, such as changes in mortality and fecundity.

This assessment uses a species-based PoE model to elucidate the linkage pathways between threats and their impacts on a particular species, which then informs a cumulative effects assessment.

PoE models provide useful outputs for the scoping phases of cumulative effects assessments as they can identify all linkage pathways (including interactions between threats), and the literature available to guide further investigations into quantifying the linkage pathways. The outputs of a PoE conceptual model consist of a visual representation of the threat linkage pathways, with supporting justification text. This can be in the form of a table or linked text presenting the evidence that is available to describe and quantify each linkage pathway shown on the diagram.

## 2.2 RESIDENT KILLER WHALE SPECIFIC POE MODEL

The first step in developing a PoE model is to scope the threats and endpoints (in this case fecundity and mortality) that the model will examine. In the present case this is not required, as the identification of the primary threats to the RKW populations has already been completed by DFO Species at Risk (DFO 2017b, 2018a). This PoE model does not explicitly include the source activities of threats as is traditionally found in such models because the focus is on specific threat impacts well defined by SARA, and we do not have sufficient knowledge or a time series of the activities to be able to include these.

The proposed PoE conceptual model (Figure 5) outlines how the potential impacts to RKW from these primary threats might manifest. In addition to describing the direct linkage pathways from threats to vital rates, as in a standard PoE model, this relatively data rich model allowed a novel approach whereby known and potential threat interactions linkage pathways to vital rates were also included. The overall PoE conceptual model (Figure 5a) identifies the important conceptual connections between threats and RKW populations, based on literature review and expert opinion. The diagrams consists of two to three rows of boxes; grey boxes represent threats of interest, dashed line boxes represent any interactions between threats, and black boxes indicate the population parameters (vital rates) affected by the impacts. Each linkage pathway is tagged with a numerical value that links it to the subsequent text to justify and provide supporting evidence for that specific linkage pathway. For clarity, direct and interaction effects are presented separately below the main diagram to simplify their interpretation and to aid the identification of the correct numbered linkage pathways, Figure 5b illustrates the direct linkage pathways between threats and vital rates, and Figure 5c the (indirect) interaction linkage pathways between threats and vital rates.

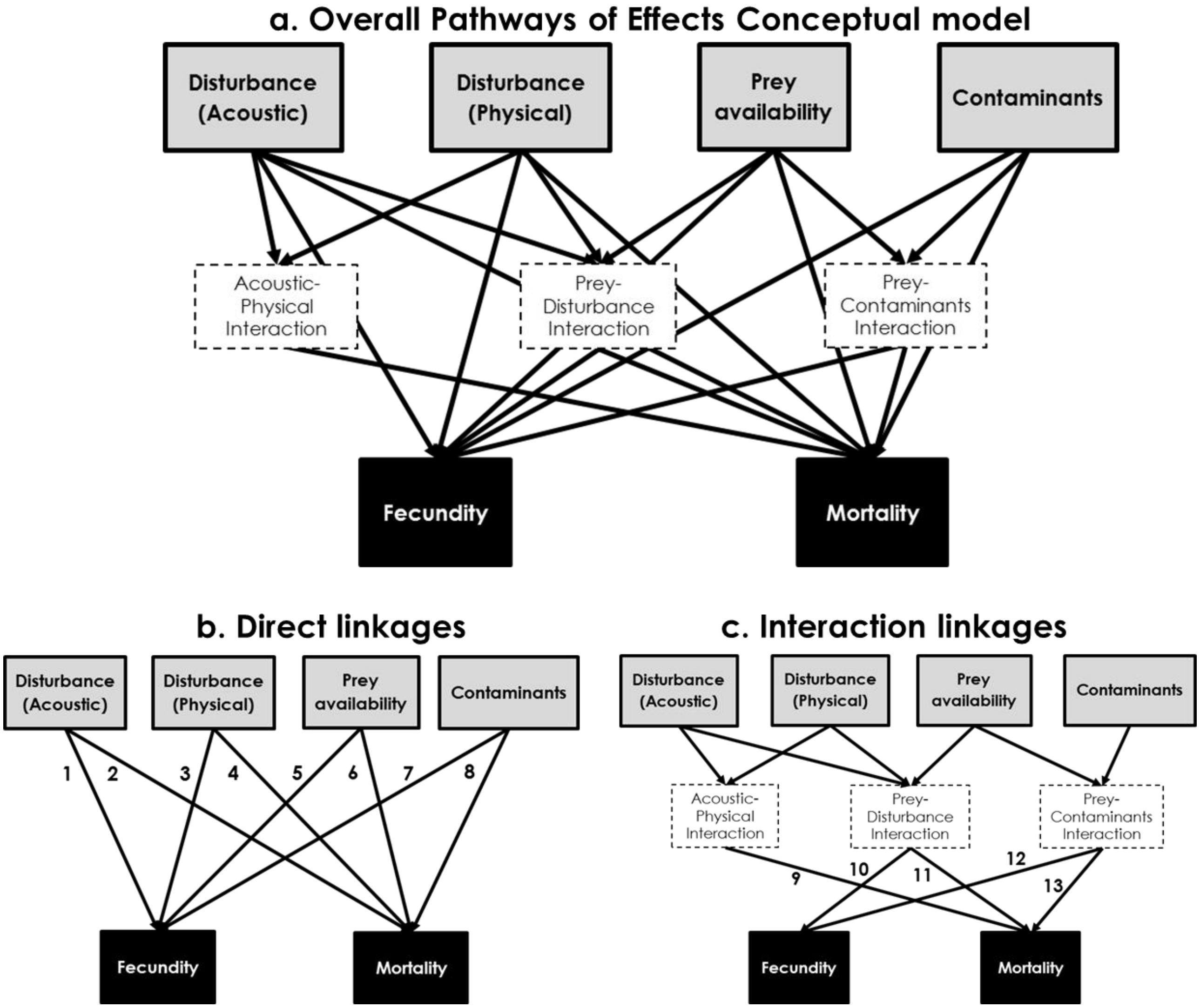


*Figure 5 - a. Overall Resident Killer Whale Pathways of Effects (PoE) conceptual model, including priority threats, interactions, and impacts on Resident Killer Whale fecundity and mortality. The main diagram (a) is broken up to clearly illustrate linkage pathways and numbering for direct linkage pathways (b) and interaction linkage pathways (c). Numbers next to each linkage pathway refer to descriptions in the text.*

This single PoE model (Figure 5a) represents both populations of Resident Killer Whale (NRKW and SRKW) as the mechanisms by which threats affect individuals in the two populations are assumed to be the same in this assessment; it is in the details and quantification of the linkage pathways where differences between populations may occur. Population differences are explored in the PVA section (Sections 3 and 4 - Population Viability Analysis Model and Results) where the same linkage pathways will be assessed for each population to identify where differences occur and will be captured in accompanying tables of evidence. The components making up the PoE model outlined above are explained and justified in detail in the following sections (2.4 - 2.7), the numbered links on the PoE diagram connect to sections of text through the numbers assigned to each in this format [1]; [2]; etc.

The subsequent PVA section (Sections 3 and 4) will contain a more specific model and will be presented based on the actual analysis that was done with linkage pathways containing the values and data used to represent each component.

## 2.3 AQUARIUM REMOVALS (A HISTORIC THREAT)

### 2.3.1 Background

The removal of killer whales from the wild for display in aquaria around the world (a ‘live capture’ fishery) was a significant historical threat to Resident Killer Whale populations and could still have residual effects on current populations. This historic threat is not included in the PoE conceptual model but will be investigated in the PVA section.

In Canada, the absence of laws to guide interactions with killer whales or regulate their capture prior to 1970 meant that killer whales were classed as ‘wildlife’, and permits were issued that allowed them to be removed and held in captivity. These permits had no catch quotas for Canadian netters until 1970 when initial protective legislation was introduced. In the USA, there were no permits or limits introduced until 1971 (Bigg and Wolman 1975; Baird 2001). In 1982 the capture of killer whales was no longer permitted in BC, as the provincial Wildlife Act was amended to exclude killer whales as ‘wildlife’, and the Canadian federal government included the protection of cetaceans under the Fisheries Act. Specific marine mammal regulations protecting killer whales in Canada were fully in place by 1994 (Baird 2001). In the USA, the Marine Mammal Protection Act prohibits the capture of killer whales without a permit, and no permits have been issued since 1989 (Tierney 2010).

In addition to live captures, there is evidence that in the years before protective regulations, killer whales regularly suffered gunshot injuries and likely deaths due to fisheries conflicts. Approximately 25% of SRKW whales from the live capture fishery in Puget Sound had evidence of bullet wounds. The shooting of killer whales in the NE Pacific is estimated to have begun around 1929, and became illegal in Canada in 1970 under the Fisheries Act and in the US in 1972 under the Marine Mammal Protection Act (Hoyt 1990; Olesiuk et al 1990; Krahn et al. 2002). However, in the US, fishermen were still allowed to shoot marine mammals to ‘protect their catch and gear’ until the Act was amended in 1988, shootings are still thought to occur in Alaska (Fraker 2013). The current status of gunshot wounds in the NRKW and SRKW populations is uncertain, though data from the NRKW survey indicate that observations of injuries suspected to be a result of gunshots mostly occurred prior to the year 2000, and in the last 10 years there has been only one observation of an injury potentially consistent with a gunshot (DFO Cetacean Research Program, unpublished data). Gun shot wound mortality at this time is assumed to be zero for both populations.

### 2.3.2 The Live Capture Fishery

The live capture fishery removed 68 killer whales from BC and Washington State waters from 1962-1977. Of these, an estimated 48 were from the SRKW population (removed between 1962-1977), 15 from the NRKW population (removed between 1965-1969), and five were Bigg’s killer whales (removed between 1970-1975) (Asper and Cornell 1977; Olesiuk et al. 1990, Table 2). These numbers represent all whales removed from the Canadian Pacific killer whale populations, including those that died during a capture, or since. The majority of individuals removed from the SRKW population were physically immature (Table 2: 30/48, 63%). More males were removed than females; of those identified to sex, there were 26 males and 18 females removed from the SRKW population (Table 2). Fewer individuals were removed from the NRKW population: nine juveniles and six adults, of which eight were males and seven were females (Table 3).

*Table 2 - Southern Resident Killer Whales (SRKW) captured or killed by pod, year, length (m) and sex during the live capture fishery in BC and WA (after Olesiuk et al. 1990). Animals that were presumed to belong to the SRKW population based on their location of capture are indicated by the superscript[1].*

| **Pod/Area** | **Year caught** | **N** | **Physically immature** | | | | | | **Mature** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | **≤3.5m** | | | **3.5-4.5m** | | | **4.5-6m** | **≥ 4.5m** | **≥6m** | |
| | | | **M** | **F** | **U** | **M** | **F** | **U** | **M** | **F** | **M** | **U** |
| South Vancouver[1] | 1962 | 2 | - | - | - | - | - | - | - | - | 2 | - |
| J01, K01 or L01 | 1964 | 1 | - | - | - | - | - | - | 1 | - | - | - |
| J01, K01 or L01 | 1965 | 2 | - | - | - | - | 1 | - | - | 1 | - | - |
| South Vancouver[1] | 1966 | 1 | 1 | - | - | - | - | - | - | - | - | - |
| K01 | 1967 | 8 | 1 | 2 | - | 2 | 1 | - | - | 1 | 1 | - |
| J01, L01 | 1968 | 5 | - | - | - | 3 | - | - | 2 | - | - | - |
| South Vancouver[1] | 1968 | 2 | - | - | - | 1 | - | - | - | 1 | - | - |
| South Vancouver[1] | 1969 | 3 | - | - | - | - | - | - | - | 1 | 1 | 1 |
| South Vancouver[1] | 1970 | 2 | - | 1 | - | - | - | 1 | - | - | - | - |
| J01, K01, or L01 | 1970 | 11 | 2 | - | 2 | 3 | 2 | - | 1 | 1 | - | - |
| Washington[1] | 1971 | 2 | - | - | - | 2 | - | - | - | - | - | - |
| L01 | 1971 | 3 | - | 1 | - | 1 | 1 | - | - | - | - | - |
| J01 | 1972 | 1 | 1 | - | - | - | - | - | - | - | - | - |
| Washington[1] | 1973 | 1 | - | - | - | - | - | - | - | 1 | - | - |
| K01 | 1973 | 1 | - | - | - | - | - | - | - | 1 | - | - |
| L01 | 1973 | 2 | - | - | - | - | - | - | 1 | 1 | - | - |
| South Vancouver[1] | 1977 | 1 | - | 1 | - | - | - | - | - | - | - | - |
| **Total** | 1962-77 | **48** | **5** | **5** | **2** | **12** | **5** | **1** | **5** | **8** | **4** | **1** |

[1] Presumed SRKW based on area captured

*Table 3 - Northern Resident Killer Whales (NRKW) captured or killed by pod, year, length (m) and sex during the live capture fishery in BC and WA (after Olesiuk et al. 1990). Animals presumed to belong to the NRKW population based on their location of capture are indicated by the superscript[2].*

| Pod/Area | Year caught | N | Physically immature | | | | | | Mature | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | ≤3.5m | | | 3.5-4.5m | | | 4.5-6m | ≥ 4.5m | ≥6m | |
| | | | M | F | U | M | F | U | M | F | M | U |
| C01 | 1965 | 1 | - | - | - | - | - | - | - | - | 1 | - |
| I11 | 1967 | 1 | - | - | - | 1 | - | - | - | - | - | - |
| NE Vancouver[2] | 1968 | 1 | - | - | - | 1 | - | - | - | - | - | - |
| A (A05) | 1968 | 6 | 1 | - | - | - | 1 | - | 1 | 3 | - | - |
| A05 | 1969 | 6 | - | 2 | - | 2 | 1 | - | 1 | - | - | - |
| **Total** | 1965-69 | **15** | **1** | **2** | **0** | **4** | **2** | **0** | **2** | **3** | **1** | **0** |

[2] Presumed NRKW based on area captured

### 2.3.3 Effects of Removals

Because of the proximity of SRKW habitat to population centres, the majority of whales (48, 71%) removed for aquaria display were from this population. This removal had a disproportionate impact on the smaller SRKW population, as small populations are more vulnerable to extinction because of stochastic events (DFO 2018a). Not only did the removals significantly reduce SRKW population size, they also skewed SRKW population structure, as removals were predominantly comprised of juveniles and young males (Vélez-Espino et al. 2014a). After most live captures ended, there was a period of growth in the SRKW population of 19% until 1980, followed by a decline of 11% that was attributed to the skewing of the population structure from preferential captures (Olesiuk et al. 1990; Giles 2014). Removals are expected to have impacted the NRKW population less significantly than the SRKW population, as fewer individuals (15) were removed from a larger population. There are no mitigation actions for this historic threat but the long-term effects should be acknowledged in any assessment of cumulative effects.

## 2.4 REDUCED PREY AVAILABILITY [5,6,10,11,12,13]

Prey availability is made up of two components, prey abundance and prey access. A full characterisation of prey availability would involve considering the components that influence both whether sufficient prey is present and whether it can be accessed for consumption, which relates to factors such as timing and ability to forage. The current assessment captures this threat by examining changes in prey abundance, using a time series of Chinook salmon data. The prey access component is partially captured in interactions between prey abundance and vessel acoustic and physical disturbance.

### 2.4.1 Background [5,6]

Resident Killer Whales are fish-eating marine mammals with a distinct preference for Chinook salmon (*O. tshawytscha*), due to their large size, high lipid content and year round availability

(Ford et al. 2005; Ford and Ellis 2006). Analysis of prey remains indicates that Chinook salmon can comprise up to 90% of the summer diet of SRKW (Ford and Ellis 2006; Ford et al. 1998; Hanson et al. 2010). Changes in RKW population parameters have been directly linked to fluctuations in Chinook salmon stocks [5,6]. Inter-annual variability in Chinook salmon is related to inter-annual variability in RKW mortality (Ford et al. 2010; Vélez-Espino et al. 2014b) [6] and fecundity (Ward et al. 2009; Vélez-Espino et al. 2014b) [6]. There is a positive correlation between RKW calving probability and Chinook salmon abundance (Ward et al. 2009) [5] and a negative relationship between RKW mortality and Chinook salmon abundance (Ford et al. 2009; 2010; Vélez-Espino et al. 2014b) [6]. These findings strongly indicate that Chinook salmon abundance plays an important role in RKW population dynamics.

Aerial photogrammetry has provided information on the link between mortality rates and body condition, or the fat stores, in individual whales (Durban et al. 2015). Declines in the Eye Patch Ratio (EPR, measured as the proportional head width) have been linked to short-term mortality. In 2008 and 2013, 43 individuals from the SRKW population were measured and eleven had significant reduction in EPR, indicating depletion of fat stores (Durban et al. 2015). Animals that were not pregnant or nursing (life stages where body metric changes are expected Kriete 1995; Kastelein et al. 2003), with reduced EPR died shortly after being photographed in this condition.

Though the majority of the summer diet of RKW consists of Chinook salmon, they also consume other species of salmonids and non-salmonids. It is estimated that overall, 96% of the RKW diet comprises salmonids, and within this, 71.5% is Chinook, 24% chum, and 0.5% other salmonids such as Coho salmon (*O. kisutch*). The non-salmonid fish in the diet are Pacific herring (*Clupea pallasi*), sablefish (*Anoplopoma fimbria*), yelloweye rockfish (*Sebastes ruberrimus*), quillback rockfish (*Sebastes maliger*), and Pacific halibut (*Hippoglossus stenolepis*) (Alava et al. 2012; Ford et al. 2006; 2009). As chum salmon can comprise 24% of the salmonids in RKW diet, its availability and abundance may also be a contributor to RKW population growth. Chum salmon become more important in the RKW diet in autumn, surpassing the contribution of Chinook salmon at that time (Ford and Ellis 2006; Ford et al. 2010). However, the two studies that have examined the role of chum and other salmon species in killer whale demography did not find any statistical evidence for a relationship between these fish stocks and RKW with mortality or fecundity (Vélez-Espino et al. 2014b; Ward et al. 2009).

### 2.4.2 Important Salmonid Stocks

There is evidence from genetic analysis of prey samples that the two Resident Killer Whale populations may exploit different combinations of Chinook salmon stocks. The dominant Chinook salmon stocks found in SRKW diet from late spring to early autumn are the Fraser River and Puget Sound Chinook salmon (Vélez-Espino et al. 2014b; Hanson et al., 2010). Over the season (late spring to early autumn), the proportion of Fraser Chinook salmon in Juan de Fuca Strait increases in relation to Puget Sound stocks as populations travel through the area on their return migrations to the Fraser river (DFO 2018b[1]). Chum salmon stocks consumed by SRKW are assumed to be from Puget Sound stocks (Vélez-Espino et al., 2014b).

For NRKW, the dominant Chinook salmon prey stocks are mainly Fraser River, but also Northern and Central BC, west coast of Vancouver Island, Georgia Strait, Puget Sound and the upper Columbia stocks, in the same season (late spring to early autumn) (Ford and Ellis 2006;

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[1] DFO. 2018b. Discussion Paper: February 15, 2018. Proposed 2018 Salmon Fishery Management Measures to Support Chinook Salmon Prey Availability for Southern Resident Killer Whales (internal).

Ford et al. 2009; Vélez-Espino 2014b). Chum salmon stocks consumed by NRKW are Fraser, East coast Vancouver Island (ECVI), and South BC Coast (Vélez-Espino et al. 2014b).

Some salmon stocks may be consumed by more than one population of RKW, such as those salmon stocks with a more northerly distribution that may encounter killer whale populations throughout their migrations. For example, Fraser Summer (age-4 ocean type) Chinook salmon migrate to the Fraser River in August through Johnstone Strait and Strait of Juan de Fuca (DFO 2018b).

### 2.4.3 Trends in Chinook Abundance

Chinook salmon production mainly happens in major river systems such as the Fraser and Yukon rivers, with some in smaller streams (Healey, 2003). Chinook salmon stocks have experienced widespread population declines, especially in the 1990s (Figure 6; Riddell et al. 2013). Since the 1980's, Chinook salmon productivity is estimated to have declined by 25-40% for many BC stocks (DFO 2018b[1]). Chinook salmon populations have also shown a trend towards smaller body sizes (Wiles 2016; Ohlberger et al. 2016). The productivity of wild chum salmon stocks has also undergone widespread declines in Washington (WA) and British Columbia (BC), with 81% of stocks having recent declines in productivity (Malick and Cox 2016).

### 2.4.4 Threshold Effects/Nonlinearities [10,11,12,13]

The ability of killer whales to successfully catch and consume prey (access to prey) may be affected by vessel disturbance, as will be discussed in Section 2.5.6. The prey-disturbance interaction [10,11] effectively reduces killer whales access to prey, which can have effects at the individual and population levels. The interaction between prey abundance and physical and acoustic vessel disturbance may also potentially include reduced access to foraging habitat, in addition to reduced foraging. It is not well understood whether prey distribution becomes increasingly patchy with reduced abundance, and whether patchy prey distribution might impact prey access / foraging for Resident Killer Whales.

The consumption of prey items contaminated with persistent, bioaccumulative and toxic chemicals (PBTs) can also affect killer whale vital rates. Salmonids have been found to contain a range of contaminants that are also found in killer whale tissues, which is further discussed in section 2.7.1. The prey-contaminants interaction [12,13] potentially affects the mortality and fecundity of these populations.

In addition, there may be other factors and non-linearities that are masking or confounding the detection of stronger interactions between RKW vital rates and prey abundance (Vélez Espino et al. 2014b). For example, large-scale climate changes, genetic factors, and other environmental and anthropogenic variables are also changing in this system and make the isolation of single threat impacts more difficult.

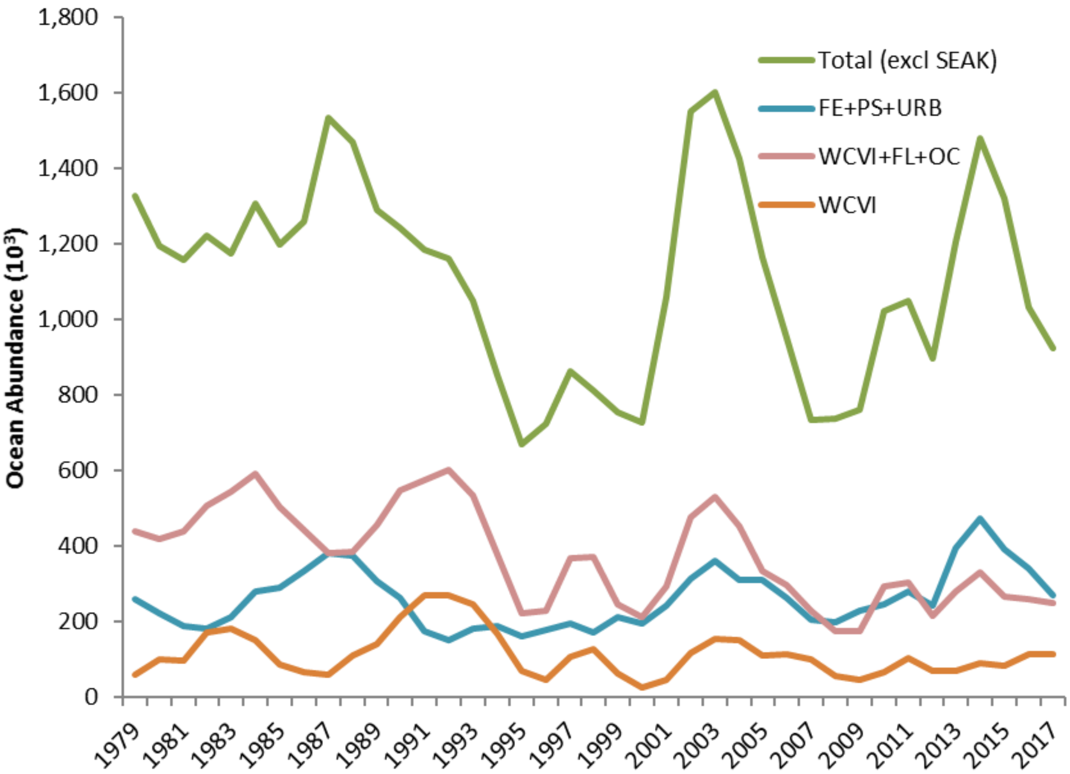

*Figure 6 – Time series of modelled Chinook salmon ocean abundance (thousands) for stocks of interest to RKW. Total is the coastwide index (excluding southeast Alaska stocks), FE+PS+URB is the Fraser Early, Puget Sound and Upper River Basin, WCVI+FL+OC is the West Coast Vancouver Island, Fraser Late, and Oregon Coast stocks; WCVI is the West Coast Vancouver Island stock.*

## 2.5 DISTURBANCE (ACOUSTIC) [1,2,9,10,11]

Increasing amounts of vessel traffic, industrial activities and other anthropogenic activities are affecting the physical and acoustic habitat of killer whales, as well as their behaviour. The impact of disturbance on cetaceans at both the individual and population level is not well understood (Nowacek et al. 2007; DFO 2011). Vessel disturbance has been identified as a principal threat to the two Resident Killer Whale populations in the DFO SARA Recovery Strategy (DFO 2011, 2018a). As it is as yet not possible to separate the impacts of vessel noise from the impacts from vessel physical presence, this threat captures both of these aspects.

### 2.5.1 Background

Vessels are one source of underwater noise; commercial ships, recreational vessels, whale watching vessels, and military vessels are active in the range of the two Resident Killer Whale populations. Noise can also come from military and research activities (e.g., sonar, explosions), aircraft overflights, construction (e.g., pile driving, blasting), and from dredging. Different types of noise can potentially result in various levels and types of disturbance, a level of complexity out of scope for this work. The impact of noise on killer whales can be acute or chronic, with the effects dependent on frequency range, source level and signal structure of the sound (Richardson et al. 1995; National Research Council 2003; Nowacek et al. 2007). Acute impacts are intermittent and less predictable in the environment. The impacts of acute sound exposure (e.g., blasting) can include direct mortality if whales are in the affected area at the time of noise emittance, and are difficult to predict in space and time [2].

Though there are many potential sources of noise disturbance that could impact Resident Killer Whales, this assessment is limited in scope to considering the impacts of acoustic disturbance from vessels, the main source of chronic underwater noise in the assessment area. Vessel-related disturbance has been specifically identified as a risk to recovery by DFO (DFO 2017b). How to discern or measure the impacts of such acoustic disturbance can be challenging and the acoustic component of vessel disturbance on RKW can cause impacts via a number of mechanisms: behavioural changes, auditory masking, fitness reduction, and resultant population consequences. Another potential impact caused by vessel disturbance is due to the physical presence of a vessel, and it is difficult to separate the impact of the physical presence of a vessel from the impact of the noise it produces. For dolphins, boat presence alone can disturb behaviour (Pirotta et al. 2015), and impact their ability to rest and reach deep sleep (Tyne 2015).

### 2.5.2 Behavioural Change [10,11]

There can be energetic costs to any disturbance that causes an animal to switch behavioural states (e.g. from resting to travelling) or results in more time spent performing energetically costly activities such as evasive or surface active behaviours (Williams et al. 2009). These costs increase with the severity and duration of the response (Erbe 2002; Williams et al. 2006; Lusseau et al. 2009; Ayres 2012; Williams et al. 2014). Coping mechanisms can range from short-term avoidance to long-term habitat abandonment (Kruse 1991; Williams et al. 2002a,b; Lusseau and Bejder 2007). In nearshore core areas in the summer and fall, Resident Killer Whales spend 40-67% of their time engaged in foraging behaviours (Ford 2006; Noren et al. 2009). The onset of behavioural changes (affecting 50% of observed killer whales) has been observed to begin at received levels of ~130 dB re: 1 µPa (broadband, root-mean-square) (Williams et al. 2014). Noise or vessel disturbance that causes an animal to reduce foraging time can have impacts on mortality and fecundity similar to that of lowered prey abundance [10,11]. Increased swimming velocity (associated with vessel avoidance behaviours) can result in an estimated 20% increase in energetic expenditures (Kriete 1995, 2002), meaning less energy is available for other vital functions and increasing caloric requirements.

### 2.5.3 Auditory Masking [10,11]

Vessel noise overlaps with the sound frequency range used by killer whales (Watkins et al. 1987; Berchok et al. 2006; Mouy et al. 2009; Tervo et al. 2011; Hatch et al. 2012), and can mask the receiving of acoustic signals used for foraging, navigation, communication and social interaction (Erbe 1997, 2016; Weilgart 2007; Clark et al. 2009; Castellote et al. 2012). Masking can interfere with echolocation of prey and the effectiveness of foraging activities [10,11]. SRKW have been shown to increase call duration and amplitude in the presence of boats, which has been suggested to be an adaptation to masking effects but one that may come with energetic costs (Foote et al. 2004; Holt et al. 2008). There is an additional energetic cost if masking prevents or inhibits successful foraging events, causing increased energy to be spent on foraging activities and less available for other life history processes, such as mating and fecundity [10,11].

### 2.5.4 Fitness Reduction due to Stress [1,9]

When individuals cannot reduce or avoid proximity to disturbance, stress levels may increase causing a reduction in fitness that may be manifested in reduced reproductive success (Lusseau and Bejder 2007) [1]. Individuals with higher levels of stress may be susceptible to other causes of mortality, such as disease, parasites, and vessel strikes (Fair and Becker 2000) [9]. Humpback whales have been shown to be more susceptible to entanglement after acoustic trauma (Todd et al 1996; Ketten et al 1993). However, Ayres et al (2012) concluded that elevated stress in SRKW, measured by a combination of hormone levels, is linked to prey abundance, rather than vessel disturbance.

### 2.5.5 Population Consequences of Disturbance [1,2,9,10,11]

Population effects of noise can manifest through behavioural or physiological changes, which can have impacts on health and vital rates [1,2]. A Population Consequences of Disturbance (PCoD) model (NRC 2005; Tolitt et al. 2017) has been used to quantify the chronic and acute impacts of noise disturbance on killer whales (Figure 7). A noise exposure model combined with the PCoD model has been used to estimate lost foraging time for SRKW as a combination of behavioural response and masking (Tolitt et al. 2017). In the Salish Sea's busy traffic areas, individual noise disturbance events can combine to have potentially substantial impacts (Tollit et al. 2017). The SMRU (2017) model predicted that in the Salish Sea, SRKW foraging time was decreased by 20-23% of each whale day (i.e. days when SRKW pods were predicted to be in the study area). Two-thirds of those effects were estimated to be from commercial vessels and one-third from whale watching vessels. Prey detection range was decreased as a result of masking of echolocation clicks by vessel noise. The combined effect of both vessel types was estimated to reduce the range of prey detection by 12-37%. Reductions in foraging time and efficiency can result in the same impacts to vital rates as that of reduced prey abundance (increased mortality and reduced fecundity) [10,11].

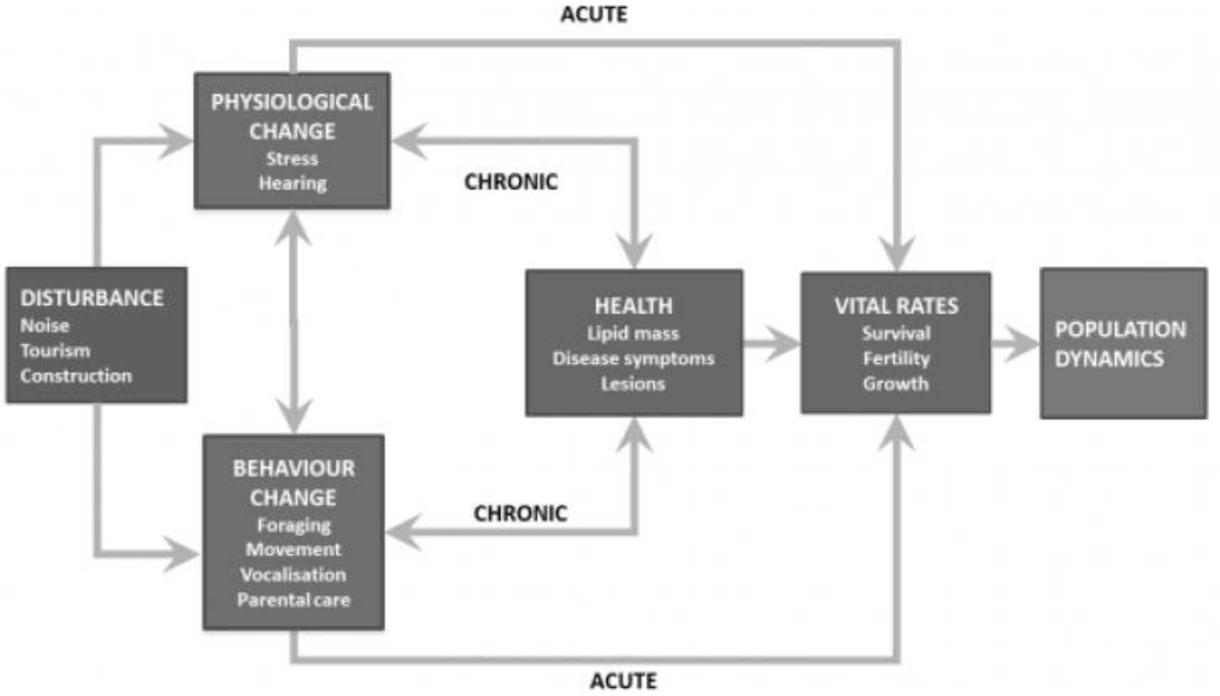

*Figure 7 - Population Consequence of Disturbance (PCoD) model and the pathways of impact that lead to changes in population dynamics (adapted from Tollit et al. 2017)*

### 2.5.6 Interactions/Thresholds/Non-linearities [9,10,11]

The impacts of vessel noise disturbance could be exacerbated when prey abundance is low, and conversely, vessel noise disturbance may have little or no effect on overall feeding efficiency when prey abundance is high [10,11]. In killer whales, glucocorticoid hormones (which increase in response to nutritional or psychological stress) in scat samples have been found to be low in summer, when vessel and salmon (Fraser River) abundance is high, and high when vessel and salmon abundance are low, indicating that short-term physiological effects of prey abundance appear to overshadow impacts from vessels (Ayres et al. 2012) [10,11]. However, the study did not collect samples from periods of time when vessels were high but salmon levels were low. The temporal relationship between vessel disturbance and stress levels has not been quantified and there may be an unobserved delay between exposure and stress hormone production that makes these results difficult to interpret.

Alternately, when prey abundance is extremely low, predators may ignore disturbance because the fitness consequences of abandoning a predation event would be higher and this may increase other mortality risks (e.g., vessel strike) if adaptive disturbance responses (e.g. startle) are overridden or ignored during pursuit of limited prey [10,11]. This is supported by basic ecological principles relating to a predator-prey relationship in the context of resource availability: prey species are only forced to forage in areas of higher risk of predation when food is scarce The basic energy requirements of an individual mean that they may have to accept predation risk to meet their energy budget in instances where conditions make the cost of predator avoidance high (Sansom 2009; Stevens 2010). Human disturbance can be analogous to risk from predators, and similar fitness impacts can result from predation and non-lethal disturbances such as noise (Frid and Dill 2002). In the case of killer whales, impacts from vessel avoidance could be analogous to those from predator avoidance. When Southern Resident Killer Whales are food limited, mechanisms of energetic impacts such as this are of concern (Lusseau et al. 2009). The effects of masking are another component that killer whales would have to overcome in the presence of vessels if they chose to take the risk to forage. An additional possible interaction is that whales could be more likely to abandon foraging activities in a low abundance/poor quality prey patch if disturbed, because the energetic returns under those circumstances are so poor (Kuningas et al. 2013; Pirotta et al 2015).

Noise may have the potential to cause accidental beaching or entrapment, and loud noises have been demonstrated to cause strandings (DFO 2018a) [9]. It has been suggested that the reason a group of SRKWs spent 30 days in an inlet in Puget Sound in 1997 was due to an aversion to passing under a noisy bridge (Shore 1998). The evidence for interaction between acoustic disturbance and other threats is sparse and not thoroughly understood for killer whales and other cetacean species.

## 2.6 DISTURBANCE (PHYSICAL) [3,4]

The second component of vessel disturbance is physical disturbance, identified as a principal threat to the Resident Killer Whale populations in the DFO SARA Recovery Strategy (DFO 2011, 2018a) [4]. For this study, this threat is captured by vessel strikes only.

### 2.6.1 Background

Killer whales can be injured or killed as a result of a vessel collision, the outcomes of the collision can be particularly damaging if moving propeller blades are encountered (Ford et al. 2000; Baird 2001) [3,4]. The spatial overlap between Resident Killer Whale populations and maritime traffic suggests that there is a risk of injury and mortality from ship strikes (Williams and O’Hara 2010). Observations have found that recreational vessels in proximity to or engaging in SRKW-watching are increasingly being driven in a manner that heightens the risk of a collision, such as stopping in the path of the whales, chasing whales, approaching whales head on or crossing their paths (Ferrera et al. 2017). The number of vessels in proximity to the whales and vessel speed are likely to have a strong influence on the rate of collisions and the severity of injury (Conn and Silber 2013; Ferrara et al. 2017).

### 2.6.2 Reports of Injuries and Mortalities

#### Injuries [3]

The frequency and severity of injuries from ship strikes on Resident Killer Whales is uncertain, but there are some anecdotal accounts available as well as observed physical evidence of injuries from regular population surveys. These sources provide some insight into the proportion of the population that may have experienced injuries from vessel strikes. Table 4 summarises information on known vessel strike incidents that resulted in injuries (that the authors were able to locate from the literature and from experts) for NRKW and SRKW. In many cases the injured killer whales recovered, even in severe cases (Ford et al. 2000; Baird 2002).

*Table 4 – Timeline of known incidents of vessel strikes causing injury in NRKW and SRKW*

| Year | Population | Individuals (#) | Description |
|---|---|---|---|
| 1995 | NRKW | 1 | Struck by a speed boat, dorsal fin was wounded, recovered (Baird 2002; Williams and O'Hara 2010) |
| 1998 | SRKW | 1 | Non-fatal strike in Haro Strait (Baird 2002; Williams and O'Hara 2010) |
| 2003 | NRKW | 1 | Injured by a high-speed boat but recovered (Federal Register 2007; Baird 2002) |
| 2005 | SRKW | 1 | K25 injured by collision with the skeg of a whale watch vessel (drifting power off) (K. Balcomb, Center for Whale Research, WA, pers. comm.) and resulted in a minor injury to the whale, which recovered (Williams and O'Hara 2010) |
| 2006 | NRKW | 2 | One calf A59 injured near Campbell River;<br>One serious injury to G39 (Williams and O'Hara 2010) |
| 2014/5 | NRKW | 1 | Superficial wounds to NRKW A61 from a propeller strike, since recovered (B. Wright, DFO, pers. comm.) |
| 2015 | NRKW | 1 | Photo evidence of relatively severe prop strike wounds across the back of NRKW A60, since recovered (B. Wright, DFO, pers. comm.) |
| 2016 | NRKW | 1 | Observed by aerial photogrammetry to have a superficial propeller wound (Ferrara et al 2017) |
| 2018 | NRKW | 1 | A109, a juvenile (born 2014) NRKW observed with a severe propeller strike injury across its back behind the dorsal fin, appeared vigorous despite injury and was re-sighted by others some days later and again in late August. Injuries appeared to be healing well (B. Wright, DFO, pers. comm.) |

If the frequency of injuries is estimated based on the time period 1995-2016, NRKW had eight reported injuries in 21 years, and SRKW experienced two in 21 years. It is likely that these reports underestimate the true frequency of propeller wounds (Williams and O'Hara 2010). If not killed by a strike, injured killer whales may suffer fitness consequences as a result of the injury (e.g. reduced ability to hunt), potentially impacting fecundity [3].

#### Mortalities [4]

Attributing cause of death in Resident Killer Whales is difficult in many cases as carcasses often sink and are lost, meaning only a small proportion are recovered for necropsy examination (Ford et al. 1998; National Marine Fisheries Service 2008; DFO 2018a). This is a reason why the cause of many calf deaths in particular is unknown (Baird 2000). Most mortality events are not directly observed and are only recorded when the animal is not seen with its matriline in subsequent encounters. Based on annual census data, it is estimated that between 1974 and 2008, 96 SRKW and 176 NRKW died, but of these only 19 SRKW and 5 NRKW carcasses were recovered, a recovery rate of 20% for SRKW and 3% for NRKW (Barbieri et al. 2013).

Data that are collected on cetaceans that are found dead, or alive on the beach and unable to return to sea are included in stranding databases. In Canada, DFO maintains a marine mammal incident database that captures stranding data (CRP unpublished data) and in the USA, NOAA maintains a database for both a national marine mammal stranding network and a west coast marine mammal stranding network which began in the 1980's. We have only presented information from stranding data that is linked to a specific killer whale population (i.e., after the population censuses began in the 1970's). Table 5 summarises the incidences of mortality events presumed to be due to vessel strike (vessels of any type were included). Using the time range in Table 5 (1974-2016), both the NRKW and SRKW populations had three mortalities [4]. Again, these are likely to be underestimated values due to the unknown causes of death in missing and presumed dead animals.

*Table 5 – Timeline of reported mortalities resulting from ship strikes in NRKW and SRKW. Data used is limited to post-1970s after population censuses had begun so that mortalities of individuals can be linked to a specific population.*

| Year | Population | Individuals (#) | Description |
|---|---|---|---|
| 1974 | NRKW | 1 | Fatal ferry strike, possibly NRKW (Baird 2002; Ford et al. 1994) |
| 2006 | NRKW | 2 | One injured near Campbell River and died following year (A82)<br>One fatal strike near Prince Rupert (C21) (Gaydos and Raverty 2010; Williams and O'Hara 2010) |
| 2006 | SRKW | 2 | One male (L98) killed by a tugboat propeller in Nootka Sound;<br>One stranded female (L112) was determined to have died from blunt trauma presumed from a ship strike (Gaydos and Raverty 2010; Williams and O'Hara 2010) |
| 2016 | SRKW | 1 | J34, an 18 year-old male found dead in Sechelt died from blunt force trauma presumed to be from a vessel strike (Ferrara et al. 2017; DFO 2017a) |

### 2.6.3 Interactions/Thresholds/Non-linearities [9,10]

Interactions of vessel strike with other threats have not been clearly demonstrated, though it has been hypothesised that killer whales might have a higher risk of vessel strike when exposed to loud sounds, which could impair the whales' ability to detect vessels (Erbe et al. 2018) [9].

In addition, as outlined in the previous acoustic disturbance section (2.5.6), when prey abundance is low, killer whales may ignore disturbances and put themselves at greater risk of ship strikes during the pursuit of prey, as has been documented in other mammal species (Frid and Dill 2002; Sansom 2009; Stevens 2010) [11].

## 2.7 CONTAMINANTS [7,8,12,13]

### 2.7.1 Background

Resident Killer Whale populations are exposed to a variety of contaminants released into the marine environment historically and currently, via sources such as rivers, wastewater, storm water and atmospheric deposition (Cullon et al. 2009). Exposure to contaminants is of particular concern for SRKW because they spend much of the spring, summer and autumn in the waters of Puget Sound and Georgia Basin (Krahn et al. 2007), areas which are influenced by the major urban centres of Vancouver and Seattle. There are a range of contaminants with the potential to be of concern to Resident Killer Whale populations, as outlined in a recent review by Environment Canada (Van Zandvoort, 2019 unpubl.[2]) and in a prioritised list of contaminants for BC killer whales (Morra and Gobas, 2017 unpubl.[3]). While any of the contaminants to which Resident Killer Whales are exposed have the potential to cause negative impacts, for most, the nature of their impacts on population parameters is uncertain and so out of scope for this assessment.

Currently, contaminants of particular concern to killer whales are those that biomagnify, reaching highest concentrations in animals at the top of the food chain, and are generally ones classified as being Persistent, Bioaccumulative and Toxic chemicals (PBTs) (Ross et al. 2000; Ross 2006). Although the manufacturing of many of these contaminants has been banned since the 1970's, they still persist in the environment. When these chemicals are consumed, they bioaccumulate in the tissues of organisms and can elicit chronic forms of immunotoxicity and neurotoxicity and can result in reproductive impairment (Ross et al. 2000; Ross and Desforges 2014, unpubl.[4]; Morra and Gobas 2017, unpubl.[3]) [7,8]. Within the PBT group, two major contaminant classes, polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) have been identified as being of ecotoxicological concern for killer whales.

### 2.7.2 Polybrominated Diphenyl Ethers (PBDEs)

PBDEs are a group of synthetic contaminants that are used as flame retardants in a range of products including many electronic and household items. PBDEs are contaminants of concern due to their effects on the immune system, reproduction and development in mammals (Ross et al. 2006). PBDEs are fat-soluble and so can be measured by sampling killer whale blubber. The levels of PBDEs in killer whale blubber is of growing concern, as concentrations have been noted to be increasing in killer whales over time (Ross 2006; Guy 2018 unpubl.[5]).

PBDEs consist of a basic diphenyl ether structure with one to 10 bromine atoms, leading to 209 different combinations (congeners). The number of bromine atoms attached to the molecule and the degree of bromination are linked to different properties and toxicity. For example, congeners with 4-8 bromine atoms typically bioaccumulate more as they bind to sediment better than those with 9-10 bromine atoms. PBDEs have been noted to have impacts on thyroid hormones and neuro-development and may be carcinogenic (US EPA 2017).

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[2] Van Zandvoort, A. 2019. Review of contaminants of concern to Southern Resident Killer Whales and/or Chinook salmon. An evergreen report prepared for Environment and Climate Change Canada. Last modified April 4, 2019.

[3] Morra, J., and Gobas, F. 2017. Recovering British Columbia's resident killer whales by tackling pollution. Report prepared for the Department of Fisheries and Oceans Canada. Unpublished

[4] Ross, P.S. and Desforges, J.P. 2014. Towards a framework for organizing a forward-looking pollution workshop for SARA-listed marine mammals in February 2015. Report prepared for Fisheries and Ocean Canada, Fisheries Management–Species at Risk, March 18, 2014. Ocean Pollution Research Program, Vancouver Aquarium Marine Science Center. 31pp. Unpublished.

[5] Guy, J. 2018. A risk analysis of legacy pollutants, PCBs, PBDEs and new emerging pollutants in the Salish Sea Killer Whales. Master's thesis, Simon Fraser University. Unpublished

### 2.7.3 Polychlorinated Biphenyls (PCBs)

Legacy PBTs, such as Polychlorinated biphenyls (PCBs), which are structurally similar to PBDEs, were assessed as being in the threat category of greatest concern to Resident Killer Whales in BC (Morra and Gobas 2017, unpubl.[3]). Further, a recent health risk-based evaluation of 25 different contaminants in RKW indicated that in terms of overall chemical exposure, PCBs were the pollutant of greatest concern to RKW (Gobas and Ross 2017 unpubl[6].).

PCBs were historically used in products such as lubricants, paints, adhesives, flame-retardants, and particularly in heat resistant oils in electrical equipment (such as transformers and capacitors) (Clark 1999; Ross et al. 2006) and were released in significant amounts into the environment from industrial practices beginning in the 1920's. It is estimated that from 1930-1993, 1.3 million tonnes of PCBs were produced worldwide (Breivik et al. 2002a) and around 1.4% of this entered the environment (Breivik et al. 2002b).

Evidence of the environmental accumulation and persistence of PCBs and their impacts lead to PCB bans in the late 1970s in the USA and Canada, and by 2001 they were banned under the Stockholm Convention (Stockholm Convention 2001[7]; Jepson et al. 2016). Although Canada banned the production, import and sale of PCBs in 1977, a ban on environmental release came later (1985) and the use of PCB-containing equipment is still allowed until the end of service life. Despite these bans, PCBs persist in the environment due to a combination of the persistent nature of these contaminants, as well as continuing PCB releases as a result of accidental spills, fires (Environment Canada 2018), river run-off, and long range atmospheric transport and deposition (Desforges et al. 2018). PCBs are also still widely present as an 'inadvertent' contaminant in the pigments and dyes of many consumer products such as newspapers, cereal packaging, plastic bags and even sidewalk chalk (Stone 2016). PCBs from these sources can enter the ocean through improper waste disposal or management.

PCBs consist of one or more combinations of man-made organic chemicals (biphenyls) similar in structure. There are 209 PCB congeners which vary based on the chlorination (number of chlorine atoms) and the position of those atoms (Heindel and Zoeller, 2006; Environment Canada 2018). The specific combination of congeners present can be an important factor in their impacts/toxicity. 136 PCB congeners have been found in killer whales (Addison and Ross 2000). The composition of PCB congeners present in SKRW and NRKW populations are similar, with congeners 153, 138, 52, 101, 118, and 180 accounting for nearly 50 percent of the total PCB load (Ross et al. 2000). PCBs are linked to cancer and nervous system problems, infant death, birth defects, and brain damage (Sullivan et al. 2007). A description of the biological impacts of PCBs is provided in section 2.7.6.

### 2.7.4 PCB Bioaccumulation in Resident Killer Whales

PCBs released into the marine environment end up in the sediment and water column, where they are taken up by sediment dwellers and plankton. From there, PCBs biomagnify up the food web (Pearce and Gobas 2018 unpubl.[8]). Consequently, those most affected by the toxicity and persistence of PCBs in the environment are the long-lived animals at the top of the food chain

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[6] Gobas, F., and Ross, P.S. 2017. Health risk-based evaluation of emerging pollutants in Killer whales (*Orcinus orca*): priority setting in support of recovery. Unpublished research report.

[7] Stockholm Convention. 2001. 2256 UNTS 119; 40 ILM 532 (2001).

[8] Pearce, R., and Gobas, F. 2018. Evaluation of trends in PCB concentration and food- web transfer of PCBs to Resident Killer Whales. Report prepared for the Department of Fisheries and Oceans Canada. Unpublished.

(such as killer whales), as PCB levels can remain elevated due to bioaccumulation. This is exacerbated by the fact that cetaceans have a limited ability to metabolise higher chlorinated PCBs (Boon et al. 1997; Ross et al. 2000). PCB levels in killer whales are influenced by age and sex (Ross et al. 2000). For example, males become increasingly contaminated as they age, while levels decrease with age in reproductively active females, as they offload a percentage of their PCB burden to their young during gestation and lactation, potentially affecting the development of young calves (Ross et al. 2000).

The consumption of Chinook salmon is a significant source of PCBs for these populations of fish-eating killer whales (Ross et al. 2000). The PCB loads of Chinook salmon stocks vary, and the majority of PCBs present in returning adult Chinook salmon are obtained while out at sea (Cullon et al. 2009; O'Neill and West 2009). The PCB concentrations in Chinook salmon sampled in BC and Washington exceed a dietary threshold (8 µg/kg) estimated as protective for 95% of killer whales (Cullon et al. 2009). Southern Chinook salmon stocks consumed by SRKW have higher PCB contamination levels than the northern stocks consumed by NRKW, in particular the PCB load of Chinook salmon sampled in Puget Sound (in the Salish Sea) was up to five times higher than other populations (O'Neill and West 2009). The higher PCB loads in southern stocks is attributed to the fact that some Chinook salmon, termed 'residents', do not migrate, but rather remain in the Salish Sea area year round (O'Neill and West 2009), which includes areas highly contaminated with PCBs (Ross et al. 2004, 2006). Approximately 29% of hatchery subyearlings and 45% of yearlings that entered Puget Sound remained as residents (O'Neill and West 2009). Further increasing the exposure of SRKW to PCBs, the lipid content of more southerly Chinook salmon stocks is also lower, and SRKW may need to increase salmon consumption to compensate (Cullon et al. 2009). A small portion of RKW diet may include local non-salmonid fish, so consumption of fish residing in the more contaminated southern habitats may also contribute to the higher PCB loads observed in the SRKW population (Ross et al. 2000; 2006). Overall, SRKW are predicted to consume 6.6 times more PCBS through their diet than NRKW (Cullon et al. 2009 Ross et al. 2006).

### 2.7.5 PCB Loads in Resident Killer Whales Over Time

The burden of fat-soluble PCBs can be measured from samples of killer whale blubber. DFO began collecting biopsy samples of Resident and Transient Killer Whale blubber in 1993 (Ross et al. 2000). Analysis of data from the first three years of sampling (1993-1996; SRKW n=6; NRKW n=26; Transients n=15) found ∑PCB levels in SRKW were three times higher compared to NRKW, likely due to different contaminant exposure from habitat and diet. SRKW have a higher overlap with the population centres and industrial areas of southern BC and northern Washington State, which contaminant studies on seals indicate is an area highly contaminated with PCBs (Ross et al. 2004, 2006, 2013). The sampling program by DFO continued, extending the time series from 1993-2009 for NRKW and 1993-2004 for SRKW (Ross et al. 2013 unpubl.[9]; Guy 2018 unpubl.[5]). A separate US study collected SRKW samples in 2004, 2006, and 2007 (Krahn et al. 2007; 2009), and analysis indicated that PCB levels exceeded thresholds for health effects in marine mammals, and also that juveniles had significantly higher concentrations of POPs than adults due to maternal transfer (Krahn et al. 2009). A decrease in PCB levels was reported for SRKW sampled in 2004/2006 compared to 1993-1995 (Krahn et al. 2007). However, the analysis done by Krahn looked at 45 PCB congeners, and the analysis of previous samples consisted of 205 congeners (Ross et al. 2004, 2006, 2013 unpubl.[9]). Analysis of SRKW

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[9] Ross, P.S., Desforges, J-P.W., Dangerfield, N.J., Crewe, N.F., Dubetz, C.P., Fischer, M.B., Fraser, T.L., and Ross, A. R. 2013. Blubber concentrations of PCBs, PBDEs, PCDDs and PCDFs from 1993 to 2009 in killer whales (*Orcinus orca*) from the North East Pacific Ocean. Unpublished

samples collected by NOAA (USA) was conducted in 2015. In these samples ∑PCBs from the SRKW population ranged from 10-48 (geometric mean = 24) mg/kg lipid in males and 3-44 (geometric mean = 10) mg/kg lipid in females (Guy 2018 unpubl.[5]; Appendix II).

The compilation of all ∑PCB data collected to date on the killer whale populations in BC, allowed an analysis of trends over the complete time range (1993-2015 for SKRW, 1993-2009 for NRKW) (Source of PCB data: Krahn et al. 2007;2009; Ross et al. 2013 unpubl; Guy 2018 unpubl.[5]). PCB concentrations accumulated in killer whales varied by dietary preference, calving order, reproductive history, birth year and matriline membership (Pearce and Gobas 2018, unpubl.[8]). Male and female SRKW ∑PCB levels did not significantly change from 1993-2015, indicating that PCBs continue to persist in SRKW (Table 6, Table 7; Gobas and Ross 2017 unpubl.[6]). NRKW females' ∑PCB levels also showed no significant change 1993-2009). However, male NRKW samples did exhibit a significant decline over the same time period (Table 6; Gobas and Ross 2017, unpubl.[6]). The lack of an observed decrease in females was attributed to a trend being masked by the higher individual variability in values in females as a result of differences in reproductive success.

The observed differences in PCB load between males and females may indicate that reproductive offloading can introduce significant variability in these observations, particularly in populations where reproduction has been affected and pregnancy failures may be occurring. Male PCB levels may be a more reliable indicator of temporal PCB changes in KW exposure to PCBs, as they are less likely to have as much variability as females with different reproductive histories. This is supported by evidence from a ∑PCB trend analysis from the larger and healthier Resident Killer Whale population in Alaska, where declines in PCB levels have been observed in both sexes over a similar time period (Gobas and Ross 2017, unpubl.[6]).

A recent food web analysis (Pearce and Gobas 2018, unpubl.[8]) incorporated sediment ∑PCB values (Guy 2018 unpubl.[5]) into an existing bioaccumulation model (Lachmuth et al. 2010; Alava et al. 2012, 2016) to examine PCB transfer from sediments to killer whales in different areas. The sediment measurements that inform the model indicate significant declines in total PCBs in samples from the North coast (2011-2015) and BC Strait of Georgia (2010-2017), whereas samples in US SKRW habitat showed an increase (2010-2016), although the data did not have a good linear fit. The model estimates of ∑PCB concentrations in adult male and female RKW based on these sediment values were generally close to the observed, except for when using sediment from the US SRKW habitat. In these cases, the model greatly over predicted ∑PCB levels in killer whales, as ∑PCB levels in the sediments of Puget Sound are not representative of the rest of the SRKW habitat. While Puget Sound sediments are highly PCB contaminated, it is estimated that SRKW spend only 6% of their time in that area (Pearce and Gobas 2018 unpubl.[8]). In the BC areas of SRKW habitat, the model better predicted PCBs in killer whales, and SKRW are thought to spend 18% of their time in BC SRKW critical habitat and 3% in the BC Strait of Georgia (Lachmuth 2010; Pearce and Gobas 2018 unpubl.[8]).

*Table 6 - Summarised ∑PCB data for male Northern and Southern Resident Killer Whales. Age categories are based on Vélez-Espino et al. 2014a; 2014b). The category for calves (<1) was excluded due to lack of data. Values under each mean represent the range of mean values within that category. Sample data where sex was unknown were excluded.*

| **Eco-type** | **Date range** | **Source** | **Congeners analysed** | **∑PCB geometric mean, and range mg·kg-1 lw** | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | **Juveniles (1-9)** | **n** | **Young males (10-21)** | **n** | **Older males (22+)** | **n** | **All adults males (10+)** | **n** |
| **NRKW** | 1993-1996 | Ross et al. 2000 | 205 | 17.27<br>10.80-27.90 | 3 | 20.6<br>20.00-21.20 | 2 | 25.2<br>2.42-49.60 | 8 | 24.28<br>2.42-49.60 | 10 |
| | 1993-2009 | Ross et al. 2013 unpubl.[9]<br>(collated by Guy 2018 unpubl.[5]) | 205 | 10.62<br>3.27-27.90 | 10 | 10.54<br>2.60 - 23.70 | 21 | 19.32<br>0.72 - 49.60 | 13 | 13.79<br>0.72-49.60 | 34 |
| **SRKW** | 1993-1996 | Ross et al. | 205 | - | - | 63.20<br>sole value | 1 | 119.80<br>5.93-192.0 | 3 | 105.78<br>5.93-192.00 | 4 |
| | 1993-2009 | Ross et al. 2013 unpubl.[9]<br>(collated by Guy 2018 unpubl.[5]) | 205 | - | - | 27.93<br>8.53 - 63.20 | 4 | 151.98<br>5.93-248.00 | 4 | 89.96<br>5.93-248.00 | 8 |
| | 2004, 2006 & 2007 | Krahn et al. 2007; 2009 | 45 | 38.00<br>34.00-41.00 | 3 | 40.43<br>22.00-74.00 | 7 | 91.33<br>38.00-180.00 | 3 | 55.70<br>22.00-180.00 | 10 |
| | 2015 | Guy 2018 unpubl.[5] | 209 | 37.60<br>27.70-47.50 | 2 | - | - | 10.30<br>sole value | 1 | 10.30<br>sole value | 1 |
| | 1996-2015 | All combined<br>(Ross et al. 2000; 2013; Krahn, 2007; 2009; Guy 2018 unpubl.[5]) | 45, 205 and 209 | 37.84<br>27.70-47.50 | 5 | 35.88<br>8.53-74.00 | 11 | 111.53<br>5.93-248.00 | 8 | 67.73<br>5.93-248.00 | 19 |
| | 1996-2015 | All combined<br>*excluding* Krahn data | 205 and 209 | 37.60<br>37.60-47.50 | 2 | 27.93<br>8.53-63.20 | 4 | 123.65<br>5.93-248.00 | 5 | 81.11<br>5.93-248.00 | 9 |

*Table 7 - Summarised ∑PCB data for female Northern and Southern Resident Killer Whales. Age categories are based on Vélez-Espino et al. 2014a; 2014b). The category for calves (<1) was excluded due to lack of data. Values under each mean represent the range of mean values within that category. Sample data with unknown sex were excluded.*

| **Eco-type** | **Date range** | **Source paper** | **Congeners analysed** | **∑PCB geometric mean, and range mg·kg-1 lw** | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Juveniles (1-9) | n | Young females (10-30) | n | Older reproductive (31-50) | n | Post reproductive (51+) | n | All adult females (10+) | n |
| **NRKW** | 1993-1996 | Ross et al. 2000 | 205 | 40.67<br>9.80-109.00 | 4 | 7.09<br>0.48-15.40 | 6 | 1.81<br>1.04-2.58 | 2 | 19.65<br>9.45-25.50 | 3 | 9.56<br>0.48-25.50 | 11 |
| | 1993-2009 | Ross et al. 2013 unpubl.[9] (collated by Guy 2018 unpubl.[5]) | 205 | 25.76<br>4.12-109.00 | 12 | 7.79<br>1.37-25.00 | 21 | 2.38<br>1.04-6.37 | 5 | 17.1<br>9.45-25.5 | 3 | 7.87<br>0.48 -25.50 | 30 |
| **SRKW** | 1993-1996 | Ross et al., 2000 | 205 | - | - | 74.70<br>sole value | 1 | 34.70<br>sole value | 1 | - | - | 54.70<br>34.70-74.70 | 2 |
| | 1993-2009 | Ross et al. 2013 unpubl.[9] (collated by Guy 2018 unpubl.[5]) | 205 | - | - | 74.70<br>sole value | 1 | 34.70<br>sole value | 1 | - | - | 54.70<br>34.70-74.70 | 2 |
| | 2004, 2006 & 2007 | Krahn et al. 2007; 2009 | 45 | 62<br>sole value | 1 | 17.97<br>4.30-45.00 | 3 | 8.90<br>sole value | 1 | 67.33<br>27.00-120.00 | 3 | 37.83<br>4.30-120.00 | 7 |
| | 2015 | Guy 2018 unpubl.[5] | 209 | - | - | 16.85<br>3.01-44.10 | 5 | 4.83<br>sole value | 1 | - | - | 14.85<br>3.01-44.10 | 6 |
| | 1996-2015 | Guy 2018 unpubl.[5] (collated Ross et al. 2000; 2013; Krahn 2007; 2009; Guy 2018 unpubl.[5]) | 45, 205 and 209 | 62<br>sole value | 1 | 23.65<br>3.01-74.70 | 9 | 16.14<br>4.83-34.70 | 3 | 67.33<br>27.00-120.00 | 3 | 30.89<br>3.01-120.00 | 15 |
| | 1996-2015 | As above, but excluding Krahn samples | 205 and 209 | - | - | 26.50<br>3.01-74.70 | 6 | 19.77<br>4.83-34.70 | 2 | - | - | 24.81<br>3.01-74.70 | 8 |

### 2.7.6 Biological Impacts of PCBs [7,8]

PCBs can have a variety of dose-dependent toxic effects on mammals including immunosuppression, reproductive impairment, and impacts to the endocrine system (Buckman et al. 2011; Jepson et al. 2016; Lundin et al. 2016; Mongilo et al. 2016). Reproductive impacts are of particular note as PCBs have been implicated in decreases in cetacean reproductive success [6] and increases in calf mortality [8], suggesting they may have an important role in population declines and suppression of population recovery in some killer whale populations (Jepson et al. 2016; Hall et al. 2018; Desforges et al. 2018). In grey seal pups, maternally-transferred PCBs disrupt glucose uptake and lactate production in fat (adipose) tissues, and POP levels in 3 week old seal pups are high enough to impact adipose function (the ability to regulate and generate blubber), potentially impacting survival (Hall et al. 2001; Robinson et al. 2018). The effects that POPs have on adipose function is considered to be highest in young marine mammals, and are modified by nutritional state and the depth of blubber (Robinson et al. 2018). For example, post-weaning, POP levels in fasting young seal pups can increase further as lipids mobilise, with lipophilic POPs concentrating in remaining blubber and less lipophilic POPs being released into the blood (Debier et al. 2003a; 2003b; 2006; Louis et al. 2016). This may be relevant to other marine mammals such as killer whales as elevated POP levels have been associated with altered adipose gene expression (Buckman et al. 2011).

However, direct health effects of PCB contamination on RKW have not been clearly demonstrated or quantified to date due to the challenge of determining causal relationships amidst other factors influencing health (Hickie et al. 2007; Pearce and Gobas 2018, unpubl.[8]). One way to address this is to infer toxicological effects from other mammals and use these relationships in bioaccumulation models. Combining findings from related studies can be used in a ‘weight of evidence’ approach to assess population-level toxicological risk, as done by Ross (2000). In the absence of killer whale-specific information, a PCB bioaccumulation/depuration model developed by Hall et al. (2006, 2018), based on experiments on a land mammal, has been used to model the impact of PCBs on population growth as a reduction in survival of killer whale calves based on the PCB levels of their mothers at the time of their birth [8]. How PCBs impact male fecundity and the way this contributes to the overall impacts of PCBs on RKW population fecundity is uncertain, though there is some evidence that high PCB levels may impact testes development. An 18 year old male SRKW (J18) that died in 2000 had high PCB levels and undeveloped testes, unusual at that age, possibly indicating maturity was affected by PCB contamination (K. Balcomb, Center for Whale Research, WA, pers. comm.). Though this aspect has not been considered here, it is an area that may be explored further using necropsy data, and potentially incorporated into future iterations.

A recent study compared PCB concentrations in worldwide killer whale populations to concentration-response relationships for reproductive impairment and immunotoxicity-related disease mortality (Desforges et al. 2018; based on Hall et al. 2018) [7,8]. The Desforges et al. (2018) study concluded that more than half of the world’s killer whale populations are at risk of long-term population level effects as a result of the impacts of PCBs on reproduction and immune function. The model linked PCB impacts on immunity to the probability of survival using relationships between immune suppression and disease mortality (Luster et al. 1993).

With normal reproductive activity, PCB levels in females should decrease over time due to offloading from mother to calf during gestation and lactation. Levels should then increase again after reproductive senescence (a U shaped pattern, as observed in NRKW by Ross et al. 2000). In cases where levels in females are not showing this decrease, but follow similar trends to males, it may indicate reproductive failure, such as is the case in some highly contaminated European populations (Jepson et al. 2016). In industrialised areas of Europe, only small killer whale populations remain, with very low reproductive rates. The one remaining resident fish eating population in southern Europe has only 36 members, with six reproducing females, who have produced only five calves (surviving past 1 year) over 13 years of monitoring (1999-2011). ∑PCB levels in this resident tuna-eating KW population in the Strait of Gibraltar are very high, ranging from 172-315 mg/kg lipid for males and 43-858 mg/kg lipid for females (Jepson et al. 2016). The mean PCB levels for Strait of Gibraltar adult females (215 mg/kg lipid) were almost four times higher than those reported for SKRW females (55.4 mg/kg lipid - Ross et al. 2000; 2013; Krahn 2007, 2009; Desforges et al. 2018; Guy 2018, unpubl.[5]) and twice as high as in Bigg's killer whales (109 mg/kg lipid - Ross et al. 2013 unpubl.[9]).

### 2.7.7 Interactions, Threshold and Nonlinearities [12,13]

The impacts from PCBs are unlikely to be characterised by a simple linear impact-effect relationship, as there are other threats and factors present for both populations (Buckman et al. 2011) that may interact with or modulate the impact of PCBs, as well as other contaminants present that may also have impacts. As noted above, the effects of PCBs on killer whales may be mediated by nutritional stress and the amount of blubber stores, as observed in seals (Robinson et al. 2018) [12,13]. The PCB contamination and prey availability threats may interact, because killer whales suffering nutritional stress (from lack of prey) will metabolise adipose (fat) tissue in blubber, resulting in the mobilisation of lipophilic toxins such as PCBs into the bloodstream and causing a toxic response (Krahn et al. 2002, Mongillo et al. 2016). It has been proposed that at these times, systematic POP concentrations and associated bioavailability to organs increases (Aguilar et al. 1999; Lundin et al. 2016). Nutritional stress resulting from a lack of Chinook salmon may act synergistically with high contaminant burden resulting in higher calf mortality and reduced fecundity (Mongillo et al. 2016) [12,13]. Though the Biggs population have higher contaminant loads in the blubber, the population is reportedly healthy and increasing (Ford et al. 2007). Blubber-bound toxin levels may be higher in Bigg's whales but they may not have the same toxic effects as in prey-limited populations that are mobilising the toxins as a result of nutritional stress. Some support for this was provided by Lundin et al. (2016b) who examined the relationship between PCB levels measured from SRKW scat and Chinook salmon abundance and found that concentrations of contaminants were highest and had the highest toxic potential when prey abundance was the lowest, and that the contaminants likely originated from endogenous lipid stores (Lundin et al. 2016).

## 2.8 PATHWAYS OF EFFECTS DISCUSSION

The development of the Pathways of Effects conceptual model provides an illustration and summary of the evidence for the structure of the system under investigation. This structure forms the basis for the population viability analysis modelling in the subsequent section of the paper. In the proposed PoE model for Resident Killer Whales, prey availability appears to be a central node, with six linkage pathways to fecundity and mortality, including two interactions with other threats. The interactions make the assessment of impacts more difficult, as they imply that impacts are not additive and may have non-linear or threshold effects.

Additional threats to Resident Killer Whale populations were identified in the SARA recovery strategy (DFO 2018a) that were not included in the current model, and include contaminants other than PCBs and PBDEs, incidental mortality in fisheries, oil spills, disease, harmful algal blooms, as well as seismic exploration and other high-intensity sounds.

Incidental injury and mortality in fisheries appears to be a rare occurrence in Resident Killer Whale populations. Stranded killer whales have been found with fishing gear in their stomachs (Ford et al. 1998) and a small number of entanglements have been reported, but most were not fatal (DFO 2018a).

There is a low probability of killer whales being exposed to a major oil spill, but if they are exposed there can be serious consequences. The 1989 Exxon Valdez oil spill in Alaska was strongly linked to an unprecedented mortality event, after killer whales were seen surfacing in the oil slick (Matkin et al. 1999). It was hypothesised that mortality was caused by the inhalation of petroleum vapors (Matkin et al. 1999).

Three pathogens have been found in wild killer whales: *Brucella*, *Edwardsiella tarda*, and cetacean poxvirus. *Brucella* can impact killer whale reproduction, potentially affecting fecundity by causing abortions. An *E. tarda* infection was the cause of death in a SRKW male in 2000, and cetacean poxvirus has been implicated in calf mortality (Gaydos et al. 2004; Ford et al. 2000; Van Bressem et al. 1999; DFO 2018a).

Harmful algal blooms have been identified as a possible threat to Resident Killer Whales (DF0 2018a). There may be a risk to killer whales if they are exposed to the toxins released from a harmful algal bloom, given that mortalities of other marine mammals have been linked to exposure to biotoxins (Krahn 2002; DFO 2018a). Mass mortality events such as that associated with a toxic bloom of the harmful alga *Alexandrium tamarense* in the St. Lawrence Estuary in August 2008 provided strong evidence for trophic transfer of algal toxins through the food web (Starr et al. 2017). Mortalities of marine fishes, birds and marine mammals including beluga whales (*Delphinapterus leucas),* harbour porpoises (*Phocoena* phocoena), a fin whale (*Balaenoptara physalus),* and several species of seals (mainly grey seals, *Halichoerus* grypus) were associated with this bloom. In BC, harmful algae have been linked to survival of wild salmonids; marine survival of sockeye salmon *Oncorhynchus nerka* (Fraser River Chilko stock) was more than four times higher in years when there were minor or no blooms of the alga *Heterosigma akashiwo* in the Strait of Georgia during the juvenile sockeye outmigration period versus years with major blooms (Rensel et al. 2010).

Seismic surveys employ airguns, which generate high intensity sounds. Cetacean species have mixed reactions to seismic surveys; some have been shown to avoid areas where seismic surveys are occurring (Stone 2003; Calambodkis et al. 1998). The current moratorium on oil and gas exploration in BC ensures that permits for seismic surveys are rarely issued. If the moratorium were lifted, this threat could become significant, especially to NRKW.

The pathways of effects conceptual model is iterative in nature, and should be reassessed at intervals to utilise new data being collected and to capture current research and understanding of the components making up this system.

## 2.9 PATHWAYS OF EFFECTS USED IN THE POPULATION VIABILITY MODEL

Based on the review of the available literature and data, only portions of the PoE conceptual model could be parameterised with any confidence in the Population Viability Analysis. The linkage pathways (both direct and interaction) that were not able to be parameterised and were removed from this assessment are identified by grey lines in the overall pathways of effects model in Figure 8. The structure of the final modified PoE diagram used in the PVA model in this assessment, (with the grey lines removed), is outlined in Figure 9. The threat names differ on the PoE diagram used to inform the PVA, as they represent the specific aspects used to parameterise threats in the assessment. The Disturbance (acoustic) threat is represented by the combined effects of vessel noise and vessel presence, as at present there is no way to tease apart impacts from vessel presence from vessel noise. Although there is some evidence in other cetaceans that there may be an interaction between acute acoustic disturbance and strandings or vessel strikes, this was not included in the PVA model. The Disturbance (physical) threat is represented by the effects from vessel strikes. Prey availability is represented by Chinook salmon abundance in the PVA model, even though it is acknowledged that other types of salmon are also consumed. For the contaminants threat, despite the evidence that other contaminants are present in Resident Killer Whales, only PCBs were included. The details of how threats were parameterised is described in the following PVA section.

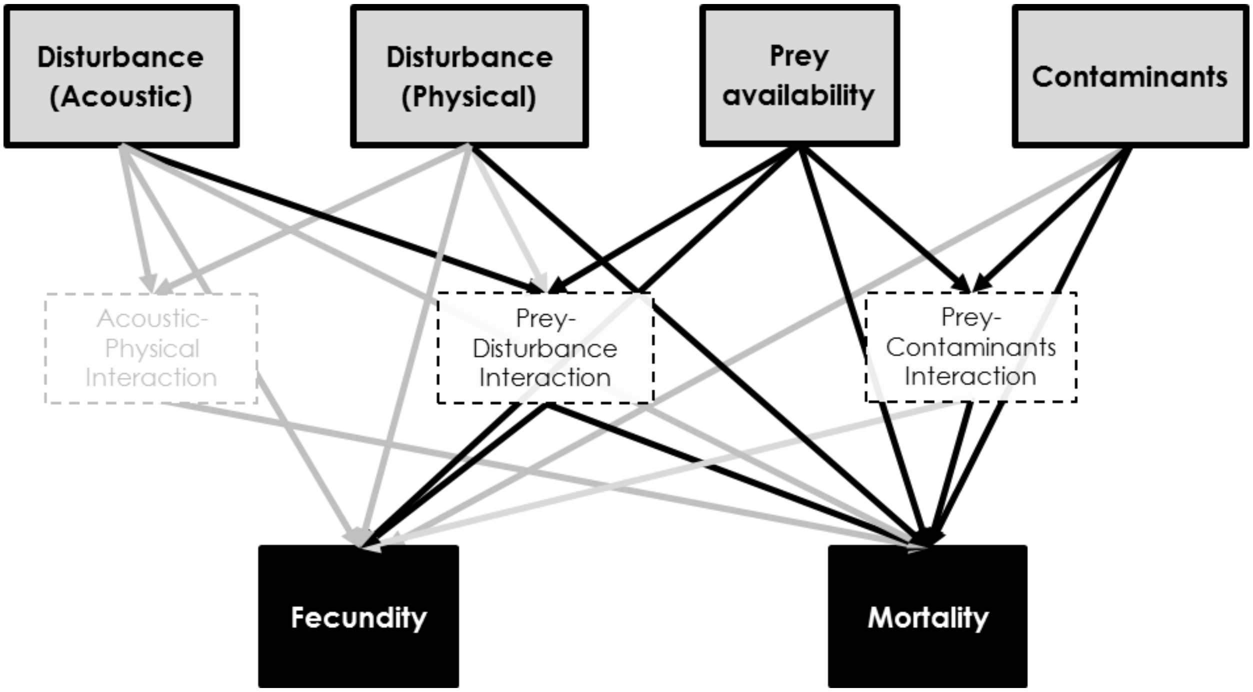

*Figure 8 – Overall pathways of effects model for Resident Killer Whale populations highlighting the direct linkage pathways and interaction linkage pathways that were able to be quantified (black lines) and those which could not be quantified in this assessment (grey lines).*

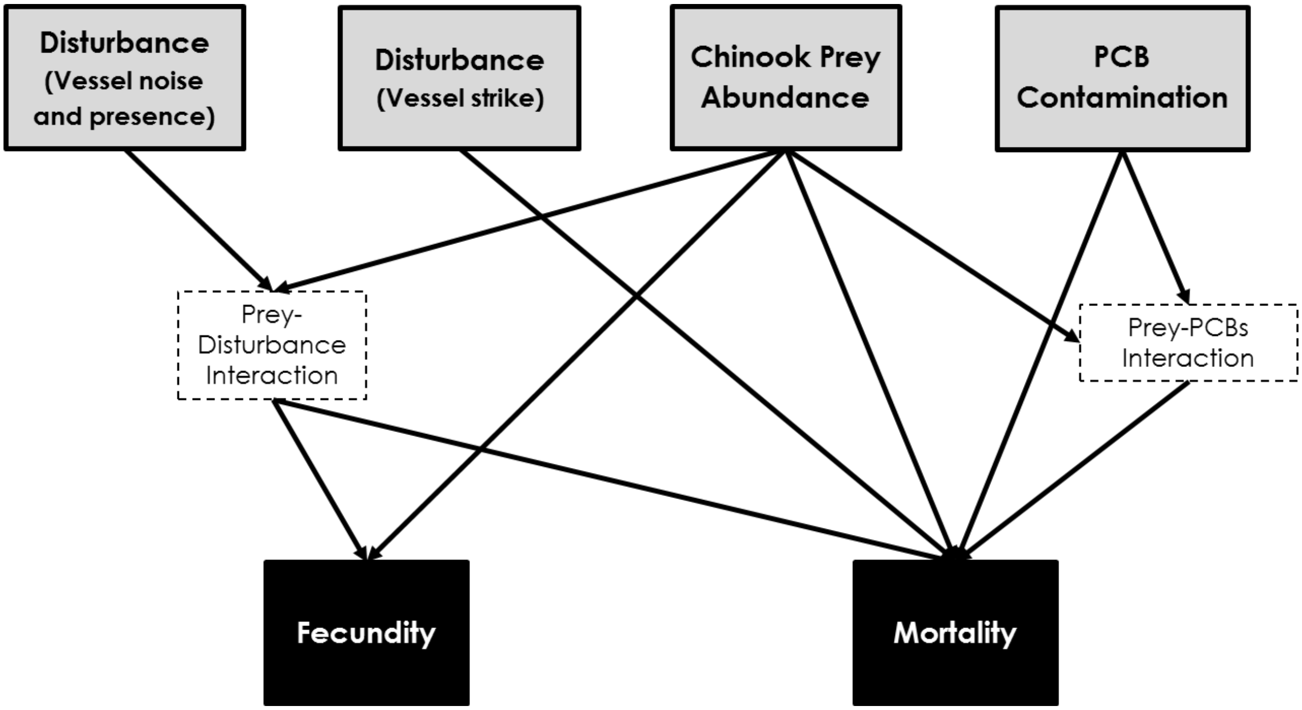

*Figure 9 - Modified PoE conceptual model for Resident Killer Whale populations used for population viability analysis (PVA).*

# 3 POPULATION VIABILITY MODEL

This cumulative effects assessment for NRKW and SRKW builds upon the methods and results of previous work (Taylor and Plater, 2001; Ward et al. 2009; Vélez-Espino et al. 2014a, 2014b; Williams et al. 2017; Lacy et al. 2017; DFO 2018a) and includes recent research advances and the compilation and review of unpublished data and results.

## 3.1 KILLER WHALE POPULATION MODEL METHODS

### 3.1.1 Killer Whale Census

Population models were constructed for each of the SRKW and NRKW populations separately using census data from DFO's Cetacean Research Program encompassing 1979-2017 (DFO CRP, unpublished data). Annual population surveys have occurred without interruption since 1973 for NRKW and 1976 for SRKW (DFO Cetacean Research Program; Center for Whale Research, CWR). For the purposes of this assessment, census data from 1979 onwards was used. By using demographic rates starting in 1979, the time series is composed mostly of data from direct observations rather than reconstructed data (Olesiuk et al. 1990).

Each annual census consists of photo-identification surveys in which individuals are identified using their unique fin shapes and saddle patch colouration (Bigg et al. 1987; Ford et al. 2000; Baird 2000, 2002). Census data were used to determine genealogical relationships and estimate life history parameters (Bigg et al. 1990; Olesiuk et al. 2005; Vélez-Espino et al. 2014a; 2014b), capturing birth and death information. A death is recorded when an individual is not observed with its matriline on several subsequent encounters where all other members of the matriline are present (this may be anywhere from one to several survey seasons, depending on the frequency that the given matriline is documented). Cause of death is difficult to assign, as killer whale carcasses are not often recovered (Olesiuk et al. 2005). When a birth is recorded, the assignment of mother to each calf is based on the observation of close associations following birth, during the same photo-identification surveys. The NRKW and SRKW populations are fully surveyed, with every individual in the population identified and tracked. Therefore there is no uncertainty around the population size numbers and any increases in population are not the result of increased sampling effort. The comprehensive census data allows for a detailed understanding of life history parameters for these Resident Killer Whale populations that can be used in population modelling.

### 3.1.2 Life History Parameters

The killer whale reproductive system was defined as polygynous and sexually dimorphic with observed population parameters (Table 8; Olesiuk et al. 2005; Vélez-Espino et al. 2014a; Ward et al. 2010).

*Table 8 - Life history parameters for Northern and Southern Resident Killer Whales used in the population models.*

| Life history parameter | Males | Females | Reference |
|---|---|---|---|
| Age of sexual maturity (y) | 12-18 | 12-18 | Olesiuk et al. 2005 |
| Maximum age of reproduction (y) | 70 | 50 | Vélez-Espino et al. 2014a |
| Maximum lifespan (y) | 70 | 90 | Vélez-Espino et al. 2014a; Olesiuk et al. 2005 |
| Maximum number of calves/brood | - | 1 | Olesiuk et al. 2005 |
| Sex ratio at birth | 0.5 | 0.5 | Olesiuk et al. 2005 |
| Gestation time (months) | - | 17 | Olesiuk et al. 2005 |

Sexual maturity can begin as young as 10 y in males (Olesiuk et al. 2005); and is evident when the dorsal fin begins growing in height (Olesiuk et al. 2005). Males are presumed to be reproductively active throughout their lives and breeding success increases with age (Barret-Lennard 2000; Olesiuk et al. 2005; Ford et al. 2011). There is some evidence that older, larger males are preferred as mates; for SRKW, only two males have sired half of the calves born since 1990 (Ford et al. 2018). It is unknown whether male breeding success is similar limited in other populations or if this is a consequence of the small population size in SRKW. In females, calving begins around age 10 years (mean age of first conception), a female's first surviving calf is most often born when she is 12-17 years old (Olesiuk et al. 2005; National Marine Fisheries Service 2008). One calf is carried per gestation, with an average gestation time of 17 months (range 15-18 months) (Olesiuk et al. 2005). Fecundity declines with age in females, with reproductive senescence occurring after age 50 y (Vélez-Espino et al. 2014a). In the model, individuals were randomly assigned an age of maturity between 12-18 y (Olesiuk et al. 2005), and the maximum age assigned for reproduction was 50 y for females (age of senescence) and 70 y for males (maximum lifespan) (Olesiuk et al. 2005; Vélez-Espino et al. 2014a).

Resident Killer Whale offspring remain with their natal family group or matriline (Bigg et al. 1990) and mating primarily occurs between individuals from different matrilines (Barrett-Lennard 2000; Ford et al. 2011). In the case of NRKW, mating typically occurs between individuals from different acoustical clans (Barrett-Lennard 2000). There is no evidence of interbreeding or dispersal between populations, and SRKW and NRKW populations are acoustically, genetically, and culturally distinct (Barrett-Lennard 2000; Barrett-Lennard and Ellis 2001; Ford et al. 1998; 2000; Ford et al. 2011). Calves are dependent on their dams for 1 year after birth, so if a mother died in the model simulations in the first year after birth of the calf, her calf also died. Sex ratio at birth was assumed to be equal.

### 3.1.3 Population Parameters

Killer whale vital rates (mortality and fecundity) have been estimated for a series of age classes within each population (Table 9a,b). Individual-based population models were constructed in the population modelling software Vortex (Lacy and Pollak 2014). The life cycles of these two populations were modeled as two-sex stage-structured models.

Population sizes and vital rates before the impact of human activities, such as aquarium removals, are unknown. Carrying capacity is therefore difficult to estimate, but is likely influenced by a combination of prey abundance, habitat requirements and limits imposed by the social structure of the populations. For this modelling exercise, carrying capacity was set sufficiently high as to have no effect on the simulated populations (carrying capacity for SRKW: 300; NRKW: 500 individuals), but the relationship between vital rates and prey abundance was included (see Section 3.3.3).

Small populations can be susceptible to the effects of inbreeding depression. A recent analysis of genotypes from the SRKW population suggested that inbreeding may be occurring, however there was limited evidence that these individuals have lower survival or fecundity than normal (Ford et al 2018). We have little information on inbreeding in cetaceans. Previous PVA efforts have used various levels of lethal equivalents to estimate the effect of inbreeding on reproductive success. Taylor and Plater (2001) used 2.0 lethal equivalents from the value for humans and chimpanzees (Ralls et al. 1988), while Manlik (2016) used the mid-range value for inbreeding (3.14 lethal equivalents) from Ralls et al (1988). In the current model, the mean value for wild species (O'Grady et al. 2009) was used: 6.29 lethal equivalents (Lacy et al. 2017).

*Table 9a - Age-specific mortality rate for each Resident Killer Whale population: Southern Resident Killer Whales (SRKW), Northern Resident Killer Whales (NRKW) and Southern Alaska Resident Killer Whales (SARKW).*

| **Age class (years)** (male and female combined) | **SRKW**[1] | **NRKW**[1] | **SARKW**[2] |
|---:|---:|---:|---:|
| 0-1 | 0.215 (SD=0.284) | 0.078 (SD=0.082) | 0.054 (SD=0.244) |
| 1-2 | 0.019 (SD=0.047) | 0.028 (SD=0.019) | 0.003 (SD=0.040) |
| 2-5 | 0.019 (SD=0.047) | 0.028 (SD=0.019) | 0.010 (SD=0.054) |
| 6-10 | 0.019 (SD=0.047) | 0.028 (SD=0.019) | 0.012 (SD=0.064) |
| 10-16 | 0.015 (SD=0.033) | 0.011 (SD=0.012) | 0.008 (SD=0.032) |
| 17-51 | 0.033 (SD=0.054) | 0.011 (SD=0.025) | 0.023 (SD=0.066) |
| 51+ | 0.072 (SD=0.108) | 0.117 (SD=0.114) | 0.217 (SD=0.292) |

[1] Vélez-Espino et al. 2014a – 1987-2011 [2] Matkin et al. 2014 – 1984-2010

*Table 9b- Age-specific fecundity rate for females in each Resident Killer Whale population: Southern Resident Killer Whales (SRKW), Northern Resident Killer Whales (NRKW) and Southern Alaska Resident Killer Whales (SARKW).*

| **Age class (years)** **Females only** | **SRKW**[1] | **NRKW**[1] | **SARKW**[2] |
|---:|:---:|:---:|:---:|
| 10-30 | 0.116 (SD=0.077) | 0.142 (SD=0.046) | 0.233 (SD=0.118) |
| 31-50 | 0.069 (SD=0.074) | 0.101 (SD=0.051) | 0.154 (SD=0.118) |

[1] Vélez-Espino et al. 2014a – 1987-2011 [2] Matkin et al. 2014 – 1984-2010

### 3.1.4 Model Structure

The Southern Alaska Resident Killer Whale (SARKW) population has a similar life history strategy but is relatively removed from the threats to which the SRKW and NRKW are exposed. To define the reference conditions for the current work, we used the mortality and fecundity rates that have been estimated from SARKW census data (census began in 1984) (Table 9a,b), as the rates expected from a population in unrestrained growth. The SARKW population is not considered to be pristine as it is exposed to anthropogenic impacts, and was notably impacted in 1989 by a major oil spill (Exxon-Valdez), resulting in a 33% loss of the resident AB matriline (Matkin et al. 2008). However, these impacts are not incorporated into the vital rates for SARKW (Table 9a,b) as these anomalous deaths were excluded from the data analysis in Matkin et al. (2014). The rates and age/sex structure of the SARKW population were found to be similar to NRKW in their period of unrestrained growth, except that the age of maturity was one year earlier for SARKW (Olesiuk et al. 2005; Matkin et al. 2014).

The SARKW vital rates data (Table 9a,b) were used in the SRKW and NRKW population models to represent the reference vital rates that determine the growth of each population in the absence of anthropogenic threats. This is an important change from the SRKW model defined by Lacy et al. (2017), where the “baseline” was defined using the mean demographic rates that were observed from recent decades and would therefore include current threat levels. Model scenarios were developed based on individual and cumulative threats. The threats (described in further detail in section 3.3) were then included in the model as modifiers of the SARKW reference vital rates.

The population genealogical and demographic data were partitioned to allow model validation and verification; the complete set of living animals in the year 2000, with their known dams, sires, calving histories, and genealogies, were used as the starting population for each of the population models (SRKW and NRKW). This allows a comparison of the modeled populations to the observed populations as an evaluation of the ability of the model scenario output data to represent observed data.

Model simulations were run on each scenario 10,000 times and summary statistics were recorded for population growth rate (*r*), population size at each time step ($N_t$), and probability of extinction. The population size at each year (mean and standard deviation) was compared to the observed (realised) population size for each population from the census survey data. Population growth rate (*r*) was quantified as the exponential rate of increase, according to the following equation:

$$r = \ln[\frac{N_{t+1}}{N_t}]$$

The model results (the predicted population size resulting from threat-modified reference vital rates) were then compared to the observed (realised) population dynamics from the census data over the same time period (2000-2017). The assumption of this approach is that if we can define a model that replicates the realised dynamics for both the SRKW and NRKW populations, we have constructed an appropriate model for the system.

The observed population growth rate (*r*) for the two populations from 2000-2017 was -0.002 for SRKW and 0.024 for NRKW. In 2017, the population size was 77 and 308 individuals for SRKW and NRKW, respectively.

## 3.2 MODEL VERIFICATION AND VALIDATION

### 3.2.1 Inspection Approach

In order to determine how representative the simulation output data were, we examined the simulated population size, population growth rate, age structure, and sex ratio for each scenario. We used the inspection approach method to validate the models (Law et al. 1991). For the model scenarios that most closely approached the observed population parameters, we ran the simulation scenario with historical input data in place of the parameter chosen from a distribution, in this case the yearly Chinook salmon index data. A valid model should closely resemble the observed killer whale survey data when the historical salmon index data are used (details of the index data in Section 4.3.2).

### 3.2.2 Sensitivity Analysis

Sensitivity analysis was conducted on key parameters in the model to test the impact of uncertainty in these parameters on the results of the study. Sensitivity analysis was conducted in Vortex using the Sensitivity Testing operations. Parameters that required sensitivity analysis included: noise impact value, vessel-KW overlap, PCB impact on calf mortality, and vessel strike.

## 3.3 THREAT SCENARIOS

### 3.3.1 Aquarium Removals / Live Capture Fishery

The removal of killer whales for display in aquaria is a historic threat that complicates population modelling, in particular for the SRKW population. Comprehensive population surveys on the SRKW and NRKW populations only began soon after the end of the live capture fishery. This means that the population composition at the start of the data set is skewed and very different to the composition at present, an issue highlighted in the predictions made by Vélez-Espino et al. 2014b. Though this is not a current threat, it is important to consider in population predictions due to its long-term influence on the population.

Because of their location closer to human population centres, most removals (48 individuals) were from the SRKW population - an estimated 36% of the total population. To illustrate the residual impact of this historic threat, the 48 animals removed from the SRKW population were added into the population model at year 1980, comprised of 30 juveniles (19 males and 11 females) and 18 mature adults (10 males and 8 females). The modified demographic structure and observed vital rates over the time period (1979-2017) from Vélez-Espino (2014a) were used to project the population growth rate from the year 1980 until the present (2017).

### 3.3.2 Chinook Salmon Abundance

The Pacific Salmon Commission's (PSC) Chinook model estimates the number of "model fish" available from each of the 30 model stocks to 25 fisheries. Ford et al. (2010) reported that RKW survival rates were related to the modelled abundance of these Chinook stocks available to six fisheries (Alaska Troll, BC North Troll, BC Central Troll, West Coast Vancouver Island Troll, Georgia Strait Sport, and Washington/Oregon Troll). Three different sources of data have been used to represent Chinook salmon abundance in RKW survival investigations (Ford et al. 2009, 2010; Vélez-Espino et al. 2014b; Stredulinsky 2016): PSC Chinook model terminal run, Coded-Wire Tag (CWT)-based terminal run reconstruction estimates, and ocean abundance estimates from the PSC Chinook model. The PSC Chinook Model terminal run estimates include both hatchery and natural production plus terminal catch estimates and is available for eight Canadian stocks. The CWT-based terminal run reconstruction estimates stock abundance for runs from northern BC through California. It uses escapement information and CWT data from fisheries across the coast to reconstruct abundance from spawning areas as well as ocean fisheries to estimate fish abundance in terminal run areas. Ocean abundance includes reconstructed estimates of numbers of fish removed in fisheries as well as those escaping to spawning grounds to estimate the number in ocean. The ocean abundance is a measure of fish sufficient in size to be vulnerable to fishing gear (larger than the minimum size limit) and is therefore not an estimate of the total number of fish. The ocean abundance, estimated by the PSC Chinook model, does not represent absolute abundance, rather it represents the vulnerable fish from specific modelled stocks that are available to certain fisheries. Therefore, relative rather than absolute changes in abundance should be used.

Updated Chinook salmon ocean abundance data were obtained from DFO Salmon Program (A. Vélez-Espino, DFO, Pacific Biological Station) (1979-2017) from the 2018 PSC Chinook model calibration (Appendix I). Ocean abundance is an adequate representation of fish available for consumption by killer whales, given that the full time series of terminal run reconstruction data was not available, and ocean abundance has statistical support in previous analyses (Vélez-Espino et al. 2014b; Stredulinsky 2016).

Yearly model ocean abundance was converted to an index of abundance by standardising the value by the mean for the full time series. The Chinook index value was assigned in each model year using either a uniform or normal distribution. The uniform distribution was defined by the full range of Chinook index values recorded during the time series (1979-2017). The normal distribution was defined by the median value (for a skewed distribution) and standard deviation from the entire time series (1979-2017). Selecting a value from a distribution in each year allows the model to represent the fine temporal structure and variation in Chinook salmon abundance, and its impacts on killer whale vital rates. However, this yearly random selection does not include the autocorrelation inherent in Chinook populations where fish from a cohort will contribute to the index value over a 4-year period.

### Mortality

The relationship between prey availability and Resident Killer Whale mortality was first described by Ford et al. (2009) and a number of significant interactions between Chinook salmon abundance and RKW vital rates have been identified (Vélez-Espino et al. 2014b). However, in the more recent analysis, the effects of these interactions on RKW population growth and viability were relatively small and uncertain (Vélez-Espino et al. 2014b), bringing into question whether Chinook salmon resource availability alone can explain the recent observed population trends.

The effects of prey availability on mortality can depend on age class. Vélez-Espino et al. (2014b) found differences between the dependence of Female 1 (young reproductive females), Female 2 (old reproductive females), and Juvenile stage survival on various Chinook salmon stocks.

The relationship between vital rates and Chinook salmon ocean abundance index values was updated using the entire time series of RKW and Chinook salmon data (1979-2017). The Coastwide Index (excluding southeast Alaskan [SEAK] stock) as well as Chinook salmon runs deemed relevant to each population, with a one-year time lag, were investigated using linear regression. For SRKW, the WCVI + FL + OC runs were used (West Coast Vancouver Island, Fraser Late, and Oregon Coastal) and for NRKW the FE + PS + URB were used (Fraser, Puget Sound, and Upper Columbia River Bights) (Table 10). The model abundance was converted to an index by standardising with the mean over the entire time series (1979-2017). The Chinook salmon stock index that best explains the mortality patterns seen in both populations was tested using model selection (Akaike Information Criterion, AIC).

*Table 10 - Chinook salmon ocean abundance indices used for mortality and fecundity analyses in each of the salmon threat models. The mean ocean abundance model estimates (OA), and the minimum and maximum values for the index are shown.*

| Killer Whale Population | Salmon Index | Mean OA (# fish) | Index min | Index max |
|---|---|---|---|---|
| **SRKW** | Coastwide (excl. SEAK) | 1,104,884 | 0.608 | 1.445 |
| | WCVI + FL + OC | 373,151 | 0.467 | 1.611 |
| | WCVI | 113,778 | 0.224 | 2.371 |
| **NRKW** | Coastwide (excl. SEAK) | 1,104,884 | 0.608 | 1.445 |
| | FE + PS + URB | 261,052 | 0.584 | 1.818 |
| | WCVI | 113,778 | 0.224 | 2.371 |

The model selection Akaike Information Criterion (AIC) values for the WCVI and NRKW-SRKW stocks are close in relative value and cannot be excluded from further investigation (Table 11; Burnham and Anderson 2004). There is little support for the use of the Coastwide index to explain mortality in the updated analysis. The relationship between killer whale mortality and Chinook salmon abundance used all of the relevant stocks for each killer whale population. As done in previous analyses, the data for both killer whale populations were combined and the linear relationship between Resident Killer Whale mortality and the Chinook salmon index is represented by ($y = 1.6773 – 0.673x$, $r^2 = 0.0889$, $p = 0.012$ (Figure 10). The previous Ford et al (2009) analysis used data up to 2003 and the additional fourteen years of data has reduced the explanatory power of the prey-mortality relationship, suggesting that additional threats or impacts may be necessary to explain the population fluctuations. To compare to previous analyses, varying stocks were used in the scenario modelling; the relevant stocks used for each model scenario are shown in Table 10.

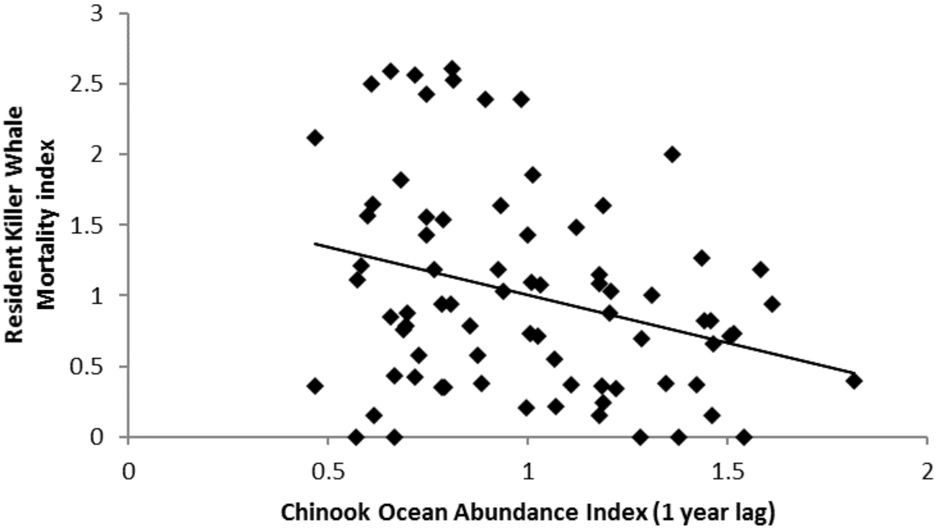

*Figure 10 - Relationship between Chinook salmon index (1 year lag) for the stocks relevant to each killer whale population (WCVI + FL + OC for SRKW and FE + PS + URB for NRKW) and mortality index (difference between mortality and the long-term mean for each population). Data from 1979-2017.*

*Table 11 - Results of linear regression model fit analysis for the three Chinook salmon ocean abundance indices and Resident Killer Whale mortality, and their Akaike Information Criterion (AIC) values. The AIC value in bold is the lowest value, and AICmin. The change in AIC (∆AIC) gives the level of support for alternate models. Models with relative AIC values greater than 10 have essentially no support, a value less than two suggests there is substantial support for the i-th model.*

| **RKW Mortality model** | **AIC** | **$\Delta AIC = AIC_i – AIC_{min}$** |
|---|---|---|
| Coastwide Chinook Index | -15,461.8 | 105.8 |
| NRKW-SRKW Chinook Index | -15,362.5 | 6.5 |
| WCVI Chinook Index | **-15,356.0** | **-** |

### Fecundity

The availability of prey can also have significant effects on reproductive success and the probability of calving. Ward et al. (2009) assessed calving probability (fecundity) of combined NRKW and SRKW females using a logistic regression model and found that RKW fecundity was highly correlated with the PSC index of Chinook salmon abundance for the WCVI troll and recreational fishery in the prior year (one year lag). The model that best supported the data included age-structured effects on reproduction and a region effect. Though the populations were combined in this study, the inclusion of a regional effect represented the lower calving rates in SRKW than NRKW.

The logistic regression analysis was repeated with the additional 10 years of data for calving probabilities and PSC Chinook model ocean abundance salmon indices, following the statistical methods of Ward et al. (2009). The WCVI stock ocean abundance was used in the update, which differs than the data series used in Ward et al. (2009). The best model to explain calving probability (lowest relative AIC value) included the relevant Chinook salmon stocks – the NRKW Chinook index for NRKW and the SRKW Chinook index for SRKW (Table 12) and included an age structure (Figure 11).

*Table 12 - Results of logistic regression model fit analysis for fecundity and the four Chinook salmon ocean abundance indices, and their AIC values. The AIC value in bold is the lowest value, and $AIC_{min}$. The change in AIC (∆AIC) gives the level of support for alternate models. Models with relative AIC values greater than 10 have essentially no support, a value less than two suggests there is substantial support for the i-th model.*

| Population | Fecundity model | AIC | $\Delta AIC = AIC_i - AIC_{min}$ |
|---|---|---|---|
| NRKW | NRKW Chinook Index | **1910.6** | **--** |
| | SRKW Chinook Index | 1912.8 | 2.2 |
| | Coastwide Chinook Index | 1911.3 | 0.7 |
| | WCVI Chinook Index | 1912.3 | 1.7 |
| SRKW | NRKW Chinook Index | 696.8 | 1.6 |
| | SRKW Chinook Index | **695.2** | **--** |
| | Coastwide Chinook Index | 696.6 | 1.4 |
| | WCVI Chinook Index | 696.71 | 1.5 |

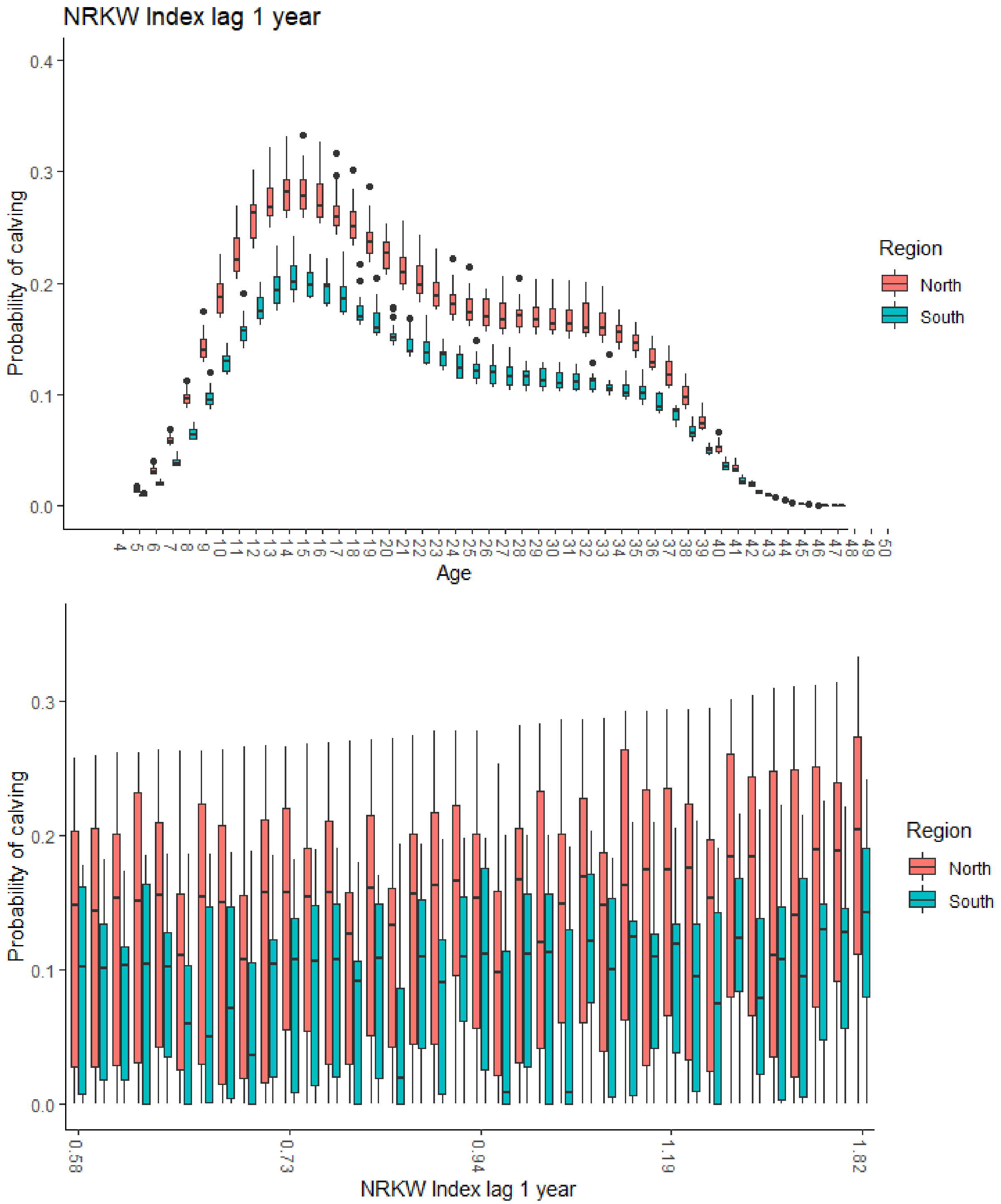

*Figure 11 - Calving probability by age (upper panel) and calving probability related to the NRKW Chinook index (lower panel)*

The PVA model used the updated binomial logistic regression coefficients, with the three Chinook index sources (Table 10). The stock used for fecundity effects were either the same population-relevant salmon stocks used for mortality (Coastwide, NRKW or SRKW), the same index as that for mortality, or the WCVI index specifically for fecundity effects. The NRKW-relevant stock (FE+PS+URB) has a lower mean abundance but a higher minimum and maximum value relative to that for the SRKW-relevant stock (WCVI+FL+OC) over the time series (Table 10). This suggests that NRKW could have greater access to Chinook salmon than the SRKW. The fecundity-relevant stock (WCVI) has a low relative mean abundance but a higher range (0.224 – 2.371), suggesting that there is higher variance in this stock relative to the others.

The percentage of adult females breeding (*Br*) was defined as a logistic function with age structure, using separate parameters for young females (< 31 years old; $Br_1$) and older females (> 30; $Br_2$). These coefficients were re-scaled for use in the model scenarios as a reduction to the reference fecundity rate.

$$Br_1 = \frac{100 * EXP(-1.88 + 0.5395 * CHINOOK)}{1 + EXP(-1.88 + 0.5395 * CHINOOK)}$$

$$Br_2 = \frac{100 * EXP(-2.96 + 0.3 * CHINOOK)}{1 + EXP(-2.96 + 0.3 * CHINOOK)}$$

### 3.3.3 Disturbance - Vessel Noise/Presence

Acoustic disturbance may come from a range of anthropogenic activities but here we focus on the impacts of vessel-associated disturbance on killer whales. In Lacy et al. (2017), it was assumed that the effect on demographic rates of reduced feeding activity is the same as a comparable reduction in prey (i.e., no behavioural compensation by killer whales). Lusseau et al. (2009) observed a 25% reduction in feeding activity when boats were present. Lacy et al. (2017) estimated that vessels are present 85% of the daytime and killer whales are foraging in the presence of vessels 78% of the time. This represents a 16.6% reduction in Chinook salmon availability in the model (25% x 85% x 78%).

A Population Consequences of Disturbance (PCoD) model (National Research Council 2005; Tollit et al. 2017) has been used to quantify the chronic and acute impacts of noise disturbance on killer whales. A noise exposure model combined with the PCoD model has been used to estimate the lost foraging time for SRKW as a combination of behavioural response and masking (Tollit et al. 2017). For the Salish Sea's busy traffic areas, where SRKW spend 23-33% of their time, individual noise disturbance events can combine to have potentially substantial impacts. The 2017 model predicted that in the Salish Sea, SRKW foraging time was decreased 20-23% of each whale day. Reductions in foraging time are based on the assumption that the impact of acoustic disturbance is the same in NRKW and SRKW. There is emerging evidence that the acoustic environment is naturally noisier in the NRKW range than in the SRKW, due to storm action, waves and other factors. This may differently affect the response of the two populations to anthropogenic acoustic disturbance (S. Vagle, DFO, Institute of Ocean Sciences, pers. comm.).

A time series of vessel activity for the study region that is comparable to the data available for killer whale population dynamics and Chinook salmon was not available. In order to estimate the relative presence of vessels for each population, we gathered data on the magnitude of vessel presence (commercial, recreational and whale watching vessels) in the range of SRKW and NRKW. This rough comparison will be used to set the vessel presence parameter for noise disturbance, which was estimated to be 85% for SRKW by Lacy et al (2017).

### Vessel Transits

Marine Communications and Traffic Services (MCTS)-Western provided summaries of commercial vessel movements in British Columbia, within the ranges of the NRKW and SRKW populations. Vessel movements are recorded for specific vessel types by MCTS at each call-in station and include all vessels using Automatic Identification System (AIS-A) that pass a call-in station (a single pass is one vessel movement). Vessel types included in the dataset include barges, tugs,  tankers, bulk carriers, container ships, cruise ships, fishing vessels, military vessels, scientific research vessels, and large yachts. For the model comparison, all vessel types were treated equally. The data collated were for Jan 1, 2017 to Dec 31, 2017 but the vessel traffic is fairly consistent across recent years with low inter-annual variability (B. Crooks, MCTS Western Region, pers. comm.). There are seasonal differences in the vessel traffic: there was 35% more vessel traffic in summer (April – September) than winter (October – March).

Vessel movements were summed for the MCTS call in points that fall within the range of each population (Figure 12). The boundary between the NRKW and SRKW was delineated by call in point seven on the west coast of Vancouver Island, and call in point 25 on the east. There are many more vessel movements (155,556) in the SRKW range compared to the NRKW range (31,065) (see map Figure 12). Based on these data, we estimate that SRKW are exposed to five times as many commercial vessel transits than NRKW.

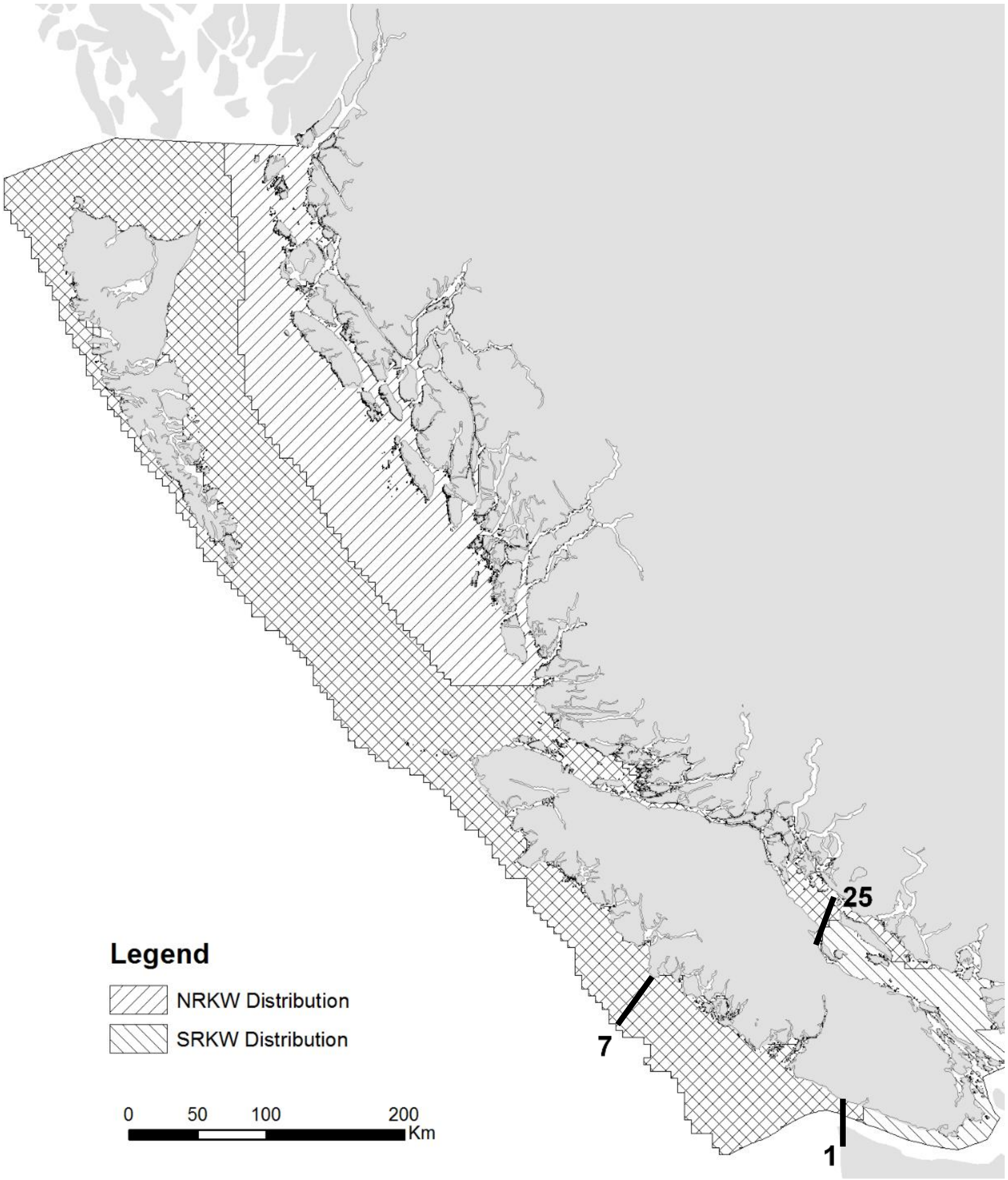

*Figure 12 - Canadian range distribution of NRKW and SRKW populations (SARA, 2015) and the three relevant numbered MCTS calling-in-points used to summarise commercial vessel movements.*

### Whale Watching Vessels

Whale watching has increased significantly, from a few boats in the 1970s to an estimated 96 active commercial whale watching vessels operating in 2015 (Osborne 1991; Seely et al. 2017). There are far more whale watching vessels in the SRKW range than the NRKW (Seely et al. 2017).

Details on the location and operations of commercial whale watch operators in BC and Washington State were catalogued in January 2019 (Serra-Sogas 2019, unpublished report[10]). The Salish Sea had the largest whale watch fleet (Canadian: 30 operators; US: 26 operators) and the highest number of vessels (Canadian: 77; US: 34). The rest of BC (Johnstone Strait, Queen Charlotte Strait, Central and North Coast and West Coast Vancouver Island) had smaller commercial fleets (31 operators,  61 vessels), suggesting that SRKW are in the presence of whale watching vessels more often than NRKW. Moreover, the SRKW population is more exposed to vessels than the NRKW population, as whale watching boats are present with the SRKW population (or part of the population) from about 09:00 to sunset, as late as 21:00 h in summer (Lusseau et al. 2009), in addition to many other types of boats that are also present engaging in opportunistic whale watching.

### Recreational Vessels

There are 55% more recreational marinas operating in the SRKW range (193 marinas) than in the NRKW range (87 marinas) (Figure 13; Clarke Murray et al. 2014).

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[10] Serra-Sogas, N. 2019. Quantifying whale watch vessel traffic in British Columbia and Washington State. Unpublished report to DFO. 23pp.

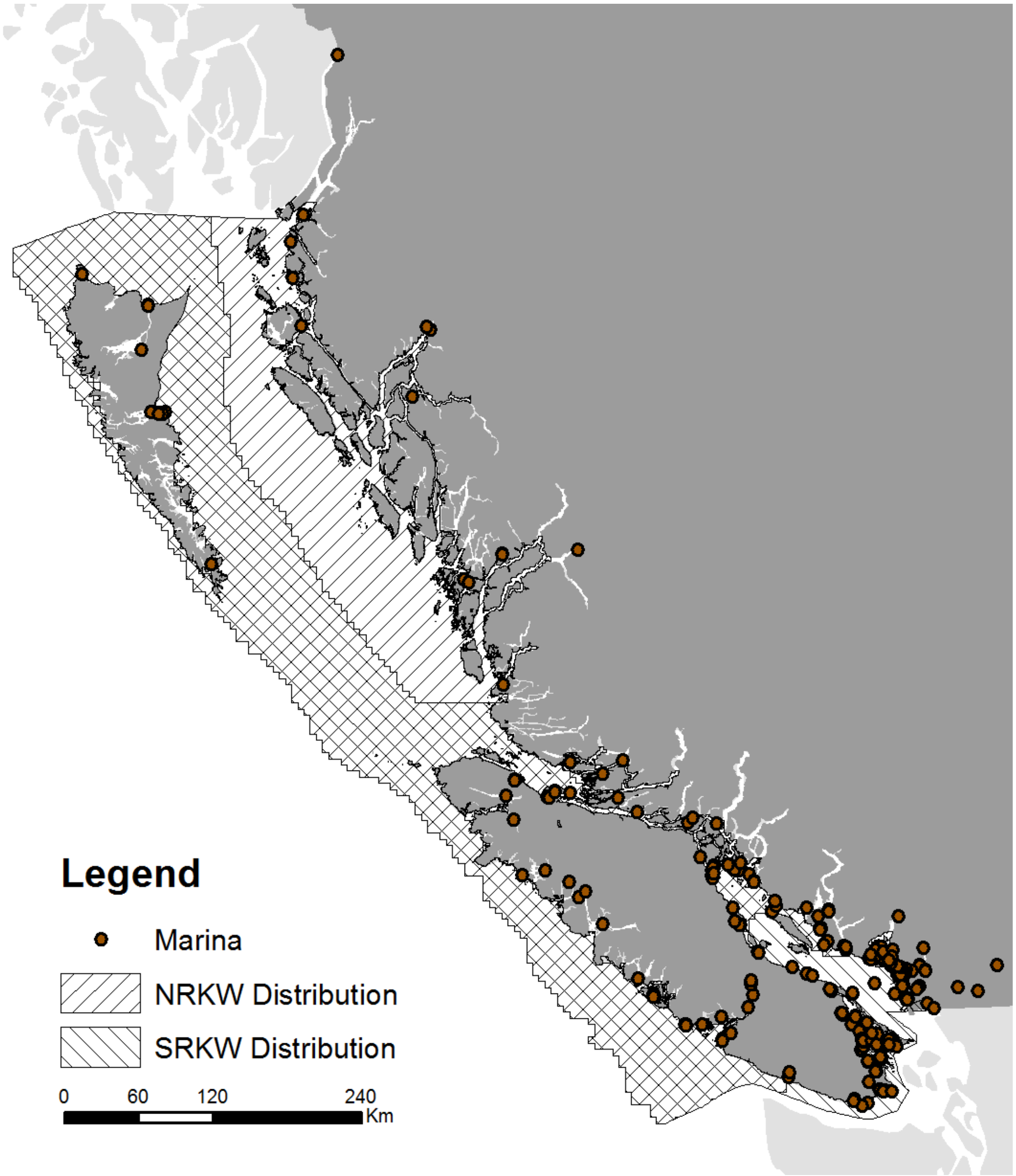

*Figure 13 - Distribution of recreational marinas in British Columbia overlaid on the Canadian range distribution of NRKW and SRKW populations*

### Vessel Noise/Presence Model Parameters

For SRKW, the noise impact was modeled as it was in Lacy et al (2017). Noise was modeled as a reduction in feeding efficiency, and was linked with the variation in Chinook salmon abundance (see Prey availability section). The noise parameter varied between 0.85 (no effect), 1.0 (current estimate of vessel presence), and 1.25 (increased noise impact) under three scenarios (Table 13). In the model scenarios, the feeding rate was used to reduce the Chinook availability and act as a modifier for the mortality rates across all age classes. In scenario one ("Noise", Table 13), noise was modelled to reduce feeding rate by 3.5%. In scenario two, ("Noise threshold", Table 13), the effect of noise was modeled as a threshold effect, where vessel disturbance only affects mortality when Chinook stocks are low. When the Chinook stock index was greater than the mean (1), there was no effect of noise disturbance but when the index was lower than 1, the noise disturbance effect was applied (3.5% reduction in feeding rate). In scenario three ("Noise threshold-high" Table 13), the effect of noise was also modeled as a threshold effect where the effect of noise increased when Chinook stock index was low. When the Chinook stock index was greater than the mean (1), the baseline effect of noise was applied (25% less feeding activity than without the presence of vessels). When the Chinook stock index was less than or equal to the mean (1), the effect of noise was increased by 25% (Table 13), which at the lowest stock index levels leads to roughly twice the mortality rate.

Based on the vessel data described above, the commercial vessel traffic in the NRKW range is five times less than that for SRKW and distributed over a much larger area, suggesting that the NRKW population spends comparably little time in the presence of vessels. Within critical habitat or key foraging areas, vessel presence may have a higher impact on foraging success of NRKW. For NRKW, noise threat parameters were modeled using the same three noise scenarios as SRKW (noise, noise threshold, and noise threshold-high) with a five-fold reduction in vessel exposure.

*Table 13 - Noise parameters and feeding rate used in the noise threat scenarios for SRKW and NRKW, and a scenario specific to NRKW (NRKW-low vessels).*

| **Scenario** | **Noise parameter** | | **Feeding rate** | |
|---|---|---|---|---|
| | High Chinook (>1) | Low Chinook (<1) | High Chinook (>1) | Low Chinook (<1) |
| Noise | 1.0 | 1.0 | 0.965 | 0.965 |
| Noise threshold | 0.85 | 1.0 | 1.0 | 0.965 |
| Noise threshold - high | 1.0 | 1.25 | 0.965 | 0.907 |
| NRKW – low vessels | 0.85 | 0.85 | 1.0 | 1.0 |

### 3.3.4 Disturbance - Vessel Strike

Current knowledge of Resident Killer Whale mortalities from vessel strikes does not indicate that there is a difference in the risk of a vessel strike threat between the two KW populations, even with different vessel densities. Limited data on cause of mortality suggests that SRKW have a slightly higher risk of strike than NRKW, 9.5% and 7.1% of cases respectively (Ford et al. 2000; Baird 2002; Williams and O'Hara 2010). Changes in the frequency of vessel transits and the characteristics of ships (quieter ships may increase strike risk) could affect this probability in the future. The vessel strike threat was modeled as a 10% probability of a fatal vessel strike each year across the entire population resulting in an animal being removed randomly from the model adult population once every ten years. The probability was shared equally between males and females of the population.

### 3.3.5 PCB Contamination

The impact of contaminants on killer whale vital rates could only include PCBs. A PCB accumulation/depuration model has been developed to link PCB levels to calf mortality in cetaceans (Hall et al. 2006; 2018). This PCB model has been used in cumulative effects assessment for SRKW (Lacy et al. 2017) and in estimating risk to global killer whale populations (Desforges et al 2018). The logistic regression model used in these studies (Figure 14; Hall et al. 2018) predicts survival based on maternal PCB level. The levels of PCBs in killer whales have been recorded from blubber samples in both populations (Ross et al. 2000), and vary greatly between sexes and through time. There were relatively few samples prior to 2000 and the high variability makes it difficult to parameterise the scenarios. The results from blubber samples obtained since 2000 have been compiled recently and analysed to update the estimated mean PCB loads, including samples from both NRKW and SRKW obtained in Canada and the US (Table 13; Pearce and Gobas 2018 unpubl.[8]; Guy 2018 unpubl.[5]; Appendix II). The PCB model simulated the accumulation of PCBs in individuals over time, based on a set accumulation rate. Females depurate (offload) an estimated 77% of PCB load to each calf during calving and nursing (Hall et al. 2006; 2018). The estimated accumulation rate can vary based on sources of PCBs in the environment and prey items (Pearce and Gobas 2018 unpubl.[8]; Desforges et al. 2018). Based on a global review and modelling of killer whale populations, Desforges et al. (2018) suggest a higher accumulation rate (6 mg/kg) for SRKW than for NRKW (1 mg/kg). Model scenarios were run using both the estimated PCB levels from Ross et al 2000 (1993-1996) and the grand mean for the entire time series (Guy 2018, unpubl.[5]; Pearce and Gobas 2018 unpubl.[8]) (Table 14), with varying accumulation rates (1, 2, and 6 mg per year). The modeled PCB concentrations were then compared to the sampled PCB levels.

The effect of PCBs on calf mortality could be in addition to the effect of prey availability. We tested an additive model which applies both mortality and fecundity impacts from prey availability and an additional impact on calf mortality from PCB accumulation. It has been hypothesised that PCBs may only be metabolised from the blubber and exhibit an impact on calf mortality when prey levels are low (Robinson et al. 2018). We next applied a threshold interaction impact so that calf mortality would only be applied when the Chinook index was less than 1 (long term mean). The modeled PCB concentrations in scenarios with various starting concentrations and accumulation rates were compared to measured killer whale PCB concentrations.

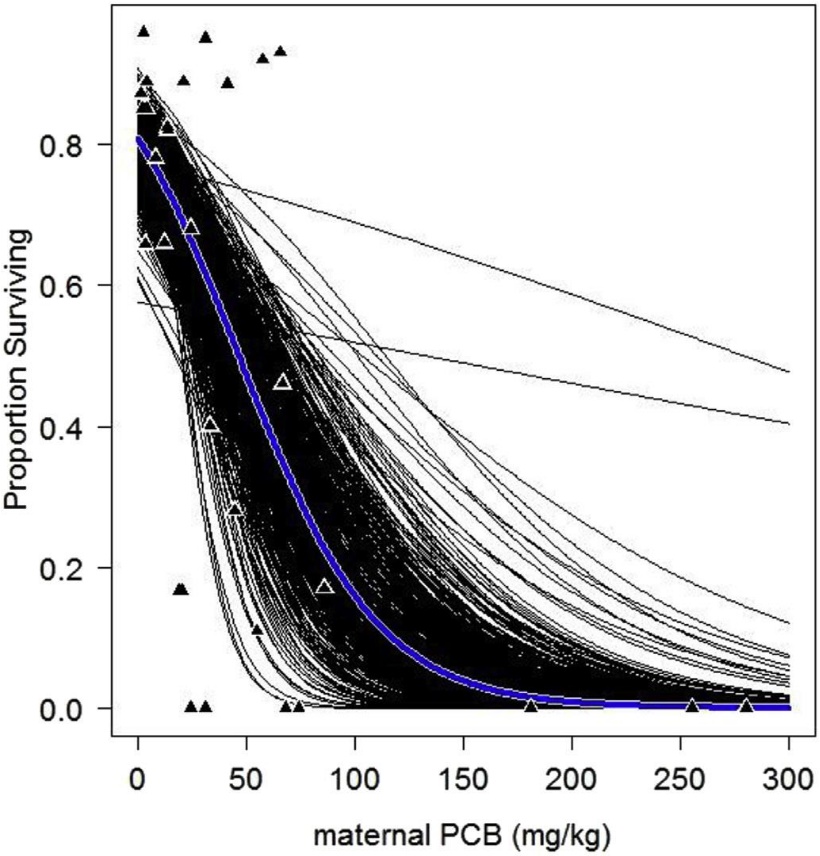

*Figure 14 - (after Fig.2 in Hall 2018) Logistic regression model predicting probability of calf survival in relation to maternal blubber PCB concentration using a subset of studies. The triangles represent the data points from the six published studies and black lines show 500 resampled regression models and the blue line shows the best fit.*

*Table 14 – Mean PCB concentration values for each Resident population by sex and the source of the values. Full dataset used to calculate means in Appendix II.*

| **Population** | **Female PCBs (mg kg$^{-1}$ lw)** | **Male PCBs (mg kg$^{-1}$ lw)** | **Source** |
|---|---|---|---|
| NRKW | 9.3 ± 2.8 (n=15) | 37.4± 6.1 (n=13) | Ross et al. 2000 |
| NRKW | 4.9 ± 2.9 (n=42) | 10.1 ± 2.4 (n=44) | Ross et al. 2000; 2013 unpubl.[9] |
| SRKW | 55.4 ± 19.3 (n=2) | 146.3 ± 32.7 (n =4 ) | Ross et al. 2000 |
| SRKW | 17.5 ± 3.2 (n=16) | 40.7 ± 2.9 (n=24) | Ross et al. 2000, 2013 unpubl.[9]; Krahn et al. 2007, 2009; Guy 2018 unpubl.[5] |

### 3.3.6 Cumulative Effects

A model including all threats (cumulative effects) was constructed using the individual threat models and the interactions between them. A representative model including all threats allows the modelling and simulation of management and mitigation actions and the effects on future population growth. Table 15 lists the details of the parameterisation of the individual threats and their interactions in the cumulative effects model.

The observed population growth is the result of the actual conditions in the environment. To test the predictive power of the cumulative model, the Chinook values for 2000-2017 were input into the model. The cumulative effects model was then projected 100 years into the future to examine the long-term forecast for the two populations under the current levels of cumulative effects.

*Table 15 - Summary of the threat parameters used in the cumulative effects model for each Resident population.*

| **Threat model** | **SRKW** | **NRKW** |
|---|---|---|
| Prey Availability | Mortality related to Chinook index relevant to SRKW:<br>Chinook WCVI + FL + OC (mean OA: 373,151, index min 0.467, max 1.611); fecundity impact related to SRKW index | Mortality related to Chinook index relevant to NRKW:<br>Chinook FE + PS + URB (mean OA: 261,052, index min 0.584, max 1.818); fecundity impact related to NRKW index |
| Vessel Strikes | One per ten years | One per ten years |
| Prey-Noise High Threshold | Feeding is expected to be reduced by 16.6% (85% × 78% × 25%) due to disturbance by boats. When the Chinook stock index was less than or equal to the mean (1), the effect of noise was increased, which at the lowest stock index levels leads to roughly twice the mortality rate. | Feeding reduced by 3% (17% x 78% x 25%) because the number of vessels was five times less in NRKW range |
| Contaminants (PCB) | Calf survival based on maternal PCB concentration (Hall et al 2018)<br>Females 17.46 mg/kg; Males 40.74 mg/kg; Accumulation rate 2 mg/kg/y; Depuration rate 0.77; When prey availability was low (less than mean index), the PCB impact was applied | Calf survival based on maternal PCB concentration (Hall et al 2018)<br>Females 4.97 mg/kg; Males 10.09 mg/kg; Accumulation rate 1 mg/kg/y; Depuration rate 0.77; When prey availability was low (less than mean index), the PCB impact was applied |

# 4 PVA RESULTS

## 4.1 REFERENCE MODEL

The reference scenario using the vital rates from the SARKW population demonstrates dramatic growth over time (Figure 15), reaching up to 178 (SD = 58.5) and 400 (SD = 88.5) individuals by 2017 for SRKW and NRKW, respectively (Figure 15).

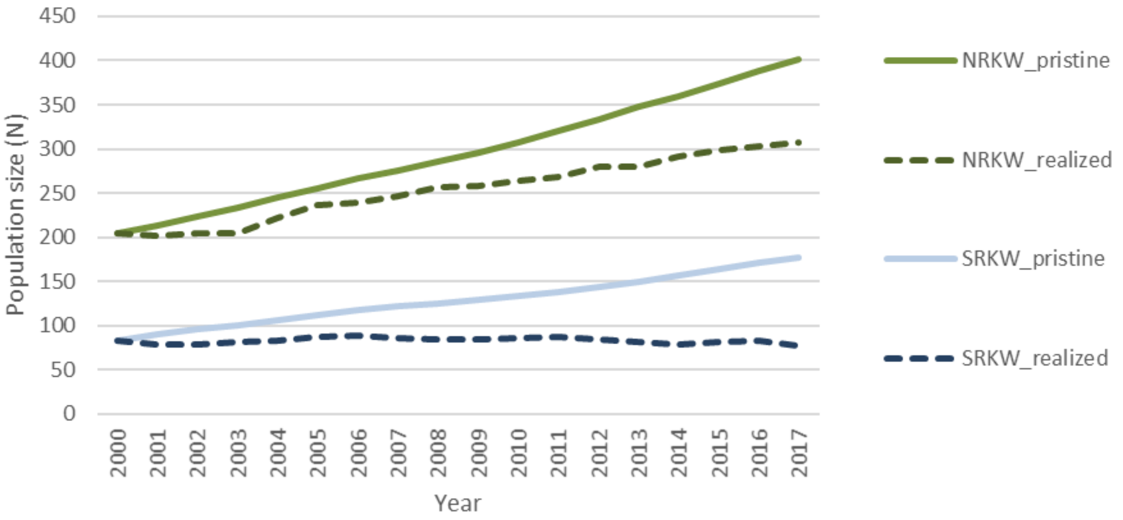

*Figure 15 - Mean reference model ("pristine") simulations for SRKW (solid blue lines) and NRKW (solid green lines), with the observed (realised) population growth (dashed lines).*

## 4.2 AQUARIUM REMOVALS / LIVE CAPTURE FISHERY

The modeled population with removed animals ("removals" scenario) used the observed SRKW mortality and fecundity rates (not the reference rates). The removals scenario shows a similar population growth trajectory (r = 0.004 ± 0.039) to the observed SRKW population (r = -0.002, 1979-2017), but remains at a higher population size (~140 animals ± 26 SD) (Figure 16).

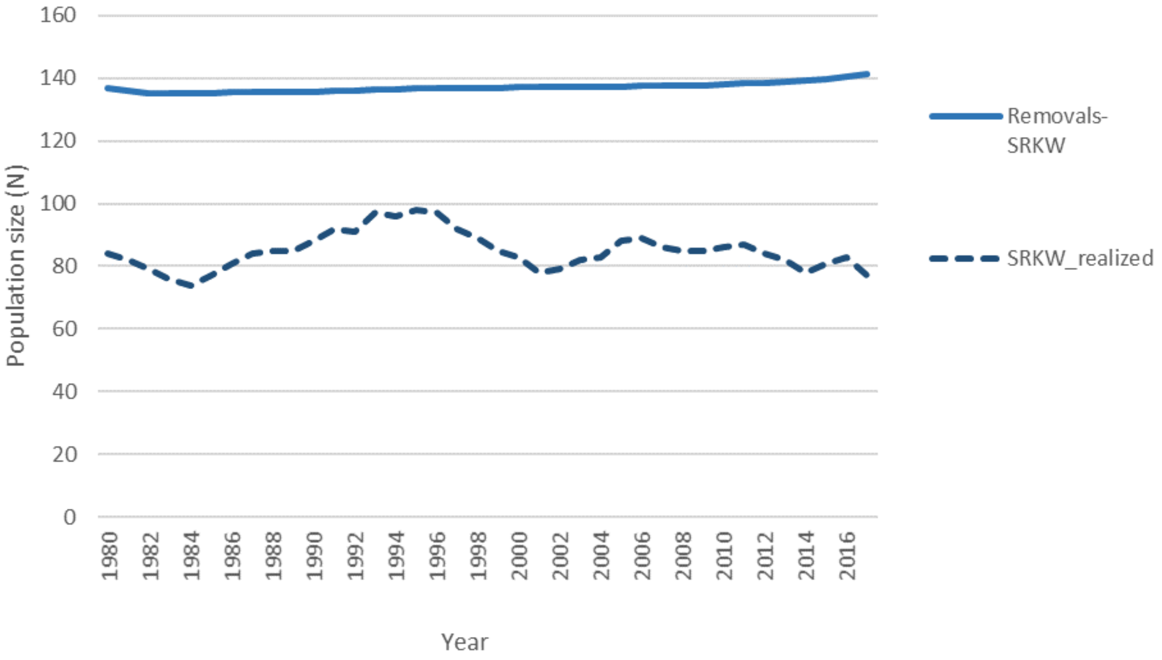

*Figure 16 – Mean modeled simulation (solid blue line) and realised (dashed line) population size for SRKW, with removed animals added back into the population in 1980.*

## 4.3 PREY ABUNDANCE

The effect of prey abundance on population size was tested with a number of scenarios to examine the effect of the Chinook index chosen, the way the index value is assigned each year, and the application of mortality and fecundity impacts (Table 16). Prey abundance effects on mortality alone does not explain the realised population growth (Figure 17). The closest model for NRKW includes the effects of prey availability on both mortality and fecundity and approaches the observed population trend, especially in the first 12 years of the simulation. For SRKW, the model scenario that incorporates impacts on both mortality and fecundity does not match the observed population growth, as it predicts slow population growth, rather than stability or decline. Therefore, salmon availability alone does not explain the population dynamics in SRKW and indicates that other threats may be affecting this population.

The distribution of Chinook stock size is not likely to be random and also not likely to be uniform across all possible values. We therefore also explored scenarios with the Chinook index being chosen from a normal distribution, with the median and standard deviation defined from the Chinook ocean abundance time series (Figure 18). Table 16 shows the results of these scenario simulations.

*Table 16 - PVA scenarios for the impact of prey abundance on both SRKW and NRKW. Scenario runs used either uniform or normal distribution to assign the Chinook abundance in each year, and impacts were on mortality, or on both mortality and fecundity. The stochastic-r, standard deviation (SD(r)), probability of extinction (PE), the number of live animals (N-extant) and the standard deviation for the number of live animals (SD(Next)) are presented for each model scenario.*

| Distribution | Mortality Index | Fecundity Index | stoch-r | SD(r) | PE | N-extant | SD(Next) |
|---|---|---|---|---|---|---|---|
| Uniform | Coastwide | | 0.0396 | 0.0900 | 0.0001 | 429.09 | 92.07 |
| Uniform | Coastwide | Coastwide | 0.0147 | 0.0889 | 0.0002 | 290.56 | 88.89 |
| Uniform | Coastwide | | 0.0396 | 0.0830 | 0 | 427.70 | 88.89 |
| Uniform | Coastwide | WCVI | 0.0243 | 0.0876 | 0 | 347.32 | 101.4 |
| Uniform | NRKW/SRKWruns | | 0.0414 | 0.0833 | 0 | 435.31 | 86.69 |
| Uniform | NRKW/SRKWruns | NRKW-runs | 0.0224 | 0.0845 | 0 | 334.82 | 97.13 |
| Uniform | NRKW/SRKWruns | | 0.0414 | 0.0823 | 0 | 435.90 | 87.01 |
| Uniform | NRKW/SRKWruns | WCVI | 0.0264 | 0.0869 | 0 | 359.36 | 101.05 |
| Normal | Coastwide | | 0.0393 | 0.0829 | 0 | 425.94 | 89.72 |
| Normal | Coastwide | Coastwide | 0.0138 | 0.0833 | 0 | 284.61 | 88.14 |
| Normal | Coastwide | | 0.0394 | 0.0831 | 0 | 427.03 | 90.26 |
| Normal | Coastwide | WCVI | 0.0235 | 0.0854 | 0 | 341.56 | 97.99 |
| Normal | NRKW/SRKWruns | NRKW-runs | 0.0416 | 0.0824 | 0 | 436.46 | 86.03 |
| Normal | NRKW/SRKWruns | NRKW-runs | 0.0218 | 0.0845 | 0 | 332.09 | 97.72 |
| Normal | NRKW/SRKWruns | | 0.0414 | 0.0825 | 0 | 436.08 | 85.71 |
| Normal | NRKW/SRKWruns | WCVI | 0.0253 | 0.0858 | 0 | 353.10 | 100.00 |

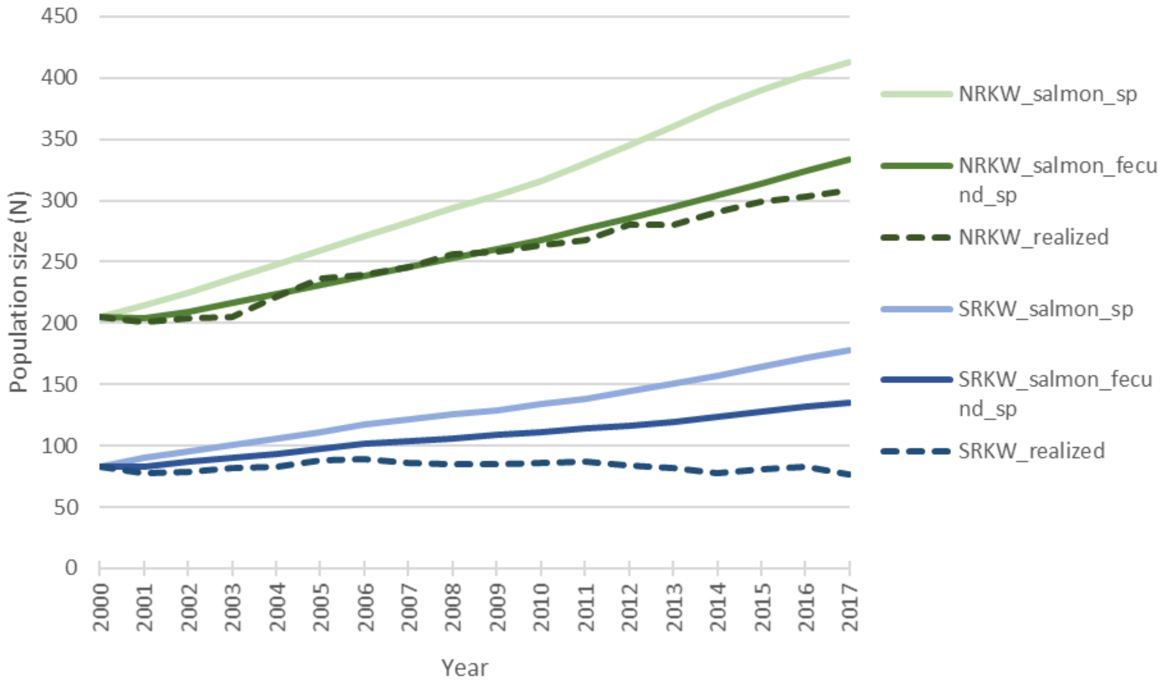

*Figure 17 - Scenarios with mean modelled simulations (solid lines) and observed ("realised" - dashed black lines) population size over time for models that include the effects of prey availability on mortality and fecundity in NRKW (green) and SRKW (blue), using a uniform distribution and specific Chinook indices for each population with impacts on mortality only (salmon_sp) and with an additional fecundity impact (using the WCVI stock index, salmon_fecund_sp).*

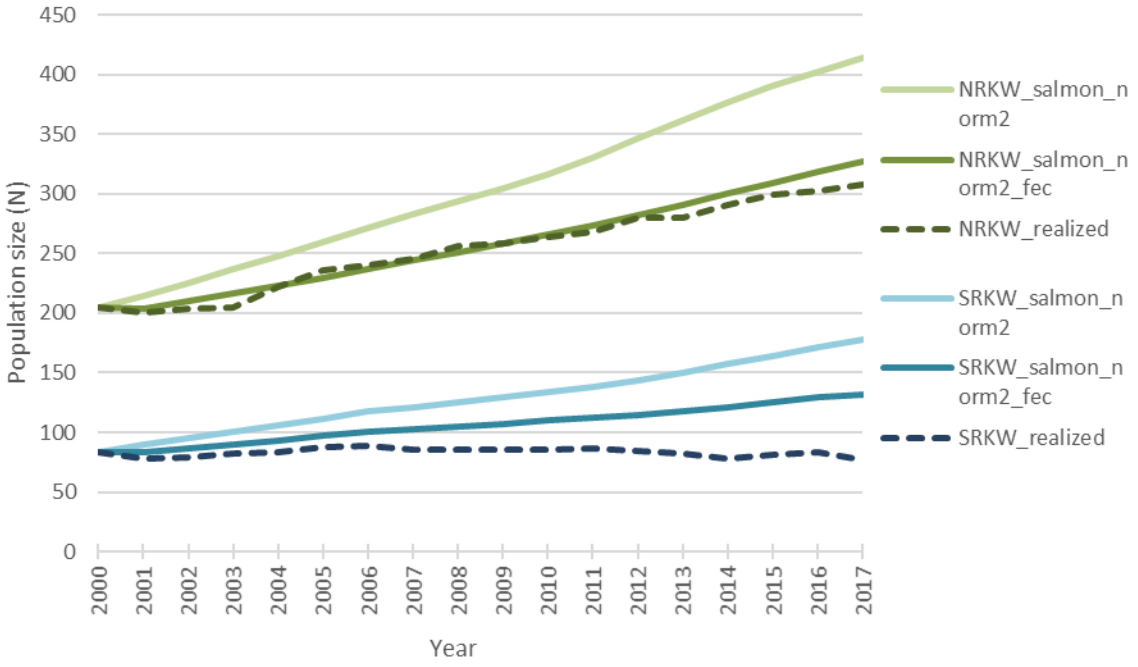

*Figure 18 – Mean scenario simulations with Chinook index chosen from a normal distribution around the mean and specific Chinook indices for each population. Impacts on mortality only (salmon_norm2) and additional impacts on fecundity using the WCVI index (salmon_norm2_fec).*

## 4.4 DISTURBANCE – VESSEL NOISE/PRESENCE

The effects of vessel noise/presence (mediated through prey abundance as a reduction in abundance of prey available) did not match the observed population dynamics (Figure 19; Table 17). The threshold scenarios were similar in impact to noise alone for SRKW, predicting higher KW abundances than the direct effect of noise scenario. The high noise threshold depressed the population more strongly in NRKW than SRKW (Figure 19). Even the high noise threshold scenario did not approach the observed population dynamics. These results suggest that vessel noise/presence disturbance alone does not control the dynamics of these populations.

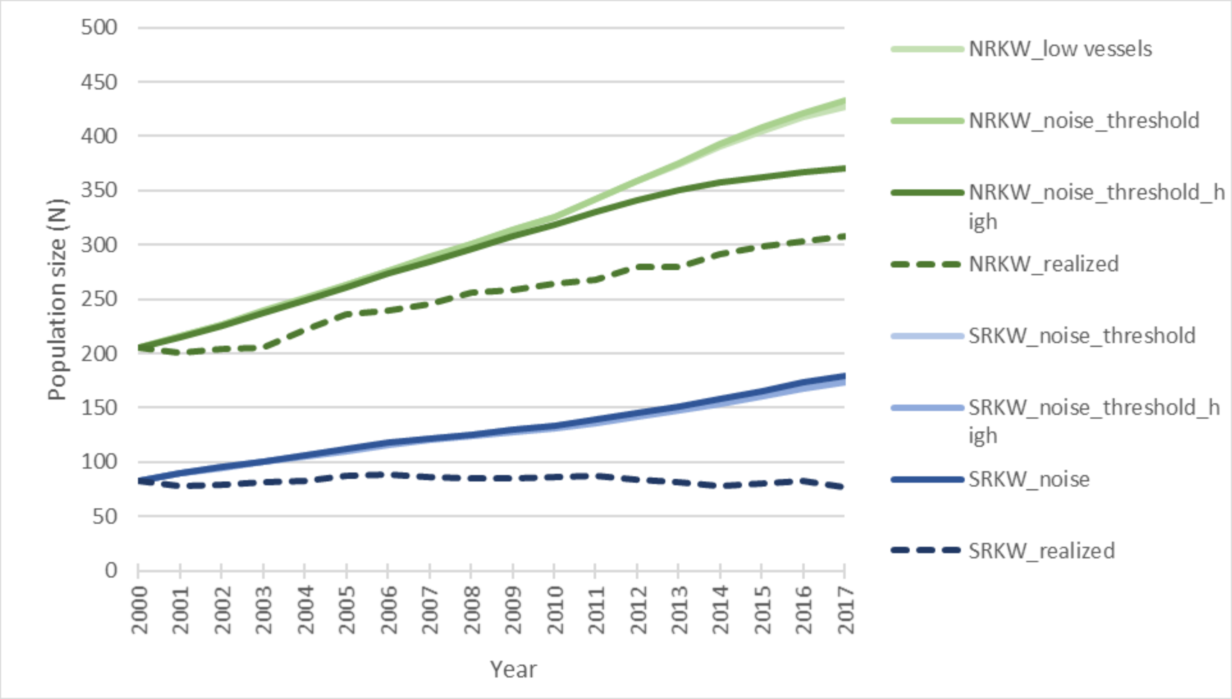

*Figure 19 – Mean modeled (solid lines) and realised (dashed lines) population size for NRKW and SRKW, with scenarios of the impacts of noise set to 25% feeding rate reduction (Noise scenario), with a threshold effect (Noise threshold) and with a higher impact when prey is low (Noise threshold high), and reduced vessel presence for NRKW (low vessels).*

*Table 17 - PVA simulation results for each noise threat scenario, including the population growth rate (stoch-r), standard deviation of r (SDr), probability of extinction (PE), and the estimated number of living animals (N-extant) and the standard deviation (SD Next).*

| Model scenario | stoch-r | SD(r) | PE | N-extant | SD(Next) |
|---|---|---|---|---|---|
| SRKW_noise | 0.0406 | 0.084 | 0 | 198.68 | 66.51 |
| NRKW_low vessels | 0.0444 | 0.075 | 0.0001 | 445.14 | 86.52 |
| SRKW_noise_threshold | 0.0409 | 0.0835 | 0 | 197.06 | 56.82 |
| NRKW_noise_threshold | 0.045 | 0.0745 | 0 | 454.83 | 74.95 |
| SRKW_noise_threshold_high | 0.0393 | 0.086 | 0.0001 | 191.52 | 57.05 |
| NRKW_noise_threshold_high | 0.043 | 0.079 | 0 | 375.73 | 51.99 |

## 4.5 DISTURBANCE - VESSEL STRIKE

The modeled populations affected by vessel strikes do not match the realised population growth (Figure 20; Table 18). These results suggest that physical disturbance alone does not control the dynamics of these populations.

*Table 18 - PVA simulation results for each strike threat scenario, including the population growth rate (stoch-r), standard deviation of r (SDr), probability of extinction (PE), and the estimated number of living animals (N-extant) and the standard deviation (SD Next).*

| Model scenario | stoch-r | SD(r) | PE | N-extant | SD(Next) |
|---|---|---|---|---|---|
| SRKW_strike | 0.0396 | 0.0849 | 0 | 192.51 | 58.01 |
| NRKW_strike | 0.0392 | 0.0799 | 0 | 367.32 | 54.64 |

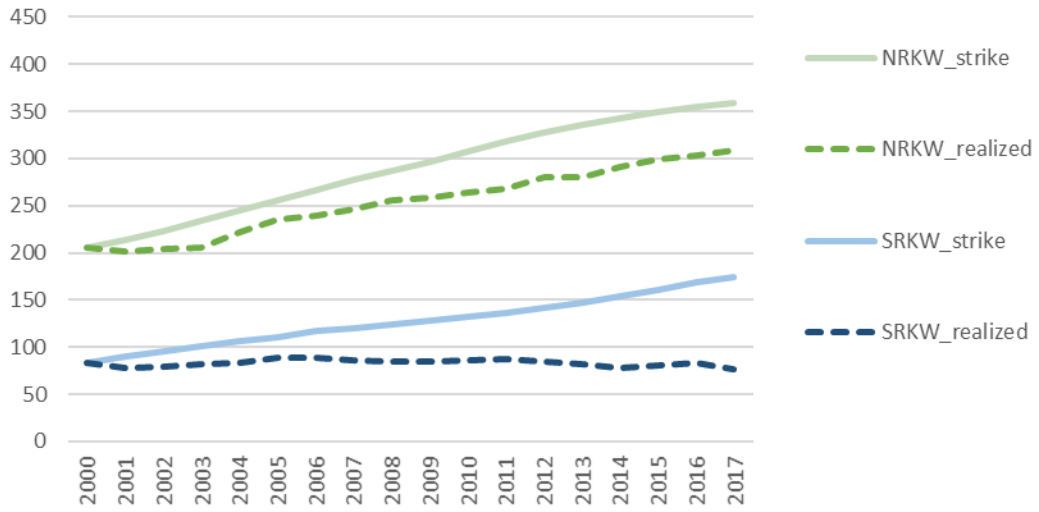

*Figure 20 – Mean modeled scenarios (solid lines) and realised population growth (dashed lines) for strike threat for the SRKW (blue) and NRKW (green) populations.*

## 4.6 PCB CONTAMINATION

The population model simulations generate a range of mean PCBs levels in adults across different initial PCB levels and accumulation rates (Table 19). The model scenarios that most closely approach the range of measured PCB levels in recent samples are those with initial PCB levels set to the grand means (Table 19), with accumulation rates slightly higher in SRKW than NRKW (2 mg $yr^{-1}$ and 1 mg $yr^{-1}$, respectively). The impact of PCBs alone does not match the realised population growth rate for either population (Figure 21; Table 19).

The results of the additive prey and PCB interaction scenario is similar to that of the prey-PCB threshold interaction impact scenario (Figure 22). The additive model applies both mortality and fecundity impacts from prey availability and an additional impact on calf mortality from PCB accumulation. In the threshold prey-pcb scenario, PCB-calf mortality was only applied when the Chinook index was less than 1 (long term mean). Both interaction scenarios resulted in population dynamics that closely resemble the realised for NRKW and are close for SRKW (Table 20). This suggests that the effects of prey availability on mortality and fecundity swamps the effect of PCB impact alone.

*Table 19 - PCB levels from blubber samples and results from model scenario simulations using varied starting PCB concentrations and accumulation rates.*

| | Sampled PCB Levels | | | Modeled PCB levels (year 2017) | |
|---|---|---|---|---|---|
| Scenario | PCB fem | PCB male | Accumulation rate | Female Mean (±SD) | Male Mean (±SD) |
| SRKW_pcb | 55.40 | 146.30 | 2 | 61.00 (± 6.3) | 112.70 (± 7.50) |
| SRKW_pcb_2mg | 17.46 | 40.74 | 2 | 48.82 (± 4.5) | 80.04 (± 3.16) |
| SRKW_pcb_3 | 17.46 | 40.74 | 6 | 142.77 (± 14.0) | 239.15 (± 7.40) |
| SRKW_pcb_1mg | 17.46 | 40.74 | 1 | 28.19 (± 2.8) | 48.88 (± 2.35) |
| NRKW_pcb_specific | 9.30 | 37.40 | 2 | 48.90 (± 4.2) | 86.00 (± 2.60) |
| NRKW_pcb_2mg | 4.97 | 10.09 | 2 | 47.75 (± 4.1) | 79.66 (± 1.75) |
| NRKW_pcb_3 | 4.97 | 10.09 | 1 | 24.65 (± 2.2) | 41.76 (± 1.00) |
| NRKW_pcb_specific_1mg | 9.30 | 37.40 | 1 | 25.80 (± 2.2) | 48.07 (± 2.01) |

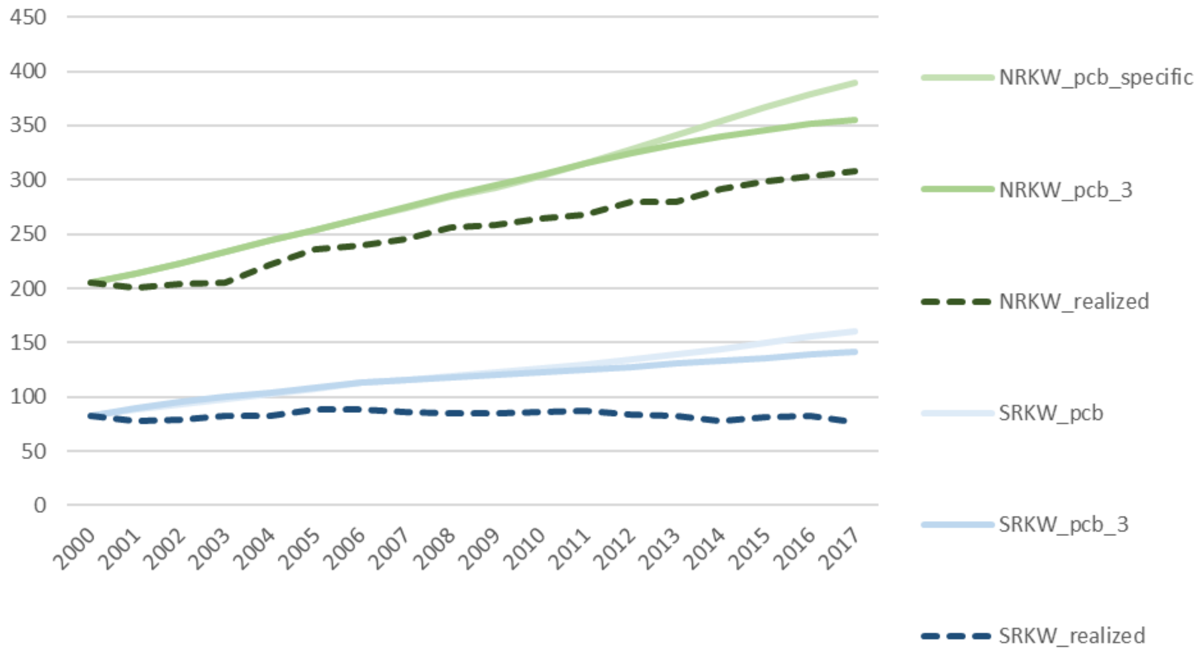

*Figure 21 – Mean model simulations of PCB impacts on NRKW (green lines) and SRKW (blue lines), with realised population size (dashed lines) and PCB levels set to levels from Ross et al. 2000 (PCB specific) or with updated values collated from Ross et al. 2000; 2013 for NRKW and Ross et al. 2000; 2013; Krahn et al. 2007; 2009; Guy 2018 unpubl.[5] for SRKW (pcb 3).*

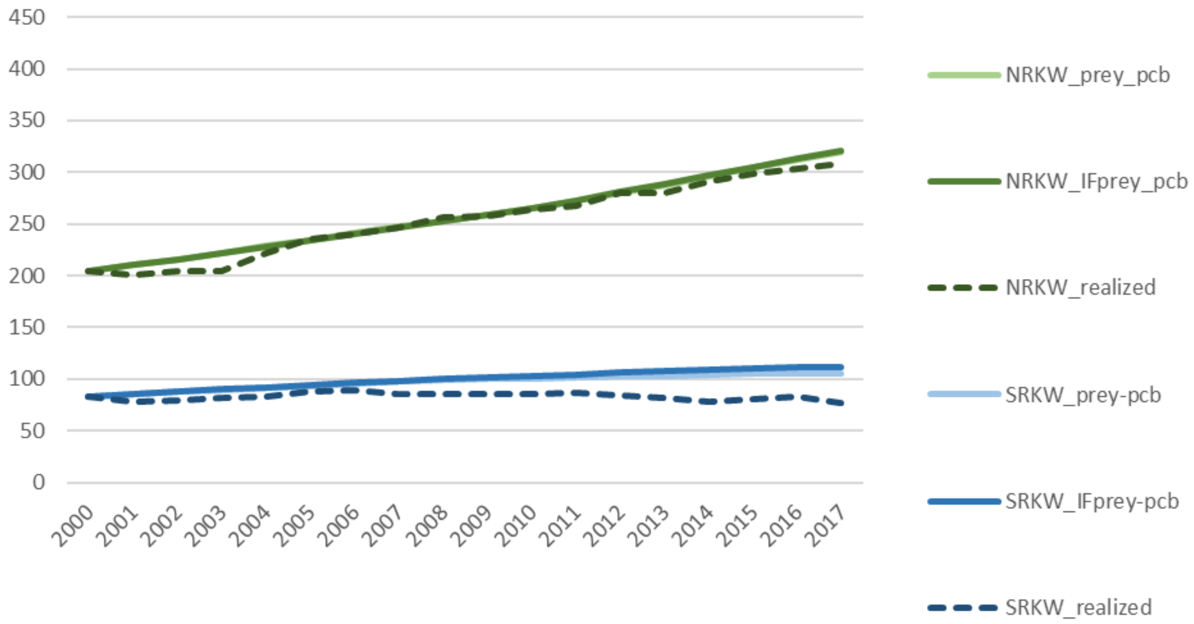

*Figure 21 – Mean model simulations of the interaction between prey and PCB threats; either as an additive model (prey abundance mortality and fecundity effects) with the additional effect of PCB effects (prey-pcb), or as an interaction between prey and PCBs, where the calf mortality impact is applied only in years where salmon index is less than 1 (IFprey_pcb).*

*Table 20 - PVA simulation results for each PCB threat scenario, including the population growth rate (stoch-r), standard deviation of r (SDr), probability of extinction (PE), and the estimated number of living animals (N-extant) and the standard deviation (SD Next).*

| Model scenario | stoch-r | SD(r) | PE | N-extant | SD(Next) |
|---|---|---|---|---|---|
| SRKW_pcb | 0.034 | 0.084 | 0.000 | 173.3 | 53.1 |
| SRKW_pcb_2mg | 0.038 | 0.087 | 0.000 | 167.4 | 49.2 |
| SRKW_pcb_3 | 0.025 | 0.083 | 0.000 | 145.7 | 46.3 |
| SRKW_pcb_1mg | 0.038 | 0.086 | 0.000 | 188.3 | 57.7 |
| SRKW_prey-pcb | 0.008 | 0.080 | 0.000 | 103.9 | 34.1 |
| SRKW_IFprey-pcb | 0.012 | 0.080 | 0.000 | 112.0 | 35.8 |
| NRKW_pcb_specific | 0.037 | 0.079 | 0.000 | 413.9 | 91.3 |
| NRKW_pcb_2mg | 0.037 | 0.080 | 0.000 | 361.6 | 58.4 |
| NRKW_pcb_3 | 0.038 | 0.081 | 0.000 | 364.2 | 56.7 |
| NRKW_pcb_specific_1mg | 0.038 | 0.080 | 0.000 | 420.2 | 88.9 |
| NRKW_prey-pcb | 0.023 | 0.081 | 0.000 | 341.0 | 96.4 |
| NRKW_IFprey_pcb | 0.024 | 0.080 | 0.000 | 342.5 | 96.9 |

## 4.7 CUMULATIVE EFFECTS

The cumulative effects model with all threats represented (prey abundance, PCBs, vessel noise/presence and vessel strikes) is closer to the observed population size than any of the single threat models alone (Figure 23). The cumulative model approaches the realised population growth for both populations closely, but particularly closely in the NRKW population (Figure 24; Table 21). The standard deviations for this model run encompass the observed population growth for NRKW. The mean model NRKW population size in 2017 was 309 (± 76 SD), the recorded NRKW population in 2017 was 308. The average model SRKW population size in 2017 was 134 (± 41), the recorded SRKW population in 2017 was 77.

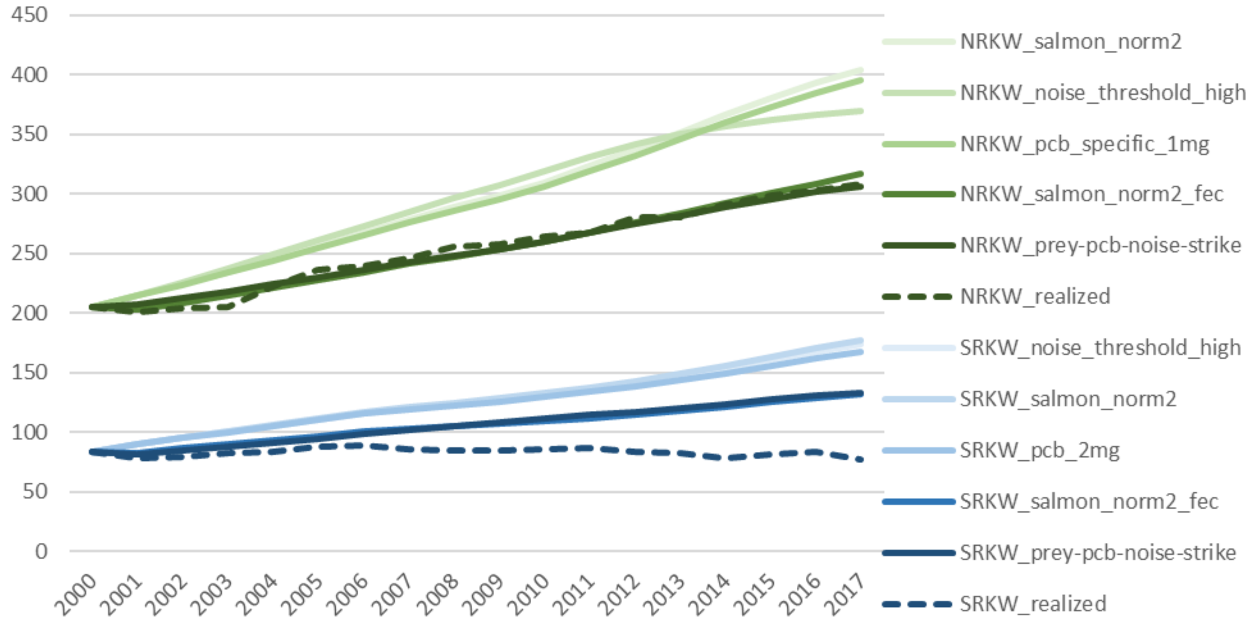

*Figure 22 - Mean model simulations of single threat scenarios (prey abundance, vessel noise/presence, PCBs) and the cumulative effects model scenario (prey-pcb-noise-strike) on NRKW (green lines) and SRKW (blue lines), with realised population size (dashed lines). Scenario names norm2: normal distribution salmon index, norm2_fec: adds a fecundity impact.*

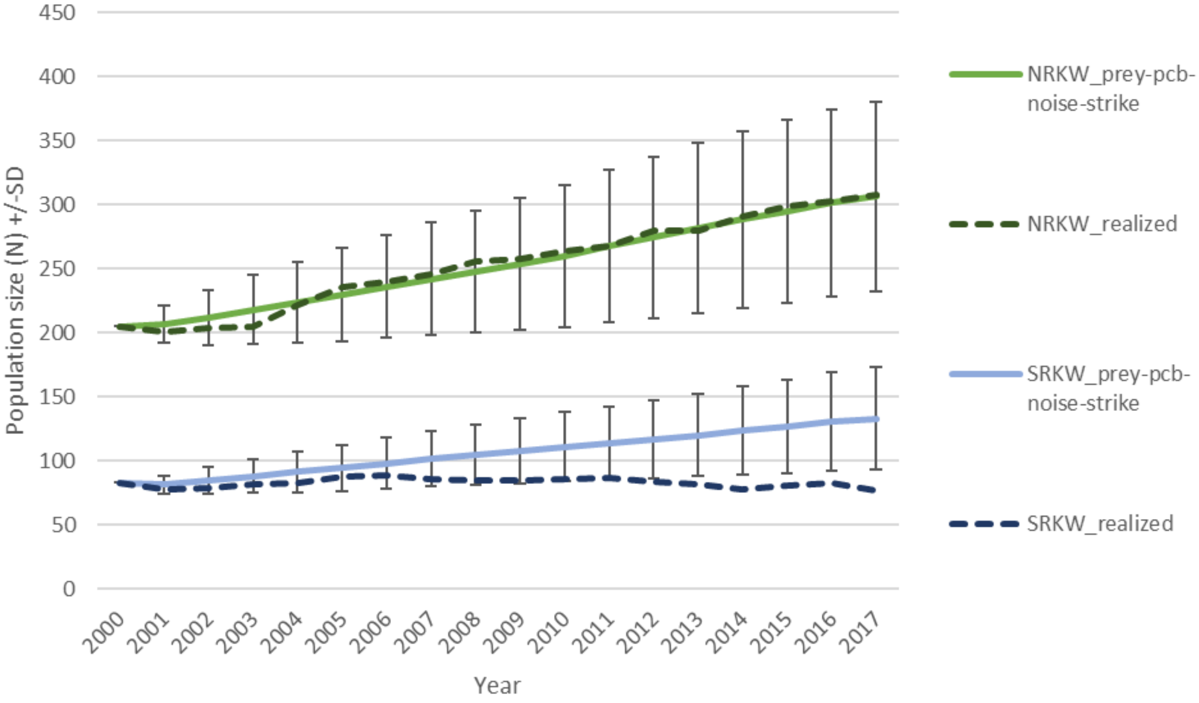

*Figure 23 - Mean model simulations of the cumulative effects scenario (prey-pcb-noise-strike) on NRKW (green lines) and SRKW (blue lines), with realised population size (dashed lines). Error bars represent the ±1 standard deviation.*

*Table 21 - PVA simulation results for the cumulative threat scenario, including the mean population growth rate (stoch-r), standard deviation of r (SDr), probability of extinction (PE), and the mean estimated number of living animals (N-extant) and the standard deviation (SD Next).*

| **Model scenario** | **stoch-r** | **SD(r)** | **PE** | **N-extant** | **SD($N_{ext}$)** |
|---|---|---|---|---|---|
| SRKW_prey-pcb-noise-strike | 0.024 | 0.083 | 0.000 | 134.4 | 41.4 |
| NRKW_prey-pcb-noise-strike | 0.023 | 0.079 | 0.000 | 309.5 | 75.7 |

## 4.8 MODEL VERIFICATION AND VALIDATION

### 4.8.1 Inspection

Using the historical (rather than drawn randomly from a distribution) Chinook index values for 2000-2017, resulted in the cumulative effects model approaching the observed population growth even more closely, especially for SRKW (Figure 25; Table 22). The cumulative effects model explains the realised population growth better than any single threat model alone.

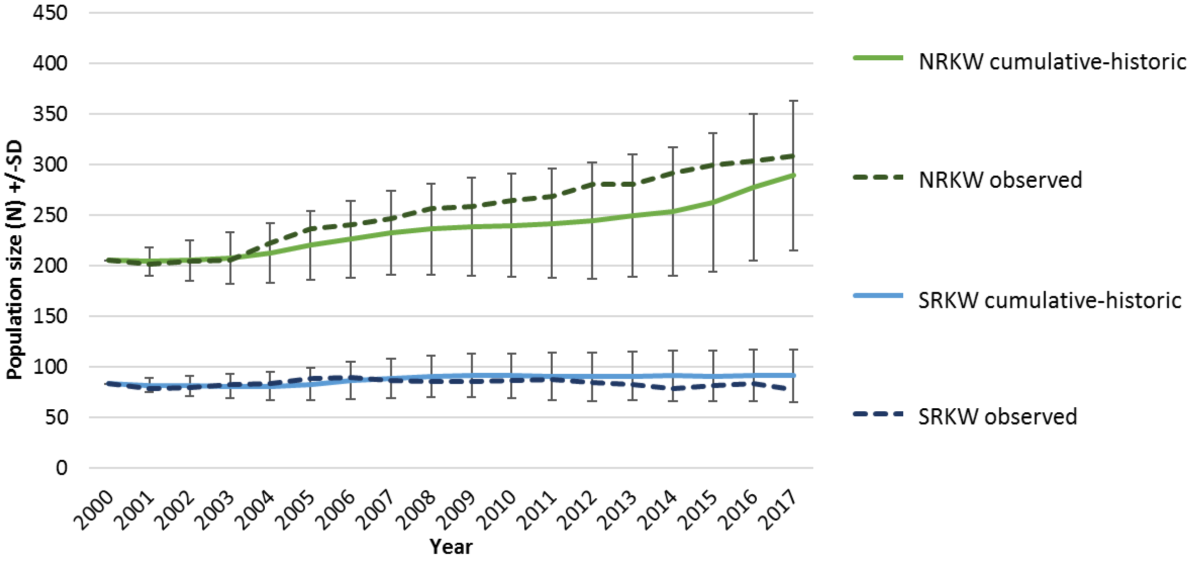

*Figure 24 - Mean model simulations of the cumulative threat scenario with the historical Chinook index values (cumulative-historic), and realised population size (dashed line) for NRKW (green) and SRKW (blue). Error bars represent* ±*1 standard deviation.*

*Table 22 - PVA simulation results for the cumulative threat scenario, using historical Chinook salmon index values, including the mean population growth rate (stoch-r), standard deviation of r (SDr), probability of extinction (PE), and the mean estimated number of living animals (N-extant) and the standard deviation (SD $N_{ext}$).*

| **Model scenario** | **stoch-r** | **SD(r)** | **PE** | **N-extant** | **SD($N_{ext}$)** |
|---|---|---|---|---|---|
| SRKW cumulative-historic | 0.002 | 0.083 | 0.000 | 91.1 | 29.2 |
| NRKW cumulative-historic | 0.019 | 0.079 | 0.000 | 296.6 | 75.3 |

### 4.8.2 Population Structure

To further validate the model, we compared the observed and simulated population structure for the cumulative effects model (prey-pcb-noise-strike). The relative proportions of juveniles and adults were similar, as were the sex ratios for both SRKW and NRKW (Table 23). The NRKW model was extremely close to the observed values in its outputs (Modeled: 102 juveniles and 207 adults; Observed: 104 juveniles and 204 adults). The sex ratios were also similar between the cumulative effects model outputs and the observed, both predicting more females than males. For SRKW the male to female ratio was modeled to be 0.813 and the observed was 0.949, more females than males. For NRKW, the modeled sex ratio was 0.866 and the observed was 0.610. A large proportion of the NRKW population has not been identified to sex, likely because of the number of juveniles and because its members are not surveyed as often as the SRKW.

*Table 23 - Observed and modeled demographic structure for the two populations (SRKW and NRKW).*

| | | **Observed** | | | | **Modeled** | | |
|---|---|---|---|---|---|---|---|---|
| **SRKW** | **Age class** | **Female** | **Male** | **Unknown** | **Total** | **Female** | **Male** | **Total** |
| | 0-9 | 7 | 13 | 0 | **20** | 22 | 22 | **44** |
| | 10+ | 32 | 24 | 0 | **56** | 52 | 39 | **91** |
| | **Total** | **39** | **37** | **0** | **77** | **74** | **61** | **135** |
| **NRKW** | 0-9 | 4 | 3 | 97 | **104** | 51 | 51 | **102** |
| | 10+ | 102 | 62 | 40 | **204** | 114 | 93 | **207** |
| | **Total** | **105** | **64** | **136** | **308** | **165** | **144** | **309** |

### 4.8.3 Projection

Model scenarios can be projected into the future to examine the long-term population growth rate and future of the populations. The projection of the cumulative effects model assumes that the current levels of threats continue into the future, with no changes in threats and no mitigation actions. When Chinook salmon abundance is taken from the long-term mean abundances (1979-2017) , the cumulative model (prey-pcb-noise-strike) projects mean positive population growth for both populations, but with uncertainty that includes negative population growth: 1.6% (±7.9 SD) for NRKW and 1.5% (±8.1 SD) for SRKW (Figure 26; Table 24). NRKW reaches the arbitrarily-set carrying capacity early in the projections and this affects the projected population growth rate. The probability of extinction (defined in the model as a single sex remaining) for both populations is zero over 100 years. Note that under SARA, extinction is defined as no individuals remaining. In contrast, when the cumulative effects model uses the recent (2008-2017) distribution of Chinook salmon abundance indices the projection is negative population growth for SRKW (-2.5%), and a slightly lowered, but still positive, growth rate for NRKW (Figure 26, Table 24). Under the recent prey scenario, SRKW have a 26.1% probability of extinction and in those simulations where extinction occurred, the mean time to extinction was 86 years (±11.3 years).

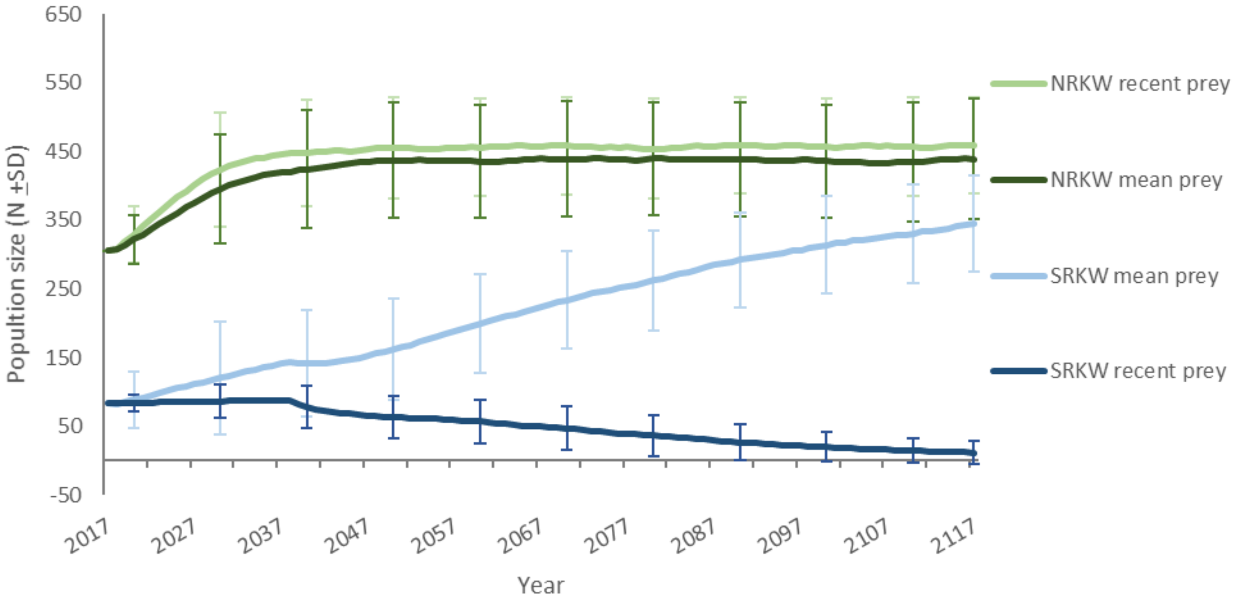

*Figure 25 - Mean projection of cumulative effects model 100 years into the future (starting in 2017) for NRKW (green) and SRKW (blue), under mean Chinook index ("mean prey": 1979-2017) or recent Chinook index ("recent prey": 2008-2017). Error bars represent +1 standard deviation.*

*Table 24 - Population growth rate, population size at year 100 ($N_{100}$) and probability of extinction of cumulative effects model projection 100 years into the future, under two scenarios of Chinook abundance: mean prey (1979-2017) or recent prey (2008-2017).*

| | **Projection Scenario** | |
|---|---|---|
| **Summary Statistics** | **Mean prey (Chinook index distribution: 1979-2017)** | **Recent prey (Chinook index distribution: 2008-2017)** |
| Population growth rate | + 1.5% | - 2.5% |
| $N_{100}$ | 344.7 (± 140) years | 15.1 (±17) years |
| Probability of extinction | 0 | 26.1% |

### 4.8.4 Sensitivity

The sensitivity of model parameters was tested to distinguish which parameters have the highest impact on long-term population dynamics. The cumulative effects scenario model projection for SRKW was used in sensitivity testing, which includes all four threats and their interactions as defined in Section 4.7. Single factor sensitivity testing was performed where the parameter of interest was varied across its range (minimum-maximum), with the base values used for all other parameters, using the full individual-based stochastic model. The base threat levels were the original values used in the cumulative effects model. Sensitivity testing for that parameter varied across the entire distribution (minimum-maximum) by set increments (Table 25). For the prey parameter sensitivity testing, the full range of Chinook index values for both stocks was tested (minimum = 0.4, maximum = 1.8). The vessel noise/presence parameter begins at the base level of noise – the value set to 0.85 has no reduction in feeding rate and increases to a maximum of 1.55, to represent the possibility that the reduction in feeding time could be higher or that the presence spent near vessels was higher than estimated. The PCB value tested included the base initial PCB concentration for females, and included the full range of measured female PCB concentrations. Male PCB concentration was not used in sensitivity testing because the impact pathway occurs via maternal transfer. Strike risk was varied from 5% to as high as 50%. The most sensitive parameter for the long-term projection of the population was prey availability (the value of the Chinook index), followed by vessel noise/presence (Figure 27).

*Table 25 - Parameters and values used for testing sensitivity in the SRKW cumulative effects (prey-pcb-noise-strike) scenario projection, including the base value, the range and increment of testing.*

| | **Base** | **Minimum** | **Maximum** | **Increment** |
|---|---|---|---|---|
| Prey (Chinook Index) | 1.00 | 0.40 | 1.80 | 0.10 |
| Noise | 0.85 | 0.85 | 1.55 | 0.10 |
| PCB-female | 17.46 | 5.00 | 200.00 | 25.00 |
| Strike risk | 0.10 | 0.05 | 0.50 | 0.05 |

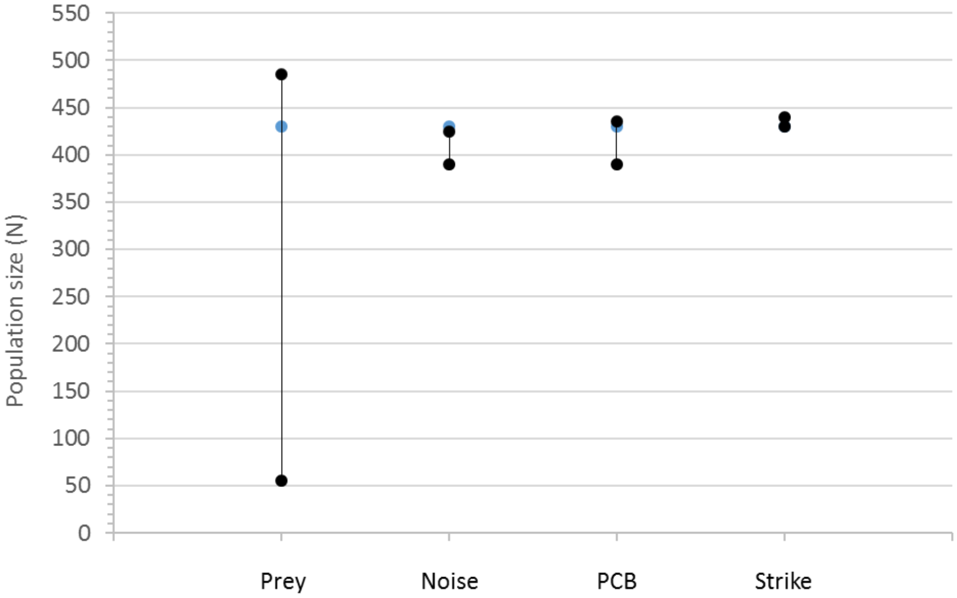

*Figure 26 - Sensitivity of SRKW projected population size in the cumulative effects scenario to changes in the threat parameters: prey abundance, vessel noise/presence, PCB concentration and strike risk. Blue circles represent the base value for each threat and the vertical bars represent the range of population size (N) for each change in threat value (Table 25).*

# 5 DISCUSSION

## 5.1 ASSESSING CUMULATIVE EFFECTS

This is the first cumulative effects assessment that includes a pathways of effects conceptual model and a population viability analysis incorporating the impacts of all the SARA priority threats (prey availability, disturbance and contaminants) on NRKW and SRKW. It is of considerable interest to investigate why these populations with similar life histories and diet exhibit such different population trajectories: the SRKW population is in decline while the NRKW population is increasing (Figure 2). The inclusion of both populations in the same assessment allows for comparisons to be made within the same set of models and parameters (assuming that the mechanisms of impact are similar, while threat exposure levels differ). The systematic assessment of both individual and combined threats in the model scenarios allows examination of which threats (or combination of threats) best explain the observed population growth and in turn may have a greater influence on the population trajectories and demographics of these killer whale populations.

This work relied heavily on the existing body of literature and data and built upon previous work and threat characterisations, including previous assessments of Resident Killer Whale populations (Lacy et al. 2017; Vélez-Espino et al. 2014b; Taylor and Plater 2001). We incorporated new and updated information and data for all threats and included potential interactions between threats. It is anticipated that this species-focused cumulative effects assessment method will be useful as a tool for managers and scientists for killer whales, as well as potentially for other species.

The first phase of the cumulative effects assessment provided an evidence-based pathways of effects conceptual model, creating the foundation for the assessment. Explicitly outlining the background and current state of knowledge of priority threats, interactions and impacts, and in particular the uncertainties and limitations, is valuable for understanding and having confidence that the model outputs represent the system of interest.

The second phase constructed a population model for each population and systematically tested the effects of individual and cumulative effects on population growth by comparing the model outputs to the observed population sizes from 2000-2017. An important change from previous PVAs is that the current model uses the observed vital rates from the relatively unimpacted Southern Alaska Resident Killer Whale population as the reference state, and adds threats to the model as modifiers of these rates. This allowed us to test the validity of the model scenarios in order to identify the model that most closely matches the observed data and assess which of the priority threats most contribute to cumulative effects. This is a different approach than that used by Lacy et al. (2017) for SRKW, where the “baseline” was defined with the mean demographic rates that were observed across recent decades (i.e., including current threats to the population), and then varied threat levels to investigate the impact on population performance. The Lacy et al (2017) baseline tests scenarios of varying threat levels, rather than explaining current trends or patterns in the historical data.

The definition of the model scenarios incorporated updated and/or new inputs for every threat in the assessment:

- Prey abundance - Updated Chinook salmon ocean abundance model data, extending the time series to 2017 from that used in previous studies (2008: Lacy et al. 2017, and 2011: Vélez-Espino et al. 2014b). These data were used in statistical and model selection analyses performed to update the relationships between Chinook salmon indices and mortality and fecundity.

- Vessel noise/presence – The proportional of loss of foraging time from vessel noise was based on a new study (Tollit et al. 2017), and the incorporation of relative vessel density for both populations estimated by the number of ship transits (MCTS-Western data), and the presence of marinas (Clarke Murray et al. 2014).
- Vessel strike – Strike risk was estimated based on collation of reports of presumed strikes.
- Contaminants – Unpublished PCB data summarised from a number of new sources (Guy 2018 unpubl.[5]; Gobas and Ross 2017 unpubl.[6]), extended the number and time range of SRKW samples incorporated in this assessment to 40 samples (collected 1993-2015), compared to the 6 samples (collected 1993 and 1996) included in previous work (Lacy et al. 2017). For the NRKW population, data from 85 PCB samples were available (28 currently available in the published literature).

Despite the updated inputs, the individual threat models did not closely align with the observed population dynamics. However, the cumulative threats model, incorporating all priority threats (Chinook salmon abundance, vessel noise/presence, vessel strike, and PCB contamination, predicted population growth closest to the observed rates for both populations, out of all individual and combined threat models tested. The cumulative effects model scenario results matched the observed data more closely for NRKW than for SRKW (Figure 24).

The cumulative threats model slightly over-estimates growth for the SRKW population. There are a number of factors that could act singly or in concert to explain this. The individual-based simulation PVA includes stochasticity in the model simulations. The Chinook salmon index assigned annually was drawn randomly from a distribution and this index value affects mortality, fecundity, and interactions with other threats. When the historical Chinook index values were used instead of the random values, the SRKW model very closely aligned with the observed RKW data which fell within the model's standard deviations for both populations (Figure 25). This suggests that the cumulative model is a valid representation of the system but the temporal range in question (2000-2017) included a number of years with Chinook availability below the long-term mean (1979-2017). Essentially, the observed data were closely aligned with the subset of model simulations where the randomly drawn Chinook values were lower than average. The development and use of a Chinook salmon model that could better represent the amount of temporal autocorrelation could be used in the model to produce better projections. There is a coastwide Chinook model by the Pacific Salmon Commission that is produces four year projections, but it will need modifications to project further into the future.

This assessment highlights the importance of considering threats collectively. Specifically, within the cumulative effects PVA assessment, Chinook salmon abundance and its interactions with vessel noise/presence and PCBs strongly influenced modelled killer whale population dynamics. The cumulative effects PVA model can be used to compare population trajectories into the future for NRKW and SKRW. These projections are best used in a comparative evaluation, rather than absolute predictions of abundance. The projected population growth was highly sensitive to the Chinook salmon abundance index. Under long-term mean Chinook abundances, the modeled SRKW population was projected to increase and when the recent Chinook salmon index values were used to set the model parameters, the average model SRKW population trajectory declined, with a chance of extinction. Model projections are based on an assumption that modeled threat conditions continue at the same levels. A continuation of the decline in the Chinook salmon stocks that SRKW depend upon can affect the future outlook of the population, and potentially increase the probability of extinction.

## 5.2 COMPARING INDIVIDUAL THREATS

Although the best fitting model was the cumulative impacts model, the single threat model for prey availability with both mortality and fecundity impacts did approach the observed data, indicating that variations in prey are an important contributor to predicting demographic rates in these populations. The sensitivity analysis demonstrated that prey availability is the most sensitive parameter for projecting cumulative effects, having a large impact on the population projections into the future. In the cumulative effects model, prey availability was the most important threat for these populations followed by vessel noise/presence.

The findings of this cumulative effects assessment strongly support the significant role of prey availability in determining the population trajectory of these populations, and are consistent with previous work (Lacy et al. 2017, Vélez-Espino et al. 2014b; Ford et al. 2009; 2010; Ward et al. 2009). The updated statistical analyses for the effect of prey availability on mortality and fecundity suggests that these impacts are still important to RKW, but the power of the single threat models have been reduced compared to previous studies. Ford et al. (2009) found that the coastwide Chinook index represented most of the variation in mortality index ($r^2 = 0.77$), while the updated analyses presented here showed that prey alone does not explain as much of the variation in mortality ($r^2 = 0.09$). This provides additional evidence that the cumulative threat model is a better representation of the current system.

The enduring effect of aquarium removals is evident in the SRKW population, as many more individuals were removed from the SRKW population than the NRKW. The SRKW population size would likely have been much higher in the present day if the large proportion of individuals had not been removed. The PVA model provided an opportunity to examine what may have happened to the SRKW population trajectory had these individuals remained in the population. The removals scenario results indicated that though the population growth rate may have been unchanged by the higher population numbers, SRKW population size likely would have stabilised at around 140 individuals ($\pm$26 standard deviation), making for a more resilient and genetically diverse population.

## 5.3 ASSUMPTIONS AND UNCERTAINTIES

An important assumption made in this work is that the pathways of effects from threats to impacts are the same for both SRKW and NRKW, in other words, that the mechanisms by which threats affect individuals are the same for both populations. This assumption is the justification for utilising the same impact model structure for both populations, with differing threat levels. The consequence of exposure to threats is assumed to be the same while the levels of exposure to threats is assumed to be population-specific. Sub-population level variation (pods/clans) may affect the exposure to threats and were not captured in the current assessment. Knowledge about the relationships between threats and Resident Killer Whale mortality and fecundity were based on information mostly obtained in the Salish Sea area in the summer/fall period but was assumed to represent threat conditions throughout the range and throughout the year.

Further, the two populations may exploit different prey stocks that themselves have varying population growth and availability to killer whale predation. All Chinook salmon stocks went through a period of decline in the 1990s but since then have experienced differing temporal variation (Figure 6). The Chinook stocks linked to SRKW (WCVI + FL + OC) had a higher mean over the time series than the stocks linked to NRKW (FE + PS + URB), suggesting that NRKW would have less prey available if they are limited in prey choice. The ability and flexibility of these populations to exploit different stocks is unknown and may vary between NRKW and SRKW. Potential competition between populations and with other marine mammals for prey may also affect prey availability and have not been included in the current models. Additionally, the proximity of SRKW to major population centres likely means greater exposure to sources of contamination and vessel traffic.

The projection of positive population growth under mean prey abundance with the cumulative effects model assumes that the current levels of threats remain the same and do not increase, which may not be the case in reality. Changing climate conditions and an increasing human population are having significant ongoing impacts on the marine ecosystem and are likely to continue to affect killer whales and their prey into the future. Reductions in threats may also affect the population trajectories, such as through mitigation and management actions. The US and Canada have taken a number of management actions in recent years to support the recovery of the SRKW population. Incorporating the effect of management actions, changing natural conditions and changes to threats into iterations of the cumulative effects assessment may provide useful insight into the potential effects on population trajectory.

It is cautioned that positive population growth is in no way assured by the results of this modelling exercise. The model scenarios produce mean population growth rate projections with uncertainty bounds around them. Uncertainty is addressed with stochasticity, including error rates explicitly in the model parameters. Consequently, the standard deviations for both populations include negative population growth rates, and was demonstrated by including the historical Chinook index values, which causes the SRKW population to exhibit negative growth. Therefore, if the Chinook index remains under the long-term mean in the future population growth would be expected to be negative.

The extinction probabilities and population projections of the model are only accurate if all relevant threats have been included and adequately parameterised. The representation and parameterisation of each threat in the model has associated uncertainties or limitations. Acoustic disturbance is the threat for which the least information was available. The data collected on the effects of vessels on killer whale behaviour are based on limited study time period and duration, typically from one season and/or one location (Tollit et al. 2017; Lusseau et al. 2009; Williams et al. 2002a,b). The presence and effects of vessels outside the summer season and in areas other than the Salish Sea are unknown. All vessels have been treated equally in the current treatment of vessel noise/presence and the size, acoustic signature, spatial and temporal distribution will affect the impacts on Resident Killer Whales. At present, there are no data to support clear distinctions in impacts from vessel presence and vessel noise. The effect of vessel presence may be similar to that of predator presence but little evidence exists for how to represent this component of the threat. There may be an interaction between vessel-related noise disturbance and increased incidences of other mortality events, from acoustic trauma from seismic testing or other more significant sounds, such as military munitions testing. This linkage pathway is difficult to quantify due to limited retrieval of dead killer whales and the secrecy of the spatial and temporal location of some of these disturbance events (DFO 2018a). The impact of echosounders on small vessels in close proximity to whales may also affect killer whales. Echosounder noise can be heard on Dtag deployments and the impacts are currently unknown (B. Wright, DFO, pers. comm.).

The representation of the contaminants threat also had limitations and uncertainties. From the suite of contaminants of concern for killer whales, only PCBs were able to be incorporated because this contaminant has been measured over time in this population and there is a model linking it to calf mortality (Hall et al. 2018). There is evidence that PCB contamination may affect reproductive development and disease susceptibility (Hall et al 2018), but these impacts were not able to be sufficiently quantified for use in the models. Killer whales also have significant concentrations of PBDEs but it was not possible to include this contaminant in the model due to a lack of evidence linking it to killer whale vital rates. The concentration of PCBs in killer whale tissue was incorporated into the PVA threat model based on a model for PCB accumulation and depuration linking declines in calf survival with increased maternal PCB concentration (Hall et al. 2018). The original model was developed based on a dose-response curve extended from laboratory responses in mink, the primary concern is the uncertainty associated with converting a physiological response from a small terrestrial mammal to much larger wild killer whale population (Witting et al. 2018).

Low probability, high consequence threats, such as oil spills or disease outbreaks, are difficult to include in simulation modelling. These threats should not be ignored in management and mitigation because they can have catastrophic consequences if the population were to be exposed. The Exxon Valdez oil spill in Alaska was linked to significant declines in the resident pods that were exposed to oil, suffering significant losses in the year following the spill (Matkin et al. 1999; Matkin et al. 2008). These losses had pod-level impacts and the affected pods had not recovered to pre-spill levels 16 years after the event. One way to address high consequence events, such as a large oil spill or disease epidemic, in model simulations could be to dramatically reduce the population to 50-75% of the current levels and test if the model population would be resilient enough to recover from such a catastrophe.

## 5.4 CONCLUSIONS

This cumulative effects assessment further advances the field by combining a detailed Pathways of Effects conceptual model and a specific Population Viability Analysis simulation model (after Lacy et al. 2017) to evaluate how the current state of human activities affects the future persistence of the two populations. The cumulative effects population viability analysis model we have developed could be used in a number of ways by managers and scientists. One important way it could be used is to determine which threat has the most impact on long-term population persistence for a particular population, and could be extended to other killer whale ecotypes and populations. Another valuable use for the model is to test the impacts of different theoretical mitigation and management scenarios for individual threats on the population trajectory, for example to test whether the complete mitigation of acoustic disturbance would cause the population trajectory to increase over time and how long it may take for a change in population trajectory to be observable. The cumulative effects PVA model can be run using different input parameters (e.g., increased shipping) in order to consider the potential impacts of proposed developments and other anthropogenic changes in the Southern and Northern RKW’s range. The ongoing research being conducted under initiatives such as Oceans Protection Plan and Whales Initiative, and others, can be used to refine the model and test possible mitigation and management actions and the impact on the long-term survival and recovery of the threatened and endangered Northern and Southern Resident Killer Whale populations. Information from ongoing and/or planned further research on RKW such as prey competition in key foraging areas, foraging efficiency, RKW diet composition, prey field analysis, underwater acoustic monitoring and modelling, contaminant sources and levels, will all help to inform future iterations of the model. Model use, and refined future versions, can help to adaptively inform and/or implement RKW recovery measures identified in the *Action Plan for the Northern and Southern Resident Killer Whale* (Orcinus orca) *in Canada*, such as investigating the benefits of management actions to protect important areas, evaluating potential impacts of disturbance and prey competition from fisheries, assessing the potential impact of salmon enhancement on RKW, and assessing project impacts on RKW and their habitat to provide advice on avoidance and mitigation measures as required.

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# APPENDIX I: CHINOOK MODEL OCEAN ABUNDANCE AND INDEX VALUES

*Appendix Table 1 - The model Chinook ocean abundance values for five stock indices, and their corresponding index values (1979-2017) with the summary statistics (mean, standard deviation, minimum and maximum values)*

| | **Model Chinook Ocean Abundance** | | | | | **Chinook Index** | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Year** | **Total** | **Total (excl. SEAK)** | **FE+PS+ URB** | **WCVI+FL+ OC** | **WCVI** | **Coastwide Index Total (excl. SEAK)** | **NRKW Index (FE+PS+ URB)** | **SRKW Index (WCVI+FL+ OC)** | **WCVI Index** |
| 1979 | 1,336,622 | 1,328,288 | 259,998 | 439,869 | 60,945 | 1.199 | 0.996 | 1.179 | 0.536 |
| 1980 | 1,203,711 | 1,194,004 | 223,034 | 418,398 | 101,538 | 1.080 | 0.854 | 1.121 | 0.892 |
| 1981 | 1,169,582 | 1,158,360 | 189,635 | 440,400 | 95,855 | 1.049 | 0.726 | 1.180 | 0.842 |
| 1982 | 1,232,148 | 1,221,029 | 180,362 | 508,465 | 171,707 | 1.106 | 0.691 | 1.363 | 1.509 |
| 1983 | 1,188,448 | 1,174,663 | 211,145 | 544,413 | 182,846 | 1.066 | 0.809 | 1.459 | 1.607 |
| 1984 | 1,323,238 | 1,307,815 | 278,484 | 590,565 | 151,996 | 1.187 | 1.067 | 1.583 | 1.336 |
| 1985 | 1,215,562 | 1,199,250 | 289,653 | 502,120 | 88,084 | 1.091 | 1.110 | 1.346 | 0.774 |
| 1986 | 1,278,693 | 1,260,447 | 335,530 | 442,340 | 66,658 | 1.147 | 1.285 | 1.185 | 0.586 |
| 1987 | 1,553,371 | 1,534,810 | 381,238 | 382,529 | 60,717 | 1.394 | 1.460 | 1.025 | 0.534 |
| 1988 | 1,483,370 | 1,468,110 | 376,360 | 385,109 | 109,780 | 1.331 | 1.442 | 1.032 | 0.965 |
| 1989 | 1,300,421 | 1,290,017 | 307,754 | 455,769 | 140,389 | 1.167 | 1.179 | 1.221 | 1.234 |
| 1990 | 1,249,674 | 1,241,650 | 263,908 | 546,608 | 213,518 | 1.121 | 1.011 | 1.465 | 1.877 |
| 1991 | 1,193,851 | 1,185,273 | 174,244 | 575,046 | 269,064 | 1.071 | 0.667 | 1.541 | 2.365 |

| | **Model Chinook Ocean Abundance** | | | | | **Chinook Index** | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Year** | **Total** | **Total (excl. SEAK)** | **FE+PS+ URB** | **WCVI+FL+ OC** | **WCVI** | **Coastwide Index Total (excl. SEAK)** | **NRKW Index (FE+PS+ URB)** | **SRKW Index (WCVI+FL+ OC)** | **WCVI Index** |
| 1992 | 1,169,643 | 1,160,515 | 152,430 | 601,057 | 270,790 | 1.050 | 0.584 | 1.611 | 2.380 |
| 1993 | 1,059,739 | 1,049,669 | 182,438 | 535,455 | 245,199 | 0.951 | 0.699 | 1.435 | 2.155 |
| 1994 | 862,123 | 853,603 | 187,404 | 377,408 | 167,428 | 0.774 | 0.718 | 1.011 | 1.472 |
| 1995 | 677,078 | 669,434 | 160,574 | 223,197 | 71,120 | 0.608 | 0.615 | 0.598 | 0.625 |
| 1996 | 732,393 | 724,537 | 178,309 | 229,071 | 47,080 | 0.657 | 0.683 | 0.614 | 0.414 |
| 1997 | 870,410 | 862,521 | 195,083 | 367,002 | 105,969 | 0.781 | 0.747 | 0.984 | 0.931 |
| 1998 | 820,971 | 812,464 | 171,553 | 372,964 | 129,090 | 0.737 | 0.657 | 0.999 | 1.135 |
| 1999 | 764,740 | 754,682 | 211,522 | 244,700 | 61,718 | 0.686 | 0.810 | 0.656 | 0.542 |
| 2000 | 737,322 | 725,553 | 195,232 | 212,933 | 25,496 | 0.662 | 0.748 | 0.571 | 0.224 |
| 2001 | 1,072,818 | 1,060,708 | 242,166 | 294,405 | 46,236 | 0.963 | 0.928 | 0.789 | 0.406 |
| 2002 | 1,559,654 | 1,549,335 | 314,406 | 477,966 | 116,719 | 1.399 | 1.204 | 1.281 | 1.026 |
| 2003 | 1,610,659 | 1,601,251 | 359,540 | 530,834 | 153,526 | 1.445 | 1.377 | 1.423 | 1.349 |
| 2004 | 1,434,584 | 1,424,749 | 310,027 | 451,426 | 150,160 | 1.287 | 1.188 | 1.210 | 1.320 |
| 2005 | 1,176,463 | 1,165,783 | 310,805 | 333,769 | 111,600 | 1.056 | 1.191 | 0.894 | 0.981 |
| 2006 | 961,736 | 949,908 | 262,978 | 295,609 | 112,908 | 0.863 | 1.007 | 0.792 | 0.992 |
| 2007 | 746,441 | 734,905 | 205,288 | 227,601 | 100,940 | 0.670 | 0.786 | 0.610 | 0.887 |

| | **Model Chinook Ocean Abundance** | | | | | **Chinook Index** | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Year** | **Total** | **Total (excl. SEAK)** | **FE+PS+ URB** | **WCVI+FL+ OC** | **WCVI** | **Coastwide Index Total (excl. SEAK)** | **NRKW Index (FE+PS+ URB)** | **SRKW Index (WCVI+FL+ OC)** | **WCVI Index** |
| 2008 | 745,175 | 738,184 | 199,959 | 174,200 | 55,353 | 0.669 | 0.766 | 0.467 | 0.487 |
| 2009 | 768,658 | 761,879 | 228,120 | 174,788 | 44,701 | 0.690 | 0.874 | 0.468 | 0.393 |
| 2010 | 1,029,980 | 1,022,934 | 245,614 | 293,418 | 65,508 | 0.924 | 0.941 | 0.786 | 0.576 |
| 2011 | 1,053,968 | 1,048,424 | 279,302 | 304,161 | 104,360 | 0.946 | 1.070 | 0.815 | 0.917 |
| 2012 | 901,835 | 897,959 | 243,656 | 214,059 | 70,964 | 0.809 | 0.933 | 0.574 | 0.624 |
| 2013 | 1,210,298 | 1,205,330 | 395,354 | 278,833 | 69,049 | 1.086 | 1.514 | 0.747 | 0.607 |
| 2014 | 1,484,397 | 1,479,438 | 474,485 | 330,696 | 90,298 | 1.332 | 1.818 | 0.886 | 0.794 |
| 2015 | 1,324,091 | 1,319,306 | 392,910 | 267,736 | 82,484 | 1.188 | 1.505 | 0.718 | 0.725 |
| 2016 | 1,033,825 | 1,030,649 | 342,221 | 259,093 | 113,298 | 0.928 | 1.311 | 0.694 | 0.996 |
| 2017 | 925,453 | 923,050 | 268,321 | 248,889 | 112,249 | 0.830 | 1.028 | 0.667 | 0.987 |
| **mean** | **1,114,440** | **1,104,884** | **261,052** | **373,151** | **113,778** | **1.000** | **1.000** | **1.000** | **1.000** |
| **SD** | **262,628** | **261,397** | **78,367** | **126,860** | **60,441** | **0.236** | **0.300** | **0.340** | **0.531** |
| **minimum** | **677,078** | **669,434** | **152,430** | **174,200** | **25,496** | **0.608** | **0.584** | **0.467** | **0.224** |
| **maximum** | **1,610,659** | **1,601,251** | **474,485** | **601,057** | **270,790** | **1.445** | **1.818** | **1.611** | **2.380** |

# APPENDIX II: MEASURED PCB CONCENTRATIONS FOR NRKW AND SRKW

*Appendix Table 2 - Summary of total polychlorinated biphenyls (ΣPCB) concentrations measured in Northern Resident Killer Whale blubber from 1993-2015 (adapted from Guy, 2018 unpubl.[5] - Appendix E, Table E1). Source of PCB data Ross et al. 2013 unpubl.[9]*

| Source | Animal ID | Sex | Age | Year | # congeners analysed | Lipid (%) | ΣPCB (mg·kg-1lw) | ΣPCB mg/kg |
|---|---|---|---|---|---|---|---|---|
| Ross et al. 2013 unpubl | A56 | F | 3 | 1993 | 205 | 64.3 | 9.80E+00 | 9.80 |
| Ross et al. 2013 unpubl | A54 | F | 4 | 1993 | 205 | 64.3 | 9.96E+00 | 9.96 |
| Ross et al. 2013 unpubl | A52 | F | 6 | 1993 | 205 | 64.3 | 3.39E+01 | 33.90 |
| Ross et al. 2013 unpubl | A48 | F | 10 | 1993 | 205 | 64.3 | 1.07E+01 | 10.70 |
| Ross et al. 2013 unpubl | A43 | F | 12 | 1993 | 205 | 64.3 | 7.41E+00 | 7.41 |
| Ross et al. 2013 unpubl | A35 | F | 19 | 1993 | 205 | 64.3 | 1.68E+00 | 1.68 |
| Ross et al. 2013 unpubl | A24 | F | 26 | 1993 | 205 | 64.3 | 4.79E-01 | 0.48 |
| Ross et al. 2013 unpubl | A11 | F | 35 | 1993 | 205 | 64.3 | 1.04E+00 | 1.04 |
| Ross et al. 2013 unpubl | A23 | F | 46 | 1993 | 205 | 64.3 | 2.58E+00 | 2.58 |
| Ross et al. 2013 unpubl | A9 | F | 53 | 1993 | 205 | 64.3 | 2.40E+01 | 24.00 |
| Ross et al. 2013 unpubl | A60 | M | 1 | 1993 | 205 | 64.3 | 1.31E+01 | 13.10 |
| Ross et al. 2013 unpubl | A59 | M | 1 | 1993 | 205 | 64.3 | 1.08E+01 | 10.80 |
| Ross et al. 2013 unpubl | A13 | M | 15 | 1993 | 205 | 64.3 | 2.12E+01 | 21.20 |
| Ross et al. 2013 unpubl | A27 | M | 22 | 1993 | 205 | 64.3 | 2.42E+00 | 2.42 |
| Ross et al. 2013 unpubl | A6 | M | 29 | 1993 | 205 | 64.3 | 1.79E+01 | 17.90 |
| Ross et al. 2013 unpubl | B2 | M | 41 | 1993 | 205 | 64.3 | 2.69E+01 | 26.90 |
| Ross et al. 2013 unpubl | B1 | M | 42 | 1993 | 205 | 64.3 | 6.90E+00 | 6.90 |

| Source | Animal ID | Sex | Age | Year | # congeners analysed | Lipid (%) | ΣPCB (mg·kg-1lw) | ΣPCB mg/kg |
|---|---|---|---|---|---|---|---|---|
| Ross et al. 2013 unpubl | A57 | F | 5 | 1996 | 205 | 64.3 | 1.09E+02 | 109.00 |
| Ross et al. 2013 unpubl | A42 | F | 16 | 1996 | 205 | 64.3 | 1.54E+01 | 15.40 |
| Ross et al. 2013 unpubl | C10 | F | 25 | 1996 | 205 | 64.3 | 6.90E+00 | 6.90 |
| Ross et al. 2013 unpubl | I2 | F | 57 | 1996 | 205 | 64.3 | 9.45E+00 | 9.45 |
| Ross et al. 2013 unpubl | C5 | F | 71 | 1996 | 205 | 64.3 | 2.55E+01 | 25.50 |
| Ross et al. 2013 unpubl | B13 | M | 9 | 1996 | 205 | 64.3 | 2.79E+01 | 27.90 |
| Ross et al. 2013 unpubl | B12 | M | 12 | 1996 | 205 | 64.3 | 2.00E+01 | 20.00 |
| Ross et al. 2013 unpubl | H4 | M | 22 | 1996 | 205 | 64.3 | 2.20E+01 | 22.00 |
| Ross et al. 2013 unpubl | A5 | M | 39 | 1996 | 205 | 64.3 | 3.82E+01 | 38.20 |
| Ross et al. 2013 unpubl | I5 | M | 42 | 1996 | 205 | 64.3 | 3.77E+01 | 37.70 |
| Ross et al. 2013 unpubl | R6 | M | 42 | 1996 | 205 | 64.3 | 4.96E+01 | 49.60 |
| Ross et al. 2013 unpubl | A70 | F | 1 | 2000 | 205 | 64.3 | 4.12E+00 | 4.12 |
| Ross et al. 2013 unpubl | I80 | F | 3 | 2000 | 205 | 64.3 | 1.94E+01 | 19.40 |
| Ross et al. 2013 unpubl | A69 | F | 4 | 2000 | 205 | 64.3 | 1.06E+01 | 10.60 |
| Ross et al. 2013 unpubl | G51 | F | 8 | 2000 | 205 | 64.3 | 1.57E+01 | 15.70 |
| Ross et al. 2013 unpubl | I63 | F | 10 | 2000 | 205 | 64.3 | 1.79E+01 | 17.90 |
| Ross et al. 2013 unpubl | I51 | F | 14 | 2000 | 205 | 64.3 | 7.85E+00 | 7.85 |
| Ross et al. 2013 unpubl | I15 | F | 48 | 2000 | 205 | 64.3 | 1.86E+00 | 1.86 |
| Ross et al. 2013 unpubl | I85 | M | 2 | 2000 | 205 | 64.3 | 5.75E+00 | 5.75 |
| Ross et al. 2013 unpubl | C20 | M | 7 | 2000 | 205 | 64.3 | 5.46E+00 | 5.46 |

| Source | Animal ID | Sex | Age | Year | # congeners analysed | Lipid (%) | ΣPCB (mg·kg-1lw) | ΣPCB mg/kg |
|---|---|---|---|---|---|---|---|---|
| Ross et al. 2013 unpubl | C17 | M | 11 | 2000 | 205 | 64.3 | 6.89E+00 | 6.89 |
| Ross et al. 2013 unpubl | D13 | F | 18 | 2002 | 205 | 64.3 | 3.48E+00 | 3.48 |
| Ross et al. 2013 unpubl | D12 | F | 20 | 2002 | 205 | 64.3 | 1.51E+00 | 1.51 |
| Ross et al. 2013 unpubl | I50 | F | 20 | 2002 | 205 | 64.3 | 3.76E+00 | 3.76 |
| Ross et al. 2013 unpubl | I21 | F | 23 | 2002 | 205 | 64.3 | 2.45E+00 | 2.45 |
| Ross et al. 2013 unpubl | C18 | M | 11 | 2002 | 205 | 64.3 | 6.28E+00 | 6.28 |
| Ross et al. 2013 unpubl | I52 | M | 16 | 2002 | 205 | 64.3 | 8.24E+00 | 8.24 |
| Ross et al. 2013 unpubl | A62 | F | 10 | 2003 | 205 | 64.3 | 3.54E+00 | 3.54 |
| Ross et al. 2013 unpubl | I68 | F | 11 | 2003 | 205 | 64.3 | 7.57E+00 | 7.57 |
| Ross et al. 2013 unpubl | D9 | F | 31 | 2003 | 205 | 64.3 | 6.37E+00 | 6.37 |
| Ross et al. 2013 unpubl | A74 | M | 3 | 2003 | 205 | 64.3 | 6.83E+00 | 6.83 |
| Ross et al. 2013 unpubl | A60 | M | 11 | 2003 | 205 | 64.3 | 1.53E+01 | 15.30 |
| Ross et al. 2013 unpubl | I67 | M | 12 | 2003 | 205 | 64.3 | 2.37E+01 | 23.70 |
| Ross et al. 2013 unpubl | A55 | M | 13 | 2003 | 205 | 64.3 | 2.60E+00 | 2.60 |
| Ross et al. 2013 unpubl | R28 | M | 15 | 2003 | 205 | 64.3 | 3.48E+00 | 3.48 |
| Ross et al. 2013 unpubl | I42 | M | 20 | 2003 | 205 | 64.3 | 7.15E+00 | 7.15 |
| Ross et al. 2013 unpubl | A33 | M | 32 | 2003 | 205 | 64.3 | 1.12E+01 | 11.20 |
| Ross et al. 2013 unpubl | C16 | F | 15 | 2004 | 205 | 64.3 | 6.65E+00 | 6.65 |
| Ross et al. 2013 unpubl | A71 | M | 5 | 2004 | 205 | 64.3 | 3.27E+00 | 3.27 |
| Ross et al. 2013 unpubl | I110 | * | 2 | 2007 | 205 | 64.3 | 5.14E+00 | 5.14 |

| Source | Animal ID | Sex | Age | Year | # congeners analysed | Lipid (%) | ΣPCB (mg·kg-1lw) | ΣPCB mg/kg |
|---|---|---|---|---|---|---|---|---|
| Ross et al. 2013 unpubl | I80 | F | 10 | 2007 | 205 | 64.3 | 2.50E+01 | 25.00 |
| Ross et al. 2013 unpubl | B14 | F | 16 | 2007 | 205 | 64.3 | 1.37E+00 | 1.37 |
| Ross et al. 2013 unpubl | I35 | F | 33 | 2007 | 205 | 64.3 | 1.21E+00 | 1.21 |
| Ross et al. 2013 unpubl | R43 | M | 5 | 2007 | 205 | 64.3 | 1.16E+01 | 11.60 |
| Ross et al. 2013 unpubl | A71 | M | 8 | 2007 | 205 | 64.3 | 8.18E+00 | 8.18 |
| Ross et al. 2013 unpubl | C22 | M | 10 | 2007 | 205 | 64.3 | 8.52E+00 | 8.52 |
| Ross et al. 2013 unpubl | R31 | M | 10 | 2007 | 205 | 64.3 | 2.12E+01 | 21.20 |
| Ross et al. 2013 unpubl | A61 | M | 13 | 2007 | 205 | 64.3 | 9.78E+00 | 9.78 |
| Ross et al. 2013 unpubl | R30 | M | 13 | 2007 | 205 | 64.3 | 6.59E+00 | 6.59 |
| Ross et al. 2013 unpubl | I64 | M | 17 | 2007 | 205 | 64.3 | 4.47E+00 | 4.47 |
| Ross et al. 2013 unpubl | I46 | M | 22 | 2007 | 205 | 64.3 | 7.22E-01 | 0.72 |
| Ross et al. 2013 unpubl | A86 | * | 2 | 2008 | 205 | 64.3 | 5.97E+00 | 5.97 |
| Ross et al. 2013 unpubl | A84 | * | 3 | 2008 | 205 | 64.3 | 9.30E+00 | 9.30 |
| Ross et al. 2013 unpubl | I110 | * | 3 | 2008 | 205 | 64.3 | 1.07E+01 | 10.70 |
| Ross et al. 2013 unpubl | A84 | * | 3 | 2008 | 205 | 64.3 | 9.99E+00 | 9.99 |
| Ross et al. 2013 unpubl | A78 | * | 5 | 2008 | 205 | 64.3 | 2.78E+00 | 2.78 |
| Ross et al. 2013 unpubl | A72 | F | 7 | 2008 | 205 | 64.3 | 1.45E+01 | 14.50 |
| Ross et al. 2013 unpubl | A75 | F | 7 | 2008 | 205 | 64.3 | 1.29E+01 | 12.90 |
| Ross et al. 2013 unpubl | C24 | F | 8 | 2008 | 205 | 64.3 | 6.14E+01 | 61.40 |
| Ross et al. 2013 unpubl | A67 | F | 12 | 2008 | 205 | 64.3 | 5.33E+00 | 5.33 |

| Source | Animal ID | Sex | Age | Year | # congeners analysed | Lipid (%) | ΣPCB (mg·kg-1lw) | ΣPCB mg/kg |
|---|---|---|---|---|---|---|---|---|
| Ross et al. 2013 unpubl | A54 | F | 19 | 2008 | 205 | 64.3 | 1.77E+00 | 1.77 |
| Ross et al. 2013 unpubl | A51 | F | 22 | 2008 | 205 | 64.3 | 1.11E+01 | 11.10 |
| Ross et al. 2013 unpubl | R43 | M | 6 | 2008 | 205 | 64.3 | 1.33E+01 | 13.30 |
| Ross et al. 2013 unpubl | I78 | M | 11 | 2008 | 205 | 64.3 | 8.52E+00 | 8.52 |
| Ross et al. 2013 unpubl | R30 | M | 14 | 2008 | 205 | 64.3 | 1.02E+01 | 10.20 |
| Ross et al. 2013 unpubl | R28 | M | 16 | 2008 | 205 | 64.3 | 9.11E+00 | 9.11 |
| Ross et al. 2013 unpubl | I67 | M | 17 | 2008 | 205 | 64.3 | 9.38E+00 | 9.38 |
| Ross et al. 2013 unpubl | I64 | M | 18 | 2008 | 205 | 64.3 | 6.81E+00 | 6.81 |
| Ross et al. 2013 unpubl | I62 | M | 20 | 2008 | 205 | 64.3 | 8.36E+00 | 8.36 |
| Ross et al. 2013 unpubl | I46 | M | 23 | 2008 | 205 | 64.3 | 1.53E+01 | 15.30 |
| Ross et al. 2013 unpubl | I42 | M | 25 | 2008 | 205 | 64.3 | 1.46E+01 | 14.60 |
| Ross et al. 2013 unpubl | A39 | M | 33 | 2008 | 205 | 64.3 | 7.68E+00 | 7.68 |
| Ross et al. 2013 unpubl | A79 | * | 4 | 2009 | 205 | 64.3 | 1.21E+01 | 12.10 |
| Ross et al. 2013 unpubl | R44 | * | 5 | 2009 | 205 | 64.3 | 1.34E+01 | 13.40 |
| Ross et al. 2013 unpubl | R39 | F | 8 | 2009 | 205 | 64.3 | 7.88E+00 | 7.88 |
| Ross et al. 2013 unpubl | R35 | F | 11 | 2009 | 205 | 64.3 | 1.33E+01 | 13.30 |
| Ross et al. 2013 unpubl | R29 | F | 15 | 2009 | 205 | 64.3 | 9.00E+00 | 9.00 |

*Appendix Table 3 - Summary of total polychlorinated biphenyls (ΣPCB) concentrations measured in Southern Resident Killer Whale blubber from 1993-2015 (adapted from Guy 2018 unpubl* [5] *- Appendix E, Table E1). Source of PCB data: Krahn et al. 2007, 2009; Ross et al. 2013 unpubl.*[9]*; Guy 2018 unpubl* [5]

| Source | Animal ID | Sex | Age | Year | # congeners analysed | Lipid (%) | ΣPCB (mg·kg-1lw) | ΣPCB Converted (mg/kg) |
|---|---|---|---|---|---|---|---|---|
| Ross et al. 2013 unpubl | J6 | M | 37 | 1993 | 205 | 64.3 | 5.93E+00 | 5.93 |
| Ross et al. 2013 unpubl | J3 | M | 40 | 1993 | 205 | 64.3 | 1.62E+02 | 162.00 |
| Ross et al. 2013 unpubl | J20 | F | 16 | 1996 | 205 | 64.3 | 7.47E+01 | 74.70 |
| Ross et al. 2013 unpubl | J18 | M | 20 | 1996 | 205 | 64.3 | 6.32E+01 | 63.20 |
| Ross et al. 2013 unpubl | J11 | F | 41 | 1996 | 205 | 64.3 | 3.47E+01 | 34.70 |
| Ross et al. 2013 unpubl | J1 | M | 46 | 1996 | 205 | 64.3 | 1.92E+02 | 192.00 |
| Ross et al. 2013 unpubl | J18 | M | 23 | 2000 | 205 | 64.3 | 2.48E+02 | 248.00 |
| Ross et al. 2013 unpubl | L78 | M | 15 | 2004 | 205 | 64.3 | 8.53E+00 | 8.53 |
| Krahn et al. 2007 | L78 | M | 15 | 2004 | 45 | 15.2 | 2.20E+01 | 22.00 |
| Ross et al. 2013 unpubl | L74 | M | 18 | 2004 | 205 | 64.3 | 2.22E+01 | 22.20 |
| Ross et al. 2013 unpubl | L71 | M | 18 | 2004 | 205 | 64.3 | 1.78E+01 | 17.80 |
| Krahn et al. 2007 | L71 | M | 18 | 2004 | 45 | 9.6 | 3.60E+01 | 36.00 |
| Krahn et al. 2007 | L74 | M | 18 | 2004 | 45 | 18 | 4.50E+01 | 45.00 |
| Krahn et al. 2007 | J39 | M | 3 | 2006 | 45 | 40.9 | 3.40E+01 | 34.00 |
| Krahn et al. 2007 | J27 | M | 15 | 2006 | 45 | 30.4 | 7.40E+01 | 74.00 |
| Krahn et al. 2007 | L85 | M | 15 | 2006 | 45 | 24.8 | 5.00E+01 | 50.00 |
| Krahn et al. 2007 | J19 | F | 27 | 2006 | 45 | 29.4 | 4.50E+01 | 45.00 |
| Krahn et al. 2007 | L57 | M | 29 | 2006 | 45 | 19.4 | 5.60E+01 | 56.00 |
| Krahn et al. 2007 | J1 | M | 55 | 2006 | 45 | 21.9 | 1.80E+02 | 180.00 |
| Krahn et al. 2009 | J38 | M | 4 | 2007 | 45 | 20.9 | 4.10E+01 | 41.00 |
| Krahn et al. 2009 | K34 | M | 6 | 2007 | 45 | 22.3 | 3.90E+01 | 39.00 |
| Krahn et al. 2009 | K36 | F | 4 | 2007 | 45 | 18.3 | 6.20E+01 | 62.00 |
| Krahn et al. 2009 | L87 | M | 15 | 2007 | 45 | 25.6 | 2.40E+01 | 24.00 |
| Krahn et al. 2009 | J22 | F | 22 | 2007 | 45 | 28.4 | 4.60E+00 | 4.60 |

| Source | Animal ID | Sex | Age | Year | # congeners analysed | Lipid (%) | ΣPCB (mg·kg-1lw) | ΣPCB Converted (mg/kg) |
|---|---|---|---|---|---|---|---|---|
| Krahn et al. 2009 | L67 | F | 22 | 2007 | 45 | 29.2 | 4.30E+00 | 4.30 |
| Krahn et al. 2009 | L73 | M | 21 | 2007 | 45 | 23.8 | 3.20E+01 | 32.00 |
| Krahn et al. 2009 | K21 | M | 35 | 2007 | 45 | 26.6 | 3.80E+01 | 38.00 |
| Krahn et al. 2009 | K13 | F | 35 | 2007 | 45 | 22 | 8.90E+00 | 8.90 |
| Krahn et al. 2009 | L26 | F | 51 | 2007 | 45 | 22.1 | 2.70E+01 | 27.00 |
| Krahn et al. 2009 | L21 | F | 57 | 2007 | 45 | 18.7 | 5.50E+01 | 55.00 |
| Krahn et al. 2009 | K7 | F | 97 | 2007 | 45 | 28.5 | 1.20E+02 | 120.00 |
| Guy 2018 unpubl | J49 | M | 4 | 2015 | 209 | 64.3 | 2.77E+01 | 27.70 |
| Guy 2018 unpubl | L103 | F | 13 | 2015 | 209 | 64.3 | 1.33E+01 | 13.30 |
| Guy 2018 unpubl | L116 | M | 6 | 2015 | 209 | 64.3 | 4.75E+01 | 47.50 |
| Guy 2018 unpubl | J37 | F | 15 | 2015 | 209 | 64.3 | 3.01E+00 | 3.01 |
| Guy 2018 unpubl | Blubber | F | 18 | 2015 | 209 | 64.3 | 4.41E+01 | 44.10 |
| Guy 2018 unpubl | K22 | F | 29 | 2015 | 209 | 64.3 | 1.42E+01 | 14.20 |
| Guy 2018 unpubl | L72 | F | 30 | 2015 | 209 | 64.3 | 9.66E+00 | 9.66 |
| Guy 2018 unpubl | K25 | M | 25 | 2015 | 209 | 64.3 | 1.03E+01 | 10.30 |
| Guy 2018 unpubl | K13 | F | 44 | 2015 | 209 | 64.3 | 4.83E+00 | 4.83 |

### References Appendix II

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