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Killer Whales (*Orcinus orca*) Face Protracted Health Risks Associated with Lifetime Exposure to PCBs

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Polychlorinated biphenyl (PCB) concentrations declined rapidly in environmental compartments and most biota following implementation of regulations in the 1970s. However, the metabolic recalcitrance of PCBs may delay responses to such declines in large, long-lived species, such as the endangered and highly PCB-contaminated resident killer whales (*Orcinus orca*) of the Northeastern Pacific Ocean. To investigate the influence of life history on PCB-related health risks, we developed models to estimate PCB concentrations in killer whales during the period from 1930 forward to 2030, both within a lifetime (∼50 years) and across generations, and then evaluated these in the context of health effects thresholds established for marine mammals. Modeled PCB concentrations in killer whales responded slowly to changes in loadings to the environment as evidenced by slower accumulation and lower magnitude increases in PCB concentrations relative to prey, and a delayed decline that was particularly evident in adult males. Since PCBs attained peak levels well above the effects threshold (17 mg/kg lipid) in ∼1969, estimated concentrations in both the northern and the more contaminated southern resident populations have declined gradually. Projections suggest that the northern resident population could largely fall below the threshold concentration by 2030 while the endangered southern residents may not do so until at least 2063. Long-lived aquatic mammals are therefore not protected from PCBs by current dietary residue guidelines.

Introduction

Fish-eating resident killer whales (*Orcinus orca*) that frequent coastal waters of British Columbia, Canada, and Washington State, U.S., face a daunting set of conservation threats, including noise and disturbance associated with vessel traffic, reduced prey abundance, and very high concentrations of potentially toxic chemicals (*1*, *2*). The "northern resident" population of approximately 200 individuals is listed as "threatened" under the terms of the Canadian Species at Risk Act (SARA), while the transboundary "southern resident" population of approximately 85 individuals is listed as "endangered" under SARA, as well as under the terms of the U.S. Endangered Species Act (ESA) (Figure 1). Legally enacted Recovery Strategy (Canada) and Conservation Planning (United States) teams have been tasked with quantifying the threats to these populations and identifying mitigative strategies to ensure the long-term survival of these iconic marine mammals (*3*−*5*).

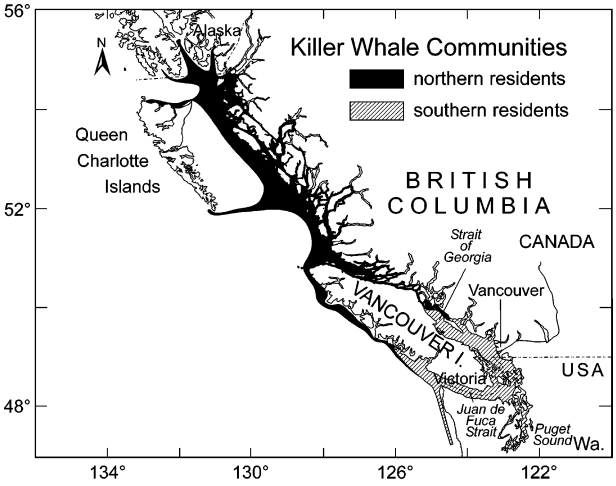

FIGURE 1. "Threatened" northern resident killer whales and "endangered" southern resident killer whales frequent the coastal waters of British Columbia, Canada, and the states of Alaska and Washington. Very high levels of PCBs measured in biopsies from these long-lived cetaceans are viewed as a major conservation concern and an obstacle to population recovery (*2*). Map adapted from ref *52*.

Given that killer whales are among the most PCB-contaminated marine mammals in the world (*6*), their protection presents two important, if daunting, challenges regarding potentially toxic chemicals: (1) how to characterize current health risks associated with contaminant exposure and/or burdens; and (2) how to determine the extent to which contaminant inputs into their habitat must be reduced to protect their health. After considering influential factors such as age, sex, dietary preferences, and calving order on observed contaminant concentrations, a weight of evidence suggests that the risk of adverse health effects is high (*6*, *7*), although direct evidence in killer whales is, understandably, lacking. However, significant unknowns remain for killer whales, with one-time biopsies providing a restricted temporal "snapshot" of contaminant concentrations stored in blubber, no samples or data from before 1993, and no direct means of assessing the contribution of exposure history to present burdens. Their very long life expectancy (up to 90 years for females and 50 years for males) complicates exposure assessments, and raises important questions about how best to evaluate adverse health effects. Given that some current members of killer whale populations were born before the widespread release of persistent organic pollutants (POPs) into the environment, lifetime contaminant exposure needs to be a component of health risk evaluation.

Risk characterizations based on the results of a variety of study designs and marine mammal species have resulted in estimated thresholds for the onset of toxic effects ranging from 10 to 77 mg/kg PCB in lipid in the blubber or liver of marine mammals (*8*−*13*). Characterizing exposure and bioaccumulation is the basis for toxicological risk assessments and for the development of guidelines to protect wildlife.

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However, current paradigms based on assumed steady-state conditions (e.g., biomagnification factors) fail to address issues such as temporal changes in environmental contaminant concentrations and the lifespan of a given species. These paradigms may therefore not protect long-lived wildlife exposed to highly persistent contaminants. Species-specific dynamic bioaccumulation models, in combination with proxy contaminant exposures derived from sediment core profiles or other sources, have been used to reconstruct the history of PCBs and other POPs in St. Lawrence beluga whales (*Delphinapterus leucas*) (*14*) and Arctic ringed seals (*Phoca hispida*) (*15*). Although such models have provided an overview of contaminant histories in these two marine mammal species, the consequence of persistent contaminants on their health is not clear.

Wildlife species at or near the top of aquatic food webs are typically contaminated with high concentrations of POPs, among which the PCBs frequently dominate. Furthermore, adverse health effects in these top predators in the northern hemisphere have been largely attributed to the PCBs, underscoring the ecotoxicological relevance of this class of chemical (*16*). Here we (1) present two models that provide quantitative assessments of the bioaccumulation of PCBs by killer whales at both individual and population levels, (2) provide comprehensive histories of PCB exposure and accumulation for two populations back to 1930, (3) examine the prospects for future declines of PCB concentrations in the populations, and (4) compare results to a toxicity threshold established in a captive feeding study for marine mammals and to current dietary tissue guidelines designed to protect aquatic wildlife.

Methods

Individual-based (IB) and population-based (PB) models were adapted from ones previously developed and used to reconstruct temporal trends of persistent organic pollutants in beluga whales (*14*) and ringed seals (*15*). The IB model provides a dynamic accounting of the pharmacokinetics and overall mass balance of a persistent organic pollutant over the entire life of an average individual and takes into account the animal's complete life history, including growth, body composition, energetics and diet, gestation, birth, and lactation. The PB model uses energy and contaminant flux budgets summarized from the IB model and quantifies the changes in contaminant levels throughout an age-structured population (up to 50 years of age) over several generations. The IB and PB models yield similar results when calibrated with the same parameter values. Details on the structure and calibration of these models for killer whales are provided in the Supporting Information, along with the estimation of an elimination rate constant of 0.02 yr^{-1} for PCBs.

We used the two models to characterize the bioaccumulation of PCBs by the two resident fish-eating killer whale populations from the northeastern Pacific Ocean from 1930 to the present and to project potential future concentrations up to 2030. Together, these models describe both the detailed and long-term effects of killer whale biology and life history on the accumulation of POPs over multiple generations. Such an approach is imperative since some killer whales can live for 90 years with mean life expectancies of about 50 years for females and 29 years for males in the northern resident population (*17*). Adults may weigh in excess of 7000 kg, but males typically weigh about 5000 kg and females weigh about 2700 kg and reach full size by about 20 years of age (*18*). Females first give birth at about 15 years of age and thence about every 5 years thereafter up to about 40 years of age (*17*). Gestation lasts about 17 months with neonates weighing about 155 kg (*19*). Calves likely nurse for 1−2 years, with milk being supplemented by solid food in the later stages (*20*). For simplicity, we assume here an exclusive nursing period of 12 months ending with instantaneous weaning in most simulations. Feeding rates of free-ranging orcas have been estimated to be about 2−4% of body weight per day (*21*).

Reconstructing the PCB History in Killer Whale Prey. Marine mammals accumulate PCBs and other POPs exclusively from their diet. Adult salmon make up about 96% of the diet of both southern and northern resident killer whale populations, with chinook (*Oncorhyncus tshawytscha*) being the most frequently identified salmonid in their diet (*22*). From this we assumed that PCB concentrations in chinook salmon reflect the average dietary exposure concentration for both populations, thus excluding the possible contribution of unknown minor diet components that may be highly contaminated. PCB concentrations in adult chinook collected from Johnstone Strait (within the range of the northern residents) in 2000 averaged 13 μg/kg PCB wet wt (sum of ~100 congeners; range 8.8−17.5 μg/kg; $n = 6$; Cullon, personal communication; analytical methods described elsewhere (*6*)). Chinook from Puget Sound collected between 1992 and 1995 averaged 54 μg/kg PCB wet wt (range 12−200 μg/kg) (*23*). We hypothesize that the lower PCB concentrations in the Johnstone Strait chinook reflect their broad-scale background exposure resulting from long-range transport and deposition to the northeastern Pacific Ocean and remote coastal waters. The higher and broader range of concentrations in Puget Sound adult chinook likely reflect a mix of less contaminated open ocean stocks returning to the region and more highly contaminated stocks that remain close to local coastal PCB sources.

Since no long-term POPs monitoring data exist for chinook or other fish species from the region we have estimated the history of PCB contamination in the diet for the northern and southern resident orcas back to 1930 using trends derived from sediment cores from coastal locations (Puget Sound (*24*), Georgia Basin (*25*), unpublished data) and several inland lakes (LRTAP lakes) which provide records of broad-scale atmospheric loadings (*26*, *27*). Concentrations of PCBs were normalized within each core by expressing them as a proportion of the concentration in the most recently deposited layer measured (range from 1989 to 1997). The normalized trends for Puget Sound, Georgia Basin, and the pooled LRTAP lakes (Supporting Information) show similar relative peak heights (ranging from 3.8 to 4.3 times higher than the most recent levels) but distinctly different loading histories with peak loadings in about 1960, 1972, and 1967, respectively. PCB concentrations appear to have changed little in the cores since the mid 1980s, similar to the trends evident in eggs collected from bird colonies in the Strait of Georgia (*28*, *29*).

Historical PCB diet concentrations for the northern residents were estimated by multiplying the normalized trend from the LRTAP lakes (Figure 2) by the recent average concentration in Johnstone Strait chinook, which yielded a peak exposure concentration of 56 μg/kg PCB wet wt in 1967, about 4.3 times higher than recent levels. Reconstructing the dietary PCB history for the southern residents is far more complex because both predator and prey are highly mobile and the sediment core profiles reveal spatial, temporal, and compositional heterogeneity in PCB loadings. In addition, the strength of the several chinook salmon stocks that run through the Strait of Georgia and Puget Sound vary considerably from year to year (*30*). The approach we took was to construct plausible diet histories using the three loadings profiles with the constraints that the recent average diet concentration equals 54 μg/kg, that ocean-run (i.e., LRTAP) chinook represent at least 50% of the diet, and that the recent concentration in fish from Puget Sound not exceed 200 μg/kg. The resulting diet histories all reach peak concentrations, ranging from 177 to 197 μg/kg, in the early 1960s and differ by less than 5% in terms of area under the curve. These peak

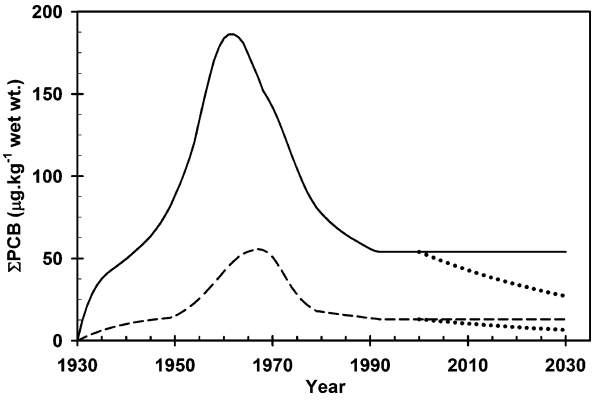

FIGURE 2. Estimated histories of PCB concentrations in the chinook salmon diet for the northern (dashed line) and southern resident (solid line) killer whales. The northern resident diet was based on PCB concentrations in chinook salmon (Cullon, personal communication) and the long-range transport of atmospheric pollutants (LRTAP) sediment profile. The southern resident diet scenario was based on weighted contributions from LRTAP (65%), Puget Sound (16%), and Strait of Georgia (19%) sediment cores, representing a combination of local and "global" sources. Two possible future diet scenarios are shown, representing a steady-state PCB exposure (solid lines extending past 2000) and a PCB decline starting in 2000 where an environmental half-life of 30 years is assumed (dotted lines extending past 2000).

concentrations are between 3.3 and 3.6 times higher than recent exposure levels, and are about 3.2 to 3.5 times higher than the peak concentrations in the diet of northern resident orcas. One historical profile close to the average (Figure 2) was used in the simulations presented here for the southern resident killer whales. While sediment core profiles provide a means of inferring past contaminant levels in biota, this procedure may be subject to bias. For example, sediment concentrations may be offset by several years from loadings and water concentrations, as noted for Lake Ontario (*31*, *32*), and sediment profiles may be lowered and broadened due to mixing processes such as bioturbation. An advective−diffusive model was applied to account for such possible influences in the case of Georgia Basin sediment cores (*25*).

Reconstructing PCB Histories in Killer Whales. Model PCB exposure simulations were run for northern and southern resident populations from the 1930s to present and then forward to 2030 to project future risks. Specifically, the simulations were designed to estimate the peak tissue concentrations achieved in the past by the two populations and the contribution of past exposure to current concentrations. Initial simulations yielded the probable history of PCB concentrations in the northern resident population back to 1930 using the reconstructed exposure history based on the LRTAP signature (Figure 2). The model skill was evaluated by comparing predicted concentrations from the 1994−2000 period with measured values from 38 whales collected by biopsy darts in 1993−1994 (*6*) and an additional 15 animals collected in 2000 (28 males, 25 females; ∼26% of the population). The contribution of PCBs from past exposure to concentrations found in the 1994−2000 period was estimated by the difference between this simulation and one run to steady state using a fixed diet PCB concentration of 13 μg/kg (ww), reflecting recent concentrations in chinook salmon from Johnstone Strait. This fixed exposure simulation also indicates what would be expected if current exposure levels do not change in the future. We also used the model to evaluate killer whale contamination response to future exposure scenarios ranging from no change to gradual declines based on environmental half-lives between 20 and 30 years.

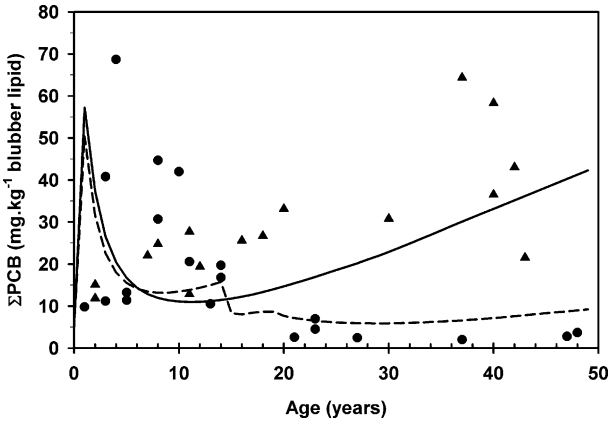

FIGURE 3. Measured PCB concentrations in male (▲) and female (●) northern resident killer whales from biopsies collected in 1994−2000 (*6*), and estimated concentrations derived from the population-based (PB) model (solid line: male; dashed line: female) using the LRTAP dietary exposure profile shown in Figure 2. Modeled concentrations agree well with observed concentrations, and show a rapid rise associated with nursing, followed by a growth dilution phase up to ∼10 years of age. Concentrations in males and females diverge following the onset of reproduction at about ∼14 years.

Similar simulations were conducted for the southern resident population using the SR diet history from Figure 2 (and others) but can only be compared to PCB data from 6 animals (4 males, 2 female; ∼7% of the population) collected in 1994 (*6*). A final simulation ran the PB model to steady state using a fixed diet PCB concentration consistent with the Canadian tissue residue guideline (TRG) for the protection of fish-eating wildlife (*33*). This TRG (0.79 ng/kg toxic equivalents (TEQs)) was converted to a PCB concentration of 50 μg/kg based on the regression

$$\text{TEQ (ng/g)} = 0.0148 \times \text{PCB (ng/g)} + 0.0431 \quad (r^2 = 0.984\ n = 12,\ p < 0.0001)$$

which was derived from chinook salmon (Cullon, personal communication). The TEQs in salmon were calculated using Toxic Equivalency Factors (TEFs) derived for mammals (*34*). It is noteworthy that 50 μg/kg is close to the current average PCB concentration in the diet of the southern resident population, and is approximately 3-fold lower than the USEPA (*35*) wildlife protection value of 160 μg/kg.

Results and Discussion

No model, be it one designed to characterize climate change (*36*), contaminant cycling (*37*), or bio-uptake (*14*, *15*), can capture complete system details. The model we present here is no different in that exposure, accumulation, and loss cannot be represented at the individual level. As with other modeling efforts, we have developed confidence in the performance of the model through numerous simulations and comparisons with data. Despite the limitations of models, generally, they can be exceptionally useful tools for synthesizing diverse data, allowing scientists to evaluate sensitivity to various factors, and enabling future projections in the context of proposed human activities, such as a reduction of contaminant emissions following regulations.

History of PCBs in Northern Resident Killer Whales. Predicted concentrations in northern residents from the 1994−2000 period of the reconstructed history agree well with observed concentrations in biopsies from the same period (Figure 3) (*6*). When matched for age and sex, 62% of observed concentrations were within a factor of 2 of predicted concentrations, and 89% were within a factor of 3. The greatest deviations were among young animals, where

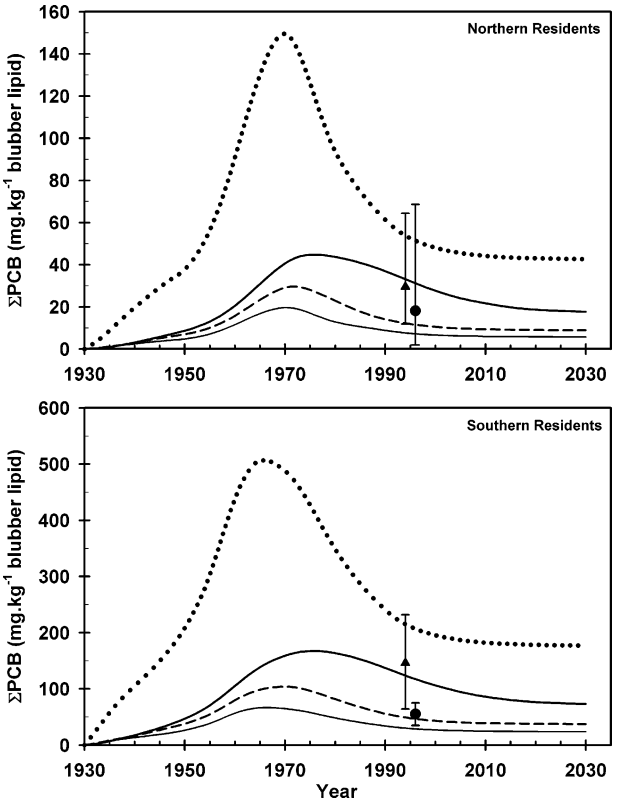

FIGURE 4. Estimated temporal trend of average PCB concentrations in four specific age classes (dotted line: 1-year-olds, dashed line: 11-year-old juveniles, thin solid line: 20-year-old females, thick solid line: 40-year-old males) for northern (upper) and southern residents (lower) using the dietary exposure profiles shown in Figure 2. Mean measured concentrations (±range) are also shown for males (▲) and females (●) as previously documented (*6*).

predicted concentrations in individual calves are sensitive to complex interactions between factors such as birth order, interval between births, duration of nursing, and early growth rate (*7*, *14*, *38*). For example, a calf born shortly after the previous one was weaned, who nurses for more than 1 year, and grows more rapidly than we have estimated, would show a shorter and wider concentration spike associated with nursing. The predicted concentrations for 1994−2000 that included past exposure history were approximately 40% higher than those from the simulation run to steady state using a fixed diet concentration from the same time period (13 μg/kg), with the difference due to the lasting contribution of more PCB-contaminated prey in the past.

The effects of age and sex are also evident throughout the reconstructed history of PCB contamination in the northern resident population as demonstrated by the temporal profiles of four representative age/gender classes (Figure 4). The curves for 1-year olds and for 20 year-old reproductively active females represent the upper and lower bounds, respectively, of predicted PCB concentrations for the population, and reflect the efficient transfer of PCBs from mother to calf. The shape of curves for adult females, 11 year-old juveniles, and 1-year olds are similar, with peak concentrations approximately three times higher than those predicted for 2006, occurring only approximately 4 years (1970−1972) after the estimated peak in dietary concentrations. Dietary concentrations were estimated to be four times higher than recently measured PCB concentrations in chinook salmon. However, PCB concentrations in 40-year-old males lag their diet by 8 years, and have a peak of only two times higher than in 2006, suggesting that a limited metabolic capacity in these long-lived animals contributes to sustained and protracted PCB body burdens.

History of PCBs in Southern Resident Killer Whales. The simulated histories of PCB contamination for select age classes in the southern resident population depict trends that are similar to those modeled for the northern residents, and show reasonable agreement to observed biopsy results from a limited sample size for this small population (Figure 4). Predicted concentrations in killer whales derived using other plausible diet PCB histories differed by less than 10% from the scenario presented here. The predicted PCB concentrations for killer whales in 1994−2000 were underestimated by 15% (juveniles and adult females) to 70% (adult males), when past exposure to higher PCB concentrations was neglected. Again, this suggests a continued contribution of past dietary exposures and limited metabolic elimination which was most evident in adult males.

We estimated that PCB concentrations in southern residents peaked in 1966−1969 for 1-year olds (507 mg/kg lipid), juveniles (104 mg/kg lipid), and reproductive females (67 mg/kg lipid), being approximately 2.6 times higher than the levels estimated for these age classes in 2006. As with the northern residents, PCB concentrations lag in 40-year-old males (peaking at 168 mg/kg lipid), occurring 10 years after the rest of the population, and with a peak of almost two times higher than 2006.

The effect of past exposure history is complex, and produces unique histories by cohort and sex within both the northern and southern resident killer whale populations. Clearly, steady-state exposure assessments that omit exposure history underestimate tissue concentrations in both populations.

Future Trend Scenarios. Assuming that dietary PCB concentrations remain stable from 1994 onward for northern and southern resident killer whale populations, average concentrations in both populations would be expected to decline by 15−22% between 1994 and 2006, and would be within 10% of steady-state by 2008. Given the variability in measured PCB concentrations in biopsy samples unaccounted for by age, sex, and reproduction, it would be difficult to detect such modest declines without a large sample size.

This "steady-state" scenario likely represents the worst case for future PCB trends. A gradual reduction in dietary exposure is more realistic, but estimates of environmental half-life ($t_{1/2}$) for PCBs vary from a few years up to 100 years (*39*, *40*). Using an illustrative $t_{1/2}$ of 30 years starting in 2000, average concentrations in both killer whale populations would be about 20% lower in 2006 and 40% lower in 2020 than those measured during the period 1994−1996. The rate of PCB decline in these simulated populations is driven primarily by the environmental $t_{1/2}$ and, to a lesser extent, by the rate of population turnover and the elimination rate constant. Because the southern residents frequent the more industrialized waters of southern British Columbia and northern Washington State, mitigation efforts such as the removal or capping of contaminated sediments might accelerate the decline of PCBs in this population.

Comparison of Contaminant Projections to Effects Thresholds. The simulations allow us to assess whether PCBs pose toxicological risks to killer whale populations either during the past or into the future. Adverse effects of PCBs on wildlife, which have been extensively studied, include changes to immune function, neurological development, endocrine endpoints, and/or reproductive health (*41*−*46*). In the absence of thresholds determined specifically for PCB-related health effects in killer whales, we selected the threshold that was established from a long-term captive feeding study designed to measure the effects of POPs (among which the PCBs dominated) on harbor seals (*Phoca vitulina*). This study determined that the immune system and endocrine endpoints were affected at 17 mg/kg PCBs in blubber lipid, and this value has been incorporated into recent risk assessments

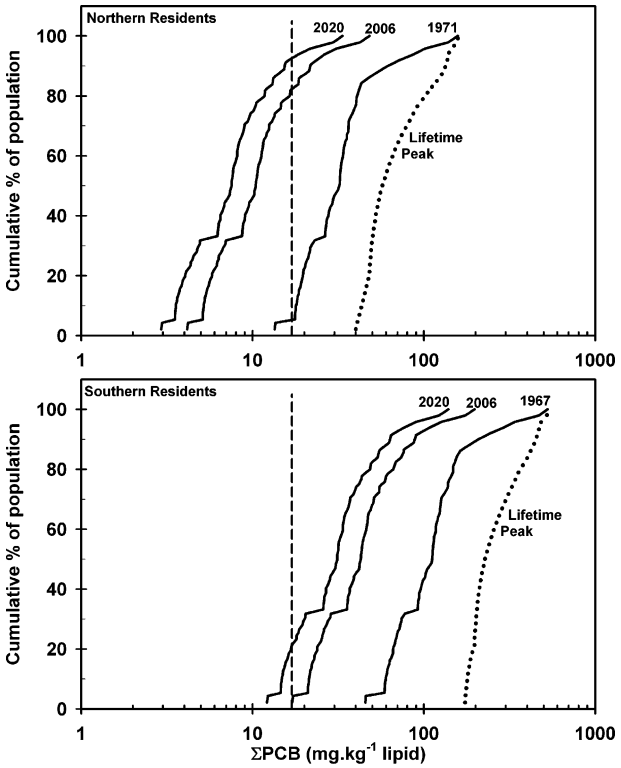

FIGURE 5. Cumulative distributions of PCB concentrations in the peak year of accumulation (1967 and 1971 for southern and northern resident populations, respectively), in the year 2006 and in 2020 (assuming a 30-year environmental $t_{1/2}$ for PCBs) shown in relation to the established health effects threshold for PCBs in marine mammals (17 mg/kg; (*8, 51*)). Cumulative distributions of peak lifetime concentrations show that all members of the two populations ca. 2006 exceeded the threshold at some point earlier in their lifetime (dotted line).

for marine mammals (*8*, *47*, *48*). This threshold value is more protective than other thresholds established for reproductive health in harbor seals (*11*) and Baltic ringed seals *Phoca hispida* (*13*), but less protective than the threshold of 10 mg/kg lipid PCBs which was associated with increased calf mortality in free-ranging bottlenose dolphins *Tursiops truncatus* (*9*).

Using thresholds to assess risk warrants caution owing to possible differences in species sensitivities, differences in exposure regime, variation in PCB composition, and the presence of additional endocrine disrupting contaminants in wildlife, including PCB metabolites, polybrominated diphenylethers (PBDEs), dioxins, and furans (*49*). Given these difficulties, we view these thresholds mainly as a standard against which to gauge the success of mitigation measures including chemical regulations, source control, and/or cleanup of contaminated areas, as well as providing a rationale for further effects-based research.

The cumulative distribution of PCB concentrations for 2006, 2020, and the year of peak burden for both the northern (1971) and southern resident (1968) populations reveals the importance of age and gender in determining health risk (Figure 5). The upper quartile comprises males over 26 years, and young juveniles from 1−4 years, with weanlings consistently being the most PCB-contaminated. Juveniles aged 5−14 years and young adult males up to 25 years of age make up the next 43% of the distribution, followed by reproductive females (∼28%), and by neonates (4%), the latter of which have the lowest PCB concentrations.

This succession shows that nursing killer whales can progress from being the least contaminated to the most contaminated members of the population in the span of approximately 1 year. After weaning, young killer whales then progress down the distribution over the next 15 years as a consequence of switching from mother's milk to a less contaminated (fish) diet and growth dilution. Once mature, females continue down the distribution due to reproductive transfer of persistent hydrophobic chemicals, while males move back up the distribution as they age and are unable to readily eliminate PCBs. Thus, individuals experience a wide range of PCB concentrations over their lifetime, underscoring the uncertainty of using a single point dose metric to assess risk rather than one which incorporates temporal change.

In the peak year of exposure for northern residents (1971), 96% of the population exceed the 17 mg/kg PCB threshold. In 2006, 18% of the population continues to exceed this threshold. All members of the population in 2006, however, would have exceeded this threshold at some time during their lives (generally as young animals). This peak lifetime distribution (Figure 5) must be viewed with the caveat that predicted concentrations in young whales have the greatest uncertainty of any age group in the model population. Furthermore, the toxicological significance of this dose metric is predicated on PCBs causing developmental effects at this life stage that may then persist through life.

Future projections show modest declines in threshold exceedences by the year 2020 for both the steady-state exposure (14% of population >17 mg/kg) and the 30 yr PCB $t_{1/2}$ (8% of population >17 mg/kg) scenarios. We estimate that the long-term PCB concentrations in killer whale prey would have to drop to approximately 8 μg/kg PCBs (wet weight) for 95% of a killer whale population to fall below the 17 mg/kg threshold. Under the 30 yr PCB $t_{1/2}$ scenario, this objective would be reached by the year 2030.

PCB concentrations in the southern resident killer whales were 2.5−3.7 times higher than those in the northern residents during the period since 1970. In the year of peak tissue concentrations (1968), all members exceeded the threshold by factors of 2.6−31.0 times. For 2006, all members are projected to exceed the threshold by factors of 1.1−11.6. A substantial proportion of the population is projected to exceed the threshold in 2020 under both the steady state (96% of population >17 mg/kg) and the 30 yr PCB $t_{1/2}$ (79% of population >17 mg/kg) exposure scenarios. Using a 30-yr environmental $t_{1/2}$ for PCBs, the model suggests that 95% of the southern resident population will drop below the threshold only after ∼2089. Since killer whales can live up to ∼90 years, many individuals alive today have passed through the era of peak PCB contamination, and/or have inherited its legacy from maternal transfer. Even assuming a 20-year $t_{1/2}$ for PCBs, we estimate that it will take at least 14 (2020) and 57 (2063) years for 95% of the northern and southern resident populations, respectively, to fall below the effects threshold.

To protect wildlife, Tissue Residue Guidelines (TRGs) are set for the maximum acceptable PCB concentration in prey. By simulating an exposure to a fixed dietary PCB concentration equivalent to the lowest published TRG found (Environment Canada diet residue guideline of 50 μg/kg PCB or 0.79 ng/kg TEQ; (*33*)), PCBs in a killer whale population would stabilize at 15−174 mg/kg lipid in approximately 50 years. A diet at the TRG concentration in our model would place 95% of the killer whale population over the threshold, suggesting that the TRG provides inadequate protection for killer whales. Transient killer whales, which feed on trophically higher marine mammals (*6*), would also not be protected.

There are a number of reasons why TRGs might fail to protect long-lived species such as killer whales. Foremost, perhaps, is that the nature of marine mammal toxicology

studies has resulted in their being excluded from the derivation of TRGs (*50*) for PCBs or other contaminants. This is because TRG criteria often require the demonstration of a dose−response relationship, a well-defined effects threshold, and clear endpoints of high ecological relevance such as survival, growth, or reproduction. Ethical, logistical, and legal constraints have generally confined marine mammal toxicology studies to (i) the use of two treatment groups, (ii) associative field studies which lack a dose−response relationship with a single contaminant class, or (iii) studies that lack endpoints of perceived ecological relevance (*11*, *13*, *51*). In addition, while TRGs are intended to protect the most sensitive life stages, their derivation does not effectively address interspecies differences in contaminant transfer via nursing, and clearly underestimate PCB exposure in killer whale calves.

Persistent contaminants such as PCBs may hinder the recovery of endangered killer whale populations by increasing vulnerability to infectious disease, reducing reproductive performance, and impeding normal growth and development. Given the constraints in conducting toxicological research on large and/or endangered marine mammals, the use of models such as the ones presented here provide conservationists, regulators, and managers with benchmarks against which the effectiveness of mitigative steps can be measured and tissue residue guidelines can be evaluated. The results of these models provide little confidence that simple dietary guidelines afford protection to long-lived animals such as killer whales.

Acknowledgments

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Supporting Information Available

Detailed descriptions of the IB and PB models, parameter values used in the simulations, and a summary of the sediment core profiles used to derived exposure histories. This material is available free of charge via the Internet at http://pubs.acs.org.

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