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The potential effect of PCBs on Killer whales using the SPOC individual based pollution model approach to estimate impacts on population growth.

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The potential effect of PCBs on Killer whales – using the 'SPOC' individual based pollution model approach to estimate impacts on population growth.

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Abstract

Preliminary results of simulations, using the IWC Pollution 2020 'SPOC' individual based pollution model to explore the effects of PCBs on Pacific killer whales populations, found that the *potential* population growth rate of Northern resident killer whales (NRKW) would only be substantially affected, through effects of PCBs on calf survival and immunity, when a high proportion of the population were subsequently exposed to a newly introduced pathogen. An annual accumulation of ~1 mg/kg total PCBs resulted in levels in the adult females comparable to the published empirical data (<10 mg/kg lipid weight total PCBs). However, for the Southern resident killer whale (SRKW) population, the model simulations suggested they were experiencing a substantially higher accumulation of approximately 5 mg/kg total PCBs per year as this level was required to produce concentrations in the adult females that were similar to the published empirical data (~ 50 m/kg lipid weight). At this level, in all scenarios where immune effects were also considered, population growth rates (lambda) estimates fell to <1.0, suggesting that PCB exposure could result in a declining population at various levels of introduced pathogen exposure. However, if the accumulation of PCBs declined by 1% per annum after year 50 of 100 years of simulated populations, this would result in a stationary population with lambda ~ 1.0.

This modelling approach has been taken to assist in assessing the risks of PCB exposure to killer whale populations under various exposure conditions. However, not all the drivers of population change have been considered and these simulations are likely to represent worst case scenarios. The concentration (dose) response functions embedded in the model are based on laboratory animal data and are therefore not species specific. In addition, direct effects of PCBs on fecundity are not considered here and density dependence is not taken into account. Thus, the results should be interpreted as impacts on *potential* rather than realised population growth. Interactions with other stressors, anthropogenic noise, contaminants and disease agents, effects of historical exploitation on the population and prey availability, to give just a few examples, would all need to be considered alongside the PCB contaminants as critical factors affecting vital rates and therefore population dynamics.

Introduction

There is a great deal of interest in the potential effect of contaminant exposure on cetacean populations, particularly the legacy pollutants such as the polychlorinated biphenyls (PCBs) which continue to have an impact on the health of individuals and populations, some 35 years after a ban on their production and use (De Voogt and Brinkman, 1989). One approach to better understand impacts at the population level has been the development of an individual based model which modifies annual survival probabilities based on the accumulation and transfer of PCBs and their estimated effects on calf survival and immunity. This approach, developed as part of the IWC Pollution 2000+ and 2020 initiatives (Hall et al. 2011, 2012, 2014), has been applied to populations of bottlenose dolphins (*Tursiops truncatus*) and humpback whales (*Megaptera novaeangliae*). Here, we apply it, using published data and population parameters, to the northern and southern resident populations of killer

whales ("NRKW" and "SRKW", *Orcinus orca*), which inhabit the coast of British Columbia (Ford et al. 2000). Both populations of resident, fish-eating killer whales are imperilled: southern residents are listed as Endangered by both Canada and the US, and northern resident killer whales are listed as Threatened by Canada (Fisheries and Oceans Canada, 2011, National Marine Fisheries Service 2008). Canada and the US have identified the same three risk factors likely to be hindering recovery of these killer whale populations, namely: contaminants; anthropogenic noise; and limitation of the preferred prey, Chinook salmon. Quantitative data are available to link the dynamics of both NRKW and SRKW populations to interannual variability in abundance of Chinook salmon through effects on fecundity and survival in all age-sex classes (Ford et al. 2010). Anthropogenic noise and boat-based disturbance have been linked to 18-25% reductions in time spent foraging (Williams et al. 2006, Lusseau et al. 2009), as well as large proportional losses of acoustic communication space (Williams et al. 2014). To date, contaminants have been treated in a qualitative way in endangered species listing, recovery and action planning for the populations. The threat is recognized, but no attempt has been made to quantify the relationship between empirically measured contaminant levels and predictions of demographic effects/population consequences. This inability to quantify the relative influence of the three risk factors on demography makes it difficult to objectively prioritize recovery actions.

The objective is to estimate the potential impact of various levels of PCB accumulation on potential population growth. The model allows for the investigation of the effect of two concentration (dose) responses (a) the effect of maternal PCBs on calf survival and (b) the effect of maternal PCBs on calf survival combined with effects on immunity, where PCBs can affect pathogen host resistance. We then compare the estimated concentration of total PCBs in the adult females with published empirical data to determine the likelihood of different annual accumulation rates and therefore the potential for impacts to be observed at the population level.

Northern resident killer whale population parameters

This is a well-studied population and estimates of their vital rates and population trends have been published (Olesiuk et al, 1990). These estimates were used to set up an initial population with a stable age structure based on historic data and trends. The parameters used in the model are given in Table 1. This represents the abundance and population trajectory for the northern resident killer whales (NRKW) as estimated in the mid-1970s and in the individual based pollution model (SPOC, Hall et al 2012) gave a growing population with an annual growth rate of ~2% (Fig. 1, mean lambda for the simulations from years 65 to 90 = 1.0185, 95% confidence interval 1.0117, 1.0236). Whilst this is not as high was estimated by Olesiuk et al (1990) at 2.62% between 1973 and 1987, it represents a similarly rapidly growing population at a rate of similar magnitude. The model first simulates the growth of the population without the effect of PCBs using a stochastic approach to capture the uncertainty in the model parameters. Fig. 1 shows 50 model simulations. All the model outputs from these simulations represent the 'potential' population growth because density dependence and carrying capacity has not been included. All mean population growth rates are estimated from 50, 100-year simulations.

Table 1. Parameters and vital rates used in the starting population for modelling the effect of PCBs on northern resident killer whales

Parameter	Value
Calf survival	0.97
Juvenile survival	0.98
Reproductive female survival	0.999
Post reproductive female survival	0.95
Fecundity	0.20
Maximum age (life expectancy)	50
Age at sexual maturity	14
Post reproductive age	38
Total population size	125

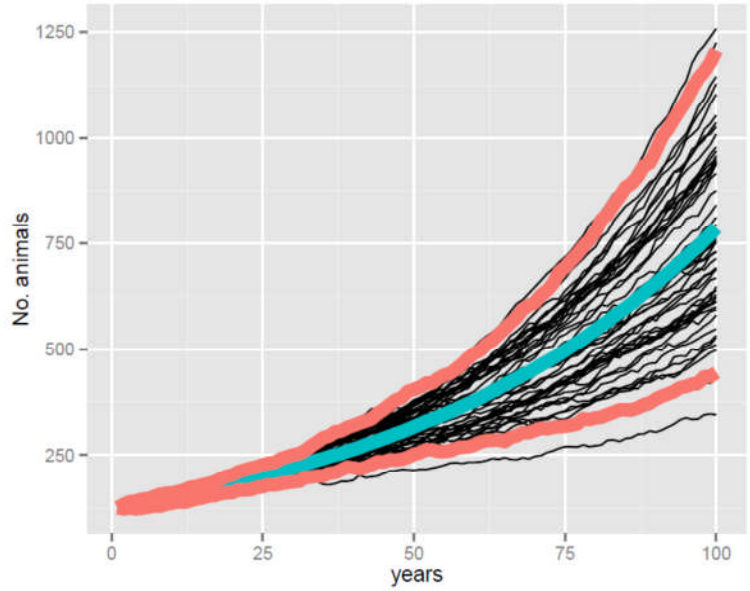

Figure 1. Baseline population representing NRKW, blue line = mean population growth, red lines = 95% confidence intervals

Effect of PCBs on calf survival

In the first set of simulations the effect of PCB uptake on calf survival was modelled. Details of the concentration (dose) response function used to modify calf survival in the individual based model are given in Hall et al. (2012). The proportions of maternal PCBs offloaded to the calf were set at 0.6 in utero and 0.77 during lactation as estimated from published data for other cetaceans species (see Hall et al. 2006 for details). The results for the 50 replicates of 100-year simulations with an annual accumulation of 1 and 3 mg/kg PCBs are shown in Fig. 2. This reduced the mean estimated potential population growth between years 65 and 90 by 0.2 and 0.6% respectively. At both accumulation rates the model indicated that the population still had the potential to increase by ~1% per annum.

These simulations also resulted in an estimate of the concentration of PCBs in the adult females between the ages of 14 and the maximum age class 50 years (Fig. 3 shows the estimated age-specific concentrations from the model). For the 1 mg/kg and 3 mg/kg annual accumulations this resulted in a mean concentration for the females of 10.43 mg/kg lipid weight and 30.53 mg/kg lipid weight, respectively. Empirical data (Ross et al 2000; Ylitalo et al 2001) indicate that the concentration of total PCBs in adult females is in the order of ~10 mg/kg lipid weight which would suggest an annual accumulation of ~1 mg/kg. This is not directly comparable to the intake of total PCBs estimated by Cullon et al (2009) of ~300 ug/d, from the consumption of Chinook salmon (*Oncorhynchus tshawytscha*), as it includes the processes of metabolism and excretion and represents what is actually assimilated into the blubber by the animals.

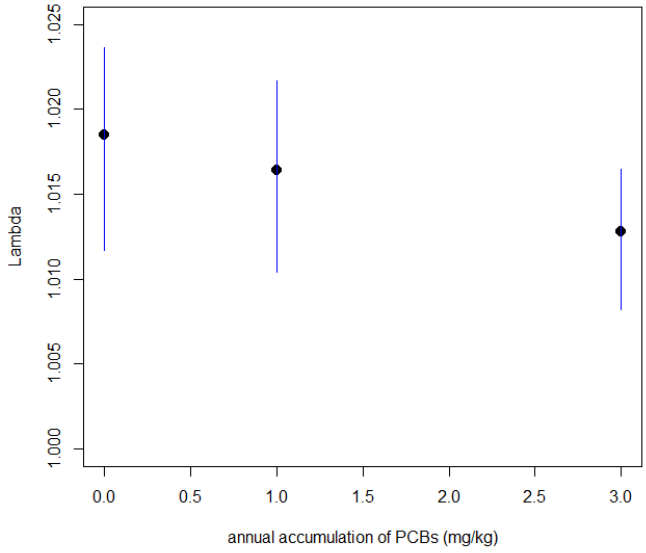

Figure 2. NRKW - Change in lambda with different PCB annual accumulation concentrations (mean and 95% confidence intervals) in model simulations with effects of PCBs on calf survival only.

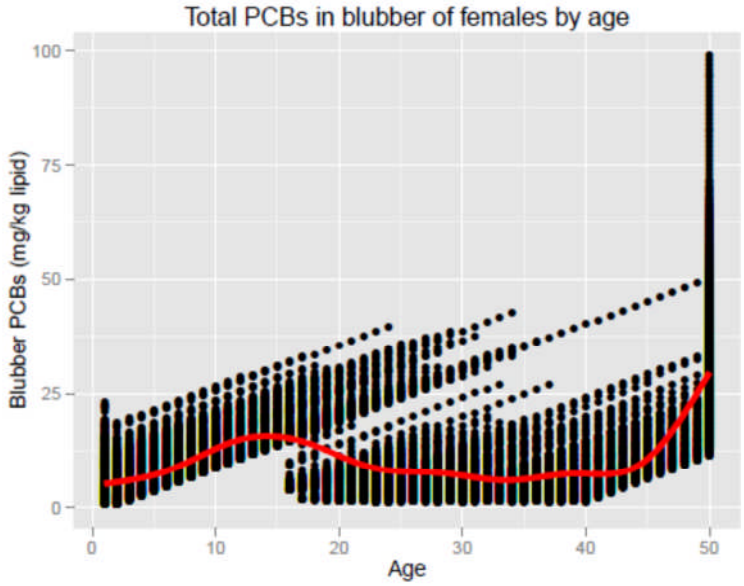

Figure 3. NRKW - Estimated concentration of PCBs in the blubber of females by age. The red line shows the mean for each age class. Concentrations in juveniles increase until females recruit into the

population, decline as females depurate their stored contaminants through lactation and increase again in senescent females.

Effect of PCBs on calf survival and immunity

In the second set of 50 100-year simulations, PCB effects on immunity were also included in the model. In these scenarios, the user can set the proportion of the population additionally exposed to a pathogen. Any baseline mortality due to infectious disease is already accounted for in the survival estimates for the starting population, so in the case of estimating the effects of PCBs on the population through modified immunity, it is assumed that an additional pathogen is then introduced into the population.

The resulting estimated lambdas from simulations with 1 and 3 mg/kg annual accumulation of PCBs and 5% of the population exposed to a low virulence pathogen are shown in Fig. 4. The potential population growth rate decreased by up to 1% per annum at the higher accumulation rate (1 mg/kg mean potential growth rate = 1.0129, 95% confidence interval 1.0178, 1.0087; 3 mg/kg mean potential growth rate = 1.0096, 95% confidence interval 1.0155, 1.0014) but remained above stationary, with a lambda >1.0. The concentrations in the adult females were, as expected, similar to the first set of simulations at 10.47 and 30.65 mg/kg lipid weight.

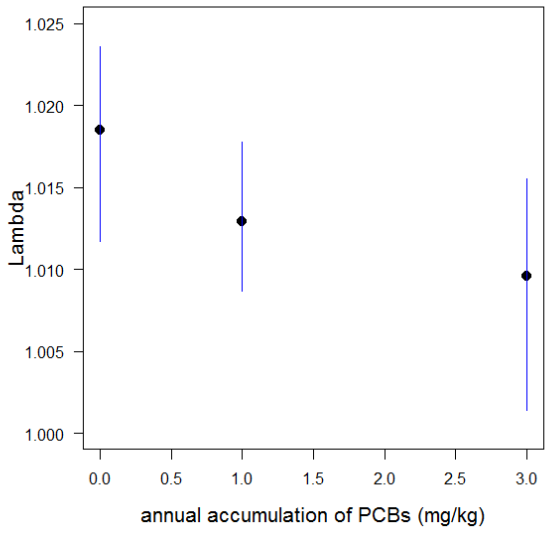

Figure 4. NRKW - Change in lambda with different PCB annual accumulation concentrations (mean and 95% confidence intervals) in model simulations with effects of PCBs on calf survival and immunity.

Additional model simulations were then carried out at 1 mg/kg annual accumulation, with a higher proportion of the population, 20%, exposed to a low virulence pathogen. This caused the population to decline with a mean lambda from years 65 to 90 = 0.9985 (95% confidence limits 0.9866, 1.0112, mean PCBs in adult females = 10.34 mg/kg lipid weight). This represents about a 2% decrease in potential population growth compared to the baseline population.

With a higher virulence pathogen and 5% of the population exposed the potential growth rate between years 65 and 90 was 1.0014 (95% confidence limits 0.994, 1.0067) with a mean concentration of

PCBs in the blubber of adult females = 9.94 mg/kg lipid. This represented ~2% decline in the potential population growth and caused the population growth to stabilise.

A similar result was observed when the proportion of the population exposed to a high virulence pathogen increased to 20%. The population declined slightly further, by 2.5% from baseline, with a mean lambda between years 65 and 90 of 0.9933 (95% confidence limits 0.9757, 1.0117, mean PCBs in adult females = 10.85 mg/kg lipid weight).

Finally, if a 1% decline in annual accumulation of PCBs was introduced into the model after year 50 with a 5 mg/kg annual accumulation (which would represent a higher concentration in the prey historically) and 5% of the population exposed to a low virulence pathogen the potential population growth rate from years 65 to 90 was 1.0128 (95% confidence limits 1.0043, 1.0197), a 0.57% decline in potential population growth compared to the baseline but still increasing.

Southern Resident Killer Whales

The population of Southern Resident Killer Whales (SRKW) has not increased at the same rate as the NRKW population and the trend from 1975-1987 indicated that the population was increasing at approximately 1.3% per annum during that period (Olesiuk et al 1990). The population has hovered below 90 individuals since the late 1990s (Center for Whale Research, unpublished data). Thus for the purposes of this modelling approach, an initial population with a stable age structure was set up with the population parameter estimates, as suggested in Olesiuk et al (1990), shown in Table 2. This resulted in a population with a mean annual rate of increase of ~1.3% (lambda=1.0129, 95% confidence interval 1.0126, 1.0136). This is in line with Olesiuk's estimate for the above mentioned 12-year period. However, it should be noted that this population was exploited (cropped) by approximately 30% beginning in the 1960s in a live-capture fishery for display in aquaria which may result in some overestimated values of lambda relative to current conditions. But the objective here is to explore the relative magnitude of effects of PCBs on a simulated population with similar vital rates and life histories to the species of interest. The resulting baseline population trajectory with unconstrained growth (potential population growth) is shown in Fig. 5.

Table 2. Parameters and vital rates used in the starting population for modelling the effect of PCBs on southern resident killer whales

Parameter	Value
Calf survival	0.97
Juvenile survival	0.98
Reproductive female survival	0.99
Post reproductive female survival	0.96
Fecundity	0.18
Maximum age (life expectancy)	50
Age at sexual maturity	14
Post reproductive age	38
Total population size	65

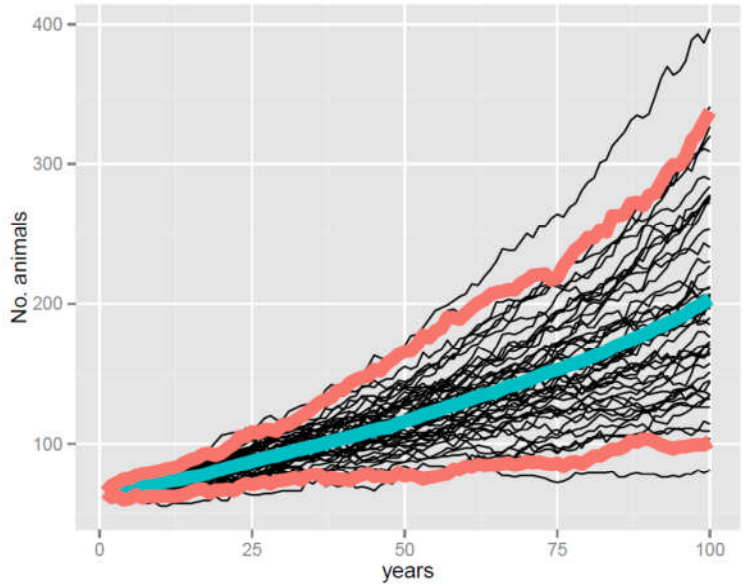

Figure 5. Baseline population representing SRKW, blue line = mean population growth, red lines = 95% confidence intervals

Effect of PCBs on calf survival

The simulations carried out were largely the same as for the NRKW above. However, the empirical data suggests that this population is exposed to higher levels of PCBs than the NRKWs (Ross et al, 2000). Thus higher annual accumulations were simulated, between 1 and 5 mg/kg per year, as shown in Fig. 6.

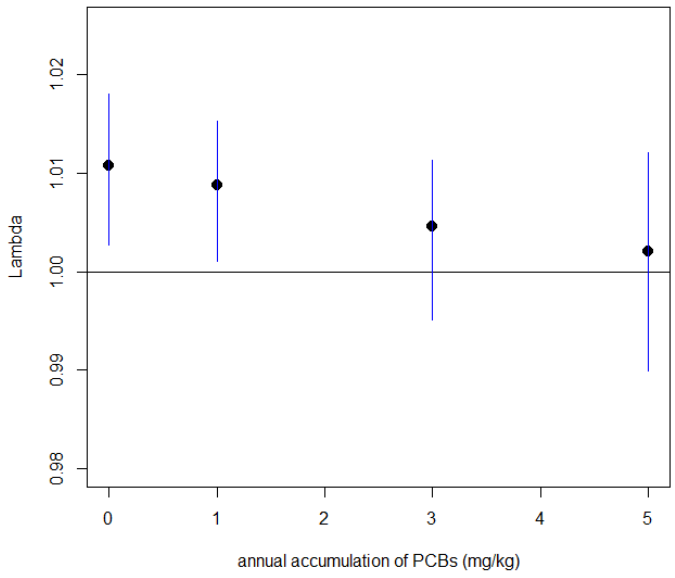

Figure 6. SRKW - Change in lambda with different PCB annual accumulation concentrations (mean and 95% confidence intervals) in model simulations with effects of PCBs on calf survival only. The horizontal line represents lambda = 1.0, a stationary population.

In these scenarios the population is still increasing, but the potential growth rate (calculated from years 65 to 90 as above) was reduced by between 0.2 and 0.9%. At annual accumulations of 5 mg/kg the population is approaching stationary conditions and at rates >5 mg/kg the model suggests the population will start to decline (results not shown). However, the model outputs suggest that accumulations are unlikely to be very much higher than ~5 mg/kg because at this rate the mean level of total PCBs in the adult females was 52.18 mg/kg lipid weight. This is in line with the small amount of published data for adult female SRKWs of ~ 55 mg/kg lipid weight (n=2, Ross et al 2000).

Effect of PCBs on calf survival and immunity

When immune effects were also included in the model simulations, as expected the potential population growth rates are affected more markedly. At 1 mg/kg annual accumulation with 5% of the population exposed to a low virulent pathogen the mean lambda was estimated at 1.0074 (95% confidence limits 0.9982, 1.0155), 0.34% lower than the baseline potential growth rate. At 5 mg/kg the potential growth rate then fell to <1.0 (0.9998, 95% confidence interval 1.0065, 0.9868), indicating a declining population. Again this accumulation resulted in a mean concentration of total PCBs in the adult females of 50.97 mg/kg (Fig. 7).

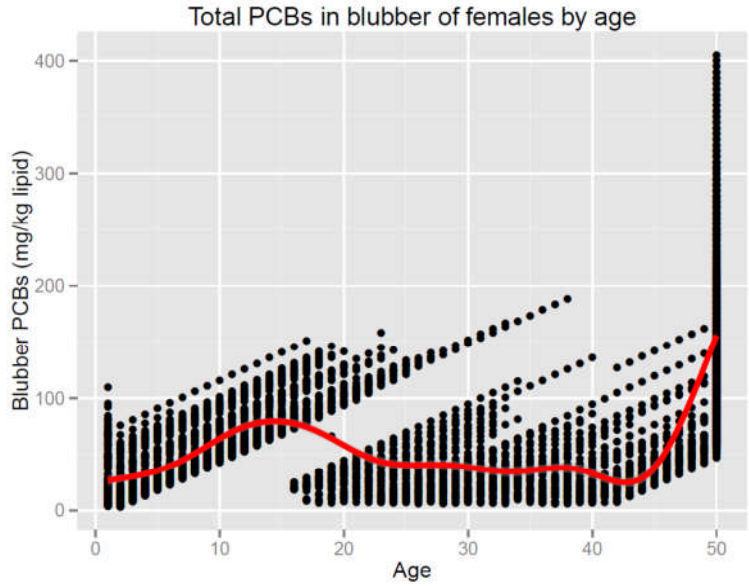

Figure 7. SRKW - Estimated concentration of PCBs in the blubber of females by age. The red line shows the mean for each age class. Concentrations in juveniles increase until females recruit into the population and again in senescent females.

When a higher proportion of the population was exposed to a low virulent pathogen (20%) the potential growth rate estimates also indicated this would result in a declining population (mean lambda = 0.9884, 95% confidence interval 0.9694, 1.0042). A similar result was observed with a higher virulent pathogen at the lower exposure proportion of 5% of the population (mean lambda = 0.9906, 95% confidence interval 0.9714, 1.0034). All scenarios at 5mg/kg accumulation led to ~ 50 mg/kg in the adult female population.

Finally when 5mg/kg annual accumulation with 5% of the population exposed, the population became stationary (between years 65 and 90 of the simulations) with a 1% per annum reduction in annual accumulation of PCBs (mean lambda = 1.006, 95% confidence interval 0.9956, 1.014).

Discussion

This analysis explores the potential effect of PCB exposure and uptake on the two populations of killer whales inhabiting the coasts of British Columbia and Washington State. In the first analysis, when only effects of PCBs on calf survival were included, the simulations suggested that the population of NRKW would not be significantly affected by PCBs at their current level of exposure, although their potential population growth rate could be depressed by around 0.2%. An annual accumulation of total PCBs of ~1 mg/kg resulted in levels in the females comparable to the empirical data (<10 mg/kg total PCBs, Ross et al 2000, Ylitalo et al 2001) although more recent data would confirm whether these concentrations are representative of the current situation. When effects on immunity were additionally included at the same annual accumulation of 1mg/kg, coupled with 5% of the population being exposed to pathogens, the potential population growth rate remained at approximately 1% or was stationary. However, when a higher proportion of the population was exposed to pathogens (20%) then the population trajectories showed a potential for decline and an estimated lambda of <1.0.

For the SRKW population, although its population was slightly increasing during the 1970s and 1980s (Olesiuk et al. 1990), it was not increasing as rapidly as the NRKWs and its abundance has remained largely stable, albeit with a notable decline in the 1990s (NOAA Fisheries, 2014). In the model simulations for this population a higher accumulation of 5 mg/kg per year was required to produce concentrations in the adult females that were similar to the published empirical data (~ 50 m/kg Ross et al 2000). At this level, with considering effects only on calf survival, the population was likely to be stationary but some simulations suggested a decline with lambda < 1.0. And in all scenarios where immune effects were also considered, at the 5 mg/kg annual accumulation level, potential population growth fell to lambda <1.0, suggesting that PCB exposure could result in a declining population at various levels of pathogen exposure and with low or higher virulence pathogens being introduced into the population. However, of some interest was that if, after year 50 of 100 years of simulated populations, the accumulation of PCBs declined by 1% per annum, it would result in a stationary population with lambda ~ 1.0.

This modelling approach has been taken to assist in assessing the risks of PCB exposure to killer whale populations under various exposure conditions. However, it should be remembered that not all the drivers of population change have been considered and that these simulations are likely to represent worst-case scenarios with regard to PCB exposure. The concentration (dose) response functions embedded in the model, which modify the survivorships based on blubber levels of total PCBs, are based on laboratory animal models that are particularly sensitive to the effects of PCBs. In addition, effects of PCBs on fecundity are not considered here. In addition, density dependence is not taken into account and thus the results should be interpreted as impacts on *potential* rather than realised population growth. Interactions with other stressors (e.g. anthropogenic noise, which can reduced prey acquisition) and disease agents, historical exploitation of the population and prey availability for example would all need to be considered alongside the contaminants as factors affecting vital rates and therefore population dynamics.

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