Title: Predicting global killer whale population collapse from PCB pollution

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One Sentence Summary:

Polychlorinated biphenyls threaten killer whales

Abstract:

Killer whales (*Orcinus orca*) are among the most highly polychlorinated biphenyl (PCB)-contaminated mammals in the world, raising concern about the health consequences of current PCB exposures. Using an Individual Based Model framework and globally available data on PCB concentrations in killer whale tissues, we show that PCB-mediated effects on reproduction and immune function threaten the long-term viability of >50% of the world's killer whale populations. PCB-mediated effects over the coming 100 years predicted that killer whale populations near industrialized regions, and those feeding at high trophic levels regardless of location, are at high risk of population collapse. Despite a near global ban of PCBs over 30 years ago, the world's killer whales illustrate the troubling persistence of this chemical class.

Main Text:

The widespread industrial use of polychlorinated biphenyls (PCBs) during the 20th century led to ubiquitous contamination of the biosphere, with significant harm among different wildlife populations (*1*). PCBs are toxic anthropogenic compounds shown to impair reproduction, disrupt the endocrine and immune systems, and increase the risk of cancer in vertebrates (*2*, *3*). While declines in PCB concentrations in the environment were evident after regulatory implementations (*4*), improvements were short-lived and PCB levels have remained essentially constant in many species since the 1990s (*5*). Still today, PCB concentrations are exceedingly high in the tissue of high trophic-level killer whales (*Orcinus orca*) and other dolphin species (*5*, *6*). It has been suggested that high PCB concentrations in killer whales may be contributing to observations of low recruitment and population decline, potentially leading to local extinctions (*5*, *7*). To date,

only one study, focusing on resident killer whales in western Canada, has investigated population risk from PCB exposure (*8*). Exposure modelling predicted protracted health risks in these resident populations over the next century, underscoring the vulnerability of this long-lived species to PCBs (*9*). With many killer whale populations facing significant conservation pressures, there is an urgent need to assess the impact of PCBs on global killer whale populations.

We compiled available data on blubber PCB concentrations (ΣPCBs, mg/kg lipid weight [lw]) in killer whales from populations around the world and compared these to established concentration-response relationships for reproductive impairment and immunotoxicity-related disease mortality using an Individual-Based Model framework (*8, 10*). This model incorporates published killer whale fecundity and survival data to construct a stable age-structured baseline population. The model then simulates the accumulation and loss of PCBs in blubber through placental and lactation transfer to the fetus and calf as well as prey ingestion after weaning. Simulated PCB concentrations are then evaluated against concentration-response relationships for calf survival and immune suppression. Immunity is linked to survival probability using relationships between immune suppression and disease mortality (*11*). We then forecast the predicted effects of PCB exposure on killer whale population growth around the world over the next 100 years.

PCB concentrations in killer whales around the world reflect proximity to PCB production and usage, as well as diet and trophic level (Fig. 1, Table S1). Global PCB production (1930 to 1993) was estimated between 1 and 1.5 million tonnes, and mostly occurring in the USA (~50%), Russia (~13%), Germany (~12%), France (~10%) and the UK (5%) (*12*, *13*). The global manufacture of PCBs corresponded well with the observed pattern of PCB levels in killer whale populations, which ranged widely from lowest values in Antarctica, <10 mg/kg lw (*14*), to values

above 500 mg/kg lw in individuals near the highly industrialized areas of the Strait of Gibraltar, the UK, and the NE Pacific (*5*, *15*, *16*). Diet is an important contributor to PCB accumulation in killer whales via biomagnification across trophic levels, resulting in sharp differences between populations feeding on marine mammals, tuna (*Scombridae*) and sharks (*Selachimorpha*) to those feeding on lower trophic level fish (Fig. 1; Table S1). This is exemplified in the NE Pacific where marine mammal-eating Bigg's killer whales carry 10–20 fold higher PCB burdens compared to fish-eating northern residents, despite sharing the same coastline (*15*, *17*). Overall, females exhibit lower blubber PCB levels than males due to maternal sequestration to young during foetal development and lactation (*18*, *19*). Exceptions have been reported in the most highly PCB-contaminated populations, including the UK, Strait of Gibraltar (*5*), and Bigg's individuals in the NE Pacific (*17*), suggesting that PCBs may be limiting successful reproduction with the consequence of reducing the maternal loss of PCBs.

Model forecasting over the next 100 years shows the significant potential impact of PCBs on population size and long-term viability of long-lived killer whales around the world (Fig. 2). Killer whale populations with similar PCB levels were grouped together and assigned to exposure groups (Fig 2C, D, Table S1, *10*). The modelled reference (unexposed) population grew by 141% (interquartile range ($25/75^{th}$) = 96.3–176.5%) over the 100-year simulation period. The least contaminated populations (group 1) included Alaskan residents, Antarctica type C, Canadian Northern residents, Crozet Archipelago, Eastern Tropical Pacific, and Norwegian populations. These are estimated to accumulate 1 mg/kg lw of PCBs per year, resulting in median blubber concentrations of 7.9 (4.7–14.0) mg/kg lw and effects causing a population decrease of 8.8% (4.1–25.3%) or 15.4% (3.5–25.2) relative to the reference population for reproductive effects alone or combined reproductive and immune effects, respectively. However, while relative population-

level effects were observed for these low exposed populations, the model still predicts a net doubling in their population size over 100 years (Fig. 2C, Fig. S2-S3). Annual PCB accumulation rates of 3, 6, 9, 15, 18, and 27 mg/kg are represented by exposure groups two through seven, which have incrementally greater blubber PCB levels (Fig. 2C, Table S1). Alaskan offshore, Faroe Islands, and Iceland whales (group 2) have similar PCB burdens (13.9–41.5 mg/kg lw) and are predicted to have modest population growth over the 100-year simulation period, albeit at reduced growth relative to the reference population; modelled PCB effects on reproduction alone or in combination with immune suppression resulted in a population reduction of 22.6% (14.0–38.3%) or 40.5% (32.6–48.7%). Alaskan transient and Canadian Southern resident populations have similar PCB burdens (group 3: 28–83 mg/kg lw), and PCB effects are predicted to inhibit population growth or cause a gradually decline of ~15% (4.3–33.9%) for reproductive or combined effects, respectively. These represent median reductions of 54.7 and 64.7% relative to unexposed populations. Greenland, Canary Islands, Hawaii, Japan, Brazil, Northeast Pacific Bigg's, Strait of Gibraltar, and UK populations all possess PCB levels above 40 mg/kg lw (Fig. 2C), and this level of exposure is predicted to cause population declines at various rates depending on the exposure group. Populations of Japan, Brazil, NE Pacific Bigg's, Strait of Gibraltar, and UK are all tending towards complete collapse in our modelled scenarios.

To quantify and compare the global risk of PCB exposure in killer whales, population trajectories from the model were used to calculate potential annual population growth rates (λ). The achievable growth rates, incorporating combined PCB effects on both reproduction and immune function, were at or below the growth threshold ($\lambda=1$) for 10 of the 19 populations for which information on PCB exposure is currently available (Fig. 2D and Table 1). These results suggest that chronic exposure to persistent PCBs has the potential to impact long-term population

viability in over half of all studied killer whale populations. Of these, Alaskan transient and Canada Southern resident populations are at moderate risk of population-level effects (λ=1), while Brazilian, NE Pacific Bigg's, Canary Islands, Greenlandic, Hawaiian, Japanese, Strait of Gibraltar, and the UK populations are at high risk of collapse over the next 100 years. The model predicted low PCB risk and stable population growth (λ>1) for the remaining nine populations (Fig. 2D and Table 1).

Our global assessment of PCB-related effects on the long-term viability of killer whale populations represents a fundamental advancement in our understanding of population impacts from chronic exposure to these legacy chemicals in a long-lived marine apex predator. More than 35 years after the onset of the ban of PCBs, killer whales still have PCB concentrations reported to be as high as 1,300 mg/kg lw (*24*). Killer whales once thrived in all oceans of the world, but only those in the less contaminated waters of the Arctic and Antarctic today appear to be able to sustain growth (Table 1) (*7*, *25*). We had no PCB data for killer whales in the Gulf of Mexico, but even before the Deep Water Horizon oil spill in 2010, estimates for killer whales in the region are consistent with a progressive population collapse from 277 individuals in 1991–1994, 133 in 1996–2001, 49 in 2003–2004, and only 28 in 2009 (*26*). Prey switching from low to high PCB-contaminated prey sources (e.g. fish to seals) has significantly increased PCB exposures in some killer whale populations like Northeast Scotland (UK) and Greenland that are now predicted to collapse (*27*, *28*). Prey switching is likely a function of prey availability as fish stocks and seal populations fluctuate over time (*27*, 28). Our finding that a single chemical class (PCBs) may represent a significant conservation threat to killer whales around the world raises concerns about the potential for other persistent contaminants to generate additional toxic injury in long-lived, high trophic level aquatic species.

The status-quo efforts to protect killer whales from conservation threats are likely to be impeded because PCBs have remained at levels associated with adverse health effects in at-risk populations over the past decades (*5*, *7*, *9*). Concerted efforts beyond those listed under the Stockholm Convention on POPs are urgently needed to reduce PCB exposure in vulnerable wildlife populations. It is estimated that more than 80% of global PCB stocks are yet to be destroyed, and at present rates of PCB elimination, many countries will not achieve the 2025 and 2028 targets as agreed upon under the Stockholm Convention on POPs (*29*). Although killer whale populations face other anthropogenic stressors such as prey limitation and underwater noise (*25*), our assessment here clearly demonstrates the high risk of collapse for many killer whale populations as a consequence of their PCB exposures alone.

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Supplementary Materials:

Materials and Methods

Figures S1-S3

Tables S1-S2

References (30-*56*)

Fig. 1. Global PCB concentrations in killer whales. A. Conceptual model of PCB bioaccumulation and magnification leading to elevated PCB concentrations in killer whale populations. **B**. Global overview of PCB concentrations in killer whale blubber. Light and dark green circles represent males and females, respectively. Also shown is population density-normalized cumulative global usage of PCBs per country from 1930 to 2000 (*12*). Number labels indicate populations with measured PCB concentrations (Table S1).

Fig. 2. Simulated killer whale population size in response to reproductive and immune effects of PCB exposure. A. Calf survival as a function of maternal adipose PCB lipid weight concentration. **B.** Immune suppression as a function of blubber PCB lipid weight concentration. **C.** Simulated effect of PCB exposure on population size (% initial size, N_0) of killer whales over the next 100 years. Simulations include the unexposed reference population (**black**), effects on reproduction (**red**), and combined effects on reproduction and immunity (**blue**). Bold lines and shading represent the median and interquartile range. Each plot represents a different PCB exposure group noted by the interquartile range of PCB concentrations in each panel (*10*). **D.** Annual population growth rates (λ) for modelled populations according to exposure group.

Table 1. Global assessment of population-level risk from PCB exposure.

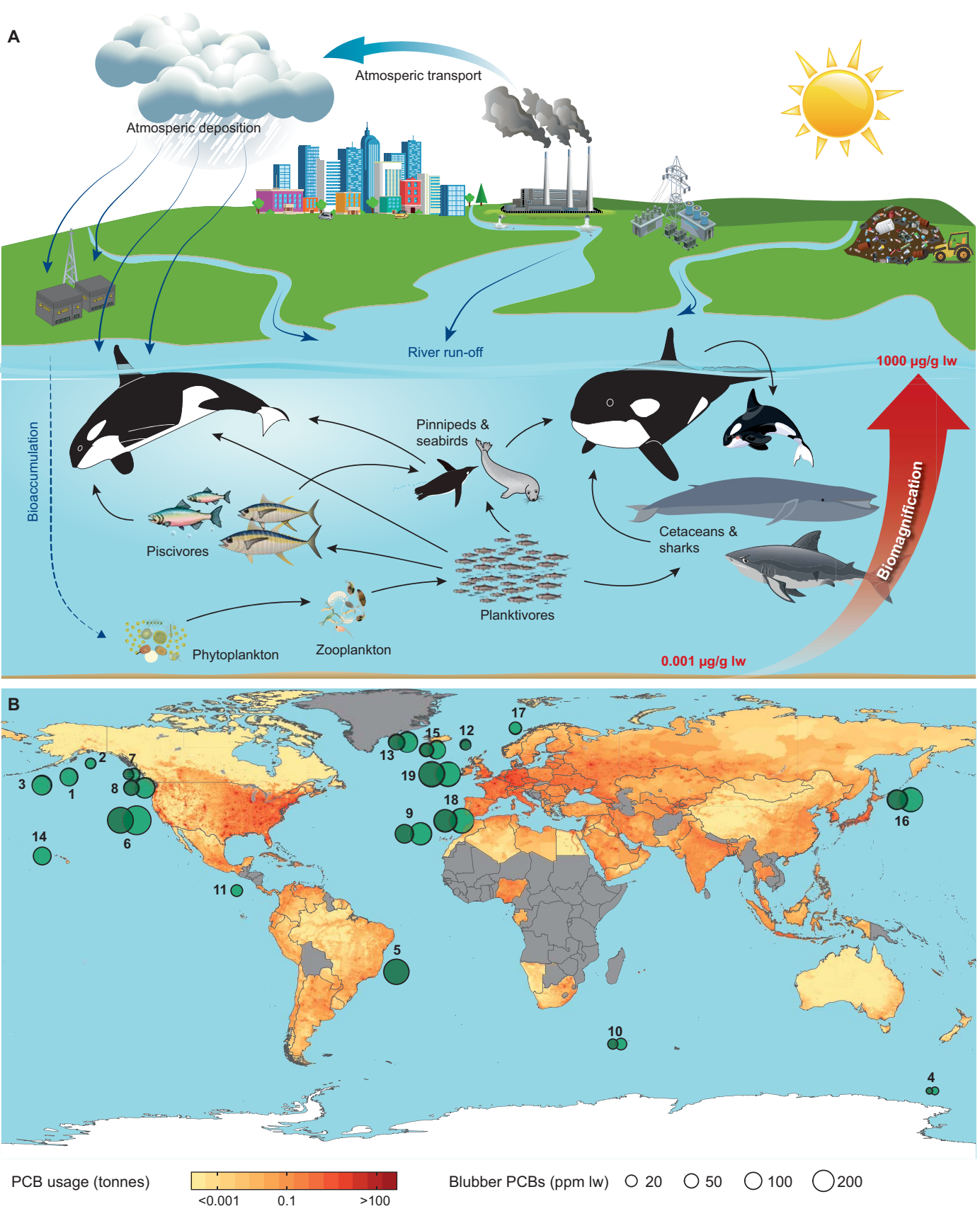
PCB risk	*Population*	*Location*	*Population size*	*Protection status*
Low (λ>1)	Alaska offshore	North Pacific	>200[†]	none[†]
	Alaska resident	North Pacific	2347[†]	none[†]
	Antarctica type C	Southern Ocean	unknown	unknown
	Northeast Pacific North resident	Northeast Pacific	290[‡]	threatened[‡]
	Crozet Archipelago	South Indian Ocean	37-98[§]	unknown
	Eastern Tropical Pacific	Tropical Pacific	8500[†]	unknown
	Faroe Islands	Northeast Atlantic	unknown	unknown
	Iceland	North Atlantic	376[¶]	NA[¶]
	Norway	Northeast Atlantic	500-1100[£]	unknown
Moderate (λ=1)	Alaska transient	North Pacific	587[†]	none/depleted[†]
	Canada South resident	Northeast Pacific	78[‡]	endangered[‡]
High (λ<1)	Brazil	Southwest Atlantic	unknown	unknown
	Northeast Pacific Bigg's	Northeast Pacific	521[†]	none[†]/threatened[‡]
	Canary Islands	Atlantic Ocean	unknown	unknown
	Greenland	North Atlantic	unknown	none
	Hawaii	Tropical Pacific	101[†]	none[†]
	Japan	Northwest Pacific	unknown	unknown
	Strait of Gibraltar	Mediterranean	36[¥]	vulnerable[¥]
	United Kingdom	Northeast Atlantic	≤9[Ψ]	none

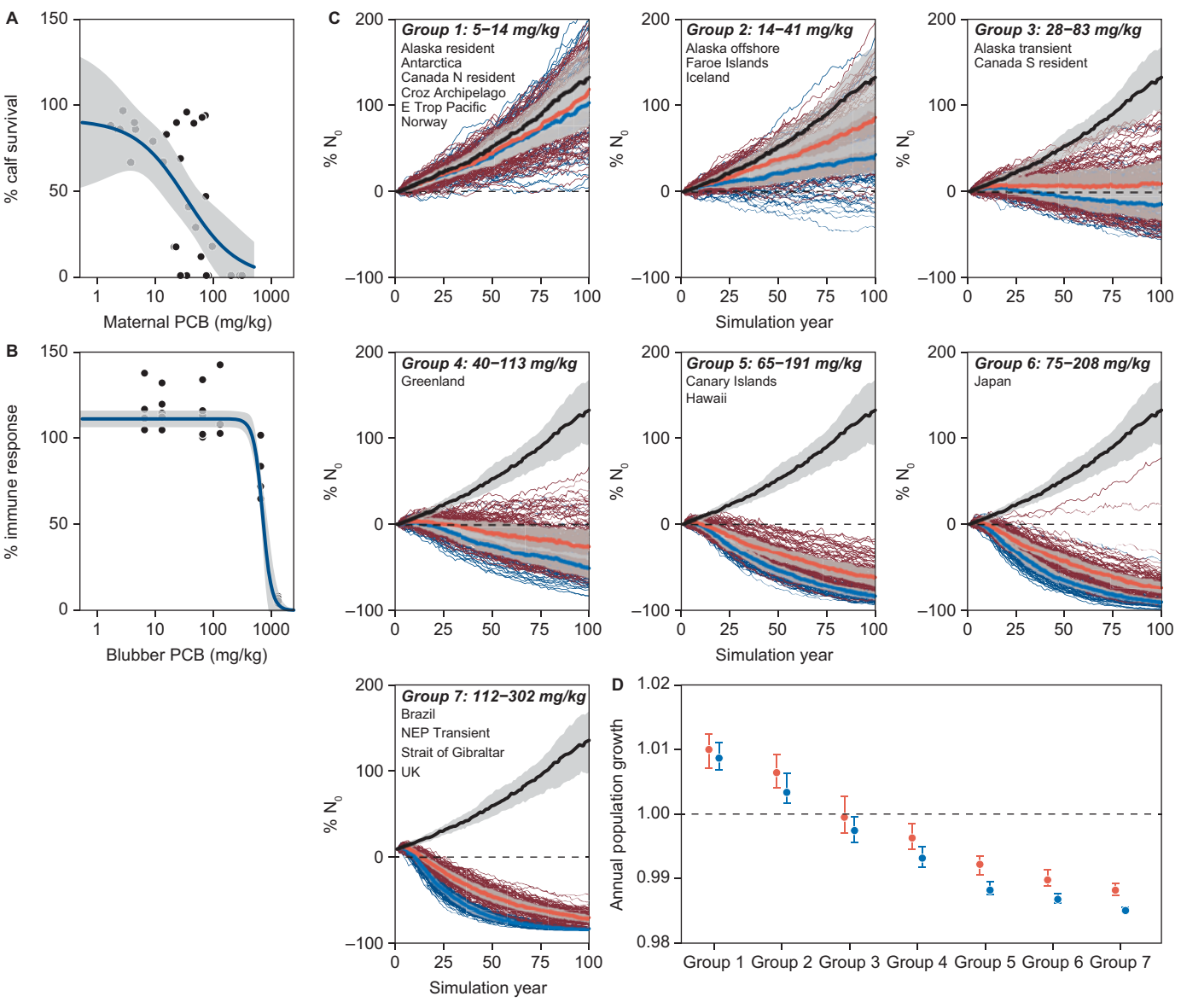
Risk categories were set based on predicted growth rates (λ) and significant difference using a one-sample t-test against a reference of no growth (λ=1): low risk (λ>1, little to no effect on population growth), moderate risk (λ=1, stagnant population growth), high risk (λ<1, population decline).

[†] National Oceanographic and Atmospheric Administration (NOAA) stock assessment reports (http://www.nmfs.noaa.gov/pr/sars/species.htm#smallwhales); AT1 transients in Alaska are a subgroup considered depleted under the US Marine Mammal Protection Act

[‡] Government of Canada, Species at Risk Public Registry (http://www.sararegistry.gc.ca/default.asp?lang=en&n=24F7211B-1)

[§] (*20*) [¶] (*21*) [£] (*22*) [¥] (*23*) [Ψ] (*5*)


A
Atmosperic transport
Atmosperic deposition
River run-off
1000 µg/g lw
Bioaccumulation
Pinnipeds &
seabirds
Piscivores
Cetaceans &
sharks
Biomagnification
Planktivores
Phytoplankton
Zooplankton
0.001 µg/g lw
B
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
PCB usage (tonnes)
<0.001
0.1
>100
Blubber PCBs (ppm lw)
20
50
100
200


A
% calf survival
Maternal PCB (mg/kg)
B
% immune response
Blubber PCB (mg/kg)
C
Group 1: 5–14 mg/kg
Alaska resident
Antarctica
Canada N resident
Croz Archipelago
E Trop Pacific
Norway
Group 2: 14–41 mg/kg
Alaska offshore
Faroe Islands
Iceland
Group 3: 28–83 mg/kg
Alaska transient
Canada S resident
Group 4: 40–113 mg/kg
Greenland
Group 5: 65–191 mg/kg
Canary Islands
Hawaii
Group 6: 75–208 mg/kg
Japan
Group 7: 112–302 mg/kg
Brazil
NEP Transient
Strait of Gibraltar
UK
% N0
Simulation year
D
Annual population growth
Group 1
Group 2
Group 3
Group 4
Group 5
Group 6
Group 7

Supplementary Materials for

Predicting global killer whale population collapse from PCB pollution

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- Summary of measured PCB concentrations in killer whales
- Dose-response data for PCB effects on calf survival in mink
- Dose-response data for PCB effects on immune suppression in killer whales

Materials and Methods

Global PCB usage

We used PCB usage data freely available online at https://www.nilu.no/projects/globalpcb/globalpcb1.htm to visualize the global distribution of PCBs. PCB usage reported is compiled from information on imports, exports and consumption of PCBs for individual countries and years, and here we take the cumulative usage over the history of PCB consumption, from 1930–1993 (*12*). Countries with no data on PCB usage are shaded in grey in Fig. 1B. The spatial distribution on the national scale is based on population densities within each country, which is considered a suitable surrogate for PCB usage (*12*). Population density data was taken from the Gridded Population of the World, Version 4 (GPWv4) (*30*). Population density for 2015 were used at a resolution of 30 arc-seconds, or ~1 km.

Model design

The Individual Based Model framework used in the present study was initially developed by (*31*) for PCB effects on calf survival, and later further developed by (*8*) to include effects on the immune system; see the later study for a detailed overview of the model design, structure, and published model code in R. Briefly, the model combines an individual based model of female killer whales, following individuals from birth to death, with a Leslie matrix model to predict the fate of individuals and thus potential population growth (not accounting for density dependent effects or variations in vital rates). The Individual Based Model simulates the exposure and accumulation of PCBs throughout the lifetime of female killer whales. The model takes into account accumulation and depuration processes such as prey ingestion and maternal sequestration (through placenta and milk). The resulting maternal blubber PCB concentrations are used in concentration-response functions to predict effects on calf survival and immune function (see below).

Several modifications to the original model were made for this study. Calf mortality from PCB exposure during nursing was randomly assigned a date between birth and weaning, and this date was used to adjust maternal depuration. We used immune dose-response data specific to killer whales in calculating the probability of disease mortality in immunosuppressed animals (see below for details). The vital parameters used in the Leslie matrix model were based on available data compiled from different killer whale populations (Table S2). We assigned the same set of vital parameters to all populations because little to no information was available for most killer whale populations. While we acknowledge that this has consequences for specific predictions of population fate, this approach has the benefit to allow the analysis to keep all things equal except for PCB exposure, the aspect of interest here. A different choice of parameters would affect the baseline population growth estimates, though the relative PCB effects would be similar; our results should therefore be interpreted on a relative basis.

An arbitrary starting abundance of 300 killer whales was chosen to ensure sufficient animals numbers in the population after 100 years. The relationship between PCB levels and age stabilized after simulation year 40, and thus the annual population growth rate (λ) was calculated from time on as:

$$\frac{final\ population\ size - initial\ population\ size}{initial\ population\ size} \Big/ 60\ years$$

The PCB modelling was done by assuming different annual accumulation rates, ranging and equally spaced between 1 and 27 mg/kg lw per year (Table S1). We termed these annual accumulation rates 'exposure groups' and refer to these throughout the main text. Each exposure group (theoretical population) is modelled separately and the model output includes blubber PCB concentrations throughout the full lifetime of each individual female killer whale simulated within that population. The results is an expected increasing blubber PCB concentrations as annual accumulation rates increase from 1 to 27 mg/kg/year. This range of accumulation rates was chosen to give a broad range of blubber PCB concentrations similar to what is observed in free-ranging killer whale populations. The individual data as well as population median and interquartile range (25/75th) for each modelled exposure group is shown in Fig. S1.

The range of blubber PCB concentrations modelled for the different exposure groups were used for the classification of actual killer whale populations. This was done by matching the model output to the observed distribution of measured blubber PCB concentrations reported in adult female killer whales in each population (Data S1), thus assigning each free-ranging population to a specific exposure group based on the actual level of PCBs in those animals (Table S1). For those populations where only male PCB data were reported, we applied an adult female to adult male conversion ratio of 3.8 to estimate blubber levels in females; this ratio represents the average calculated for northern residents, southern residents, and transients in the North-East Pacific as reported in (*15*), the study with the highest sample size available for both sexes and several populations. Full model simulations were run for each exposure group and the resultant achievable population growth was estimated as described above. Note that while all annual accumulation rates (1–27 mg/kg/year) were modelled, not all were assigned killer whale populations and therefore numbered exposure groups (one through seven) are reported only for the assigned accumulation rates (Table S1, Fig S1-S3).

PCB concentration-response

The effects of PCBs on population growth in the present study were assessed via influences on calf survival and immune suppression. Details on the concentration-response relationship between maternal PCBs and calf survival has been fully described elsewhere (*8*), displayed graphically in Fig. 2A, and raw data available in Data S2. The immune concentration-response relationship used here was derived from an in vitro exposure study using isolated killer whale immune cells (*32*); full details on sample collection and immune assays can be found therein and raw data available in Data S3. In brief, a complex cocktail of environmental pollutants, including most importantly PCBs, was extracted from killer whale blubber and used in exposure studies on killer whale blood derived lymphocytes to generate a concentration-response for T-lymphocyte function. The PCB concentration from the in vitro study was converted here to blubber equivalents assuming the nominal exposure concentration was equivalent to wet weight blood concentration; using a typical blood lipid content of 1% and a blood to blubber conversion factor for sum of PCBs (lw) of 1.5 (*33*), we estimated the lipid weight concentration of PCBs in blubber. A generalized linear quasi-binomial model with a logit link function was fitted to the data and used to predict the probability of immune suppression from blubber PCBs in killer whales. The resulting tissue-concentration relationship is shown in Fig. 2B, and used in the model to estimate the consequence of PCB exposure on the survivability of an infectious disease.

The association between contaminant exposure, immune suppression, and survival following pathogen exposure was derived from the extensive studies carried out by the U.S. National Toxicology Program (*11*). Those studies quantified the relationship between T lymphocyte proliferation in response to concanavalin A (Con A) stimulation and host-resistance in mice, demonstrating that changes in immune function are predictive of host outcomes. Here we followed the steps detailed in (*8*) to associate the immune concentration-response in killer whales (proportional change in T-lymphocyte response) to the immunosuppressant effect of a chemical on disease resistance. The host resistance model was assessed in this study by assuming 10% of individuals in a given population are exposed to a novel pathogen each year and the chance of survival is estimated by applying a multiplier to the probability of survival given the current PCB blubber levels. The 10% exposure choice can be viewed as somewhat arbitrary, but was chosen as the approximate average of pathogenic bacteria detection in killer whale respiratory microbiomes (*34*). Little to no information is available on yearly novel pathogen exposure in killer whales or other cetaceans. Serological surveys in other toothed whales commonly report prevalence of pathogens (viral/bacterial) in the range of 2-40% of sampled individuals, but typically between 5-20% (*35*–*37*). Our use of 10% population exposure here is therefore a realistic conservative estimate.

Supplementary Text

Population trajectories

The results of the model simulations for population growth over a 100-year period are shown for each annual exposure group for PCB effects on reproduction alone (Fig. S2) and combined reproduction and immune effects (Fig. S3). These figures follow the number of individuals in each population, from a start of 300 individuals. For both scenarios, the lowest two PCB accumulation rates do not prevent the number of individuals to increase over time; albeit growth rates are reduced relative to a control population (Fig. 2C). All the populations in the remaining annual accumulation rate groups (>3 mg/kg/year) are shown to decrease from the initial 300 individuals over time, with the rate of decrease proportional to blubber PCB levels (Fig. S2-S3).

Model Assumptions and Caveats

We acknowledge several important caveats in the present study. Due to the lack of data for each killer whale population and temporal trends, we applied the same general killer whale vital parameters to all modelled populations and assumed them constant through time. It is also likely that these baseline vital rates based on actual killer whale demographics represent already PCB exposed parameter values; nonetheless the values chosen are the best currently available and provide positive population growth estimates in our control population. Ultimately, the modelled population growth estimates should be viewed as relative, not absolute. Another limitation is that average PCB concentrations in certain killer whale populations are based on information from only few individuals. Such lack of data or skewed data towards stranded animals can introduce uncertainty in correctly assigning killer whale populations to modelled exposure groups. In most part, the risk of skewed PCB distributions is mitigated by the fact that the majority of data come from biopsied (healthy) animals for most populations and stranded animals are often killed by

traumatic causes and therefore still in good nutritive condition (*5*). In addition, little is known about population structure and foraging preferences for many of the less studied killer whale populations, resulting in potentially erroneous risk categorization here if subsets of populations are more or less exposed than others. For instance, individuals within a region may actually be part of two separate populations that feed on different prey, but we are currently lack the information to distinguish these.

Our model assumed fixed annual exposures over time, which may or may not be accurate. Killer whales, and other marine mammals, are long-lived species with rich blubber stores and a limited ability to metabolically eliminate PCBs. The result is very slow PCB elimination rates (e.g. high PCB half-life) in individual animals and populations (*9*). While marine mammal populations in certain areas in the world, particularly those that have implemented effective mitigation measures for contamination hot spots such as the USA (*38*), have undergone constant PCB declines, levels of PCBs in killer whales and other marine top predators in Europe and parts of the Arctic have stabilized over the past two decades (*5,7*). Time trend studies in marine mammals have found decreasing, stable or increasing PCB levels depending on region and ecological and dietary changes over time (*39*). Prey switching in killer whales, from fish to seals, has been reported in the North Atlantic as populations of seals and fish stocks fluctuate over time (*27, 28*), which is likely to have significant consequences for PCB exposure in these killer whales. Given the global complexity of PCB time trends in marine mammals and the likely confounding influence of dietary changes (i.e. changes in PCB exposure) over time and space, we believe the assumption of fixed temporal exposure is fair and introduces the least uncertainty, especially on a global scale exercise such as in this study. We also acknowledge different approaches to calculating sum PCB concentrations (i.e. congener composition), but this is not likely to appreciably influence our modelling since the total sum of PCBs is primarily dominated by few commonly reported congeners; inclusion of other minor constituents is not likely to have a major influence on the total load.

While the model used the best available toxicity data for PCB effects and incorporated uncertainty in the dose-response relationship, it included datasets from surrogate species and in vitro studies where interspecies/in vitro differences in PCB toxicokinetics and dynamics may introduce unquantified uncertainty in the model. However, of all chemicals, PCBs may be particularly well suited for in vitro studies because of their stability and relatively low rate of elimination. Lastly, the model only included effects of parent PCB congener compounds on reproduction and immunity in female individuals, ignoring similar effects in males, effects of PCBs on other physiological endpoints (*2*) and synergistic or antagonistic effects of PCB metabolites and the complex mixture of other environmental pollutants present in killer whale tissues. Including such complexities is currently not possible due to lack of relevant concentration-response data, but future studies could help elucidate these interactions and improve estimates of cumulative risk.

There are a number of model parameter uncertainties that we have tried to capture, however not all potential sources of error have been included. Stochasticity is included in the model through randomization of birth and survival outcomes; birth and survival outcomes are determined by whether a random number (from a uniform distribution) is less than or equal to the probability associated with that event. Uncertainty around the dose-response relationship for calf survival as well as immune suppression was included by using resampling with replacement (n=500) of the generalized linear quasi-binomial model

(logit link function) of the data. The results of these uncertainties are evident in the range of simulated individual blubber PCB concentrations (Fig. S1), survival trajectories (Fig. 2C), and population growth rates (Fig. 2D). Uncertainties that we have not accounted for include changes in vital rates over time, depuration and maternal transfer rates, density dependence, fixed temporal PCB exposure, surrogate species data, and pathogen exposure rates. Inclusion of the uncertainty associated with these parameters would increase the variability of population growth and effect estimates. Some parameters are not likely to be important, such as density dependence since most killer whale populations are small relative to historic levels. Uncertainty in most other parameters are difficult to include due to lack of empirical data. The choice of 10% population exposure to pathogens is described (*10*), but several realistic levels could have been used instead. The consequence of these choices would have somewhat predictable effects given the use of a constant dose-response relationship for immune suppression. The effect of different pathogen exposures was tested in a similar model in three cetacean species (*8*).

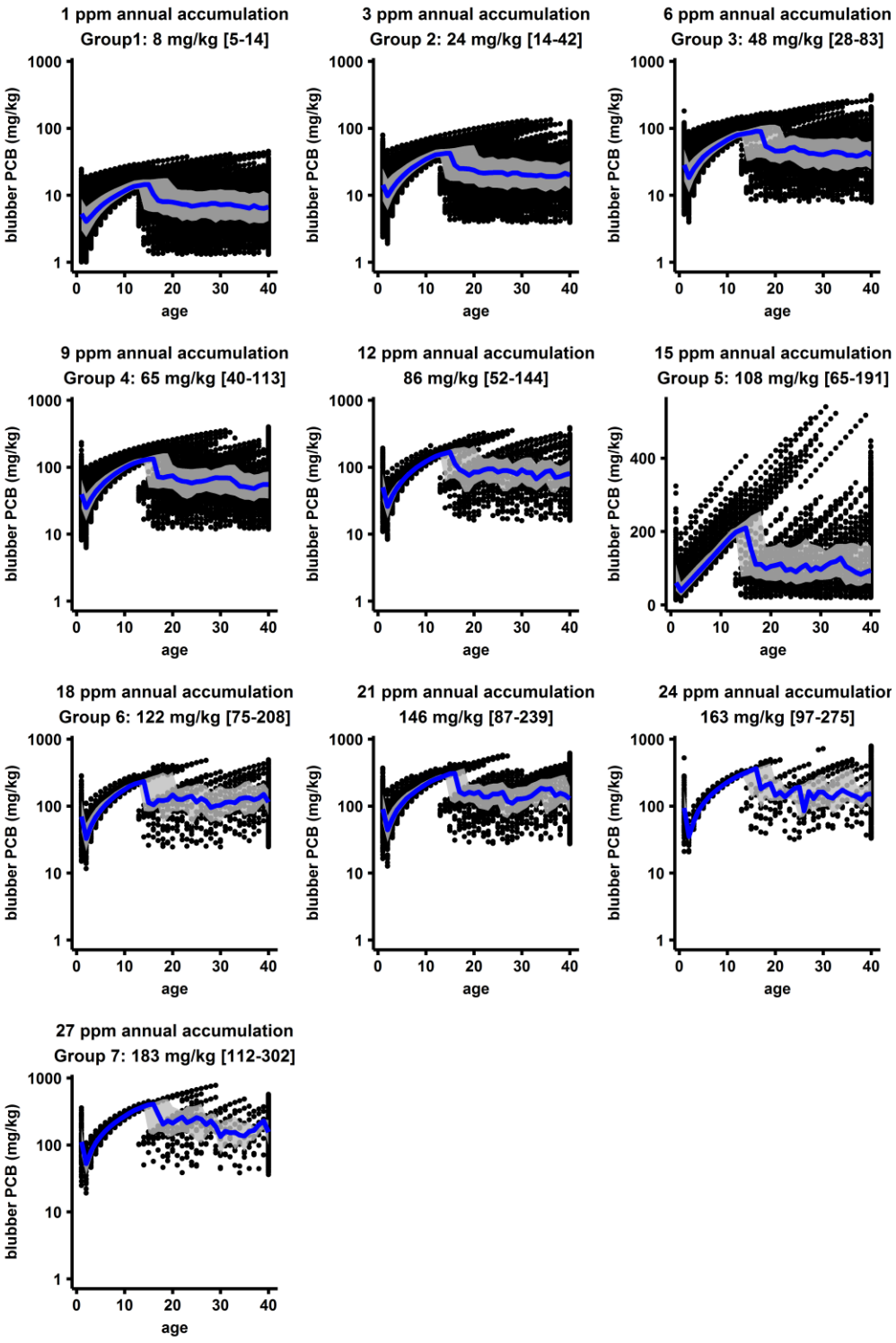

Fig. S1.

Modelled age PCB profile for different annual accumulation rates in killer whales. The bold blue line represents the median and grey shaded areas define the interquartile range (25/75th) of individual killer whale PCB age profiles.

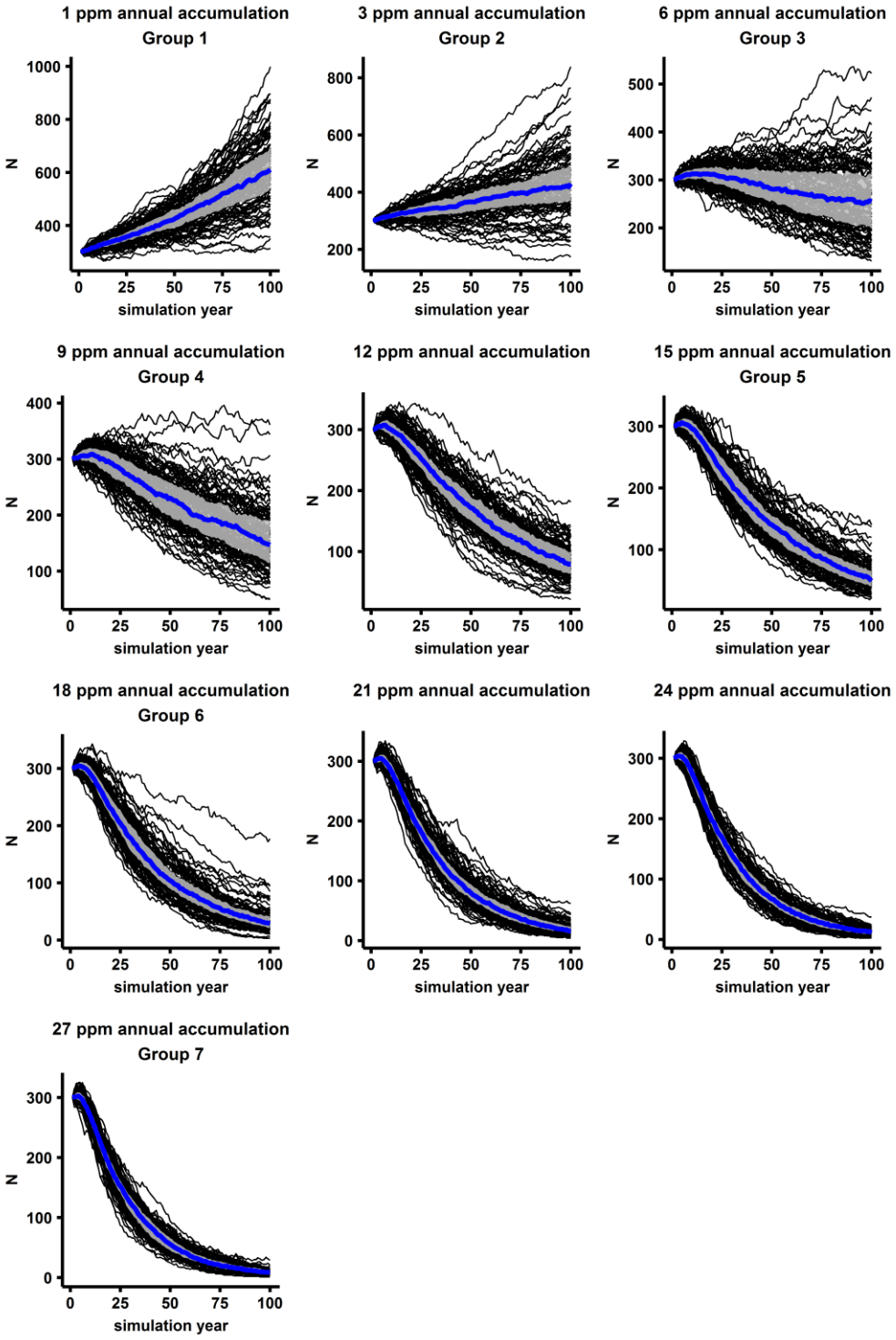

Fig. S2

Simulated reproductive effects of PCBs on population size (N, number of individuals) of killer whales over 100 years. The bold blue line represents the median and grey shaded areas define the interquartile range (25/75th) of individual killer whale trajectories.

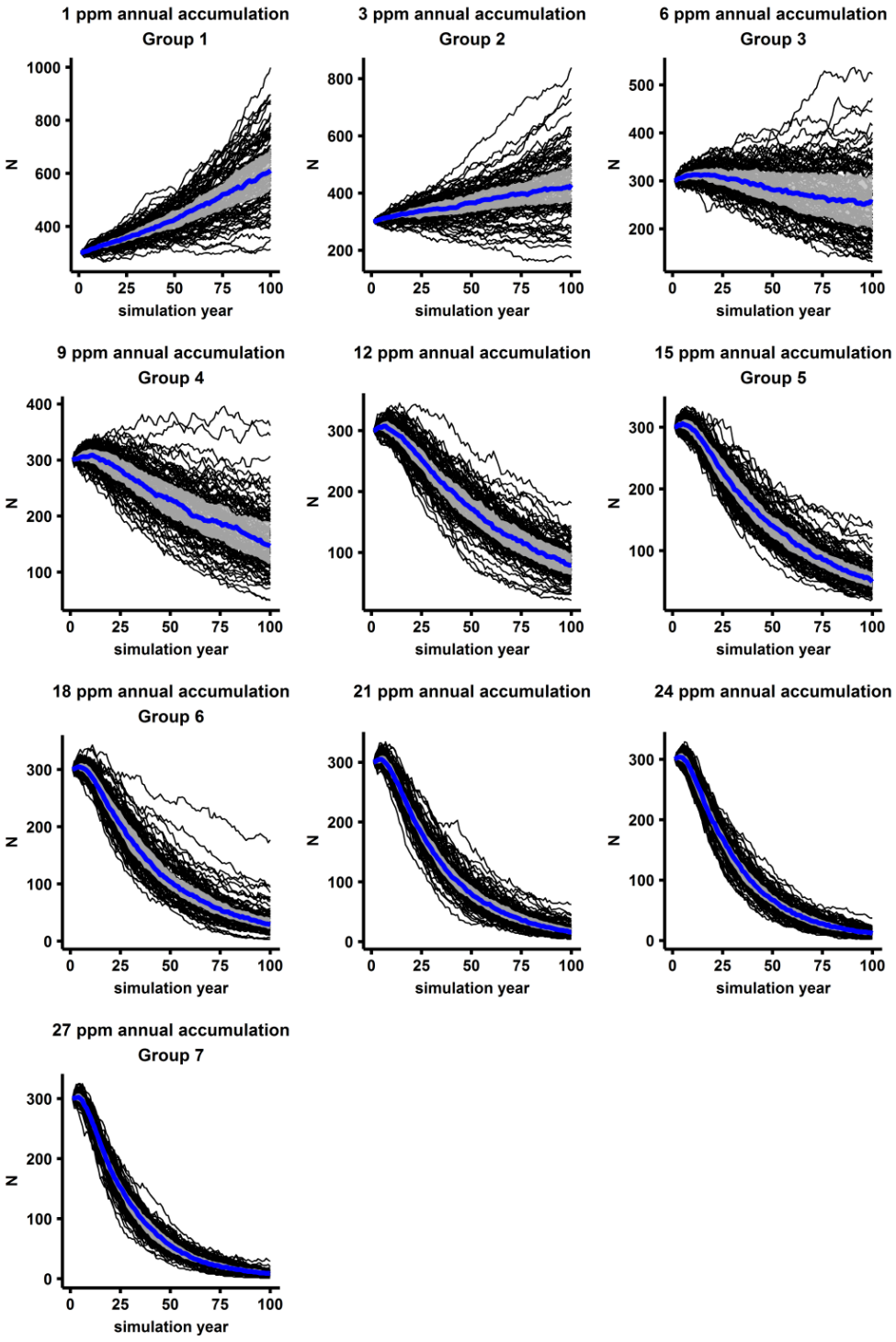

Fig. S3

Simulated combined reproductive and immune effects of PCBs on population size (N, number of individuals) of killer whales over 100 years. The bold blue line represents the median and grey shaded areas define the interquartile range (25/75th) of individual killer whale trajectories.

Table S1.

Summary of exposure groups and classification of killer whale populations according to blubber PCB concentrations.

modelled annual accumulation rate	exposure group	modelled adult blubber PCB (mg/kg lw) †	killer whale population
1 mg/kg	group 1	8 (5-14)	Alaska resident Antarctica Canada North resident Crozet Archipelago Eastern tropical pacific Norway
3 mg/kg	group 2	24 (14-42)	Alaska offshore Faroe Islands Iceland
6 mg/kg	group 3	48 (28-83)	Alaska transient Canada South resident
9 mg/kg	group 4	65 (40-113)	Greenland
12 mg/kg	--	86 (52-144)	--
15 mg/kg	group 5	108 (65-191)	Canary Islands Hawaii
18 mg/kg	group 6	122 (75-208)	Japan
21 mg/kg	--	146 (87-239)	--
24 mg/kg	--	163 (97-275)	--
27 mg/kg	group 7	183 (112-302)	Brazil Northeast Pacific Bigg's Strait of Gibraltar United Kingdom

† median and interquartile range in brackets

Table S2.

Model parameters and summary of vital population parameters in killer whales.

	Calf survival	Juvenile survival	Adult survival	Fecundity	Age at sexual maturity	Age at sexual senescence	Maximum age	Reference
Model	**0.859**	**0.951**	**0.966**	**0.169**	**11**	**40**	**80**	***this study***
Canadian S. resident	0.785	0.981	0.960	0.116	10	50		(*50*)
	0.964	0.964	0.996	0.137	16	40	100	(*51*)
Canadian N. resident	0.923	0.972	0.952	0.142	10	50		(*50*)
Canadian resident[†]	0.57	0.982	0.989	0.214	14.9	40	80	(*52*)
Norway		0.768	0.977	0.197				(*22*)
Alaska resident	0.946	0.990	0.783-0.996	0.210	12	40.5	90	(*53*)
Crozet Archipelago			0.901-0.942	0.064-0.195				(*20*, *54*)
Strait of Gibraltar		0.966	0.901-0.991	0.14	10	46		(*55*)
Seaworld - captive	0.966	0.988	0.955-1.00	0.27	7.5	40	65	(*56*)

[†]mixed resident population

Data S1. (separate file)

Summary of measured PCB concentrations in killer whales. Diet is marked as fish (F) or marine mammal (M) eating.

Data S2. (separate file)

Dose-response data for PCB effects on calf survival in mink. Data are presented graphically in Fig. 2a and explained in detail elsewhere (*8*).

Data S3. (separate file)

Dose-response data for PCB effects on immune suppression in killer whales. Data are presented graphically Fig. 2b and explained in detail elsewhere (*32*).