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SUPPLEMENTARY MATERIALS

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PERSISTENT CHEMICALS

Predicting global killer whale population collapse from PCB pollution

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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 more than 30 years ago, the world's killer whales illustrate the troubling persistence of this chemical class.

The widespread industrial use of polychlorinated biphenyls (PCBs) during the 20th century led to ubiquitous contamination of the biosphere, with substantial 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*). National and international regulatory actions succeeded in reducing PCB contamination of the environment primarily in the first decades after the bans (*4*); however, PCB concentrations remain high in many long-lived wildlife species because of their environmental persistence and efficient biological cycling (mother-calf transfer), as well as dietary shifts in some species over time to more contaminated prey (*2*, *5*). For example, 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 modeling 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 growing 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

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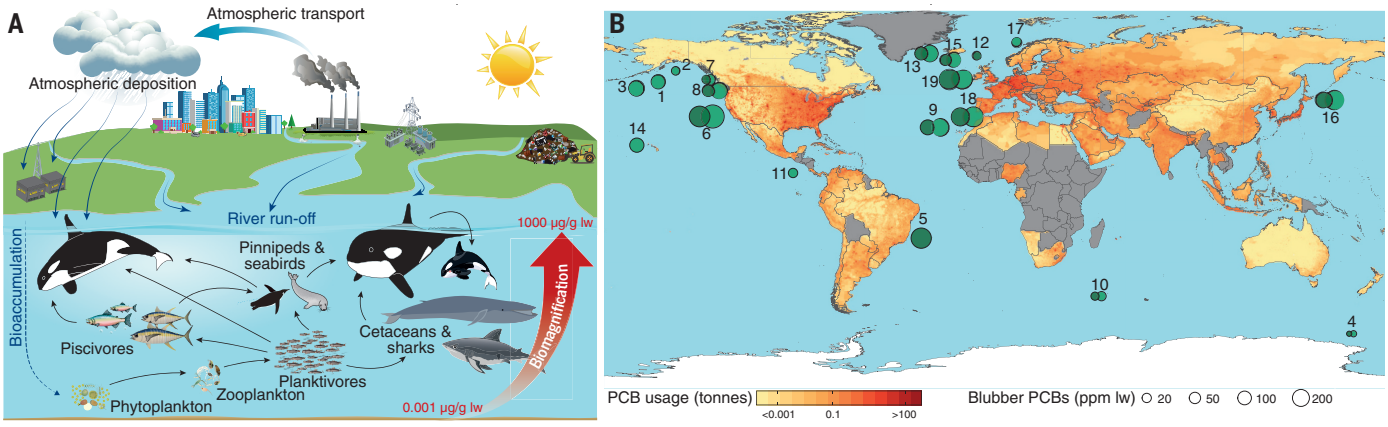

Fig. 1. Global PCB concentrations in killer whales. (**A**) Conceptual model of PCB bioaccumulation and magnification, leading to elevated PCB concentrations in killer whale populations. (**B**) Global overview of PCB concentrations in killer whale blubber (ppm, parts per million). Light and dark green 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).

Table 1. Global assessment of population-level risk from PCB exposure. Risk categories were set based on predicted growth rates (λ) and significant difference by 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).

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§	None§
	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#	None

*National Oceanographic and Atmospheric Administration (NOAA) stock assessment reports (www.fisheries.noaa.gov/species/killer-whale); AT1 transients in Alaska are a subgroup considered depleted under the U.S. Marine Mammal Protection Act. †Government of Canada, Species at Risk Public Registry (www.sararegistry.gc.ca/default.asp?lang=en&n=24F7211B-1). ‡(*27*). §(*28*). ‖(*29*). ¶(*30*). #(*5*).

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 based on 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 and table S1). Global PCB production (1930 to 1993) was estimated to be between 1 and 1.5 million metric tons (tonnes), and mostly occurring in the United States (~50%), Russia (~13%), Germany (~12%), France (~10%), and the United Kingdom

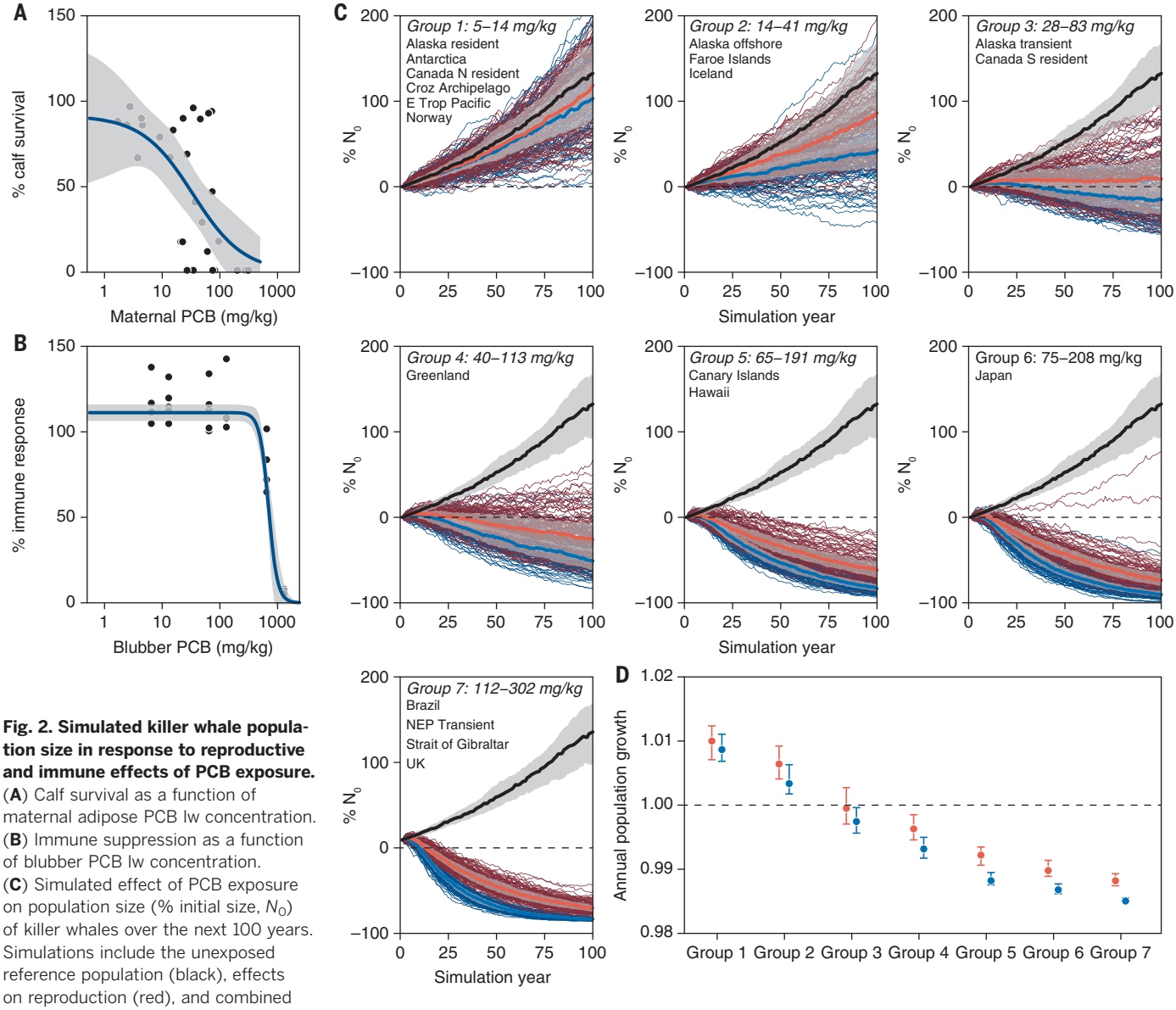

Fig. 2. Simulated killer whale population size in response to reproductive and immune effects of PCB exposure. (**A**) Calf survival as a function of maternal adipose PCB lw concentration. (**B**) Immune suppression as a function of blubber PCB lw concentration. (**C**) Simulated effect of PCB exposure 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 modeled populations according to exposure group. Symbols and error bars represent the median and interquartile range.

(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 United Kingdom, and the Northeast 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*) and those feeding on lower–trophic level fish (Fig. 1 and table S1). This is exemplified in the Northeast Pacific where marine mammal–eating Bigg's killer whales carry 10- to 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 because of maternal sequestration to young during fetal development and lactation (*18*, *19*). Exceptions have been reported in the most highly PCB-contaminated populations, including in the United Kingdom, Strait of Gibraltar (*5*), and Bigg's individuals in the Northeast Pacific (*17*), suggesting that PCBs may be limiting successful reproduction and consequently reducing the maternal loss of PCBs.

Model forecasting over the next 100 years shows the large 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. 2, C and D, and table S1) (*10*). The modeled reference (unexposed) population grew by 141% [interquartile range (25/75th) = 96.3 to 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 to 14.0) mg/kg lw and effects causing a population decrease of 8.8% (4.1 to 25.3%) or 15.4% (3.5 to 25.2%) relative to the reference population for reproductive effects alone or combined reproductive and immune effects, respectively. However, although 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 and figs. S2 and S3). Annual PCB accumulation rates of 3, 6, 9, 15, 18, and 27 mg/kg are represented by exposure groups 2 through 7, which have incrementally greater blubber PCB levels (Fig. 2C and table S1). Alaskan offshore, Faroe Islands, and Iceland whales (group 2) have similar PCB burdens (13.9 to 41.5 mg/kg lw) and are predicted to have modest population growth over the 100-year simulation period, albeit at a reduced rate relative to the reference population; modeled PCB effects on reproduction alone or in combination with immune suppression resulted in a population reduction of 22.6% (14.0 to 38.3%) or 40.5% (32.6 to 48.7%). Alaskan transient and Canadian Southern resident populations have similar PCB burdens (group 3: 28 to 83 mg/kg lw), and PCB effects are predicted to inhibit population growth or cause a gradual decline of ~15% (4.3 to 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 U.K. 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, Northeast Pacific Bigg's, Strait of Gibraltar, and United Kingdom are all tending toward complete collapse in our modeled scenarios.

To quantify and compare the global risk of PCB exposure in killer whales, we used population trajectories from the model 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 affect long-term population viability in more than half of all studied killer whale populations. Of these, Alaskan transient and Canada Southern resident populations are at moderate risk of population-level effects ($\lambda = 1$), whereas Brazilian, Northeast Pacific Bigg's, Canary Islands, Greenlandic, Hawaiian, Japanese, Strait of Gibraltar, and U.K. populations are at high risk of collapse over the next 100 years. The model predicted low PCB risk and stable population growth ($\lambda > 1$) 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 on PCBs, killer whales still have PCB concentrations reported to be as high as 1300 mg/kg lw (*20*). 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*, *21*). 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 (*22*). Prey switching from low to high PCB-contaminated prey sources (e.g., fish to seals) has been documented in some killer whale populations like Northeast Scotland (United Kingdom) and Greenland (*23*, *24*), which is likely to have important consequences for PCB exposure in these already vulnerable populations. Prey switching is likely a function of prey availability as fish stocks and seal populations fluctuate over time (*23, 24*). Our finding that a single chemical class (PCBs) may represent a substantial 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. Indeed, a long list of additional known and as yet unmeasured contaminants are present in killer whale tissues, including biologically active compounds like perfluoroalkyl acids, brominated and organophosphate flame retardants, and polychlorinated naphthalenes (*25*), and although these are less well characterized, they may contribute to reproductive and immune failure or other health endpoints not included here.

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 Persistent Organic Pollutants (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 (*26*). Although killer whale populations face other anthropogenic stressors such as prey limitation and underwater noise (*21*), 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

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