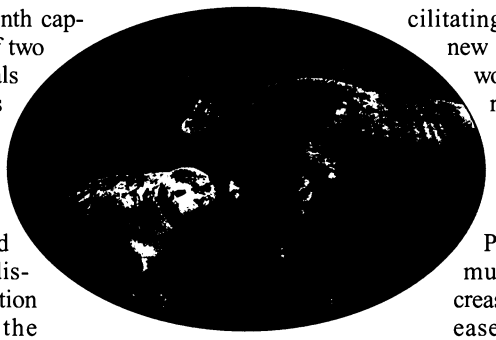
contributory role in the severity of the event.

About 20,000 European harbor and gray seals died during this event, representing up to 60% of local populations. A newly isolated, identified, and characterized *Morbillivirus* (phocine distemper virus) was assigned as primary cause of the event (*1*). However, concerns lingered that persistent organic pollutants (POPs), many of which are demonstrated immunotoxicants (including dioxins, the "dioxin-like" PCBs, and furans), played a role. We therefore carried out a 30-month captive feeding study of two groups of harbor seals in which 22 seals were fed herring from either the relatively uncontaminated Atlantic Ocean or the contaminated Baltic Sea. POPs disrupted immune function in the seals fed the Baltic Sea herring, causing diminished T cell function in vitro and in vivo and reduced natural killer cell function (*2, 3*), both of which are crucial to antivirus defenses in vertebrates.



Although not the primary cause of the harbor seal die-off in Europe in 1988, PCBs appear to have had a contributory role.

Our study seals were not directly dosed with pure PCBs, but this would have been unethical and unnecessary. Our aim was to mimic "real world" conditions for free-ranging populations of seals exposed to POPs with a carefully controlled captive feeding study, rather than to create artificial and unrealistic exposure regimes. In the case of the European seal die-off, the following lines of evidence implicate the PCBs: (i) immunotoxicity observed in our captive seals was typical of dioxin-like contaminants, not the other POPs; (ii) PCBs were the overwhelming dioxin-like chemical measured in samples taken from our study seals (93% of total dioxin Toxic Equivalency Quotient) compared with dioxins and furans; (iii) laboratory rat studies using the identical herring diets and a positive control group exposed to dioxin confirmed that the dioxin-like PCBs were largely responsible for the effects observed (*4*); (iv) an extensive laboratory animal literature underscores the immunotoxicity of the dioxin-like compounds in particular; (v) most European seal populations in 1988 had PCB levels that exceeded those found to be immunotoxic in our captive study (*2*) and would have been exposed in a manner (that is, perinatally) that would ensure a more profound immunotoxicity; and (vi) the several *Morbillivirus*-associated mass mortalities observed since 1987 have been largely restricted to the more contaminated marine mammal populations (*5*).

Human and wildlife toxicologists face the challenge of identifying specific chemicals responsible for toxic effects in cases of complex dietary exposures. Ultimately, it is the weight of evidence from laboratory, semi-field, and epidemiological studies that highlights PCBs as a significant threat to the health of wildlife and humans (*6*). Indeed, anthropogenic factors, including environmental contaminants, may be facilitating the emergence of new diseases around the world (*5*). In addition, recent studies of nursing human infants have found that even "background" levels of PCBs lead to immunotoxicity and increased incidence of disease (*7*). The valuable lessons learned from the more highly exposed wildlife species compel us to better integrate human and ecological research to assess the continued health risks of POPs to all consumers.

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* This letter does not reflect EPA policy.

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CORRECTIONS AND CLARIFICATIONS

Letters: Corrections and Clarifications (3 Sept. 1999, p. 1493). The trace in Fig. 4C reproduced in this correction of the Report "Molecular identification of a eukaryotic, stretch-activated nonselective cation channel" by M. Kanzaki *et al.* (6 Aug. 1999, p. 882) was offset slightly in the negative direction. A correct panel has been published (26 May 2000, p. 1347).

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