

cluding rhesus monkeys (1). This issue deserves further study.

However, I must disagree with Southwick's conclusion that I reject innate aggressiveness. Animals would have had no need for mechanisms to control aggression, and to repair the damage it inflicts on social relationships, if open conflict had not been pervasive. Attention to conflict resolution needs to go hand in hand with attention to the sources of aggression, genetic and otherwise.

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Cause of Seal Die-off in 1988 Is Still Under Debate

Peter Ross and his colleagues review in their letter (15 Sept., p. 1878) their findings on immune disruption by polychlorinated biphenyls (PCBs) in harbor seals, a rare example of controlled experimental research much needed in marine mammal toxicology. There is no question that PCBs can pose threats to humans and wildlife. Their letter was prompted by my comment (Letters, 16 June, p. 1965) that a 1988 die-off of seals in Europe was due to a morbillivirus epizootic. This contrasted with a report by Jocelyn Kaiser (News Focus, 21 Apr., p. 424) that implied PCB exposure was the underlying factor spurring this epizootic. I cited evidence that this generalization was inaccurate. Ross and colleagues note that my points were technically correct, but that I did not consider a broader "weight of evidence." They say that "[c]urrent scientific consensus supports the idea that PCBs played a contributory role in the event."

There seems to be, however, no such scientific consensus. This is evident in recent proceedings from two major interdisciplinary, international scientific workshops on marine mammals and contaminants (1, 2). The summary chapter (3) from a workshop in Norway sponsored by the International Whaling Commission noted the capacity of morbilliviruses to produce high mortality in immunologically naïve populations, well documented in terrestrial mammals before the synthesis of PCBs, and concluded, "at this stage it is unclear whether contaminants had a role in morbillivirus epizootics in marine mammals." The plenary chapter on morbilliviruses in marine mammals (4) stated, "given the lethal effects of morbilliviruses, it is...unlikely that organochlorine tox-

icity had anything other than a marginal effect on mortality" and "there is no evidence that [PCBs] have affected mortality or morbidity due to morbilliviral infection." This review concluded, however, that further investigation is warranted.

The group report on immunotoxicology (5) from a second workshop sponsored by the U.S. Marine Mammal Commission also noted an insufficiency of evidence to establish a cause-and-effect relation between exposure to environmental contaminants and injury to the immune system that might affect marine mammal populations. In a recent independent review (6), J. R. Geraci and colleagues observed that during the European seal die-off "there was much speculation that the deaths were the result of pollution, and this remained an obsession for the media," but they concluded that the evidence for a causal relation or compounding effect between contaminants and susceptibility to disease in marine mammals was inconclusive.

Thus, much current thinking emphasizes the virulence of morbillivirus infections rather than major roles for PCBs. This has been underscored by this year's die-off of Caspian seals, ascribed to a morbillivirus, with any role for organochlorines currently downplayed (News of the Week, "Canine virus blamed in Caspian seal deaths" by R. Stone, 22 Sept., p. 2017). A precautionary approach certainly should be taken in human and wildlife health issues, and a "weight of evidence" evaluation has a role in environmental management decisions. However, from a scientific standpoint, application of such an approach to the contributory role of PCBs in the severity of the 1988 seal die-off has generated a hypothesis that is difficult to test. Several attempts thus far have failed to find support over an alternative that fits within the bounds of existing data on morbillivirus infections. Lack of consensus has resulted.

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