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Global Warming and Increasing Population Densities: a Prescription for Seal Plagues

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Application of Ockham's Razor to data from documented mass mortalities in pinniped populations suggests that unseasonably warm temperatures and high densities of seals, and not 'invading harp seals', marine pollution and suppressed immune systems, triggered the 1988 seal plague in the North Sea.

During the summer of 1988, more than 17 000 harbour seals died along the coasts of Scandinavia and Europe (Dietz *et al.*, 1989) in the most recent mass mortality to strike a pinniped (seals, sea lions, fur seals and walrus) population (Geraci *et al.*, 1982). The ultimate cause of this 'seal plague' has been identified as a previously unknown morbillivirus (Family Paramyxoviridae), now called phocine distemper virus (PDV) (Mahy *et al.*, 1988; Cosby *et al.*, 1988). Other morbilliviruses cause distemper in dogs, measles in humans, and rinderpest in ungulates (Mahy *et al.*, 1988; Miller, 1989). Where PDV originated and why it was so virulent in harbour seals remains the subject of much speculation and controversy (Anon., 1988a; Dickson, 1988; Goodhart, 1988; Harwood & Reijnders, 1988; Holdgate, 1988; McGourty, 1988a,b; Eis, 1989; Harwood, 1989; Holt, 1989; Law *et al.*, 1989; Osterhaus & Vetter, 1989; Simons & Johnson, 1989a,b).

It has been suggested that the 1988 epizootic began with an 'invasion' of non-endemic harp seals, *Phoca groenlandica*, which transmitted PDV to previously uninfected harbour seals (Goodhart, 1988; Harwood, 1989; Miller, 1989). It has also been argued that these harbour seals may have been particularly susceptible to infection because their immune systems were suppressed by the action of environmental contaminants, particularly, polychlorinated biphenyls (PCBs) (Anon., 1988a; Dickson, 1988; Harwood & Reijnders, 1988; Holdgate, 1988; McCourty, 1988a,b; Eis, 1989; Harwood, 1989; Holt, 1989; Law *et al.*, 1989; Osterhaus & Vetter, 1989; Simons & Johnson, 1989a,b). Yet, consideration of previous mass mortalities of pinnipeds suggests that, once again (see Geraci *et al.*, 1982), other factors may have been more important, especially during the initial stages of the outbreak.

We have re-examined the major mass mortalities that have been documented in pinniped populations (Table 1). In those cases where the relevant observations were recorded, population sizes were said to be increasing or seal densities on shore were described as being unusually high. In the majority of cases, ambient temperatures were reported to be unseasonably warm.

To investigate the possible relationship between environmental temperatures, seal densities and the outbreak of epizootics, we compiled data on local mean monthly air temperatures during the years of the mass mortalities and compared these with the mean monthly air temperatures for the preceding ten years. In the five instances with available data, mean monthly temperatures preceding the mass mortalities were higher than the ten-year averages (Fig. 1). In each case, the mass mortalities began following three months when mean air temperatures were 1.0–3.0°C higher than the preceding ten-year average. Even in Iceland, where 1918, the year of their seal plague, is reported to have had the coldest winter on record (Dietz *et al.*, 1989), two of the three months immediately preceding the detection of the outbreak in June were warmer than the ten-year monthly averages (Fig. 1A).

There are no quantitative data to link environmental

TABLE 1

Population densities and environmental temperatures preceding major mass mortalities in pinniped populations, as they were originally described.

Location of outbreak	Date	Species	Population density	Environmental temperature	Pathogen/ Disease
Iceland[a,b]	1918	harbour seal, *Phoca vitulina*	nd*	extremely cold	pneumonia
Antarctica[c]	1955	crabeater seal, *Lobodon carcinophagus*	10 × normal	mild	'virus'
Alaska[d]	1978	walrus, *Odobenus rosmarus*	high	nd	none
New England[e]	1979/80	harbour seal, *Phoca vitulina*	unusually large	unseasonably warm	virus
Lake Baikal[a]	1987/88	Baikal seal, *Phoca sibirica*	nd	nd	virus[f,g,h,i]
Kattegat–Skagerrak[a]	1988	harbour seal, *Phoca vitulina*	growing population[j]	unusually mild	virus[k,l]

Sources: [a]Dietz *et al.* (1989), [b]Bárdarson (1931), [c]Laws & Taylor (1957), [d]Fay & Kelly (1980), [e]Geraci *et al.* (1982), [f]Anon. (1988c), [g]Grachev *et al.* (1989), [h]Osterhaus *et al.* (1989), [i]Likhoshway *et al.* (1989), [j]Heide-Jørgensen & Härkönen (1988), [k]Mahy *et al.* (1988), [l]Crosby *et al.* (1988).
*nd—no data provided.

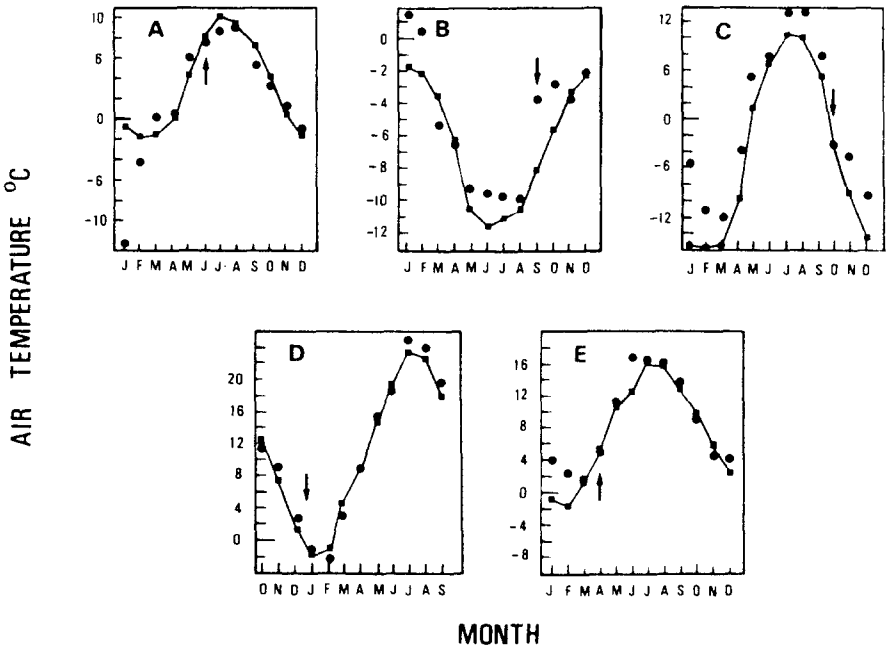

Fig. 1 Comparison of local mean monthly air temperatures during mass mortalities in the seal populations listed in Table 1 (circles), with mean monthly air temperatures for ten years preceding the outbreaks (squares). Temperature data were obtained from Ruffner & Bair (1987) (for New England and Alaska) and from the Meteorological Institute of Iceland (Iceland), the National Meteorological Archive, UK (Hope Bay, Antarctica), and the Danish Meteorological Institute (Kattegat–Skagerrak). We have been unable to obtain temperature data for Lake Baikal. A. harbour seals, Iceland, 1918; B. crabeater seals, Hope Bay, Antarctica, 1955; C. walruses, Alaska, 1978; D. harbour seals, New England, 1979; E. Kattegat–Skagerrak, 1988. Arrows indicate month in which each mass mortality began.

temperatures and densities of seals on shore prior to a mass mortality. Relevant data are, nonetheless, available for harbour seals at Sable Island, Canada (Boulva, 1973). In this population, animals usually remain in the water until air temperatures rise to about −1°C. As temperatures continue to increase, the seals begin to 'haul out' on the beaches to bask and, between −1 and 1°C, densities of animals on shore increase by orders of magnitude. They remain at high levels to at least 5.6°C (Fig. 2). Thus, if temperatures are unseasonably warm, harbour seals may haul out when they would otherwise be in the water. During times when they are normally on shore, higher temperatures may also cause them to congregate at greater densities and for longer durations than usual.

Consistently, the proportion of walruses, *Odobenus rosmarus,* on shore has been observed to increase from zero to about 100% as air temperatures increase from −25 to −20°C and densities remain high at temperatures up to about 5°C (Ray & Fay, 1968).

Contrary to recent claims (Dietz *et al.,* 1989), our assessment suggests that there is a common mechanism triggering mass mortalities in pinnipeds. We propose that high densities of pinnipeds on land, frequently mediated by unseasonably warm temperatures, set the stage for the rapid invasion of an opportunistic pathogen by increasing the availability of potential hosts. This is the most parsimonious explanation for mass mortalities in pinnipeds, including the recent seal plague that devastated European harbour seals, *Phoca vitulina,* in 1988. Such a scenario is entirely consistent with documented epizootics in other animals, including epidemics in humans (Cooper & MacCallum, 1984).

Of course, high densities alone will not necessarily result in an epizootic. This will only occur if a pathogen is present and becomes established in the population. Indeed, the walrus mortalities in Alaska were largely the result of pregnant females and pups being crushed because animals were overcrowded. No disease agent was identified (Fay & Kelly, 1980). Nonetheless, if a highly contagious disease, like phocine distemper, does become established in a population, it can easily spread to susceptible animals, either by direct contact between individuals or by airborne transmission (Andrewes *et al.,* 1978; Cooper & MacCallum, 1984). This may account for the widespread dispersal of phocine distemper during the seal plague of 1988.

The suggestion that marine pollution played a significant role in the 1988 seal plague cannot be ruled out entirely. Nonetheless, this particular epizootic began in a population that had been growing at an annual rate of about 12%, in spite of whatever contaminants might have been present (Heide-Jørgensen & Härkönen, 1988). Moreover, contaminant levels in the affected seals appeared to be lower than in the previous decade (Anon., 1988b) and the plague failed to penetrate far into the Baltic Sea where contaminant levels are high (Helle *et al.,* 1976) but seal densities are relatively low (Almkvist, 1982). Furthermore, pollution is not a common factor in mass mortalities of pinnipeds; it is unlikely, for example, to have been involved in those in Iceland in 1918 and at Hope Bay, Antarctica in 1955.

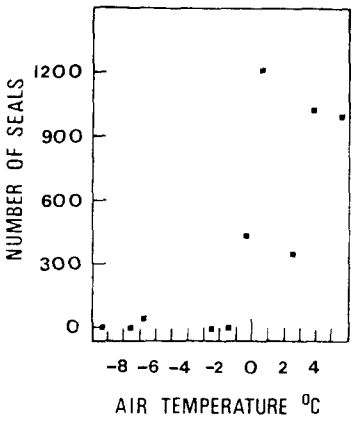

Fig. 2 Relationship between ambient air temperatures and animal abundance on shore for harbour seals on Sable Island. Plotted from data provided in Boulva (1973). This isolated population of harbour seals numbered 1200–1500 animals at the time of the study (Boulva, 1973).

Of course, if environmental contaminants (Thomas & Faith, 1985; Vos *et al.,* 1988) or exposure to unusually hot or cold temperatures (Novak *et al.,* 1989) were to induce immunosuppression, something that has yet to be observed in seals prior to an epizootic, the chances of a disease outbreak would certainly be increased.

Similarly, the notion that the seal plague of 1988 was initiated by harp seals, which carried PDV from the Barents Sea to the Kattegat–Skagerrak and infected harbour seals (Goodhart, 1988; Harwood, 1989; Miller, 1989), is not consistent with the available data. The most recent major 'invasion' of harp seals occurred in northern Norway in 1987 (T. Øritsland, pers. comm.). If these harp seals had been capable of initiating an epizootic in harbour seals, they should have done so in 1987, not 1988, especially since the typical incubation period for a morbillivirus like canine distemper is about ten days (Andrewes *et al.,* 1978). Furthermore, if harp seals were the source of PDV, the initial outbreak should have occurred in harbour seals in northern Norway and subsequently spread south. Instead, the plague began in the south (Kattegat–Skagerrak) and never reached harbour seals in northern Norway (Dietz *et al.,* 1989).

That small changes in ambient temperatures can increase the likelihood of an epizootic in pinniped populations has profound implications for the future. If we are indeed entering a period of global warming, because of natural events or the greenhouse effect, we would predict that the incidence of mass mortalities in pinnipeds will increase, at least in those species that exhibit marked changes in density in response to relatively small changes in environmental temperatures. Consistently, four of the six documented mass mortalities of pinnipeds have occurred since 1978 (Table 1), a period that includes some of the warmest years in the 20th century (Barron, 1989; Revkin, 1989). Moreover, current models of global climatic change predict that the greatest temperature difference will occur in polar and subpolar regions (Revkin, 1989; Barron, 1989), precisely where pinnipeds live (Davies, 1958; Lavigne & Kovacs, 1988).

Candidates for future epizootics include northern elephant seals, *Mirounga angustirostris,* and antarctic fur

seals, *Arctocephalus gazella*. Their numbers are increasing exponentially (Cooper & Stewart, 1983; McCann & Doidge, 1987; Heide-Jørgensen & Härkönen, 1988) and local densities could be further inflated during an unseasonably warm spell.

In contrast, it is unlikely that endangered Mediterranean monk seals, *Monachus monachus*, are threatened by PDV from North Sea harbour seals (Harwood, 1989). They are far removed from the infected area and they are currently living at very low densities (Sergeant *et al.*, 1978), being one of the most endangered mammalian species on the planet.

For related reasons, we would not have expected the seal plague of 1988 to have repeated itself during the summer of 1989 (Harwood et al., 1989), even though temperatures were again unseasonably high (air temperature data for 1989 were obtained from the Danish Meteorological Institute, Copenhagen, Denmark and The Meterological Office, Berkshire, UK). Because of the reduction in population size due to the epizootic and because many of the surviving animals had developed immunity to PDV (Harwood *et al.*, 1989), the number of potentially susceptible animals remaining in the population would likely be too small to sustain another epizootic. Consistently, only a few deaths attributed to PDV were reported in 1989.

In recent years, there have also been a number of mass mortalities involving cetaceans (whales, dolphins and porpoises). In two cases—humpback whales, *Megaptera novaeangliae*, at Cape Cod in 1987, and bottlenosed dolphins, *Tursiops truncatus*, along the US eastern seaboard in 1987–88—the deaths were attributed to the consumption of toxic algae (Perrin, 1988; Geraci, 1989). Widespread blooms of toxic algae are, however, also associated with abnormally high temperatures (Underdal *et al.*, 1989). As with pinnipeds, changes in environmental temperatures can have marked effects on the distribution and local densities of some whales, e.g. beluga whales, *Delphinapteras leucas* (Hansen, 1987).

Accordingly, we wonder if the temporal associations of red tides and recent mass mortalities of marine mammals, including both harbour seals (Dietz *et al.*, 1989) and dolphins (Geraci, 1989), are merely coincidental. As in the case of the pinnipeds, the mass mortalities of humpback whales (December 1987) and dolphins (June 1987) along the US east coast began at times when both air temperatures (NOAA National Climatic Data Center, Asheville, NC, pers. comm.) and sea surface temperatures (Strong, 1989) were unseasonably warm. Perhaps our hypothesis that increased environmental temperatures provide requisite conditions for mass mortalities also applies to cetaceans?

Regardless, the identification of a pattern in the mass mortalities of pinnipeds provides a prescription for anticipating such events in the future.

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Long Term Risks of Recurrent Seal Plagues

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There has been widespread speculation about the factors which contributed to the mass mortality of North Sea harbour seals during 1988, and the threat that these factors pose to this and other marine mammal populations. We identify the questions which need to be answered before these speculations can be transformed into testable hypotheses and outline an epidemiological context for the preliminary evaluation of such hypotheses.

The epizootic which killed more than 17 000 harbour seals (*Phoca vitulina*) around the coasts of Europe in 1988 is the largest and best documented case of mass mortality in marine mammals (Harwood & Hall, in press). It is of particular scientific interest for a number of reasons: the size of the affected local populations had been monitored before the epizootic and will be monitored now that it is over; a high proportion of the animals which died were recovered and subject to pathological, bacteriological, and virological examination; and the speculation which surrounded the initial investigations received extensive coverage in the specialist and general press. Many of these speculations would not normally have been exposed to public view but, because they were widely publicized, they have been elevated to the status of working hypotheses (Harwood, 1990).

It is now widely accepted that the primary cause of the deaths was infection with a previously undescribed paramyxovirus from the morbilli family, which has been christened phocine distemper virus—PDV (Mahy *et al.*, 1988; Cosby *et al.*, 1988). However, a number of important questions about this particular epizootic remain unanswered. Persistent questions are: where did the virus come from? why did the epizootic occur when it did? why did it cause so many deaths? what was the contribution of other environmental factors? and what are the risks of future epizootics of this kind among marine mammal populations? In this paper we review