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SEA OTTER MORTALITY AT KODIAK ISLAND, ALASKA, DURING SUMMER 1987

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A die-off of sea otters (*Enhydra lutris*) occurred in the Kodiak Archipelago, Alaska, during summer 1987. Previous die-offs of sea otters were related to a combination of food stress and inclement weather during winter (Kenyon, 1969). The magnitude and timing of the recent die-off at Kodiak Island were unexpected because large numbers of sea otters have occupied the area only since the late 1970s and food available to sea otters is patchy but still abundant. In addition, with exception of a 2-week-long period of storms during June, the weather was moderate throughout the summer. We have not observed incidents of mortality of similar magnitude at Kodiak Island during 3 other years of field work of equal or greater intensity.

Between 14 June and 5 October 1987, we found 34 sea otter carcasses north of Kodiak Island, and south of Afognak and Raspberry islands, during routine censuses of the study area and systematic beach walks. Through interviews of local residents and fishermen, we learned of an additional 26 carcasses that we did not collect or were unable to find. Based on the locations of reported carcasses, they unlikely were the same as those recovered. We estimated the condition of each carcass by the following criteria established by the marine-mammal-stranding program of the National Marine Fisheries Service: fresh dead; moderate decomposition,—organs distinct; advanced decomposition—organs indistinct or the same color; and old—mummified or skeleton only. Based on these approximations, most of the mortality occurred during early to midsummer (Fig. 1). Sex of dead sea otters was determined and necropsies were performed as soon as possible after discovery. Length and weight were recorded for all intact carcasses. A lower premolar was extracted from 32 carcasses. The teeth were sectioned and stained, and the age of each otter was estimated by counting the number of cementum annuli (Garshelis, 1984). Samples of tissue from two relatively fresh carcasses were collected, preserved in buffered 10% formalin, and examined by pathologists at the National Wildlife Health Center, Madison, Wisconsin. Fluid contents of two stomachs from freshly dead sea otters were analyzed for paralytic shellfish poison by standard mouse bioassay (Quayle, 1969).

The advanced state of decomposition of most carcasses and loss of tissues to scavengers hindered our efforts at determining cause of death. Necropsies of intact or partially intact carcasses revealed no obvious pathology, but four sea otters had foam in their lungs suggesting they had aspirated water before dying. Cause of death was determined for only four of the 33 sea otters. One male was shot, one female and one male drowned in salmon (*Oncorhynchus* sp.) gill nets, and one male died from complications resulting from vegetative valvular endocarditus. The latter diagnosis was confirmed by tissue analysis. Based on net marks, cuts, and loose teeth, we suspect that one other adult male may have drowned in a salmon gill net. Two male pups, classified as dependents on the basis of size, tooth eruption, and pelage, may have died after abandonment or death of their mothers. An unidentified toxin was found in stomach fluids from two relatively fresh carcasses. We received three reports from residents and fishermen of sick sea otters and we observed one moribund sea otter in the water that was able to dive only feebly at the approach of our boat.

Of 28 sea otters for which sex was determined, 24 (86%) were males and four (14%) were females. Sea otters often segregate by sex (Garshelis et al., 1984). From the high proportion of males we infer that the source of mortality was highly localized—for example, in one of two large male groups that exist in the study area.

The estimated ages of the dead male sea otters ranged from 1–12 years, with 7- and 8-year olds the most frequent. The average ($\pm SD$) estimated age of the males (6.8 $\pm$ 3.0 years, $n = 24$) was significantly greater than that of live males that we captured in the same area in 1986 and 1987 (3.7 $\pm$ 2.6 years, $n = 13$, $t = 3.07$, $P < 0.01$). Estimated ages of four female sea otters were 1-year old ($n = 2$) and 7-years old ($n = 2$).

The age structure of sea otters in this die-off differs from that found at Amchitka Island in the Aleutian Chain, where losses were greatest among dependent pups, recently weaned otters, and old otters (Kenyon, 1969). Mortality at Amchitka Island probably was related to food stress, which was unlikely at Kodiak Island. The difference between the age structure of carcasses and the sample of live otters suggests that older sea

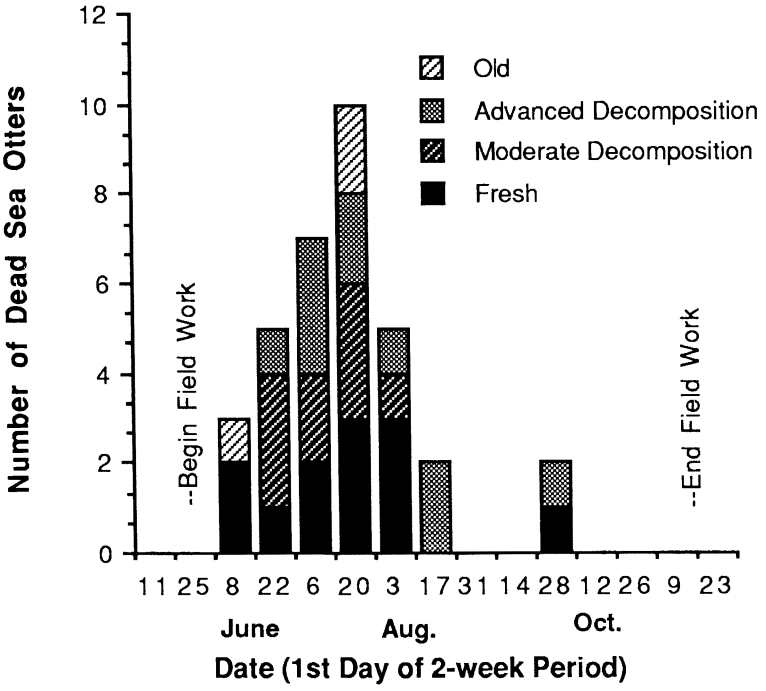

FIG. 1.—Condition of sea otter carcasses and the timing of their recovery at Kodiak Island, summer 1987.

otters were more susceptible to mortality; however, we cannot discount the possibility that the tangle nets we used to capture live otters may have selected young, inexperienced animals.

The die-off occurred during summer when water temperature and sea state are most favorable for sea otters. Kenyon (1969) found positive relationships between mortality, particularly the number of emaciated carcasses recovered, and inclement, winter weather. Between 15 and 31 June 1987, a series of storms passed through Kodiak Island causing strong winds and heavy rainfall. These storms may have reduced the time sea otters could feed, but they unlikely were responsible for the mortalities. We found fresh carcasses before, during, and long after the period of stormy weather, and only two carcasses obviously were emaciated: an 8-year-old male whose teeth were in poor condition, and the male suffering from vegetative valvular endocarditus. Three relatively fresh carcasses of males, however, were about 15% lighter than expected based on a linear regression of weight by length for live male sea otters at Kodiak Island ($Y = 2.96X - 87.16$; $r^2 = 0.83$).

Drowning, shooting, and disease, known sources of mortality for sea otters elsewhere in Alaska and California (Cornell et al., 1979; Hennessey and Morejohn, 1977; Kenyon, 1969; Morejohn et al., 1975; Rausch, 1953), were the only conclusive sources of mortality that we identified. Kodiak Island is one of the principal sites of a legal harvest of sea otters by Alaskan natives (Rotterman and Simon-Jackson, 1988) but this mortality is concentrated during winter. An unknown amount of illegal shooting also occurs. If shooting contributed substantially to this die-off, we would have found more than one sea otter with bullet wounds. An unknown but small number of sea otters die in salmon gill nets in our study area during the summer fishing season; however, few sea otters were observed at fishing sites in 1987.

Coincident with our discovery of dead sea otters, two people on Kodiak Island became sick from eating blue mussels (*Mytilus edulis*) collected in our study area. A sample of blue mussels collected independently from the same site contained >5,800 μg of paralytic shellfish toxin/100 g of mussel tissue (80 μg paralytic shellfish toxin/100 g tissue is considered the upper limit for human consumption in Alaska; M. Ostasz, pers. comm.). From this, the presence of an unidentified toxin in stomachs of two sea otters, and the ability of butter clams (*Saxidomus giganteus*), a principal food of sea otters in our study area, to sequester paralytic shellfish toxins (Kvitek and Beitler, 1989), we suggest that paralytic shellfish poisoning could have been a source of the sea otter mortality at Kodiak Island. Although paralytic shellfish toxins have been implicated in the deaths of sea otters in the Commander Islands (Sidorov, 1987), and as a cause of death of other marine

vertebrates (Adams et al., 1968; Armstrong et al., 1978; Buergelt et al., 1984; Coulson et al., 1968; Grindley and Taylor, 1962; Hockey and Cooper, 1980; Keyes, 1965; McKernan and Scheffer, 1942; Nisbet, 1983; White, 1981), only in fish (White, 1981) and starlings (*Sturnus vulgaris*, Kvitek and Beitler, 1989) have biotoxins produced from dinoflagellates and mortality been experimentally and conclusively linked. From recent experimental evidence from Kodiak Island, we suggest that sea otters can detect, and are vulnerable to paralytic shellfish toxins, but probably are not at mortal risk from the toxins provided sufficient quantities of low-toxicity foods are available, the situation in 1987. Of known or suspected causes of mortality of sea otters at Kodiak Island during summer 1987, only disease cannot be ruled out as the principal source.

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