Killer Whale Predation on Sea Otters Linking Oceanic and Nearshore Ecosystems

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After nearly a century of recovery from overhunting, sea otter populations are in abrupt decline over large areas of western Alaska. Increased killer whale predation is the likely cause of these declines. Elevated sea urchin density and the consequent deforestation of kelp beds in the nearshore community demonstrate that the otter’s keystone role has been reduced or eliminated. This chain of interactions was probably initiated by anthropogenic changes in the offshore oceanic ecosystem.

Apex predators often initiate forces that cascade across successively lower trophic levels, sometimes reaching the base of the food web (1). Plant-herbivore interactions vary predictably with trophic complexity in such systems, being weak or strong when the number of trophic levels is odd or even, respectively (2). Sea otters (Enhydra lutris) and kelp forests provide a well-known example of this pattern (3). After being protected from overhunting, recovering otter populations transformed nearshore reefs from two- to three-trophic-level systems by limiting the distribution and abundance of herbivorous sea urchins, thereby promoting kelp forest development (4).

Sea otters abounded across the North Pacific rim until unregulated exploitation in the maritime fur trade reduced the species to near-extinction by the early 20th century (5). Population regrowth began when protection was afforded under the International Fur Seal Treaty. A geographically discordant recovery pattern ensued because of the fragmented distribution of surviving colonies, the discontinuous nature of their habitat, and the otter’s limited dispersal ability (5, 6). Consequently, by the 1970s otter populations had recovered to near maximum densities in some areas of their historic range, were growing rapidly in others, and remained absent from still others (7). The sea otter’s predatory role in kelp forest ecosystems was discovered by contrasting inhabited with uninhabited areas (8) and by observing changes over time as the uninhabited areas were recolonized and their founding populations grew (4, 9). In addition to showing the influence of sea otters on North Pacific kelp forests, this approach has demonstrated a breadth of indirect effects on coastal ecosystems (10). The sea otter’s reputation as a keystone species (11) is based on these interactions and processes.

Recently, sea otter populations have declined precipitously and unexpectedly over large areas of western Alaska. We first detected this decline through population surveys at Adak Island in the central Aleutian archipelago, which indicated that the otter population decreased ~25% per year through the 1990s, resulting in nearly an order-of-magnitude overall reduction by 1997 (Fig. 1). Additional surveys of Little Kiska, Amchitka, and Kagakaska Islands all show population declines of similar timing and rate to that which occurred at Adak (Fig. 1). Aerial surveys of the Aleutian archipelago conducted by the U.S. Fish and Wildlife Service in 1965 and 1992 further indicate that these declines are occurring throughout the region (12). The concurrent and widespread nature of these declines strongly suggests a causal link with the oceanic environment.

Demographic explanations for the sea otter population declines are limited to reduced fertility, increased mortality, or redistribution. Of these, reduced fertility and redistribution can be excluded. Studies of radiotagged sea otters at Amchitka Island in 1992–94 and Adak Island in 1995–96 show that birth rates of adult females and pup survival rates from birth to weaning were similar to those of stable populations. Redistribution is equally unlikely because the declines were synchronous over large areas—there have been no population buildups on some islands to account for the losses on others—and radio-tagged otters at Amchitka and Adak islands provided no indication of redistribution during the declines (13). From this we conclude that the sea otter population declines were caused by increased mortality.

Three lines of evidence point to increased predation by killer whales (Orcinus Orca) as the reason for this mortality. First, although killer whales and sea otters have been observed in close proximity for decades, the first attack on a sea otter was seen in 1991. Subsequently, nine more attacks have been reported (14). We evaluated the likelihood that this cluster of recent observations was due to chance alone by summing the number of person-days spent in the Aleutian Islands by our research team before and after 1990 (3405 person-days before; 4005 after), estimating the attack rate from the post-1990 data (0.0015 attacks per day), and then calculating the probability of no attacks being seen before 1990 if the attack rate remained constant over the 27-year period. By modeling the expected number of observed attacks as a Poisson process, the probability of zero attacks being seen before 1990 is 0.006 (15).

Second, we evaluated the impact of killer whales on sea otter populations at Adak Island by contrasting otter population trends and survival rates between Clam Lagoon, an area uniquely inaccessible to killer whales, and adjacent Kuluk Bay, an open coastal environment (Fig. 2). Sea otter numbers were stable from 1993 through 1997 in Clam Lagoon, whereas in Kuluk Bay they declined by 76%. In 1995, we marked 17 otters in Clam Lagoon and another 37 in Kuluk Bay with flipper tags and surgically implanted radio transmitters in order to compare their behavior and demography. There was virtually no movement of the marked animals between these areas. However, through year 1 of the study, the disappearance rate of sea otters in Kuluk Bay (65%) was greater than five times that of Clam Lagoon (12%), a trend that continued through year 2.

Finally, we estimated how many otters must have been eaten by killer whales to drive the decline rates, and then compared the actual number of observed attacks with the expected number of observed attacks based on this estimate. This analysis was done for the area between Kiska and Seguam Islands. Before the onset of the decline, an estimated 52,656 otters inhabited this area (16). Life table statistics (age-specific birth and death rates) were estimated from data collected during earlier field studies to construct a Leslie matrix for a stationary population. We then added an age-constant death rate (17) from killer whale predation sufficient to reduce the population by 78% over 6 years—the observed rate and magnitude of decline at Adak. The simulation was run by holding the number of individuals that died from killer whale predation constant over time, which produced a loss estimate of 6788 otters per year. The expected number of observed attacks produced by this approach is 5.05 for this 6-year period (18). This compares favorably with the 6 attacks that were seen.

Disease, toxins, and starvation, which are three other causes of elevated mortality in wildlife populations, can be dismissed as causes of the population declines. Any one of these should have produced substantial numbers of beach-cast carcasses, whereas very
few were found. Marked increases in sea urchin biomass during the population decline at Adak (Fig. 1) are further evidence against starvation, because sea urchins are the principal prey of sea otters in the Aleutian Islands (19). Although we looked specifically for signs of disease, none were found (20). Elevated contaminant concentrations have been reported in the Aleutian Islands (21), but subsequent analyses from 39 sites across the Aleutian archipelago have shown that these are restricted to a few small areas (22), which is inconsistent with the widespread declines in otter numbers.

The collective evidence thus leads us to conclude that increased killer whale predation has caused the otter declines. Although the population size and status of killer whales in the Aleutian Islands are unknown, these animals are commonly seen. From the energetic requirements of free-ranging killer whales and the calorific value of sea otters, we estimate that a single killer whale would consume 1825 otters per year and thus that the otter population decline could have been caused by as few as 3.7 whales (23).

Strikingly rapid changes in the kelp forest ecosystem have accompanied the sea otter population declines (Fig. 1). In 1987, when otters at Adak Island were near equilibrium density, the kelp forests were surveyed at 28 randomly selected sites (4). Otters were still numerous at Adak in 1991, when five of these sites were randomly chosen for the measurement of plant tissue loss to herbivory (24). Using similar procedures at the same sites in 1997, we resurveyed the kelp forest and repeated the measurements of plant tissue loss to herbivory. Over the 10-year interim, sea urchin size and density increased to produce an eight-fold increase in biomass, while kelp density declined by more than a factor of 12 (Fig. 1). Observations made in August of 1997 revealed similar changes at Kiska, Amchitka, and Kagalaska Islands.

Killer whales and sea otters have co-inhabited the west-central Aleutian archipelago for much of the past half century, and probably for millennia before. Thus, it is necessary to ex-

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**Fig. 1.** (A) Changes in sea otter abundance over time at several islands in the Aleutian archipelago and concurrent changes in (B) sea urchin biomass, (C) grazing intensity, and (D) kelp density measured from kelp forests at Adak Island. Error bars in (B) and (C) indicate 1 SE. The proposed mechanisms of change are portrayed in the marginal cartoons—the one on the left shows how the kelp forest ecosystem was organized before the sea otter’s decline and the one on the right shows how this ecosystem changed with the addition of killer whales as an apex predator. Heavy arrows represent strong trophic interactions; light arrows represent weak interactions.

**Fig. 2.** Population trends and survival rates of sea otters in Clam Lagoon (solid squares) and adjacent Kuluk Bay (open circles), Adak Island, Alaska. (A) The rate of population change \( r \), calculated as the slope of the linear best fit to the natural log of the number of otters counted versus year, for Kuluk Bay between 1993 and 1997 was \( -0.345 \) (SE = 0.058), which is significantly different from 0 \( (R^2 = 0.946, P = 0.027) \). In Clam Lagoon, the rate of change over this same period was 0.006 (SE = 0.034), which is not significantly different from 0 \( (R^2 = 0.011, P = 0.867) \). (B) Survival rates of marked sea otters differed significantly \((x^2 = 27.26, 1 \, df, P < 0.001)\). In Clam Lagoon, the rate of change over this same period was 0.006 (SE = 0.034), which is not significantly different from 0 \( (R^2 = 0.011, P = 0.867) \). (B) Survival rates of marked sea otters differed significantly \((x^2 = 13.52, 1 \, df, P < 0.001)\).
plain why the behavior of killer whales toward
sea otters has recently changed. The most likely ex-
planation is a shift in the prey resource base
for killer whales. Some killer whale groups or
individuals feed on marine mammals (25), in-
cluding Steller sea lions and harbor seals, and
populations of both these species recently have
collapsed across the western North Pacific. Sea
lion populations began to decline in the late
1970s, and their numbers had reached mini-
mum levels in the Aleutian islands by the late
1980s (26), a time that coincides with the onset of
otter declines. Although the exact cause of the
pinioned decline is uncertain (27), it prob-
ably relates to reduced abundance and altered
species composition of their prey (28). Recent
population declines of piscivorous marine birds
are consistent with this explanation (29). Why
forage fish stocks have shifted is not well un-
derstood, although the change was likely caused by some combination of effects from the
region’s burgeoning fisheries, increased ocean
temperature, and depletion of baleen whales
(30).

Regardless of the ultimate cause, sea otter population declines and the consequent collapse of
kelp forest ecosystems almost certainly have
been driven by events in the offshore oceanic
realm. Our proposed explanation involves a
chain of ecological interactions, beginning with reduced or altered forage fish stocks in the
oceanic environment, which in turn sent pin-
nippled populations into decline. Pinniped num-
bers eventually became so reduced that some of
the killer whales who once fed on them expand-
ed their diet to include sea otters. This shift in
killer whale foraging behavior created a linkage
between oceanic and coastal ecosystems and in
so doing transformed coastal kelp forests from
three- to four-trophic-level systems, thereby re-
leasing sea urchins from the limiting influence
of sea otter predation. Unregulated urchin pop-
ulations increased rapidly and overgrazed the
kelp forests, thus setting into motion a host of
effects in the coastal ecosystem.

Parts of this scenario are well documented,
others are more speculative, and still others have yet to be evaluated. Nonetheless, the data are
sufficient to make several points of broader
ecological significance. First, our findings af-
ford evidence of the often underappreciated
difference that uncommon and transient spe-
cies can have in controlling community struc-
ture, demonstrating further that such species
can link interactions across ecosystems. Al-
though intersystem linkages are becoming in-
creasingly well known (31), this example is
unusual because the linkage is formed through the
activities of a top-level carnivore. Addition-
ally, our results are relevant to understanding
food web dynamics, because they demonstrate
that adding another apex predator to a system
under top-down control has predictable effects
on plant populations at the base of the food
chain. Finally, results from this long-term study
have implications for both the approach to and
scale of other ecological field studies. The
events reported here could not have been chron-
icled or even detected in a short-term study,
were unanticipated, and thus seem poorly suited
for analysis by a priori hypothesis testing. These
points emphasize the potential signific-
ficance of large-scale ecological events and the
consequent need for large-scale approaches in
ecological research.

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12. By 1965, otter populations had recovered to pre-
exploitation levels at most of the Aleutian islands,
from Kiska in the west to Adak in the east (5). Of
the 21 islands in this region that were surveyed in both
1965 and 1992, sea otter counts decreased at all but
eight, for an average reduction of 58%. The 1965 data
are from (5); the 1992 data are from J. A. Estes, in
Technical Report MMM 97-5 (U.S. Fish and Wildlife
Service, Anchorage, AK, 1997).
13. Among resightings of 57 tagged otters at Adak (1635
resightings of 52 otters) and Amchitka (3711
resightings of 98 otters), the maximum distances
moved were 4.31 and 6.95 km, respectively. From
1965 aerial survey (5) and adjusted upward by a factor
of 5.62 to account for the proportion of animals that
were unanticipated, and thus seem poorly suited
for analysis by a priori hypothesis testing. These
points emphasize the potential signific-
ficance of large-scale ecological events and the
consequent need for large-scale approaches in
ecological research.

17. The age-constant death rate was inferred from the
5.62 to account for the proportion of animals that
were unanticipated, and thus seem poorly suited
for analysis by a priori hypothesis testing. These
points emphasize the potential signific-

15. This probability was calculated from the Poisson
probability density function f(x) = e−μx/x!, for μ =
5.1 [the expected number of attacks seen] and x = 0
(the number of attacks actually seen).
16. This number was obtained. Som counts made during
a 1965 aerial survey (5) and adjusted upward by a factor
of 5.62 to account for the proportion of animals that
were not seen. The resighting factor was calculated
from a 1972 estimate of sea otter abundance at
Amchitka Island [estimate, 6432; from J. A. Estes, in
The Environment of Amchitka Island, M. L. Merritt and R. G.
Fuller, Eds. (TID-26712, U.S. Energy Research and De-
511–526] divided by the number of otters counted at
Adak Island.
17. The age-constant death rate was inferred from the
environmental analysis of individual otters on the
calculated that an adult female killer whale feeding
on sea otters provides 41,630 kcal. From this,
we calculated that an adult female killer whale feeding
exclusively on sea otters would need three male
or five female sea otters per day. One adult male
would require five male or seven female otters per
day. The average consumption rate (five otters per
whale per day) was divided into the sea otter loss
estimate to determine how many killer whales
would be needed to account for the losses. Based
on this approach, 3.7 killer whale feeding exclu-
sively on sea otters would be sufficient to drive the
population decline.
18. These measurements of plant tissue loss were ob-
tained by placing preweighed pieces of tissue from
blades of the four most common kelp spe-
cies—Alaria fascilus, Agarum gigas, Aga-
rum cribrosum, and Thallasiophyllum clathrus—on
the seafloor and recording their change in mass
over 24 hours relative to that of adjacent caged
controls. Five replicates were done for each species
at each site.
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Induction of Antigen-Specific Cytotoxic T Lymphocytes in Humans by a Malaria DNA Vaccine

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CD8⁺ cytotoxic T lymphocytes (CTLs) are critical for protection against intracellular pathogens but often have been difficult to induce by subunit vaccines in animals. DNA vaccines elicit protective CD8⁺ T cell responses. Malaria-naïve volunteers who were vaccinated with plasmid DNA encoding a malaria protein developed antigen-specific, genetically restricted, CD8⁺ T cell–dependent CTLs. Responses were directed against all 10 peptides tested and were restricted by six human lymphocyte antigen (HLA) class I alleles. This first demonstration in healthy naive humans of the induction of CD8⁺ CTLs by DNA vaccines, including CTLs that were restricted by multiple HLA alleles in the same individual, provides a foundation for further human testing of this potentially revolutionary vaccine technology.

During 1990–1994, the administration of “naked” plasmid DNA encoding a specific protein antigen was shown to induce expression of the protein in mouse myocytes (1), to elicit antibodies against the protein (2), and to manifest protection against influenza (3) and malaria (4) that was dependent on CD8⁺ T cell responses against the expressed protein. Hundreds of publications have now reported the efficacy of DNA vaccines in small and large animal models of infectious diseases, cancer, and autoimmune diseases (5).

DNA vaccines elicit antibodies and CD4⁺ T cell responses in animals, but their major advantage at the immunological level has been their capacity to induce antigen-specific CD8⁺ T cell responses, including CTLs, which is a major mechanism of protection against intracellular pathogens. Important to our method of developing a malaria vaccine is the induction of CD8⁺ T cell responses against Plasmodium falciparum–infected hepatocytes (6). The lysis of cells in a standard chromium release assay was used as a surrogate for antihaptoocyte responses, because it has been established that CD8⁺ CTLs, which recognize peptide-pulsed target cells, also recognize and eliminate parasite-infected hepatocytes (6). On the basis of our work with rodents (4, 7) and our work and that of others with rhesus monkeys (8, 9), we have developed a plan for manufacturing and testing the efficacy of a multigene P. falciparum liver-stage DNA vaccine in humans (10). This has been contingent on establishing that DNA vaccination of humans is safe and induces antigen-specific, genetically restricted, CD8⁺ T cell–dependent CTLs. Recently, the presence of CTL responses in human immunodeficiency virus (HIV)–infected individuals after vaccination with plasmid DNA encoding the nef, rev, or tat genes or the env and rev genes of HIV was reported (11). Interpreting these results is difficult because of the concurrent HIV infection, which has been demonstrated to prime individuals for a CTL response that is independent of immunization.

Accordingly, 20 healthy, malaria-naïve adults were recruited and randomized into four dosage groups of five individuals. Three injections of 20, 100, 500, or 2500 μg of plasmid DNA encoding the P. falciparum circumsporozoite protein (PICSV) (12) were administered at 4-week intervals in alternate deltoids (13). The details of recruitment, safety, and tolerability were reported elsewhere (14). To assess CTL responses, we collected peripheral blood mononuclear cells (PBMCs) from each volunteer before vaccination, 2 weeks after the second immunization, and 2 and 6 weeks after the third immunization. These cells were either assayed while fresh for recall antigen-specific CTL responses (15) or were frozen (16) for subsequent study. In parallel, CTL assays were carried out with PBMCs from nonimmunized control volunteers. Cytolytic activity was assessed after both primary and secondary in vitro restimulation against HLA-matched and HLA-mismatched PCSP-specific and control targets. The percent lysis and the percent specific lysis were determined as described (15). The most sensitive and specific method (17) for demonstrating the presence of CTLs was with effector cells that were expanded in vitro by exposure to cells infected with canary pox (ALVAC) expressing the PCSP (18) and with target cells that were sensitized with PCSP-derived synthetic peptides (19). There was no apparent difference between the primary and secondary assays (20) or between the fresh and frozen specimens (21).

For logistical reasons, fresh PBMCs were studied only before vaccination and after the second immunization in the 20- and 100-μg-dosage groups but were studied before vaccination and after all immunizations in the 500- and 2500-μg-dosage groups, with the exception of one individual (13). For 14 individuals, adequate amounts of frozen PBMCs were available for further analysis. A typical pattern of CTL responses is presented in Fig. 1A. These responses were peptide-specific and genetically restricted because there was little or no recognition of autologous targets that were incubated with the control peptide or of HLA class I–mismatched targets that were incubated with the specific peptide. This activity was shown to be CD8⁺ T cell–dependent by restimulating...