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FISHING FOR LOBSTERS INDIRECTLY INCREASES EPIDEMICS IN SEA URCHINS

KEVIN D. LAFFERTY[1]

USGS Western Ecological Research Center, c/o Marine Science Institute, University of California, Santa Barbara, California 93106 USA

Abstract. Two ecological paradigms, the trophic cascade and the host-density threshold in disease, interact in the kelp-forest ecosystem to structure the community. To investigate what happens when a trophic cascade pushes a host population over a host-threshold density, I analyzed a 20-year data set of kelp forest communities at 16 sites in the region of the Channel Islands National Park, California, USA. Historically, lobsters, and perhaps other predators, kept urchin populations at low levels and kelp forests developed a community-level trophic cascade. In geographic areas where the main predators on urchins were fished, urchin populations increased to the extent that they overgrazed algae and starvation eventually limited urchin-population growth. Despite the limitation of urchin population size by food availability, urchin densities, at times, well exceeded the host-density threshold for epidemics. An urchin-specific bacterial disease entered the region after 1992 and acted as a density-dependent mortality source. Dense populations were more likely to experience epidemics and suffer higher mortality. Disease did not reduce the urchin population at a site to the density that predators previously did. Therefore, disease did not fully replace predators in the trophic cascade. These results indicate how fishing top predators can indirectly favor disease transmission in prey populations.

Key words: Channel Islands (California, USA); disease epidemics; emerging disease; host-density threshold for disease transmission; kelp forest ecosystem; lobster fishing; sea urchins; trophic cascade.

INTRODUCTION

Predation and disease are ubiquitous components of ecosystems but are usually considered to operate on prey/host populations through distinctly different and unrelated processes. A classic interpretation is that predators may weed sick individuals out of the prey population, increasing the health of the herd (Slobodkin 1974, Hudson et al. 1992, Lefcort and Blaustein 1995). Alternatively, predation may keep prey at densities too low for efficient disease transmission (Packer et al. 2003).

Trophic cascades can result when predators reduce the abundance of their prey to the extent that the prey's food source (plants or other prey) indirectly increases in abundance (Polis et al. 2000). This can affect ecosystem processes and the structure of entire communities (Strong 1992). An example of a community-level trophic cascade occurs where predators such as sea otters, fishes, lobsters, and sea stars normally limit sea urchin populations and, with this grazer suppressed, large standing stocks of macroalgae persist (Sala et al. 1998). Fishing on the predators of sea urchins adds a fourth level, fishers, to the top of the trophic cascade. Such changes at the top of the food chain can greatly alter population densities at lower trophic levels. Released from predation, urchin densities increase so that they overgraze the standing stock of attached plants (Estes and Duggins 1995, Tegner and Dayton 2000). Evidence of the effect of fishing for lobsters on trophic cascades is apparent in comparisons between a marine reserve and fished areas (Babcock et al. 1999, Pinnegar et al. 2000, Shears and Babcock 2002).

Population density is a key factor determining disease processes. In particular, host-density thresholds for epidemics are typical when disease transmission requires contact between individuals. If the frequency of contact between infected and susceptible hosts is lower than the death or cure rate of infected hosts, the prevalence of disease decreases. This process is why epidemics eventually fade out and can also be what prevents their initiation. For example, measles epidemics rarely occur below a minimum human population density (Black 1966). There are two sequential aspects to an epidemic that depend on host density. The first is the probability of an epidemic. Below a certain host threshold, this probability is theoretically zero; above the threshold, the probability increases directly with host density (Anderson and May 1986). The second component is the extent to which an epidemic reduces host density. The impact of an epidemic, expressed as the fraction of the susceptible host population removed, should also increase with host density (Swinton 1998). The importance of host density for epidemics potentially allows for strong interactions between disease and other sources of population regulation such as predation. Because host populations that reach equilibrium

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[1] Lafferty@lifesci.UCSB.edu

at low densities are less likely to experience damaging host-specific epidemics, a release from predation should increase the risk and impact of disease in a prey population. In this sense, disease could act as a redundant population regulator, coming into play if other regulatory effects (competition, predation) are too weak to cap populations at low levels. Because predators can directly or indirectly alter population densities at lower trophic levels, they may affect disease processes at these levels even when the predator is not a host in the life cycle of the disease.

What happens when a trophic cascade pushes a host population over a host-density threshold? To investigate this question, I studied how the fishing of predators alters conditions for epidemics in prey populations in a kelp forest ecosystem. I analyzed a replicated time series of sea urchin populations in the Channel Islands National Park off southern California, USA. In this region, large spiny lobsters, *Panulirus interruptus,* prey on the purple urchin, *Strongylocentrotus purpuratus* (Tegner and Levin 1983), but the predatory seastar *Pycnopodia helianthoides* is also an important predator at colder water sites where lobsters are rare (Duggins 1983, Lafferty and Kushner 2000). Furthermore, a large labrid fish, the sheephead (*Semicossyphus pulcher*), can also reduce urchin densities in southern California (Cowen 1983). The effect of these predators on urchin populations can be limited by fisheries. In southern California, commercial and recreational fisheries take a very high fraction of spiny lobsters over the legal size limit (Tegner and Levin 1983). In addition, sheephead are speared and trapped outside of reserves (Tegner and Dayton 2000). The larger red sea urchin, *S. franciscanus,* is also subject to an intensive fishery.

Diseases have depressed urchin abundance in many areas around the world, sometimes dramatically so (Scheibling and Stephenson 1984, Harrold and Reed 1985, Pearse and Hines 1987, Azzolina 1988, Lessios 1988, Andrew 1991, Hughes 1994, Scheibling and Hennigar 1997). In addition to the direct link between host density and disease-transmission rates, crowded, starved urchins may not able to commit sufficient resources (such as acid phosphatase, [Shimizu and Nagakura 1993]) to battling infections. Also, high densities may force urchins from their crevices in search of food, exposing them to surge that may damage them such that they are more susceptible to infection (Gilles and Pearse 1986). Such increases in susceptibility could increase disease-transmission efficiency at high host density. Disease symptoms at the Channel Islands (spine loss, tissue damage represented by dark blotches on the test [Richards and Kushner 1994]) are most consistent with *Vibrio* bacteria (Gilles and Pearse 1986), but the etiological agent of disease has not, as yet, been formally determined (as per Ritchie et al. 2001). The most common host is the purple urchin, *Strongylocentrotus purpuratus*. Two less common sympatric species (*S. franciscanus, Lytechinus anameus*) also suffer from disease. The disease is often fatal as evidenced by the high number of freshly dead and dying animals during the peak of an epidemic, although the presence of urchins re-growing spines after an epidemic indicates that some sick urchins recover (Richards and Kushner 1994). Disease similarly affects sympatric sea stars, though it is not known whether the urchin disease is related to the sea star disease. Understanding the host specificity of the disease is important for being able to make predictions about the association between host density and transmission efficiency.

I asked the following questions: (1) Is there evidence for top-down control? (2) Do host-density thresholds affect epidemics? and (3) How do trophic cascades and epidemics interact? As evidence for top-down control, I expected to find negative associations between predator density and urchin density as well as between urchin density and algal cover. I also tested the prediction that fishing would decrease top predators (lobsters), increase prey (urchins) and decrease plants (kelp). To determine the importance of host-density thresholds, I first tested for a correlation between disease in urchins and disease in seastars to determine whether the disease was likely to be specific to urchins. I then evaluated whether the probability of an epidemic increased with host density, starvation, or warm temperatures. Next, I considered whether epidemics reduced urchin density and if the magnitude of this effect increased with the urchin density before the epidemic. To look at the interactions between trophic cascades and epidemics, I evaluated whether epidemics were less frequent in reserves than in fished areas.

Methods

The Channel Islands National Park (California, USA) long-term kelp forest monitoring program, established in 1982 (Davis et al. 1997), provided data from 1992 through 2001 for analysis (these data are available via request from the Superintendent of the Channel Islands National Park). This program annually samples 16 sites from the north and south sides of Santa Barbara, Anacapa, Santa Cruz, Santa Rosa, and San Miguel Islands (1–3 visits per summer, Fig. 1). Fishing is allowed at each site except for two sites on Anacapa Island that have been in a marine reserve since 1978 (Fig. 1). Each site consists of a 100-m permanent transect. Divers use a variety of methods to quantify taxa such as urchins, sea stars, lobsters, and fishes. Annual reports (e.g., Richards and Kushner 1994) describe each year's monitoring efforts. For example, divers measure urchin densities using 12–40 quadrats spaced along each transect. Data on a particular taxa are averaged to one value per year, per site. The kelp forest monitoring program also collects recruitment data from caged concrete modules placed at 10 of the 16 sites. Individual purple urchins smaller than 15 mm were classified as ''recruits.''

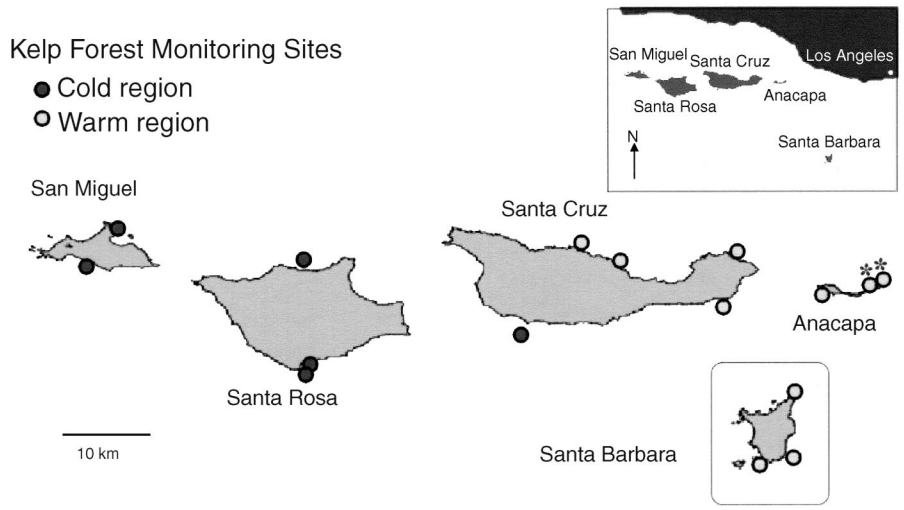

FIG. 1. Kelp Forest Monitoring Program sites located at the California (USA) Channel Islands. Two sites in a small marine reserve that were established in 1978 are denoted by stars. Sites used for comparison with the reserves include the southeast sites on Santa Cruz and Santa Barbara Island. The fished site on Anacapa was not used in this comparison because it is substantially deeper than the reserve sites.

To evaluate the presence of a trophic cascade, I looked for associations in density among trophic levels using all 16 sites (with year as a covariate). I also used repeated-measures analyses to compare the densities of lobsters, sea urchins, and algae under fished and no-fishing conditions, site was a random factor nested in reserve designation and year was the repeated variable (the repeated-measures design accounted for the non-independence of sampling each site in sequential years). Unfortunately, the reserve sites were both on Anacapa Island, making it difficult to separate a fishing effect from a biogeographic effect. For this reason, when testing the effect of fishing I compared the two sites in the reserve only with the seven sites outside the reserve that matched the geographic province and depth of the reserve (reserve and matching sites as noted in Fig. 1).

Because disease affects all three common urchin species, I defined ''host'' density as the sum of the densities of the three urchin species. This was done after confirming that epidemics in urchins were not associated with similar epidemics in seastars. Prior to 1992, disease was not reported, though, since neither the field effort nor the personnel greatly changed, it presumably would have been seen had it been prevalent. For this reason, unless otherwise noted, I restricted analyses of disease to 1992 through 2001. Since 1992, the divers categorize the health of the urchin population according to disease prevalence: disease free, fewer than five individuals sick (rare), between five individuals and 25% sick (common), between 25 and 75% sick (abundant), and greater than 75% sick (very abundant). This provided five prevalence categories for 16 sites for 10 yr. For the analyses, I considered reports of no disease to indicate that an epidemic did not occur. As reports with fewer than five sick urchins were neither clearly an epidemic nor evidence that an epidemic did not occur (i.e., they could have represented the head or tail end of an epidemic), I did not consider them in the analyses. The monitoring program also collects in situ temperature using data loggers that record temperature several times per day. I calculated monthly means from these data (filling occasional gaps in the record by interpolating among sites and dates), from which I calculated a mean summer temperature (June–July) for each year.

To calculate the association between epidemics and density, I grouped the density data (sorted by density) into bins of 10 observations and, for each bin, calculated the proportion of observations where an epidemic was evident ($\pm$95% confidence limits calculated from percentages). I fit this relationship with a logarithmic regression. To independently estimate the host-density threshold, the logarithmic regression excluded the lowest density bin where the proportion of observations with an epidemic was zero. I then used an ordinal-logistic regression model to determine what independent variables were associated with epidemics. The presence or absence of an epidemic served as the dependent variable. I used urchin density (log 10 of the sum of the three host species), summer temperature, algal cover (combined percent cover of all edible fleshy algae, i.e., excluding *Desmerestia ligulata* and crustose corralines) as continuous, independent variables and island and year as nominal, independent variables.

To explain variation in urchin density, N, between years, I expressed change in density as population growth rate, $r = \ln(N_{t+1}/N_t)$, as this provided a standard and relative measure of density change that was normally distributed. I used r as a continuous, dependent variable in a multivariate, least-squares model using

prevalence as an ordinal independent variable, temperature, algal cover, and predator density (log 10 of the sum density of predatory sea stars, lobsters, and sheephead) as continuous independent variables, and island and year as nominal independent variables. I conducted a similar analysis to evaluate the prediction that the change in urchin density in the year following an epidemic should decline with the density of the urchin population in the year of the epidemic (using log density and algal cover as independent variables).

I investigated the effects of fishing on disease by comparing the proportion of dates where disease was observed inside and outside the reserve using a chi-square test of independence.

Results

Correlations among the densities of consumers and their food resources were consistent with prediction from the trophic-cascade hypothesis. After controlling for year as the repeated measure, there was a negative association between edible algal abundance and urchin abundance (log density all species summed) among the 16 sites (model df = 20, effect df = 1; F ratio = 76.4, $P < 0.0001$). At the warm and shallow sites, urchin abundance was strongly negatively associated with the density of lobsters and sheephead (warm shallow sites, model df = 18, effect df = 1; F ratio for lobster = 15.8, $P = 0.0001$; F ratio for sheephead = 13.6, $P = 0.001$). At the other sites (cold or deep), urchin abundance was negatively associated with the predatory seastar *Pycnopodia helianthoides* (model df = 19, effect df = 1; F ratio = 28.1, $P < 0.001$). The average purple urchin density at a site did not increase with the average amount of purple urchin recruitment at a site (for the 10 sites where recruitment was measured, $R = -0.42$, $P = 0.22$). Although algal abundance was higher at cold temperatures (model df = 20, effect df = 1; F ratio = 42.7, $P < 0.0001$) and urchin abundance was higher at warm temperatures (model df = 20, effect df = 1, F ratio = 29.3, $P < 0.0001$), the cascade effects were significant whether or not temperature was used as a covariate. Removal of an urchin predator (e.g., lobsters) by fishing provides further evidence that a trophic cascade occurs in this system (Fig. 2). Compared with outside the reserve, lobster densities were 5.5 times higher inside the reserve (model df = 33, effect df = 1; F ratio = 6.7, $P = 0.02$). Suprisingly, there was no difference in the density of sheephead inside and outside of the reserve (model df = 31, effect df = 1; F ratio = 0.27, $P = 0.61$). Despite equal rates of recruitment inside and outside of the reserve, urchin densities were 4 times higher outside of the reserve than inside the reserve (log total urchin density, model df = 27, effect df = 1; F ratio = 8.4, $P = 0.023$). Algal biomass was 3 times higher inside the reserve than outside the reserve (model df = 26, effect df = 1; F ratio = 6.2, $P = 0.042$).

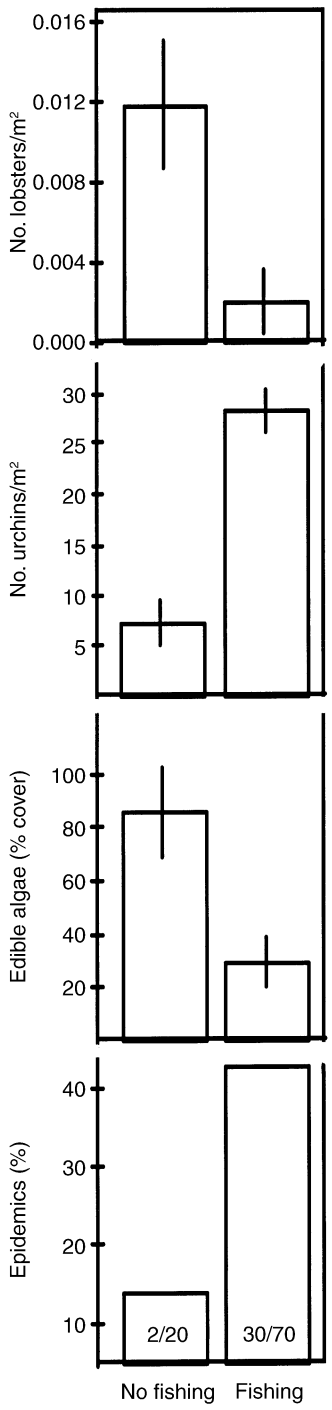

FIG. 2. Comparison of lobster (predator), purple urchin (prey), and algae (food) abundance and occurrence of urchin disease inside and outside of marine reserves (Channel Islands, California, USA). Data are means ± 1 SE (except ''Epidemics (%)'', where the numerator [number of dates/sites with epidemics] and denominator [total number of dates/sites] are given). All comparisons were significantly different as analyzed by a repeated-measures, general linear model or chi square test.

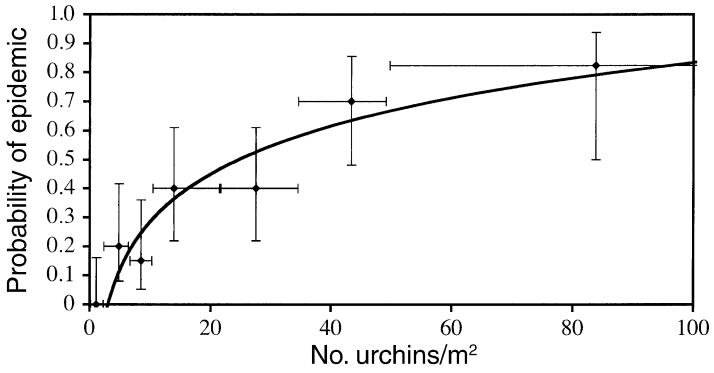

FIG. 3. The relationship between the probability of an epidemic and the density of sea urchins. Urchin density is the sum of three host species. Each point represents the mean probability of an epidemic (and 95% CI) across 10 observations for a particular mean density (range in urchin density is indicated by horizontal bars). The line follows a logarithmic regression [$y = 0.2386 \ln(x) - 0.2647$] with the lowest density data point removed. The host-density threshold for epidemics is the x intercept and equals 3.7 urchins/m^2.

Urchin density (all species pooled) at a site ranged from 0 to 145 urchins/m^2. Disease was only seen when density was >3.2 urchins/m^2 and was always noted if the density exceeded 74 urchins/m^2. The disease was specific to sea urchins (disease in urchins was not associated with disease in sea stars, $R = 0.09$, $P > 0.05$). The logarithmic regression showed a significant positive association between urchin density and disease (Fig. 3, $R^2 = 0.89$, $P < 0.005$) and estimated that the host-density threshold for epidemics was 3.72 (±1.4 urchins/m^2 (mean ± 1 SE). The ordinal-logistic regression also found that epidemics were positively associated with urchin density (df = 1, Wald chi square = 11.6, $P = 0.0006$). No other effects, including food and temperature, were significantly associated with epidemics.

The growth rate of the urchin population decreased as food became scarce (df = 1, F ratio = 8.34, $P = 0.0046$) and disease became more prevalent (Fig. 4; df = 1, F ratio = 3.5, $P = 0.01$). The magnitude of the relative decline in urchin populations following an epidemic increased with urchin density, even after accounting for the significant effect of food limitation (for algal cover, df = 1, F ratio = 10.6, $P = 0.001$).

Epidemics were four times as frequent outside the reserve than inside the reserve (Fig. 2, $N = 90$, chi square = 7.3, $P = 0.007$), supporting the hypothesis that fishing predators increases epidemics in prey populations. However, the persistence of urchin ''barrens'' following epidemics indicated that disease did not replace predators in the trophic cascade.

DISCUSSION

The results appear to support the following scenario. Historically, lobsters, and perhaps other predators (such as sea otters), kept urchin populations at low levels and kelp forests developed as a result of a community-level trophic cascade. Where the main predators were fished, urchins overgrazed algae and ultimately starvation limited urchin population growth. In 1992 an urchin-specific disease entered the area that had urchin populations well exceeding the host-threshold density for epidemics. Epidemics were more probable and led to higher mortality in dense urchin populations so that disease acted as a density-dependent mortality source. Epidemics did not reduce urchin populations to the extent that predators did, and algae remained overgrazed. In this sense, the disease did not replace the ability of predators to control grazer populations.

As predicted by epidemiological theory, the probability of observing an epidemic during a particular year was strongly associated with the density of urchins at a site. One alternative explanation for this association is that observers were more likely to come across a sick urchin in areas where they saw many urchins. Although an effect of sampling effort (hosts seen) could operate to increase the chance of observing sick individuals, the extent to which this could fully explain the observed patterns seen here seems small and would be difficult to determine. Neither food availability nor temperature appeared to influence disease. Although temperature was associated with disease in Nova Scotia (Scheibling and Hennigar 1997), this was not the case in other studies (Margosian et al. 1987, Lessios 1988) nor did it seem to be the case in this study.

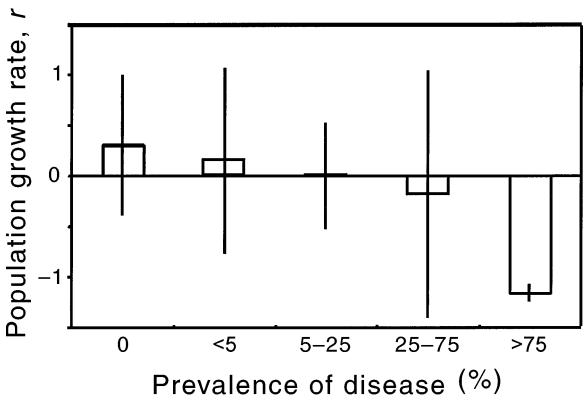

FIG. 4. Association between the prevalence of sick urchins observed and the subsequent growth rate of the urchin population based on 16 sites and 10 years of observations. Growth rate, r, is calculated as the change in density, N, between years t and $t + 1$ according to $r = \ln(N_{t+1}/N_t)$. Data are means ±1 SD. For log density of urchins, df = 1, F ratio = 10.6, $P = 0.0014$.

It was not surprising that factors besides disease were associated with the year-to-year change in density. The substantial year-to-year variation could have been affected by high annual fluctuations in urchin recruitment (Estes and Duggins 1995), though, as mentioned before, this did not explain comparisons between fished and protected sites. Low food availability also led to reductions in density, though this effect may not be as simple as starvation. Although some urchins may starve to death, urchins can persist on little food and feed well on drift algae (Harrold and Reed 1985). However, hungry urchins move to exposed locations where they are more susceptible to predators and surge (Ebeling et al. 1985).

There was evidence that populations declined following an epidemic and that the magnitude of this decline was associated with the prevalence of the disease. This was most clear for cases where over 75% of the urchins were seen to be sick. The negative association between urchin density and the subsequent change in density the year following an epidemic is consistent with epidemiological theory, but this pattern can be difficult to distinguish from random variation around mean annual changes in density (e.g., on average, high densities will be followed by a decrease in density).

Although disease contributes to population regulation by reducing urchin population growth rates at high density, it did not result in widespread mass mortalities. For this reason, disease did not replace predation in the trophic cascade; despite epidemics, urchin populations remained dense and algae were still overgrazed. One potential reason is that disease and predation differ in the extent to which they are specific to host or prey. Spiny lobsters are effective predators on urchins (especially small purple urchins), and, because they can switch to a wide range of prey when urchins are rare (unlike disease), lobsters can still thrive where urchin densities are low (Tegner and Levin 1983). Diseases are more likely to be host specific than predators are to be prey specific and this makes diseases less effective at persisting at low host densities than a predator that can switch to a new prey species. An important exception is when large domestic populations of closely related hosts are kept near wild hosts; the majority of diseases of conservation concern occur in this manner (Lafferty and Gerber 2002). In addition, unlike the effects of predation, the disease is not 100% lethal and an unknown, but potentially substantial, proportion of the infected hosts survive. There is some evidence that catastrophic diseases can lead to cascades in other systems. For example, urchin epidemics in Nova Scotia are followed by the recovery of kelp beds (Scheibling 1986), and epidemics nearly extirpated *Diadema* urchins from the Carribean (Lessios 1988), resulting in recent increases in macroalgae (Hughes 1994).

In the Channel Islands, urchins were dense enough to have experienced just as much disease prior to 1992 as they did after 1992. Before the disease was reported (1982–1991), 48 out of 151 (or 32%) site–year combinations had urchin densities over the host-density threshold for epidemics. After the disease was reported (1992–2001), 52 out of 160 site–year combinations had urchin densities over the threshold. Therefore, urchin populations could have supported a disease if it was there and it seems very unlikely that a common disease in an abundant species would have been completely overlooked by the trained park biologists. It seems plausible that this disease arrived in the region from somewhere else. A very similar disease was observed in the 1980s several hundred kilometers to the north in Santa Cruz County, California (Gilles and Pearse 1986). The year 1992 was a strong El Niño year and the associated change in current patterns could have established links between the normally isolated Channel Islands and already diseased urchin populations elsewhere (presumably on the mainland, near the eastern Channel Islands). If this was the case, the spread was rapid as it occurred at all islands in 1992 (except San Miguel Island where urchin densities were relatively low and disease has not been observed by the monitoring program). Interestingly, an intracellular bacterial pathogen of unknown origin led to mass mortalities of black abalones at many of these same sites during the period 1985–1992 (Lafferty and Kuris 1993); this disease also came on the heels of a large El Niño event. In other cases where urchin diseases have been reported, urchin predators have been heavily fished and urchins reach high densities (Hughes 1994, Sala et al. 1998, Babcock et al. 1999, Tegner and Dayton 2000, Shears and Babcock 2002) that may have created opportunities for diseases to invade.

Conclusions

The paradigms of trophic cascades and host-density thresholds for the transmission of disease have population density as a common factor. The lobster–urchin–kelp community-level cascade alters conditions for epidemics in urchins. Only in marine reserves are lobsters apparently still abundant enough to prevent sea urchins from greatly exceeding the host-density threshold for epidemics and experiencing disease dynamics. These results indicate that overfishing can have unanticipated indirect effects. This dramatic shift in community states would be difficult to predict from a single-species management approach and could have broad-scale effects on other species of ecological and economic interest. Increased disease, primarily in non-fished purple urchins, also affects red urchins, which have historically supported a lucrative export fishery. The indirect effect of lobster fishing on the health of red sea urchins has not been considered.

There has been substantial interest in how environmental change, in general, can contribute to emerging diseases (Daszak et al. 2001). Some have suggested that environmental change has led to a general increase in disease in the ocean (Ritchie et al. 2001). There has

been a significant increase in the reports of disease in urchins in the scientific literature (normalized to overall annual publication rates on urchins) over the last three decades (Ward and Lafferty 2004). It seems plausible that increased reports of disease in urchins could be related to the fact that urchin predators are often fished. But this does not mean that fishing increases disease. More typically, fishing should decrease disease of fished species by reducing host densities (Lafferty and Kuris 1999); it is only when fishing interacts with trophic cascades that disease may emerge in the prey of fished species. Managing marine resources increasingly requires knowledge and understanding of indirect effects. For kelp forest ecosystems, loss of kelp forests and disease in urchins are two indirect consequences of fishing that may have economic and ecological costs. This underscores the value of having protected areas, both for preserving historical conditions for future generations and also for permitting a better understanding of ecological dynamics.

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