

The iconic keystone predator has a pathogen

In any community, a keystone species is one that has an effect that is disproportionate to its abundance. A species that acts as a keystone predator consumes the competitively dominant species, leading to an overall increase in local species diversity. The earliest and still one of the best examples of such a predator is the ochre seastar (*Pisaster ochraceus*). Along western North America's rocky shorelines from Alaska to Baja (Figure 1), *Pisaster's* appetite for the California mussel (*Mytilus californianus*) is well documented. Foundational experiments on Makah Tribal Lands in Washington State by one of us (RTP) in the 1960s showed that when *Pisaster* was intentionally excluded from a rocky intertidal slope, the mussel population advanced toward the lower reaches of the intertidal zone and competitively eliminated a rich diversity of other species (Paine 1966, 1974). Two of the mussel's attributes – the strength of its byssal threads (used to attach itself firmly to surfaces) and the ability to grow in dense aggregations – enabled the mollusk to dominate mid and lower intertidal areas when their *Pisaster* predator was removed. In contrast, when the *Pisaster* population was left undisturbed, the resultant food web comprised a mosaic of species, including limpets, chitons, anemones, hydrocorals, sponges, barnacles, and seaweeds. Because *Pisaster* does not consume anemones, sponges, or seaweeds, these and other organisms benefit indirectly from the mussel-free space created by *Pisaster*. Furthermore, anemones receive indirect feeding benefits (dislodged food items) when *Pisaster* is present (Dayton 1973).

But what happens when a keystone predator is threatened by an important new enemy? In recent years, a pathogen of seastars has swept through the northeast Pacific Ocean. Once its characteristic lesions are observed on infected individuals (Figure 2), seastar populations (including those of *Pisaster*) decline drastically within weeks to months. At locations where there were once densities of 6.9 seastars per square meter (Paine 1974), our surveys show there is now only an individual or two across 100 square meters of rocky shore, a 92% decline in density. Citizen scientists have been crucial in recording incidences of this so-called “seastar wasting syndrome”, which has reduced seastar populations to historical lows (<http://bit.ly/1o5bWNi>).

Echinoderm populations are notoriously sensitive to pathogen-driven fluctuations in abundance. In California during the 1970s, populations of the red sea urchin (*Strongylocentrotus franciscanus*) suffered from disease-like symptoms that led to mass mortality (Pearse *et al.* 1977). Likewise, in the northwest Atlantic during the 1980s and 1990s, the closely related green sea urchin (*Strongylocentrotus droebachiensis*) was subject to similar die-off events; in this

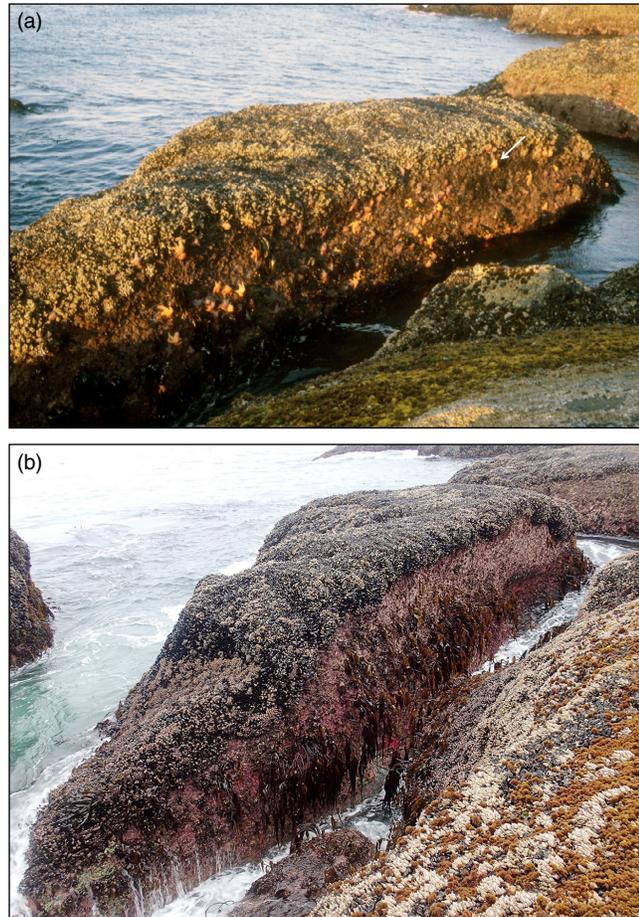


Figure 1. Images from Tatoosh Island, WA, showing changes in the relative abundance of *Pisaster ochraceus* over time. (a) Numerous *Pisaster* (one identified with an arrow) are distributed across a large central rock in this August 1986 photograph. (b) The same rock in June 2015, with no evidence of *Pisaster* presence. The rock remained devoid of *Pisaster* through August 2015.

case, a temperature-sensitive amoeba was identified as the causative agent (Scheibling and Hennigar 1997). Other seastars with a history of disease include the crown-of-thorns seastar (*Acanthaster planci*) in the southern Pacific (Pratchett 1999); the sun star (*Heliaster kubiniji*) in the Gulf of California (Dungan *et al.* 1982); and the black-spined sea urchin (*Diadema antillarum*) in the Caribbean, which succumbed basin-wide to a pathogen – with a ~98% mortality rate – in the early 1980s (Lessios 1988). As with *Pisaster*, *Diadema* has strong influences on food web structure. The loss of the herbivorous *Diadema*, which consumed seaweeds, is thought to have exacerbated coral reef decline by allowing macroalgae to increase in abundance (Carpenter 1990).



Figure 2. *Pisaster ochraceus* with arm lesions (denoted by arrow) on Tatoosh Island, WA, circa September 2014. The seastar also exhibits relatively “limp” muscle tone, which characterized many affected seastars.

Although a virus is implicated in seastar wasting syndrome (Hewson *et al.* 2014), questions remain about the origin of the agent, its transmission, and prevalence. Has this virus been a persistent part of the seastar’s microbiome and evolutionary history? Has an environmental trigger ignited the new epidemic? What are the expected consequences to this food web?

On the basis of research conducted in coastal Washington, Oregon, and British Columbia, we expect that the California mussel’s abundance will increase and that its range will extend lower into the intertidal zone. Seastars are not the only cause of death for mussels; although constantly replenishing the plankton on which mussels filter-feed, waves and currents become an agent of mortality during storms. Thus, in areas impacted by waves that physically dislodge mussels and generate open patches in the mussel bed (Paine and Levin 1981), the other members of the intertidal community will persist in this changing spatial mosaic of opportunity. In calmer embayments, however, mussels may become dominant until *Pisaster* populations recover. California mussels may also grow to a large enough size to escape predation. Very large mussels can resist the tenacious grip of the seastar and its remarkable ratcheting muscles that take only seconds to activate as it tries to pry open the bivalve’s shell.

The outcome of this pathogen–*Pisaster*–prey interaction in the northeast Pacific will depend on three key aspects, the first of which is to determine if the current pathogen outbreak represents a singular occurrence or a periodic event driven by climate fluctuations and/or host–pathogen dynamics. As we learn more about the

virus in natural settings, we can determine whether it will always be a driver of disease outbreak.

The second key aspect of this three-way interaction is whether *Pisaster* can recover. Some areas of the north-east Pacific are already seeing the recruitment of young *Pisaster*, suggesting that successful spawning must have occurred even as the pathogen moved through the population. At Tatoosh Island, some adults remain. However, three factors – the separation of the sexes (ie self-fertilization is not possible), the current low density of adults, and broadcast spawning of gametes – could result in depressed fertilization rates (eg Levitan *et al.* 1992) and a reduced likelihood of population recovery.

The third aspect of this interaction is whether the wide scale of *Pisaster* extirpation will change the capacity of the community to respond. What if mussel body size can surpass a threshold so as to impede *Pisaster* predation and limit *Pisaster* population performance? Delayed recovery and possible transitions in ecological state might result. Further ecological twists are possible in a future where calcium carbonate shells may be more costly to construct as ocean acidification continues, where there is the potential for changing seawater temperatures, and where sea level is rising. The vast scale of this pathogen provides an opportunity to ask whether community response is similar to the smaller-scale *Pisaster* experimental removals of the past, in which RTP and others assumed the role of “pathogen”, or if the depletion of *Pisaster* over large spatial scales, compounded with global change in the ocean, changes the rules of coexistence.

Although we have focused on *Pisaster*, one of the allures of studying rocky shores occasionally exposed to heavy wave action is their amazing and conspicuous species richness. *Pisaster* shares the Tatoosh intertidal zone with at least nine other seastar genera, eight of which have shown similar disease symptoms, and four of which appear to have gone locally extinct. Currently, the esthetic loss is obvious; however, the future reassembly of the entire community will be influenced by their recovery. Seastar wasting syndrome offers a large geographic scale to probe the dynamics of recovery and change. We have much to learn by observing the aftermath of the loss of a keystone predator.

Supporting Information

References may be found in the online version of this article at <http://onlinelibrary.wiley.com/doi/10.1002/fee.1292/supinfo>

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