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Starfish Baby Boom Surprises Biologists

Hundreds of babies have appeared off the Oregon coast, but it's unclear how the youngsters will fare as they face a deadly wasting disease.



Sea stars off the Oregon coast.
PHOTOGRAPH BY AURORA VIA ALAMY



By **Michelle Z. Donahue**

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The devastation caused by a viral infection of sea stars along the United States Pacific coastline shows few signs of abating, continuing to [kill its victims](#) in a swift and grisly fashion.

Yet against those odds, a huge new crop of sea star babies popped up after last year's spawning season, surprising researchers who have been tracking the epidemic.

[Bruce Menge](#), an Oregon State University marine ecologist who has studied sea stars along the coast for the past 30 years, found an unprecedented surge of babies, mostly ochre sea stars, at nine study sites in Oregon. In most of the areas, that meant around 300 times as many babies as in previous years.

"It was a pretty big surprise, actually," Menge says. "We thought there could have been a response, but to see the extent of it, the magnitude, was very surprising."

In some areas, the young sea stars were so abundant "you could hardly miss them," despite the fact they are each roughly the size of a dime, he says.



Why Are So Many Starfish Dying? Sea stars (starfish) along North America's west coast have been dying at an alarming rate. A syndrome known as sea star wasting disease causes the animal to lose limbs and eventually disintegrate, leaving behind a pile of white goo.

But it's still unclear whether the virus thought to be at the heart of the epidemic, the sea star-associated densovirus, will affect these juveniles as it has the adults.

The wasting disease can kill an adult animal in as little as three days. Early symptoms appear as pockmarked lesions on the sea star's body, followed by twisted, contorted arms and the inability to cling to rocks and other surfaces.

At the end, the stars disintegrate into a slimy white sludge. Since 2014, approximately 20 species of sea stars along the Pacific coast have seen population losses between 60 and 90 percent.

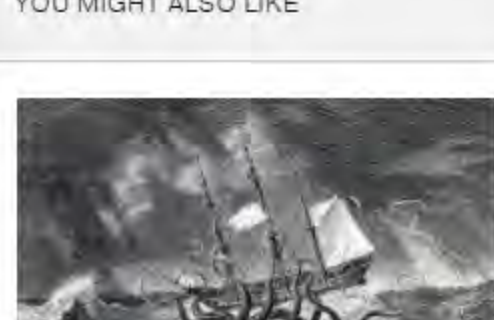
Make Way for Sea Starlets

Though the scale of the current epidemic is unlike anything seen before, sea star wasting disease isn't new, nor is the virus that is suspected of causing it. Similar, smaller outbreaks occurred during El Niño events in the 1980s and '90s, and the densovirus was found in preserved sea star specimens dating to the 1940s.

Such diseases were classically associated with warm-water regions of the world, such as in the Caribbean, where a major epidemic wiped out sea urchins in the 1980s. Why the virus has shown up in cooler northern waters is still a topic of investigation, though warming ocean trends have been tagged as a possible cause. It could also be a combination of factors, including ocean acidification, that is making the sea stars more vulnerable.

"Echinoderms are a boom-bust phenomenon," says [Ian Hewson](#), the Cornell researcher who first identified densovirus as the probable culprit of the Pacific epidemic. He's currently investigating how the virus works and how it's transmitted.

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"They undergo these massive mortality events and swing back strongly. If you talk to fishermen in the Gulf of Maine, they'll tell you they see sea star die-offs every 20 to 30 years. But we've never seen anything quite so bad [as the current outbreak]."

The working theory to explain the latest swell of babies is the very fact that there are fewer adults. Voracious eaters, adult sea stars mow their way through full-size mussels, barnacles, and other prey in the first half of the year, moving on to smaller animals by the summer.

It's at this time of year when young sea stars, newly transformed from their larval stage, begin to migrate out of the water column in greater numbers. Fewer hungry adults means more food for growing babies, allowing more of them to survive.

Baby booms like the one in Oregon [have been seen in previous years](#) in California, but they were based only on anecdotal observations and not formally quantified, says Hewson. Tallying up the absolute numbers of new sea stars gives researchers a better insight into how the remaining populations might be responding to the onslaught.

[Pete Raimondi](#), a University of California, Santa Cruz, marine ecologist who has worked with Menge in the past, says that though it's too early to toss around words like "turnaround," the surge in sea star babies is a positive sign.

"One thing's for sure: You won't get populations to rebuild unless you have babies," Raimondi says. "It's a huge, good sign. But it's too soon to be optimistic, because we don't know their fate. We'll see what happens in a couple of years when they grow past the baby stage."

Evolving Resistance?

The hope now is that, with such a large and well-documented surge in new sea stars, some of them might be found to show resistance to the disease.



For instance, recent work by [John Wares](#) of the University of Georgia and graduate student Lauren Schiebelhut at the University of California, Merced, has revealed that some sea stars carry a gene that seems to be connected with viral resistance.



The gene, known as elongation factor 1-alpha, or EF1-A, has a version that shows up more often in healthy animals than diseased animals, though researchers still aren't sure why, or even what specific role the gene plays.

In mammals, EF1-A is associated with cell growth, among other functions, though in sea stars it may be linked to a heightened immune response. Ironically, the presence of the gene in a slightly different form impairs protein synthesis and is usually lethal to sea stars.

"It's like sickle cell disease. It's in higher proportions in the population than you'd expect normally because it has this lethal effect," says [Michael Dawson](#), a UC Merced evolutionary ecologist and Schiebelhut's advisor. "This disease outbreak allowed us to test the question of why."

The next step is to do a full genomic analysis of *Pisaster* that might show other positive immune response mutations related to the EF1-A gene.

"Unfortunately, it's a watching and waiting game," Menge says. "Any kind of mitigation efforts would be thwarted by the scale of the problem. The only thing we can really do is try to understand it. But all is not lost."

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