Sulfurous chemical known as 'smell of the sea' serves as clarion call for coral pathogens

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Roman Stocker dives among corals.

Coral is really an ecosystem within the reef ecosystem: a colony of invertebrate polyps that excrete a calcium-carbonate skeleton. Living within the polyps are photosynthetic algae that produce nutrients the polyps use as food. When a coral is stressed by rising water

temperatures, it expels the algae, causing bleaching. This consequently increases the coral's stress to a greater extent, making it more susceptible to attack by pathogenic bacteria.

While the steep decline in the health of coral reefs has prompted additional scientific study, little is known about the ecological interactions between the pathogens and the weakened coral at the microscale.

However, researchers at MIT have identified one mechanism by which pathogenic bacteria identify their prey: They've found that stressed *Pocillopora damicornis* coral produce up to five times more of a sulfurous compound called dimethylsulfoniopropionate (DMSP). The abundance of DMSP appears to serve as a clarion call, inciting the pathogen cells, which sense the amplified chemical and charge in for attack, changing their swimming direction and speed as they home in on the weakened coral.

"This is the first time we've been able to sneak a peek at a coral pathogen's behavior in real time, as it responds to the chemical cues leaking into the seawater from its host," says postdoc Melissa Garren of MIT's Department of Civil and Environmental Engineering (CEE), first author on a paper about this work that appears Dec. 12 in the *International Society for Microbial Ecology Journal*. Professor Roman Stocker of CEE is lead researcher on the project.

"By doing so, we discovered that these tiny cells have an amazing array of tricks up their sleeves for finding a host, and that they can preferentially navigate toward hosts that are stressed," Garren says.

DMSP—whose sulfur smell is familiar to anyone who's been on a beach at low tide—is just one of the many molecules in the mucus covering the coral's surface. The coral produces the mucus continuously as a means of

cleansing and defense, and DMSP is produced both by the coral polyp and its symbiotic algae. However, no one knows exactly why the mucus contains elevated levels of DMSP during periods of stress or why the pathogens home in on that specific molecule.

Stocker had shown in earlier research unrelated to corals that ocean microbes are attracted to DMSP and will swim along chemical gradients—a behavior called chemotaxis—to reach it. However, this is the first study to show that DMSP attracts coral pathogens and that the pathogens are able to alter their swimming direction as well as their speed—a behavior called chemokinesis—to reach their targets.

In the field, Stocker and Garren collected small amounts of coral from the Great Barrier Reef and performed experiments at Heron Island Research Station, subjecting coral samples to a water temperature increase of 1.5 degrees daily over a week. Coral fragments from all donor colonies displayed the same response to the heat, exuding five times more DMSP in their mucus than the control samples, which remained at ambient temperatures.

Back in the lab, the researchers used microfluidics and videomicroscopy to test the swimming directions and speeds of the coral pathogen *Vibrio corallilyticus*. When in the presence of the mucus, the bacterium increased its swimming speed by up to 50 percent. Once the cells reached the richest layer of mucus, their movement went back to normal speed, about 50 body lengths per second.

Surprisingly, unlike many other marine bacteria that use DMSP as an important source of food, this *Vibrio* didn't metabolize DMSP at all, indicating that the chemical compound may serve purely as a signal to attack.

"I'm intrigued by how certain key processes keep popping up in

oceanography—as if there was some universality to them," Stocker says. "Because DMSP is involved in chemical signaling among so many other marine animals—ranging from birds to turtles to fish to seagulls, and now coral pathogens—it seems that the chemical must be the currency of signaling and sensing in the sea, though no one really knows why."

To ensure that what they were seeing was actually chemokinesis, Kwangmin Son, a graduate student in Stocker's lab, created a mathematical model that simulated what the scientists had observed: the swimming of bacteria toward the mucus layer. In the model, the accumulation of bacteria in the mucus layer was 50 percent higher and 50 percent faster than if the microbes had not changed their swimming speed.

"This research by Dr. Garren and her colleagues has tremendous implications for the way we study coral disease and host-pathogen interactions," says Courtney Couch, a postdoc at the Hawaii Institute of Marine Biology who specializes in coral disease ecology. "Thanks to their innovative research, we can for the first time visualize how microbes migrate toward corals, which is a fundamental part of the infection process."

Co-authors on the paper, in addition to Garren, Stocker, and Son, are postdocs Roberto Rusconi and Filippo Menolascina and former MIT postdoc Orr Shapiro of Stocker's lab; Justin Seymour and Jessica Tout of the University of Technology, Sydney; and David Bourne and Jean-Baptiste Raina of the Australian Institute of Marine Sciences. The research was funded by the National Science Foundation's Human Frontiers in Science Program.

"These experiments have helped us get one step closer to understanding the mechanisms for coral disease, because we have been able to directly visualize the microscopic pathogens of the corals as they swim with great

vigor towards the coral surface," Stocker says. "It goes without saying that this access to the microscale provides a whole new appreciation for the mechanisms of disease."

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