Coral reefs in warming seas

December 9 2011, By Steven Powell

Disease outbreaks are often associated with hot weather. Because many bacteria typically multiply more rapidly in warmer conditions, it's a commonly held notion that warm-weather outbreaks are a straightforward consequence of greater numbers of the microbial culprit.

But a team of researchers, led by the University of South Carolina's Pamela Morris, has uncovered some unexpected details in the temperature-dependent virulence of *Vibrio coralliilyticus*, a marine pathogen found across the globe. It's not an increase in bacterial abundance, but rather the way a small temperature increase modifies protein expression that makes the bacterium so destructive.

"We have a lot of pathogens that we think of as being more dangerous in the summer months," said Morris, a marine scientist with USC's Belle W. Baruch Institute of Marine and Coastal Sciences. "Vibrio cholera is one--there are more cholera outbreaks when it's warm. We also have more coral diseases in the warmer summer months."

"But surprisingly enough," she added, "when you start looking at what is known about the effect of temperature on the virulence of an organism--any organism--very little is known about what's really going on. Most people assume that we have more diseases in the summer months because the pathogens grow faster--there's more of it. But we have demonstrated in this study that at two different temperatures, the abundance of the organism was the same, but that certain virulence-related genes were more highly expressed at the warmer temperature."

The focus of the study was Vibrio coralliilyticus. This bacterial species has been shown to be able to cause coral diseases like bacterial bleaching, where an infection of healthy coral results in the loss of pigmented algae (hence the term bleaching).

The Vibrio coralliilyticus infection is highly temperature dependent: at 24 degrees Centigrade, one particular strain is not virulent, but it has been shown that in water warmed just 3 degrees to 27 degrees C, it will invade and damage the tissue in the coral species Pocillopora damicornis.

In a report published Dec. 8 in Nature Publishing Group's ISME Journal, Morris's team established that both the cell's growth curve and the total protein produced by the pathogen were essentially identical at the two temperatures. But when they looked at the individual proteins that Vibrio coralliilyticus makes, 27 degrees C turned out to be a hothouse for producing the wrong sort of enzymes.

The team, which also included Nikole Kimes of USC as well as Christopher Grim and Rita Colwell of the University of Maryland, sequenced the entire genome of Vibrio coralliilyticus and characterized its proteome, identifying more than 600 virulence-related genes that fall under five different categories: chemotaxis or motility, host degradation, microbial resistance, secretion, and transcriptional regulation. Fewer than 30 of these were significantly up-regulated at 24 degrees C, but more than 130 genes were significantly up-regulated when the temperature was increased just 3 degrees C.

Several lines of evidence were used to conclude that the temperature-dependent virulence of the Vibrio coralliilyticus could be attributed to the change in protein expression. "The key to the study was carefully choosing environmentally relevant temperatures for comparison," said Morris. "What we see is a temperature effect, not an abundance effect.

Unlinking those two things was critical."

They see their results as important not just for assessing coral vulnerability in the face of increasing sea surface temperatures, but also for addressing the largely under-explored means by which modestly higher temperatures trigger virulence in a much wider wide range of pathogenic organisms.

"We're really proposing Vibrio coralliilyticus as a model pathogen for temperature-dependent virulence, not just in corals, but more broadly," said Morris. "Even in cholera, they don't know much about the temperature trigger. We're trying to find the mechanism responsible for the initial trigger, which then results in a cascade of virulence factors that result in disease."

One way to do that is to use the new genomic and proteomic information against the pathogen. "We can generate targeted mutants and then do coral infection studies," said Morris. "We hope to find out which of these genes we could knock out that would make this organism no longer pathogenic. That allows you to know what to target if you were going to develop a technique to inhibit the organism."

Provided by University of South Carolina