# Temperature influences gene expression, life cycle in vibrio cholerae

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*Vibrio cholerae* infects roughly four million people annually, worldwide, causing severe diarrheal disease, and killing an estimated 140,000 people. Its success as a pathogen belies the challenges this bacterium faces. The waters this bacterium inhabits when it's not infecting H. sapiens can be 40 degrees Fahrenheit cooler than our normal body temperature. Now a team of investigators from the University of California, Santa Cruz provides new insights into how different temperatures in the bacterium's environment control expression of genes required for life at those temperatures. The research is published ahead of print May 20, 2016 in *Applied and Environmental Microbiology*, a journal of the American Society for Microbiology.

In the study, the investigators grew *V. cholera* at human body temperatures, and then shifted them to the temperatures of the waters they normally inhabit when they are not infecting people, said corresponding author Fitnat Yildiz, PhD, professor of Microbiology and Environmental Toxicology, the University of California, Santa Cruz.

Loni Townsley, Yildiz' student who performed the experiments, used microarrays to determine which genes were expressed at the various temperatures. Low environmental temperatures of around 15oC (59oF), turn on genes that regulate biofilm formation, genes that code for a molecular weapon that secretes toxins, and genes that enable adaptation to cold.

Biofilms are tough microscopic mats of bacteria. These enable *V.*

*cholerae* to abide hardily at the low environmental temperatures temperatures, growing on a species of zooplankton, Daphnia magna, which is a tiny crustacean. "*V. Cholerae* in biofilms are protected, sheltered," said Yildiz. (Growing on millions of zooplankton makes the V. cholerae independently mobile, raising the chances of encountering a human host to infect.)

As for the molecular weapon, a device called a "type VI secretion system," it was discovered only recently, said Yildiz. Although knowledge about the secretion system is increasing rapidly, much remains to be learned. "We do know that it is used to kill both bacterial and eukaryotic cells," said Yildiz. And *V. cholerae* is known to be able to take up DNA from these dead cells, in order to acquire new genes.

Beyond that, it's not certain what purposes killing other bacteria may serve. One speculation is that killing members of other bacterial species that inhabit the surface of Daphnia opens up more space for *V. cholerae* to grow, said Yildiz. Another speculation is that *V. cholerae* may procure nutrients—carbon and nitrogen—from the dead bacteria.

In any case, the investigators found that expression of a protein component of the type VI secretion system was highest at 25$^{o}$C (77$^{o}$F).

The investigators also found that expression of a major regulator of virulence is notably reduced at low temperature. That means that expression of virulence factors is curtailed at low temperatures, which makes sense since they are only needed during infection of a human host—which happens at body temperature, said Yildiz.

Linking the genes that promote biofilm formation, cold adaptation, and production of the toxin-producing system to the Vibrio-zooplankton association, as was done in this study, was a breakthrough in understanding the environmental lifecycle of *V. cholerae*, said Yildiz.

"Our research focuses on environmental pathogenesis, with the goal of increasing our understanding of molecular detection, survival mechanisms, and dissemination dynamics of human pathogens into the environment," said Yildiz. "Our long-term goal is to develop strategies for disease prevention and treatment by identifying targets to reduce environmental survival and infectivity."

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