A Specific Protein, Possibly NO Enable *D. radiodurans* To Cope with UV

Following exposure to ultraviolet (UV) radiation, the ObgE protein of *Deinococcus radiodurans* apparently acts in concert with elevated levels of nitric oxide (NO) “as a general checkpoint system for telling cells to go forward and divide,” enabling them to recover from the stresses of that exposure, says Brian Crane of Cornell University in Ithaca, N.Y. ObgE, a previously uncelebrated gene product, he adds, “probably acts at many different points that involve both protein and DNA synthesis.”

*D. radiodurans*, which can withstand 1,000 times more radiation than can mammalian species, also relies on NO synthase to make NO while recovering from UV radiation. Yet the precise role of NO in this bacterial species as it recovers following radiation is not known. Expression of NO synthase in wild-type *D. radiodurans* increases within 30 minutes following exposure to radiation and remains high for about 6 hours, according to Crane and his collaborators. Cells begin producing NO about 3 hours after being irradiated and high levels persist for another 8 hours. Meanwhile, for mutants lacking NO synthase, growth rates slow by 95%, and such cells take six hours longer to recover exponential growth than do nonmutants following irradiation.

ObgE is another gene that comes into play when cells recover from radiation, the Cornell researchers report. After cells are irradiated, ObgE levels rise in wild-type *D. radiodurans*, but not in mutants missing NO synthase. Adding NO to mutant cells increases expression of ObgE. When irradiated with UV light, *D. radiodurans* cells rapidly make NO, which activates a gene that proves important for accelerating growth and coping with stress. Details appear in the October 27, 2009 *Proceedings of the National Academy of Sciences* (106:18183–18188).

First identified in *Escherichia coli*, ObgE belongs to a superfamily of microbial GTPases whose cellular roles are not well understood. However, where studied, ObgE typically proves essential for cellular life. In *E. coli*, for instance, ObgE controls chromosome segregation and progress through the cell cycle, whereas in *Bacillus subtilis* an ObgE-like protein regulates DNA replication and activates stress response transcription factors.

Although ObgE seems to belong to “a general checkpoint system for telling cells to go forward and divide,” Crane says that this protein likely “acts at many different points that involve both protein and DNA synthesis.” Whether NO activates ObgE in other bacterial species is not known. Bacteria lacking NO synthase might obtain NO through other means such as denitrification, a common event in microbial environments, Crane points out.

The findings “help nail down one way that bacteria use their NO production, which has mostly been a mystery,” says Dennis Stuehr, a professor of molecular medicine at the Cleveland Clinic in Cleveland, Ohio. Crane and colleagues “show fairly conclusively that NO confers the trademark UV resistance to *Deinococcus*.”

Producing NO might be a widely distributed bacterial stress response. For example, some gram-positive bacteria release NO when exposed to antibiotics, a response that increases their resistance to a broad spectrum of antimicrobial agents, according to Evgeny Nudler of New York University Medical School in New York City, whose findings appear in the September 11, 2009 *Science* (325:1380–1384). Crane’s study “expands the unexpected roles for NO synthase,” Nudler says.

“NO [synthesis] could be a general way for bacteria to survive different types of stressors,” Crane says. “In *Deinococcus* it’s radiation, but it could be antibiotic stress in other bacteria.”

Carol Potera

Protozoan *T. gallinae*: Plausible Culprit for Dinosaur Downfalls

The protozoan *Trichomonas gallinae* sometimes infected theropod dinosaurs, including *Tyrannosaurus rex*, riddling their jawbones with erosive lesions, or “holes,” according to Ewan Wolff at the University of Wisconsin, Madison, and collaborators from several U.S. and Australian institutions. Thus, they conclude, microscopic parasites rather than Mesozoic-era predators might explain some otherwise puzzling damage being detected in dinosaur fossils, and also might account for the demise of some dinosaur species.

“We first became interested in trichomonosisis as a possible culprit for the holes that we were finding in tyrannosaur jaws when we evaluated modern crocodile and bird diseases,” Wolff says. “The diseases we were seeing in birds matched up best with what we were seeing in tyrannosaurs. In particular, trichomonosisis, a recurrent problem in birds of prey, caused very similar holes in their jaws.” He discounts an alternative explanation that the holes in dinosaur bones are from bite-marks and related traumas. *T. rex* bite marks are “unmistakable” and not “what we were seeing,” he says.

Among 61 tyrannosaurid specimens, Wolff and his team identified 10 *T. rex* individuals with comparable full-thickness erosive lesions. The specimens occupied a 10-million-year stretch from about 75 million years ago to 65 million years ago, and they were found within a geographical region in North America that encompasses the province of Alberta in Can-