For many insects, winter survival is in the genes

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Many insects living in northern climates don't die at the first signs of cold weather. Rather, new research suggests that they use a number of specialized proteins to survive the chilly months.

These so-called "heat-shock proteins" ensure that the insects will be back to bug us come spring.

A study of flesh flies and a handful of other insects suggests that they have an arsenal of protective heat-shock proteins that are turned on almost as soon as the temperature dips. Until this new study, researchers knew of only two such proteins that were activated in flesh flies during cooler weather.

"Insects need heat-shock proteins in order to survive," said David Denlinger, the study's lead author and a professor of entomology at Ohio State University. "Without these proteins, insects can't bear the cold and will ultimately die."

Denlinger and his colleagues found nearly a dozen additional heat-shock proteins that are activated during diapause, a hibernation-like state that insects enter when temperatures drop. Insects can stay in this state of arrested development for several months.

"We certainly didn't expect to find that many proteins active during diapause," Denlinger said. The researchers report their findings in the current online early edition of the Proceedings of the National Academy

of Sciences.

Insects and other animals, including humans, produce heat-shock proteins in response to extremely high temperatures. The proteins are so named because they were initially discovered in fruit flies that were exposed to high heat. Humans make these proteins when we run a high fever.

"But insects make these very same stress proteins during times of low temperature as well as during exposure to high levels of toxic chemicals, dehydration and even desiccation," Denlinger said.

He and his colleagues first figured out how many genes were turned on only during the flesh fly's dormant state. The researchers extracted and compared RNA from both dormant and non-dormant fly pupae – the developmental stage between larva and adulthood. They used a laboratory technique that let them separate out genes that were turned on only in the flies in this dormant state.

The researchers found 11 previously undiscovered genes that turn on heat-shock proteins during diapause. Until this study, they had only known of two such proteins.

Denlinger and his team also examined the expression of one of those previously discovered heat-shock proteins, Hsp70, in five additional insect species that aren't related to the flesh fly. Each insect is a fairly common agricultural pest: the gypsy moth, the European corn borer, the walnut husk maggot, the apple maggot and the tobacco hornworm. Collectively, these species cause millions of dollars of damage annually.

Hsp70 was active while all of the insects were in diapause.

When Denlinger's team knocked out the Hsp70 gene that makes the heat-

shock protein, the insects were unable to survive at a low temperature (in this case, insects were exposed to -15°C, or 5°F.)

"This underscores the essential role of this gene for winter survival, suggesting that this particular heat-shock protein is a major contributor to cold tolerance in insects," Denlinger said. "It's highly likely that the other heat-shock proteins we found during diapause in the flesh fly are also important to an insect's ability to endure months of cold temperatures."

Denlinger has no plans to develop a method to get rid of heat-shock proteins in insect pests, but he says that it is important to understand how insects survive through the winter.

"There may be steps we can take to disrupt the diapause process and make an insect vulnerable to low temperatures," Denlinger said. "At this point, the findings broaden our palette of players that contribute to cold tolerance in insects."

He said the next step is to figure out the unique functions of each heat-shock protein.

"We assume it's not simply redundancy in the system, but that each protein makes a unique contribution somehow," Denlinger said. "This protective mechanism is much more complex than we envisioned."

Source: Ohio State University