# K-State biologist hopes mosquito can break viral chain

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Most people do their best to avoid mosquitoes. But this summer Rollie Clem will play the wary host to his own homegrown swarm of Aedes aegypti, the yellow fever mosquito. He's made a room ready for them, and even a menu.

"Sheep's blood or cow's blood," said Clem, an associate professor of biology at Kansas State University. "This particular species is less finicky than others," so Clem won't need to stock their cages with sweaty socks. (Some mosquitoes won't feed without the persuasive scent of humans in the air.)

Clem, who studies molecular virology, is going out of his way to accommodate A. aegypti in hopes of learning more about how viruses disrupt the programmed death of cells, or apoptosis.

"Millions of cells are dying at any given moment in our body," Clem said. "And that's a good thing."

Programmed cell death is tidier than necrosis, in which injury prompts inflammatory cells to rush in and clean up. In contrast, apoptosis relies on a cell's genes to trigger an orderly disassembly.

It's the body's way of removing tissue that has done its job, such as the webbing between the developing fingers of an embryo, or cells whose DNA is damaged. Malfunctions in apoptosis are associated with cancer, neurological diseases such as Alzheimer's and immune disorders such as

AIDS and rheumatoid arthritis.

Though scientists knew of apoptosis as long ago as the late 1800s, interest in the field has intensified only in the last 15 years, Clem said.

"It was very obscure" when he was a graduate student at the University of Georgia in the early 1990s, he said. "Now it's taught to undergraduates."

Clem's current experiment, helped by a grant from the National Institutes of Health, grew from his work with moths that were naturally immune to fatal viruses. Clem chose A. aegypti for this round because it spreads such diseases as dengue fever, the most important of the world's mosquito-borne viruses. The Centers for Disease Control estimate that 100 million cases occur annually.

But the mosquito's effectiveness in spreading the disease varies from place to place. Clem wants to find out whether apoptosis plays a role in that variability.

Once his mosquitoes are safely housed in their lab, Clem plans to infect them with genetically altered strains of Sindbis, a virus related to those that cause equine encephalitis. Some strains will contain genes that block apoptosis, Clem said, and others will encourage the process.

He will go on to test other mosquitoes with unaltered strains of Sindbis. The results should suggest whether A. aegypti can be made immune to viruses.

Clem stresses that A. aegypti's living arrangements will be anything but casual. His lab's insectary is approved by the CDC to conform to "arthropod containment level 2," which specifies such things as screened drainage, extensive caulking, and heat-sterilization of equipment and

waste. So whatever you're swatting this summer, it won't be one of Clem's mosquitoes.

"Our work will be taking place in a very secure environment," he said.

In the Western Hemisphere, A. aegypti is widespread in Latin America but occupies only bits of the southern United States, particularly south Florida. The species has made a comeback since eradication programs ended in the 1970s, and dengue fever has expanded along with it.

In Kansas, Culex tarsalis and other members of the Culex group do most of the biting. They don't transmit dengue, but C. tarsalis is known to spread West Nile and western equine encephalitis. The world's most troublesome mosquito-borne disease, malaria, is spread by Anopheles gambiae.

One of Clem's students is investigating a dozen or so genes in A. aegypti because of their similarity to genes that control apoptosis in the fruit fly. "But even in humans, the genes are similar in their sequence," Clem said. This phenomenon of "gene homology" means that insects have a lot to tell the species on the other end of the microscope about its own genetic workings.

Source: Kansas State University