



niversity of Arizona researchers have sown the seeds of a virus' destruction in its own

genetic code—or rather, in the genetic code of the organisms it seeks to infect. Their work could improve both the understanding of how viruses work as well as the ability to make plants and animals more virus-resistant.

Working with a virus that infects bacteria, Bentley Fane, a professor in the division of Plant Pathology in the Department of Plant Sciences and a member of the BIO5 Institute, and

The research casts light on the biology of how viruses work and how the proteins they create interact with one another.

“We all have an interest in better understanding both how viruses work and how to stop them from working,” Fane explained.

Viruses are little more than strands of DNA or RNA surrounded by a protein coat; they can't reproduce on their own. Instead they invade the cells of more complex host organisms—everything from bacteria to plants and animals—and hijack the machinery

view of evolution in action. Over the course of 200 generations, Fane and Cherwa have watched the virus evolve a mutant strain that not only can replicate in spite of the inhibitory protein, but that may also, according to some very preliminary research, be somewhat dependent on the protein. In other words, the original strain's inhibitory protein poison just may be the resistant strain's medicine.

That resistant strain would have a hard time surviving outside the lab, because its resistance is pretty much the only thing it has going for it—it is

DEBORAH DAUN



Bentley Fane in laboratory

Engineering a Self-destructing Virus

A tool to learn how viruses work

By University Communications

James Cherwa, a graduate student in Fane's lab, pinpointed a region of a protein that's crucial to building the virus' structure, designed a modified version of that protein, and engineered the bacteria's cells to produce the modified protein.

When the virus infected cells of the bacteria, it “recognized” the modified protein and tried to incorporate the altered protein into copies of itself. Instead the protein gummed up the works of the replication process, causing the virus to die without producing any progeny.

“We were shocked by just how potent the inhibitory protein was,” Fane said.

inside those host cells in order to replicate.

Along the way, viruses can cause any number of diseases, including blights in plants and colds, flu and HIV in humans. Fane hopes to begin using what he's learned to engineer virus-resistant plants. While similar work has been done with plant viruses before, none of those viruses had the icosahedral shape [a solid figure with 20 triangular facets] and structure that is the focus of Fane and Cherwa's research.

The virus they're working with also reproduces quickly—a generation lasts all of about 20 minutes—which means their research provides an up-close

otherwise less healthy than the original virus. “When something mutates, it does so at a cost to its usefulness,” Fane said. “It's always illuminating to see how a virus adapts to something like this, though, because it always manages to. That's the power of evolution and selection.” 🌟

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