Researchers examine 'invading' bacteria in DNA

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Call it advanced warfare on the most elemental of levels.

Researchers at Texas A&M University's Artie McFerrin Department of Chemical Engineering have discovered how certain types of bacteria integrate the DNA that they have captured from invading enemies into their own genetic makeup to increase their chances of survival.

To be more accurate, the genetic material isn't really captured as much as it is simply utilized after it's injected into the bacteria by an invading virus, says Professor Thomas K. Wood, who along with colleagues Xiaoxue Wang and Younghoon Kim has published the findings in Nature's 2009 International Society for Microbial Ecology Journal.

Wood's findings shed light on a millions-of-years-old battle between bacteria and bacteria-eating viruses known as "phages." Locked in an epic struggle, the two life forms, Woods explains, are constantly developing new ways to win the war. One such approach undertaken by a phage is to attach to a bacterial cell and, using a syringe-like tail apparatus, inject its genetic material into the bacterial cell. Once inside, the phage replicates itself and eventually exits the cell to find new bacteria to infect.

But as is the case with men, the best-laid plans of phages can also go astray.

Examining *E. coli* bacteria, Wood found that the bacteria developed a

means of not allowing the phage to replicate and leave the cell of its own volition. Once the phage was effectively "captured," the bacteria incorporated the phage's DNA material into its own chromosomes. This new diverse blend of genetic material, Wood says, has helped the bacteria not only overcome the phage but also flourish at a greater rate than similar bacteria that have not incorporated the phage DNA.

"The bacteria are alive and doing well, and in fact the bacteria are doing better because it captured its enemy," Wood said. "Our research shows that if these bacteria didn't have this particular set of 25 genes that belonged to the old phage it wouldn't be able to grow as fast. If you removed the phage remnant, the bacteria grows five times slower on some carbon sources."

This distinct advantage is helping scientists understand why bacteria carry about 10-20 percent of genes that aren't their own. Simply put, carrying the virus DNA allows bacteria to increase their chances of survival by producing diverse progeny - something Wood says is extremely important when the bacteria choose to move to a new environment through a process known as dispersal.

Dispersal occurs, Woods says, when the bacterium can no longer glean the nutrients it needs from its surroundings or when other environmental conditions, such as temperature, have become unfavorable. Wood found that through an elaborate regulation method, the bacteria are able to retain the virus DNA or expel it. It's an interesting trade off, as retaining the virus DNA helps the bacteria grow faster but reduces its motility, which is needed when seeking out new environments, Wood explains.

Further exploring this dynamic, Wood and his research group were able to link this regulation process to the formation of bacterial communities called biofilms.

A biofilm, Wood says, is a protective, adhesive slime created by bacteria that have joined together to form a community and reap the benefits of a "strength-in-numbers" approach. Biofilms can grow on a variety of living and nonliving surfaces, including submerged rocks, food, teeth (as plaque) and biomedical implants such as knee and hip replacements.

The National Institutes of Health estimate that about 90 percent of infections in humans are caused by biofilms, and the Centers for Disease Control estimate biofilm to be present in 65 percent of hospital-acquired (nosocomial) infections. Biofilms typically are the cause of fatal infections that develop post surgery. More commonly, they are the source of persistent ear infections among children.

In addition to finding that biofilm formation relies heavily on virus genes present within the bacteria, Wood's research has shown the mechanism for how this takes place. A protein within the bacterium called Hha has the ability to control whether virus genes are kept within the bacterium or jettisoned. When Hha is basically "turned on," the bacteria expel the virus genes, opting for motility over the ability to form biofilms. Likewise, when Hha is not expressed, the bacteria move slower but grow biofilms at a much faster rate, Wood explains.

It's a finding that could impact everything from health care to research into alternative fuel production.

"If we can understand how biofilms are formed, we can begin to manipulate forming them where we want and getting them to not form where we don't want them," Wood says. "We have found a regulator - this Hha - that controls the genes related to biofilm formation. Now we can begin to envision ways to turn on that Hha gene if we want to get rid of biofilms, and that is what we are working on. That's the long-term goal - as engineers to make biofilms where we want them.

"For example, if we want to remediate soil, we'd form a biofilm on the roots of plants, plant the tree, and wherever the tree root goes we clean the soil. That's a beneficial biofilm. If I want to make hydrogen with *E. coli*, I'll probably want to do it in a biofilm, so I would want to promote the growth of the biofilm.

"We're one of the first labs in the world that has begun to not only try to understand how biofilms form but to control them."

Source: Texas A&M University