

Sugar on bacteria surface serves as base for a web of resistance

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The bacteria responsible for chronic infections in cystic fibrosis patients use one of the sugars on the germs' surface to start building a structure that helps the microbes resist efforts to kill them, new research shows.

Scientists have determined that the bacterial [cell-surface](#) sugar, a polysaccharide called Psl, is anchored on the surface of the bacterium as a helix, providing a structure that encourages cell-to-cell interaction. When multiple [bacterial cells](#) join together with the help of such a structure, they form what is called a biofilm, a persistent community of bugs that is able to resist the effects of a human [immune response](#), as well as antibiotic drugs.

In the case of the bacterium being studied, *Pseudomonas aeruginosa*, the results of biofilm development can be lethal. Chronic infection with these bacteria is what causes the death of most patients with [cystic fibrosis](#), a genetic disease characterized in part by excess mucus in the lungs - a condition that can foster creation of the biofilm. Some critically ill hospitalized patients also are susceptible to infection by *Pseudomonas*, which can lead to sepsis or bacteremia, common causes of death in very sick intensive-care patients.

With Psl clearly identified as the foundation of the matrix, or the architectural glue that holds a biofilm together, scientists hope the research could lead to therapies that target the sugar and thus prevent the development of these masses of bacterial cells.

"From a clinical perspective, if we can figure out ways to inhibit or destroy Psl, we could free up these organisms and they'd be much more readily killed by antibiotics or by the host's defense," said Daniel Wozniak, professor of internal medicine and microbiology at Ohio State University and senior author of the study.

"Many antibiotics target actively growing cells, but biofilms go into a dormant mode of growth. This comes into play with resistance."

The research appears in a recent issue of the journal [PLoS Pathogens](#), published by the Public Library of Science.

Wozniak and colleagues had singled out Psl as the likely culprit in Pseudomonas biofilm creation a few years ago, but weren't certain about how it worked until this new series of experiments allowed them to view the entire matrix and biofilm development process.

The researchers are the first to note that the actual shape of Psl resembles a helix, a ladder-like structure, which stabilizes cell-to-cell interaction critical to the formation of a biofilm. Wozniak's group also observed that free-floating DNA, with its double-helix shape, exists within the Pseudomonas biofilm, offering additional web-like structural support to the mass of cells.

"The extracellular DNA works like another matrix. It's a long polymer that attracts all the bacteria together," said Luyan Ma, lead author of the study and a research scientist in Ohio State's Center for Microbial Interface Biology (CMIB).

The biofilm eventually grows into a three-dimensional mushroom-shaped mass. And then, in a programmed series of events, the biofilm initiates a process to create another microcolony of cells.

"At some point, there is either a nutritional or physiological cue that says, 'this is no longer advantageous and we need to start this process over again'," said Wozniak, also an investigator in CMIB.

As part of this process, some bacterial cells in the biofilm kill themselves to create a cavity from which seed cells can be released to begin the formation of a new mass. This kind of programmed cell death, called apoptosis, is typical of higher organisms, but has rarely been seen in bacteria, Wozniak said.

The researchers believe this cell death disrupts the matrix holding the existing biofilm together, releasing Psl and a host of nutrients and enzymes that support new "swimming" cells that move out of the biofilm to occupy a new surface and begin matrix and biofilm development all over again. The cell death is also believed to be the source of the extracellular DNA that helps maintain the biofilm structure during this release of seed cells.

"We all think of DNA as an information storage element in our cells, but it's becoming clear that DNA has roles outside of that capacity," Wozniak said.

Wozniak and colleagues introduced a number of mutant genes and enzymes to the process to either block or generate extra Psl to test its role in matrix formation. All signs point to Psl as the key scaffolding component of the Pseudomonas biofilm matrix, he said.

It's too early to tell whether strategies to block Psl in these lab experiments would be a safe way to disrupt the polysaccharide's actions in the human body, Wozniak noted.

Surfaces to which bacterial biofilms can attach are key to their success, meaning that in addition to thriving in congested lungs, they also grow on

catheters, ventilators and tracheal tubes - any medical device that enters the body. But preventing bacterial biofilm development has implications beyond medicine.

Fortified by the moist environment, plentiful nutrients and easy access to the human mouth, biofilms are also responsible for tooth decay. These microcolonies, when they are able to contact water or soil, also attach to certain industrial surfaces, causing corrosion and other damage.

"Not all bacteria use biofilms, but many do," Wozniak said. "Biofilms have been recognized for some time, but only in the last decade have people been able to apply modern molecular techniques to address the problem."

Source: The Ohio State University ([news](#) : [web](#))

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