Public release date: 12-Oct-2006

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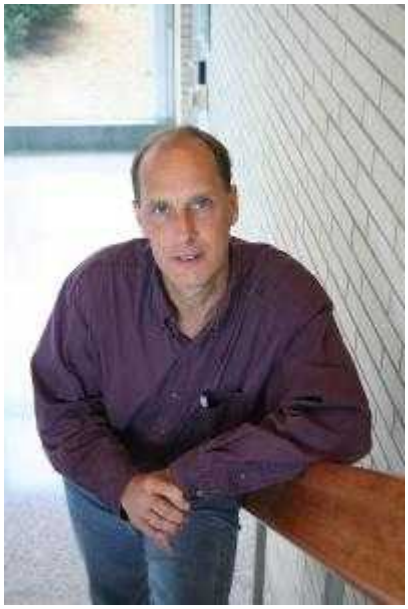
Binghamton University researcher makes major biofilm dispersion breakthrough

A Binghamton University biologist's discovery of a molecule that induces the dispersion of biofilms will likely mean a sea change in health care, manufacturing, shipping and pharmaceutics over the coming years.



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David Davies has found and is in the process of synthesizing a compound that will cause biofilm colonies to disperse, thus leaving individual bacteria up to 1,000 times more susceptible to disinfectants, antibiotics and immune functions. It's a discovery that will most certainly drive worldwide biofilm research in new directions and that could help put some of the most virulent cells in all of nature out of business.

Biofilms are complex aggregations of bacteria marked by the excretion of a protective and adhesive matrix. They develop almost anywhere that water and solids, or solids and gases meet, which means they are virtually everywhere. They are formed when individual microorganisms embed themselves in a gelatinous structure of their own making. When traveling alone in planktonic form, most bacteria are of small consequence and generally easy to manage, even with antibacterial hand soaps. But when they form biofilms, bacteria seem to gain super powers. In human terms the characteristic "slime" of biofilms, which comprises organic polymers that can grow to several centimeters thick and cover large areas, spells all kinds of big trouble.

Biofilms, for instance, fog your contacts, help to rot your teeth, and cause a host of diseases from cystic fibrosis and ulcers to colitis and ear infections. They are a leading cause of hospital infections and non-healing wounds, and were even at the root this past summer of corrosion that forced the replacement of 16 miles of the Alaska pipeline. As a result of that incident, 400,000 barrels a day of production from the largest oil field in the United States was suspended. The indefinite shut down, at a cost equal to 8 percent of U.S. petroleum output, led to immediate increases in the price of crude oil, and drove up fuel oil and gas prices.

Annual worldwide costs of biofilm infection and remediation are in the high billions, even according to the most modest estimates, and they are costs borne by industries and consumers worldwide. Name a manufacturing process and biofilms are probably a serious and costly issue. They have even been discovered in pipes at factories producing prepadine, the anti-bacterial, iodine-based solution that doctors swab on patients to "prep" them for surgery.

The small molecule Davies is working with appears to be one of the few known examples anywhere in nature of a communication signal that remains effective across species, family and phyla. In fact, though the evidence isn't yet in on that, Davies predicts the compound may also prove to have communicative effect even across bacterial kingdoms.

"I consider this the Holy Grail of research in biofilms," he said. "It's a new paradigm in the way we look at how bacteria regulate their behavior."

An associate professor of biology at Binghamton University, Davies' prominence in his field was already secured when he showed in the late 1990s that bacteria "talk to one another" through cell-to-cell communication and that such signaling is key to biofilm formation. Davies discovered the molecular medium of that communication in Pseudomonas aeruginosa, a biofilm-forming microorganism that is arguably the most common organism on the planet.

The dispersion autoinducer Davies is now investigating has shown itself to be effective in dispersing biofilms containing Pseudomonas aeruginosa, Streptococcus mutans (strep), Escherichia coli (E coli) and Staphylococcus aureus (staph) whether those bacteria exist in a pure or mixed-culture biofilm.

The dispersion-inducing molecule provokes genetic and physiological changes in the biofilm bacteria, causing them to disperse and return to a planktonic state. In lay terms, Davies has discovered at the very least how to tell four of the most problematic organisms around to pack up and get out of Dodge. And in so doing, the bacteria become easier to kill than the average

mosquito. Davies' feels certain his discovery will dramatically change the way infections are treated.

"I think people will start inducing dispersion to disaggregate biofilms and, then, treat them concurrently, and with significantly greater efficacy, with antibiotics."

He envisions his discovery first making its way to market as a topical treatment for cuts, lacerations and minor burns, perhaps even as an additive in adhesive bandages. But his major interest, and something he hopes to turn his attention towards in earnest in the coming year, is the area of non-healing wounds. Davies watched his diabetic great-aunt lose both of her feet to amputation after bacterial biofilm infections set in.

"If we can treat those kinds of wounds and clear up the infection, they will heal. We know that from wound debridement studies," he said. "I really think we can make a difference with these people, and if that was the only thing we did, it would be worth everything we're doing."

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