'Zinc Zipper' Plays Key Role In Hospital-Acquired Infections

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(PhysOrg.com) -- Hospital-acquired infections that are resistant to traditional antibiotic treatment have become increasingly common in recent years, confounding health care professionals and killing thousands of Americans.

Now, in studies that could lead to new ways to prevent this growing public health danger, a team of University of Cincinnati (UC) researchers is exploring a "zinc zipper" that holds bacterial cells together and plays a key role in such infections.

Hospital-acquired infections affect about 1.7 million people per year in the United States and result in an estimated 99,000 deaths annually, according to the Centers for Disease Control. About two-thirds of all hospital-acquired infections can be traced to two staphylococcal species, Staphylococcus aureus—including methicillin-resistant strains (MRSA) that are particularly difficult to treat—and Staphylococcus epidermidis.

In an article appearing in the Dec. 1 online edition of *Proceedings of the National Academy of Sciences*, researchers in UC's department of molecular genetics, biochemistry and microbiology detailed findings that the presence of zinc is crucial to the formation of infection-causing biofilms.

Staphylococci can grow as biofilms, which are specialized communities of bacteria that are highly resistant to antibiotics and immune responses. They are remarkably adhesive and can grow on many surfaces, including

implanted medical devices such as pacemakers, heart valve replacements and artificial joints. Preventing or inhibiting the growth of such biofilms would dramatically reduce the incidence of staph infections.

UC researchers in the lab of Andrew Herr, PhD, an assistant professor and Ohio Eminent Scholar in structural biology, found that zinc causes a protein on the bacterial surface to act like molecular Velcro, allowing the bacterial cells in the biofilm to stick to one another. Zinc chelation, or removal, prevented biofilm formation by Staphylococcus epidermidis and Staphylococcus aureus. The researchers used a chelation agent called DTPA (diethylenetriamine pentaacetic acid) to remove the zinc from a sample biofilm.

"We've shown that if you remove the zinc, you prevent the biofilm from forming, and if you add zinc back, the biofilm can grow," says Herr. "So we're hopeful that we can use this sort of approach to prevent these biofilms from ever taking hold in the first place."

The most practical applications, Herr says, might involve coatings for implanted medical devices, or rinses that a surgeon could use to clear the area around the implant.

Systemic removal of zinc, such as through an intravenous injection, is impractical for now because DTPA is approved by the U.S. Food and Drug Administration only for people with radio isotope poisoning. In addition, zinc is known to activate immune cells and play many other important roles in the body, so a proper balance would need to be developed.

Provided by University of Cincinnati

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