Stopping harmful oral bacteria in its path

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The best way to keep bacteria from doing any damage is to stop them in their tracks before they can start down their pathological road to destruction.

Yiping Han, associate professor at the Case Western Reserve University School of Dental Medicine, aims to understand how to build roadblocks for a common bacterium that's harmless in a mother's mouth but can turn deadly when it reaches an unborn child. She has received a five-year, $1.85 million grant from the National Institute of Dental and Craniofacial Research (NIDCR) at the National Institutes of Health to fund the effort.

This is Han's second NIDCR RO1 award. She's published more than 10 papers from previous research related to the bacterium, *Fusobacterium nucleatum*, that creates havoc once it leaves the mouth and enters the blood stream.

She has discovered an adhesin protein molecule, called FadA, in the genes of *F. nucleatum*. This adhesin, or binding agent, on the bacteria allows them to connect with receptors on epithelial cells in the mouth and later the endothelial cells of the placenta.

In tests, bacteria without FadA had less binding capability compared to those with the adhesin, Han and a team of researchers report on this finding in the July issue of the journal Infection *and Immunity*.

"With this new grant, we will be able to continue a functional analysis of

FadA," said Han. Her research group will look not only at the binding agent but the receptors on the host epithelial and endothelial cells that promote the binding of the oral bacteria.

"In some way, the receptors on the host cell activate a signal that puts into action a cascade of processes that allow the bacteria to penetrate the epithelial and endothelial linings and then colonize," explains Han.

"We want to block the bacteria before it can do any damage," Han says. "It's an upstream approach to go back to where the whole process begins and stop it from starting its destruction."

Once it leaves the mouth, the invasion of the bacteria through the placenta allows the bacteria to multiple rapidly in the immune-free environment that protects the fetus from being rejected by the mother's body. The rapid bacterial growth causes the placenta to become inflamed. In turn, the inflammation can trigger preterm birth and fetal death.

According to Han this research into the mechanisms of bacterial transport not only has potential to prevent preterm and stillborn births, it may have implications in preventing periodontal disease. Periodontal disease has been linked to such health problem as arthritis, diabetes and heart disease.

Source: Case Western Reserve University (news : web)