

Protein plays key role in infection by oral pathogen

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Scientists at Forsyth, along with a colleague from Northwestern University, have discovered that the protein, Transgultaminase 2 (TG2), is a key component in the process of gum disease. TG2 is widely distributed inside and outside of human cells. The scientists found that blocking some associations of TG2 prevents the bacteria *Porphyromonas gingivalis* (PG) from adhering to cells. This insight may one day help lead to novel therapies to prevent gum disease caused by PG.

Periodontal, or gum, disease is one of the most <u>common infectious</u> <u>diseases</u>. In its more severe forms, such as periodontitis, it causes loss of the bone that supports the teeth. Approximately 65 million adults in the United States are affected by some form of the disease. PG is the major causative agent of periodontitis, and it may also be involved in the development of systemic diseases such as atherosclerosis and <u>rheumatoid</u> <u>arthritis</u>.

The findings in this study indicate that TG2 is a key mediator in *Porphyromonas gingivalis* infection. In this research, the scientific team examined the critical role that TG2 plays in enabling *Porphyromonas gingivalis* to adhere to cells. Using confocal microscopes, clusters of TG2 were found where the bacterium was binding to cells. When the team silenced the expression of TG2, *Porphyromonas gingivalis* was diminished.

This study, which will be published in *Proceedings of the National Academy of Sciences* on March 24, 2014, was led by Dr. Heike Boisvert,



Assistant Member of the Staff, Department of Microbiology at Forsyth. The work was done in collaboration with Dr. Laszlo Lorand from Northwestern University Feinberg Medical School and Dr. Margaret Duncan, Senior Member of Staff at The Forsyth Institute.

"Once established, *Porphyromonas gingivalis* is very hard to get rid of" said Boisvert. "The bacterium changes conditions in the surrounding environment to ensure perfect growth; unfortunately, those changes, if untreated, can result in a loss of supportive tissue for our teeth. Also, as has been recently reported, manipulations of host proteins by PG may be involved in the development of systemic diseases such as atherosclerosis and rheumatoid arthritis. The more we know about the relationship of PG with us, the host, the better we can work on how to prevent disease and disease progression. " In the next phase of research, Boisvert will be examining TG2-knockout mice to test their susceptibility to *Porphyromonas gingivalis* infection and periodontal disease.

More information: Transglutaminase 2 is essential for adherence of Porphyromonas gingivalis to host cells, www.pnas.org/cgi/doi/10.1073/pnas.1402740111

Provided by Forsyth Institute

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