Insight - February 2020

Dugoni School of Dentistry

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Welcome to the winter 2020 issue of *Insight*, a quarterly newsletter celebrating the accomplishments of our community of scholars. We aim to spotlight insights from people at the Dugoni School working in all areas of scholarship, including clinical or biomedical research, the scholarship of teaching and learning, improvement of the health care system, and professional partnerships that advance the field.

**Systemic Immunologic Consequences of Chronic Periodontitis**

**What is it?**
Research into the effects of chronic gum disease on the entire body.

**What problem does it aim to solve?**
Nearly one in two American adults — 46% — have chronic gum disease. This is a problem for the health of the mouth, since periodontal problems can lead to tooth loss. But the negative effects aren’t confined confined to the mouth. Evidence has accumulated that there is a link between periodontal conditions and cardiovascular disease, diabetes, dementia, cancer and miscarriages/premature labor.

**How does it work?**
This was a study that started with 30 participants, half of whom had chronic gum disease that was being treated, and half who were healthy control subjects. Follow-up study took place three weeks after scaling, root planing and/or prophylaxis, and at that time, only nine patients and seven controls were available. Tissue samples were taken both at the initial session and the follow-up and analyzed. The main finding: “the capacity of specific immune cell subsets to respond to an inflammatory challenge is altered in peripheral blood samples from patients with chronic periodontitis (ChP). The most prominent differences between the immune features … were observed in innate immune cells and included heightened TLR-2/4 responses to *Porphyromonas gingivalis* LPS and depressed signaling responses to TNF, IFN and IL-2/4/6 and GM-CSF.”
What are the real-world implications?
“These results suggest that the systemic immune signature of ChP identified in this study is driven, at least in part, by local pathologic processes that are sensitive to disinfection of the oral cavity.” In short, chronic gum disease causes detectable changes to the immune system and its response, while treatment can reverse those changes.

What are the next steps?
Additional immunotherapy research and also studies on larger populations.

Source
Systemic Immunologic Consequences of Chronic Periodontitis, *Journal of Dental Research*, Volume 98, Issue 9, 1 August 2019, Pages 985-993

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A cell signaling-based analysis identifies immunological features associated with chronic periodontitis (ChP). (A) Immune features from previous publications were inserted into the mathematical model for signal transduction pathways (csEN, cell signaling-based elastic net model). (B) Results of the mathematical analysis shows the association between immune features revealed by experimental data (left panel). Further analysis shows that ChP patients have a higher level of immune activation than healthy controls (right panel), consistent with higher levels of inflammation in periodontitis.
Sabbatical at Jill Helms Research Laboratory at Stanford University

During the 2018-19 academic year, Dr. Steven Sadowsky spent six months with a team of 17 post-docs engaged in research on implants in the mouse model, with a focus on bone biology and implant biomechanics. Specifically, the team investigated mechano-adaptive responses of alveolar bone to oral implant hyper-loading in mice. A number of counterintuitive articles had previously been published that found an anabolic response in the bone when crown-to-implant ratios are between 2 and 3. The Stanford team created a model by inserting a 0.65 mm diameter retopin in a healed extraction site in a mouse maxilla, added a composite “crown” to engage the antagonist tooth for normal loading and then extracted adjacent maxillary molars to simulate hyper-loading. The histological results of increase in bone volume over total volume in this condition corroborated the Mechanostat theory, which suggests that a higher-than-normal level of strain state in bone triggers heightened bone modeling, which in turn should increase net bone area, thus decreasing the strain. Dr. Sadowsky’s role during this period at Stanford helped focus the research design towards clinical relevance.
Noteworthy Publications

Congratulations to Dugoni School faculty, staff, student and resident researchers involved in the following research publications within the last three months as sourced by Scopus, the abstract and citation database of peer-reviewed literature. Visit the abstract links to learn more.

Sadowsky, S.J.
View ›

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EFLA 945 restricts AIM2 inflammasome activation by preventing DNA entry for psoriasis treatment (2020) Cytokine, 127, art. no. 154951.
View ›

View ›

Peters, O.A., Du, D., Ho, M.Y., Chu, R., Moule, A.
View ›