

Why this dentist pays people to Not Brush Their Teeth for Two Weeks



Michael Glogauer

From his periodontal practice in Hamilton, Ontario, Dr. Michael Glogauer regularly sees patients with periodontal disease, some with an aggressive form of the disease that is resistant to treatment. Although disease-free patients typically don't present at his practice, he estimates that about 50% of people are protected—at some level—against developing periodontal disease. He says there are no obvious explanations for why some people are more susceptible to periodontal disease, while others seem to be inexplicably protected from it. But research he's conducted with colleagues at the University of Toronto faculty of dentistry has revealed a possible explanation: it's all about neutrophils, the most numerous blood cells in circulation.

Dr. Glogauer is a professor in the faculty of dentistry and member of the Matrix Dynamics Group at the University of Toronto. He is the immediate past-president of the Canadian Association for Dental Research and a member of the JCDCA.ca Editorial Advisory Group.

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"I'm fascinated by neutrophils," says Dr. Glogauer. Our common understanding of neutrophils views them as the short-lived "infantry" of the immune system and first line of defence against pathogens. "Everyone assumes neutrophils are one homogenous population of cells; they're resting, in circulation, and then they rush to the site of infection and are fully activated to ingest pathogens," Dr. Glogauer explains. "One of the problems we recognize with the neutrophil response for all sorts of inflammatory diseases, such as periodontal disease, arthritis and inflammatory bowel disease, is that sometimes the response is a little too exuberant and ends up causing a lot of redness, swelling and tissue damage."

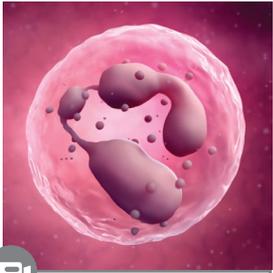
To Dr. Glogauer, this generally held view of neutrophils didn't add up with his research observations of neutrophils constantly flowing in the mouth *without* causing tissue damage. "Neutrophils are supposed to be inflammation factories," he says. "So it was a little heretical to have neutrophils entering a tissue without causing inflammation."

A recent study published by Dr. Glogauer and his collaborators in the *Journal of Dental Research*¹ explains his puzzling observations. His team, for the first time, identified different kinds of neutrophils, each with a special function. The study describes three predominant types of neutrophils: two types are found in healthy mouths, called Para-inflammatory (Para) 1 and 2, and one is found in mouths during chronic periodontal disease, called Proinflammatory (Pro).

"Para 1 neutrophils are somewhat quiescent, so they're not causing a lot of damage," he says. "Para 2 neutrophils are going around ingesting and killing bacteria, but they're doing this in what I would call a controlled manner; it's a condition where the immune system can control the



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To watch the full interview with Dr. Glogauer, visit:

oasisdiscussions.ca/2016/06/10/ons/

This interview has been condensed and edited.

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biofilm without any visual signs of inflammation. So there's no pain, no destruction to the tissue." Dr. Glogauer believes the neutrophil profiles he's identified in a healthy mouth—neutrophils controlling the biofilm without causing damage—can serve as a model for what's happening in other parts of the body where tissues connect with biofilm.

Patients with chronic periodontal disease, on the other hand, have only one kind of neutrophil—Pro neutrophils. These are described by Dr. Glogauer as "neutrophils with a lot of bacteria inside of them, spitting out enzymes that are toxic to the tissue." They superactivate the immune system process, bringing on inflammation, bleeding, bone loss, or detachment loss.

It's groundbreaking work that promises to help us better understand the pathogenic mechanisms of periodontal disease. How do pathogenic and healthy bacteria interact to elicit a switch from health to disease, from parainflammatory to proinflammatory neutrophils? To understand how this switch occurs, Dr. Glogauer and colleagues are conducting a study to see how neutrophil types change during the development of gingivitis. To accomplish this, he is paying people \$450 to not brush their teeth for 2 or 3 weeks and then monitors neutrophil profiles in their mouths. Despite neglecting their teeth for weeks, Dr. Glogauer is certain he'll find patients who seem to be protected against developing periodontitis. "I'm quite sure we're going to find patients who won't develop even a few proinflammatory neutrophils," he says. "And I'm sure we'll see some of our gingivitis patients

who are more susceptible to developing periodontal disease."

In addition to developing risk profiles for the development of periodontal disease, his work has the potential to create new diagnostics for early detection of inflammatory disease anywhere in the body. "The exciting part of this work is that the mouth is an easy site to access," says Dr. Glogauer. "It's much easier to develop diagnostics for an oral condition than, say, inflammatory bowel disease, because we can collect cells easily in rinses."

It also raises the possibility of developing diagnostic tools that would allow dentists to differentiate among various periodontal conditions. "As clinicians, we sometimes forget that periodontal disease is actually a group of probably more than 30 conditions," says Dr. Glogauer. "For example, a 40-year-old patient comes in with periodontal disease. Is it a rapidly progressing form? A chronic form? As a periodontist, being able to identify challenging disease in patients early on would allow for better treatment outcomes and better quality of life for patients."

For Dr. Glogauer's patients, knowledge of their susceptibility to periodontal disease could help them mitigate their risks. "Hopefully they would continue to brush their teeth, either way," he smiles.

REFERENCE

1. Fine N, Hassanpour S, Borenstein A, Sima C, Oveisi M, Scholey J, et al. Distinct oral neutrophil subsets define health and periodontal disease states. *J Dent Res*. 2016 Jul;95(8):931-8. Epub 2016 Jun 6.



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