

Researchers find susceptibility for caries, gum disease in genes

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Certain genetic variations may be linked to higher rates of tooth decay and aggressive periodontitis, according to two recently published papers by researchers at the University of Pittsburgh School of Dental Medicine and their collaborators.

Alexandre R. Vieira, D.D. S., Ph.D., senior author of both papers and an assistant professor of oral biology, and his colleagues at the School of Dental Medicine found that the rate of [dental caries](#) was influenced by individual variations, or polymorphisms, in a gene called beta defensin 1(DEFB1), which plays a key role in the first-line [immune response](#) against invading germs. The findings are available online in the [Journal of Dental Research](#).

"We were able to use data gathered from our Dental Registry and DNA Repository, the only one of its kind in the world, to see if certain polymorphisms were associated with the development of caries," Dr. Vieira said. "This could help us find new ways to treat people who are particularly susceptible to [tooth decay](#), a problem that afflicts millions of Americans."

For the study, the researchers analyzed nearly 300 anonymous dental records and accompanying saliva samples from the registry, assigning each case a DMFT score based on the presence of decayed teeth, missing teeth due to caries, and tooth fillings, as well as a DMFS score, based on decayed teeth, missing teeth, and filled surface of a tooth. In general, individuals with fewer caries have lower DMFT and DMFS

scores.

Saliva samples contained one of three variants, dubbed G-20A, G-52A and C-44G, of the DEFB1 gene. Individuals who carried a G-20A copy had DMFT and DMFS scores that were five-times higher than for people who had other variants. The G-52A polymorphism was associated with lower DMFT scores.

"It's possible that these variations lead to differences in beta defensin's ability to inhibit bacterial colonization," Dr. Vieira said. "In the future, we might be able to test for these polymorphisms as clinical markers for caries risk."

In a second paper, published last week in *PLoS One*, Dr. Vieira, colleagues at Pitt and collaborators in Brazil studied saliva samples of 389 people in 55 families to look for genetic links to aggressive periodontitis, which is rapid and severe destruction of the gums and bone that starts at a young age and is thought to be more common in Africans and those of African descent. Brazil's population is composed primarily of Caucasians of Portuguese ancestry, Africans and native Indians.

They found hints of an association between the disease and the FAM5C gene. While further testing did not find any mutations or [polymorphisms](#) that bore out a relationship, other experiments showed elevated levels of FAM5C expression, or activation, in areas of diseased periodontal tissue compared to healthy tissue.

"The FAM5C gene recently was implicated in cardiovascular disease, in which inflammation plays a role, just as in periodontitis," Dr. Vieira said. "More research is needed to see if variation in the gene is associated with different activity profiles."

Provided by University of Pittsburgh Schools of the Health Sciences

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