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Significance of Predicting Periodontal Disease Susceptibility by Single Nucleotide Polymorphism (SNP) Profile

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Objectives Periodontal disease is the destruction of the teeth supporting tissues leading to tooth loss in older adults and diseases such as cardiovascular disease, rheumatoid arthritis, premature birth, and dementia. If gingivally healthy volunteers stop oral hygiene for a week, gingivodentally adherent biofilms induce gingival crevicular fluid (GCF), an inflammatory exudate. Epithelial turnover at the crevice base maintains a barrier to biofilm downgrowth but requires the essential amino acid lysine from GCF, which is depleted to cadaverine by bacterial lysine decarboxylase (Ldc). Biofilm accumulation also requires GCF lysine. Hosts with most Ldc have the least biofilm and weakest epithelial barrier to bacteria, whereas hosts with the least Ldc have the most biofilm, but the strongest epithelial barrier. The GCF exudation rate gives 2 parallel arch-shaped curves (a weak and a strong GCF flow) which both increase and decrease depending on how much lysine remains unconverted to cadaverine.
Methods To find out the cause of the dual GCF response, we investigated the SNPs in 7 genes previously associated with periodontitis in buccal scraping samples from 15 volunteers using real-time PCR with SNP genotyping using the TaqMan method.
Results Responders with a weak GCF inflammatory response were completely separated from strong responders based primarily on gene interleukin1B SNPs at -511 and +3954 and sometime also on an SNP in gene IL6, or IL10 or CD14.
Conclusions Protective, strong GCF expels bacteria into saliva, whereas weak GCF incubates the bacteria and slowly converts the salivary microbiome in gingival crevices into a pathogenic microbiome responsible for periodontal inflammation. The significance of this study is that it may now be possible to use this SNP profile to identify the 40% of periodontitis-susceptible individuals apparently in the US population. Adequate treatment such as immunizing with periopathogens could slow periodontitis development enough to prevent comorbidity development in old age.