

Smoking, diabetes and oral microbiome: Implications for periodontal and implant health

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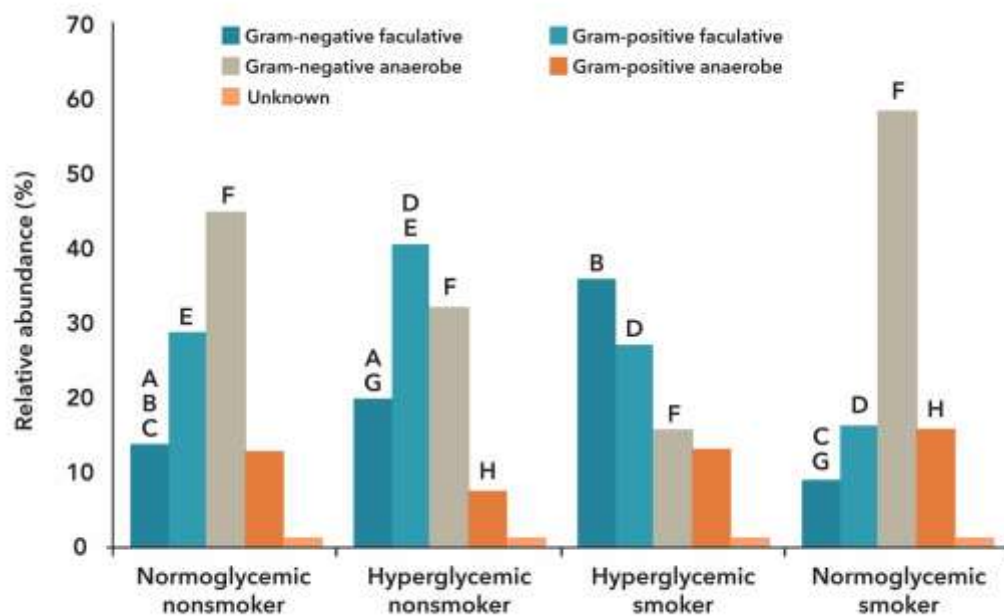
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The oral cavity is a complex, open ecosystem that houses hundreds of microbial species co-existing with the host cells. This collection of microbial members, including bacteria, viruses, fungi, and **archaea**, constitute the oral microbiome.

In health there is **homeostasis**, a **harmonious**, continuous interaction between the microbial and host. Disruptions in this homeostasis define periodontal and peri-implant diseases. Specific factors, such as cigarette smoking, and type 2 Diabetes (T2D) alter this **ecosystem** in the short term or long term. While smoking and T2D are firmly established as risk factors for periodontal disease, each impacts the oral environment differently.

For instance, smoking leads to peripheral vasoconstriction, decreased vascular permeability, and lower oxygen tension, while diabetes increases vascular permeability. Therefore, smoking and diabetes are associated with **distinct microbial colonization in the oral cavity, and specifically, in the subgingival environment.**

In T2D, the subgingival environment exhibits lower levels of anaerobes (bacteria that survive in oxygen-free conditions) and **microaerophiles** (**bacterial that survive in very little free oxygen**), including bacteria belonging to the genera *Parvimonas*, *Prevotella*, and *Tannerella*, *Lachnoanaerobaculum*, *Lactovacillus*, *Fretibacterium*, *Dialister*, *Eubacterium*, *Fusobacterium*, *Atopobium*, *Alloprevotalla*, and *Porphyromonas*, and higher levels of **facultative genera** (**bacteria that can survive in both oxygen-rich and oxygen-free environments**), like *Homophilus*, *Pseudomonas*, *Neisseria*. In contrast, smokers demonstrate higher abundances of anaerobes and lower levels of facultatives, corresponding to the environmental changes caused by cigarette smoking.



Although inconclusive, uncontrolled diabetes and smoking may be associated with increased risk for peri-implant diseases. Smoking alters the clinically healthy peri-implant microbiome even before the development of peri-implant mucositis or peri-implantitis with clinically healthy smokers showing a depletion of commensals and enrichment for anaerobes and pathogens. This pattern of loss of beneficial microbial members and increased pathogens further develops during the in peri-implantitis. Evidence on the impact of T2D on the peri-implant microbial environment is scarce and inconclusive, consisting mostly of cross-sectional studies. Alterations in the levels of *C. rectus*, *P. gingivalis*, *A. actinomycetemcomitans*, and *T. forsythia* have been reported. Overall, more rigorous, frequency-matched prospective studies that control confounding factors are needed to determine the impact of smoking and diabetes on the peri-implant environment. Regardless, current evidence demonstrating enrichment of pathogens and depletion of commensals suggests that smoking, and possibly T2D, can disrupt microbial homeostasis in the peri-implant environment, priming implant sites for development of clinical disease. Therefore, these high-risk cohorts may require more frequent monitoring and recall appointments to remove the enriched pathogens from the environment and re-establish the commensal microbiome.

REFERENCES ARE AVAILABLE UPON REQUEST.