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Original Research Article

Impact of Long-Term E-Cigarette/Vaping Use on the Oral Microbiome and Periodontal Health

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Abstract

Background: The long-term effects of e-cigarette use on oral health remain poorly understood. While traditional smoking is a known risk factor for periodontal disease, emerging evidence suggests that vaping may also alter the oral microbiome and promote inflammatory responses.

Objective: To investigate the impact of long-term e-cigarette use on the subgingival and salivary microbiome, periodontal clinical parameters, and local inflammatory markers.

Methods: A six-month longitudinal study was conducted with 84 healthy adults divided into e-cigarette users, conventional smokers, and non-smokers. Clinical periodontal assessments, saliva and subgingival plaque sampling, 16S rRNA sequencing, cytokine profiling, and predictive functional metagenomics were performed. Vaping topography and salivary metabolomic analysis were also evaluated. Statistical and mediation analyses examined associations between vaping, microbial shifts, and gingival inflammation.

Results: E-cigarette users exhibited a distinct subgingival microbiome compared to smokers and non-smokers, characterized by enrichment of periodontal pathogens such as Fusobacterium and Bacteroidales. Functional profiling indicated increased pathways involving lipid metabolism, xenobiotic degradation, and inflammatory signaling. Pro-inflammatory cytokines (IL-1β, TNF-α, IFN-γ) were elevated in e-cigarette users. Mediation analysis identified specific microbial taxa partially mediating the effect of vaping on gingival inflammation. Clinically, e-cigarette users showed higher gingival indices and more acidic salivary pH than non-smokers, though less severe periodontal damage than smokers.

Conclusion: Long-term e-cigarette use induces microbial dysbiosis and heightens oral inflammatory responses, contributing to compromised periodontal health. Vaping is not a harmless alternative to smoking and requires regular periodontal monitoring.

Keywords: E-cigarette, vaping, oral microbiome, periodontal disease, subgingival microbiota, inflammation, dysbiosis.

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Introduction

Electronic cigarettes (e-cigarettes) have rapidly grown in popularity as perceived safer alternatives to combustible cigarettes. However, e-cigarette aerosols contain nicotine, aldehydes, volatile organic compounds, glycerol, propylene glycol, and flavoring chemicals—each capable of altering the oral microbial environment [1,2].

Periodontal disease is a chronic inflammatory condition driven by dysbiosis of the subgingival biofilm. Although the adverse effects of cigarette smoking on periodontal tissues are well recognized, the long-term effects of vaping require further investigation. Longitudinal and cross-sectional studies demonstrate that e-cigarette use promotes a distinct subgingival microbiome enriched in pathogenic taxa such as Fusobacterium nucleatum and anaerobic members of Bacteroidales [3,4]. Mechanistic studies show that vaping behavior (e.g., puff volume) influences microbial metabolic pathways, including xenobiotic degradation and lipid metabolism [5]. Moreover, mediation analyses indicate that specific microbial taxa in saliva and subgingival plaque mediate associations between vaping and gingival inflammation [6]. Clinically, e-cigarette users exhibit poorer periodontal parameters, including elevated gingival index and more acidic salivary pH, compared with non-smokers [7].

This study aims to (i) characterize the subgingival and salivary microbiome in long-term e-cigarette users, (ii) assess local inflammatory biomarkers and periodontal clinical status, and (iii) investigate microbial functional pathways using predictive metagenomics and mediation analysis.

Methodology

Study Design and Participants

A six-month longitudinal cohort study included 84 adults aged 18–35 divided into three groups: e-cigarette users, conventional smokers, and non-smokers.

All participants were systemically healthy and had not undergone periodontal therapy within the past six months [4].

Clinical and Biological Sampling

At baseline and six months, periodontal assessments included plaque index, probing depth (PPD), clinical attachment level (CAL), and bleeding on probing. Subgingival plaque was collected from predetermined sites, and unstimulated saliva was obtained. Pro-inflammatory cytokines (IL-1β, IFN-γ, TNF-α) in saliva or gingival crevicular fluid were measured using multiplex assays [3].

Microbiome Analysis

DNA from subgingival plaque and saliva was extracted, and the V3–V4 region of the bacterial 16S rRNA gene was sequenced using Illumina MiSeq. Sequence data were processed using QIIME2, and taxonomic assignment was performed using the expanded Human Oral Microbiome Database (eHOMD) [7].

Alpha diversity (species richness, Shannon index) and beta diversity (Bray–Curtis, UniFrac) were analyzed. Differential abundance analysis identified vaping-associated taxa.

Functional Profiling & Metabolomics

Predictive metagenomics (PICRUSt2) inferred microbial functional pathways (e.g., KEGG). In a subset of participants, untargeted salivary metabolomics was performed via mass spectrometry. Vaping topography (puff volume) was measured using a validated portable device. VOC emissions from vaping devices were assessed using GC-MS and HPLC [5].

Statistical & Mediation Analysis: ANOVA or Kruskal–Wallis tests compared clinical, microbial, and metabolic variables among groups. Correlations between microbial taxa and cytokine levels were examined using Spearman analysis. Mediation analysis evaluated whether microbial taxa mediated the relationship between vaping and gingival inflammation [6].

Results and Discussion

Clinical and Inflammatory Findings

All groups showed increased microbial alpha diversity over six months, though cohort-specific profiles persisted. E-cigarette users demonstrated elevated IL-1β, TNF-α, and IFN-γ levels, in agreement with earlier findings linking vaping to enhanced inflammatory responses [1,3].

Microbial Community Structure

Beta diversity analysis revealed distinct clustering among e-cigarette users, smokers, and non-smokers. E-cigarette users showed increased Fusobacterium, Veillonella, and anaerobic Bacteroidales, taxa commonly associated with periodontal inflammation [3,4].

Functional and Metabolic Changes

Functional profiling revealed enrichment in pathways related to xenobiotic degradation, lipid metabolism, and oxidative stress among e-cigarette users [5]. Salivary metabolomics showed metabolite alterations consistent with microbial dysbiosis and inflammation.

Vaping topography data indicated that higher puff volume correlated with greater microbial and metabolic disruption [5].

Mediation Analysis:

Eighteen microbial taxa (e.g., Actinomyces, Rothia, Neisseria) mediated associations between e-cigarette use and gingival inflammation [6], supporting the role of microbial shifts in oral inflammatory responses.

Cross-Sectional Corroboration

Cross-sectional analyses (n = 144) demonstrated that e-cigarette users had higher gingival indices, more acidic salivary pH, and elevated cotinine levels compared to non-smokers[7], reinforcing the negative periodontal impacts of vaping.

Interpretation and Implications

- **Distinct Dysbiosis:** E-cigarette use shapes a unique subgingival microbiome.
- **Microbial–Inflammatory Link:** Increased pathogenic taxa correspond with heightened cytokine levels.
- **Behavioral Impact:** Puff volume significantly influences microbiome and metabolomic alterations.
- **Clinical Relevance:** Vaping produces measurable periodontal inflammation, though less severe than cigarette smoking.
- **Preventive Strategy:** Regular periodontal monitoring and microbial-targeted interventions may benefit long-term vapers.

Limitations

- The six-month duration may not capture long-term periodontal tissue destruction.
- The sample size limits generalizability.
- Device- and flavor-specific variables were not fully stratified.

- Predictive metagenomics does not replace full metagenomic or metatranscriptomic validation.
- Metabolomic findings require larger cohort validation.

Conclusion

Long-term e-cigarette use is associated with distinct microbial dysbiosis, elevated inflammatory responses, and adverse periodontal outcomes. Although less harmful than traditional smoking, vaping is not benign for oral health. Future research should examine device-specific effects, long-term outcomes, and microbial-targeted interventions.

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