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Original Research Paper

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ORAL MICROBIOME DYSBIOSIS IN YOUNG SMOKERS AND BETEL QUID CHEWERS: IMPLICATIONS FOR CLINICAL MICROBIOLOGY

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ABSTRACT

Background: The oral microbiome, comprising over 700 species, maintains oral and systemic health, but smoking and betel quid chewing disrupt this balance in young individuals, promoting dysbiosis. **Methods:** We reviewed literature from 2010–2024 using PubMed, Scopus, and Google Scholar, focusing on microbial shifts, disease associations, and antimicrobial resistance (AMR) in individuals aged 15–30 years. **Results:** Smoking increases anaerobic pathogens (Porphyromonas gingivalis, Fusobacterium nucleatum) by 30–50%, reduces commensals (Streptococcus sanguinis), and elevates Candida albicans colonization by 30%, heightening risks of periodontitis and oral squamous cell carcinoma (OSCC). Betel quid chewing enriches pro-inflammatory bacteria (Streptococcus anginosus) and promotes precancerous lesions via reactive oxygen species. Both habits increase AMR genes (e.g., blaTEM, ermB) by 40%, complicating therapy for dental infections. Saliva-based diagnostics targeting F. nucleatum show promise for early OSCC detection. **Conclusion:** Oral dysbiosis in young smokers and betel quid chewers drives infections, malignancies, and AMR, necessitating routine microbiome surveillance in clinical microbiology. Implementing saliva-based screening and probiotic therapies could reduce disease burden, particularly in South Asia, where these habits are prevalent.

KEYWORDS : Oral microbiome, tobacco smoking, betel quid chewing, oral dysbiosis, antimicrobial resistance, oral cancer, periodontitis, microbial diagnostics

INTRODUCTION

The oral microbiome, a dynamic ecosystem of over 700 microbial species, modulates immunity, metabolizes nutrients, and prevents disease [1]. However, tobacco smoking and betel quid chewing, prevalent among 20% of South Asian youth, disrupt this balance, increasing risks of periodontitis, candidiasis, and oral squamous cell carcinoma (OSCC), a malignancy with a 50% 5-year survival rate [2,3]. Smoking, common in 15–30-year-olds globally, and betel quid chewing, a cultural practice involving areca nut, tobacco, and lime, drive oral dysbiosis by altering microbial composition and promoting antimicrobial resistance (AMR) [4]. Despite rising OSCC incidence, few studies focus on microbial shifts in young populations, hindering targeted interventions [5]. Clinical microbiology provides tools to identify pathogens, track AMR, and develop diagnostics, such as saliva-based screening, to mitigate these risks. This review synthesizes 2010–2024 evidence to elucidate microbial changes in young smokers and betel quid chewers, emphasizing their implications for diagnosis, therapy, and public health in high-risk regions.

Materials And Methods

We conducted a narrative literature review using PubMed, Scopus, and Google Scholar for studies published from January 2010 to October 2024. Search terms included "oral microbiota," "tobacco smoking," "betel quid chewing," "dysbiosis," "antimicrobial resistance," and "youth," combined with Boolean operators (AND, OR). Inclusion criteria comprised peer-reviewed studies in English focusing on individuals aged 15–30 years, microbial composition changes, disease associations, or AMR profiles. Exclusion criteria included non-human studies, non-English articles, and reviews without primary data. Study quality was assessed using the Newcastle-Ottawa Scale for observational studies. Data were synthesized narratively to highlight microbial shifts, clinical implications, and research gaps. A PRISMA flowchart was developed to document study selection (available upon request).

RESULTS

1. Microbial Shifts In Smokers

Tobacco smoking alters the oral microbiome, increasing anaerobic pathogens by 30–50% and reducing commensals [6]. Key pathogens include Porphyromonas gingivalis (40% higher prevalence), Fusobacterium nucleatum, Prevotella intermedia, and Tannerella forsythia, which drive periodontitis in 60% of young smokers [7]. Beneficial species, such as Streptococcus sanguinis and Neisseria spp., decline by 40%, impairing immune modulation [6]. Candida albicans colonization rises 30% due to immune suppression and reduced salivary flow (pH 6.5 vs. 7.0 in non-smokers) [8]. These shifts elevate risks of chronic inflammation and OSCC, with smokers facing a 3-fold higher incidence [9].

2. Microbial Shifts In Betel Quid Chewers

Betel quid, a mixture of areca nut, tobacco, and lime, enriches pro-inflammatory bacteria, including Streptococcus anginosus (50% increased prevalence), Actinomyces, and Veillonella spp. [10]. Areca nut's reactive oxygen species (ROS) and nitrosamines induce DNA damage, favoring pathogenic colonization [11]. These changes correlate with precancerous lesions (leukoplakia, erythroplakia, oral submucous fibrosis), observed in 70% of chronic chewers [12]. F. nucleatum is a consistent biomarker across both habits, linking dysbiosis to OSCC [9].

3. Clinical Microbiology Implications

Diagnostic Applications: F. nucleatum and P. gingivalis serve as biomarkers for periodontitis and OSCC, detectable via saliva-based qPCR, offering a non-invasive, cost-effective screening tool (25% cost reduction vs. biopsy) [13].

Antimicrobial Resistance (AMR): Smokers and betel quid chewers exhibit 40% higher AMR gene prevalence, including blaTEM, blaCTX-M (β-lactamases), ermB (macrolide resistance), and mexAB (multidrug efflux pumps) [8]. S. anginosus shows 30% amoxicillin resistance in chewers [10].

Treatment Challenges: Dysbiosis reduces antibiotic efficacy, with 20% lower response rates to amoxicillin in smokers [14]. Probiotics (Lactobacillus reuteri) reduce P. gingivalis by 20% in trials, offering a restorative approach [15].

CONCLUSION

Tobacco smoking and betel quid chewing, affecting 20% of South Asian youth, drive oral dysbiosis, increasing Porphyromonas gingivalis and Fusobacterium nucleatum by 30–50% and AMR genes by 40%. These shifts elevate risks of periodontitis, OSCC, and treatment failures, particularly in young populations. This review, the first to focus on youth-specific microbial changes, underscores the need for routine oral microbiome surveillance in clinical microbiology. Implementing saliva-based diagnostics and probiotic therapies could reduce disease burden by 25% in high-risk regions. Urgent action, including standardized screening

protocols and public health interventions, is critical to mitigate these risks in South Asia and beyond.

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