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MODERN NICOTINE DELIVERY SYSTEMS (E-CIGARETTES AND NICOTINE POUCHES) AND THEIR EFFECTS ON ORAL TISSUE: A LITERATURE REVIEW

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ABSTRACT

Introduction and Purpose: The global decline in traditional tobacco smoking has been accompanied by a rapid increase in the popularity of modern, tobacco-free nicotine delivery systems, primarily electronic cigarettes (ENDS) and oral nicotine pouches. Marketed as safer alternatives and available in numerous flavors, these products highly appealing to adolescents and young adults. The purpose of this narrative review is to evaluate and compare the impact of electronic cigarettes and tobacco-free nicotine pouches on oral health, focusing specifically on the oral microbiome, mucosa, periodontium, and dental **Materials and Methods:** A comprehensive review of the current literature was conducted. Relevant scientific articles, including in vitro studies, clinical evaluations, and systematic reviews, were analyzed to assess the pathophysiological mechanisms, cytotoxicity, and clinical oral manifestations associated with the exposure to e-cigarettes aerosols and the chemical components of tobacco-free nicotine pouches.

Conclusions: Modern nicotine delivery systems are not safe smoking alternatives. ENDS disrupt the oral microbiome promoting periodontal pathogens and cariogenic bacteria. E-cigarettes induce mucosal lesions and enamel demineralization, while masking periodontal inflammation via vasoconstriction. Conversely, nicotine pouches cause cytotoxicity, mucosal keratosis, and severe, site-specific gingival recession independent of oral hygiene. Given these risks, there is an urgent need for long-term clinical studies and proactive patient education by dental professionals to debunk misconceptions regarding their safety.

KEYWORDS

Electronic Cigarettes, Nicotine Pouches, Oral Health, Oral Microbiome, Gingival Recession, Oral Mucosa, Dental Caries

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Introduction

Recent epidemiological data indicate a significant shift in trends regarding nicotine consumption. According to the World Health Organization (WHO), the global prevalence of traditional tobacco smoking decreased from 22.8% in 2007 to 17.0% in 2021, representing a relative reduction of 25% over 14 years (1). On the other hand, the market has witnessed a rapid rise in the popularity of modern tobacco-free nicotine delivery systems among teenagers and young adults - especially Electronic Nicotine Delivery Systems (ENDS) and nicotine pouches. The scale of this rapid adoption is unprecedented; for instance, Euromonitor International reported that global sales of nicotine pouches reached an estimated 6.8 billion units in 2021, representing a growth of over 2000% compared to 2018 (2).

Currently, the market for alternative nicotine products is dominated by two primary, entirely tobacco-leaf-free delivery systems. The first type comprises an inhalator form, widely recognized as Electronic Nicotine Delivery Systems (ENDS) or e-cigarettes. These battery-powered devices operate by heating a liquid carrier solution- most commonly a mixture of propylene glycol and/or vegetable glycerin- containing nicotine and various flavoring agents, thereby generating an aerosol intended for inhalation (3). The second type involves a topical, intraoral delivery method utilizing tobacco-free nicotine pouches. These are small, discreet white pouches based on plant fibers, containing a cellulose matrix infused with nicotine that can be either naturally or synthetically sourced (4). They are designed to be placed directly in the oral cavity, typically between the upper lip and gingiva, which facilitates the rapid and direct absorption of the active substance through the highly vascularised oral mucosa.

Understanding the popularity of electronic cigarettes and nicotine pouches requires analyzing user motivations. A dominant factor is the common belief that these products are less harmful than traditional cigarettes. In fact research shows that a substantial part of consumers (including 28.2% in a Polish study) consider nicotine pouches as a safer alternative (5), often leading people to use them as an aid to quit smoking (6,7). Furthermore, both ENDS and nicotine pouches are heavily promoted with thousands of flavoring (mint, menthol, fruit). These flavors are especially appealing to adolescents, because they effectively mask the bitter

taste of nicotine (2). Additionally, the convenience and high level of discretion play a major role in choosing these alternative methods. E-cigarettes are relatively easy to hide, while modern smokeless nicotine pouches do not require spitting and produce no emissions. This allows for unnoticed use in public places where smoking is strictly prohibited (5,6). Finally, aggressive digital marketing further normalizes their consumption (8), peer influence - such as e-cigarette use among friends- plays a significant role in encouraging the use of these devices (7).

Despite their growing popularity and the common public perception of reduced harm, the exact effects of these products on the oral cavity are still being researched. Therefore, the aim of this narrative review is to summarize and compare the impact of these two tobacco-free products- electronics cigarettes and nicotine pouches- on oral health. Specifically, this paper focuses on how they affect periodontal tissues, the oral mucosa, the oral microbiome and the development of dental caries.

Electronic Nicotine Delivery Systems and Oral Health Implications

1.1 The Oral Microbiome

The oral microbiome continuously maintains a microbial equilibrium that is crucial for overall oral health. However, electronic nicotine delivery systems destabilize this state, promoting the proliferation of pathogenic microorganisms and leading to dysbiosis. Exposure to electronic cigarette aerosols has been associated with an increased abundance of specific anaerobic bacteria and pathogens linked to periodontal diseases. These include: *Fusobacterium*, *Bacteroidales* (G-2), *Treponema*, *Prevotella*, *Clostridium*, *Veillonellaceae*, *Tannerella*, *Oribacterium* (9,10). Furthermore, expanded population of microorganisms such as *Alloscardovia*, *Cryptobacterium*, *Dialister*, as well as the pathogenic species *Olsenella uli* (associated with endodontic infection), was observed in the saliva of e-cigarette users (11). In the subgingival microbiome, an increased abundance of *Selenomonas*, *Saccharibacteria*, and *Leptotrichia* (including *L. wadei* and *L. hongkongensis* correlated with dental caries, gingivitis, and periodontitis) was noted compared to non-smokers. These bacteria are correlated with the pathogenesis of oral diseases and inflammatory markers (specifically the cytokine IL-1 β) (9). In contrast, studies also reveal a decline in the population of many commensal bacteria among e-cigarette users, for instance *Streptococcus*, *Actinomyces*, *Rothia*, *Neisseria*, *Haemophilus*, *Kingella*, *Cardiobacterium*, *Scardovia*, *Granulicatella*, *Bergeyella*, *Catonella*, and *Enterococcus* (9,11). Another significant issue is the effect on the proliferation of cariogenic bacteria. It has been shown that e-cigarette aerosols are toxic to oral commensals such as *Streptococcus gordonii*, *Streptococcus mitis*, *Streptococcus intermedius*, and *Streptococcus oralis*. At the same time, they promote the proliferation of *Streptococcus mutans*, which directly causes tooth decay (12). Moreover, dysbiosis facilitates the colonization of the oral cavity by *Candida albicans*, the primary pathogen responsible for problematic fungal infections for example thrush and denture stomatitis (13).

1.2 Cytotoxicity and Oral Mucosal Lesions

The disrupted microbiome and direct exposure to toxins within the aerosol primarily compromise the protective layer, the oral mucosal epithelium. The heated aerosol serves as a carrier for the toxic thermal degradation products of the base (glycol and glycerin), heavy metals, nicotine, and chemical flavoring agents (14). E-cigarettes aerosols contain significant concentrations of carcinogenic residues, including formaldehyde, acetaldehyde, and acrolein. Furthermore, the heating of the device's filament facilitates the transfer of heavy metals such as nickel, tin, lead, and chrome into the aerosol, which can then infiltrate tissues and damage cellular health (14). At the intracellular level, the penetrating aerosol particles induce pronounced oxidative stress and active inflammatory cascade, which manifests as the enhanced secretion of pro-inflammatory cytokines, particularly IL-1 and IL-6 (15). Studies confirm that exposure to these emissions exerts profound cytotoxic and genotoxic activities. This leads to direct damage to the genetic material (including the formation of DNA adducts and strand breaks) (16), a significant reduction in cell viability, and the intensification of apoptotic processes accompanied by DNA fragmentation and nuclear chromatin condensation (15). These alterations are clearly reflected in cytomorphological observations, revealing a spectrum of structural anomalies such as nuclear hypertrophy, an elevated nuclear-to-cytoplasmic ratio, as well as the formation of micronuclei and the presence of binucleated cells (17). These phenomena are accompanied by concurrent dysbiosis of the oral environment, characterized by a decline in commensal bacteria and an overgrowth of pathogens. These cellular damages directly impair the protective function of the epithelial barrier (15). The

clinical manifestation of this includes an increased incidence of ulcerations, xerostomia, and the presence of characteristic white lesions on the buccal and lingual mucosa (17). From a long-term perspective, the accumulated genotoxic defects, in close correlation with chronic inflammation, are recognized as a significant and early marker predisposing the oral epithelial cells to malignant transformation (17).

1.3 Periodontal and Peri-implant Health

Beyond the mucosal epithelium and the oral microbiome, the detrimental effects of e-cigarettes extend significantly to the periodontal and peri-implant tissues (18,19). E-cigarette users exhibit poorer periodontal health than non-smokers, yet generally present less severe tissue destruction than conventional cigarette smokers (20). Clinical evaluations reveal that e-cigarette use is associated with higher plaque index (PI) values and increased periodontal pocket depths compared to the control group (18,21). However, a critical clinical paradox is observed regarding gingival bleeding; similar to traditional smoking, the continuous exposure to aerosolized nicotine in e-cigarettes induces local vasoconstriction and reduces gingival blood flow. Consequently, e-cigarette users often present with markedly lower bleeding on probing (BOP) and gingival indices than non-smokers, which can mask underlying chronic inflammatory processes and delay the clinical diagnosis of periodontitis (21,22). An analogous phenomenon is observed in peri-implant tissues, where despite reduced bleeding, elevated concentrations of pro-inflammatory markers, such as IL-1 β and TNF- α , are found in the peri-implant sulcular fluid (PISF) (23). Although results regarding marginal bone loss (MBL) are sometimes inconsistent, comprehensive network meta-analyses indicate that e-cigarette users are still at an elevated risk for peri-implant marginal bone loss and deteriorated clinical parameters compared to individuals completely free of nicotine exposure (18,19,23).

1.4 Effects on Tooth Structure and the Risk of Dental Caries

The negative impact of electronic nicotine delivery systems is not limited to the oral mucosa and periodontal tissues, but also encompasses the direct destruction of dental hard tissues. The pathomechanism of this phenomenon is initiated by the physicochemical properties of the vapors themselves. Many commercially available e-liquids are characterized by an acidic pH (falling below the critical threshold of 5.5 for enamel dissolution), which imparts a strong erosive potential and leads to significant reduction in enamel microhardness (6,24). The demineralization process is further exacerbated by the specific high viscosity of the generated aerosol and the ubiquitous presence of cariogenic sweeteners (e.g., sucrose or sucralose) in flavoring additives (24,25). The highly viscous residue easily adheres to tooth surfaces, particularly within retentive pits and fissures, creating an ideal environment for the adhesion and colonization of *Streptococcus mutans* (12,25). The primary cariogenic pathogen rapidly metabolizes the carbohydrates contained in the vapor, as well as the thermal degradation by-products of the base (glycol and glycerin), into acids, directly driving enamel dissolution (12,23). The extent of this destruction is strongly catalyzed by vaping-associated xerostomia, which deprives the oral cavity of the natural buffering and remineralizing properties of saliva (6,24,25). Clinically, this sequence of adverse mechanisms results in an increased incidence of carious lesions, dentin hypersensitivity, and an increased risk of mechanical microtrauma and dental crown fractures (12,23).

Nicotine Pouches and Their Impact on Oral Tissues

2.1 Oral Dysbiosis

Current research regarding the direct impact of oral nicotine pouches on the oral microbiome remains limited and necessitates further investigation to establish their independent or concomitant effects (26). However, existing evidence suggests that flavored nicotine pouches possess the potential to induce microbial dysbiosis within the oral cavity and periodontium, which can subsequently inhibit local innate immune responses (26). Although the literature concentrating exclusively on tobacco-free nicotine pouches is still in its infancy, studies on comparable smokeless tobacco products clearly indicate that their use correlates with an overall increase in bacterial diversity and the proliferation of periodontal pathogens, such as *Actionmyces*, *Aggregatibacter*, *Streptococcus*, and *Staphylococcus* (26). Furthermore, it has been established that elevated nicotine concentrations are significantly linked to a higher prevalence of cariogenic *Streptococcus mutans* and greater caries severity (27). Additionally, the prolonged physical placement of the pouch may lead to localized plaque accumulation, further altering the flora and potentially contributing to site-specific dysbiosis and increased caries risk (28).

2.2 Cytotoxic Effects and Mucosal Changes

The use of tobacco-free nicotine pouches exerts a direct impact on the oral mucosa, promoting inflammation and demonstrating cytotoxic effects. Nicotine, the primary ingredient in these products, interacts with host cells and alters inflammatory responses to microbial challenges (26). The use of pouches is associated with elevated levels of pro-inflammatory salivary biomarkers, including interleukins (IL-1, IL-6, IL-8) and tumor necrosis factor TNF- α (4,29). Furthermore, *in vitro* studies show that nicotine pouch extracts cause a direct toxic response in gingival epithelial cells. Exposure to nicotine and flavoring additives, such as menthol, represses the viability of periodontal ligament fibroblasts by increasing the generation of cellular reactive oxygen species (ROS) (26). This physiological sequence leads to oxidative stress, DNA damage, and ultimately cell death (apoptosis) (26). On a histological level, dense inflammatory infiltrates composed of lymphocytes and macrophages are visible in the subepithelial tissue (30). As a result of these intracellular pathologies and the long-term direct contact between the pouch and the tissues, specific clinical symptoms develop at the application site, accompanied by histopathological changes such as parakeratosis, epithelial hypertrophy (acanthosis), and intraepithelial edema (30). These oral lesions range from slight wrinkling to distinct white, grainy, or leathery mucosal changes consistent with smokeless tobacco keratosis (4,29). A significant clinical concern is the potential development of precancerous conditions such as leukoplakia, which requires thorough diagnosis due to the risk of progression to cell carcinoma (31).

2.3 Periodontal Consequences and Localized Gingival Recession

The close contact between nicotine pouches and oral tissues is detrimental to the periodontium, with localized gingival recession-the process of tooth root exposure-being one of the most frequent outcomes (28,29). These lesions usually develop in the area of the upper canines and premolars, corresponding to the most common placement sites for the product (31). Importantly, research suggests that development of recession caused by these products appears to occur independently of the patient's overall oral hygiene; it can manifest even in individuals who maintain excellent dental health, highlighting the direct impact of the product itself (32). This is because keeping the pouch in the oral vestibule causes constant mechanical and chemical irritation, leading to localized soft tissue breakdowns. Nicotine further lowers the survival rate of periodontal ligament (PDL) fibroblasts and stem cells, which impairs the natural regenerative capacity of the periodontium (26). Evidence also shows that nicotine accelerates alveolar bone loss by boosting the production of inflammatory factors like RANKL and TNF- α (26,29). Diagnosis is further complicated by nicotine-induced vasoconstriction, which masks underlying inflammation by reducing bleeding on probing (BOP) (33).

Conclusions

Based on a comprehensive review of literature, it is evident that modern tobacco-free nicotine delivery systems are not harmless alternatives to traditional smoking. Electronic nicotine cigarettes profoundly disrupt the oral microbiome by reducing beneficial commensal bacteria and promoting the proliferation of periodontal pathogens and cariogenic species like *Streptococcus mutans*. The acidic, viscous, and sweetened nature of e-cigarettes aerosols, combined with vaping-induced xerostomia, creates a highly cariogenic environment that directly drives enamel demineralization. Furthermore, exposure to these toxic aerosols induce oxidative stress and cellular toxicity, leading to general mucosal lesions, while nicotine-induced vasoconstriction masks underlying periodontal and peri-implant tissue breakdown by significantly reducing gingival bleeding. On the other hand, tobacco-free nicotine pouches exert their detrimental effects through prolonged, direct physical contact with the oral tissues. This concentrated chemical exposure leads to smokeless tobacco keratosis and potentially malignant conditions such as leukoplakia. Crucially, nicotine pouches cause severe, localized gingival recessions. Given the rapid increase in the popularity of both products, particularly among youth, there is an urgent need for long-term clinical studies and proactive educational efforts by dental professionals to definitively dispel the myth that these modern nicotine systems are safe for the oral cavity.

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