Review Article Pol J Public Health, Vol. 136 (2026): 100-104

E-cigarettes, Oral Microbiome, and Periodontal Disease Risk: A Narrative Review of Pathogenic Mechanisms

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A – Development of the concept and methodology of the study; B – Query – a review and analysis of the literature; C – Submission of the application to the appropriate Bioethics Committee; D – Collection of research material; E – Analysis of the research material; F – Preparation of draft version of manuscript; G – Critical analysis of manuscript draft version; H – Statistical analysis of the research material; I – Interpretation of the performed statistical analysis; K – Technical preparation of manuscript in accordance with the journal regulations; L – Supervision of the research and preparation of the manuscript

Abstract

Introduction. Electronic nicotine delivery systems (ENDS) are global alternatives to tobacco, particularly popular among adolescents. Although they are marketed as a smoking cessation aid, their long-term impact on the oral cavity remains unclear. This review synthesizes current knowledge regarding the impact of ENDS on oral microbiota composition and periodontal disease risk.

Methods. The literature search covering 2015–2025 was conducted using terms: e-cigarettes, oral microbiome, periodontal disease and aerosol toxicity. Eligible peer-reviewed studies addressed aerosol properties, microbial shifts, periodontal outcomes or pathogenic mechanisms. Brief description of the state of knowledge: ENDS aerosols containing nicotine and humectants (PG/VG) decompose into toxic carbonyls and reactive oxygen species (ROS). Vaping induces xerostomia, oxidative stress, and pH alterations, triggering rapid microbial dysbiosis. In this state, bacteria utilize PG/VG as a carbon substrate, favoring anaerobic taxa. Aerosols inhibit beneficial commensals while enhancing the virulence and biofilm formation of S. mutans and S. aureus. Host immune defenses are compromised through reduced cytokine secretion, leading to impaired bacterial clearance and greater probing depths.

Conclusions. The use of E-cigarette disrupts oral homeostasis, promoting pathogenic biofilms and chronic inflammation. This unique dysbiotic state, coupled with DNA damage, significantly elevates the risk of aggressive periodontal disease and potential oral malignancy. However, marked methodological and product heterogeneity underscores the urgent need for well-controlled, longitudinal human clinical investigations.

Keywords: periodontitis, microbiota, oral health, dysbiosis, electronic nicotine delivery systems.

DOI: 10.12923/2083-4829/2026-0018

INTRODUCTION

Electronic nicotine delivery systems (ENDS) have been gaining popularity worldwide as a seemingly less harmful alternative to conventional tobacco use. ENDS are battery-powered devices that contain a heating element and a cartridge filled with "e-liquid", that generally comprises nicotine, flavouring compounds, and humectants such as propylene glycol (PG) and vegetable glycerin (VG) [1,2]. Aggressive marketing of these products, growing social acceptance and perception of electronic cigarettes as less detrimental to health resulted in their widespread use, particularly among adolescents [3]. Epidemiological data from 2020 identified approximately 19.6% of US high school students as current e-cigarette users, highlighting a significant public health challenge among teenagers and young adults [4]. In Poland, 5.9% of respondents to a 2024 nationwide cross-sectional survey declared themselves as daily e-cigarette users, while 15.2% admitted that they had used an e-cigarette at least once in the past month [5]. Although ENDS are often promoted as effective smoking cessation aids, their long term safety profile remains unclear, especially their local effects within the oral cavity [6,7]. It is the mouth that serves as the primary point of contact for inhaled xenobiotics, with nicotine concentrations in saliva found to be up to 10.5 times higher than those in plasma [8]. There is a growing consensus, from preliminary human and lab models, that e-cigarette use induces a unique state of microbial dysbiosis, that differs from both non-smokers and conventional smokers [9]. The disruption in the microbial ecosystem within the oral cavity is associated with disease, such as periodontitis, and has been linked mechanistically with numerous cancer types [2,10]. Additionally, significant knowledge gaps among

dental professionals regarding the regulatory status, chemical composition of e-cigarettes and their harmful effects on oral health, potentially compromise clinical advice [11]. An important interpretive issue in this field is the marked heterogeneity of ENDS products, usage patterns, and research methodologies. These factors complicate comparisons across studies and should be taken into account when evaluating apparently inconsistent microbiological or periodontal findings. This narrative review aims to synthesise the current knowledge on the impact of ENDS on the composition of oral microbiota and periodontal disease risk.

METHODS

This manuscript was designed as a narrative review intended to summarize the current evidence on the relationship between e-cigarette use, the oral microbiome, and periodontal disease. The literature search was conducted in PubMed, Scopus, and Google Scholar and covered publications from 2015 to 2025. The search strategy was based on combinations of the following key concepts: "e-cigarettes" OR "electronic nicotine delivery systems" OR "vaping", AND "oral microbiome" OR "oral dysbiosis" OR "subgingival microbiome", AND "periodontal disease" OR "periodontitis" OR "oral health", with additional terms such as "aerosol toxicity", "flavoring agents", and "oxidative stress" used when relevant to mechanistic sections.

Studies were considered eligible if they were peer-reviewed and addressed at least one of the following areas: (1) physicochemical properties of ENDS aerosol relevant to the oral cavity, (2) effects of ENDS on oral microbial composition or function, (3) periodontal or peri-implant outcomes in e-cigarette users or (4) cellular and molecular mechanisms potentially linking ENDS exposure with oral inflammation and tissue damage. Reviews, human observational studies, clinical studies, in vitro experiments and selected preclinical studies were included when they contributed directly to the topic.

Due to the narrative nature of the review, study selection was interpretive rather than protocol-driven, and no PRISMA flow diagram was prepared. Double, independent screening and formal risk-of-bias assessment were not performed.

Aerosol Composition and the Oral Microenvironment

ENDS function by heating a liquid solution ("e-liquid") to temperatures ranging from 100ºC to 350ºC in order to produce an inhalable aerosol [6,12]. Individual "e-liquid" components are typically classified as safe for ingestion, however, this designation does not account for the toxicity associated with their inhalation or their thermal decomposition into deleterious degradation products. Both PG and VG, which serve as carriers for the aerosolized delivery of nicotine and flavorings, can disintegrate into toxic carbonyls, including formaldehyde, acetaldehyde, and acrolein [6]. In a human clinical setting, the 2018 pilot study by Samburova et al. reported elevated breath carbonyls, with a mean retention and deposition within the respiratory and oral tracts reaching 99.7% ± 0.9% for formaldehyde and 91.6% ± 9.9% for acetaldehyde [13]. Thermal decomposition of e-liquid base materials also results in the formation of reactive oxygen species (ROS).

Furthermore, chemical analyses show that metallic heating elements and internal components of e-cigarettes lead to heavy metal exposure, as toxicants such as nickel, cadmium, chromium, and lead are transferred into the e-liquid. Other toxic emissions present in the aerosol largely depend on the flavoring compounds used [6,14]. Crucially, the wide variety of available ENDS products complicates risk assessment. Variations in device generations (e.g., disposable cigalikes vs. customizable mod systems), differences in battery voltage, variable nicotine concentrations (ranging from low-dose freebase nicotine to high-concentration nicotine salts), and an immense array of flavoring agents directly alter the chemical payload delivered to the oral cavity [6].

As illustrated in Table 1, aerosol constituents can substantially alter the physicochemical environment of the oral cavity. The most significant changes include nicotine deposition, xerostomia, oxidative stress and pH alterations. Approximately 45% of the nicotine released from ENDS is deposited directly within the oral cavity [8]. The hygroscopic nature of PG and VG has been linked to mucosal dehydration and mouth dryness with clinical observations identifying increased salivary viscosity in vapers [15,16]. Furthermore, in vitro studies demonstrate that ENDS aerosols generate significant quantities of ROS – roughly 7 × 1011 free radicals per puff – which is comparable to traditional cigarettes. This process may overwhelm the salivary antioxidant system (including enzymes such as peroxidase and glutathione), resulting in potential irreversible modification and oxidative DNA damage [8]. Vaping also modifies salivary pH patterns, with some observational studies reporting significantly higher pH values in e-cigarette users in comparison to both smokers and non-smokers [17].

TABLE 1. Impact of ENDS aerosol constituents on the oral environment.

Category	Specific Stressor	Primary Source	Impact on Oral Tissues & Microbiota
Carbonyls	Formaldehyde, Acetaldehyde, Acrolein [6]	Thermal decomposition of PG/VG and flavoring compounds [14]	DNA damage and impaired DNA repair activity; DNA adduct formation; protein cross-linking; [18] inhibition of commensal bacterial growth [4].
Heavy Metals and elements	Ni, Cd, Cr, Pb, As, Si [14]	Metallic heating coils and internal components of ENDS [6]	Genotoxicity, mutagenesis, generation of ROS (oxidative stress), epigenetic modifications, inflammation [8]; can lead to the development of periodontitis and oral cancer; [14] inhibiting osteoblast proliferation and a chemokine-mediated reduction in bone-forming processes [19].
Alkaloids	Nicotine [1,2]	E-liquid (nicotine salts/ freebase) [1,2]	Vasoconstriction of gingival capillaries; [19] impaired wound healing; increased biofilm formation and *S. mutans* viability [4].
Carrier Liquids	PG, VG [1,2]	Base e-liquid solution [1,2]	Mucosal dehydration (xerostomia); increased biofilm viscosity; altered pH patterns favoring acidogenic species [8]. Cell envelope biogenesis, extracellular matrix production, structural and metabolic roles, adaptive signaling [2].
Toxic Flavoring Agents	Cinnamaldehyde, Menthol, Vanillin, Diacetyl, Pyrazine derivatives, Benzaldehyde derivatives, Maltol [8]	Flavor additives [8]	Direct cytotoxicity to periodontal ligament cells; disruption of mitochondrial function in oral epithelial cells; irritants and sensitizers known to cause occupational asthma [6,20,21].*

* Plethora of cytotoxic, inflammatory, and oxidative effects dependent on specific toxicants

Disruptions of Oral Microbiota Homeostasis in E-cigarette Users

Characteristics of E-cigarette-induced Dysbiosis

The use of e-cigarettes appears to rapidly alter both the oral microbiome and its underlying biofilm architecture. In controlled in vitro experiments, the transition from a health-associated microbial community toward a pathogen-enriched, disease-promoting state can take place within just 24 hours of exposure to propylene glycol or vegetable glycerin vehicle. These compounds act as a catalyst for microbial transformation, largely because certain bacterial species can use them as a carbon-rich substrate to build cellular structures [2].

In cross-sectional clinical evaluations of e-cigarette users without periodontal disorders, the subgingival microbiomes show pathogen overrepresentation and virulence signatures that resemble those observed in profiles of patients suffering from severe periodontitis. The resulting metagenomic signature is unique enough for machine-learning models to reliably separate e-cigarette users from both non-smokers and traditional tobacco smokers [22]. While the overall number of bacterial species (alpha diversity) does not significantly differ between users and non-users, the structural community of the microbiome (beta diversity) is distinctly altered, reflecting a fundamental ecological shift [9].

Clinical human cohort data show that e-cigarette users develop a distinctive, stable microbial profile consisting of an overgrowth of anaerobic taxa such as Fusobacterium and Bacteroidales, which are heavily linked to periodontal inflammation and tissue damage [22]. There is also a significantly higher relative abundance of Veillonella, a genus of bacteria known to act as an opportunistic pathogen and stimulate the growth of other harmful organisms in the oral cavity [23]. This bacterial shift correlates with elevated inflammatory markers; TNF-α levels exceed those observed in both conventional smokers and non-smokers, while IFN-γ, IL-2, and IL-10 concentrations are notably higher in comparison to non-smokers [22]. However, because these human studies are primarily cross-sectional rather than longitudinal, it is difficult to definitively establish whether vaping directly causes this dysbiosis over time or if confounding lifestyle habits contribute to the shift.

More specifically, cross-sectional profiling suggests that vaping leads to a decrease in beneficial bacterial groups such as Bacteroidota, alongside an increased abundance of potentially harmful bacterial phyla including Spirochaetota and Synergistota, creating a dysbiotic environment that is may be less resilient to oral diseases [24]. The shifts observed in the oral microbiome suggest an increased risk for oral diseases, particularly in individuals exposed to multiple risk factors [9, 24]. Importantly, clinical research shows that dual users of both e-cigarettes and conventional cigarettes exhibit a microbiome profile distinct from exclusive vapers, with higher diversity and enrichment of multiple bacterial taxa [23]. Among these are Tannerella forsythia and Porphyromonas endodontalis, which have strong associations with periodontitis and endodontic infections [23]. This indicates that the combination of vaping and smoking conventional cigarettes creates an environment that favors pathogenic microbes over healthy, commensal bacteria [23].

One of the mechanisms responsible for alterations in the composition of the microbiota identified in cell and tissue cultures is the negative impact of e-cigarette aerosols on symbiotic bacteria that are normally present in the oral cavity. In vitro exposure significantly inhibit the growth and biomass accumulation of beneficial commensal bacteria, particularly S. sanguinis and S. gordonii. This effect can be observed regardless of the presence of nicotine or menthol flavorings in the aerosol [4]. As a result, an ecological niche is created, which begins to be occupied by other bacterial species. The growth rate and viability of the pathogenic S. mutans remain completely unchanged in these laboratory models, giving it a clear competitive advantage [4].

Moreover, in vitro researchreveal that exposure to e-cigarette aerosols further enhances the ability of S. mutans to form biofilm and increases the hydrophobicity of its cell surface. At the same time, the biofilm-forming capacity of S. sanguinis and S. gordonii does not improve or undergo significant changes under exposure to e-cigarettes. Consequently, S. mutans gains a greater ability to adhere to oral epithelial cells compared to commensal bacteria [4]. While these laboratory findings provide convincing insight these mechanisms, caution must be exercised when translating these results to living human patients, as the complex buffering capacity of natural human saliva and physiological shear forces are difficult to fully replicate in vitro. Environmental stress induced by e-cigarette use disproportionately damages the beneficial oral microbiota in vitro, while not negatively affecting – and even enhancing – the pathogenic traits of harmful bacteria. By hindering commensal strains and promoting the attachment of S. mutans, vaping could potentially lead to opportunistic infections, dental decay and other oral health issues [4].

Another example of a pathogenic bacterium that gains an advantage upon exposure to e-cigarette aerosols in laboratory setups is S. aureus. Similarly to S. mutans, its ability to form biofilm and adhere to oral epithelial cells increases, which constitutes a key initial step in colonization [25]. However, the development of dysbiosis is also influenced by the effects of e-cigarette aerosols on the host itself, particularly on its defense mechanisms. Cultured human oral epithelial cells exposed to the aerosol show a significant decrease in the secretion of key pro-inflammatory cytokines- such as IL-8, IL-6, and IL-1β. Under physiological conditions, these cytokines are responsible for recruiting immune system cells to eliminate bacterial infections. Their reduced secretion leads to an impaired ability of epithelial cells to eliminate S. aureus, whose uncontrolled growth remains largely unimpeded [25].

It has also been demonstrated in vitro that e-cigarette aerosols induce dose-dependent toxicity and DNA damage in oral epithelial cells, regardless of the presence of nicotine in the liquid. Moreover, simultaneous in vitro exposure to the aerosol and S. aureus results in increased expression of the inflammatory regulator COX-2. The resulting chronic inflammatory state, combined with delayed cellular repair and DNA damage, promotes conditions that increase the risk of mutagenesis and the progression of precancerous lesions and oral cancers [25]. Animal models exposed to e-cigarette vapor have supported these inflammatory findings, demonstrating compromised mucosal integrity and localized soft-tissue damage; however, animal tissue layout and metabolic pathways differ from human oral biology, which limits direct translational clinical assumptions [4,6,10].

Beyond compositional changes, e-cigarette aerosol exposure alters the functional activity of the subgingival microbiome. Functional profiling of oral plaque shows enrichment

of biological pathways related to inflammation, lipid metabolism and the degradation of xenobiotics. Salivary testing also identifies significant metabolic disruptions, including abnormal shifts in amino acid and nucleotide metabolism, which directly correlate with the altered microbial composition [24].

Importantly, the severity of these changes appears to be influenced by vaping behavior. Higher puff volumes are associated with more pronounced decreases in microbial diversity and more severe functional alterations. This demonstrates a potential dose-dependent relationship, where more intensive e-cigarette use leads to a progressively unhealthier oral environment and greater disruption of microbial homeostasis [24].

Dysbiosis-Driven Periodontal Pathology

The use of electronic nicotine delivery systems carries profound and well-documented consequences for oral health and is associated with significantly elevating the risk of both hard and soft tissue diseases. Observational patient data indicate that e-cigarette users demonstrate a distinct susceptibility to aggressive dental caries and advanced periodontal disease [26, 27]. Exposure to e-cigarette aerosols disrupts the oral microbiome, leading to increased biofilm adhesion on tooth surfaces and predisposing individuals to the development of caries and periodontitis [26]. This effect may be further compounded by the sweet flavorings in e-liquids, which actively increase the risk of tooth decay [27] .

Compared to non-smokers, cross-sectional clinical trials indicate that e-cigarette users exhibit significantly poorer periodontal parameters, including an elevated plaque index (PI), increased probing depth (PD), and advanced marginal bone loss (MBL) [15,19]. The tissues surrounding dental implants appear to be similarly vulnerable to destructive inflammatory processes associated with e-cigarette use, which also increasesthe risk of peri-implantitis. Consequently, aesthetic defects also emerge, manifesting as the discoloration of both natural teeth and dental prostheses [26,27] .

E-cigarette use frequently inflicts damage on the oral soft tissues. The most commonly reported adverse effect in epidemiological surveys is xerostomia, accompanied by oral and pharyngeal irritation [28]. E-cigarette smokers also face a higher incidence of oral mucosal lesions, nicotine stomatitis (smoker's palate), hairy tongue, and opportunistic fungal infections caused by Candida albicans [27,28]. Furthermore, in vitro studies have demonstrated that ENDS liquids and aerosols are cytotoxic to human gingival fibroblasts and periodontal ligament cells, inducing apoptosis and triggering inflammatory responses that actively contribute to the breakdown of periodontal tissues [28,29]. Nevertheless, the wide variance in clinical trial outcomes means that these clinical risks must be interpreted with caution.

The diagnosis of periodontal pathology in smokers is often significantly delayed. Bleeding on probing (BoP) in e-cigarette users is substantially reduced compared to non-smokers, closely mirroring the suppressed bleeding seen in conventional smokers. This scant or absent bleeding effectively masks the clinical signs of latent inflammation and early periodontal disease, hindering a prompt and accurate diagnosis [19].

Beyond local tissue destruction, ENDS aerosols exhibit significant carcinogenic properties in lab assays [26,27]. Carcinogens present in the aerosol,such as formaldehyde and nitrosamines, induce single-strand and double-strand DNA breaks and promote abnormal cellular migration in vitro [28]. Although the nicotine present in the aerosol is not traditionally classified as a direct carcinogen, cellular models suggest that it actively inhibits apoptosis in precancerous oral lesions, allowing damaged, potentially malignant cells to survive and proliferate [28, 29]. Similarly, clinical reports have also emerged documenting aggressive oral squamous cell carcinoma among young, otherwise healthy e-cigarette users [28]. However, these case studies are descriptive and cannot be utilized to prove direct and epidemiological causality.

CONCLUSIONS

ENDS can modify the physicochemical environment of the oral cavity through the significant deposition of nicotine, toxic carbonyls and heavy metals. The remarkably high retention of formaldehyde and acetaldehyde within the oral and respiratory tracts, combined with the massive generation of ROS, has been shown in laboratory models to overwhelm salivary antioxidant defenses and induces oxidative DNA damage. These alterations facilitate oral dysbiosis and shift the microbial balance toward periodontal pathogens, thereby increasing the risk of chronic inflammatory conditions and tissue degradation. Consequently, e-cigarette use should be regarded as an environmental stressor and a significant risk factor for the development of periodontal pathologies. However, the current body of literature is characterized by significant methodological heterogeneity, limited long-term clinical data and a heavy reliance on in vitro and animal models. Product variations in voltage, e-liquid bases and nicotine levels further confound direct clinical cross-comparisons. Therefore, well-controlled, longitudinal human clinical investigations are absolutely essential to fully elucidate the long-term impact of these emerging nicotine delivery systems on the stability of the oral microbiome and overall oral health.

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