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REVIEW ARTICLE **OPEN ACCESS**

E-Smoking and Periodontal Diseases: Current Insights and Future Directions

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Abstract:

Introduction/Background: Periodontal diseases are long-term inflammatory diseases of the gingiva and alveolar bone that can result in the loss of teeth in the case of untreated conditions. Despite the fact that conventional smoking is an established risk factor, the widespread adoption of electronic cigarettes (e-cigarettes) has raised new concerns about periodontal health.

Methods and Materials: PubMed, Scopus, and Google Scholar were utilized for carrying out this narrative review. Keywords were e-cigarettes, vaping, electronic nicotine delivery systems, periodontal disease, periodontitis, oral microbiome, gingival inflammation, and oxidative stress. Publications up to February 2026 were considered, with systematic reviews and meta-analyses (2023–2025) prioritized. Evidence on clinical periodontal parameters, inflammatory biomarkers, and changes in the microbiome was synthesized.

Results: E-cigarette smoking is linked to elevated levels of gingival inflammation, oxidative stress, immune dysregulation, enrichment of inflammatory pathogens like Porphyromonas gingivalis and compromised tissue repair. Periodontal parameters tend to be poorer than in non-smokers but better than in traditional smokers, which demonstrates a moderate risk profile.

Discussion: E-cigarettes seem to be an intermediate risk factor of periodontal disease. The mechanisms involved are oxidative stress, cytotoxicity, neutrophil dysfunction and microbial dysbiosis, though usually at reduced intensity compared to traditional smoking. Nevertheless, the existing evidence is constrained by cross-sectional designs, brief follow-ups, and confounding variables, including multiple uses or prior smoking, which makes it challenging to reach logical conclusions regarding long-term effects.

Conclusion: Current evidence suggests that e-cigarette use is associated with adverse changes in periodontal health and should not be assumed to be biologically inert for oral tissues. Its long-term periodontal effects are not well understood, although it is not as harmful as combustible tobacco. More longitudinal research is required, and dental practitioners are advised to counsel patients on the dangers of vaping and ways of quitting.

Keywords: E-cigarettes, Vaping, Periodontal diseases, Periodontitis, Oral microbiome, Inflammation, Oxidative stress.

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1. INTRODUCTION

Periodontal diseases, also known as gum diseases, are long-term inflammatory disorders that are mainly caused by bacterial infection of the gums and the alveolar bone that supports the teeth. In the absence of appropriate intervention, the progression of these infections causes a progressive loss of periodontal tissues, which causes tooth mobility and ultimately loss of teeth [1].

Oral diseases represent a significant global health problem, affecting an estimated 3.7 billion individuals worldwide, according to the latest estimates [2], revised based on the WHO 2025 fact sheet and GBD 2021 analyses. Untreated caries in permanent teeth is the most common of these conditions, and severe periodontal disease (probing pocket depth of 6 mm or more or clinical attachment loss of 6 mm or more) occurs in more than 1 billion adults worldwide, the prevalence of which is about 12.5% of the age-specific population [3-5]. This is particularly true of low- and middle-income nations, as access to preventive and curative dental services is limited, which adds to the pain, functional disability, cosmetic issues, and diminished quality of life.

Periodontal diseases are etiologically multifactorial, with dysbiotic oral biofilms, genetic predisposition, and environmental and modifiable risk factors such as poor oral hygiene, systemic conditions (*e.g.*, diabetes, obesity), and tobacco use [6]. Traditional cigarette smoking is a well-established, significant risk factor, which enhances the onset, progression, and severity of the disease by affecting the body via host immune deficiency, vascular alterations, and alterations in the subgingival microbiota [7].

Electronic Nicotine Delivery Systems (ENDS) or e-cigarettes or vapes have been rapidly gaining popularity over the past few years as an apparently less harmful substitute for conventional tobacco smoking, especially among younger demographics. Despite a body of evidence that ENDS can be less harmful than combustible cigarettes in general, recent studies suggest that they can have negative effects on oral health, such as gingivitis and periodontal microbiota changes, increased probing depths, and poorer responses to non-surgical periodontal therapy than non-users [8, 9]. Systematic reviews and meta-analyses (2023–2025) consistently report that e-cigarette users exhibit intermediate periodontal risks, worse than never-smokers but better than conventional smokers in some parameters (bleeding on probing, plaque accumulation), yet long-term data remain limited [10-12].

The rapid increase in vaping, with its incentives of flavored products, aggressive advertising, and the belief of harm reduction, raises significant concern regarding periodontal health, particularly among adolescents and young adults, whose susceptibility to nicotine addiction and frailty of oral tissue are increased [13]. Other risks are systemic effects, like E-cigarette or Vaping product use-Associated Lung Injury (EVALI), nicotine dependence disrupting brain development in adolescents, and high cardiovascular parameters [14-16].

This review seeks to critically synthesize and critique the existing evidence on e-cigarette use and periodontal health, comparing outcomes across e-cigarette users, traditional smokers, and non-smokers, clarifying underlying biological pathways, and identifying gaps to guide future research and clinical practice.

2. REVIEW METHODOLOGY

This study was developed as a narrative review. To identify relevant articles on the effect of electronic cigarette (e-cigarette) use on periodontal health, a literature search was conducted across electronic databases, including PubMed, Scopus, and Google Scholar. The search terms were based on combinations of the following terms: e-cigarettes, vaping, electronic nicotine delivery systems, ENDS, periodontal disease, periodontitis, probing depth, clinical attachment loss, bleeding on probing, plaque index, oral microbiome, gingival inflammation, oxidative stress, inflammatory biomarkers, Porphyromonas gingivalis, systematic review. Articles published in English up to February 2026 were included, with an emphasis on systematic reviews and meta-analyses published in 2023-2025 to obtain the most recent evidence. Eligible studies included clinical parameters of periodontal health and inflammatory and oxidative stress biomarkers of interest, adaptations in the oral microbiome, and comparisons of individual use of e-cigarettes, conventional smoking, and non-smoking. Case reports, editorials, conference abstracts, non-peer-reviewed materials, and purely animal or *in vitro* studies lacking human clinical correlation were removed. Study selection was conducted in two stages: titles and abstracts were first screened for relevance, followed by full-text review of potentially eligible articles. The chosen articles were combined to give a general perspective of the existing evidence on e-cigarette use and periodontal disease. A total of 84 articles met these criteria and were incorporated into this review.

2.1. Pathophysiology and Systemic Risk Factors of Periodontal Disease

Periodontal disease is an inflammatory disease with many factors, which are systemic, environmental, and genetic. Although microbial biofilm is the major etiologic agent, there are several host-related factors that modify the disease severity and progression. Chronic alcohol consumption has been associated with impaired neutrophil and immune function, disrupted vitamin K levels, and reduced bone formation, all of which may increase susceptibility to periodontal disease [10, 11, 17, 18]. Diabetes mellitus has a well-established bidirectional relationship with periodontal disease, whereby hyperglycemia impairs immune response and vascular integrity while periodontal inflammation adversely affects glycemic control [13]. Obesity has been linked to periodontal tissue breakdown through elevated pro-inflammatory cytokines, oxidative stress, and endothelial dysfunction [19]. Vitamin D and calcium deficiency have similarly been associated with impaired gingival immune defense and increased severity of periodontal destruction

[20, 21]. Genetic polymorphisms in inflammatory and matrix-degrading genes (*e.g.,* IL-1, IL-6, IL-10, MMP variants) have also been linked to periodontal disease susceptibility [22].

3. TRADITIONAL SMOKING AND PERIODONTAL DISEASES

One of the most modifiable risk factors of periodontal diseases is tobacco smoking, which has a tremendous impact on the onset of the disease, its progression, severity, and response to treatment. Since the mid-20th century, the detrimental association between smoking and periodontal health has been well established, and the available evidence indicates that smokers have a higher disease prevalence, more severe disease (deeper pockets, greater attachment loss), and faster alveolar bone loss than non-smokers [23].

It is dose-related, and the more cigarettes are smoked per day and the longer the period of exposure, the worse the periodontal outcome. Sex differences are also observed, with males are mostly affected more severely, which may be associated with increased consumption levels, hormonal effects, or behavioral variables [24]. Smoking's pathogenic effects are mediated by several interrelated mechanisms.

Altered host immune-inflammatory responses: smoking impairs neutrophil function (decreased chemotaxis, phagocytosis, and oxidative burst), lowers salivary IgA levels, and dysregulates cytokine production (high TNF-alpha, IL-1β; low IL-10), resulting in ongoing chronic inflammation and inability to eliminate infection.

Weakened tissue healing and vascular homeostasis: Nicotine and other smoke components inhibit fibroblast growth, collagen production, and angiogenesis and stimulate vasoconstriction and hypoxia of gingival tissues, retarding wound healing and increasing bone resorption.

Microbiome dysbiosis: smoking changes the microbial composition of the subgingival area, promoting the growth of keystone and red-complex pathogens and reducing the growth of beneficial species [25]. *P. gingivalis,* a black-pigmented, gram-negative anaerobe (phylum Bacteroidetes), is a pivotal pathogen central to this dysbiosis and to periodontitis development. As a low-abundance keystone species, *P. gingivalis* coordinates community-wide changes via virulence factors, including gingipains (cysteine proteases that degrade host proteins and complement components), lipopolysaccharide (LPS), and fimbriae, which facilitate immune evasion and synergistic dysbiosis [26-28]. *In vitro* models have consistently shown that cigarette smoke extract increases *P. gingivalis* biofilm formation, expression of virulence genes (gingipain upregulation), and host cell invasion, intensifying tissue destruction [29-31].

The epidemiological evidence supports the claim that smokers have increased subgingival loads of *P. gingivalis* and *Aggregatibacter actinomycetemcomitans*. In some cases, the latter has threefold higher odds of occurrence [32, 33]. Recent systematic reviews and meta-analyses (2024-2025) support the role of smoking in immunosuppression, microbial alterations, and poorer treatment outcomes (reduced gains in probing depth and clinical attachment following scaling and root planing). These results align with traditional smoking as a leading cause of periodontal loss and present a powerful comparator for newer nicotine delivery methods such as e-cigarettes.

4. E-SMOKING: BACKGROUND AND USAGE TRENDS

Electronic cigarettes (e-cigarettes, or vapes) are battery-powered, rechargeable machines that aerosolize e-liquids to be inhaled, providing nicotine (or non-nicotine versions) without burning tobacco. Fundamental elements consist of a refillable/disposable cartridge or pod with e-liquid (propylene glycol, vegetable glycerin, flavorings, and optional nicotine), an atomizer coil to heat the liquid into vapor, a lithium-ion battery, an airflow sensor to activate the device, and, in most cases, an LED indicator light [34].

Chinese pharmacist Hon Lik, who was inspired to create the device by his father's death from lung cancer caused by tobacco, invented it in 2003. Precursors became widespread in China and then in the UK (2005) and the USA (2007) as commercially sold harm-reduction devices. Although their use has been increasing, the World Health Organization (WHO) has repeatedly cautioned against promoting e-cigarettes as a smoking-cessation aid, citing insufficient long-term safety and efficacy data. It notes, however, that switching from combustible cigarettes to e-cigarettes may still offer some benefit for certain adult smokers.

The consumption of nicotine products in the world remains high, with an estimated 1.2 billion tobacco users in 2024 (compared to 1.38 billion in 2000). The prevalence of e-cigarette/vaping has risen sharply: the WHO gave the first global estimate (October 2025) of over 100 million vapers globally (at least 86 million of them adults, the largest number in high-income countries) and 15 million teenagers aged 13-15. Young people are disproportionately affected, being up to nine times more likely to vape than adults in countries with available data. Regionally, usage is highest in Europe (~20–25 million users), followed by the Americas and the Western Pacific [35].

5. E-SMOKING HEALTH OUTCOMES

The aerosols of e-cigarettes are formed after the heating of e-liquids that include propylene glycol (PG), vegetable glycerin (VG), nicotine (variable amounts), flavors, and other additives. There is no smoking of tobacco, but complex forms of potentially harmful compounds are produced by thermal degradation in the vaporization process. Several studies (qualitative and quantitative) have detected numerous chemicals in e-liquids, cartridges, and aerosols, which are usually higher than those in the parent liquid as a result of pyrolysis and oxidation reactions [36-38].

Complete analyses (2024-2025) report 60-113+ compounds per product, such as nicotine and related alkaloids, solvent carriers PG/VG, and carbonyls (formaldehyde, acetaldehyde, acrolein), Volatile Organic Compounds (VOCs such as benzene), phenolic compounds, and polycyclic aromatic hydrocarbons. These constituents depend on the type of device (pod vs. mod), power settings, puff topography, and e-liquid formulation, and higher temperatures and more complex flavors yield more toxicants [39]. Although generally lower than in cigarette smoke, cumulative exposure is problematic in terms of chronic effects, especially in the oral cavity, as it is the main location of deposition.

5.1. Nicotine

Nicotine delivery has driven the rise in e-cigarette popularity, and high-concentration preparations (nicotine salts in pods) facilitate quick systemic delivery and dependence, particularly in youth who have never smoked combustible tobacco [40]. There is prenatal and second/third-hand exposure that is dangerous to the development of the body; there are risks of neurotoxicity in adolescents and teratogenicity with prenatal exposure. Nicotine exhibits dual, context-dependent effects: systemically (*e.g.*, in models of sepsis or arthritis), it acts as an anti-inflammatory agent via cholinergic pathways. In contrast, in oral conditions, it is pro-inflammatory, which increases gingivitis and periodontitis in pathogen-rich environments [41]. It is associated with vasoconstriction (endothelium-dependent/independent), decreasing the flow of blood in the gums and nutrient transfer and increasing vascular remodeling by the proliferation of smooth muscle cells and the deposition of a matrix [42]. Gingival perfusion studies have demonstrated transient, situation-dependent effects: intra-arterial nicotine decreases the flow in animal models [43], but laser Doppler flowmetry in humans demonstrates temporary elevation of flow in young/occasional smokers (because of an increase in blood pressure and heart rate) but tolerance in chronic smokers with no net effect [44, 45]. These vasodilatory effects disrupt tissue homeostasis and conceal evidence of inflammation despite the persistence of tissue damage [46].

Nicotine interferes with fibroblast function, increases ROS/cytotoxins, modulates the microbiota, and dysregulates miRNAs, all of which contribute to inflammation and tissue destruction in periodontal contexts. In general, even though it is less than combustible sources, chronic nicotine from vaping maintains periodontal progression risks.

5.2. Propylene Glycol (PG)

PG, the primary humectant (typically 30–60% of e-liquids), produces irritant aerosols when heated. Inhalation can result in mucosal irritation (throat/lung symptoms in minutes), and productive cough, fever, and dyspnea are reported, similar to lipoid pneumonia-like dyspnea [47]. PG increases the risk of asthma and enters the mucous membranes/skin and is metabolized in the liver to acetone/pyruvic acid and overloads the kidneys. High-dose inhalation (heavy vaping) causes toxicity through oxidative stress and organ burden [48].

Oral effects include dry mouth, gingival/mucosal irritation, and possible contributions to dysbiosis or inflammation. Recent reviews have attributed decreased mucociliary clearance and cytotoxicity of airway/oral epithelium to the use of PG/VG aerosols, raising concerns about periodontal vulnerability. Although ingestion of PG is GRAS, there is concern regarding chronic aerosol exposure in oral health.

5.3. Metals

Trace metals (coils, solders) leak into aerosols, such as chromium, nickel, tin, cadmium, lead, aluminum, copper, and others. These are known carcinogens/irritants that are associated with lung/sinonasal cancers and chronic inflammation [49]. Oral tissue metals can build up in the mucosa/periodontal ligament, potentially contributing to periodontitis through cytotoxicity, ROS production, and immune dysregulation.

Copper is a respiratory irritant that facilitates macrophage migration, eosinophilia, granulomas, fibrosis, and pulmonary inflammation, which are more pronounced than those observed with other transition metals [50-53]. Cadmium (even at 0.1-10 μM) suppresses IgE production, disrupts B-cell activation/survival/proliferation, and has immunotoxic/modulatory effects [54, 55]. Although there is limited data regarding oral-specific effects, metal exposure can increase periodontal inflammation and cause oral carcinogenesis. The levels depend on the age/power of the device and the need to monitor them in the long term.

Tobacco-Specific Nitrosamines (TSNAs), such as NNK and NNN, are strong carcinogens associated with pancreatic, lung, esophageal, and oral cancers in tobacco smokers [56]. TSNAs occur as nicotine impurities or thermal by-products in e-cigarettes, but in minute amounts [57-60]. Recent systematic reviews (2025) indicate that e-cigarette aerosol TSNA levels were lower than cigarette smoke (greater than 99% lower), with NNK/NNN being the foremost compounds of primary concern. Organ-specific carcinogenesis (NNK: lung/nasal/liver tumors; NNN: nasal/esophageal) is confirmed in animal models, where these carcinogens undergo metabolic activation to form DNA adducts [59, 60].

Although less than that of combustible tobacco, chronic low-level exposure increases the risk of oral cancer, particularly with flavored/high-nicotine products. Youth/adolescent use increases concern because there is a lifetime exposure.

E-smoking brings a variety of chemicals, which are associated with addiction, inflammation, oxidative stress, and possible carcinogenesis. Although aerosols are less dangerous than combustible tobacco, they have oral risks (periodontal inflammation, dysbiosis, mucosal changes). Long-term studies, standardized aerosol testing, and regulation are crucial in reducing harm, especially to vulnerable populations.

6. E-SMOKING AND PERIODONTAL DISEASES ASSOCIATION

6.1. Periodontal Clinical Parameters

The systemic and oral impacts of e-cigarette use are harmful, and systematic reviews and meta-analyses of 2024-2025 invariably observed mouth and throat irritation and periodontal changes as frequent consequences [61]. Mucosal irritation is commonly reported in new users who have never smoked before, and in those who switch from vaping to combustible cigarettes [62]. Vaping also has periodontal repercussions, such as elevated probing depths, elevated plaque accumulation and gingival inflammation, and clinical attachment loss. However, such effects are typically not as severe as those seen in traditional smokers [63].

6.2. Oral Microbiome and Inflammatory Mechanisms

The association between vaping and periodontal pathology has three main mechanisms that are similar but milder than those associated with tobacco smoking. To start with, e-cigarette aerosols have been shown to induce cytotoxicity and DNA damage in oral keratinocytes and gingival fibroblasts and chronic inflammation by promoting oxidative stress in the presence of Reactive Oxygen Species (ROS) and aldehydes; neutrophil dysfunction; and increased pro-inflammatory cytokines (IL-1β, IL-6, and TNF-α). Second, vaping contributes to oral microbiome dysbiosis by stimulating the growth of periodontal pathogens (*e.g., P. gingivalis* and *Aggregatibacter actinomycetemcomitans)* and reducing overall microbial diversity, therefore promoting pathogenic biofilms; research shows an increase in subgingival pathogen loads in vapers compared to non-smokers [64-66]. Third, soft and hard tissue healing is compromised as a result of decreased fibroblast activity, loss of collagen synthesis, and temporary changes in the vascularity that change the blood flow in the gingivae and eventually inhibit wound healing and increase bone loss.

6.3. Inflammatory and Oxidative Stress Biomarkers

These biological alterations translate into clinical effects: short-term smoke-to-vape switchers (over two weeks) show increased gingival inflammation [67, 68]. Randomized trials have shown that harmful or potentially harmful constituents, oxidative stress, and inflammation biomarkers are reduced with the switch to particular electronic nicotine delivery systems or with abstinence [12]. There are also lower biomarkers of potential harm and better inflammatory and oxidative stress markers in long-term users of products like JUUL than in those who smoke [69].

6.4. Treatment Response

Vapers frequently exhibit clinical signs, including high plaque index, bleeding index, probing depth, and clinical attachment loss, that together augment the risk of tooth loss in the absence of treatment [70-75]. Moreover, non-surgical periodontal therapy like scaling and root planing has poorer responses in e-cigarette users due to aggravated host reactions and sustained changes in the microbiome, a trend that resembles that of smokers [76-78].

The major inconsistencies in existing literature are due to the cross-sectional nature of the study designs, the presence of confounding variables such as previous smoking history, variability of devices and e-liquids, and lack of long-term data. High-quality longitudinal studies are therefore urgently needed to clarify the long-term impact of vaping on periodontal disease.

7. LESSONS ON CONTROVERSIES, GAPS IN RESEARCH AND FUTURE DIRECTIONS.

It is still controversial to consider e-cigarettes as harm-reduction tools. Health authorities are divided. The U.S. CDC emphasizes the insufficient evidence available to draw firm conclusions, whereas the U.K. NHS encourages change among adult smokers (providing free devices) according to the reduced harm profiles. Australia requires prescriptions; there are countries that prohibit sales altogether. The involvement of tobacco industries raises skepticism, as there is a history of misinformation; experts stress the need for clear, evidence-based communication to minimize risks [79-81].

Although much work has been done, there are gaps: the majority of studies are cross-sectional/short-term, which restricts causal inference. Longitudinal study designs are required to determine chronic periodontal progression, establish causal relationships, and assess interactions with device/e-liquid factors. Vaping needs to be thoroughly compared with smoking in terms of microbiome changes, inflammation, and treatment response, particularly given its promotion as a cessation/harm-reduction tool. The roles of individual e-liquid components (PG/VG, flavors, metals) are under-researched; the discovery of the harmful ones may inform safer formulations.

Future directions: large-scale, prospective cohorts using standardized vaping measurements; mechanistic (aerosol exposure modeling) studies; prevention (targeting youth) studies; policy (flavor bans) studies. The evidence-based regulation will be informed by high-quality RCTs/meta-analyses that will shed light on the relative risks.

8. RESULTS

A total of 84 human studies met the inclusion criteria for this review and were synthesized across four outcome domains: periodontal clinical parameters, oral microbiome composition, inflammatory/oxidative stress biomarkers, and treatment response following periodontal therapy. Across these domains, e-cigarette users consistently demonstrated an intermediate periodontal risk profile, worse than non-smokers but generally less severe than conventional cigarette smokers, although the strength of this pattern varied by outcome and study design. Studies assessing clinical periodontal indices consistently reported elevated probing depth, plaque index, bleeding on probing, and clinical attachment loss among e-cigarette

users compared with non-smoking controls [61-63, 82]. These differences were most pronounced in cross-sectional comparisons of established vapers versus never-users, while studies including dual users or recent smoking-to-vaping switchers showed more variable magnitudes of effect. Across the reviewed populations, clinical severity among e-cigarette users did not typically reach the levels observed in conventional smokers, supporting a graded rather than binary risk relationship [63]. Microbiome-focused studies demonstrated that e-cigarette use was associated with increased subgingival loads of periodontal pathogens, including *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans*, alongside a measurable reduction in overall microbial diversity [64-66, 83]. This shift toward a dysbiotic, pathogen-enriched biofilm mirrors, though generally to a lesser degree, the microbial changes reported in traditional smokers and appeared consistent across the populations and sampling methods used in the included studies. Biomarker studies revealed increased pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) and oxidative stress markers among e-cigarette users relative to non-smokers, though generally at lower levels than those observed in combustible-cigarette smokers [12, 67, 68]. Short-term smoke-to-vape switchers (over periods of two weeks or more) showed measurable increases in gingival inflammation during the transition period [67, 68], whereas longer-term users of specific products (*e.g.*, pod-based systems) exhibited more favorable biomarker profiles relative to continued smokers in comparative and randomized designs [12, 69].

Data on periodontal treatment outcomes indicated that e-cigarette users exhibited poorer clinical responses to non-surgical periodontal therapy (scaling and root planing) than non-smokers, with reduced gains in clinical attachment and smaller reductions in probing depth [70-75, 84]. These attenuated treatment responses were attributed to sustained host inflammatory reactivity and persistent microbiome alteration despite therapy, a pattern resembling, though generally milder than, that reported among conventional smokers [76-78]. Collectively, the reviewed evidence indicates a consistent, dose- and duration-dependent association between e-cigarette use and adverse periodontal outcomes across clinical, microbial, biomarker, and treatment-response domains. However, this body of evidence is predominantly cross-sectional, drawn from heterogeneous populations and device/e-liquid types, and frequently confounded by dual use or prior smoking history, limiting causal inference. Table **1** summarizes the key characteristics of the main included human studies, including study design, population and sample size, vaping exposure definition, comparator group, periodontal outcomes measured, key findings, and study limitations.

Table 1. Summary of major studies on e-cigarette use and periodontal health.

Author, Year	Study Design	Population	Vaping Exposure	Comparator	Main Periodontal Findings	Key Limitations
Thiem *et al.*, 2023 [5]	Systematic review & meta-analysis	Adults	E-cigarette use	Smokers & non-smokers	Increased probing depth, clinical attachment loss (CAL), and bleeding on probing	High heterogeneity
Charde *et al.*, 2024 [6]	Scoping review	General population	E-cigarette smoking	Non-users	Gingival inflammation and plaque accumulation	Limited long-term data
BinShabaib *et al.*, 2019 [48]	Cross-sectional	Smokers, e-cigarette users, and never-smokers	Electronic cigarette use	Cigarette smokers and never-smokers	Elevated inflammatory cytokines in gingival crevicular fluid	Small sample size
Xu *et al.*, 2022 [49]	Microbiome analysis	Periodontal sites	E-cigarette aerosol	Non-exposed individuals	Enrichment of periodontal pathogens	Focused mainly on microbiome changes
Pushalkar *et al.*, 2020 [52]	Experimental	E-cigarette users	Aerosol exposure	Non-users	Oral microbiome dysbiosis and increased infection risk	Short-term exposure
Shabil *et al.*, 2024 [12]	Systematic review & meta-analysis	E-cigarette users	E-cigarette use	Non-users and conventional smokers	Increased risk of periodontitis and adverse periodontal outcomes	Variability among included studies
Alkattan *et al.*, 2025 [79]	Systematic review & meta-analysis	Vapers	E-cigarette use	Non-vapers	Increased risk of periodontal disease and poorer periodontal health	Variable exposure definitions

CONCLUSION

The increased popularity of electronic cigarettes has raised new issues about their possible effects on periodontal health. The existing evidence suggests that e-cigarette use could be less toxic than traditional cigarette smoking, but it is not biologically neutral. Recent clinical, microbiological, and molecular evidence suggests that vaping is linked to augmented inflammatory reactions, oxidative stress, oral microbiome dysbiosis, and reduced healing of periodontal tissue, consistent with the broader recognized association between periodontal disease and systemic health [11]. Taken together, these results put e-cigarette use as a possible intermediate risk factor of periodontal disease higher than non-smoking, but overall lower than traditional tobacco exposure. Nonetheless, the literature at hand is constrained by the prevalence of cross-sectional designs, limited follow-up periods, diverse definitions of exposure, and confounding variables, including dual use and history of previous smoking. Therefore, long-term causal associations are yet to be determined. Clinically speaking, dental practitioners cannot assume a safe replacement of e-cigarettes as a periodontal health option. Patients using vaping products should be informed of the potential risks, monitored closely for early signs of periodontal inflammation, and counseled as part of comprehensive tobacco cessation programs. Public health messaging must be based on a balanced, evidence-based approach taking into consideration the harm-reduction controversies, as well as the emerging biological issues. The next generation of research needs to focus more on the design of longitudinal and mechanistic studies to help elucidate dose-response connections, isolate vaping-related impacts, and evaluate the long-term periodontal results. Until this kind of data is made available, electronic cigarette use must be handled carefully within clinical practice, with clear patient counseling regarding potential periodontal risks.

AUTHOR'S CONTRIBUTION

This is a single-author paper. All contributions were made by WY.

LIST OF ABBREVIATIONS

PD	=	PERIODONTAL DISEASE
CAL	=	Clinical Attachment Loss
BI	=	Bleeding Index
PI	=	Plaque Index
GCF	=	Gingival Crevicular Fluid
ENDS	=	Electronic Nicotine Delivery Systems
TSNA	=	Tobacco-Specific Nitrosamines
NNK	=	Nicotine-Derived Nitrosamine Ketone
NNN	=	N'-Nitrosonornicotine

CONSENT FOR PUBLICATION

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CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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