REVIEW ARTICLE

Impact of E-cigarettes and vaping on periodontal health: A narrative review

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ABSTRACT

The use of electronic cigarettes, "e-cigarettes," or vaping is growing in popularity, especially among adolescents and young adults. While the effects of cigarette smoking on oral health are well-established, the exact impact that e-cigarettes may have on dental tissues is still uncertain. The current review aimed to summarize evidence related to the effect of vaping on the periodontal health status of e-cigarette users. A comprehensive electronic search was conducted on the PubMed, Scopus, and Embase databases using the following search terms: Electronic cigarettes OR vaping OR electronic nicotine delivery systems OR e-cigarettes AND periodontitis. The search was limited to studies published from 2022 to 2025. The full-text articles published in the English language were selected for the review purpose. Thirty-nine clinical studies focusing on the effect of e-cigarette smoking on the periodontal clinical parameters, levels of inflammatory mediators, alteration in periodontal micro flora, and response to periodontal treatment were found to be eligible for inclusion in the review. Vaping may be associated with greater clinical attachment loss compared to non-smokers. Moreover, ECs are also associated with unfavorable effects on periodontal microbial counts, biomarkers of inflammation, and oxidative stress. Electronic cigarettes, "e-cigarettes," or vaping may play a role in the initiation and progression of periodontal disease by altering the host response, resulting in the release of inflammatory cytokines and periodontal microflora. Clinical studies show deleterious effects of vaping on periodontal health, as well as less favorable response to periodontal treatment is observed in e-cigarette users compared to non-smokers.

Key words: E-cigarettes, periodontal health, vaping

INTRODUCTION

Since being introduced in 2004, vaping or e-cigarettes has seen a striking worldwide increase in popularity, especially among teens and young adults.[1,2] "Vaping can be defined as inhaling an aerosolized "e-liquid," produced by an electronic vaporization device, which does not require combustion. Instead of burning tobacco, as with traditional cigarettes, e-cigarettes heat up and vaporize nicotine and other flavoring products.[3,4] Because of containing fewer ingredients and the absence of combustion, e-cigarettes and vaping products were, for a while, considered a safer alternative to cigarettes and even a potential tobacco-cessation product.[5,6]

An e-cigarette device contains a battery, a reservoir holding liquid, and a vaporization chamber with a heating element. The liquid is a solvent comprising nicotine, propylene glycol, glycerine, flavoring additives, and sweeteners.[7,8] While several of the e-liquid formulations contain mostly nicotine, they can also contain other drugs such as tetrahydrocannabinol (THC) and other substances such as methamphetamine, methadone, and vitamins.[9,10] When this liquid is heated, the e-cigarette creates an aerosol of fine particles that are systemically absorbed through the oral tissues and lungs for the nicotine to be delivered to the brain within a couple of seconds.[11,12]

Tobacco smoking is a major risk factor for several oral conditions including periodontal diseases and oral cancer. Cigarette smoke contains at least 500 potentially toxic substances, including hydrogen cyanide, carbon monoxide, free radicals, tar, nitrosamines, nicotine, and several oxidant gases.[13,14] The periodontal effects of tobacco smoke are thought to occur through the systemic effects of these toxic

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constituents on immune function and the inflammatory response within the periodontal tissues.[15,16] Nicotine, the main psychoactive, chemically addictive component in tobacco smoke, is a primary ingredient in e-cigarette liquids. Vaping, or the use of e-cigarettes, has been the subject of increasing scientific scrutiny regarding its potential impact on systemic and oral health. Several studies have attempted to discuss the impact of vaping on oral health and dentition.[17,18]

In vitro studies have demonstrated that aldehydes and free radicals contained in the aerosols of e-cigarettes cause oxidative stress, alterations in cellular antioxidant activity and DNA damage, changes that are expected to ultimately lead to periodontal tissue destruction and alveolar bone loss, characteristics of periodontal diseases. Some *in vivo* studies and clinical trials, however, had conflicting findings.[15,16] Some reports have suggested that e-cigarettes are less harmful than conventional cigarettes but might still affect periodontal health.[15,16] Other studies had reported significant oral health implications including vaping-related cancerous changes, more infections, xerostomia, and traumatic injuries or burns.[15,16] The aim of the current study was to systematically review the available evidence on the effects of vaping on periodontal disease/periodontitis.

BACKGROUND

Traditional smoking and periodontal health

A direct causal relationship between smoking exposure and the prevalence and the severity of periodontal disease has been firmly established (American Academy of Periodontology 1996, Grossi *et al*. 1994).[17] According to the National Health and Nutrition Examination Survey III, smokers were four times as likely to have periodontitis as persons who had never smoked after adjusting for age, gender, race/ethnicity, education, and income/poverty ratio. The use of tobacco products, in general, and smoking products, in particular, is the major preventable risk factor for the initiation and progression of periodontal diseases.[5-7] A past indicated that current smokers were nearly 3 times more likely to have severe periodontitis than non-smokers. The detrimental impact of long-term smoking on the periodontal and dentate status of older adults has been clearly demonstrated.[8] The most marked difference between smokers and nonsmokers in probing depths (PDs) or attachment loss (AL) occurs in the maxillary lingual area and mandibular anterior teeth, suggesting a local effect of smoking.[11]

It has also been firmly established that smoking cessation is associated with decreased mortality, lower risk of developing a variety of diseases, and increased life expectancy.[1]

Several cross-sectional investigations have indicated that smokers may present with lower levels of gingival inflammation at a specific level of plaque than non-smokers. This was evidenced using both the gingival index and the dichotomous evaluation of bleeding on probing (BOP).[17] Nair *et al*. followed 27 individuals for 4–6 weeks during a verified successful period of quitting smoking and found bleeding doubled (from 16% to 32%) during this period.[18] Pindborg (1947) was one of the first investigators to study the relationship between smoking and periodontal disease. He determined that tar in the smoke exerted a direct irritating effect on the gingiva giving rise to gingivitis and that nicotine could cause contraction of the capillaries, thus interfering with the nutrition of the gingiva which consequently became less resistant to infection.[19] Smokers have a higher proportion of sites with deeper PDs and clinical attachment loss (CAL) compared with nonsmokers.[17-19] The observed effects have been confirmed in different studies and in different populations after correcting for a variety of potential confounders.[14,17]

Although the direct cause for periodontitis is oral bacterial infection, its progression and severity depend on a number of genetic and environmental factors.[14] Several epidemiological studies in different populations demonstrate a relationship between smoking and periodontal disease.[15,16] Cigarette smoking is arguably the strongest behavioral risk factor for the incidence and progression of periodontitis.[17] It is also important to note that although non-smokers universally respond better to periodontal treatment than do smokers, there is nevertheless substantial evidence of clinical improvement in smokers after treatment, indicating that smoking as a risk factor will compromise rather than prevent tissue healing.[18]

E-cigarettes and vaping devices

In 2006, electronic cigarettes (e-cigarettes) made their debut on the international scene. The term Electronic Nicotine Delivery Systems (ENDS) was introduced by the World Health Organization (WHO) in 2009 to refer to the many kinds of e-cigarettes that include nicotine.[13,14] Adults between the ages of 18 and 24 were found to be the highest users of e-cigarettes and over half of them had never smoked cigarettes. In recent years, vaping has become more common, especially among young adults (18–24 years old) and adolescents (11–17 years old). The introduction of new disposable devices has been largely blamed for this rise in vaping among these age groups. Since they initially hit the market more than 10 years ago, ENDS has gained widespread acceptance as a less dangerous option to traditional cigarette smoking. Similar to smoking when referring to the usage of combustible tobacco cigarettes, the term “vaping” describes the practice of using e-cigarettes.[15,16] Vaping has significantly risen worldwide due to the quick spread of innovative ENDS products and clever marketing strategies, raising substantial public health issues. More than 400 e-cigarette brands and more than 7000 flavor options were offered both in-store and online. Since flavored vaping liquid (or juice) is popular among teenagers, flavored vape goods have been specifically

targeted toward teenage consumers. Because components such as marijuana and concentrated THC can be utilized in all devices that are now on the market, the use of these substances by adolescents has significantly increased. One in three high school kids and one in four middle school students who used e-cigarettes reported utilizing cannabis in their devices, according to a 2018 California research.[17,18]

E-cigarettes are electronic devices that operate through rechargeable batteries in the rechargeable ones and consist of an atomizer or heating element to heat the e-liquid and produce a vapor and consist of a cartridge filled with e-liquid. They feature similar components but differ in size and shape. The various components in the e-cigarette and the products produced after heating are elaborated in Table 1.

Typically, the e-liquid comprises flavorings and humectants, either with or without nicotine. When the atomizer heats the liquid, the aerosol (vapor) creates a sensation like smoking tobacco, but it is said to have no negative consequences. However, it is said that the process of heating can result in the creation of new, potentially dangerous chemicals.

Since the chemist Hon Lik began manufacturing e-cigarettes in China in 2003, the product's design has undergone rapid modifications. E-cigarettes come in four different generations that vary in size, shape, and price.[16,20,26] When e-cigarettes were first released, they looked a lot like regular cigarettes. The second generation, known as cleoatomizers, had a larger detachable tank that could hold e-liquid, a multi-voltage battery, and a detachable filament. The third-generation devices, often known as mods, are identified by modified batteries with variable voltages, wattages, and power capacities. The fourth-generation devices, such as USB flash drives, employ fixed-voltage batteries, which are shown in Figure 1 in a variety of shapes and sizes.[21,22]

OXIDATIVE STRESS AND INFLAMMATORY MARKERS

In a narrative review, we examined the potential effects of heated tobacco products and e-cigarettes on oxidative stress and atherosclerosis. Several studies have shown that e-cigarettes and heated tobacco increase oxidative stress through the activation of enzymes such as NADPH oxidase. One of the primary effects of these products is their pro-thrombotic and pro-atherosclerotic impact on endothelial cells and platelets, which promotes inflammatory processes within the arteries. Furthermore, the chemicals found in e-cigarette liquids may exacerbate inflammation and cause endothelial dysfunction.[23,24]

In vitro studies have evaluated the effects of traditional cigarette smoke and newer smoking products (e-cigarettes and heat-not-burn cigarettes [HNBC]) on endothelial function. Giebe *et al*. reported significant impairment in cell viability and the repair capacity of endothelial damage only with traditional cigarette extracts, with increased oxidative stress.[25,26] On the other hand, HNBC and e-cigarette extracts determined increased monocyte adhesion to endothelial cells and enhanced the expression and synthesis of pro-inflammatory genes and proteins, even if lower compared to traditional cigarettes. Certain e-cigarette flavors also appear to be associated with increased endothelial dysfunction.[27,28] A recent study on aortic endothelial cells showed that low concentrations of vanillin, menthol, cinnamaldehyde, eugenol, and acetylpyrazine induced increased reactive oxygen species (ROS) production, pro-inflammatory mediators, and reduced NO bioavailability.[26]

Recent clinical studies have shown data consistent with *in vitro* and *ex vivo* findings. One study compared the acute effects of traditional tobacco smoke with e-cigarette smoke on endothelial function, oxidative stress, and vitamin E levels in smokers and non-smokers. Both types of smoke were associated with elevated oxidative stress markers, reduced flow-mediated dilation, lower nitric oxide levels, and decreased vitamin E levels, with no statistically significant difference between traditional tobacco and e-cigarettes.[14,16] Vaping generates ROS and elevates cytokines (interleukin [IL] 1β, tumor necrosis factor-alpha [TNF α], IL 6), although to lesser degrees than smoking. A 2025 oxidative stress review highlights shared but distinct pathways between e-cigs and cigarettes. Salivary IL-band TNF-α was increased ENDS than non-smokers (NS).[16,26] IL-6 and IL-8 levels were similar in ENDS users and NS. Metabolite profiling determined that 368 salivary metabolites were expressed differently in ENDS users compared to NS. The levels of salivary prostaglandin

Table 1: The various components in the e-cigarette and the products produced after heating are elaborated

e-liquid contents	Device contents	After heating e-liquid (aerosol) contents	New components produced
Propylene glycol	Wire	Propylene glycol	Propylene oxide
Glycerol	Atomizer	Glycerol	Acrolein
Nicotine	Fibreglass wicks	Nicotine	Acetaldehyde
Flavoring		Flavoring	Formaldehyde
			Acetamide, copper, nickel, silver, silicate particles

e-liquid: Electronic liquid, e-cigarette: Electronic cigarette

E2 and gingival crevicular fluid (GCF) inflammatory mediators were significantly higher in CS compared with NS, EC, and DS. Chronic E-cigs are associated with an adverse effect on periodontal health although to less extent than the effects of traditional cigarettes.[19,20] Plaque index (PI) and levels of IL-1β, IL-10, and IL-1RA were higher among e-cigarette users. E-cigs exert a powerful, detrimental effect on the subgingival ecosystem, altering the immunotolerance of the host. Levels of pro-inflammatory cytokines IL-2, IL-6, granulocyte-macrophage colony-stimulating factor, TNF-α, and INF-γ were highest among CS followed by ES and NS. The level of anti-inflammatory cytokine IL-10 was lowest in CS followed by E-cigs and NS.[21,22]

GENE EXPRESSION AND EPIGENETIC CHANGES

Aerosol generated by ECIGs is composed of various toxic agents. Some are reported to exert an effect at the cellular level similar to that of tobacco smoke, principally increased levels of oxidative stress and inflammation, and to lead to changes in gene expression.[15,17] For this reason, it is essential to determine whether ECIG aerosol exposure can cause DNA damage to pulmonary and oral epithelial cells. Ganapathy *et al*. determined the genotoxicity and mechanisms induced by ECIG aerosol extract in human epithelial normal bronchial cells (Nuli1) and human oral squamous cell carcinoma (UM-SCC-1).[18-20] After 1 h of aerosol exposure in both cell types, oxidative and alkylation DNA lesions were observed. It was also shown that DNA damage was dose-dependent, as more damage was evidenced as exposure to ECIG aerosols increased. These harmful effects may be associated with formaldehyde and the ROS levels in aerosols.[21-23]

The significant mutagenic capacity of toxic compounds in ECIGs has not yet been described, at least to the best of our knowledge. However, conventional smoking has been proven to cause multiple epigenetic alterations. Given that ECIGs contain numerous noxious chemical compounds, they can alter epigenetic mechanisms that regulate gene expression as well.[24,26]

In vitro models reveal that flavored ecig aerosols modulate gene expression in epithelial and periodontal ligament cells, altering stress response and extracellular matrix regulation – mirroring some cigarette smoke effects but often less severe.[27,28]

NICOTINE EFFECTS ON CELL FUNCTION

Nicotine induces various biological effects, such as neoangiogenesis, cell division, and proliferation, and it affects neural and non-neural cells through specific pathways downstream of nicotinic acetylcholine receptors (nAChRs). Specific effects mediated by α7 nAChRs are highlighted.[17,19]

Nicotine impairs fibroblast proliferation, collagen synthesis, and wound repair through vasoconstriction and enzyme induction. These disruptions compromise periodontal healing akin to cigarette exposure.[20,22]

PROPOSED MECHANISMS FOR NEGATIVE PERIODONTAL EFFECTS OF NICOTINE

Vascular alterations
Altered neutrophil function
Decreased immunoglobulin G production
Decreased lymphocyte proliferation
Increased prevalence of periopathogens
Difficulty in eliminating pathogens by mechanical therapy
Altered fibroblast attachment and function
Negative local effects on cytokine and growth factor production

Nicotine, a major component and most pharmacologically active agent in tobacco is likely to be a significant contributing factor for the exacerbation of periodontal diseases. Available literature suggests that nicotine affects gingival blood flow, cytokine production, neutrophil and other immune cell function, connective tissue turnover, which can be the possible mechanisms responsible for the overall effects of tobacco on periodontal tissues. Inclusion of tobacco cessation as a part of periodontal therapy encourages dental professionals to become more active in tobacco cessation counseling. This will have far-reaching positive effects on our patients' oral and general health.[23,24]

MICROBIOLOGICAL EFFECTS

A study showed that e-cig use promotes a unique periodontal microbiome, one that contains distinctive features yet shares similarities with those of both conventional cigarette users and nonsmokers. The duration of e-cig use is a strong driver of subgingival microbiome composition over flavoring additions or nicotine concentration, indicating that basal e-cig components exert specific selection pressures on the SGP microbial community. Indeed, while this longitudinal study of chronic e-cig users demonstrated an increase in α-diversity with ongoing use, a unique e-cig user microbial community was maintained compared to those of conventional smokers and non-smokers.[17,18] Two uniquely dominant taxa in e-cig users, *Fusobacterium* and *Bacteroidales* (G-2), are anaerobic and known to be associated

with periodontitis, with *Bacteroidales* (G-2) more associated than *Porphyromonas gingivalis*. *Fusobacterium* positively correlated with IFN-γ, IL-12p70, and IL-2 and is known to be enriched in periodontitis as an important component of periodontal biofilms.[19,20] The significant enrichment of these organisms provides evidence that e-cig use may promote an SGP community enriched in pathogens but in a uniquely dysbiotic manner compared to chronic conventional cigarette use. A previous study looking at the salivary microbiome and e-cig use found that the phylum *Fusobacteria* is significantly depleted in conventional smokers, with similar relative abundances between e-cig users and nonsmokers, suggesting that habitual use may have habitat-specific effects.[21,22]

Previous results demonstrate that the e-cig user's subgingival microbiome is a unique amalgamation of microbiota, containing similarities to those of both conventional smokers and nonsmokers. Due to many shared features with the conventional smoker's microbiome and considering the widespread promotion of e-cigarettes as a "healthier" alternative to or replacement for conventional cigarettes, our results show that e-cigarette use may promote a healthier SGP microbiome with respect to that of smokers but not compared to that found with never smoking in the first place.[23,24] The uniqueness of the e-cigarette periodontal microbiome indicates a need for further research into this relatively novel microbial consortium, obtained through the adoption of a newly acquired human habit, and how biotic and abiotic components synergistically impact oral health and disease.[21,22,25,26] e-cigarette use altered the oral microbiome in periodontitis patients, enriching members of the *Filifactor*, *Treponema*, and *Fusobacterium* taxa. For patients at the same periodontal disease stage, cigarette smokers (CSs) and e-cigarette smokers shared more similarities in their oral bacterial composition.[27,28] E-cigarette smoking may have a similar potential as cigarette smoking at altering the bacterial composition of saliva over time, leading to an increase in the relative abundance of periodontal disease-associated pathogens such as *Porphyromonas gingivalis* and *Fusobacterium nucleatum*.[27-30]

BIOFILM FORMATION

Shabil *et al*.[31] found that there is a significant difference in PI between e-cigarette users and non-users, with e-cigarette users exhibiting an increased PI. Tattar *et al*.[32] carried out an SRMA and five studies in this review investigated microbiome changes and found that e-cigarette use is associated with alterations in the oral microbiome, with specific microbial shifts tied to oral health risks.

According to Vámos *et al*.,[33] e-cigarette smokers showed significantly higher PIs compared to non-smokers. It was also concluded that most nicotine-containing product users presented worse clinico-radiographic and immunological peri-implant parameters compared to non-smokers. The analysis revealed no significant heterogeneity across the studies. Zięba *et al*.[34] found that E-cigarette use can increase the levels of cariogenic bacteria, such as *Streptococcus mutans* and *Lactobacilli*, in saliva. Nicotine in e-cigarettes is believed to contribute to this by promoting the adhesion of bacteria to the tooth biofilm and stimulating the synthesis of extracellular polysaccharides, which thickens the biofilm. This process may increase the risk of dental caries, as the activity of these bacteria leads to higher levels of lactic acid production. While some studies show a correlation between e-cigarette use and increased bacterial counts, more research is needed to fully understand the extent of e-cigarettes' impact on oral health.[31,32]

Youssef *et al*.[35] found that e-cigarette users had significantly higher PI compared to non smokers. E-cigarettes may have a negative effect on the clinical, radiographic, and pro-inflammatory profile of dental implants. Further studies, including longitudinal investigations in diverse patient populations, are required to confirm these outcomes. The studies showed moderate but non-significant heterogeneity.

A meta-analysis combining data from 6 studies on self-perceived gingivitis found an association between e-cigarette use and self-reported gingivitis. They also noted that flavored e-liquids from e-cigarettes are detrimental to the enamel, similar to what is caused by gelatinous sweets or acidic drinks. E-cigarettes have a potentially detrimental effect on periodontal and peri-implant parameters, and laboratory tests confirmed the presence of carcinogenic and inflammatory biomarkers. Flavored e-liquids may be a caries risk factor.[30-36] Thiem *et al*.[36] found that the PI was significantly higher in CSs compared to e-cigarette users and non-smokers in six studies. One study showed no difference between e-cigarette users and non-smokers, and another study showed increased plaque in e-cigarette users compared to non-smokers. E-cigarette use may be considered a healthier alternative to cigarette smoking in terms of periodontal health; however, harmful effects of e-cigarette use on periodontal health have also been observed.

CLINICAL PERIODONTAL EFFECTS

Gingival inflammation and bleeding

The meta-analysis from 5 studies by Shabil *et al*.[31] compared the BOP between e-cigarette users and non-users across various studies. The studies included were diverse, encompassing various countries and sample sizes. The analysis revealed a significant difference in BOP scores between the groups, with e-cigarette users generally having lower scores. The overall pooled mean difference was −14.233 (95% CI: −20.424–−8.043), indicating a statistically significant lower BOP score among e-cigarette users compared to non-users. The studies exhibited high heterogeneity (I^2 = 99%), suggesting considerable variation in the results across the studies.

Xu *et al*.[37] found that BOP and average PDs similarly increased over time in all three groups, but CAL uniquely increased in e-cigarette smokers. Rates of severe periodontal disease were higher in CSs and e-cigarette users than in non-smokers, but interpretation is confounded by the older age of the CSs.

AlZamil *et al*.[38] found that there was a statistically significant difference in BOP between healthy and periodontitis patients ($P < 0.001$). The mean PI, PD, and CAL were considerably higher in heavy smokers than light smokers and non-smokers ($P < 0.001$). In contrast, the mean GI and BOP were significantly lower in heavy smokers than in light smokers and non-smokers. There was a statistically significant difference in GCF between healthy and periodontitis patients ($P < 0.001$). The mean GCF readings were higher in heavy smokers than light smokers or non-smokers ($P < 0.001$). The present study confirms the influence of smoking on periodontal clinical parameters. Smoking was associated with increased PD, PI, CAL, and GCF readings; however, GI and BOP were decreased in smokers. The number of cigarettes played a key role in the volume of GCF and periodontal clinical parameters.

Ibraheem *et al*.[39] conducted a study to compare the levels of receptor activator of NF-kappa B ligand (RANKL) and osteoprotegerin (OPG) in the GCF of cigarette- and waterpipe-smokers and ENDS-users. Clinical periodontal parameters (PI, BOP, PD, and CAL) were measured; GCF samples were collected from the deepest periodontal pocket of the mandibular right first molar. The GCF volume was determined and levels of RANKL and OPG were determined. Cigarette- and waterpipe smoking and ENDS usage are associated with an increased expression of RANKL and OPG in the GCF showing greater gingival inflammation and BOP.

AL and pocket depth

Javed *et al*.[40] evaluated PD and CAL on radiographs among CSs (group 1) individuals exclusively vaping e-cigarettes (group 2) and never-smokers (NSs) (group 3). PD ≥4 mm was significantly higher in groups 1 and 2 than in group 3. There was no difference in clinical AL among all groups.

From a periodontal perspective, it has been reported that PI, CAL, and PD and marginal bone loss (MBL) are higher in ENDS users than controls (individuals that have never used tobacco in any form).

It is hypothesized that periodontal status is compromised, and whole salivary (WS) IL-15 and IL-18 levels are higher among cigarette-smokers and ENDS-users than never-smokers.

Ali *et al*.[41] conducted case–control study to compare the periodontal status and WS IL-15 and -18 levels among cigarette-smokers, ENDS-users, and controls (never-smokers). Participants were divided into 4 groups as follows: Group-1 – Current cigarette-smokers; Group-2 – ENDS-users; Group-3 – Never-smokers with periodontitis; and Group-4 – Never-smokers without periodontitis. Scores of PI, clinical AL, PD, and number of missing teeth were elevated in groups 1 and 3 than -4. Scores of PI, clinical AL, PD, MBL, and missing teeth were comparable among patients in groups 1, 2, and 3. Levels of IL-15 and IL-18 were elevated in groups 1 and 2 than groups 3 and 4. The levels of IL-15 and -18 were higher in Group-3 than in Group-4 ($P < 0.001$). Clinically, cigarette-smokers and never-smokers demonstrate similar periodontal statuses; however, WS immunoinflammatory biomarkers (IL-15 and -18) are elevated in these individuals than non-smokers.

Bone loss

The meta-analysis comprising eight studies focused on comparing distal MBL between e-cigarette users and non-users. This analysis incorporated studies with a total pooled sample size of 182 e-cigarette users and 173 non-users. The overall mean difference observed was 0.531 (95% CI: −0.565–1.627), indicating a non-statistically significant trend toward greater bone loss in e-cigarette users compared to non-users, though the results varied across studies ($P = 0.342$). The heterogeneity was high ($I^2 = 100\%$).[31]

The meta-analysis, including eight studies, assessed mesial MBL comparing e-cigarette users with non-users.[31] This analysis summarized findings from a total pooled sample size of 182 e-cigarette users and 173 non-users. The overall mean difference for mesial MBL was 0.516 (95% CI: −0.533–1.564), which indicates a non-statistically significant effect ($P = 0.3353$). Notable heterogeneity was observed across the studies ($I^2 = 100\%$).

Mokeem *et al*.[42] conducted a study to compare the radiographic (MBL) periodontal parameters and WS cotinine, IL-1β, and IL-6 levels among cigarette-smokers, waterpipe-smokers, e-cig users, and never-smokers. MBL was measured in digital intraoral radiographs. There was no difference in mesial and distal MBL. In conclusion, clinical and radiographic parameters of periodontal inflammation were poorer in cigarette and waterpipe smokers than e-cig users and never-smokers, and WS cotinine levels were similar in all groups. Whole salivary IL-1β and IL-6 levels were higher in cigarette- and waterpipe-smokers than E-cig users and never-smokers.[41,42]

Contrasting results were found when Javed *et al*.[40] evaluated MBL on digital radiographs among CSs (group 1), individuals exclusively vaping e-cigarettes (group 2), and never-smokers (NSs) (group 3). PD ≥4 mm ($P < 0.01$) was significantly higher in groups 1 and 2 than in group 3. There was no difference in clinical AL among all groups. It has been reported that MBLs are higher in ENDS users than in controls (individuals that have never used tobacco in any form).

IMPACT ON YOUNG POPULATIONS

Worldwide, youth e-cigarette use (vaping) has risen significantly over the past decade. This public health concern has spurred many high-quality studies characterizing country-specific prevalence, risk factors, physical and behavioral health complications, and optimal methods of assessment and counseling for youth vaping. Clinicians remain underexposed to this recent work, limiting translation of evidence into higher-quality patient care.[43-45] This review aims to provide pediatricians and other clinicians working with youth a clinically focused survey of key research findings and considerations based on recent evidence. This narrative review surveys emerging trends in EC use across different countries, reasons for youth vaping, characteristics of vaping materials that promote youth use, associations with combustible cigarette use, relations with cannabis and other illicit substances, physical and behavioral health risks associated with vaping, and methods of assessment, counseling, and intervention for problematic vaping in youth. Since vaping remains a relatively new phenomenon, long-term health consequences remain unknown.[46-49]

Youth uptake of ECs has occurred in the context of youth-targeted marketing, social media promotion of ECs, and peer influences. A study of US high school seniors identified three main motivations to vape: taste and entertainment (63%), experimentation (29%), and to replace CCs (7%). In comparison, a German study found that the vast majority of German youth (aged 14–19) cited curiosity (73.1%) as their reason for EC use, followed by quitting tobacco use (14.9%) and as a complement to tobacco use (7.5%).[50-52] In a systematic review of six studies among young adults in varied settings (i.e., USA, Romania, France, New Zealand, and Saudi Arabia), curiosity and EC use by friends were the primary reasons for EC initiation among non-smokers. Among former and current CC users, ECs were used due to perceptions of harm reduction, to aid smoking cessation, to use in smoking-restricted areas, for lower cost compared to CCs, or for flavoring. Studies in Europe and Taiwan have identified male gender, older age, and parental and peer smoking as risk factors for EC use and dual use of ECs and CCs.[44-46]

Some countries have been slow to regulate EC marketing, leaving youth vulnerable to advertisers. Advertising has consistently been associated with youths' intention to use ECs. A systematic review found that in addition to traditional marketing, often utilizing youth-directed strategies, social media platforms have been widely used for public discussion of ECs in a manner that produces largely positive or neutral EC portrayals.[45-48]

Youth vaping is now a well-studied phenomenon with various physical and behavioral health risks, some of which differ from traditional smoking. Although vaping-specific treatments remain underdeveloped, pediatricians and other youth clinicians can apply the lessons of recent research to counsel youth and their families and prevent long-term complications of vaping-related nicotine addiction.[50-52]

COMPARISON SUMMARY

Parameter	Vaping	Traditional smoking
Inflammatory Markers	Elevated (IL-1β, TNF-α), less than smokers	Strong elevation, high ROS
Microbiome Changes	Dysbiosis with subgingival pathogens	Well-documented dysbiosis
Clinical Signs	Increased plaque and inflammation; mild AL	High PI, PD, AL, BoP, bone loss
Youth Usage	Rapid rise, limited awareness	Declining, better awareness

GAPS AND FUTURE DIRECTIONS

- Long-term longitudinal studies, especially in youth cohorts
- Standardization of clinical endpoints and biochemical verification of usage
- Randomized controlled trials comparing end-point outcomes in smoking cessation versus never-users
- Public health education integrating periodontal risks into anti-vaping messaging.

CONCLUSION

E-cigarettes are not risk-free. They produce measurable oxidative stress, inflammatory responses, and microbiome shifts, increasing risk for periodontal pathology – though generally less severe than traditional cigarettes. Periodontal clinicians should advise both smokers and vapers about these risks, especially among younger demographics, while advocating for more rigorous research. The data suggest a gradient of risk where non-smokers have the lowest risk, e-cigarette users have a moderate risk, and CSs have the highest risk for periodontal health issues.

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REGISTRATION

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