

## E-Cigarettes/Vaping and Oral Health: Effects on Periodontal Tissues, Caries Risk, and the Oral Microbiome

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### Abstract

The number of users of e-cigarettes and vaping devices has been increasing rapidly, especially in adolescents and young adults, raising increasing concern over both the short-term and long-term effect on oral health. The narrative review is a synthesis of the existing evidence on the impacts of vaping on the periodontal tissues, dental caries risk, and the oral microbiome. Existing clinical and experimental literature indicates that e-cigarette aerosol may induce inflammation of the gingiva, reduce the ability to heal periodontally, and be the cause of dysregulated host immune responses due to oxidative stress and pro-inflammatory pathways. New evidence further suggests that vaping can change saliva composition and flow, which could alter buffering capacity and antimicrobial activity and that some flavored e-liquids and nicotine-containing aerosols can alter enamel demineralization processes and bacterial adhesion and thus is plausibly likely to lead to caries susceptibility. Microbiome-centered studies show

that vaping can be linked with quantifiable changes in the structure of oral microbial population such as the enrichment of opportunistic and inflammation-related taxa, which may predispose to the development of periodontal diseases and irritation of oral mucosal tissues. Nevertheless, the evidence base is heterogenous, and the limitations are small samples, imprecise measurement of exposure, short follow-up, and dual use of conventional tobacco confounding. Altogether, existing data confirms that vaping is biologically plausible in its association with unfavorable oral health effects, specifically, disrupted microbiome and inflammatory periodontal reactions. Standardized exposure definitions and outcomes of clinical importance require further longitudinal research to elucidate dose-response relationships, take out nicotine and flavoring agents, and provide guidance on dental public health and chairside counseling.

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## Introduction

E-cigarettes and vaping products have quickly become one of the most popular methods of nicotine delivery systems, and they gain popularity among different age categories, and adolescents and young adults, in particular, use them significantly (Gholap et al., 2020). Sold in most locations as less harmful alternatives to combustible cigarettes, the consumer generates aerosols by heating e-liquids which normally include different ratios of nicotine, propylene glycol (PG) and vegetable glycerin (VG) as well as a wide variety of flavoring chemicals (Li et al., 2020). Although the respiratory and cardiovascular outcomes of vaping have received significant scientific and regulatory concern, its effects on the oral cavity (Matsuoka et al., 2025). Two of the most prevalent and clinically important oral diseases in the world, periodontal diseases and dental , which is anatomically the initial exposure area, are now being viewed as an important area of concern (Rose et al., 2023). The unique vulnerability of the oral environment, due to direct impact of vaping aerosols on teeth, gingival tissues, mucosal surfaces, and saliva, and the potential effect of vaping aerosols on the oral microbiome underlying health or disease, makes the oral environment particularly vulnerable (Paweł Szumilas et al., 2022).

The outcomes of oral health are not local phenomena but are the results of the interactions between host tissues, saliva, immune and inflammatory responses and microbial communities caries, are largely dependent on modifiable behaviors, such as the use of tobacco and nicotine (Giacaman et al., 2022). Traditional smoking has traditionally been recognized to be a significant risk factor in the development of periodontal breakdown, poor wound healing, and distorted microbial colonization (Jiang et al., 2020). The growing replacement of the conventional cigarettes with the e-cigarettes poses an important clinical and scientific question: does the vape truly lead to the meaningful reduction of the oral harm, or does it pose its own unique threats through novel mechanisms (Ebersole et al., 2020). There is some developing evidence that vaping is not biologically neutral. Its aerosols may include irritants, reactive substances (including carbonyls formed in the heating process), PM and trace metals (resulting in the use of device parts). Moreover, nicotine per se can have an impact on the blood flow of the gums, the activity of the immune cells, and inflammatory messages (Ebersole et al., 2020). Exposure to these may cause clinically relevant changes in the oral tissue and the microbial dysbiosis. profile differs from combustible tobacco (Al Bataineh et al., 2020).

In comparison with the majority of systemic exposures where oral cavity is indirectly exposed, vaping implies repetitive direct contact of the oral structures with the heated aerosols (Paweł Szumilas et al., 2022). All the puffs include the chemicals that can be dissolved in the saliva, placed on the tooth surfaces and the restorative drugs and come in contact with the gingival and mucosal tissues (P Szumilas et al., 2022). The oral cavity is a dynamic ecosystem as well: the saliva frequently moisturizes the tissues, the acid is neutralized, the minerals are remineralized, antimicrobial agents are present (e.g., enzymes, immunoglobulins, peptides) (Can & Güvenç, 2025). Any alteration of any of the combinations of saliva volume, pH, buffering capacity or antimicrobial efficacy may trigger a tip towards disease (Birkhed & Heintze, 2021). Similarly, the periodontal tissues need the preservation and restoration of a balanced immune system and adequacy of blood supply. Repeated minor insults include thermal, dehydration, chemical irritant as well as oxidative stress, which may have cumulative effects on integrity of gingival epithelium, burden of inflammation and susceptibility to bacterial challenge (Knight et al., 2016).

Hundreds of microbes are present in the mouth and are arranged in complex biofilms on the mucosal surfaces as well as on the teeth (Mosaddad et al., 2019). These microbial communities are at a homeostatic state in health resulting in normal immune training and homeostasis. The pathophysiology of disease is altered towards the dysbiosis or enrichment of acidogenic and aciduric bacteria in caries or rising prevalence of inflammatory-related organisms in periodontitis (Moussa et al., 2022).

It is important to note that dysbiosis does not only mean the increase of the number of bacteria, but it suggests the shifts in the functions of the microorganisms and the processes of their interactions and interrelation with their hosts. As vaping aerosol is capable of regulating the local oxygen tension, nutrient, salivary composition and epithelial responses it is possible that they can change microbial composition and behavior-modulating caries and periodontal disease pathways (Levy et al., 2017).

Periodontal health requires a fine balance between microbial challenge and host response. Plaque deposition triggers gingivitis, although the development of periodontitis depends on host vulnerability, inflammatory control and environmental risk factors (Di Stefano et al., 2022). Older methods of smoking cigarettes are linked to more severe periodontal pockets, more attachment loss, and worse treatment outcome, in part because of a compromised neutrophil activity, distorted cytokine response, and decreased vascularity (Apatzidou, 2022).

Nicotine, which is an active ingredient of cigarettes, can potentially lead to periodontal risk through a mechanistic pathway, involving alterations in fibroblast activity, collagen metabolism and release of inflammatory mediators (Sundar et al., 2016). In addition to nicotine, the aerosol of the PG and VG may lead to mucosal drying and irritation, which may lead to mucosal barrier impairment (Kim et al., 2022). The reactive carbonyl compounds can be produced by heating, and these compounds can result in oxidative stress. In its turn, oxidative stress has been taken as a known contributor to periodontal tissue damage, which enhances inflammatory signals and benefits extracellular matrix destruction (Mano, 2012). Clinically, these processes may be expressed in the form of heightened inflammation of the gingivae, changes in the bleeding pattern, inability to heal the periodontal treatment, and possibly increased vulnerability to periodontal breakdown during the course of time (Herrera et al., 2014). Although the evidence base remains immature, the overlap of viable biological pathways and early clinical findings is causing periodontal effects to be a priority in oral health studies on vaping.

### **1.1 Caries risk: beyond traditional sugar exposure**

Dental caries essentially is a biofilm-mediated/sugar-induced disease wherein the acid synthesis by bacteria causes enamel demineralization (Liu et al., 2025). Nonetheless, diet is not a determinant of caries risk, but is influenced by saliva, exposure to fluoride, oral hygiene, microbial ecology and oral environmental influencing behaviors. Vaping can cross caries pathways in various ways (Gaur & Agnihotri, 2023). Recurrent aerosol exposure can cause a change in the composition and volume of salivary flow, which can decrease the buffering capacity and the protective qualities of saliva (Birkhed & Heintze, 2021). Some of the flavoring chemicals and additives can react with the formation of biofilms or adhesion of bacteria (Shi & Zhu, 2009). The alterations in the microbiome might mediate acid-producing bacteria or the increase in metabolic energy of the existing cariogenic bacteria (Ilhan et al., 2017). Vaping habits can be co-located with lifestyle habits that indirectly heighten caries risk, including snacking habits, poor oral care or co-use of other drugs (Iacob et al., 2024).

The development of caries is cumulative. Even small changes towards lower pH levels, less salivary protection or a more cariogenic biofilm, provided over time, can predispose lesionogenesis and development. This is why the assessment of vaping in the context of caries risk must not rely only on the assessment of direct chemical impact but also indirect ecological and behavioral factors. These involve the investigation of the role of vaping in the symptoms of xerostomia, the alteration of the frequency of exposure to acidic environments, or the ability of a biofilm to promote demineralization (Bardow & Vissink, 2015).

The new developments in the sequencing platforms have allowed to further investigate the ecology of the mouth, beyond culture-based identification, and community-level characterization. This is especially pertinent to vaping since most of

its possible outcomes can be associated with the ecologic effect: changing the environment that microbes exist in and not necessarily killing or merely adding certain organisms. There is a growing evidence of vaping possibly being linked to alterations in the community structure and activity of microbes. Examples of such changes are changes in the abundance of taxa associated with inflammation, the diversity pattern, or changes in the expression of genes that underlie stress-tolerance, biofilm formation or metabolic activity (Merecz-Sadowska et al., 2020). Both periodontal disease and caries have downstream effects of dysbiosis. Flora In periodontitis, inflammatory states facilitate the growth of organisms that grow in inflamed conditions, and this leads to a self-perpetuating cycle of inflammation promoting dysbiosis and dysbiosis promoting inflammation. Repeated acid stress in caries causes the selection of acid-tolerant bacteria and amplifies the production and resistance of the biofilm to acidic environments. Provided that vaping creates an environment where one or more of inflammatory or acidogenic oral microbial phenotypes proliferate: either by changing saliva, by oxidative stress, or by compromising epithelial barriers, it may become an upstream determinant of the risk of oral disease. Notably, the oral microbiome is linked to a host immune system; alterations in microbial patterns have the potential to affect cytokine signaling, epithelial responses, and tissue remodeling (Baima et al., 2025). Clinically, dentists and hygienists are getting more and more patients who either only vape, use both vaping products and cigarettes (dual use), or change products with time. This complication brings about the difficulties of risk profiling and counseling. Other patients will view vaping as harmless or they may not reveal use unless requested to do so, especially when they do not consider themselves to be smokers (Banerjee). Thus, dental history-taking should be modified with specific questions on the use of e-cigarettes, the type of device used, the level of nicotine, number of times per, and the use of flavored products. The education of the patients must be informed by references to evidence, balanced, and patient-centered--that something is less harmful than smoking does not mean that it is safe, particularly of the oral tissues in direct contact (Loewen & Relich, 2019). On a population level, the question of oral health in vaping overlaps with the question of youth nicotine initiation, the marketing of flavored products, and health equity. Teens who start with vaping can become addicted to nicotine, which can affect health behaviors in the long run. In the meantime, differences in technology of devices, composition of e-liquid and patterns of use make exposure assessment and interpretation of research more difficult. Preventive messaging, early identifying tissue changes, and incorporation of vaping factors into caries and periodontal prevention interventions are part of the role of the oral health community (Scherer et al., 2024).

## **1.2 Rationale, scope, and objectives**

Regardless of increased attention, the evidence on the topic of vaping and oral health is still uneven. The studies mentioned above differ in their design, with in vitro experiment, animal model, cross-sectional clinical research, as well as small longitudinal observations. Causal inference may be obscured due to confounding factors, including previous history of smoking, dual use, alcohol consumption, oral hygiene, socioeconomic status and diet. Moreover, the products are highly differentiated: the devices vary in terms of heating temperature, coil type, aerosol particle size and efficiency of nicotine delivery; e-liquids will vary in terms of PG/VG ratios, in terms of nicotine salts versus free-base nicotine and thousands of flavouring compounds. These variations are important as an exogenous effect of exposure could be affected by the chemical profile and intensity of use. Considering this complexity, a narrative review would prove useful in order to combine mechanistic knowledge with clinical observations, define recurrent trends,

and pinpoint gaps in knowledge that would need further investigation. In this regard, this review will seek to:

The present narrative review of the existing evidence on the impact of the use of e-cigarettes and vaping on periodontal tissue as a type of non-smoking tissue will summarise the reported tissue and inflammatory responses on the tissue. It also addresses realistic directions of action that include saliva, biofilm behaviour, and enamel demineralisation, and analyses emerging evidence on the vaping-induced alterations in the oral microbiome and their possible implications in risk of oral disease.

## **2. Overview of E-Cigarettes/Vaping Products and Oral Exposure Pathways**

E-cigarettes (also referred to as electronic nicotine delivery systems, ENDS) are devices designed to produce an inhalable aerosol by heating an e-liquid. Although products vary widely, most devices share common components: a power source (battery), a heating element (coil), an e-liquid reservoir (pod/tank), and a mouthpiece (Brown & Cheng, 2014; DeVito & Krishnan-Sarin, 2018; Ward et al., 2020). From an oral health perspective, these devices are clinically relevant because the oral cavity is the *first anatomical site* exposed to the aerosol—receiving repeated contact with heated vapors, dissolved chemicals, and fine particulates that can interact with saliva, dental biofilms, and mucosal and gingival tissues (Ward et al., 2020).

### **2.1 Device types and their relevance to exposure**

The e-cigarette technology has developed at a tremendous pace and has created various types of different devices, which differ in terms of the aerosol production, the effectiveness of nicotine delivery, and the manner of puffing by the user. The cigalike-type devices of the first generation that are similar to traditional cigarettes usually have a lower aerosol output. Vape pens are refillable or cartridge-based devices and produce a medium level of aerosol and give the user more control. More sophisticated tank or mod system are larger and can be customizable with higher power output which causes significantly higher aerosol generation. On the contrary, pod-based are miniature-sized units that utilize pre-filled, or refillable pods and are typically related to nicotine salt formulations, allowing a more efficient delivery of nicotine. These differences are also clinically important as the devices with the higher power output and larger aerosol volume can result in the enhanced chemical exposure to the oral tissues. Also, variations in nicotine formulation and frequency of puffing by its user may also add to local effects of nicotine by increasing or reducing the level and duration of exposure (Eaton et al., 2018a).

### **2.2 E-liquid composition: nicotine, carriers, and flavoring agents**

The vast majority of e-liquids contain three primary ingredients, including nicotine (with or without), carrier solvents, including propylene glycol (PG) and vegetable glycerin (VG), and flavoring agents, each of which can have a different biological impact on oral health. Nicotine is a pharmacologically active compound that is known to have an impact on vascular tone, inflammatory signaling, and immune response (DeVito & Krishnan-Sarin, 2018). According to tobacco research evidences, exposure to nicotine in periodontal tissues is linked to disturbed host response and dynamic healing. Even though e-cigarettes could lead to a lower exposure to some of the combustion-related toxicants relative to conventional cigarettes, nicotine via vaping can still play a role in biological effects referring to gingival inflammation and periodontal wound healing (Bonilla et al., 2025). Propylene glycol and vegetable glycerin are also humectants and carriers of aerosols and are the ones that give the feeling of the throat and aerosol dispensal visible results. Oral-health wise, the regular (repeated) inhalation of the PG and VG has been linked with oral dryness, and alternations in the salivary properties, which could influence the plaque formation, pH-buffering ability, and general mucosal comfort. Additives and flavoring agents are

especially an issue in that they differ in chemical composition and strength. Some flavoring compounds have been shown to be irritant potential or capable of effecting on microbial adhesion and biofilm behavior in the laboratory. Furthermore, flavored e-liquids could alter user behavior because of their pleasantness and enjoyment, which will increase the frequency of vaping, which in turn will increase and extend exposure of oral tissues during the day (Mangulkar, 2020).

**2.3 Aerosol characteristics and oral deposition**

Vaping produces an aerosol containing ultrafine particles, dissolved chemicals, and heat-generated byproducts. When this aerosol enters the mouth, it can deposit on tooth surfaces and restorations where it interacts with the acquired pellicle and dental plaque, dissolve into saliva and potentially alter salivary pH, viscosity, buffering capacity, and antimicrobial function, and directly contact oral mucosa and gingival tissues, which may influence epithelial barrier integrity and trigger local inflammatory responses. Because many users vape repeatedly throughout the day, these exposures occur in frequent cycles, meaning that even small changes in dryness, inflammatory tone, and microbiome balance may accumulate over time and contribute to clinically relevant oral effects (Eaton et al., 2018b; Kaisar, 2018).

**2.4 Patterns of use and confounding exposures**

Clinical interpretation of vaping-related oral outcomes is complicated by real-world patterns of use, because “vapers” are not a uniform group. Many individuals vape exclusively without cigarette smoking, others are former smokers who have switched to vaping, and a substantial proportion are dual users who both vape and smoke combustible cigarettes. Dual use is particularly important because conventional smoking is a well-established risk factor for periodontal disease and can strongly confound findings attributed to vaping. Moreover, oral health outcomes in these populations may also be influenced by oral hygiene behaviors, dietary habits (especially sugar frequency), alcohol use, socioeconomic status, and access to routine dental care. For these reasons, accurate exposure classification and detailed documentation are essential to correctly evaluate and interpret the oral health effects of vaping (Chen, 2024; Hampsher-Monk et al., 2024).

**2.5 Conceptual framework linking vaping to oral health outcomes**

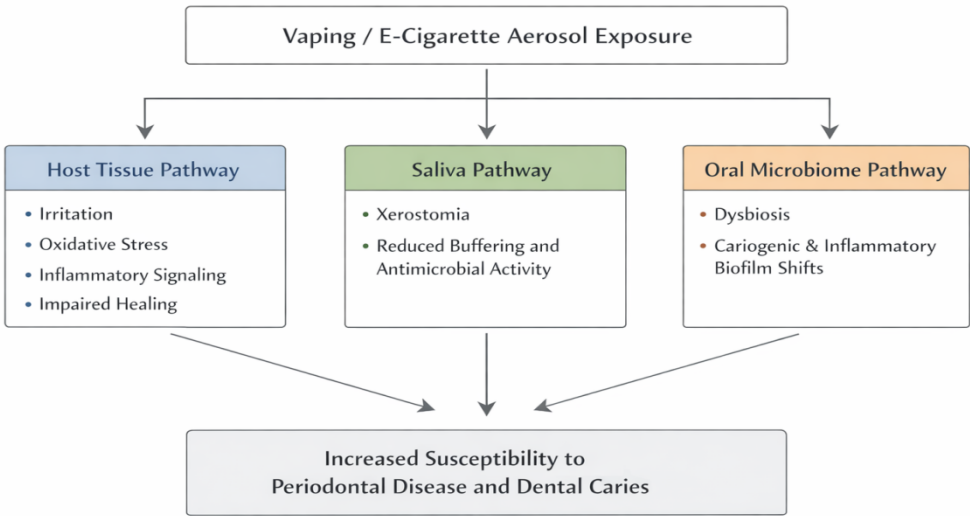


figure 1: Conceptual framework linking vaping to oral health outcomes

### **3. Effects of E-Cigarettes/Vaping on Periodontal Tissues**

Periodontal tissues (gingiva, periodontal ligament, cementum and alveolar bone) are healthy as the immunity is maintained and balance between the host and the microbe is maintained. Perturbation Periodontal disease starts with inflammation of the gingiva caused by plaque, and the disease moves on to loss of attachment, which depends on the presence of systemic and behavioral risk factors, such as exposure to nicotine. Though vaping and cigarette smoking differ on the basis of chemical profile, the immediate and recurrent contact of the gingiva to the e-cigarette aerosol increases the clinically significant issues on inflammation, vascular and immune recording, as well as tissue healing (Hathaway-Schrader & Novince, 2021).

#### **3.1 Clinical periodontal outcomes reported in the literature**

The clinical research of the effects of vaping on the oral and periodontal health regularly employs the conventional periodontal parameters to determine the health and disease condition of tissues (Thiem et al., 2023). They usually comprised of indicators of plaque accumulation and oral health (including the plaque index or PI) and signs of gingival inflammation (including the gingival index or GI) and bleeding on probing (BOP) (Chaves et al., 1993). Probing pocket depth (PPD), clinical attachment level (CAL), and the existence or severity of gingival recession are structural periodontal outcomes that can be evaluated using patient-reported symptoms of oral irritation or dryness (Carvalho et al., 2023). A common finding, in the observational studies, is that vaping is linked to the evidence of gingival inflammation in some groups, but the results do not apply to all studies. This heterogeneity is probably caused by the differences in the study design, exposure definition (i.e., exclusive vaping and dual use with conventional cigarettes), length and intensity of vaping, and baseline oral hygiene habits (Patel et al., 2023).

#### **3.2 Nicotine-related periodontal effects: vascular and healing implications**

Nicotine has long been associated with altered periodontal outcomes in tobacco research, and several nicotine-mediated mechanisms are considered relevant to periodontal tissues. Nicotine can induce changes in microvascular dynamics, potentially affecting tissue perfusion, oxygenation, and healing capacity within the gingiva (Silva, 2021). It may also modulate immune cell behavior, particularly the function of neutrophils and macrophages, which play a central role in host defense and inflammatory regulation in periodontal disease. In addition, nicotine has been shown to influence fibroblast activity and collagen metabolism, processes that are critical for connective tissue maintenance and repair (Centner et al., 2020). Nicotine exposure can further affect the production of inflammatory mediators, thereby altering the balance between tissue destruction and repair (Zhang et al., 2022).

#### **3.4 Periodontal healing and response to therapy: why it matters clinically**

Even if vaping does not result in the same magnitude of periodontal damage as combustible cigarette smoking, an important clinical consideration is whether vaping influences periodontal treatment outcomes (Apatzidou, 2022). Specifically, concerns have been raised regarding its potential effects on the resolution of gingivitis following improvements in oral hygiene, the response to non-surgical therapy such as scaling and root planing, post-treatment inflammation and tissue repair, and healing outcomes after periodontal or regenerative surgical procedures. If vaping contributes to persistent inflammatory signaling or increased oxidative stress, it may compromise the predictability and consistency of periodontal healing. These potential effects carry practical implications for clinical decision-making, including patient counseling, the selection of maintenance intervals, and the need for closer follow-up, particularly in individuals undergoing active periodontal therapy or implant-related interventions (Chaffee et al., 2021).

#### **4. Effects of E-Cigarettes/Vaping on Caries Risk**

Dental caries refers to a chronic biofilm-controlled condition that is occasioned by recurrent acid challenges that result in net loss of enamel and dentine (Timm Maske, 2018). Despite sugar exposure being a key determinant, other protective and risk modifying factors have a strong influence on caries risk, such as salivary flow and buffering capacity, exposure to fluoride, oral hygiene, and oral microbial ecology. The possible indirect pathways, including xerostomia and saliva coverage, biofilm-based pathways (e.g., changes in bacterial adhesion and plaque retention), and chemical pathways (e.g., flavoring compounds and aerosols-induced alterations in local pH and oral environment) are all potential mechanisms by which e-cigarettes/vaping can impact caries risk. The existing evidence is still developing and somewhat conflicting, but there is growing biological plausibility of the argument that vaping might be a risk factor in some users- particularly when used many times in a day and also with other risk factors present (Lagerlöf, 1994).

##### **4.1 Saliva as a primary protective factor in caries prevention**

Saliva plays a central protective role in preventing dental caries by buffering acids produced by plaque bacteria, supplying calcium and phosphate ions necessary for enamel remineralization (García-Godoy & Hicks, 2008), enhancing the activity of fluoride, mechanically clearing food debris and microorganisms, and delivering antimicrobial components that help regulate biofilm composition. Any behavior that reduces salivary flow, alters salivary composition, or disrupts salivary pH can shift the oral environment toward conditions that favor demineralization and caries progression. Vaping has commonly been associated with sensations of dry mouth in some users, and repeated inhalation of aerosol carriers may contribute to oral dryness by promoting dehydration of mucosal surfaces or altering the subjective perception of dryness. From a caries biology perspective, even modest but persistent reductions in salivary protective function can increase plaque retention, prolong acid exposure following meals, and diminish the capacity of enamel to recover through natural remineralization processes (Hicks et al., 2004).

##### **4.2 Vaping-related xerostomia and its caries implications**

Xerostomia as the perception of having a dry mouth is of clinical interest due to its tendency to correlate with increased levels of plaque retention, mucosal irritation, changes in taste perception, and a shift in eating habits, such as becoming more fond of sugary foods or drinks (Cassolato & Turnbull, 2003). All these lead to a higher risk of dental caries especially cervical and root caries in vulnerable populations. Vaping could contribute to xerostomia in a number of linked mechanisms, such as the local drying potential of inhaled aerosol and the high frequency of the behavior in most vape users leading to repeated dry cycles of the body, and nicotine-based effects that could contribute to the experience of oral dryness in a proportion of users. In the clinical setting, vapers with a high frequency and complain of dry mouth symptoms can engage in compensatory strategies, including more intake of sweetened drinks, snacking frequently, or use of flavored chewing products. Such behaviors may also increase the risks of caries by raising the regularity of sugar exposure and raising durations of acidic distress in the mouth (Han et al., 2015).

##### **4.3 Salivary pH and buffering capacity: why small shifts matter**

The development of caries is time-dependent in the number of acid attacks below the critical pH level that causes enamel demineralization. The buffering mechanism of saliva, which is mostly reliant on bicarbonate, assists in replenishing the pH level to neutral following acidic exposures. Should vaping have a role in slowing or changing buffering or salivary flow the mouth could spend more time in an acid environment so that net mineral loss is higher. Although vaping may not directly acidify saliva in a lasting effects, it can lead to a weaker oral ecosystem in which the recovery of the

oral saliva pH following the intake of dietary sugar is slower. It is especially vital in patients at the borderline risk of caries (i.e., inadequate plaque care, inadequate exposure to fluoride, orthodontic devices, or high rate of carbohydrate consumption) (Ilie et al., 2012).

#### **4.4 Plaque retention and biofilm behavior in vapers**

Dental caries is essentially a disease of biofilm and anything that elevates biofilm thickness or alters the microbial metabolism to greater acid production may hasten the process of enamel demineralization. Vaping can affect the accumulation of the plaque and biofilm behavior in a number of interconnected mechanisms. The vaping-related dry mouth may decrease the natural oral cleansing property of saliva, causing more plaque to stick on the surfaces of the teeth. The induced alteration of mucosal and tooth surface condition can also have an impact on acquired pellicle formation and bacterial adhesion and thus alter early biofilm formation. Moreover, oral hygiene habits may vary across vaping groups, which includes behavioral confounding which could indirectly impact on plaque levels. Flavor preferences and palatability can also amplify the occurrence and extended duration of vaping that is experiencing prolonged exposure of oral tissues and biofilms to aerosol components. Ecologically, the recurring exposure to aerosol constituents can cause a change in the oral milieu that would affect the microbial "fitness" possibly choosing some organisms more adapted to environmental stressors, changed oxygen gradients, and changing nutrient supply. Assuming that such changes are biased towards the acidogenic and acid-tolerant communities of bacteria, the general vulnerability to dental caries can be higher (Anil et al., 2022; Mosaddad et al., 2019).

#### **4.5 Flavoring agents and “sweet” e-liquids: potential relevance to caries**

Another notable difference between vaping and traditional cigarette smoking is the popularity of flavoured products, such as fruit-flavoured, dessert-flavoured, and candy-flavoured flavours (Anil et al., 2022). Flavoring has been a cause of concern both behaviorally and biologically as far as oral health is concerned. Sweet and palatable tastes, behaviorally, can prompt more often vaping, commonly referred to as grazing behavior, that results in more cycles of exposure per day and potentially supports diets that have an elevated frequency of sugar. Under experimentation, some flavoring compounds have been shown to affect the adhesion of bacteria, biofilm formation, or the behavior of epithelial cells biologically and may affect the oral environment. Even though the sweet taste is not necessarily a pointer of the presence of fermentable sugars, the general trend of repeated exposure, flavor-oriented application, and potential oral ecology changes is also clinically significant. Practically, clinicians tend to observe clustered behaviors, including vaping and regular drinking of sweetened drinks, and irregular oral cleansing, which in combination can synergistically elevate the possibility of caries on the teeth (Anil et al., 2022).

#### **4.6 Enamel demineralization and remineralization balance**

Dental caries occurs when the demineralization and remineralization balance becomes disturbed during the course of time towards mineral loss. Vaping cannot be considered a direct demineralizing factor, on the contrary, it can affect this balance by impairing a number of protective processes in the oral environment. The possible directions are the loss in salivary remineralization mediated by salivary flow or mineral supply, the extension of the duration of acidic environment after meals as a consequence of slower buffering capacity, the growth of plaque layers and consequent increase in biofilm acidogenicity, and lowered efficacy of fluoride in cases when the plaque layers are thick or as a result of a frequent and prolonged pH fluctuation (Anil et al., 2022; Fejerskov & Larsen, 2015).

#### **4.7 Clinical evidence: what outcomes are typically measured**

Clinical trials of the connection between vaping and dental caries risk usually measure established caries and oral health outcome indicators, such as DMFT and DMFS indices, representing decayed, missing, and restored teeth or surfaces, or the appearance of visible caries lesions or a clinical history of restorations (Gatrelnada, 2021). Other tests might involve including plaque indices, salivary flow rate measurements, subjective xerostomia reports, and/or salivary pH or buffering capacity. These findings are however difficult to interpret due to the overwhelming influence of cumulative and long-term behaviors on dental caries. The cross-sectional studies can only establish a relationship but not a cause. In addition, most studies have difficulty separating the independent impact of vaping and key confounding variables, such as previous or current cigarette smoking, nutrition patterns and frequency of sugar consumption, fluoride exposure and oral health behavior, and more general socioeconomic variables and access to dental services (Saraf, 2018).

#### **5. Effects of E-Cigarettes/Vaping on the Oral Microbiome**

Oral microbiome is a complex community of bacteria, fungi and viruses that inhabit specific niches in the mouth, including saliva, the tongue dorsum, supragingival plaque and subgingival plaque. These communities help in maintaining ecological stability in the health environment, as well as normal homeostasis of immunity. Microbial dysbiosis-community composition change and function change- may facilitate cariogenic (acidogenic/aciduric) or periodontal (enrichment of anaerobe-related inflammation) dysbiosis. Since vaping presents recurrent exposures to heated aerosols and chemical components personally into the mouth, it has a high chance of affecting microbial ecology by modifying the local environment (e.g., drying, nutrient availability alteration, oxidative stress, and epithelial reactions). Recent findings are increasingly beginning to support the notion that the use of e-cigarettes is linked to quantifiable changes in the oral microbial profiles, however, the direction and the extent of change depends on the site of sampling, the characteristics of the user, and the pattern of exposures (Sedghi et al., 2021).

##### **5.1 Why vaping can plausibly reshape oral microbial ecology**

Vaping can have a variety of effects on the oral microbial communities in many interconnected ways which modify the local oral ecosystem. One of these is microenvironmental alterations associated with saliva and surface conditions because propylene glycol and vegetable glycerin, the dominant carrier solvents in most e-liquids, has the potential to cause oral dryness and lower salivary protection. Reduced salivary clearance and buffering ability can predispose to retention of the plaque and to the establishment of an environment favoring microbial dysbiosis, especially in people who smoke their vapes regularly (Ganesan et al., 2020). The second mechanism is altering the host response, which is the inflammatory signaling and the epithelial barrier activity. The experimental evidence indicates that exposure to e-cigarette aerosols could modulate the cytokine responses in oral cell which might result in the shift of the host-microbe relationship to the inflammation-related microbiome. A third route is associated with more immediate actions on microbes and biofilms because aerosol exposure can impact microbial growth, adhesion, and the structure of biofilms. It has been observed in the laboratory that such exposure may suppress some commensal organisms and promote cariogenic characteristics, such as an increase in biofilm formation and adhesion, in acidogenic microbes, such as *Streptococcus mutans*. Notably, such mechanisms do not make vaping as detrimental as traditional cigarette smoking to be clinically significant, even small ecological changes, repeated several times per day, can favour microbial patterns that predispose to gingival inflammation and enamel demineralization (Bakerywala et al., 2024).

## **5.2 What human studies show: saliva and subgingival microbiome differences**

Comparative studies between e-cigarette users and non-users in vivo have found relationships between vaping exposure and changes in microbial community composition, as well as found associations between particular microbiome phenotypes and inflammatory marker (Walsh, 2024). Particular consideration is given to subgingival plaque as a periodontal risk factor because it is the most immediate microbial niche that deals with gingival inflammation and periodontitis development. Recent studies have thus been interested in the effect of vaping habits on the subgingival microbiome and its metabolic nature, with the emphasis being on the intensity, frequency, and habits of use instead of whether people are users or non-users. It is interesting to note that comparative studies tend to propose that the microbial profiles of vaping are intermediate between never-smokers and traditional cigarette smokers, which can support the idea of an intermediate ecological state instead of the microbiome-specifically-smoker type (Manoil & Belibasakis, 2025).

## **6 Key limitations in the current microbiome evidence**

Although the research interest has increased, there are a number of major limitations that limit the capacity to come up with strong causal conclusions concerning the impact of vaping on the oral microbiome and periodontal health. Exposure misclassification is widespread with numerous studies relying on self-reported vaping status with no standardized measure of the duration of vaping, frequency of use, nicotine level, or device power assessments. Dual use and a history of smoking are further complications to interpretation, as traditional cigarette smoking is a potent modifier of head microbiome and periodontal risk, and it is hard to believe that changes can be caused only by vaping. The research on the topic that is available is mostly based on cross-sectional design and can give observational snapshots only, which do not allow to get directionality or to understand whether vaping can cause dysbiosis or whether microbial profiles are caused by pre-existing behaviors related to vaping. Also, sampling site may lead to different results because saliva, supragingival and subgingival plaque are different microbial ecosystems, which are dependent on the time of collection and oral health status. The variability of products is also a problem since variations in the composition of e-liquids and flavoring agents can result in different microbial pressures, restricting generalizability.

## **7. Future research priorities**

To better define the true impact of vaping on the oral microbiome and its clinical implications, future research should adopt more rigorous and standardized approaches. Priority should be given to clearly defined exposure metrics, including vaping frequency, duration, nicotine formulation, and device type or power, to reduce misclassification. Studies should also distinguish exclusive e-cigarette use from dual use and prior smoking to allow clearer attribution of observed effects. Longitudinal designs are needed to link microbiome changes with clinically meaningful outcomes, such as bleeding on probing, changes in probing depth or clinical attachment level, and caries incidence. In addition, site-specific sampling of saliva and dental plaque using consistent and reproducible protocols will improve comparability across studies. Finally, integrating multi-omics approaches, such as metagenomics and metabolomics, may help connect microbial composition with functional activity and host inflammatory pathways, providing a more comprehensive understanding of vaping-related oral health effects.

## **8. Research Gaps and Future Directions**

Although the evidence base on vaping and oral health has been growing in importance, it still contains significant gaps that preclude certainty and complicate comparison between studies. Standardized exposure definitions with the clear differentiation between exclusive vaping and dual use and former smoking and reporting of

frequency, duration, nicotine formulation, device type, and flavor categories are one of the key needs. Until there is a consistent exposure reporting, two studies can both describe one thing, which in the real world is a very different exposure.

The longitudinal research is also required since periodontal disease progression and caries development are time-related processes. Associations can be determined but the directionality or dose-response can not be dependable in short-term or cross-sectional studies. The future work needs to monitor the changes in the clinically meaningful outcomes in the long term, including the alterations in the bleeding on probing and the level of attachment, the response to periodontal therapy, the appearance of new lesions in caries, and the maintenance of changes in salivary functions. Research isolating the effects of nicotine, as opposed to the effects of solvents and flavours, would also reinforce causal inferences, as there is a variety of product formulations and usage behaviors.

In the microbiome literature, there is a pressing requirement to go beyond describing the change in which organisms occur to functional implications, such as metabolic activity in the acid production, biofilm stability, and inflammatory signaling. The sampling (saliva, supragingival plaque, subgingival plaque) needs to be standardized since each niche represents a different ecosystem, and the mixed approach may actually blur the effect of a particular site. Lastly, the research always ought to adjust the key confounders of oral health, frequency of sugar, exposure to fluoride, socioeconomic status, and dental attendance, as these have a strong impact on caries and periodontal outcomes and can cluster together with vaping habits.

## 9. Conclusion

Vaping products and e-cigarettes are an exposure with a fast development of relevance in oral health since the oral cavity is the primary point of contact with aerosols. The existing data confirm that biologically plausible mechanisms exist in which vaping can impact oral health, specifically by facilitating gingivally inflammatory reactions, undermining salivary protective measures by causing alterations in dryness, and playing a role in changes in oral biofilm ecology that can change periodontal stability and caries risk. Although the ratio of risk to combustible tobacco is still a significant question of concern, available evidence does not substantiate the opinion that vaping is not harmful to the mouth.

In the case of dental practice, the vaping history must be regularly recorded and incorporated into personal risk assessment. Vapors that use high-frequency and patients with unexplainable gingival inflammation, despite their use of vaping, must be provided with enhanced preventive efforts, closer supervision, and routine counseling in a balanced and patient-centered approach. Future studies ought to focus on standardized exposure measurement, longitudinal clinical outcome, and combined host-microbiome studies to explain causal relationships and support evidence-based dental public health practices.

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