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REVIEW ARTICLE

Effects of tobacco smoking and electronic nicotine delivery systems on oral health – a narrative review

Natalia Paulina Bielecka-Kowalska[1,A-C], Sandra Bielicka[1,B,D], Natalia Lewkowicz[1,E-F]

[1] *Medical University, Łódź, Poland*

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Abstract

Introduction and Objective. Smoking is associated with periodontal disease, potentially malignant disorders and oral cancer. Recent increase in the electronic cigarette use creates new, yet undiscovered consequences for both general and oral health. Although the negative effects of nicotinism have been known for years, new information and correlations of the effects of tobacco smoking continue to emerge. The objective of this article is to provide an update on effects of both conventional smoking and electronic nicotine delivery systems on the oral health.

Review Methods. A review of the recent literature (from 01.2000 to 06.2022) was conducted using PubMed, Web of Since and Google Scholar databases.

Brief description of the state of knowledge. Smoking alters immune function promoting inflammation and impairs tissue healing. Typical problems for smokers include poor oral hygiene, halitosis and periodontal disease. Nicotinism contributes also to the oral cancer. Modern heat-not-burn nicotine delivery systems, which operate with lower temperatures and do not exude typical cigarette smoke, are increasingly popular alternative to traditional cigarettes. It is likely that e-cigarettes have fewer carcinogenic effects, but they contain nicotine and other substances that negatively affect the oral cavity. Similarly, IQOS cigarettes generate harmful products, but generally at lower concentrations than traditional cigarettes.

Summary. Oral pathologies are predominantly associated with the use of traditional cigarettes as a result of the oral tissue and immune system exposure to composition of cigarette smoke. Although modern electric nicotine delivery systems seem to be less harmful, their long-term effects on oral health are still under-recognized. Medical professionals, especially dentists should identify the addicted patient and counsel on quitting nicotine addiction.

Key words

periodontitis, smoking, oral diseases

INTRODUCTION AND OBJECTIVES

Cigarette smoking has a negative impact on general health and on the oral cavity as an example [1–3]. This harmful addiction can lead to various oral diseases, including periodontitis and cancer [1–4].

Cigarette smoke contains over 7,000 chemical compounds, many of which are toxic and/or carcinogenic, such as benzopyrene, volatiles, hydrogen cyanide, aldehydes, phenolics, aromatic amines, ammonia, carbon monoxide, nitrogen oxides and nicotine (Tab. 1). Tobacco smoke exhibits potent toxic, antigenic, cytotoxic, mutagenic and carcinogenic effects [5, 6].

Nicotine is the most well-known harmful factor which is absorbed by the tissues of the human body. One of its mechanisms of action is to increase the number of acetylcholine receptors in the central nervous system (CNS), showing a stimulant effect. This is one of the causes of the development of nicotine addiction. Over time, the addicted person experiences shorter and shorter periods of good well-being. This contributes to increased frequency of smoking [6].

Cigarette smoking contributes to deterioration of periodontal health. The toxic substances form tobacco smoke can disrupt the microbiome of the oral cavity through oxygen deficiency and other mechanisms; for instance, smoking leads to a reduction of saliva flow and thus promotes xerostomia which, in turn, results in increased accumulation of dental plaque [7, 8, 9]

Smoking disturbs the microbiota and immune response of the body [10]. It may also increase the level of certain pathogens and interfere with neutrophil function, the primary defence against infection, and antibody production [10]. The heat emitted from cigarettes during smoking, and toxins such

Table 1. Chemical compounds identified in tobacco smoke [7]

Compound	Toxicant yields in cigarette smoke (μg/cigarette)
Benzo[a]pyrene	3.9 ± 2.6
Hydrogen cyanide	22.4 ± 10.6
Acetaldehyde	360.8 ± 187.6
Phenol	13.5 ± 6.5
1-Naphthylamine	10 ± 4.9
Ammonia	10.1 ± 5.1
Carbon monoxide	9.0 ± 4.9
Nitrogen oxides	38.4 ± 20.6
Nicotine	0.8 ± 0.4

Address for correspondence: Natalia Bielecka–Kowalska, Medical University, Łódź, Poland
e-mail: natalia.bielecka-kowalska@umed.lodz.pl

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as nicotine, carbomonoxide and hydrogen cyanide, cause vasoconstriction [11]. Tobacco smoke contains carbon monoxide (CO) which displaces oxygen from haemoglobin [12]. Smoking greatly affects the periodontal tissues as a result of toxicity, promotion of pathogenic bacteria growth, oxygen deficiency, and disruption of the blood supply. Systemic disorders, a consequence of smoking, result in even more rapid degeneration of the structures supporting teeth [1, 13, 14]. Smoking generally leads to delayed tissue healing and therefore renders the regenerative mechanisms of bone and soft tissue less predictable [11, 15]. Conversely, quitting smoking slows down the progression of periodontitis and improves treatment response [10, 16].

REVIEW METHODS

Literature from the last 20 years – January 2000-June 2022 – was searched using key words 'smoking', 'periodontal disease', 'oral disease', 'risk factor for oral cancer', 'electronic cigarettes', 'heat-not-burn tobacco', 'nicotine', and 'nicotine addiction treatment' on the PubMed, Web of Since and Google Scholar databases. After having read and collected valuable data, the knowledge was divided into subsections as follows: gingivitis and periodontitis, influence of smoking on periodontal treatment, oral mucosal diseases, smoking as a risk factor of oral cavity cancer, electronic cigarettes, heat-not-burn tobacco and treatment of nicotine addiction.

DESCRIPTION OF THE STATE OF KNOWLEDGE

Periodontal diseases and smoking. Gingivitis is an inflammatory response to bacteria present in dental plaque [17]. Studies have shown that the clinical signs of gingivitis, erythema, oedema and bleeding, are not as pronounced in smokers as in non-smokers [13, 18]. The masking of gingival inflammation in smokers is a result of impaired gingival blood circulation [19, 20]. Nicotine stimulates the production of catecholamines by sympathetic ganglia (vasoconstrictor effect) and affects alpha-receptors on blood vessels, hence promotes vasoconstriction [8, 9]. In addition, the heat emitted from cigarettes during smoking, and toxins such as carbomonoxide and hydrogen cyanide cause vasoconstriction [11], and carbon monoxide (CO) displaces oxygen from haemoglobin [12]. Therefore, signs of gingival inflammation such as bleeding on probing (BOP) or erythema are less apparent than in non-smokers [13, 21]. Despite the less apparent manifestations of gingivitis, such as lower rate of BOP due to decreased gingival blood flow, studies have revealed a more frequent presence of periodontitis among smokers [22].

Cigarette smoking is a significant risk factor for periodontal disease, manifested by greater loss of attachment and progression of periodontitis [1, 10, 23]. Smoking has been proven to increase the likelihood of periodontal disease by at least five to six times [1, 24]. Smokers have also been proven to undergo a more severe form of the disease [1, 10, 25]. Various studies presented that there is a correlation between tobacco smoking and periodontal destruction, which is revealed by increased pocket depth (PD) among smokers [10]. It is estimated that heavy smokers' risk of developing periodontitis is twice as high as in moderate smokers, who still have two times greater chance of the disease than non-smokers [10]. As a result of greater loss of periodontal tissues and more advanced form of the disease, smokers have higher number of teeth lost due to periodontitis than non-smokers [20, 26, 27].

Smoking exerts a major impact on the pace of progression of periodontal disease [28, 29]. While a non-smoker who maintains good oral hygiene is rather likely to develop a rate of bone loss lower than 0.25 (% of bone loss divided by age), a patient who smokes more than 10 cigarettes a day can be suspected to lose over four times more bone [30, 31]. In addition, an average Polish smoker is estimated to smoke between 15 and 30 cigarettes daily, and the numbers are similar in other countries. The World Health Organization (WHO), in *On the Global Tobacco Epidemic, 2021 – Addressing New and Emerging Products*, reported that the prevalence of smoking among people over 15 years decreases from 22.7% to 17.5% [29, 30, 32]. Furthermore, worldwide, 1.1 billion people are considered smokers, and by 2025 this number is expected to increase to as much as 1.3 billion [33]. By combining the two higher mentioned facts, the conclusion is reached that an average smoker is in the group at risk of periodontal disease progression.

Several mechanisms can be responsible for increased susceptibility and disease progression in smokers. Smoking increases the incidence and severity of periodontal destruction by altering the host's immune response, which results in increased proliferation of dangerous bacterial species in the oral cavity [1, 13, 34]. Smoking causes impaired chemotaxis, adhesion, phagocytosis and antibody production [19,20,35]. IgG levels against periodontal bacteria, such as *Prevotella intermedia*, and *Fusobacterium nucleatum, Aggregatibacter actinomycetemcomitans,* were lower in smokers. Smoking also reduces the number of antigen-specific T cells [19, 35]. The extinction of beneficial oral bacteria due to smoking is another mechanism leading to increase in the population of pathogens and ultimately to the progression of periodontitis [1]. Carbon monoxide (CO), a powerful reducing agent contained in tobacco smoke, immediately reduces the potential for redox on the surface of the mucosa [36], which leads to the growth of anaerobic bacteria [33,37]. The reduced amount of oxygen in the periodontal pocket can promote the development of anaerobic conditions [38, 39].

Smoking affects subgingival microbiota. Bacteria from the red and orange complexes, such as *B. forsythus, P. gingivalis, T. denticola, E. nodatum, F. nucleatum S. vincentii, P. intermedia, P. micros* and *P. nigrescens,* have been shown to be more prevalent in active smokers than in former smokers or non-smokers. However, the differences between smokers and non-smokers were demonstrated only at the level of prevalence of certain bacterium, while the number or proportions did not differ [1, 38].

Key enzymes associated with periodontal inflammation are matrix metalloproteinases (MMPs) [13, 39], which are responsible for enhancing inflammation as well as degrading host tissues [40, 41]. MMP-8 is the most important collagenolytic metalloproteinase present in the oral cavity [13]. Neutrophils are the main source of MMP-8, and its release is regulated by neutrophil protein degranulation. High levels of MMP-8 correlate with increased PD levels and smoking [13, 40]. Moreover, smoking can increase bone destruction in periodontal disease by increasing the levels of pro-inflammatory cytokines and reducing the levels of

anti-inflammatory and antiresorptive factors such as IL-10. Nicotine in combination with periodontal pathogens has been shown to lead to increased production of IL-6 and IL-8 by gingival fibroblasts. TNF-α and IL-8 levels were also increased in the gingival crevicular fluid of smokers [42].

Influence of smoking on periodontal treatment. The response of smokers to periodontal treatment is less positive compared to non-addicted patients. Smokers have a greater loss of periodontium, a more advanced form of the disease and, in consequence, a higher number of teeth lost than non-smokers [20, 26, 27].

Studies showed that smoking has a negative effect on healing after various forms of treatment of periodontitis [20, 43]. Non-smokers and those who quit had a similar response to treatment, which shows that quitting smoking may be beneficial for treatment [20, 44]. However, this corresponded with longer duration of nicotine abstinence, and the reversal of the negative effects of smoking may atill be visible 10 years after quitting smoking [45]. Smokers, on the other hand, benefitted less after treatment [10, 20, 43]. Non-smokers were shown to have a 50% greater improvement in PD and clinical attachment level (CAL) after non-surgical periodontal therapy than smokers [20, 37]. Toxins and chemicals produced by cigarette smoking can delay wound healing by inhibiting the basic cellular functions responsible for tissue repair and regeneration [20]. Smoking impairs soft tissue and bone revascularization which have a significant impact on wound healing, especially following regenerative procedures in periodontology and implantology. Volatile substances from cigarettes, acrolein and acetaldehyde, inhibit the adhesion and proliferation of gingival fibroblasts [20, 37]. Exposure of fibroblasts to nicotine causes them to produce less fibronectin and collagen, which negatively affects periodontal tissue healing and causes progression of periodontal disease [20, 37]. Complications with implant failure are twice as high as in non-smokers [37]. Altogether, smoking leads to delayed tissue healing and, thus, making bone and soft tissue regenerative procedures less predictable [11, 15]. Conversely, quitting smoking slows down the progression of periodontitis and improves treatment response [10, 16].

Oral mucosal diseases. The combined negative impact of smoking products and high temperature can contribute to the keratosis of the oral mucosa and leukoplakia (Fig. 1) [24]. Leukoplakia is the most common potentially malignant disorder found in the oral cavity. It occurs on the mucous membrane of the cheeks, in the bite line of the teeth, in the area of the alveolar processes, on the lip, and on the lateral/ventral sides of the tongue or floor of the mouth [46]. The latter two are characterized by a high probability of malignant transformation, as much as a 43% chance [45]. Leukoplakia is clinically manifested as a white, well-demarcated lesion surrounded by a normal mucosa [46]. Leukoplakia is a clinical diagnosis that can only be made by exclusion, therefore this definition is misleading. The clinical diagnosis should typically be confirmed by excisional or incisional biopsy of the oral lesion and histopathologic examination. In the microscopic image, moderate hyperkeratosis and hyperplasia of the oral epithelium occur most often. Occasionally, epithelial dysplasia may be detected, suggesting a higher probability of malignant transformation in future [47, 48]. Leukoplakia needs to be differentiated from oral lichen planus (especially the plaque type), frictional keratosis, and of course from oral carcinoma [49,50].

'Smoker's palate' or nicotine stomatitis is another keratotic disorder seen in smokers. It is caused by the high temperature in the oral cavity that occurs during smoking cigarettes and cigars. Initially, palate mucosa become erythematous, and with time it becomes white and dry with numerous scattered irritated salivary gland openings on the hard palate, manifesting as red dots [51, 52].

As a result of smoking, discolouration of the soft tissues and teeth, together with halitosis, can be also be observed. Black hairy tongue and melanosis are most commonly seen (Fig. 2, 3, 4) [51]. Smoker's melanosis, which manifests as dark spots on the oral mucosa, is most commonly located on the gingiva (Fig. 3). This is a mild and reversible condition caused by stimulation of melanocytes by smoking products. The duration and the amount of smoking play a role in the development of this condition [52].

Smoking as a risk factor of oral cavity cancer. Tobacco use is recognised as one of the most influential risk factors of oral

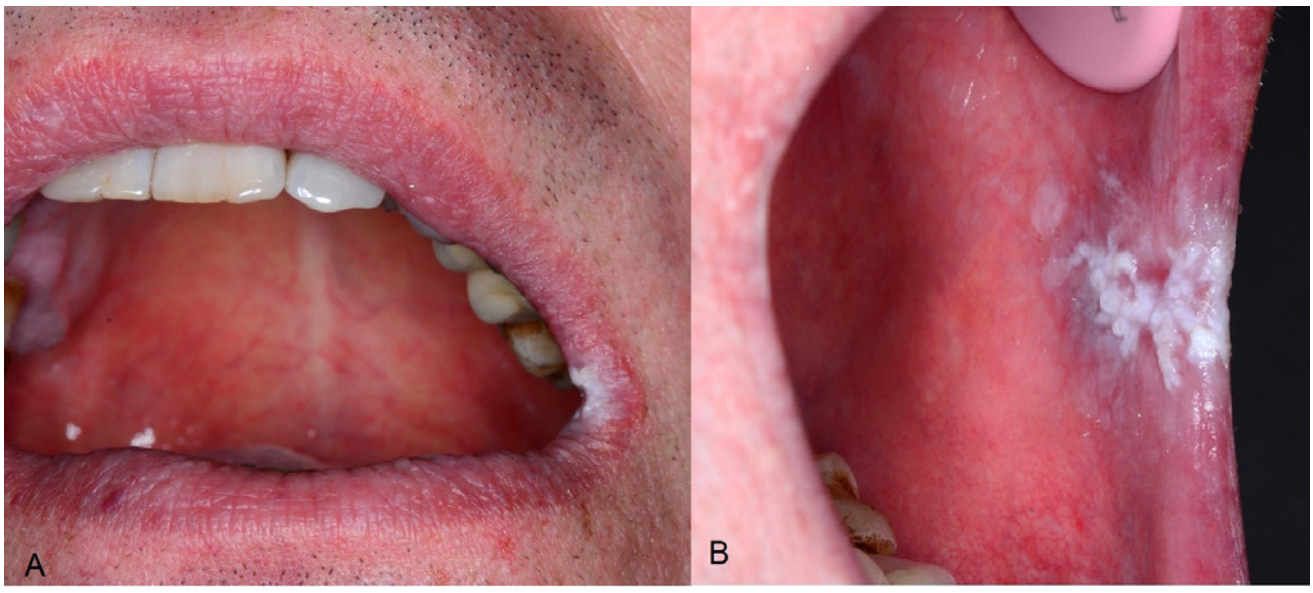

Figure 1. Leukoplakia on the mucosa of the labial commissure. A. Extraoral view; B. Intraoral view.

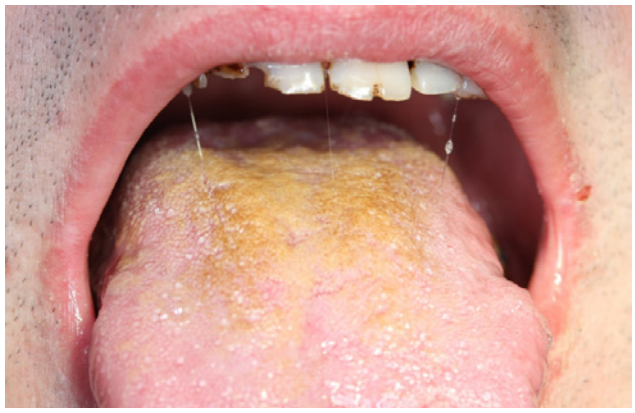

Figure 2. Filiform papilla overgrowth and discoloration in a smoking patient

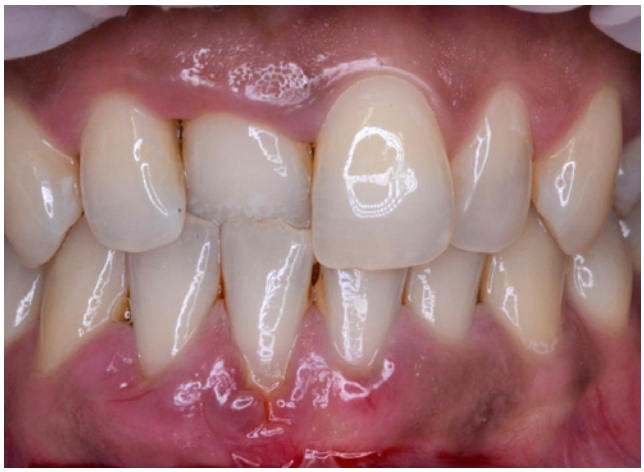

Figure 3. Gingival melanosis and periodontitis in a smoker

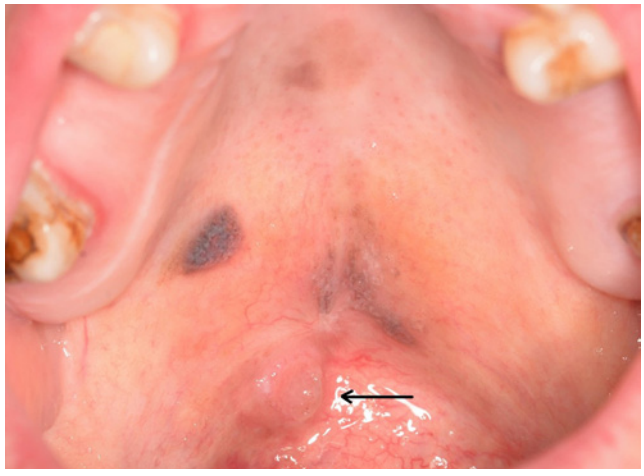

Figure 4. Smoker's melanosis on the palatal mucosa and a solitary ulcer/induration (arrow)

and oropharyngeal cancer; this refers to all forms of tobacco, including, for instance, chewing tobacco. Cigarette smoking is a factor that significantly increases cancer risk, and many people with a history of cancer are previous smokers [53, 54]. The components of cigarette smoke that are implicated in carcinogenesis include: 1.3-butadiene, isoprene, acrylonitrile, benzene, formaldehyde, acetaldehyde, quinoline, lead, cadmium, 1-naphthylamine, 2-naphthylamine, 4-aminobiphenyl, benzopyrene, N-nitrosonornicotine (NNN) and (methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) [5, 55, 56].

Oral squamous cell carcinoma (OSCC) is a predominant type of oral cancer. Cancers can develop from a potentially malignant disorder or *de novo*. The risk of developing OSCC based on leukoplakia is higher when the lesion is located on the tongue, floor of the mouth or lip [1, 3, 57]. the global incidence of OSCC in leukoplakia biopsies is estimated to be 11.11% [57]. The common symptoms of oral cancer mainly include ulceration or tumour, induration, wound or rupture. Patients may report a feeling of numbness, reduced tongue mobility, impaired speech, difficulty swallowing, foreign body sensation in the throat, discomfort, hoarseness, denture mismatch, swelling, lack of post-extraction healing, impaired mandibular mobility, bad breath, and voice change [49, 56]. In the early stages, pain is sometimes present and may be ignored [58, 59]. The lesions often remain asymptomatic for a long time.

The incidence of oral, laryngeal and pharyngeal cancer of 11.5/100,000 people in Central and Eastern Europe is lower than the incidence worldwide, or than the incidence in the more developed countries. The biggest changes between male mortality in 2012 and mortality between 2000 – 2004 were as follows: in Poland 90.63%, Bulgaria – 181.91% and Romania – 94.19%. For females, the rates were: Czech Republic – 471.69%, Bulgaria – 307.44% and Poland – 275% [60].

Despite the widespread access to diagnostics and the ease of histopathological examination, these tumors are most often diagnosed at an advanced stage, which reduces the chance of good prognosis [61].

Dissolution of toxic substances of toxic substances from cigarette smoke in alcohol leads to a stronger penetration into the epithelial cells, which leads to a higher chance of developing a malignant tumour. Smoking promotes carcinogenic transformation in two ways: by affecting the immune response and by interfering with the DNA. Nicotinic acetylcholine receptor agonists, or activation of the β-adrenergic receptor by substances contained in cigarette smoke, compromise the ability of the immune system to eliminate cancer cells, and may increase cell proliferation, angiogenesis, migration or invasion, and reduce cell death upon exposure to cytotoxic agents such as chemotherapy or radiation therapy – adversely affecting treatment [61, 62, 63]. Chronic exposure to a mixture of aerosol chemicals and tobacco substances inhibits growth factors [59]. Carcinogenic substances inhaled during smoking can also interfere with cell DNA, cause damage, and promote carcinogenic transformation. NNK, NNN and their metabolites are responsible for critical mutations involved in DNA replication. It is likely that genetic polymorphisms in genes encoding cytochrome P450 enzymes and conjugation by glutathione transferase play a significant role in the genetic predisposition to tobacco-induced cancers of the head and neck [59].

Cigarette smoking and alcohol consumption are two major risk factors of potentially malignant disorders and oral cancer. Other important risk factors include the habit of betel quid chewing, human papillomavirus (HPV) infection, diet, exposure to asbestos and polycyclic aromatic hydrocarbons [64]. These risk factors have a synergic effect; for example, HPV infection is favoured by deep kissing, the number of sexual partners, the number of oral sex partners, alcohol and tobacco consumption [64]. Eating small amounts of fruits and vegetables or a high consumption of meat, together with alcohol and smoking, are associated with a 10- to 20-fold increase in the risk of developing oral cancer [64].

The identification and controlling of different risk factors of oral cancer is therefore an important, but challenging issue. Smoking cessation is beneficial and highly recommended for the prevention of oral cancer, as well as during cancer treatment as it brings a greater chance of positive results, such as lower risk of death and smoking-related adverse events, reduces the risk of metastases and increases the effectiveness of oncological treatment [59, 61, 63].

Electronic cigarettes. In recent years, so-called electric cigarettes have gained popularity on the pretext of less harm to health, compared to traditional cigarettes. In fact, the exact effect of these cigarettes has yet to be studied in depth. The first studies indicated that they might be just as harmful as traditional cigarettes [65, 66]. The differences between traditional and electronic cigarettes are mainly that tobacco is smoked in the first place, the opposite to electric cigarettes. Electric cigarettes are considered to have a limited carcinogenic effect because they do not generate carbon monoxide and polycyclic tar hydrocarbons. The concentration of certain substances that are found in electric cigarettes, such as formaldehyde, acetaldehyde and acrolein, depends on the temperature at which they are burnt. With an increase in temperature, the amount of carcinogens becomes greater and even similar to the amount secreted by traditional cigarettes. However, temperature itself is not the only factor. The taste of e-liquid also seems to matter. For example, cherry-flavoured liquids contain large amounts of benzaldehyde, which is responsible for the feeling of shortness of breath, burning sensation in the throat, tearing and conjunctivitis [66].

As electric cigarettes also contain nicotine, which affects microcirculation, the risk of developing periodontitis may be as high as when traditional cigarettes are used. Heated flavouring agents and other ingredients, such as propylene glycol and glycerine, have an inhibiting effect on cell growth [67]. Although the influence of electric cigarettes on the oral cavity still needs further research, it cannot be excluded that alterations caused in oral tissues by electric cigarettes may be greater than those caused by regular cigarettes [68]. Moreover, in some particular cases, e-cigarettes seem to be even more addictive among young individuals than regular cigarettes [69].

Heat-not-burn (HnB) tobacco. Heat-not-burn tobacco (HnB), also known as IQOS, heat tobacco to a significantly lower temperature (about 350 °C) than traditional cigarettes, which, in comparison, burn at about 900 °C [70, 71]. Pagano analysed the cell cycle, viability, morphology, migration and apoptosis oral fibroblasts and keratinocytes exposed to HnB tobacco. The smoke stimulated cell proliferation because there were longer and more intense phases of the S and G2/M cell cycle, greater viability and migration, and more adhesive characteristics in keratinocyte morphology. However, it did not damage oral fibroblasts and keratinocytes because it did not modify survival or morphology. The IQOS aerosol was additionally tested and proved to also contain harmful products, although in lower concentrations than conventional cigarettes, and therefore less toxic [72]. However, HnB devices appear to produce more carbonyls aldehydes and free radicals compared to conventional cigarettes. Their toxic effects affect the upper gastrointestinal tract and lungs, including exhibiting carcinogenic effects. There are a limited number of studies that also included major carcinogens (Biomarker of Exposure, BoE) [70, 72].

A systematic review by Drovandi et al. reported that the potential adverse effects associated with smoking using HnB devices were less than those of traditional cigarettes. The level of BoEs generated by HNBs was shown to be lower. The greatest reduction was shown for carboxyhaemoglobin (COHb), 2-aminoaftalene (2-AN), 4-aminobiphenyl (4-ABP) and 2-cyanoethylmercapturic acid (CEMA). Thus, it can be concluded that IQOS are less harmful than conventional cigarettes [16]. In general, cigarettes such as HnB, emit much lower concentrations of tar, carbonyls, volatile organic compounds (VOCs), CO, free radicals and nitrosamines. For this reason, compared to a conventional cigarette, HnB smoking reduces exposure to pollutants, and thus reduces the risk of developing tobacco-related diseases, but does not eliminate the risk of their development [73, 74].

Treatment of nicotine addiction. The best approach to anti-smoking intervention is employment of the 5A rule (Tab. 2) [75]. The degree of nicotine addiction can also be assessed by the Fagerström Test for Nicotine Dependence (FTND) (Tab. 3). Assessment is on the basis of six questions for which

Table 2. Anti-smoking intervention 5A rule [72]

1A	Ask if the patient smokes
2A	Advise patient to stop smoking
3A	Accompany a diagnosis of exposure, dependency, and patient willingness to quit smoking
4A	Assist to choose the optimal treatment that will effectively help the patient
5A	Repeat the recommendations when the patient attends a follow-up visit

Table 3. Fagerström test for nicotine dependence [73,74]

Question	Answer
1) Do you currently smoke cigarettes?	No Yes* *If 'yes', read each question below. For each question, enter the answer choice which best describes your response.
2) How soon after you waking-up do you smoke your first cigarette?	Within 5 minutes 6 – 30 minutes 31 – 60 minutes After 60 minutes
3) Do you find it difficult to refrain from smoking in places where it is forbidden, e.g., in church, library, cinema?	No Yes
4) Which cigarette would you hate most to give up?	The first one in the morning Any other
5) How many cigarettes per day do you smoke?	10 or less 11 – 20 21 – 30 31 or more
6) Do you smoke more frequently during the first hours after waking than during the rest of the day?	No Yes
7) Do you smoke when you are so ill that you are in bed most of the day?	No Yes

Interpretation:
– Yes/no items are scored on a scale of 0 – 1
– Multiple choice questions are scored from 0 – 3
Items are summed-up to obtain a total score of 0 – 10.
The higher the total score, the more intense the patient's physical dependence on nicotine.

there are an appropriate number of points. The questions relate to, among other things, whether it is difficult for the respondent to quit smoking and how many cigarettes are smoked. The number of points is then added-up and the degree of advancement of addiction is determined [76, 77].

Nowadays, there are efficient treatment methods for cigarette addiction, either behavioural or based on pharmaceutic measures, which facilitate smoking cessation. Behavioural counselling should usually be performed by specialized consultants during several sessions. This therapy is found to be helpful also for those using cessation medicines, and is available in multiple variants [73]. It can be chosen between cognitive behavioural therapy sessions which depicts triggers and show how to cope with them without reaching for a cigarette, motivational interviews which support those who are ambivalent about quitting, mindfulness sessions in which thoughts, sensations and feelings that trigger the craving for a cigarette are purposely attended to an than they are being worked on so as to finally become reframed as tolerable**.** Furthermore, there are also various non-face-to-face supporting measures, such as by telephone and quit-lines, as well as text messaging and web-based services including social media support.

Apart from behavioural therapy, medication-including treatment can be chosen [56]. Nicotine replacement therapy (NRT) is based on the application of a nicotine substitutes in the form of a transdermal patch, spray, gum or lozenges. This treatment method helps to relieve cravings for a cigarette as the substitutes stimulate brain receptors for nicotine. Other available medications include bupropion, varenicline, some antidepressants (e.g. nortriptyline), as well as combination therapy. The combination of varenicline and NRT has been shown to be more effective in quitting smoking than varenicline alone. Similar results were obtained with bupropion with NRT. This kind of therapy is useful in individuals with a history of failure to quit smoking [56].

SUMMARY

Cigarette smoking possesses many risks to oral health, although new types of cigarettes, such as HnB, are likely to be less harmful. However, more research if need to indicate whether HnB could be a less harmful alternative for use by nicotine addicts. The degrading effects of tobacco in its different types on both the oral cavity and the entire body have been known for many years, and the only reliable way to reduce the adverse effects to health is to quit smoking.

Dental professionals should be actively involved in counselling their patients about smoking cessation and screen for the presence of periodontal disease, potentially malignant disorders or oral cancer. Self-observation and regular examination of the oral cavity is mandatory in patients who refuse to quit the smoking habit. Prevention should be focused mainly on regular office and home hygiene, and general oral health care. The education of patients about the harmful effects of smoking on oral health, together with motivation for quitting smoking and subsequent follow-up, should be included as routine procedures in dental professional activities.

Declaration

The authors declare that there are no conflicts of interest.

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