



NICOLAUS COPERNICUS
UNIVERSITY
IN TORUŃ



Journal of Education, Health and Sport. eISSN 2391-8306.

Journal Home Page

<https://apcz.umk.pl/JEHS/index>

ADAMCZYK, Adrianna, GAŁUSZKA, Maja, GAŁUSZKA, Aleksandra, KRYCIA, Kamila, MAZUR, Karolina, RÓŻYCKA, Karolina, MULAŁA, Magdalena, PIASZCZYŃSKA, Emilia, MATA CZ, Dominika and DRABIK, Sandra. The Impact of Cigarette Smoking on the Oral Mucosa. Journal of Education, Health and Sport. 2026;90:70553. eISSN 2391-8306. <https://doi.org/10.12775/JEHS.2026.90.70553>

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences). Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu). © The Authors 2026; This article is published with open access at Licensee Open Journal Systems of Nicolaus Copernicus University in Toruń, Poland
Open Access. This article is distributed under the terms of the Creative Commons Attribution Noncommercial License which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author (s) and source are credited. This is an open access article licensed under the terms of the Creative Commons Attribution Non commercial license Share alike. (<http://creativecommons.org/licenses/by-nc-sa/4.0/>) which permits unrestricted, non commercial use, distribution and reproduction in any medium, provided the work is properly cited.
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 02.04.2026. Revised: 20.04.2026. Accepted: 20.04.2026. Published: 23.04.2026.

The Impact of Cigarette Smoking on the Oral Mucosa

Adrianna Adamczyk¹ ORCID <https://orcid.org/0009-0009-3462-7972>

E mail ada.adamczyk@onet.pl

¹”Nowa Europa”, Łódź, Polska

Maja Gałuszka², ORCID <https://orcid.org/0009-0003-2659-0811>

E-mail am.galuszka@wp.pl

²Medical University of Lublin, Polska

Aleksandra Gałuszka² ORCID <https://orcid.org/0000-0003-1749-0811>

E-mail aleksandra.galuszka@interia.pl

²Medical University of Lublin, Polska

Kamila Krycia² ORCID <https://orcid.org/0009-0007-2586-1746>

E-mail kamilakrycia27@gmail.com

²Medical University of Lublin, Polska

Karolina Mazur² ORCID <https://orcid.org/0009-0001-9827-2084>

E-mail mazurinka2001@gmail.com

²Medical University of Lublin, Polska

Karolina Różycka² ORCID <https://orcid.org/0009-0004-0505-3280>

E-mail karar11@onet.pl

²Medical University of Lublin, Polska

Magdalena Mulawa² ORCID <https://orcid.org/0009-0004-2515-9533>

E-mail [magdalena.mulawa123@gmail.com](mailto:magdalenamulawa123@gmail.com)

²Medical University of Lublin, Polska

Emilia Piaszczyńska² ORCID <https://orcid.org/0009-0009-1416-6566>

E-mail piaszczyńskaemilia@gmail.com

²Medical University of Lublin, Polska

Dominika Matacz² ORCID <https://orcid.org/0009-0005-1975-3795>

E mail dominikam011@gmail.com

²Medical University of Lublin, Polska

Sandra Drabik² ORCID <https://orcid.org/0009-0003-9708-4625>

E mail sandradrabik01@gmail.com

²Medical University of Lublin, Polska

Corresponding Author

Adrianna Adamczyk E-mail ada.adamczyk@onet.pl

Abstract

Introduction. Tobacco smoking is the most common addiction worldwide. It is estimated that there are currently around 1.2 billion people addicted to this product globally (Reibel, 2005). The situation in Poland is not more optimistic - approximately 27–28% of adult Poles declare this habit (TNS Polska, n.d.). Each year, tobacco is responsible for the deaths of over 7 million people worldwide (World Health Organization, n.d.). Tobacco smoke contains approximately 4,000 chemical compounds, many of which are carcinogenic substances (Piekoszowski & Florek, 2001; Rudzinski & Banach, 2008). **Aim.** The aim of this study is to present the harmful effects of tobacco smoke on the condition of the oral mucosa and to discuss the most common pathological changes associated with exposure to tobacco smoke. **Material and methods.** The research material consisted of scientific publications concerning the impact of cigarette smoking on the oral mucosa. The literature review was conducted based on the PubMed and Google Scholar databases. Publications in both Polish and English, published between 1987 and 2023, were included.

Key words: Smoking, tobacco, oral mucosa

1. Introduction

The main active component is nicotine, which has a stimulating effect on the central nervous system (CNS), resulting in its strong and rapid addictive potential. Experimental studies, both *in vitro* and *in vivo*, have shown that this substance is distributed to all tissues of the human body (Tutka et al., 2005). Tobacco has systemic effects, for example on the respiratory and cardiovascular systems, and also exerts local effects, causing pathological changes in the oral cavity. Tobacco smoke first comes into contact with the oral mucosa. It has long been known that smoking negatively affects the condition of the oral mucosa. Pathological changes begin at the molecular level and are associated with a series of reactions that affect the entire body (Fontes et al., 2008). Cigarette smoking causes vasoconstriction, which drastically reduces blood supply to the gums. The delivery of nutrients and immune cells is limited, which impairs wound healing and tissue regeneration. Tobacco smoke affects the mucosal tissues through thermal, mechanical, and chemical irritation. The mucosa responds to this process through defensive processes such as keratinization, hyperkeratosis, atrophy, or cellular transformation (metaplasia). Tobacco also alters the structure of saliva as well as its antibacterial and antioxidant properties, and reduces its secretion. Dry mouth, in turn, promotes infections, mechanical damage, and the development of dental caries. Components of cigarette smoke intensify inflammation in oral tissues and oxidative stress, leading to the development of

periodontal diseases. It affects the metabolism of proteins and nucleic acids, disrupting the body's defense and repair processes. The physical effects of tobacco smoke are also harmful, as it increases the temperature in the oral cavity and can lead to tissue burns. It may also increase the risk of congenital defects in children, such as cleft palate or cleft lip. Smoking promotes the growth of pathogenic bacterial flora affecting periodontal tissues, causes destruction of the alveolar bone, and increases tooth loss. Moreover, smokers exhibit discoloration of teeth, oral mucosa, fillings, and prosthetic restorations, as well as bad breath (halitosis) and impaired taste perception. The most serious oral health risk associated with smoking is oral cancer. A dentist is often the first person to notice the consequences of tobacco use. In nearly three-quarters of smokers, lesions of the oral mucosa are observed (Chandra & Govinadraj, 2012). The most common include lichen planus, leukoplakia, melanosis, hairy black tongue, and others. These changes are usually located on the buccal mucosa and least frequently on the floor of the mouth (Aljabab et al., 2015; Taybos, 2003).

2. Leukoplakia

Leukoplakia is defined as a lesion in the form of white patches or streaks resulting from excessive keratinization of the epithelium. A characteristic feature is that it cannot be mechanically removed (Petkowicz et al., 2004; Axell, 1987). Histopathological changes in leukoplakia show considerable variability, ranging from typically benign lesions to carcinoma in situ (Waškowska & Koszowski, 2006). The occurrence of leukoplakia depends, among others, on the number of cigarettes smoked daily, the duration of addiction and simultaneous alcohol abuse. Leukoplakia occurs most often in men between 40–60 years of age and appears in different localizations in the mouth. In smokers, leukoplakia occurs up to 21 times more frequently than in non-smokers. The most common locations in smokers are the buccal mucosa and the floor of the mouth, whereas in non-smokers it is most often found on the dorsal surface of the tongue. This distribution results from increased accumulation of toxins from tobacco smoke in saliva (Banoczy et al., 2001). Lesions located on the floor of the mouth show a greater tendency for malignant transformation (Lima et al., 2012). The risk of cancerous transformation of leukoplakia also increases with the number of smoked cigarettes. Early diagnosis and appropriate treatment of leukoplakia, including complete smoking cessation, is an important element of cancer prophylaxis and to a large extent prevents the development of oral cancer. The management of leukoplakia depends on the clinical presentation of the lesion, the presence and severity of epithelial dysplasia, and associated risk factors. A fundamental component of treatment is the elimination of etiological factors, particularly tobacco use and alcohol consumption, as well as the removal of local mechanical irritants such as sharp tooth edges or ill-fitting dental prostheses. Improvement of oral hygiene is also essential. In certain cases, especially when no dysplasia is present, lesions may partially or completely regress following the elimination of these factors. Conservative management is primarily recommended for lesions without dysplasia or with low-grade changes and involves regular clinical follow-up and, when necessary, periodic histopathological evaluation. Management also includes the treatment of coexisting conditions, such as candidiasis, and in some cases the use of chemopreventive agents, including vitamin A or beta-carotene, although their long-term effectiveness remains limited. Surgical intervention is indicated particularly in lesions exhibiting epithelial dysplasia or those at high risk of malignant transformation. Treatment options include conventional surgical excision, laser ablation (most commonly with a CO₂ laser), and less frequently cryosurgery. All excised tissues should be subjected to histopathological examination to confirm the diagnosis and assess the degree of dysplasia

(Sarkar & Rathod, 2014; Yu et al., 2014). Adjunctive therapeutic approaches, such as photodynamic therapy, immunomodulatory agents, and retinoids, may be considered in selected cases; however, their use is often limited by variable efficacy and potential adverse effects (Nagao et al., 2015). Regardless of the chosen treatment modality, long-term follow-up is essential. Patients should undergo regular clinical examinations to monitor for recurrence and to enable early detection of potential malignant transformation. Effective management of leukoplakia therefore requires a comprehensive approach combining risk factor elimination, appropriate therapeutic intervention, and continuous monitoring.

3. Erythroplakia

Erythroplakia is an oral lesion characterized by a red, smooth, or slightly raised area on the mucous membrane. Unlike leukoplakia, which is usually white, erythroplakia has a distinct red color and is more suspicious for malignancy. It is most commonly located on the floor of the mouth, the lateral surface of the tongue, the soft palate, and the area behind the last molar (retromolar triangle). The lesions typically appear as single, vividly red patches with an oval or round shape and a shiny, granular, or smooth surface. This condition most often occurs in individuals over 40 years of age and is more common in men. People who are chronic smokers are at the highest risk. Erythroplakia is more difficult to diagnose than leukoplakia and is considerably more dangerous. The lesions are usually asymptomatic, which often delays detection. Patients may notice a persistent red patch that does not heal, sometimes accompanied by mild burning or discomfort. In more than 40% of cases, the lesion develops into a malignant tumor, making erythroplakia the most potentially dangerous of all oral mucosal pathologies. To establish a diagnosis, a biopsy is necessary; it should be performed in the perilesional area. Histopathological features of erythroplakia include epithelial atrophy with varying degrees of atypia. Treatment usually involves surgical removal of the lesion followed by histopathological evaluation. Precancerous lesions in the oral cavity may also be treated with laser surgery or photodynamic therapy. Due to the risk of recurrence, continuous patient monitoring is essential. Complete smoking cessation and limiting alcohol consumption significantly reduce the risk of disease progression (Warnakulasuriya et al., 2007; Mortazavi et al., 2014; Vedtofte et al., 1987).

4. Melanin Pigmentation

Cigarette smoking stimulates melanocytes in the oral mucosa to increase melanin production, resulting in melanin pigmentation (EU-Working Group on Tobacco and Oral Health, 1999). Smoking-related hyperpigmentations in the mouth mucosa or on the lips are observed mainly in women and it is believed that they are related to female sex hormones. Melanin has antioxidant properties; therefore, hyperpigmentation represents a defensive response of tissues to the elevated temperature generated in the oral cavity during smoking (Bharath et al., 2015). These changes are reversible and tend to resolve after smoking cessation. Melanosis is the most common lesion observed in the oral mucosa prior to the development of squamous cell carcinoma and leukoplakia (Behura et al., 2015).

5. Halitosis

The problem of unpleasant breath odor is associated with volatile sulfur compounds present in tobacco smoke. A significant relationship has been observed between the severity of this condition and both the duration of the habit and the intensity of smoking. The causes of halitosis may be local or systemic; the latter include diseases of the respiratory, digestive, and urinary

systems, haematological diseases, psychiatric diseases, vitamin deficiencies and medicines. The local causes are: reduced saliva secretion, smoking, diet (consumption of onion, garlic, alcohol), and bad oral hygiene. Habitual smokers often feel that they are permeated with the smell of cigarettes. Due to this, addicted individuals frequently use oral fresheners which not only provide temporary relief, but may also promote the development of dental caries (as they usually contain sugars) (Piątkowska & Szymańska, 2004; Kamińska et al., 2005).

6. Fungal infections

Oral candidiasis is an opportunistic infection caused by fungi of the *Candida* genus. An increase in the number of fungi inhabiting the oral cavity may lead to local inflammation of the oral mucosa, discomfort, burning sensation, dryness, altered taste perception, and even dysphagia. Smoking is one of the local factors contributing to the development of *Candida* infections. The exact mechanism by which smoking promotes fungal infections has not been fully clarified. Most likely, cigarette smoke induces changes in the epithelium, weakening its protective properties. Smoking also reduces blood flow through the oral mucosa due to vasoconstriction, which decreases the regenerative capacity of the mucosa (Górska, 2023; Słowińska et al., 2000). The management of oral candidiasis is primarily based on antifungal therapy and the elimination of predisposing factors. Depending on the severity of the condition and the patient's general health, either topical or systemic treatment may be applied. In mild cases, topical therapy is usually sufficient and includes antifungal agents in the form of suspensions, gels, or lozenges containing substances such as nystatin, miconazole, clotrimazole, or amphotericin B. These agents act directly on the oral mucosa; therefore, prolonged contact with the affected tissues is recommended to enhance therapeutic effectiveness. In more severe cases, in patients who do not respond to topical treatment, or in immunocompromised individuals, systemic antifungal therapy is required. Fluconazole is the most commonly used drug, while itraconazole, posaconazole, or systemic amphotericin B may be considered in refractory cases. The choice of treatment depends on the severity of infection, patient age, and the presence of underlying conditions. An essential component of management is also the elimination of causative factors. This includes improving oral hygiene, proper cleaning or replacement of dental prostheses, reducing predisposing factors such as prolonged antibiotic or corticosteroid use, and controlling systemic diseases such as diabetes mellitus. These measures significantly reduce the risk of recurrence and improve treatment outcomes. The duration of therapy typically ranges from 7 to 14 days and should be continued for at least 48 hours after the resolution of clinical symptoms to prevent relapse. In recurrent or treatment-resistant cases, microbiological testing is recommended to identify the causative organism and guide targeted therapy (Łabuś et al., 2018).

7. Xerostomia

Smokers frequently experience xerostomia, or troublesome dry mouth. Saliva becomes foamy, thick, and sticky, and may form strands on the tongue, cheeks, and gums. The mucosa appears thin and pale. Dry mucosa is significantly more prone to injury, and such injuries show a reduced tendency to heal. The risk of secondary fungal and bacterial infections increases. Xerostomia may also be accompanied by angular cheilitis and halitosis. Prolonged dryness of the mucosa impairs epithelial renewal, leading to atrophy of the tongue papillae (Górska, 2023). Additionally, clinical examination may reveal a smooth or wrinkled tongue as well as fetor ex ore (Mielnik-Błaszczak et al., 2009). Among the many diseases of the oral cavity, dryness of the mucous membranes significantly affects patients' quality of life and is a difficult problem to treat for both dentists and general practitioners. Dry mouth is a condition that negatively

impacts patients' emotional well-being. It contributes to low mood, the development of anxiety states, depressive symptoms, and cancer-related phobia. For effective treatment of dry mouth, the pathogenesis is determined based on a thorough medical history. The treatment of xerostomia may be either causal or symptomatic. Good results can be achieved by chewing sugar-free gum or sucking lozenges containing citric acid, as well as by selecting an appropriate diet that requires chewing. The use of vitamin A, B vitamins, and iron also has beneficial effects (Jańczuk & Banach, 1998).

8. Hairy Black Tongue

Chronic chemical irritation and colonization by yeast may lead to discoloration, hypertrophy, and keratinization of the filiform papillae of the tongue. In such cases, the condition is referred to as hairy black tongue (Maciąg & Bachanek, 2006). Its prevalence among heavy smokers is estimated at 58% in men and 33% in women (Gurvits & Tan, 2014). The condition is often associated with poor oral hygiene, smoking, excessive coffee or tea consumption, alcohol use, and certain medications such as antibiotics. It can also occur in individuals with a dry mouth or after radiation therapy. Although typically painless, it may cause bad breath (halitosis), an altered taste sensation, or a tickling sensation on the tongue. Treatment focuses on improving oral hygiene, gently brushing or scraping the tongue to remove the buildup, reducing contributing factors like smoking or certain foods, and staying well-hydrated.

9. Neoplastic Lesions

Smoking is the main risk factor for the development of oral cancers. Approximately 75% of head and neck cancers are associated with cigarette smoking, tobacco chewing, or correlated with alcohol consumption. Oral cancer ranks 11th in terms of global incidence. Smoking contributes to about 25% of all cancers in men and approximately 4% in women (Stewart & Kleihues, 2003). Squamous cell carcinoma (SCC) is the most common type, accounting for as many as 90% of cases. It usually occurs on the ventrolateral surface of the tongue and the floor of the mouth, which together with the retromolar trigone forms a horseshoe-shaped area where 75–85% of extraoral cancers are located (Mirbod & Ahing, 2000). SCC develops on the basis of epithelial dysplasia. The main risk factor is prolonged exposure to carcinogens such as tobacco. Importantly, even after cessation, the risk of developing cancer remains statistically higher due to DNA changes such as deletions, rearrangements, and point mutations (Kолоkythas et al., 2011). Epithelial dysplasia clinically manifests as leukoplakia (homogeneous or non-homogeneous) or erythroplakia. The risk of malignant transformation ranges from 6.6% to 36.4% (Ho et al., 2012). It should be stressed that the treatment of craniofacial cancer is often related to considerable facial tissue loss, which causes a decrease in patients' quality of life, their self-esteem, and may lead to severe depression. Malignant neoplasms in the oral cavity are often diagnosed too late, which reduces the chances of full recovery and survival time.

10. Nicotine Stomatitis

Nicotine stomatitis (stomatitis nicotinic), also called smoker's palate, manifests itself as whitish-grey foci, likened to cobblestones, that appear on the palate in addicted cigar and cigarette smokers. The affected palate is thickened and hyperkeratotic. On its surface, one can often observe inflamed duct openings of minor salivary glands in the form of red papule-like elevations. Depending on the severity of the process, the lesions can take various forms – ranging from subtle white spots, to clustered or diffuse papules and plaques, and up to red, raised nodules. Those lesions are caused by local exposure to high temperature. There is a minor

risk of the described lesions becoming malignant (Brzeziński, 2010). Particularly dangerous is so-called reverse smoking, which involves holding the lit end of a cigarette or cigar inside the mouth (a practice common in South America, India, and the Philippines).

11. Implant Prosthetic Treatment

The long-term stability of dental implants relies on successful osseointegration, defined as a direct structural and functional connection between the implant surface and the surrounding bone. Numerous studies indicate that this process is significantly compromised in smokers compared to non-smokers, which is why tobacco use is considered a major risk factor for implant therapy failure. One of the leading causes of implant loss is peri-implantitis, a pathological inflammatory condition associated with an imbalance between pathogenic biofilm and the host's immune response. Smoking has been shown to increase the prevalence of peri-implantitis, contribute to chronic inflammation of peri-implant mucosa, and accelerate marginal bone loss. A dose-dependent relationship has also been observed, with heavier smokers experiencing greater bone resorption around implants. While bacterial colonization remains the primary etiological factor in peri-implantitis, smoking exacerbates disease progression by impairing host defense mechanisms. It negatively affects vascularization, reduces tissue perfusion, and limits oxygen supply, which in turn compromises healing and regenerative capacity. Additionally, smoking interferes with osteoblast function and new bone formation, further undermining implant integration and stability. The combined effect of increased microbial load, diminished immune response, impaired healing, and reduced bone-forming potential significantly raises the risk of implant failure in smokers. Importantly, smoking cessation has been demonstrated to substantially improve treatment outcomes and reduce the likelihood of complications (Jaworska-Zaremba et al., 2007; Szpak et al., 2012; Koszuta et al., 2012). For this reason, thorough patient education is essential during treatment planning. Dentists should clearly communicate all modifiable risk factors and strongly encourage smoking cessation, as it greatly enhances the probability of successful osseointegration and long-term implant survival.

12. Periodontal Problems

Numerous studies indicate that smokers retain fewer teeth compared to non-smokers, while the rate of tooth loss is significantly higher among individuals addicted to cigarette smoking. Greater destruction of the alveolar bone and more pronounced loss of epithelial attachment have also been observed in this group. Moreover, smokers are at a much higher risk of developing severe forms of periodontal disease, and the overall intensity of these conditions tends to be greater. Periodontitis is strongly associated with the effects of nicotine and tar components of tobacco. In smokers, reduced salivary flow impairs the natural self-cleansing mechanisms of the oral cavity, leading to increased accumulation of subgingival plaque and, consequently, a higher load of pathogenic bacteria affecting periodontal tissues. Smoking also lowers the pH of dental plaque, which promotes its mineralization and facilitates the formation of both supra- and subgingival calculus. The presence of calculus contributes directly to the development of gingivitis and periodontitis. An increased number of potentially pathogenic bacteria is consistently found in smokers, while the host immune response to these microorganisms is weakened, resulting in less effective microbial control. Research by Semlali et al. (2009) demonstrated that even a single exposure to tobacco smoke can significantly disrupt both the morphology and function of gingival fibroblasts. Additionally, cigarette smoke inhibits epithelial cell proliferation and interferes with their migration, thereby impairing the healing of

periodontal tissues. Nicotine also exerts systemic effects by reducing blood flow within tissues. It stimulates the adrenal glands to release higher levels of adrenaline and noradrenaline, which cause constriction of peripheral blood vessels and alter gingival circulation. As a result, clinical examination using a periodontal probe may reveal reduced bleeding from periodontal pockets in patients with chronic periodontitis, potentially masking disease symptoms. Vasoconstriction further limits oxygen delivery to periodontal tissues, creating hypoxic conditions that favor inflammation. In such an environment, destructive enzymes targeting the periodontal ligament become active, ultimately leading to the breakdown of the attachment between tooth roots and bone, increased tooth mobility, and eventual tooth loss. Additionally, nicotine-induced stress negatively impacts periodontal health, significantly increasing the risk of periodontal disease. Importantly, smoking cessation has been shown to markedly improve periodontal condition, reduce the risk of disease development, and enhance the effectiveness of periodontal treatment (Piekoszowski & Florek, 2001; Moś & Ogłodek, 2009; Kowalski & Dragan, 2006).

13. Taste Disorders

Proper taste perception is one of the most important sensory experiences, yet numerous studies indicate that it is often impaired in smokers, a condition known as dysgeusia. The sense of taste plays a significant role in both physical and psychological well-being. Taste buds on the tongue are responsible for detecting flavors. In smokers, reduced salivary flow leads to altered taste perception, causing discomfort and affecting the enjoyment of food. Research has shown that in 85% of individuals addicted to smoking, the threshold for detecting saltiness is 12 to 14 times higher than in non-smokers (Barczewska & Nowak, 2000).

14. Superficial Tongue Inflammation

Tobacco smoke contains numerous irritants and toxic substances that directly damage the oral mucosa. Chemical irritation caused by nicotine can lead to superficial inflammation of the tongue (glossitis superficialis). The tongue becomes painful, reddened, and sometimes even swollen. Patients often report sensations of pain and burning. The filiform papillae may undergo excessive shedding or increased keratinization (Górska, 2007). While supportive care can alleviate symptoms, continued smoking often leads to persistence or recurrence of the condition.

15. Wound Healing

Smoking has a significant negative impact on wound healing. Chemicals in tobacco smoke, including nicotine, carbon monoxide, and cyanides, reduce blood flow to tissues and decrease oxygen delivery, both of which are essential for tissue repair. Nicotine also constricts blood vessels and impairs the function of fibroblasts and other cells responsible for collagen production and tissue regeneration. Smokers are also at higher risk of postoperative complications, including infection and delayed recovery. By-products of tobacco combustion delay wound healing, contributing to a higher rate of complications following oral surgical and periodontal procedures. The occurrence of a dry socket after tooth extraction is reported to be four times more frequent in smokers compared to non-smokers (Gonul et al., 2011).

16. Smokeless Tobacco Products

In Poland, the most popular forms of tobacco are powdered tobacco (snuff) and chewing tobacco. Snuff is highly addictive, as it contains about 20 times more nicotine than cigarettes. Its use can cause changes in the oral mucosa, gum recession, and root caries. Among chewing tobacco users, common oral changes include keratosis, leukoedema, and a condition known as “Pan-chewer’s lesion” (observed in 21.8% of cases). This appears as a brownish-black lesion on the oral mucosa, typically in areas where the tobacco or betel is held (most often on the cheeks), and it can be wiped off with a gauze pad. The mucosa in these areas tends to become roughened, wrinkled, and thickened, with yellowish or reddish-brown deposits caused by pigments (National Cancer Institute & Centers for Disease Control and Prevention, 2014). In Scandinavian countries, an alternative to cigarettes is snus, a smokeless product placed under the upper or lower lip, from which nicotine is absorbed (Kallischnigg, Weitkunat, & Lee, 2008). The popularity of snus is increasing, especially among young people, partly due to marketing portraying it as a product that can be used in smoke-free environments such as restaurants or airplanes. Snus use can cause gum inflammation and pain at the site where it is held in the mouth, and it may also increase susceptibility to dental caries. The most common lesion associated with snus is leukoplakia (Underner, Perriot, & Peiffer, 2012).

17. Smokeless Tobacco in Eastern Countries

In Eastern countries, smokeless tobacco is often mixed with betel leaves or areca nuts, increasing the mutagenic potential of these products for oral tissues by up to 15 times. Other prepared forms include toombak, shamma, naswar, khaini, and zarda. These are placed between the teeth and cheek mucosa and are chewed or held in the mouth for several hours. The alkaloids released from areca nuts produce a euphoric effect. Oral mucosal changes result both from the chemical composition of the chewing mixtures and from the prolonged mechanical action of chewing. Individuals who chew only areca nuts may have clinically normal oral mucosa without changes in texture or color. In contrast, 89% of betel chewers develop white, linear lesions known as oral submucous fibrosis (Avon, 2004).

18. Oral Submucous Fibrosis (OSMF)

The most common oral condition among betel chewers is oral submucous fibrosis. This disease is primarily observed in populations from South Asia and is characterized by inflammation, excessive collagen deposition in the submucosal layer, and the formation of fibrous bands (Auluck, Rosin, Zhang, & Sumanth, 2008). In its early stages, OSMF presents as slight mucosal opacification accompanied by a burning sensation, particularly when consuming spicy foods. In advanced stages, white horizontal and circular fibrotic bands appear on the buccal mucosa of the oral vestibule. These changes can lead to restricted mouth opening as well as difficulties with swallowing and speaking (Sabharwal, Gupta, Kapoor, Puri, Rajpal, 2013). Management of OSMF focuses on relieving symptoms, improving mouth opening, and preventing malignant transformation. The first step is cessation of betel quid chewing, tobacco use, and alcohol consumption, which slows disease progression. Early or moderate cases are treated conservatively with intralesional corticosteroids, sometimes combined with enzymes to reduce fibrosis, along with antioxidants and regular mouth-opening exercises to maintain oral mobility. In advanced cases, surgical excision of fibrotic bands and reconstruction with grafts may be necessary. Long-term follow-up is essential due to the risk of progression to oral cancer, highlighting the importance of early intervention and a multidisciplinary approach (Arakeri et al., 2017; Gopinath et al., 2022).

19. Summary

Tobacco smoking is associated with numerous serious consequences for both overall and oral health. Its harmful effects range from minor inconveniences, such as bad breath and dry mouth, to mild mucosal lesions like hairy black tongue, and can progress to life-threatening precancerous lesions and cancers. Smoking negatively affects not only the oral cavity but also the respiratory, cardiovascular, and immune systems, and it impairs tissue repair and wound healing. Cigarette smoking is one of the main preventable risk factors for cancer. Early detection of precancerous oral lesions, such as leukoplakia and erythroplakia, during routine dental exams can significantly reduce the risk of progression to malignancy. Smoking also contributes to periodontal disease, tooth loss, impaired taste, and delayed healing after dental procedures. Changing long-standing habits requires strong motivation and discipline, as nicotine addiction and behavioral patterns make quitting challenging. Dentists play a key role in this process, often being the first healthcare professionals to notice the consequences of tobacco use. Their responsibilities include raising patient awareness about the risks of tobacco, educating patients on its impact on oral health, and guiding them toward smoking cessation programs, counseling, and supportive interventions such as nicotine replacement therapy. In addition to clinical management, promoting proper oral hygiene, a balanced diet, and regular dental check-ups are essential preventive measures. Reducing tobacco use not only improves individual health but also lowers the broader societal burden of tobacco-related diseases, making patient education and intervention a critical aspect of public health.

Disclosure:

Author Contribution Statement.

Conceptualization: Adrianna Adamczyk, Aleksandra Gałuszka

Methodology: Adrianna Adamczyk, Karolina Różycka, Maja Gałuszka

Formal analysis: Adrianna Adamczyk, Emilia Piaszczyńska, Kamila Krycia

Investigation: Adrianna Adamczyk, Aleksandra Gałuszka, Maja Gałuszka, Karolina Mazur

Data curation: Sandra Drabik, Magdalena Mulawa, Emilia Piaszczyńska

Writing – original draft preparation: Adrianna Adamczyk

Writing – review and editing: Adrianna Adamczyk, Kamila Krycia, Karolina Mazur, Karolina Różycka, Emilia Piaszczyńska, Dominika Matacz, Magdalena Mulawa, Aleksandra Gałuszka, Maja Gałuszka, Sandra Drabik

Visualization: Adrianna Adamczyk, Karolina Różycka, Dominika Matacz

Project administration: Adrianna Adamczyk

Supervision: Adrianna Adamczyk, Karolina Mazur, Magdalena Mulawa

All authors have read and agreed to the published version of the manuscript.

References

Reibel, J. (2005). Tobacco or oral health. *Bulletin of the World Health Organization*, 83, 643–644.

TNS Polska. (05.02.2026). Report from a nationwide survey on attitudes toward tobacco smoking conducted for the Chief Sanitary Inspectorate (Poland).

World Health Organization. (05.02.2026). Tobacco. <https://www.who.int/news-room/fact-sheets/detail/tobacco>

- Piekoszowski, W., & Florek, E. (2001). Markery narażenia na dym tytoniowy. Poznań: Katedra i Zakład Toksykologii Akademii Medycznej w Poznaniu.
- Rudzinski, R., & Banach, J. (2008). Wpływ nawyku palenia tytoniu na stan przyzębia oraz toksyczne oddziaływanie nikotyny i jej metabolitów na tkanki przyzębia. *Czasopismo Stomatologiczne*, 61, 635–643.
- Tutka, P., Mosiewicz, J., & Wielosz, M. (2005). Pharmacokinetics and metabolism of nicotine. *Pharmacological Reports*, 57, 143–153.
- Fontes, P. C., Correa, G. H., Issa, J. C., Brandao, A. A., & Almeida, J. D. (2008). Comparison of exfoliative pap stain and AgNOR counts of the tongue in smokers and non-smokers. *Head and Neck Pathology*, 2, 157–162.
- Chandra, P., & Govinadraj, P. (2012). Prevalence of oral changes among tobacco users. *Oral Health & Preventive Dentistry*, 10, 149–153.
- Aljabab, M. A., Aljabab, A. A., & Patil, S. R. (2015). Evaluation of oral changes among tobacco users of Aljof Province, Saudi Arabia. *Journal of Clinical and Diagnostic Research*, 9, 58–61.
- Taybos, G. (2003). Oral changes associated with tobacco use. *American Journal of the Medical Sciences*, 326, 179–182.
- Górska, R. (2023). Choroby błony śluzowej jamy ustnej. Podręcznik dla studentów i do LDEK (pp. 102–104). Wrocław: Edra Urban & Partner.
- Kaliakatsou, F., Hodgson, T. A., Lewsey, J. D., Hegarty, A. M., Murphy, A. G., & Porter, S. R. (2002). Management of recalcitrant ulcerative oral lichen planus with topical tacrolimus. *Journal of the American Academy of Dermatology*, 46(1), 35–41.
- Patil, S., Khandelwal, S., Sinha, N., Kaswan, S., Rahman, F., & Tipu, S. (2012). Treatment modalities of oral lichen planus: An update. *Journal of Oral Diagnosis*, 1(2), 47–52.
- Petkowicz, B., Skiba, M., Tomaszewski, T., & Wysokinska-Miszczuk, J. (2004). Leukoplakia błony śluzowej jamy ustnej w aspekcie klinicznym i epidemiologicznym – analiza przypadków. *Dental and Medical Problems*, 41(4), 635–641.
- Axell, T. (1987). Occurrence of leukoplakia and some other oral white lesions among 20,333 adult Swedish people. *Community Dentistry and Oral Epidemiology*, 15, 46–51.
- Waškowska, J., & Koszowski, R. (2006). Ocena wyników leczenia kriochirurgicznego leukoplakii błony śluzowej jamy ustnej w materiale Katedry i Zakładu Chirurgii Stomatologicznej ŚAM. *Czasopismo Stomatologiczne*, 59(6), 438–445.
- Banoczy, J., Gintner, Z., & Dombi, C. (2001). Tobacco use and oral leukoplakia. *Journal of Dental Education*, 65, 322–327.
- Lima, J. S., Pinto, D. dos S., Jr., Sousa, S. O., & Correa, L. (2012). Oral leukoplakia manifests differently in smokers and non-smokers. *Brazilian Oral Research*, 26, 543–549.
- Sarkar, R., & Rathod, G. P. (2014). Clinicopathologic assessment of Candida colonization of oral leukoplakia. *Indian Journal of Dermatology, Venereology and Leprology*, 80, 413–418.
- Yu, C. H., Lin, H. P., Cheng, S. J., Sun, A., & Chen, H. M. (2014). Cryotherapy for oral precancers and cancers. *Journal of the Formosan Medical Association*, 113, 272–277.
- Nagao, T., Warnakulasuriya, S., Nakamura, T., et al. (2015). Treatment of oral leukoplakia with a low-dose of beta-carotene and vitamin C supplements: A randomized controlled trial. *International Journal of Cancer*, 136, 1708–1717.
- Warnakulasuriya, S., Johnson, N. W., & van der Waal, I. (2007). Nomenclature and classification of potentially malignant disorders of the oral mucosa. *Journal of Oral Pathology & Medicine*, 36(10), 575–580.

- Mortazavi, H., Baharvand, M., & Mehdipour, M. (2014). Oral potentially malignant disorders: An overview of more than 20 entities. *Journal of Dental Research, Dental Clinics, Dental Prospects*, 8(1), 6–14.
- Vedtofte, P., Holmstrup, P., Hjørting-Hansen, E., & Pindborg, J. J. (1987). Surgical treatment of premalignant lesions of the oral mucosa. *International Journal of Oral and Maxillofacial Surgery*, 16(6), 656–664.
- EU-Working Group on Tobacco and Oral Health. (1999). Tobacco and oral diseases: Report of EU Working Group. *Journal of the Irish Dental Association*, 46, 12–23.
- Bharath, T. S., Kumar, N. G., Nagaraja, A., Saraswathi, T. R., Babu, G. S., & Raju, P. R. (2015). Palatal changes of reverse smokers in a rural coastal Andhra population with review of literature. *Journal of Oral and Maxillofacial Pathology*, 19, 182–187.
- Behura, S. S., Masthan, K. M. K., & Narayanasamy, A. B. (2015). Oral mucosal lesions associated with smokers and chewers – A case-control study in Chennai population. *Journal of Clinical and Diagnostic Research*, 9, 17–22.
- Piątkowska, A., & Szymańska, J. (2004). Przykry zapach z ust – halitosis. *Zdrowie Publiczne*, 114(3), 383–387.
- Kamińska, K., Emeryk, B., & Kamińska, K. (2005). Halitosis – przykry zapach z ust – problem pacjenta i lekarza. *Nowa Stomatologia*, 4, 220–221.
- Górska, R. (2023). Choroby błony śluzowej jamy ustnej. Podręcznik dla studentów i do LDEK (pp. 49–51). Wrocław: Edra Urban & Partner.
- Słowińska, S. M., Pierzynowska, E., & Foik, T. (2000). Występowanie grzybów z rodzaju *Candida* w jamie ustnej u pacjentów z zapaleniem dziąseł i zapaleniem przyzębia. *Nowa Stomatologia*, 3, 51–54.
- Łabuś, M., Sekuła, B., Kubicka-Musiał, M., et al. (2018). Kandydoza jamy ustnej – diagnostyka i leczenie. *Twój Przegląd Stomatologiczny*, 3, 22–26.
- Górska, R. (2023). Choroby błony śluzowej jamy ustnej. Podręcznik dla studentów i do LDEK (pp. 123–127). Wrocław: Edra Urban & Partner.
- Mielnik-Błaszczak, M., Kosek, K., Hoehne, D., Ciurysek, M., & Schabowski, J. (2009). Kserostomia – problem dotyczący coraz większą populację. *Medycyna Ogólna*, 15(3), 445.
- Jańczuk, Z., & Banach, J. (1998). Choroby błony śluzowej jamy ustnej i przyzębia. Warszawa: Wydawnictwo Lekarskie PZWL.
- Maciąg, W., & Bachanek, T. (2006). Wpływ alkoholu i nikotyny na stan zdrowia jamy ustnej. *Magazyn Stomatologiczny*, 11, 28–29.
- Gurvits, G. E., & Tan, A. (2014). Black hairy tongue syndrome. *World Journal of Gastroenterology*, 20, 10845–10850.
- Stewart, B. W., & Kleihues, P. (2003). *World cancer report*. Lyon: WHO International Agency for Research on Cancer.
- Mirbod, S. M., & Ahing, S. I. (2000). Tobacco-associated lesions of the oral cavity: Part II. Malignant lesions. *Journal of the Canadian Dental Association*, 66, 308–311.
- Kolokythas, A., Schwartz, J. L., Pytynia, K. B., Panda, S., Yao, M., et al. (2011). Analysis of RNA from brush cytology detects changes in B2M, CYP1B1 and KRT17 levels with OSCC in tobacco users. *Oral Oncology*, 47, 532–536.
- Ho, M. W., Risk, J. M., Woolgar, J. A., Field, E. A., Field, J. K., et al. (2012). The clinical determinants of malignant transformation in oral epithelial dysplasia. *Oral Oncology*, 48, 969–976.
- Brzeziński, P. (2010). Nikotynowe zapalenie jamy ustnej u żołnierzy. *Forum Medycyny Rodzinnej*, 4(5), 319–322.

- Jaworska-Zaremba, M., Domagała, J., Łomżyński, Ł., & Trębacki, J. (2007). Wpływ palenia tytoniu na proces osteointegracji wszczepów stomatologicznych. *Nowa Stomatologia*, 2/3, 91–94.
- Szpak, P., Szymańska, J., & Koszuta, A. (2012). Niepowodzenia w leczeniu implantoprotetycznym związane z paleniem tytoniu. *Zdrowie Publiczne*, 122(3), 310–315.
- Koszuta, A., Szymańska, J., & Szpak, P. (2012). Leczenie protetyczne z zastosowaniem implantów zębowych. *Zdrowie Publiczne*, 122(2), 217–221.
- Moś, D., & Ogłodek, E. (2009). Ocena wskaźnika wypływu płynu dziąsłowego u kobiet palących nikotynę. *Nowa Stomatologia*, 4, 128–131.
- Kowalski, J., & Dragan, M. (2006). Wpływ czynników miejscowych i ogólnoustrojowych na stan kliniczny tkanek przyzębia. Opis dwóch przypadków. *Nowa Stomatologia*, 1, 42–46.
- Barczewska, E., & Nowak, A. (2000). Zaburzenia smakowe – dysgeusia. *Nowa Stomatologia*, 1–2, 3–8.
- Górska, R. (2007). *Choroby błony śluzowej jamy ustnej*. Med. Tour Press International.
- Gonul, M., Gul, U., Kaya, I., Kocak, O., Cakmak, S. K., Kilic, A., et al. (2011). Smoking, alcohol consumption and denture use in patients with oral mucosal lesions. *Journal of Dermatology Case Reports*, 4, 64–68.
- National Cancer Institute & Centers for Disease Control and Prevention. (2014). *Smokeless tobacco and public health: A global perspective* (NIH Publication No. 14-7983). U.S. Department of Health and Human Services, Centers for Disease Control and Prevention and National Institutes of Health, National Cancer Institute.
- Kallischnigg, G., Weitkunat, R., & Lee, P. N. (2008). Systematic review of the relation between smokeless tobacco and non-neoplastic oral diseases in Europe and United States. *BMC Oral Health*, 8, 13.
- Udner, M., Perriot, J., & Peiffer, G. (2012). Smokeless tobacco. *Presse Médicale*, 41(3), 3–9.
- Avon, S. L. (2004). Oral mucosal lesions associated with use of quid. *Journal of the Canadian Dental Association*, 70, 244–248.
- Auluck, A., Rosin, M. P., Zhang, L., & Sumanth, K. N. (2008). Oral submucous fibrosis, a clinically benign but potentially malignant disease: Report of 3 cases and review of the literature. *Journal of the Canadian Dental Association*, 74, 735–740.
- Sabharwal, R., Gupta, S., Kapoor, K., Puri, A., & Rajpal, K. (2013). Oral submucous fibrosis: A review. *Journal of Advanced Medical and Dental Sciences Research*, 1, 29–37.
- Arakeri, G., Rai, K. K., Boraks, G., Patil, S. G., Aljabab, A. S., Merks, M. A. W., et al. (2017). Current protocols in the management of oral submucous fibrosis: An update. *Journal of Oral Pathology & Medicine*, 46(6), 418–423.
- Gopinath, D., Lai, M. H., Veettil, S. K., Balakrishnan Nair, A., & Maharajan, M. K. (2022). Comparative efficacy of interventions for the management of oral submucous fibrosis: A systematic review and network meta-analysis. *Journal of Personalized Medicine*, 12(8), 1272.