ORAL MANIFESTATIONS OF NICOTINE POUCH USE: CURRENT KNOWLEDGE

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ABSTRACT

Nicotine dependence remains one of the most complex and widespread global public health challenges. Despite extensive prevention efforts and regulatory actions, its prevalence continues to be high. In recent years, there has been rapid development of non-combustible nicotine delivery products, including nicotine pouches (NP), which represent a newer category within the smokeless, tobacco-free market. These pouches are placed in the oral vestibule, between the lip and the gum, allowing nicotine to be gradually absorbed over approximately 30 minutes per use. However, their effects on oral health are still not well understood. This narrative review therefore aims to summarize and clarify the current evidence regarding the oral manifestations associated with NP use.

Key words: Nicotine pouches, Oral health, Narrative review, Oral lesions.

Introduction

To date, nicotine dependence is considered one of the global most complex and widespread public health problems [1]. As the main psychoactive component of tobacco, nicotine leads to pronounced physical and psychological dependence. Despite numerous preventive campaigns and regulatory measures, the prevalence of this condition in the general population remaining high [1–3]. Nicotine dependence does not represent solely an individual health problem, but also a significant social and economic burden to healthcare systems and contributes to the rise of non-communicable diseases [1].

Epidemiological studies indicate a consistently high prevalence of use of both, conventional tobacco products and modern nicotine alternatives [4].

The mechanism of action of nicotine is based on its selective binding to nicotinic cholinergic receptors in the brain, which stimulates the release of dopamine and produces a sense of pleasure. The cessation of nicotine intake disrupts the activity of the reward centre and triggers withdrawal symptoms, making this dependence particularly difficult to treat [5–7]. In addition to conventional tobacco products, nicotine is today consumed through various alternative forms.

Non-combustible nicotine-containing products have seen intensive development in recent years, with nicotine pouches (NP) emerging as a new category within the smokeless, tobacco-free product market. They appeared on the United States market in 2016, and have been available in Europe since 2018. They appeared on the market of Bosnia and Herzegovina relatively recently, with wider regional availability recorded from 2021 onwards [8, 9]. In terms of method of use and external appearance, they are similar to Swedish snus. However, unlike snus, NP do not contain tobacco leaves. They most commonly consist of a microcrystalline cellulose matrix impregnated with nicotine, which may be derived from tobacco leaves (natural nicotine) or obtained synthetically. The addition of flavourings, plant fibres, and other additives contributes to their functional characteristics and sensory properties [10].

NP are used by being placed in the vestibule (between the lip and the gum), thereby ensuring the gradual absorption of nicotine over a period of 30 minutes per use [10–12]. The absorption of nicotine from the NP depends on the pH value of the pouch, which is why pH regulators are also added to these products. The higher the pH, the greater the capacity of nicotine to be absorbed through the oral mucosa. Thus, if the pH of the NP is close to 10, nicotine can be transformed into almost 99% freebase nicotine, which is more bioavailable, can pass through the oral mucosa more rapidly, and increase the concentration of nicotine in the bloodstream [13, 14].

NPs are available in a variety of flavours (e.g. peppermint, spearmint, tobacco, liquorice, citrus, berry) and strengths (low, medium, strong, extra strong), which differ from one another in terms of their nicotine content. The concentration of nicotine in smokeless tobacco products is high. The average nicotine level in conventional cigarettes is approximately 6–12 mg per cigarette, depending on the brand. Disposable e-cigarettes contain 39–48 mg of nicotine per cartridge [15]. The nicotine level in NP ranges from 4 mg to 14 mg per pouch (depending on the strength), and an average of 3 to 10 pouches are used per day, resulting in a high daily nicotine intake [15–17].

Since their appearance on leading global markets, NP have recorded a sharp rise in sales and growing prevalence, which has raised serious public health concerns, particularly due to their frequent use among young people. Their popularity among adolescents and younger adults is significantly associated with intensive marketing strategies on social media, a wide selection of appealing flavours, as well as the possibility of discreet use in various settings (e.g. at work, in school classrooms, during training sessions, or indoors). Although they are often presented as a less harmful alternative to conventional smoking, given that they involve neither combustion nor tobacco, these products still enable nicotine intake. Exposure to nicotine in this

form may have adverse effects on the cardiovascular system, while in younger populations it may negatively impact brain development [9, 12, 18].

The oral effects of NP, however, remain insufficiently studied. Existing research and clinical observations indicate that prolonged direct contact of the NP with oral tissue may cause keratosis at the site of application, leukoplakia, and periodontal disease [6, 15, 16, 19–22]. However, the majority of available studies are limited by small sample sizes, short follow-up periods, and heterogeneity in methodological approaches, which prevents the drawing of reliable clinical conclusions. It is precisely this gap in the literature that makes the systematisation of existing evidence not only useful, but essential.

Materials and methods

The methodology of this paper was based on a systematic search of the available scientific literature, with the aim of identifying relevant studies examining the effects of NP on oral health and changes in the oral mucosa. The search was conducted in the PubMed/MEDLINE electronic database.

The search strategy encompassed a combination of Medical Subject Headings (MeSH) and free-text keywords. The following terms were used as keywords: "nicotine pouches", "oral mucosa", "smokeless tobacco“, "smokeless tobacco keratosis", "periodontitis", "gingival recession", "oral cancer" and "oral leukoplakia". Boolean operators AND and OR were used to combine search terms, enabling more precise retrieval of relevant studies.

Inclusion Criteria

Studies were considered eligible for inclusion if they were observational (cross-sectional, cohort, or case studies), systematic reviews, meta-analyses, or experimental studies examining the effects of NP on oral health. Research conducted on participants of all age groups who use NP was included. Relevant outcomes pertained to changes in the oral cavity, including changes in the oral mucosa, periodontal disease, gingival recession, leukoplakia and potentially malignant and malignant lesions. Only peer-reviewed publications written in English and published between January 2020 and January 2026 were included. Alongside contemporary sources, earlier studies providing a foundation for understanding pathophysiological mechanisms were also integrated into the methodological framework.

Exclusion Criteria

Studies that did not directly examine the effects of NP or smokeless tobacco products on oral health were excluded from the analysis, as were papers not available in full text. Duplicates, conference abstracts, letters to the editor, and studies published in languages other than English were also excluded.

Study Selection and Data Extraction

The selection process involved independent assessment of titles and abstracts by two researchers (T. A, V. P), followed by full-text analysis to determine eligibility based on the inclusion criteria. Disagreements were resolved through consensus and discussion. The initial search identified a total of 115 records. Identified records were exported from the PubMed/Medline database into the reference manager Zotero, where duplicates were removed through a combination of automated detection and manual verification. After removal of duplicates and application of inclusion and exclusion criteria, 21 studies were included in the final analysis. The remaining 13 references used in this paper refer to institutional reports, guidelines, books, and review articles included as background literature for contextualisation of the topic and explanation of pathophysiological mechanisms, and were not subject to systematic selection. The study selection flow is presented in accordance with PRISMA guidelines (Figure 1).

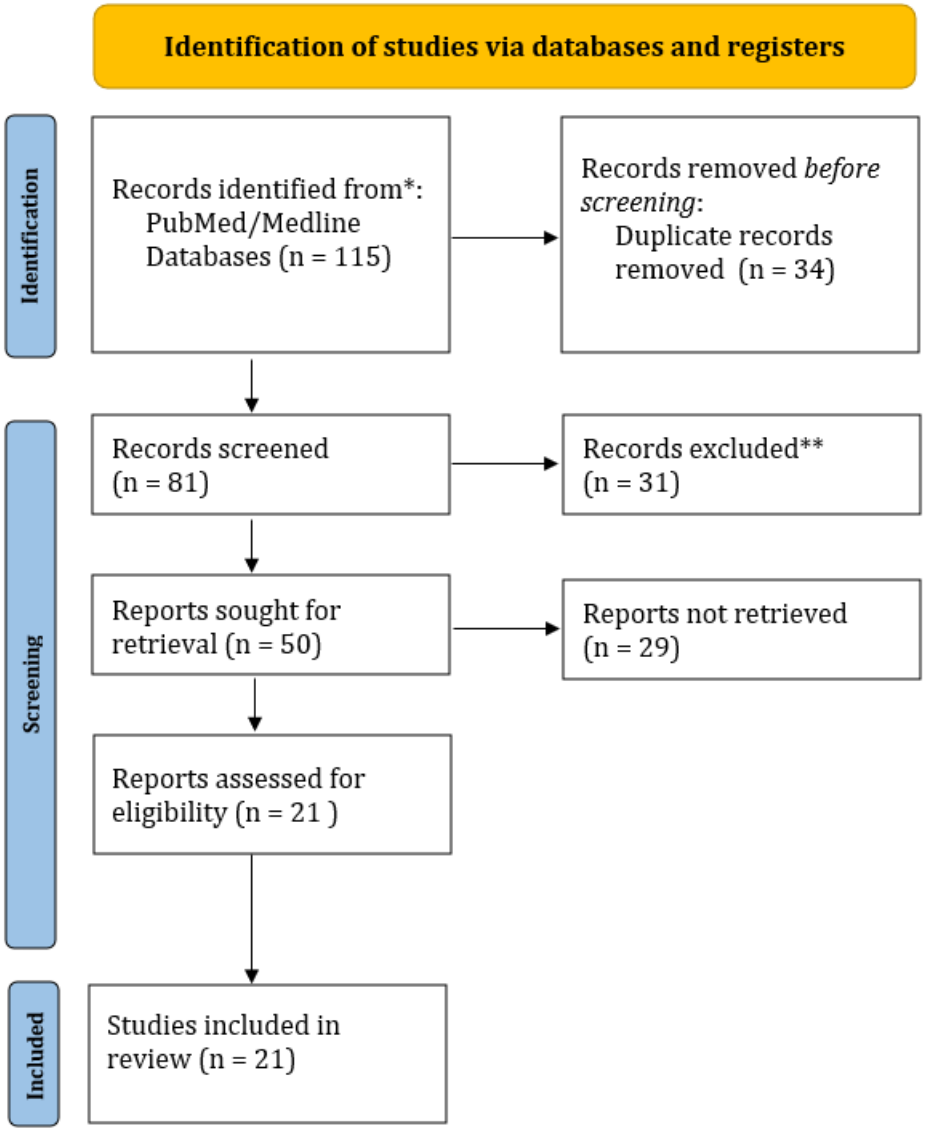

Figure 1. PRISMA flowchart for narrative review

Quality Assessment

Due to the exploratory nature of this paper and the heterogeneity in the design of the included studies, a formal assessment of the risk of bias using standardised tools was not conducted. The methodological quality of the studies was considered descriptively.

Data Synthesis

Due to heterogeneity in study design, definition of exposure, and product composition, a meta-analysis was not conducted. The results were synthesised narratively and grouped into thematic sections: changes in the oral mucosa, potentially malignant and malignant lesions, and periodontal disease.

Results and Discussion

The results of the conducted analyses indicate that the emergence of NP on the market represents a significant public health challenge, with the promoted concept of "reduced harm" frequently failing to align with data on nicotine concentration and pharmacokinetics.

Pharmacokinetics and Bioavailability

When a NP is placed in the oral cavity, between the gum and the lip, nicotine is released under the action of saliva and passes by diffusion through the oral mucosa into the bloodstream. The passage of nicotine into the bloodstream occurs primarily through the non-keratinised buccal mucosa.

Nicotine is a weak base, and its absorption depends on the pH of the surrounding environment. At higher pH values, a greater proportion of nicotine converts to its unionised, free (freebase nicotine) form, which is more readily absorbed through the oral mucosa. The process of nicotine dissolution and mucosal permeation may also be influenced by the volume of saliva produced [13]. Increased salivary secretion may enhance nicotine release, whereas excessive salivation can lead to swallowing of saliva, thereby reducing local absorption and intensifying unpleasant sensory effects such as throat irritation. Placement of the NP in an area with a greater mucosal absorption surface, such as the upper lip, may increase nicotine uptake. As a consequence of the conversion of nicotine to its free base form, users of NP are exposed to a continuous and elevated level of this alkaloid, which favours the development of pronounced physiological dependence, often more intense than that observed in conventional smokers [13, 23, 24].

NP do not, as a rule, contain classical tobacco-specific nitrosamines (TSNAs) to the extent characteristic of combustible tobacco products; however, certain toxicological risks persist. TSNAs may occur in various forms, including N-nitrosoanatabine (NAT), N-nitrosonornicotine (NNN), N-nitrosoanabasine (NAB), and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK). Mallock and colleagues identified the presence of tobacco-specific nitrosamines (TSNAs) in 26 out of a total of 44 analysed NP products, with the highest recorded concentrations of N-nitrosonornicotine (NNN) and N-nitrosamino ketone (NNK) amounting to 13 ng and 5.4 ng per pouch, respectively [8]. In addition to nicotine and TSNAs, other potentially harmful constituents were detected in certain products, including chromium and formaldehyde, further highlighting the need for a thorough safety assessment. Such a composition may have potential implications for oral and periodontal health, acting through various biological and pathophysiological mechanisms [8, 11, 16, 25].

Changes in the Oral Mucosa: Keratoses and Precancerous Lesions

The use of NP is considered a significant risk factor for the development of various white lesions in the oral cavity [26]. Certain white lesions may carry the potential for malignant transformation, which is why timely recognition and diagnosis are of critical importance in the prevention of oral cancer. There are two key reasons why the use of NP is associated with potentially harmful effects on the oral mucosa. First and foremost, their application between the lip and the gum allows prolonged and direct contact with the mucosa, which favours more frequent and extended use compared to other tobacco or nicotine products. In addition, the high nicotine content, predominantly absorbed through the oral mucosa, further increases the risk of local adverse changes [19].

Repeated application of NP in the vestibular region of the oral cavity may give rise to subjective and objective signs on the oral mucosa, including redness, tingling, itching and a burning sensation. These changes are not necessarily confined to the site of contact, but may extend to other parts of the oral cavity and even the pharynx. Furthermore, in a certain number of users, disturbances in taste perception may occur. In long-term and heavy users of NP, changes in the quantity and quality of salivary secretion may also develop, which may further affect the integrity and function of the oral mucosa [20].

One of the most common conditions associated with the use of NP is smokeless tobacco keratosis (STK). It develops as a consequence of prolonged mechanical irritation of the oral mucosa caused by the application of NP. The degree of keratinisation is determined by the frequency and duration of the habit. In the early stages, STK presents clinically as a whitish-greyish plaque with a slightly wrinkled surface. In advanced stages, pronounced hyperkeratosis occurs, accompanied by the formation of a characteristic indentation, a mucosal "pouch" [26, 27]. When the pouch is placed closer to soft, mobile tissues, such as the frenulum, the lesions take on the appearance of a white line. In other cases, the lesions appear more cloud-like, with varying intensities of white colouration.

Histopathological findings reveal cyclic parakeratosis associated with epithelial acanthosis. In some cases, signs of epithelial dysplasia and the presence of hyperchromatic nuclei in the basal layer have been recorded. Changes in the connective tissue are subtle and are mainly reflected in collagenous sclerosis around blood vessels and nerve structures. Clinical experience has shown that if a healthy individual ceases to use NP, oral lesions may resolve within a period of six weeks to six months. Should the lesion fail to resolve or change in appearance, surgical excision with biopsy is indicated [15, 26, 28].

Leukoplakia

Leukoplakia is one of the possible consequences of prolonged NP use, a potentially malignant disorder that manifests clinically as white lesions on the oral mucosa. This condition is of particular clinical significance as it is regarded as a precancerous state, given its potential for progression to squamous cell carcinoma [2]). Leukoplakia is predominantly recorded in the population over the age of 40, with epidemiological data indicating a higher prevalence among men and an approximately six-fold greater frequency among users of smokeless tobacco products compared to non-users. Prolonged mechanical pressure and continuous exposure to chemical constituents may represent predisposing factors for the development of leukoplakia, although a reliable causal relationship with NP use cannot yet be unequivocally established. Clinically, leukoplakia may encompass a spectrum of changes ranging from benign hyperkeratosis to epithelial dysplasia and carcinoma in situ, which is why histological verification is of paramount importance for adequate risk assessment and the establishment of a definitive diagnosis [20, 30]. Experimental studies demonstrate that smokeless tobacco products may increase oxidative stress, stimulate the production of nitric oxide, and lead to the suppression of tumour-protective pathways, providing a biological explanation for potential malignant transformation [21].

Oral Cavity Carcinoma

Long-term epidemiological data that would clearly define a direct association between the use of NP and the development of oral carcinoma are currently limited, primarily due to the relatively recent emergence of these products on the market and an insufficient number of studies. Existing research indicates that NP may lead to localised changes in the oral mucosa, including the appearance of white lesions and inflammatory reactions at sites of application. However, reliable evidence confirming a direct causal relationship between their use and malignant transformation has not yet been clearly established in long-term studies. In contrast, for traditional smokeless tobacco products containing tobacco-specific carcinogens, such as snus, a clear association with the development of premalignant lesions of the oral mucosa and oral carcinoma has been established across various population groups [19, 20]. The intensity and distribution of risk vary depending on the chemical composition and type of product, as well as regional patterns of consumption, including frequency, duration and method of use.

Periodontal Disease

The use of smokeless tobacco products is considered one of the significant risk factors for the development of periodontal diseases such as gingivitis, periodontitis and peri-implantitis [31].

The most widely accepted explanation for the relationship between NP and periodontal disease proceeds from the assumption that nicotine, as their principal toxic constituent, interacts with host cells and modifies the inflammatory response to microbial stimuli. Such mechanisms may result in direct or indirect damage to periodontal tissues.

Experimental *in vitro* studies have demonstrated that exposure to nicotine leads to inhibition of periodontal ligament fibroblast function and reduced stem cell viability, alongside increased production of reactive oxygen species (ROS). Nicotine also participates in the development of periodontal disease through modulation of the cytokine response, stimulating increased secretion of IL-1β and IL-8, as well as IL-6, IL-10, and IFN-γ, and prostaglandin PGE2, while simultaneously reducing the expression of MMP-2. Experimental models demonstrate that nicotine, both in healthy periodontium and in cases of periodontitis, causes

significant bone loss, particularly in furcation regions, while simultaneously reducing alveolar bone regeneration by increasing the number of osteoclasts and enhancing the expression of nuclear factor kappa-B ligand (NF-κB ligand) [22].

In addition to nicotine, the constituents of NP, including flavourings, raise concern due to their potential impact on general and oral health. More than 15 000 different flavourings are available for smokeless products, among which 2,3-hexanedione (creamy, fruity), ethyl butyrate (fruity, apple) and DL-menthol (menthol, mint) are notable examples. These flavourings may accelerate the development of physical nicotine dependence in adolescents and increase emotional attachment to NP through enhanced positive and reduced negative subjective experiences. Menthol, as a common additive, also affects the viability and proliferation of human periodontal ligament fibroblasts, inducing increased oxidative stress, pro-inflammatory reactions, and accelerated cellular senescence. Furthermore, the menthol flavouring facilitates the passage of toxic substances, such as nitrosonornicotine (NNN) and nicotine, through the buccal and sublingual mucosa, increasing the risk of soft tissue damage in the oral cavity. These effects indicate that flavouring additives in NP may represent a potential risk to the health of the periodontium and oral tissues. Flavoured nicotine products may also lead to an imbalance in the oral and periodontal microbiota, interfere with the local innate immune response and contribute to the development of periodontitis through interaction between the respiratory microbiota and the immune system [16, 32–34]. Changes in the pH value and chemical composition of saliva create a microenvironment conducive to the proliferation of anaerobic bacteria, which are aetiologically associated with the development of periodontal disease. The results of the study by Miluna-Meldere and colleagues indicate a possible association between NP use and the presence of periodontopathogenic bacteria in saliva [15]. The bacteria *Tannerella forsythia*, *Prevotella intermedia*, and *Porphyromonas gingivalis* were detected in NP users, but were absent in the control group. It was particularly noted that their presence was more pronounced in subjects with long-term and heavy use (more than ten pouches per day over several years). These findings suggest that NP may contribute to oral microbiota dysbiosis and potentially increase the risk of periodontal disease [15, 16].

The study by Alkhatib suggests a potential association between the use of NP and the occurrence of localised pathological changes in the oral cavity, such as gingival recession [21]. The changes are localised precisely at the sites where the pouches are regularly applied, pointing to a clear topographical relationship. Continuous and repeated exposure to these products may cause mechanical irritation and chemical irritation of the gingiva and oral mucosa, leading to soft tissue damage. Although the precise pathophysiological mechanism has not yet been fully elucidated, similarities can be observed with the effects of smokeless tobacco products, which have been shown to be associated with gingivitis, gingival recession, and the formation of periodontal pockets [16, 19, 21].

Conclusion

This narrative literature review systematised the available evidence on the effects of NP on oral health, with particular emphasis on changes in the oral mucosa, periodontal tissue and potential malignant risk. The results indicate that the use of NP may lead to local mucosal changes at the site of application, including keratotic white plaque-like lesions and localised gingival recession. The reversibility of some of these changes following cessation of use represents a clinically relevant finding; however, it does not preclude the need for regular monitoring. Periodontal effects remain inconsistent in the literature and are difficult to separate from the influence of confounding factors, such as the level of oral hygiene and concurrent cigarette smoking. Reliable long-term data on the malignant potential of NP are not yet available, necessitating the application of the precautionary principle in clinical practice.

Dental practitioners play a key role in the early detection of lesions, patient counselling and timely referral for biopsy in cases of persistent, indurated, or morphologically irregular lesions. Particular attention should be paid to the younger population, which is most exposed to aggressive marketing strategies on social media.

Future research should be methodologically rigorous, with long-term follow-up and clearly defined exposure criteria. At the same time, it is essential to introduce more stringent regulatory control over the composition of NP, with particular emphasis on upper limits for pH values and nicotine concentrations, in order to reduce addictive potential and minimise the risk of developing oral pathologies.

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