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ORAL AND SYSTEMIC HEALTH EFFECTS OF NICOTINE POUCHES: A NARRATIVE REVIEW WITH PUBLIC HEALTH IMPLICATIONS

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ABSTRACT

Background and Aim: Oral nicotine pouches (ONPs) have rapidly emerged as popular, non-combustible, tobacco-free nicotine delivery systems. While marketed as reduced-risk alternatives, their health impacts and epidemiological trends raise significant concerns. This narrative review critically synthesizes current evidence, regarding the localized oral and systemic health effects of ONPs and also their broader public health implications.

Findings: Locally, prolonged ONP use induces mucosal inflammation, cytotoxicity and clinically observable lesions such as gingival recession and leukoplakia, which can be exacerbated by different kind of flavors and pH adjusters. Systemically, ONPs deliver high plasma nicotine concentrations comparable to or exceeding combustible cigarettes, triggering acute cardiovascular strain, gastrointestinal distress, also posing severe acute toxicity and neurodevelopmental risks to youth. From a public health perspective, while ONPs may offer a theoretical harm-reduction pathway for established adult smokers, real-world data indicate a troubling prevalence of poly-tobacco use and a rapid surge in youth uptake. Driven by aggressive marketing and regulatory loopholes - including the use of synthetic nicotine analogs - the current trajectory of ONPs closely mirrors the early e-cigarette epidemic.

Conclusion: ONPs are not risk-free. A comprehensive, evidence-based regulatory approach, including strict product standards, flavor bans, and digital marketing restrictions, is urgently required. Independent prolonged research is essential to ensure that adult harm reduction strategies do not inadvertently catalyze a new generation of youth nicotine addiction.

KEYWORDS

Nicotine Pouches; Health; Nicotine Exposure; Oral Health; Systemic Health; Smokeless Nicotine Products

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1. Introduction

The nicotine products market has evolved over the years to attract a broad new customer base, offering discreet, non-combustible alternatives. Among these are oral nicotine pouches (ONPs), which became available on European markets in 2019, excluding Scandinavia [1]. These products are placed in the oral cavity, typically between the gums and lip, allowing nicotine to be absorbed through the oral mucosa without the need for combustion or inhalation [2]. Unlike traditional smokeless tobacco products such as snus, nicotine pouches do not contain tobacco leaf, although they may include tobacco-derived or synthetic nicotine, along with various excipients such as flavorings, fillers, and pH-modifying agents [2, 3]. The increasing use of nicotine pouches has been attributed to several factors, including their discreet mode of use, wide variety of flavors and marketing strategies that emphasize their “tobacco-free” composition and potential as reduced-risk alternatives to combustible tobacco products. Also compared to snus, nicotine pouches are legal and contain less heavy metals and other chemical compounds, which also makes them more likely to be used [2, 3, 4, 5].

Although nicotine pouches eliminate exposure to combustion-related toxicants, they are not devoid of potential health risks. Nicotine is a pharmacologically active substance with well-documented effects on the cardiovascular system, central nervous system and pathways of addiction [6]. Evidence from pharmacokinetic studies indicates that nicotine pouches can deliver clinically relevant doses of nicotine, with absorption occurring via the oral mucosa and influenced by factors such as nicotine content and formulation characteristics, including the proportion of freebase nicotine [7, 8]. Depending on these factors, systemic exposure to nicotine may approach levels observed with other nicotine delivery systems, raising important considerations regarding dependence potential and physiological effects [6].

In addition to nicotine, these products may contain other biologically active substances, including flavoring agents and trace toxicants such as metals and tobacco-specific nitrosamines, although generally at lower levels than those found in combustible tobacco products [3, 9]. Experimental and toxicological studies

suggest that nicotine pouches exhibit lower cytotoxic and genotoxic potential compared to traditional smokeless tobacco products; however, their long-term safety profile remains insufficiently characterized [9, 10]. Nicotine pouches deliver nicotine buccally with slower uptake and lower peak plasma concentrations than cigarettes, while total nicotine exposure is dose-dependent and can approach smoking levels at higher contents [11]. The presence of nicotine analogs such as 6-MN and nicotinamide (NA) is also noteworthy, with 6-MN exhibiting greater cytotoxic and inflammatory responses in bronchial models, raising regulatory concerns about undisclosed additives [12].

From a systemic perspective, nicotine exposure has been associated with acute cardiovascular effects, including increases in heart rate and blood pressure, as well as potential longer-term impacts on cardiometabolic health [3, 6]. Recent scientific statements emphasize that, while non-combustible nicotine products may reduce exposure to certain harmful constituents, they are not risk-free and their overall health impact depends on patterns of use, product characteristics, and user populations [13]. This duality reduced exposure to combustion products but continued exposure to nicotine and other chemicals complicates the assessment of their role in harm reduction strategies.

Recent epidemiological data show increasing nicotine pouch use among youth with regional variation in adult uptake and substantial dual-use with other nicotine products. In Great Britain, the United States and Poland, awareness and use metrics have shifted over the past few years [1, 4, 14]. According to an international study conducted via an online survey, the vast majority of ONP users also use other nicotine products at the same time [15]. The rise of nicotine pouch sales is also concerning, as evidenced by a rise of up to 641% in the United States between 2019 and 2022 [16].

Despite the growing prevalence of nicotine pouch use, the current evidence base remains limited and heterogeneous. Much of the available research consists of short-term clinical studies, toxicological assessments, and observational data, with a lack of long-term epidemiological studies evaluating clinically relevant health outcomes [2, 17].

In this context, nicotine pouches represent a complex and evolving issue for both clinical practice and public health. While they may offer a potentially lower-risk alternative for individuals who switch completely from combustible tobacco products, their increasing uptake among younger populations and non-smokers raises concerns regarding nicotine initiation and sustained dependence [1, 18].

2. Aim of the study, materials and methods

The aim of this narrative review is to critically synthesize current evidence on the oral and systemic health effects of nicotine pouches and to evaluate their implications for public health. The scope of the materials included scholarly articles in English and one in Czech, published in 2022–2026, searched in the PubMed database. The search terms: ”nicotine pouches” AND ”health influence”, and ”nicotine pouches” AND ”public health”. The following work addressing the health effects of oral nicotine pouches and their impact on public health, full text available. No formal quality assessment tool was applied due to the mixed nature of the sources; the assessment of credibility was based on the transparency of methods and the reputation of the sources.

3. Literature review

3.1. Oral health influence of nicotine pouches

Because oral nicotine pouches are placed directly in the vestibule, the oral cavity is the primary site of both chemical exposure and pharmacological absorption. While these products are heavily marketed as safer, non-combustible alternatives, their localized impact on the oral microenvironment warrants significant clinical concern. The prolonged and repeated contact of the oral mucosa with nicotine, pH adjusters, and flavorings can lead to physical irritation, local pH alterations and disruption of the oral microbiome [19, 20].

The most immediate consequences of ONP use are observed on the mucosal soft tissues.

A high prevalence of adverse oral events has been documented with nearly half of users reporting mouth lesions as well symptoms of soreness and irritation [15]. These self-reported symptoms align with emerging clinical observations. Recent case reports highlight a direct, specific relationship between habitual ONP placement and the development of localized gingival recession and leukoplakia in young, otherwise healthy individuals [21].

These macro-level clinical findings are strongly supported by histopathological analyses. Miluna-Meldere et al. (2024) demonstrated that ONP use induces white, homogeneous lesions at the placement site, characterized microscopically by parakeratosis, acanthosis, and subepithelial chronic inflammatory infiltration

consisting of lymphocytes and macrophages [22]. Such tissue degradation is likely the result of both mechanical trauma and chemical toxicity, mirroring the well-documented localized gingival recession associated with traditional snus use, yet occurring even without the presence of tobacco leaf [23].

To understand the etiology of these mucosal lesions, several authors have investigated the molecular and cellular mechanisms triggered by ONPs. In vitro studies on human gingival epithelial cells reveal that ONP extracts induce significant cytotoxicity, generate reactive oxygen species (ROS) and promote apoptosis [10, 24]. This oxidative stress leads to a robust pro-inflammatory response. Shaikh et al. (2022) reported elevated release of interleukins (IL-6, IL-8) and tumor necrosis factor-alpha (TNF-α) in epithelial cells exposed to ONPs [10].

Crucially, this in vitro mechanism is corroborated by clinical biochemical analyses. Miluna et al. (2022) found that users of snus and ONPs exhibited the highest salivary concentrations of IL-1, IL-6, IL-8 and TNF-α compared to both smokers and non-tobacco users [25]. Moreover, Miluna (2022) established a statistically significant correlation between elevated salivary IL-6 levels and the clinical presence of localized white mucosal lesions, providing a direct link between ONP-induced molecular inflammation and observable tissue damage [25].

The toxicity of ONPs is heavily modulated by their flavoring additives. Flavorings, particularly fruit and menthol/mint varieties, have been shown to exacerbate cytotoxicity and impair the innate immune system of the periodontium [10, 22]. Furthermore, menthol additives not only increase oxidative stress but also facilitate the mucosal penetration of toxic chemicals, including trace tobacco-specific nitrosamines (TSNAs) like NNN, which are still detectable in some ONP formulations [19, 24].

It can be assumed that the development of oral cancer in connection with the use of oral nicotine pouches is a matter of time, but so far, due to their short time on the market, there are no studies that could confirm this [22].

Beyond soft tissue damage, the extreme concentrations of nicotine delivered by certain ONPs - sometimes up to ten times higher than conventional cigarettes - pose a severe threat to the deeper periodontium [26]. Nicotine locally upregulates the expression of osteoclastogenesis factors such as RANKL and TNF-α, which can lead to alveolar bone loss and severe periodontal disease [24]. Nevertheless, far-reaching research is necessary to confirm this effect.

However, a distinct contrast emerges when it comes to evaluate the impact of ONPs on dental hard tissues. While ONPs are highly detrimental to the periodontium, their cariogenic risk appears relatively low. Because ONPs utilize artificial sweeteners (such as maltitol) instead of fermentable sugars, they do not provide a substrate for acid-producing bacteria in the same manner as traditional sweets [23]. In a clinical crossover study, Alizadehgharib et al. (2026) demonstrated that usage of ONPs actually decreased dental plaque acidogenicity over time, maintaining a favorable pH environment that lowers the risk of enamel demineralization, even despite an observed increase in Streptococcus mutans populations. This fact could have a positive effect on reducing the development of caries [27].

3.2. Systemic health effects of nicotine pouches

Beyond the localized impact on the oral microenvironment, the rapid absorption of nicotine from oral nicotine pouches (ONPs) through the mucosal capillaries precipitates a profound systemic physiological response. Because nicotine accounts for the vast majority of health effects, these adverse effects are likely to be largely similar to those observed with the use of other nicotine-containing products. The systemic bioavailability of ONPs is remarkably high. Clinical assessments indicate that the nicotine extraction rate during a typical 60-minute application reaches approximately 50% of the product's total nicotine content [3]. Consequently, high-dose ONPs can achieve maximum plasma nicotine concentrations that are statistically equivalent to or even exceed those produced by traditional smokeless tobacco (SLT) and combustible cigarettes [8]. The cumulative systemic nicotine exposure can exceed 6800 μg/kg of body weight per day, vastly surpassing established safety thresholds [3].

The cardiovascular system is particularly vulnerable to this massive influx of systemic nicotine. The primary acute cardiovascular effect of ONPs stems from the intense activation of the sympathetic nervous system, leading to the rapid release of catecholamines (epinephrine and norepinephrine) [14]. This triggers immediate dose-dependent increases in heart rate, blood pressure and myocardial contractility, simultaneously inducing endothelial dysfunction and arterial stiffness [3, 14].

While the acute hemodynamic effects are well-documented, the long-term cardiovascular consequences of ONPs must currently be extrapolated from epidemiological data on traditional snus. Chronic sympathetic

activation and persistent resting tachycardia are established risk factors for heart failure and cardiovascular mortality [3]. Besides, nicotine's potential to induce sustained endothelial dysfunction, oxidative stress and adverse lipid profile alterations suggests a plausible role in accelerating atherogenesis. In the end, this could lead to an increased risk of arrhythmias, development of hypertension and also contribute to remodeling of the heart [14]. Pooled analyses of Swedish cohorts demonstrate that while SLT use may not directly initiate acute myocardial infarction (MI), it is significantly associated with increased short-term mortality following an MI or stroke, likely due to the arrhythmogenic effects of nicotine during recurrent acute ischemia [13].

In addition to cardiovascular strain, the systemic absorption of nicotine and the inevitable ingestion of nicotine-infused saliva significantly affect the gastrointestinal (GI) tract and the central nervous system. A large-scale cross-sectional study revealed an exceptionally high prevalence of adverse GI events with 80.8% of ONP users reporting symptoms such as bloating, nausea and heartburn [28]. Furthermore, the potent psychoactive nature of systemic nicotine carries notable psychological implications. Al-Nafisi et al. (2025) reported that nearly 40% of ONP users experience mild to moderate psychological symptoms, including anxiety and distress, which were found to be inversely correlated with socioeconomic status and education levels [28].

The extreme nicotine concentrations in modern ONPs have also introduced a severe risk of acute poisoning, particularly among nicotine-naive individuals and minors. Gketsi et al. (2026) documented cases of severe acute nicotine poisoning in pediatric patients (aged 13 and 15) following ONP use [29]. The clinical manifestations - including severe dizziness, vomiting, and loss of consciousness - occurred rapidly, with serum nicotine levels reaching up to 266 ng/ml [29]. These incidents highlight a dangerous variability in individual sensitivity and challenge outdated toxicological models, emphasizing that while the actual lethal dose of nicotine may be higher than historically presumed (500–1000 mg), the threshold for severe acute toxicity is easily breached by modern ONPs [29].

Finally, the systemic exposure to high-dose nicotine poses a critical threat to adolescent neurodevelopment. Adolescence is a period of significant remodeling in the prefrontal cortex and limbic system and nicotine exposure during this neuroplastic window disrupts normal developmental trajectories, altering dopaminergic and serotonergic pathways. This disruption not only creates a heightened, lasting vulnerability to addiction but could also lead to long-term cognitive deficits, impaired impulse control and an increased susceptibility to anxiety and depression in adulthood [14].

3.3. Public health implications

The rapid increase in the use of oral nicotine pouches (ONPs) has sparked a lot of discussion within the global public health community, centering on the tension between adult harm reduction and youth addiction. From a toxicological perspective, ONPs possess a significantly reduced toxicant profile compared to combustible cigarettes and traditional smokeless tobacco (SLT) [30]. Clinical pharmacokinetic studies demonstrate that ONPs can deliver appreciable levels of plasma nicotine capable of suppressing tobacco-related withdrawal symptoms and cravings, particularly among adult SLT users [7]. Consequently, for adult smokers or SLT users who are unable or unwilling to quit, ONPs are frequently marketed as a less harmful, non-combustible alternative that may facilitate product switching [31, 32]. However, under the population-level public health standard applied by regulatory bodies like the Food and Drug Administration (FDA), any theoretical cessation benefits for adults must be rigorously weighed against the substantial risk of fostering nicotine addiction among nicotine-naive individuals, particularly youth [2].

Recent epidemiological data suggest that the intended harm-reduction substitution is frequently failing in real-world settings. In the United States, awareness and experimentation have surged rapidly; between 2020 and 2021, awareness among adult tobacco users jumped noticeably, with nearly half reporting familiarity and 16.4% reporting ever-use [33]. Crucially, Sparrock et al. (2023) observed that current users of cigarettes, e-cigarettes and traditional SLT are the most likely to initiate ONP use [33]. Similarly, European data from Poland reveal that the highest risk of initiation is among young adults and current e-cigarette users [4]. This indicates a troubling trend of poly-tobacco consumption rather than complete substitution, which may ultimately deepen overall nicotine dependence rather than reduce population-level health risks.

Public health experts warn that now is a critical inflection point. As Zamarippa (2025) astutely notes, the current trajectory of ONPs is highly reminiscent of e-cigarettes circa 2010–2013 [2]. Driven by aggressive marketing, characterizing flavors (e.g., mint, fruit), and implicit "tobacco-free" health claims, ONPs are highly appealing to youth. In the US, certain brands like ZYN command nearly 68.7% of the youth market, fueled by social media campaigns and endorsements from influencers [34].

What is more, manufacturers actively exploit significant regulatory loopholes. For instance, because ONPs were not included in the US Comprehensive Smokeless Tobacco Health Education Act of 1986, they uniquely evade federal bans on television and radio advertising, allowing for pervasive promotion [33]. The industry is also utilizing synthetic cooling agents - which reduce the harshness of nicotine without being classified as characterizing flavors and novel nicotine analogs to bypass existing FDA tobacco regulations entirely [13]. The discreet, spit-free nature of ONPs has further led to their proliferation in traditionally tobacco-free environments, normalizing nicotine consumption for younger generations.

Consequently, the global regulatory landscape for ONPs remains highly fragmented. While the US FDA authorized select ONP products for market in 2025, a vast array of unauthorized brands continues to proliferate [34]. Conversely, other nations have adopted far more restrictive stances - France for example, implemented a nationwide decree in 2025 banning the production, distribution and usage of all ONPs [34].

To prevent a repetition of the e-cigarette epidemic, major health organizations advocate for a multiple, evidence-based public health response [14]. Key recommendations include establishing strict product standards like maximum nicotine concentrations (escpecially to avoid nicotine poisoning) and pH regulations to limit abuse liability, implementing comprehensive flavor bans, introducing plain packaging and restricting digital marketing [5, 13, 14, 34]. Additionally, healthcare professionals must begin routinely screening adolescents for ONP use and offering evidence-based cessation support [14]. Ultimately, regulatory actions must be guided by independent, non-industry-funded research to ensure that ONPs do not become another catalyst for a youth nicotine epidemic [2].

4. Conclusion

Oral nicotine pouches (ONPs) present a complex challenge to modern clinical practice and public health. While they eliminate exposure to combustion-related toxicants and theoretically offer a harm-reduction pathway for adult smokers, the current body of evidence unequivocally demonstrates that these products are not benign.

Clinically, the prolonged mucosal exposure to high-dose nicotine, pH adjusters and characterizing flavorings induces significant localized inflammation, cytotoxicity and tissue degradation, frequently manifesting as gingival recession and leukoplakia. Systemically, the rapid and highly efficient absorption of nicotine triggers robust sympathetic activation, posing notable cardiovascular risks, gastrointestinal distress and psychological side effects. Also the extreme nicotine concentrations found in modern ONPs introduce a severe risk of acute toxicity and threaten adolescent neurodevelopment.

From a public health perspective, the narrative of ONPs as an exclusive cessation tool is heavily compromised by real-world epidemiological trends. The alarming growth in youth uptake, driven by aggressive digital marketing, influencer endorsements and the appeal of diverse flavors, closely mirrors the early trajectory like the one of the e-cigarette epidemic. Additionally, the high prevalence of nicotine products co-use among current consumers suggests that ONPs often suplement, rather than replace another products, thereby deepening overall nicotine dependence.

Moving forward, it is crucial that global regulatory frameworks adapt rapidly to close existing legislative loopholes, such as the unrestricted use of synthetic nicotine analogs and targeted media advertising. Policymakers must prioritize stringent product standards, especially maximum nicotine limits. Finally, independent, non-industry-funded longitudinal research is urgently required to fully clarify the long-term health impacts of ONPs. Only through robust, evidence-based regulation can the public health community ensure that efforts to reduce adult smoking do not inadvertently catalyze a new generation of nicotine addiction.

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