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(REVIEW ARTICLE)

The correlation between smoking habits and sensitivity and taste function disorders: A literature review

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Abstract

Introduction: Smoking habits can cause various health problems. Smoking can have a direct or indirect negative impact on taste function, and the severity of the impact depends on the level of exposure to smoking. Smoking causes benign and sometimes malignant changes in the oral cavity which is the first part of the body exposed to cigarette smoke and its harmful substances.

Method: Descriptive research method literature review. The research includes articles published from 2016 to 2023 using databases in the form of Google Scholar, Pubmed, and Science direct which discuss the "Correlation of Smoking Habits to Taste Sensitivity".

Results: Smoking can cause changes in the taste buds and vascularization of the fungiform papillae, reducing the ability to taste. As the amount and duration of smoking increases, a direct proportional increase in EGM taste threshold is seen due to a reduced number of fungiform papillae on the dorsum of the tongue in smokers. In addition, nicotine from cigarettes can cause vasoconstriction and hypoxia, thereby reducing taste sensitivity while increasing systolic blood pressure.

Conclusion: Smoking has a negative effect in the form of decreased taste and dysfunction, the severity of the impact depends on the level of exposure to smoking. This situation is caused by a decrease in the number of papillae and the number of taste buds per papilla.

Keywords: Smoking; Taste sensitivity; Taste impairment; EGM; Taste buds

1. Introduction

Currently, smoking is considered a public health issue due to its high prevalence and the mortality rate associated with tobacco-related diseases. Tobacco addiction exposes smokers to 4,720 toxic substances in tobacco smoke, 60 of which are carcinogenic and proven harmful to individual health. When the senses of smell and taste are exposed to these substances, they may suffer reversible or permanent damage. The severity of this damage is related to the duration of exposure, concentration, and tobacco toxicity [1]. Tobacco use, such as smoking, can create both psychological and physiological addiction among users. Nicotine, the active ingredient in tobacco, is highly addictive and can result in prolonged tobacco use [2].

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Indonesia remains one of the most serious tobacco use epidemics in the world. According to the 2019 National Socioeconomic Survey (SUSENAS), the adult smoking prevalence in 2019 was 32.8%, with the prevalence among adult males reaching 64.5%. The smoking prevalence among children also increased, with the percentage of children aged 10–18 who had ever smoked rising from 7.2% in 2013 to 9.1% in 2018. Around 51.3% of all adults and 66.2% of young people aged 13–15 years are regularly exposed to secondhand smoke [3].

Tobacco control efforts in Indonesia are still suboptimal, exacerbated by the lack of a national response framework. The WHO Framework Convention on Tobacco Control (FCTC) is an international treaty offering a regulatory framework for tobacco control. According to WHO FTCT data, Indonesia is the only country in the Asia-Pacific region that has not yet ratified the FCTC. In 2013, 192 WHO member countries agreed to the FCTC treaty, which has been in force since 2005 and is viewed as a critical protocol for enforcing tobacco control regulations. Comparing Indonesia to other countries with similar economies that have ratified the FCTC reveals a significant difference in smoking prevalence, even after the FCTC was implemented [4]

Taste dysfunction results from changes in the shape, quantity, and vascularization of taste buds, which can be caused by tobacco consumption [1]. Taste perception involves the binding of taste molecules to taste receptors in the oral cavity and the initiation of taste transduction pathways, which convey taste signals to the central nervous system via several nerves. Damage along this pathway can alter taste perception [5].

Smoking can have both direct and indirect negative effects on taste function, with the severity depending on the level of exposure to cigarette smoke. Greater exposure to nicotine and cigarette smoke has been shown to reduce the number of taste-related anatomical structures (taste buds) in both animals and humans. Smoking is also associated with various risk factors for taste dysfunction [5]. Smoking negatively affects the oral cavity, similar to other parts of the body. The first part of the body to be affected by exposure to cigarette smoke and its harmful substances is the oral cavity. Smoking causes benign and sometimes malignant changes in the oral cavity. Other side effects of smoking on the oral cavity include hairy tongue, leukoedema, melanin pigmentation, tooth discoloration, cervical caries, periodontal disease, reduced taste sensation, halitosis, nicotinic stomatitis, xerostomia, Candida infections, premalignant lesions, and oral cancer [6].

Several studies have shown that chronic smokers have relatively lower sensitivity to certain tastes compared to non-smokers. Regional testing using electrogustometry has shown that smokers have a higher taste threshold. Based on intervention studies, smoking and taste function showed that quitting smoking for 2 to 9 weeks was associated with increased taste threshold and perceived bitterness, indicating that smoking not only damages taste but that former smokers can experience improvements in taste over time [5]

2. Methods

The method we used is a descriptive literature review. A descriptive literature review is one of the methods employed as the basis for evaluating the writing of this research's findings. This method is used to briefly identify and explain the material obtained from several journals discussing the "Correlation Between Smoking Habits and Taste Sensitivity." The research includes articles published from 2016 to 2023 using the following databases: Google Scholar, Pubmed, and Science Direct. The chosen search terms are based on several keywords that serve as references, such as Smoking, Taste Sensitivity, Taste Disorders, and combinations of related terms.

3. Results and discussion

3.1. Taste Nerve System

Compared to non-smokers, both regular and heavy smokers experience higher rates of respiratory infections and an increased risk of taste disorders. Chronic smoking can also reduce salivary flow and cause xerostomia, leading to downstream effects on taste and oral sensation. Several cranial nerves interact to maintain taste function throughout the oral cavity, including the chorda tympani branch of the facial nerve (cranial nerve VII), which innervates the tip of the tongue, the glossopharyngeal nerve (cranial nerve IX), which innervates the posterior and lateral sides of the tongue, and the superior laryngeal branch of the vagus nerve (cranial nerve X), which innervates taste from the throat. Due to redundancy in the peripheral innervation of the taste system, total loss of taste (ageusia) or severe loss (hypogeusia) is rare, and regional taste loss throughout the mouth often goes unnoticed during eating and swallowing. However, regional taste loss at the tip of the tongue (resulting from viral damage or other environmental harm to the chorda tympani branch of cranial nerve VII) may block input from other taste nerves, leading to intense whole-mouth

sensations or phantom taste sensations (dysgeusia). Previous studies have shown that reduced taste intensity at the tip of the tongue—whether assessed directly or relative to whole-mouth sensation—can be linked to important dietary and health outcomes. For example, reduced bitterness at the tongue's tip is associated with poorer diet quality, while lower bitter sensations at the tongue's tip relative to the whole mouth are linked to increased central adiposity. Given the various implications of smoking on taste and health, research is needed to comprehensively evaluate regional and whole-mouth taste intensity among individuals with varying levels of smoking exposure [5].

3.2. Nicotine Content in Cigarettes

The addiction caused by nicotine in cigarette smoke is the fundamental reason why individuals tend to continue smoking. Prolonged exposure to nicotine significantly reduces amiloride-sensitive sodium currents in type A cells, as these currents are mediated by epithelial sodium channels, which serve as salt receptors in taste cells. In addition, prolonged nicotine exposure reduces the number of three types of taste cells, resulting in smaller mushroom-shaped taste buds. It has also been theorized that nicotine affects taste by altering neuronal responses in the nucleus of the solitary tract, the primary central relay in the gustatory pathway from the taste buds on the tongue. Other studies have reported that nicotine influences taste by activating nicotinic acetylcholine receptors manifested in the taste buds [7].

Evidence suggests that smoking can cause changes in taste buds and vascularization of the fungiform papillae, reducing the ability to taste. While the scientific literature shows inconsistent associations between nicotine product use and taste dysfunction, tobacco smoking and inhaling smoke have been linked to an increased risk of respiratory infections and dental problems, which can affect one's ability to taste. However, taste can play a compensatory role by introducing chemosensory sensations such as heat, tingling, burning, or cooling. Cigarettes and other tobacco products contain bitter compounds like nicotine, which contribute to tobacco's chemosensory properties, and genetic variants of bitter taste receptors have been shown to be associated with smoking status, depending on the population studied [8]

3.3. Effects of Smoking on Electrogustometry Thresholds

Electrogustometry (EGM) is a simple test used to assess taste function, introduced as a clinical assessment of taste sensitivity in the 1950s. It has good reliability. Compared to chemical solution-based tests, EGM is an efficient clinical tool used in evaluating taste disorders. However, few experimental studies provide data on the effects of smoke on the number of fungiform papillae. Therefore, the effects of smoking on the density of fungiform papillae and EGM thresholds were evaluated [9].

As the number and duration of smoking increased, a direct proportional increase in the EGM taste threshold was observed. The reason behind these findings is the reduced number of fungiform papillae on the dorsum of the tongue in smokers. Endoscopic contact findings of smokers' tongues revealed morphological changes in fungiform papillae and changes in vascular supply, leading to higher EGM thresholds. Previous researchers compared taste thresholds in smokers and non-smokers using EGM and reported a significant increase in taste thresholds with age in both groups. This was attributed to a decrease in the number of papillae and taste buds per papilla [9].

Since ancient times, nicotine has been linked to changes in gustatory function under chronic exposure conditions. It has been found that EGM thresholds increase with age, starting around 60 years old in areas innervated by the chorda tympani and glossopharyngeal nerves, and beginning around age 70 in areas innervated by the greater petrosal nerve. Previous research has illustrated differences in EGM thresholds, the morphology, and vascularization of fungiform papillae (fPap) between smokers and non-smokers. Earlier studies demonstrated variations in EGM thresholds among smokers before and after quitting. Although EGM thresholds decreased after quitting smoking, the morphology and vascularization of fPap appear to be influenced by long-term nicotine exposure [10].

3.4. Effects of Smoking on the Number of Fungiform Taste Buds

Research by Khan et al. (2016) showed that the more cigarettes smoked, the fewer fungiform papillae were present. There was also a significant reduction in the density of fungiform papillae in smokers compared to non-smokers. However, in similar studies on smokers and non-smokers, the number of papillae in non-smokers was reported to be 27 ± 7.6; 26±6 fungiform papillae/cm^2, and in smokers, it was 25.7±4.2; 24.9 ± 5.9 fungiform papillae/cm^2. These findings suggest that nicotine has long been associated with gustatory changes under chronic exposure conditions.

3.5. Effects of Smoking on Salt Taste Sensitivity

According to a study by Tjahajawati et al. (2020), data showed that higher salt taste thresholds correlated with higher systolic blood pressure. Smoking was found to be a key factor influencing this correlation. Studies have established that salt intake is the most significant cause of increased blood pressure, and nicotine from cigarettes can cause

vasoconstriction and hypoxia, reducing taste sensitivity while increasing systolic and diastolic blood pressure. Thus, systolic blood pressure may be an early predictor of decreased salt taste sensitivity.

The study by Ma & Lee (2022) confirmed a link between smoking and preference for salty tastes. Adjusted Odds Ratios (AOR) for daily smokers were higher for salt intake than for occasional smokers, with former smokers having the lowest AOR. Previous research also found that some former smokers partially regained their ability to taste salt after quitting smoking, and differences in taste perception may be due to the frequency and quantity of smoking.

In a cohort study conducted in France, a large web-based observation also reported a correlation between smoking and salt taste preference. However, the link remains unclear as previous studies failed to identify a connection between smoking and excessive sodium intake. In Korea, smokers who consumed alcohol showed a significant relationship with excessive sodium intake [7].

4. Conclusion

Based on sources from several studied journals, there is a strong correlation between smoking habits and sensitivity disorders in taste function. Smoking has negative effects, including a reduction and dysfunction in taste, with the severity of these impacts depending on the level of exposure to smoking. This condition is caused by a decrease in the number of papillae and taste buds per papilla.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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