

# The estimation of the viable count of mutans streptococcus in waterpipe smokers and cigarette smokers

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## ABSTRACT

**Background:** Waterpipe and cigarette are two products of tobacco consumption; tobacco use has detrimental effects on the oral cavity. Numerous studies around the world have reported a significant relationship between smoking and increase dental caries and viable count of cariogenic bacteria,

**Materials and Methods:** unstimulated saliva was collected from 84 subjects and divided equally into three groups: waterpipe smokers, cigarette smokers, and non-smokers. All of the participants are adult male aged between 25-60 years; dental caries was measured by use DMFT index, while *S.mutans* and *S.sobrinus* were isolated by using a selective medium SB 20M (Sugar bacitracin-20 modified) agar

**Results:** The present study showed a significant ( $p \leq 0.01$ ) higher DMFT, DT, MT, and FT among cigarette smokers group than both waterpipe smokers and non-smokers groups. The viable count of *S mutans* was significantly ( $p \leq 0.01$ ) higher in the cigarette smokers group followed by the waterpipe smokers group and then the non-smoker's group, while the viable count of *S sobrinus* showed no statistical differences ( $P > 0.05$ ) between groups. The correlation of DMFT with *S.mutans*, and *S sobrinus* count were significantly positive ( $p \leq 0.05$ ) in the cigarette smokers group only.

**Conclusion:** Dental caries increase in cigarette smokers, where the DMFT and *S mutans* viable count are less affected by waterpipe than cigarette smoking. A positive correlation is found between DMFT and mutans streptococcus count only in cigarette smokers

**Keywords:** Waterpipe, tobacco, *S.mutans*, *S sobrinus*, SB 20M. (Received: 9/7/2021, Accepted: 8/8/2021)

## INTRODUCTION

Tobacco is a product that grows commercially in many countries. Studies suggest that tobacco's first use was by Maya people in the center of America in the first centuries BC. With the migration of Maya to the Mississippi Valley started spreading to the south of America. Native Americans afterward, Portuguese and Spanish navigators served to spread various kinds of tobacco to be used throughout the world,<sup>(1,2)</sup> Another level of tobacco use started when cigarette had been invented; it fired this dramatic increase in tobacco using<sup>(3)</sup>. There is a difference form of tobacco consuming either smoking like waterpipe, cigarette, non-smoking chewing tobacco like snuff /naswar (roasted and finely powdered for inhalation<sup>(1)</sup>).

One of the most common use and most favored product of tobacco is cigarette, more than six trillion cigarettes are produced annually and about one billion smokers consume these products in the world<sup>(4)</sup>.

Another type of tobacco consumption is waterpipe which has different designs according to regional and cultural reasons; it also has different names, narghile in east Mediterranean countries like Turkey and Syria, shisha in Egypt, and hookah in India<sup>(5)</sup>. Recently waterpipe tobacco use is spreading rapidly worldwide, with reports of more youth being waterpipe users compared to adults. In many areas of the world<sup>(6)</sup>, the highest prevalence of smoking waterpipe is in Arab countries of Africa, the eastern Mediterranean plus Southeast Asia and rises in European countries. It appears higher among youth than adults<sup>(7,8)</sup>.

In the middle of the twenty century, widely read and admitted scientific reports decided that smoking is the major cause of lung cancer<sup>(9)</sup>. Evidence exhibits that the use of tobacco in all its types substantially raises the risk of premature death from many chronic diseases<sup>(10)</sup>.

Smoking has many adverse effects on oral health in addition to the contribution in the development of lung cancer and cardiovascular disease. There is plenty of evidence that it has a strong impact on the mouth; it may develop some oral diseases such as periodontal disease, dental caries, oral candidosis, implant failure, oral precancer, and cancer<sup>(11)</sup>.

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Dental caries or decay can be defined as the most widely distributed infectious, chronic and multifactorial disease. It is the result of interactions over time between oral bacteria that produce acid, a substrate, that the bacteria can metabolize, and many host factors that include teeth and saliva<sup>(12)</sup>.

The mechanism of action of the caries process happens by the production of a weak acid by cariogenic bacteria such as *Streptococcus* and *Lactobacillus*, as a result of the fermentation process of carbohydrate which leads to a decrease in local pH below the critical value and demineralization of the tooth surface<sup>(13,14)</sup>.

Studies have strong evidence that emphasizes this significant association between the degree of caries activity and the salivary levels of *Streptococcus mutans*<sup>(15,16)</sup>.

*S. mutans* is considered as the main component of the oral microbiota and one of the key elements of the dental plaque.<sup>(17)</sup> Although *S.sobrinus* is a minor component, *S.mutans* usually predominates; but both are causative pathogens of dental caries and strongly implicated in plaque or oral biofilm formation<sup>(18)</sup>.

Mutans streptococci detection is typically dependent on selective media, growth on Mitis Salivarius Agar (MS), colony morphology, and Biochemical features<sup>(19)</sup>.

The SB-20M medium is also a selective medium that is efficient for the identification and direct morphological recognition of *S mutans*, *S.sobrinus*<sup>(20)</sup>.

In addition to their hazards to general health, tobacco use has detrimental effects on the oral cavity both soft and hard tissue, about half a million oral cancer recorded in 2002 around the world with tobacco as the main cause. A clear relationship was observed with periodontal disease, and also it is associated with the increase of dental caries and is considered as a risk factor<sup>(21)</sup>.

Numerous epidemiological reports all around the world have recorded a near association between smoking and dental caries. In Portugal, a study confirms smoking as a risk factor for tooth decay. Further, avoiding exposure to smoking leads to a 7% reduction in caries occurrence.<sup>(22)</sup> Research in Scotland found that if a pregnant woman smokes cigarettes, her infant can have a higher prevalence of caries than a child born to a non-smoking mother<sup>(23)</sup>.

Microbial analysis of saliva showed that there was a substantial increase in the microbial load of *S.mutans* among tobacco users which might increase

the caries severity; however, the association between smoking and *S.mutans* growth is controversial. Some research has revealed a positive relationship between smoking and *S.mutans* growth as nicotine in tobacco has been shown to enhance the adhesion of *S.mutans* to the acquired pellicle and increase biofilm formation and thus increase the incidence and severity of dental caries.<sup>(24,25)</sup>

On the other hand, one recent study shows that tobacco consumers had fewer caries than healthy adults, which was contrary to several previous studies.<sup>(26)</sup> Supposing that smoking tobacco helps to reduce caries, this was supported by the presence of a higher concentration of Thiocyanate (SCN). In the blood and saliva of the smoker, Thiocyanate is a constituent of tobacco smoke which has caries inhibiting effect<sup>(27,28)</sup>.

## MATERIALS AND METHODS

This comparative study consisted of 84 males aged between 25 to 60 years old. The study consisted of 56 smokers subjects divided into waterpipe smokers group<sup>(28)</sup>, and cigarette smokers group<sup>(28)</sup>, and 28 non-smoker subjects as a control group. The study included subjects who smoke more than five times weekly for waterpipe smokers, while more than 10 cigarettes daily for cigarette smokers, all those with systemic diseases, such as (diabetes mellitus, chronic heart disease), or taken antibiotics within the last 3 months; or had periodontal treatment during the last 6 months were excluded.

Oral examination of each subject was carried out following criteria recommended by World Health Organization, 2013<sup>(29)</sup>, the subjects were examined while seated on a dental chair. The clinical examination of dental caries was conducted using a dental explorer and mouth mirror.

Unstimulated saliva was collected from subjects in the early morning between 8-10 am. The subject was asked to avoid eating or drinking for three hours before the procedure of saliva collection, then asked to wash his mouth with distilled water for one minute and to relax for five minutes directly before starting saliva collection. Subjects were also asked to spit saliva into the sterilized cups that possess graduations<sup>(30)</sup>.

**RESULTS**

Table 1 :showed that the median values of DMFT, DT, MT, and FT (8.0, 2.5, 1.0, 3.5 ) respectively were higher among cigarette smokers group than both waterpipe smokers and non-smokers groups; all these differences were statistically highly significant ( $P \leq 0.01$ ).

Although the median value of DT and MT were equal (1.5, 0.0) respectively in both the waterpipe smokers group and non-smokers group, the mean rank values of DT and MT were higher in the waterpipe group (38.018, 37.143), respectively than non-smokers group (35.78, 33.929) with a non-significant differences ( $P > 0.05$ ) between both groups as clarified by Mann-Whitney test.

The median of DMFT and FT were higher in the non-smoker's group (3.5, 1.0 respectively) than in waterpipe smokers group (3.0, 0.0), with a non-significant difference ( $P > 0.05$ ) in DMFT, and statistically highly significant ( $p \leq 0.01$ ) in FT.

Regarding the viable count of *S.mutans* results showed that median values of *S.mutans* calculated in (CFU/ml) were higher in the cigarette smokers group (23.00), followed by the waterpipe smokers group (12.50) and then non-smokers group

(11.65). These differences were statistically highly significant ( $p \leq 0.01$ ). Mann-Whitney test clarified that despite that the median value of colony count in the waterpipe group was higher than the non-smoker's group, statistically there were no significant differences ( $P > 0.05$ ) between them.

According to *S.sobrinus* viable count (CFU/ml), the results showed that mean rank values in the cigarette smokers group (47.732) have the higher value followed by the non-smoker's group (40.054) and waterpipe smokers group (39.714), but statistically, there were no significant differences ( $P > 0.05$ ).

In table 2, results showed that the correlation of DMFT with *S.mutans* and *S.sobrinus* viable count, was a weak positive correlation in the non-smoker's group and the waterpipe smokers group, with a non-significant statistical difference ( $P > 0.05$ ), whereas in the cigarette smokers group the correlations was statistically significant. It was moderate positive with *S.mutans* count ( $P \leq 0.01$ ) and weak positive with *S.sobrinus* ( $P \leq 0.05$ ).

**Table 1: The differences between, waterpipe smokers, cigarette smokers and non-smokers groups considering DMFT index and viable count of mutans streptococcus**

Variables	Groups	Descriptive statistics				Group difference			
		Median	Mean Ran	KWH test	p-value	Groups	Mann-Whitney U	p-value	
DT	Control	1.5	35.786	9.362	0.009	Control	Waterpipe	377	0.800
	Waterpipe	1.5	38.018				Cigarette	219	0.004
	Cigarette	2.5	53.696				Waterpipe	251.5	0.019
MT	Control	0	33.929	16.907	0.000	Control	Waterpipe	364.5	0.587
	Waterpipe	0	37.143				Cigarette	179.5	0.000
	Cigarette	1	56.429				Waterpipe	214.5	0.002
FT	Control	1	39.964	32.945	0.000	Control	Waterpipe	224.5	0.004
	Waterpipe	0	25.589				Cigarette	153.5	0.000
	Cigarette	3.5	61.946				Waterpipe	86	0.000
DMFT	Control	3.5	32.750	33.633	0.000	Control	Waterpipe	356	0.550
	Waterpipe	3	30.589				Cigarette	83	0.000
	Cigarette	8	64.161				Waterpipe	94.5	0.000
<i>S.mutans</i> count x 10 <sup>5</sup> (CFU/ml)	Control	11.65	29.839	32.970	0.000	Control	Waterpipe	355	0.543
	Waterpipe	12.50	33.696				Cigarette	74.5	0.000
	Cigarette	23.00	63.964				Waterpipe	108.5	0.000
<i>S.sobrinus</i> count x 10 <sup>5</sup> (CFU/ml)	Control	4.00	40.054	1.954	0.376	Control	Waterpipe	387	0.934
	Waterpipe	4.00	39.714				Cigarette	318.5	0.226
	Cigarette	5.00	47.732				Waterpipe	319	0.229

DMFT= decay, missing, filling tooth, SFR= salivary flow rate,  $P \leq 0.01$  highly significant,  $P \leq 0.05$  Significant,  $P > 0.05$  Non-significant

**Table 2: correlation of DMFT index with *S mutans*, *S sobrinus***

Variables	DMFT Index		
	Control	Waterpipe	Cigarette
<i>S Mutans</i>	0.289	0.226	0.565
	0.291	0.294	0.002
<i>S Sobrinus</i>	0.094	0.054	0.385
	0.633	0.783	0.043

**P≤ 0.01 highly significant, P≤0.05 Significant, P>0.05 Non-significant**

## DISCUSSION

Although the oral cavity contains numerous types of microorganisms, the present study has chosen *S.mutans* and *S.sobrinus* as these two cariogenic bacteria are the most common dental pathogens responsible for the development of caries. In this study, Sucrose-bacitracin, 20 Modified (SB-20M) culture medium was used to culture the aforementioned bacteria, as a selective medium is reliable for detection and direct morphological differentiation of *S.mutans* and *S.sobrinus*.

Results showed that dental caries increased in cigarette smokers and that median values of DT, MT, FT and, DMFT, were significantly higher among cigarette smokers than waterpipe smokers and non-smokers, These results were in agreement with the former studies (25, 31, 32) and could be due to smoking influences on saliva as it reduced the buffer capability, changing its chemical agent and bacterial components, as well as the existence of nicotine in tobacco which enhances the adhesion of *S.mutans* to the acquired pellicle and increases biofilm formation that increases the incidence of dental caries (33, 34).

On the other hand, results also revealed that the median value of DT and MT were slightly higher in waterpipe group than the non-smokers but without significant differences. Similarly, previous researches assumed that waterpipe smokers were more susceptible to the development of dental caries than non-smoker because of high scores of plaque and calculus indices (35,36), while FT was higher in control groups than waterpipe smokers in the present work. This result agrees with the results of Sahib *et al.*, (2018) (37).

The present work showed that median of *S mutans* viable count (CFU/ml) in cigarette smokers group have the highest value followed by waterpipe smokers group and then non-smokers groups with significant differences and this may be explained by

the effect of nicotine and tar which improves the growth and attachment of *S. mutans* (38,39).

The result of the present study disagrees with another previous study that found that the number of *S.mutans* in the saliva is not related to the smoking status (40).

Furthermore, the current results showed that median values of *S.sobrinus* count in cigarette smokers group had the higher value followed by waterpipe smokers group and non-smokers groups but statistically there were no significant differences. This corresponds with the previous study which recorded higher counting levels of *S.sobrinus* in smokers than non-smokers (41). This slight increase in colony count of *S sobrinus* in cigarette smokers may be due to the same aforementioned reason that leads to an increase in *S.mutans* as both *S.mutans* and *S. sobrinus* share several traits and virulence factors (42).

It can be concluded that waterpipe smokers are less affected by dental caries when compared with cigarette smokers. This could be due to the lower daily nicotine exposure which is equivalent to 10 cigarettes, for daily smoking and equal to 2 cigarettes per day in non-daily smoking (43). Moreover, the addition of artificial flavoring; like honey, glycerin and other flavors in the preparation of Moassel contributed to lowering the nicotine level of each gram of moassel (44).

On the other hand, the correlation of dental caries with *S.mutans* and *S.sobrinus* revealed that the relationship of DMFT with *S.mutans* count was positive non-significant in the non-smokers group and waterpipe smokers group, while it was significantly positive in the cigarette smokers group. This agrees with other previous studies which showed a positive association of caries scores with *S.mutans* (45-49),

Additionally, this study noted a positive non-significant relationship of DMFT with *S sobrinus* count in non-smokers group and waterpipe smokers group, while it was positive significant in the cigarette smokers group, and this agree with a recent study (50).

This positive correlation with *S.mutans* and *S.sobrinus* could be due to the role of mutans streptococcus in the initiation of dental caries as both of these bacteria are well-known primary cariogenic microorganisms associated with dental caries (51).

## CONCLUSION

Dental caries were affected by cigarette smoking more than waterpipe smoking, where dental caries and *S.mutans* viable count (CFU/ml) were significantly higher among cigarette smokers than waterpipe smokers or non-smokers.

A cigarette smoker group revealed a significant positive association between dental caries with both *S.mutans* and *S.sobrinus* count.

Regarding the *streptococcus sobrinus* count, there were no significant differences between the three studied groups despite a slight increase in colony count of *S.sobrinus* in cigarette smokers.

**Conflict of interest:** None.

## REFERENCES

- Slade J .. Historical Notes on Tobacco. Prog Respir Res ; 1997; 28 :1-11.
- Ren N, Timko MP. AFLP analysis of genetic polymorphism and evolutionary relationships among cultivated and wild Nicotiana species. Genome. 2001 Aug 1;44(4):559-71.
- Tyrrell I, Allan m, Brandt. The Cigarette Century: The Rise, Fall, and Deadly Persistence of the Product That Defined America. New York: Basic Books. 2007
- Giovino GA, Mirza SA, Samet JM, et al. Tobacco use in 3 billion individuals from 16 countries: an analysis of nationally representative cross-sectional household surveys. Lancet. 2012 Aug 18;380(9842):668-79.
- Maziak W. Commentary: the waterpipe—a global epidemic or a passing fad. Int J Epidemiol. 2010 Jun 1;39(3):857-9.
- Lopez AA, Eissenberg T, Jaafar M, et al. Now is the time to advocate for interventions designed specifically to prevent and control waterpipe tobacco smoking. Addict Behav. 2017 Mar 1;66:41-7.
- Khattab A, Javaid A, Iraqi G, et al. Shahrour N, Taright S, Idrees M, Polatli M, Rashid N. Smoking habits in the Middle East and North Africa: results of the BREATHE study. Respir Med. 2012 Dec 1;106:S16-24.
- Jawad M, Charide R, Waziry R, et al. The prevalence and trends of waterpipe tobacco smoking: A systematic review. PloS one. 2018 Feb 9;13(2):e0192191.
- World Health Organization. Guidelines for controlling and monitoring the tobacco epidemic. World Health Organization; 1998.
- Holford TR, Meza R, Warner KE, et al. Tobacco control and the reduction in smoking-related premature deaths in the United States, 1964-2012. JAMA. 2014 Jan 8;311(2):164-71.
- Reibel J. Tobacco and oral diseases. Update on the evidence, with recommendations. Med Princ Pract. 2003;12 Suppl 1:22-32.
- Bowen WH, Burne RA, Wu H, et al. Oral biofilms: pathogens, matrix, and polymicrobial interactions in microenvironments. Trends Microbiol.. 2018 Mar 1;26(3):229-42.
- Selwitz RH, Ismail AI, Pitts NB. Dental caries. Lancet. 2007 Jan 6;369(9555):51-9.
- Yadav K, Prakash S. Dental caries: A microbiological approach. J Clin Infect Dis Pract. 2017;2(1):1-5.
- Thenisch NL, Bachmann LM, Imfeld T, et al. Are mutans streptococci detected in preschool children a reliable predictive factor for dental caries risk? A systematic review. Caries Res. 2006;40(5):366-74.
- Eşian D, Man A, Burlibasa L, et al. Salivary level of Streptococcus mutans and Lactobacillus spp. related to a high risk of caries disease. Rom Biotechnol Lett. 2017;22(2):12496-503.
- Gomez A, Espinoza JL, Harkins DM, et al. Host genetic control of the oral microbiome in health and disease. Cell host microbe. 2017 Sep 13;22(3):269-78.
- Mitrakul K, Akarapipatkul B, Thammachat P. Quantitative Analysis of Streptococcus Mutans, Streptococcus Sobrinus and Streptococcus Sanguinis and their Association with Early Childhood Caries [Internet]. 2020 February [Cited September 13, 2021];14(2):ZC23-ZC28.
- Hirasawa M, Takada K. A new selective medium for Streptococcus mutans and the distribution of S. mutans and S. sobrinus and their serotypes in dental plaque. Caries Res. 2003;37(3):212-7.
- Saravia ME, Nelson-Filho P, Ito IY, et al. Morphological differentiation between S. mutans and S. sobrinus on modified SB-20 culture medium. Microbiol Res. 2011 Jan 20;166(1):63-7.
- Benedetti G, Campus G, Strohmenger L, et al. Tobacco and dental caries: a systematic review. Acta Odontol Scand.. 2013 Jan 1;71(3-4):363-71.
- de Araújo Nobre M, Maló P. Prevalence of periodontitis, dental caries, and peri-implant pathology and their relation with systemic status and smoking habits: Results of an open-cohort study with 22009 patients in a private rehabilitation center. Journal of dentistry. 2017 Dec 1;67:36-42.
- Bernabé E, Macritchie H, Longbottom C, et al. Birth Weight, Breastfeeding, Maternal Smoking and Caries Trajectories. J Dent Res.; 96(2)171-178.
- Liu S, Wu T, Zhou X, et al. Nicotine is a risk factor for dental caries: an in vivo study. J Dent Sci.. 2018 Mar 1;13(1):30-6..
- Koul M, Pandey A. Association of tobacco habits with dental caries and Streptococcus mutans count. Int. J. Appl. Dent. Sci.. 2019;5(2):479-83..
- Modi A, Kalra D, Bhate P, et al. Assessment of Oral Health Status & Association of Caries Experience, Oral Hygiene and Dental Plaque with Tobacco Habits: A Cross Sectional Analytical Study. Indian Journal of Contemporary Dentistry January-June 2020; 8(1): 5-10.
- Johnson NW, Bain CA. Tobacco and oral disease. Br Dent J. 2000 Aug;189(4):200-6.
- Madiyal A, Ajila V, Babu SG, et al. Status of thiocyanate levels in the serum and saliva of non-smokers, ex-smokers and smokers. Afr Health Sc. 2018 Aug 15;18(3):727-36.

29. World Health Organization. Oral health surveys: basic methods. World Health Organization; 2013.
30. Henson BS, Wong DT. Collection, storage, and processing of saliva samples for downstream molecular applications. *Methods Mol Biol.* 2010 (pp. 21-30). Humana Press, Totowa, NJ.
31. Campus G, Cagetti MG, Senna A, et al. Does smoking increase risk for caries? A cross-sectional study in an Italian military academy. *Caries Res.* 2011;45(1):40-6.
32. Jiang X, Jiang X, Wang Y, et al. Correlation between tobacco smoking and dental caries: A systematic review and meta-analysis. *Tob Induc Dis.* 2019;17.
33. Wu J, Li M, Huang R. The effect of smoking on caries-related microorganisms. *Tob Induc Dis.* 2019;17.
34. El-Ezmerli NF, Gregory RL. Effect of nicotine on biofilm formation of *Streptococcus mutans* isolates from smoking and non-smoking subjects. *J Oral Microbiol.* 2019 Jan 1;11(1):1662275.
35. Al-Mufti SM, Saliem SS. Waterpipe Smoking Effect on Clinical Periodontal Parameters, Salivary Flow Rate and Salivary pH in Chronic Periodontitis Patient. *J Bagh College Dentistry.* 2018;30(1).
36. Javed F, Al-Kheraif AA, Rahman I, et al. Comparison of clinical and radiographic periodontal status between habitual water-pipe smokers and cigarette smokers. *J Periodontol.* 2016 Feb;87(2):142-7.
37. Sahib AM, Radhi NJ. Salivary constituent in relation to dental caries among coffee-shop workers in Najaf city/Iraq. *Int. j. med. health res.* 2018 Jan 1;7:86-92.
38. Courtney R. The Health Consequences of Smoking-50 Years of Progress: A Report of the Surgeon General, 2014Us Department of Health and Human Services Atlanta, GA: Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for. *Drug Alcohol Rev.* 2015; 34(6):694-695.
39. Zonuz AT, Rahmati A, Mortazavi H, et al. Effect of cigarette smoke exposure on the growth of *Streptococcus mutans* and *Streptococcus sanguis*: an in vitro study. *Nicotine Tob Res.* 2008 Jan 1;10(1):63-7.
40. Nakonieczna-Rudnicka M, Bachanek T. Number of *Streptococcus mutans* and *Lactobacillus* in saliva versus the status of cigarette smoking, considering duration of smoking and number of cigarettes smoked daily. *Ann Agric Environ Med.* 2017;24(3):396-400.
41. Belstrøm D, Holmstrup P, Nielsen CH, et al. Bacterial profiles of saliva in relation to diet, lifestyle factors, and socioeconomic status. *J Oral Microbiol.* 2014 Jan 1;6(1):23609.
42. Conrads G, de Soet JJ, Song L, et al. Comparing the cariogenic species *Streptococcus sobrinus* and *S. mutans* on whole genome level. *J Oral Microbiol.* 2014 Jan 1;6(1):26189.
43. Neergaard J, Singh P, Job J, et al. Waterpipe smoking and nicotine exposure: a review of the current evidence. *Nicotine Tob Res.* 2007 Oct 1;9(10):987-94..
44. Chaouachi K. Hookah (shisha, narghile) smoking and environmental tobacco smoke (ETS). A critical review of the relevant literature and the public health consequences. *Int J Environ Res Public Health.* 2009 Feb;6(2):798-843.
45. Loyola-Rodriguez JP, Martinez-Martinez RE, Flores-Ferreyra BI, et al. Distribution of *Streptococcus mutans* and *Streptococcus sobrinus* in saliva of Mexican preschool caries-free and caries-active children by microbial and molecular (PCR) assays. *J Clin Pediatr Dent.* 2007 Dec 1;32(2):121-6..
46. Pannu P, Gambhir R, Sujlana A. Correlation between the salivary *Streptococcus mutans* levels and dental caries experience in adult population of Chandigarh, India. *Eur J Dent.* 2013 Apr;7(02):191-5.
47. Oda Y, Hayashi F, Okada M. Longitudinal study of dental caries incidence associated with *Streptococcus mutans* and *Streptococcus sobrinus* in patients with intellectual disabilities. *BMC Oral Health.* 2015 Dec;15(1):1-5.
48. Lee YJ, Kim MA, Kim JG, et al. Detection of *Streptococcus mutans* in human saliva and plaque using selective media, polymerase chain reaction, and monoclonal antibodies. *Oral Biol Res* 2019;43(2):121-129
49. Veena RL, Nagarathna C.. Correlation of streptococcus mutans and streptococcus sobrinus colonization with and without caries experience in preschool children. *Indian J Dent Res.* 2020;31(1):73-79
50. Al-Anbari A A, Al-Ani M A . The association between severity of dental caries and salivary immunoglobulins in asthmatic adult patients. *Indian Journal of Forensic Medicine & Toxicology.* 2021; 15(1) :2446-2450
51. Garcia SS, Blackledge MS, Michalek S, et al. Targeting of *Streptococcus mutans* biofilms by a novel small molecule prevents dental caries and preserves the oral microbiome. *J Dent Res.* 2017 Jul;96(7):807-14..

### المستخلص

الخلفية: النرجيلة والسجائر نوعان من استهلاك التبغ ، حيث ان تعاطي التبغ له آثار ضارة على تجويف الفم ، وقد أفادت العديد من الدراسات حول العالم بوجود علاقة كبيرة بين التدخين وزيادة تسوس الأسنان والعدد الحيوي للبكتيريا المسببة للتسوس المواد والطرق: تم جمع اللعاب غير المحفز من 84 شخصاً وقسم بالتساوي إلى ثلاث مجموعات من مدخني النرجيلة ومدخني السجائر وغير المدخنين ، جميع المشاركين هم من الذكور البالغين الذين تتراوح أعمارهم بين 25-60 عاماً ، وتم قياس تسوس الأسنان باستخدام مؤشر DMFT ، بينما تم عزل المكورات المسبحية الميوتنس و المكورات المسبحية السوبرينوس باستخدام وسط انتقائي SB 20M

النتيجة: أظهرت الدراسة الحالية ارتفاعاً مؤثراً ( $p \leq 0.01$ ) DMFT و DT و MT و FT بين مجموعة مدخني السجائر مقارنة بمجموعات مدخني النركيلة وغير المدخنين. حيث كان العدد القابل للحياة من المكورات المسببية الميوتنس مؤثراً ( $p \leq 0.01$ ) وأعلى في مجموعة مدخني السجائر تليها مجموعة مدخني النركيلة ثم مجموعة غير المدخنين ، بينما لم يظهر العدد الحيوي للمكورات المسببية السوبرينوس فروق ذات دلالة إحصائية بين المجموعات ( $P > 0.05$ ) وكانت العلاقة بين DMFT و المكورات المسببية الميوتنس و المكورات المسببية السوبرينوس موجبة معنوياً ( $p \leq 0.05$ ) في مجموعة مدخني السجائر فقط. الاستنتاجات: زيادة تسوس الأسنان في مدخني السجائر ، حيث ال DMFT و عدد المكورات المسببية الميوتنس أقل تأثراً بالنركيلة من تدخين السجائر ، و يوجد علاقة إيجابية بين DMFT و عدد العقديات الطافرة فقط في مدخني السجائر.



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