

The Caries Experience in the Smoker and Non-Smoker among College Student

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Abstract

Dental caries is the scientific term for tooth decay or cavities. It is caused by specific types of bacteria. They produce acid that destroys the tooth's enamel and the layer under it, the dentin. The bacteria turn sugar and carbohydrates (starches) in the foods we eat into acids. Smoking is also a global and national health problem and is one of the most significant health risks that impact all age groups.

The Study Aims to investigate the effects of different tobacco smoking methods and their relation to dental caries in comparison to non-smoker students.

Material and Method: A random sample of 336 students from Tikrit university aged 19-24 years, the sample grouped to cigar, shisha, cigar and shisha together and non-smoker groups. Dental caries was recorded according to WHO criteria (1987) were decayed, missing and filled with permanent teeth (DMFS). The data were analyzed as a descriptive statistic includes frequency, percentage, mean, and for statistical analysis of the data using T- test and one- way ANOVA test to detect the significance of the relation between various variable.

Result: the result of this study showed that the severity of dental caries higher in smoking cigar and smoking shisha than in the non-smoking group with statistically highly significant.

Conclusion Nicotine may be increasing the incidence and severity of dental caries. So that, nicotine might be considered as a risk factor for inducing of dental caries.

Key words: dental caries, cigar, shisha, non-smoker

Introduction

Dental caries is a well-known infectious disease that affects all ages ⁽¹⁾. Caries results from the interplays between enamel and dentin tooth structures, diet, and cariogenic bacteria. These interactions make the bacteria to release acidic byproducts resulting in degrading the hard tissues of the teeth ⁽²⁾. Without treatment, caries lesion has the potential to advance and reach the pulp, which can result in severe pain ⁽³⁾. Cariogenic bacteria such as *Streptococcus mutans* that colonize on the firm surfaces of the oral cavity performs a vital role in plaque development and dental caries formation. *Streptococcus mutans* competes with other bacteria to attach to the tooth surface. *S. mutans* is remarkably aciduric and acidogenic. It produces organic acids, mainly lactic acid,

which damages the enamel by demineralization ⁽⁴⁾.

Smoking dates to 5000–3000 BC, when the farm product began to be cultivated in South America and Mesoamerica; consumption later developed into burning the plant material either by chance or with the intent of exploring other ways of consumption ⁽⁵⁾. Tobacco was first introduced in France in 1560 by a Frenchman named Jean Nicot (from whose name the word nicotine is derived). Tobacco then spread from France to England ⁽⁶⁾. Tobacco products are extensively used by cigarettes, which are often smoked widely ⁽⁷⁾. about 1.1 billion adults (29% of the adult population) are cigarette smokers throughout the world ⁽⁸⁾. Tobacco use forces a huge and increasing burden on public health worldwide. Approximately 5 million death annually is attributed

to tobacco use. According to the current trends, it is expected that by the year 2030, this number will increase to 10 million, with about 70% of these deaths occurring in low- and middle-income nations ⁽⁹⁾.

The main consistency of tobacco smoke is the nicotine, which is an alkaloid from the oxygen-free alkaloid cluster ⁽¹⁰⁾. There is no clear explanation of why smoking increases the risk of dental caries. The relationship between dental caries and smoking has been the subject of debate. A common belief in the earlier literature was that smoking helps to reduce dental caries ^(11, 12, 13). Schmidt, in 1951, held this belief when he proclaimed that an increase in tobacco smoking was succeeded to decrease caries rate ⁽¹⁴⁾. Thiocyanate concentration, a constituent of tobacco smoke and in normal saliva, was found a higher in the saliva of the smokers, thiocyanate caries inhibiting effect on dental caries ⁽¹⁵⁾. So, one might predict fewer dental caries in smokers. However, the reduced buffering effect and probable lower pH of the smoker's saliva and the more significant number of Lactobacilli and *S. mutans* may indicate a higher susceptibility to caries ^(15, 16). Several types of research had recorded tobacco smoking as a risk factor. In the UK, there was a considerable increase in root and coronal caries due to exposure tobacco products for years ⁽¹⁷⁾. also, there was a dose-dependent relation between root caries and tobacco in the USA ⁽¹²⁾ other studies significantly showed the effect of smoking can lead to dental caries, tooth discoloration and gum recession which may lead to uneven margins on the crown and other restoration ⁽¹⁸⁾.

There is limited information concerning the consequences of nicotine on oral bacteria and *S. mutans*, and important microbe in the etiology of dental caries. Nicotine has been described within the saliva of smokers and can stimulate the growth of *S. mutans* and likely place smokers at increased chance for dental caries ^(19,20).

Material and Method

Study participants were constituting of a random sample of 336 students from Tikrit university aged 19-24 years, the student in the sample was from the different colleges of the university, the duration of data collection was from October 2018 to May 2019. Smoking status was obtained through a questionnaire. The students with a history of systemic diseases which may affect the result

were excluded. The tooth was diagnosed to be caries according to the criteria suggested by WHO, 1987 ⁽²¹⁾. were decayed, missing, and filled with permanent teeth (DMFS). The examination of dental caries was conducted by using plane mouth mirrors and bland explorers. Radiographic studies were not used. The students were categorized into three groups as follows: non-smoker 80 students, smoker cigarette only 156, smoker shish 100 students and the duration of smoking range between (1-3) years, all the students are male-only. Data analyzed using a statistical program (SPSS - 10); were descriptive statistic includes frequency, percentage, mean, standard deviation, and standard error and statistical analysis of data using T-test and ANOVA test used to detect the significance of the relation between the variable. P-value <0.05 was considered as statistically significant while P-value >0.05 was considered as statistically not significant and P-value < 0.01 was considered as highly significant.

The Results

A total of 336 students were included in this study. The age of study participants was between 19 and 24 years old. The distributions of the total sample by types of smoking are seen in Table (1).

Tables (2) illustrates the mean values, standard deviation and standard errors of caries severity for permanent dentition in the non-smoker and smoking cigar, DMFS mean value for the smoking cigar students was found to be higher (19.53 ± 0.50) than in non-smoker students (9.20 ± 0.45). The differences were found to be statistically highly significant (T-test value =15.39, P-value = 0.001).

The mean values of caries experience for permanent dentition (DMFS), were found to be higher in the students smoking shisha (12.50 ± 0.47) than non-smoking students, statistically, there were highly significant differences founded between them (T-test = 5.05, P-value = 0.015) as seen in table (3), while the mean value of DMFS was higher in the students were smoking cigar than in students who smoking shisha. The difference was reported to be statistically highly significant (T-test = 10.25, P-value = 0.002) as shown in table (4). Analysis using One-way ANOVA between three groups showed statistically highly significant F-value = 123.91, P-value = 0.00006 (smoking cigar

19.53 a, smoking shisha 12.50 b and non-smoking 9.20 c) this illustrated by table (5) and diagram (1).

Table (1) distribution of total sample according to the types of smoking.

Type of smoking	number	Percentage %
Non-smoker	80	23.81
Smoking cigar	156	46.43
Smoking shisha	100	29.76
total	336	100

Table (2) Caries experience of permanent dentition (mean, standard deviation and standard error of DMFS index) among non-smoking group and smoking cigar.

Types of smoking	mean	SD	± SE
Non -smoker	9.20	0.78	0.45
Smoking cigar	19.53	0.86	0.50

T-test value = 15.39, P-value = 0.001

Table (3) Caries severity of permanent dentition DMF index (mean, standard deviation and standard error) among non-smoking group and smoking shisha.

Types of smoking	mean	SD	± SE
Non -smoker	9.20	0.78	0.45
Smoking shisha	12.50	0.82	0.47

T-test value = 5.05, P-value = 0.015

Table (4) Caries severity of permanent dentition DMF index (mean, standard deviation and standard error) among smoking cigar group and smoking shisha.

Types of smoking	mean	SD	± SE
Smoking cigar	19.53	0.86	0.50
Smoking shisha	12.50	0.82	0.47

T-test value = 10.25, P-value = 0.002

Table (5) the differences between the total sample (smoking cigar, smoking shisha and non-smoking) by using One-way ANOVA (Analysis of Variances).

Types of smoking	numbers	Mean	SD
Smoking cigar	3	19.53 a	0.86
Smoking shisha	3	12.50 b	0.82
Non-smoking	3	9.20 c	0.78

F-value = 123.91, P-value = 0.00006

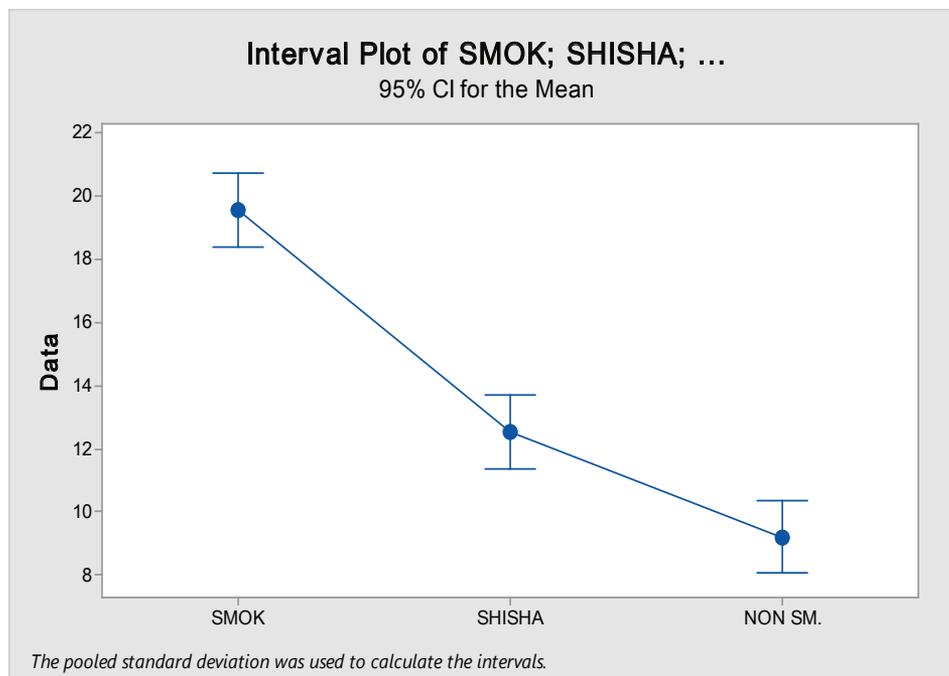


Diagram (1) the caries experience for the three groups

Discussion

The present study is the first Iraqi study that deals with the relation between different types of smoking and dental caries in Tikrit city. The caries experience (DMFS) in this study was found to be higher in the smoker students (cigarette and shisha) than in the non-smoker students which agrees to the other studies^(22,23,24). Also, this study agrees with the study was conducted in Japan 2013, where clinical data of smokers and non-smokers from 753 clinics concluded that patients who smoked had significantly more treatment procedures than non-smokers⁽²⁵⁾. This is may be due to that nicotine stimulates biofilm of metabolic activity of oral pathogens such as *Streptococcus mutans*, a Gram-positive oral bacterium; as a result, this stimulation increases *S. mutans* biofilm formation and metabolism of sucrose into lactic acid, consequently contributing to dental caries⁽²⁶⁾ in addition, various sugars and sweeteners are added deliberately during tobacco production method up to 4%wt or can be up to 13%wt of sugars. Sugars used as a cigarette additive include glucose, fructose, invert sugar (glucose/fructose mixture) and sucrose. Also, many tobacco additives contain a high amount of sugars, for example, fruit juices, honey, molasses extracts, cones, and maple

syrup and caramel. The added sugars are usually reported to serve as flavor/casing and humectants. However, sugars also encourage tobacco smoking, because they create acids that neutralize the sharp taste and throat impact of tobacco smoke. furthermore, the sweet taste and the pleasant smell of caramelized sugar flavors are appreciated by starting adolescent smokers⁽²⁷⁾. Smokers usually had bad oral hygiene and less primitive outlook on health, besides, they usually have different eating habits, presumably consuming a high number of sugar-containing products like soft drinks and snacks^(22,28). Daily smoking has been reported to be associated with an increase in the use of sugar in coffee or in tea, and with more frequent alcohol consumption^(29,34,36).

The current study added to multiple other studies to confirm the harmful effects of smoking and disagree with early studies reported reduce dental caries in individuals with a smoking habit, which reported that smoking reduces dental caries^(12,14,30). In contrast is the case reported by Zitterbart et al., which did not find any relation between dental caries and nicotine^(31,32). Some researchers estimated the percentage of the highest amount of caries-promoting such as sugar and caries-inhibiting chemicals such as fluoride in commercially

available^(33,35).

Conclusion

Our Study conducted that dental caries higher in the smoker than in the non-smoker student and dental caries showed higher prevalence in the student that smoke cigar than smoking shisha. Smoking with cigar or shisha may be increased the incidence and severity of dental caries. So, nicotine might be a risk factor for smoking-induced caries.

Ethical Considerations:

All Research participants haven't been subjected to any kind of harm in any way and the dental caries was recorded according to WHO criteria (1987) were decayed, missing and filled with permanent teeth (DMFS).

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Conflict of Interest: The author declare no conflict of interest regarding this research.

References

- 1- Dye BA, Thornton-Evans G, Li X, Iafolla TJ. Dental caries and sealant prevalence in children and adolescents in the United States, 2011–2012. NCHS Data Brief 2015; 191:1–8.
- 2- Li MY, Huang RJ, Zhou XD, Gregory RL. Role of sortase in Streptococcus mutans under the effect of nicotine. Int J Oral Sci. 2013; 5:206–211.
- 3- Huang R, Li MY, Gregory RL. Effect of nicotine on grow and metabolism of streptococcus mutans. Eur J Oral Sc. 2012; 120: 319-325.
- 4- El-ezmerli NF. Effect of nicotine on biofilm formation of Streptococcus mutans isolates from smoking versus non-smoking subjects [Master's Thesis]. Indianapolis, Indiana: Indiana University School of Dentistry; 2015.
- 5- Gately, Iain (2004) [2003]. Tobacco: A Cultural History of How an Exotic Plant Seduced Civilization. Diane. pp. 3–7. ISBN 978-0-8021-3960-3. Retrieved 22 March 2009.
- 6- Giovino, GA; Mirza, SA; Samet, JM; Gupta, PC; Jarvis, MJ; Bhala, N; Peto, R; Zatonski, W; Hsia, J; Morton, J; Palipudi, KM; Asma, S; GATS Collaborative, Group (18 August 2012). "Tobacco use in 3 billion individuals from 16 countries: an analysis of nationally representative cross-sectional household surveys". Lancet. **380** (9842): 668–79.
- 7- Prokhorov AV, Winickoff JP, Ahluwalia JS, Ossip-Klein D, Tanski S, Lando HA et al. Youth tobacco use: A global perspective for child health care clinicians. Pediatrics 2006;118: 890-903.
- 8- Anderson P. Global use of alcohol, drugs and tobacco. Drug Alcohol Rev 2006; 25:489-502.
- 9- Jha P, Chaloupka FJ, Corrao M, Jacob B. Reducing the burden of smoking world-wide: effectiveness of interventions and their coverage. Drug Alcohol Rev. 2006 Nov;25(6):597-609.
- 10- Henningfield, J. E. & Goldberg, S. R. Progress in understanding the relationship between the pharmacological effects of nicotine and human tobacco dependence. Pharmacol. Biochem. Behav.1988; 30 (1):217-20.
- 11- Hart AC. Prevention of decay of the teeth. Dent Items Interest. 1899;21(3):153-63.
- 12- Gibbs MD. Tobacco and dental caries. J Am Coll Dent. 1952; 19:365-7.
- 13- Zitterbart PA, Matranga LF, Christen AG, Park KK, Potter RH. Association between cigarette smoking and the prevalence of dental caries in adult males. Gen Dent. 1990 Nov-Dec;38(6):426-31.
- 14- Schmidt HJ. Tobacco smoke and the teeth. Stoma (Heidelb). 1951 May;4(2):111-25.
- 15- Johnson NW, Bain CA. Tobacco and oral disease. EU-Working Group on Tobacco and Oral Health. Br Dent J. 2000 Aug 26;189(4):200-6.
- 16- Kassirer B. Smoking as a risk factor for gingival problems, periodontal problems and caries. Univ Tor Dent J. 1994;7(1):6-10.
- 17- Hart A. Prevention of decay of the teeth. Dent Items Interest. 1899; 21:11
- 18- Shiyu Liu, Tianmu Wu, Xuedong Zhou, Bo Zhang, Sibe Huo, Yutao Yang, Keke Zhang, Lei Cheng, Xin Xu, Mingyun Li. Nicotine is a risk factor for dental caries: An in vivo study. Journal of Dental Sciences. 2018;13 (1): 30-36
- 19- Keenck K, Johnson RB. The effect of nicotine on growth of streptococci mutans, Dental association Journal. Jan 1999; 55(4):38-39.
- 20 - Ruijie Huang, Mingyun Li, Richard L. Gregory. Effect of nicotine on grow and metabolism of streptococci mutans. European Journal of Oral

- Science.2012; 120 (4): 319-352.
- 21- WHO. Oral health survey. Basic methods 3rd ed. 1987 WHO Geneva
- 22- Hirsch JM, Livian G, Edward S, Noren JG. Tobacco habits among teenagers in the city of 7- Goteborg, Sweden and possible association with dental caries. *Swedish Dent J.* 1991; 15:117-23.
- 23- Athra M AL-Weheb. Smoking and its relation to caries experience and salivary lactobacilli count. *Bagdad j college dentistry* 2005; 17(1): 92-95
- 24- Campus G, Cagetti MG, Senna A, Blasi G, Mascolo A, Demarchi P et al. Does smoking increase risk for caries? A cross-sectional study in an Italian military academy. *Caries Res* 2011; 45:40- 6
- 25- Ojima M, Hanioka T, Shimada K, Haresaku S, Yamamoto M, Tanaka K. The role of tobacco uses on dental care and oral disease severity within community dental clinics in Japan. *Tob Induc Dis.* 2013; 11:13
- 26 - Huang R, Li M, Gregory RL. Nicotine promotes *Streptococcus mutans* extracellular polysaccharide synthesis, cell aggregation and overall lactate dehydrogenase activity. *Arch Oral Biol.* 2015; 60:1083–1090.
- 27- Talhout R, Opperhuizen A, van Amsterdam JG. Sugars as tobacco ingredient: effects on mainstream smoke composition. *Food Chem Toxicol.* 2006 Nov;44(11):1789-98.
- 28- Axelsson P, Paulander J, Lindhe J. Relationship between smoking and dental status in 35-, 50-, 65-, and 75-year-old individuals. *J Clin Periodontol.* 1998 Apr;25(4):297-305.
- 29- Telivuo M, Kallio P, Berg MA, Korhonen HJ, Murtomaa H. Smoking and oral health: a population survey in Finland. *J Public Health Dent.* 1995;55(3):133-8.
- 30- Simon-Soro A, Belda-Ferre P, Cabrera-Rubio R, Alcaraz LD, Mira A. A tissue-dependent hypothesis of dental caries. *Caries Res.* 2013; 47:591-600.
- 31- Eintraub JA, Burt BA. Periodontal effects and dental caries associated with smokeless tobacco use. *Public Health Rep* 1987; 102:30-5.
- 32- Zitterbart PM, Marlin DC, Christen AG. Dental and oral effects observed in a long-term tobacco chewer: Case report. *J Indiana Dent Assoc.* 1983; 62:2.
- 33- Nallan CSK Chaitanya, Mamata Boringi, Ramakrishna Madathanapalle, Alekya Renee, Surabi V Sree, Nancy Priyanka, Triken Sowetha, Kondaiah Marella. The Prevalence of Dental Caries in Smokers and Smokeless Tobacco Users. *Dental hypotheses* 2018; 9 (2) 36-40
- 34- Jameel, M. I., Yaseen, A. H., Al-Samarraie, M. Q., & Abdulla, B. A. (2018). Visualization and Analysis of Tumor Protein P53 by VMD. *International Journal for Sciences and Technology*, 143(6042), 1-9
- 35- Abdulazeez, M., Hussein, A. A., Hamdi, A. Q., & Mustafa, M. A. (2020). Estimate the Complications That Resulting from Delayed Management of Dental Trauma in Tikrit City. *Journal of Cardiovascular Disease Research*, 11(2), 80-82
- 36- Mustafa, M.A & AL-Samarraie, M.Q . (2020) .SECONDARY MENOPAUSE and its RELATIONSHIP to HORMONAL LEVELS AMONG WOMEN at SALAH AL-DIN HOSPITAL . *European Journal of Molecular & Clinical Medicine* . Volume 7, Issue 09, PP 96-104.