

Association between Dental Caries and Passive Smoking and Its Related Factors in Children Aged 3–9 Years Old

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ABSTRACT

Aims: Dental caries is the most common chronic and infectious childhood disease. Several studies have investigated the side effects of cigarette smoke on oral health. Caries appear to be higher in children exposed to environmental cigarette smoke, but this relationship has not yet been established. The purpose of this study was to investigate the relationship between dental caries and passive smoker children aged 3–9 years old.

Materials and methods: This descriptive analytical study was performed on 75 children aged from 3 to 9 years. The study included two groups (first group: control, second group: passive smoker). Demographic data, frequency of toothbrushing and using toothpaste, sugar consumption, and number of dental visits were recorded in a questionnaire. Then, the children were examined, and dmft and DMFT index and plaque index were recorded. Children's saliva was then collected and sent to the laboratory for determination of cotinine levels. Then, the relationship between passive smoker and caries index and other factors were statistically analyzed.

Results: Studying dmft and DMFT, it was found that except for component M (Missing) which was zero in both groups, the other components of these two indices were significantly higher in the group exposed to passive smoker. Plaque index and salivary cotinine level were also significantly different in the two groups and were lower in the control group.

Conclusion: According to the results of this study, children exposed to passive smoker have a higher rate of caries than other children. It seems that it is primarily due to its effect on increasing plaque accumulation and then the effect of passive smoker on the physiological structures of the mouth and so on.

Clinical significance: We can perform preventive care for children if we know that passive smoking may cause more dental caries.

Keywords: Children, Cigarette smoking, Cross-sectional study, Dental caries.

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INTRODUCTION

Dental caries is an infectious, multifactorial disease and a perennial problem worldwide. Among all the causes of disability in the years of life that were studied in the World Disease Study in 2010 (GBD), the global prevalence of untreated dental caries was highest, without decreasing trend between 1990 and 2010.¹

The caries process is due to a combination of major and secondary factors such as microbial plaque containing carcinogenic microorganisms, caries substrate, time, saliva, immune mechanisms, dental tissues, and oral and fluoride ecological conditions. Behavioral factors such as poor oral hygiene and consumption of sweet meals and snacks also stimulate dental caries and decay.² Other factors are also involved in dental caries such as environment, genetics, nutrition, age, sex, etc. Cigarette smoke also appears to be one of the contributing factors to caries. Researchers say 4 of every 10 children in the world are exposed to cigarette smoke in their surroundings; one of the behaviors that researchers declare will have an important role in increasing children's dental caries in the future. About 20% to 80% of the world population is exposed to cigarette smoke, and according to the 2006 World Health Organization statistics, 42% of Iranian children are exposed to cigarette smoke. Disadvantages of passive smoking are approximately equal to being an active smoking.³ Some researchers believe that smoking can lead to dental caries along with other misconduct, including poor socioeconomic status, poor nutritional status, and poor dental and oral hygiene.^{4,5}

Although there are studies that do not show a link between passive smoking and caries incidence,⁶ other studies confirm

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this association.^{7,8} Tanaka et al. showed that exposure to passive smoking significantly increased the prevalence of dental caries in children that depends to dose. Negative effects of passive smoking include inflammation of the oral mucosa, impaired salivary gland function, a decrease in serum vitamin C, and decrease in immune function. Children exposed to passive smoke also have lower immunoglobulin A (IgA) levels and increased levels of sialic acid in their saliva.⁹

Based on the evidence cited, exposing the Iranian race to cigarette smoke in order to investigate the existence of this association in Iranian society, in this study, we investigated the relationship between children's dental caries and their parents' smoking in 3–9-year-old children referred to Dental school of Tehran University of Medical Sciences.

MATERIALS AND METHODS

First, the protocols of this descriptive analytical study were approved by the Regional Faculty Ethics Committee. This study was performed on 3–9-year-old children referred to Tehran University Dental faculty. The study consisted of two groups: The first group of children were not exposed to passive smoke (control), and the second group comprised children exposed to passive smoke. Sampling was randomized, and children who had a history of systemic disease and specific drug use or were undergoing orthodontic treatment were excluded.

After obtaining informed consent from parents, demographic information including age, sex, number of toothbrushes and toothpaste, amount of sugar consumed, and number of dental visits were recorded using a designed questionnaire. Then, samples were examined by a trained dentist on the dental unit, and dmft, DMFT index, and plaque index were recorded. The examination was done early in the morning and before eating. Then, the children were asked to rinse their mouth thoroughly with water and dip their saliva and finally collect it into a special sterile container (15 mL Falcon tube) within 5 minutes.⁸

These saliva samples were then inserted into test tubes containing intermediate material placed in ice-packed containers and transferred rapidly to the laboratory. The salivary cotinine content was calculated with the accuracy of 0.15 ng/mL by Salimetrics Salivary Cotinine ELISA kit. Samples with cotinine above 0.5 ng/mL were exposed to passive smoker group, and the other samples were considered as non-passive smoker. Using multiple pass regression pass 11 software, with considering $a = 0.05$, $B = 0.2$, $R2 = 0.2$, at least 75 children were considered an acceptable sample size.⁹ Thus, sampling continued until the total number of children reached 75.

All examinations and sampling were performed, and finally all data were loaded in SPSS v24 software, and quantitative data were analyzed using regression, *t* test, and analysis of variance.¹⁰ Then, to determine the effect of passive smoking on plaque index, personal hygiene information, and exposure to passive smoking with and caries rate, regression equation was measured without the plaque index. The effect of the abovementioned factors on plaque index was also investigated.

RESULTS

This study was performed on 75 children referred to the Dental School of Tehran University of Medical Sciences: 37 (49%) were female and 38 (51%) were male. There were 32 children in passive smoker group and 43 children in the control group. Descriptive data of the samples regarding variables affecting dental caries are given in Tables 1 and 2. There was no significant difference between the two groups in terms of age.

The results of dmft and DMFT studying in the two groups showed that in comparison to DMFT, F (Filling) was significantly higher in the passive smoker group than the control group. M (Missing) in both groups was zero, and D (Decayed) was higher in the inactive smokers group, but no significant difference was found in the control group (Table 3).

The absence of M in both groups is justified by age of the studied samples. In addition, the absence of a significant difference in component D can be justified by the fact that permanent teeth have recently erupted in this age range; so, children with permanent teeth are less exposed to passive smoking. Thus, the effect of passive

Table 1: Descriptive variables of parental cigarette and hookah consumption per year and age, plaque index and salivary cotinine level in the studied samples ($n = 75$)

Variables	Min	Max	Mean	Standard deviation
Age (year)	4	9	6	1/000
Cigarette pocket/year	0	365	98	142/0
Hookah/year	0	144	12	36/02
Plaque index	16/70	63/00	42/32	12/02
Salivary cotinine (ng/mL)	0/000	6/55	1/58	2/05

Table 2: Descriptive analysis of variables studied ($n = 75$)

Variables	n	
Relation of tobacco consumer to child	No contact with smoker	43
	Father	19
	Mother	1
	Both	10
	Others*	2
The type of tobacco used	None	43
	Cigarettes	24
	Hookah	3
	Hookah and cigarettes	5
Using toothbrush per day	No toothbrush	36
	1 to 2 times	34
	More than 2 times	5
Using toothpaste	No	36
	Yes	39
Sugar snacks consuming per day	None	1
	1 to 2 times	52
	More than 2 times	22
Regular dental visits	Yes	9
	No	66

*It means people who are not part of the family but have daily contact with children such as: uncle, grandfather, grandmother, etc.

Table 3: Mean DMFT and its components in each group (control group $n = 43$; passive smoker group $n = 32$)

Variable	Group	Mean	Standard deviation	Sig.
D (decay)	Control	0/56	0/88	0/117
	Passive smoker	0/94	1/19	
M (missing)	Control	0/00	0/00	0/227
	Passive smoker	0/00	0/00	
F (filling)	Control	0/09	0/29	0/021
	Passive smoker	0/38	0/70	
DMFT	Control	0/65	0/99	0/026
	Passive smoker	1/31	1/51	

smoking has not yet been shown on dental caries. Comparing dmft, all components of m, d, and f were significantly higher in the passive smoker group than in the control group (Table 4).

In the next step, regression analysis was used to investigate the relationship between all variables and dental caries. The results showed that among all the studied variables, smoking, hookah per year, sugary snacks, and plaque index were significantly correlated with dmft (Table 5), whereas in DMFT, only significant relation was reported for plaque index (Table 6).

Table 4: Mean dmft and its components in each group (control group n = 43; passive smoker group n = 32)

Variable	Group	Mean	Standard deviation	Sig.
d (decay)	Control	2/00	1/00	0/001
	Passive smoker	5/00	1/00	
m (missing)	Control	0/00	0/00	0/002
	Passive smoker	1/00	0/00	
f (filling)	Control	1/00	1/00	0/004
	Passive smoker	2/00	1/00	
dmft	Control	4/00	2/00	0/001
	Passive smoker	9/00	1/00	

Since it is possible that the variables studied can be affected by plaque index on dental caries, the relationship between the variables and plaque index was evaluated by regression analysis. Brushing, toothpaste, consumption of sugary snacks, and exposure to cigarette and hookah smoke were found to be significantly associated with plaque index (Table 7).

To investigate the effect of variables without affecting of plaque index, all variables except the plaque index were again included in the regression equation. By removing the plaque index variable, the previous study was re-examined, and it was found that, unlike the previous model, which only the plaque index effected on permanent DMFT, the annual consumption rate of the cigarette pack and brushing were also affected by DMFT (Table 8).

When the plaque index variable was removed in the dmft study, in addition to smoking and hookah, which had a significant relationship with the dmft in the previous model, in the new model, brushing, the use of toothpaste, and regular dental care also significantly correlated with the dmft (Table 9).

These results show that the effect of annual smoking and brushing on DMFT and brushing and use of toothpaste and regular dental care on dmft is indirect. This means that these factors change the plaque index and affect the caries rate. Finally, in this study,

Table 5: Regression test results in determining the effects of different variables related to dmft in the studied samples

Variable	Standard coefficients		Unstandardized coefficients		
	β	β	The standard error	t	Sig. (<0.1)
Cigarette use/year	0/413	0/009	0/002	4/96	0/001
Hookah/year	0/155	0/013	0/006	2/068	0/043
Toothbrushing/day	-0/175	-0/873	1/090	-0/801	0/426
Using toothpaste	0/218	1/331	1/168	1/122	0/266
Sugar snacks consuming per day	0/105	0/668	0/519	1/288	0/022
Regular dental visits	-0/103	-0/965	0/854	-1/129	0/263
Plaque index	0/418	0/107	0/029	3/700	0/001

Table 6: Regression test results in determining the effects of different variables related to DMFT in the studied samples

Variable	Standard coefficients		Unstandardized coefficients		
	β	β	The standard error	t	Sig. (<0.1)
Cigarette use/year	0/119	0/001	0/001	0/908	0/367
Hookah/year	0/051	0/002	0/004	0/435	0/665
Toothbrushing/day	-0/128	-0/265	0/712	-0/372	0/711
Using toothpaste	0/083	0/212	0/775	0/274	0/785
Sugar snacks consuming per day	-0/027	-0/070	0/339	-0/208	0/836
Regular dental visits	0/041	0/160	0/558	0/286	0/776
Plaque index	0/324	0/034	0/019	1/826	0/072

Table 7: Regression test results in determining the effects of different variables in relation to plaque index in the studied samples

Variable	Standard coefficients		Unstandardized coefficients		
	β	β	The standard error	t	Sig. (<0.1)
Cigarette use/year	0/333	0/028	0/007	4/172	0/000
Hookah/year	0/154	0/051	0/026	1/950	0/035
Toothbrushing/day	-0/509	-9/000	4/420	-2/240	0/028
Using toothpaste	-0/004	-0/091	4/987	-0/018	0/000
Sugar snacks consuming per day	0/340	8/494	1/920	4/418	0/000
Regular dental visits	-0/081	-2/968	3/574	-0/830	0/409

Table 8: Regression test results in determining the effects of different variables in relation to DMFT in the studied samples (without intervention of plaque index variable)

Variable	Standard coefficients		Unstandardized coefficients		t	Sig. (<0.1)
	β	β	The standard error			
Cigarette use/year	0/227	0/002	0/001		1/909	0/044
Hookah/year	0/00	0/004	0/004		0/000	0/369
Toothbrushing/day	0/000	-9/000	0/000		0/000	0/080
Using toothpaste	0/082	-0/091	0/000		0/018	0/792
Sugar snacks consuming per day	0/084	8/000	0/000		0/000	0/468
Regular dental visits	0/015	0/058	0/000		0/000	0/919

Table 9: Regression test results in determining the effects of different variables in relation to dmft in the studied samples (without intervention of plaque index variable)

Variable	Standard coefficients		Unstandardized coefficients		t	Sig. (<0.1)
	β	β	The standard error			
Cigarette use/year	0/552	0/012	0/002		6/000	0/001
Hookah/year	0/219	0/019	0/007		2/000	0/007
Toothbrushing/day	-0/172	-0/855	0/454		-1/000	0/064
Using toothpaste	0/216	0/932	1/050		1/023	0/010
Sugar snacks consuming per day	0/242	1/545	0/497		3/000	0/002
Regular dental visits	-0/170	-1/594	0/874		-1/000	0/073

Table 10: Pearson's correlation coefficient test to examine the relationship between cotinine with dmft and DMFT

	Cotinine	DMFT	dmft
DMFT	*0/323	-	-
dmft	*0/746	0/419	-

*p < 0.05

salivary cotinine was considered as an indicator of inactivity of smoker. In all equations, salivary cotinine was used instead of the annual amount of cigarette and hookah. All results were similar, and with increasing salivary cotinine levels, there was an increase in permanent and primary dental caries. This indicates that smoke of either cigarette or hookah increase caries through the increase in salivary cotinine or that cotinine is a very good marker for detecting exposure to cigarette smoke/hookah (Table 10).

DISCUSSION

Passive smoking is actually inhaling the smoke of others, which is one of the key issues that can lead to a ban on smoking in indoor public places, such as restaurants, airplanes, subway, etc.¹⁰ About 65–85% of the world's tobacco use constitutes smoking, and about 20% to 80% of the world's population is exposed to cigarette smoke and its harm. Passive smoking is approximately equal to active smoking, and children in their early years are more vulnerable than adults.³

In the present study, the effect of passive smoking on dental caries in 4–9-year-old children was investigated. The results show that children exposed to cigarette smoke have higher DMFT and dmft index, and this increase in caries is more evident in primary teeth. Many studies have yielded similar results to our study and have noted that passive smoking increases caries of primary teeth.^{4,5,11,12} In the case of permanent teeth, the effect of increased peripheral cigarette smoke has also been proven by studies.¹³

In their study of 1–14-year-old children, Tanaka et al. demonstrated a positive association between passive smoking and the experience of deciduous and permanent tooth decay in children. But unlike the present study, all components of the DMFT index were increased in their study. While in our study the overall DMFT values were higher, in the passive smoker group the only difference in the F component was significant in the fractional study. The reason for the difference in our study population is probably due to pain caused by dental caries, and more frequent follow-up and more treatments have been applied. The absence of M in samples of our study may also be related to our age-group, as younger teeth are less likely to lose permanent teeth (except in congenital absences).⁹

Leroy in 2008 showed that there was no significant relationship between parental smoking and dental caries in the age-group of 3 years, whereas this relationship was significant at 5 years. This may indicate that the effect of passive smoking on caries may be shown over time. In Leroy's study, smoking was measured by a questionnaire, while in this study we also measured salivary cotinine.⁶

In explaining the effect of environmental cigarette smoke on the caries rate of different studies, they have cited different overlaps, of course. Sakki states that passive smoking increases *Streptococcus mutans* and lactobacilli levels in the mouth. Nicotine from cigarette smoke also increases extracellular polysaccharides, which can attract microorganisms to the plaque.^{14–16} Or in a Swedish study, cigarette smoke has been implicated in reducing saliva buffering capacity.¹² People exposed to passive smoking also have lower sIgA levels.¹⁷

Studies have also shown that there is no relationship between passive smoking and dental caries. Nakayama et al. in the study of 5-year-old children showed that the main factor of caries in these children was the frequent consumption of sugary snacks, and no direct relationship with smoking was found.¹⁸

The differences in the results of different studies can be due to differences in factors such as type of study, controlling the confounding factors, populations of study with different cultures, different lifestyles and diet, etc. As shown in the results of this study, there seems to be a direct and net effect of passive smoking on caries rate, but it is low. Children in smoker families may be more likely to get caries because of other factors. Crawley states that these children have lower levels of fiber, vitamin C, vitamin E, and magnesium.¹² Hu also points out that children in these families eat breakfast more irregularly.¹⁹ These may confirm the different nutritional status of these families.

On the other hand, children in smoker families have been reported to have a lower social and cultural status and are less likely to deal with general and oral health status and good oral hygiene.^{6,20} This was also observed in our study, and children exposed to passive smoking had higher plaque index. It is clear that dental caries is a multifactorial disease caused by various physical, biological, and environmental factors. Passive smoking is one of the factors that can affect the rate of decay, but there are many other factors that have a greater impact. In the present study, salivary cotinine was considered as a function of passive smoker and had similar results compared to the rate of parental cigarette smoking. This may indicate that cotinine is an accurate marker for detecting passive smoker or show that cotinine itself, one of the metabolites of cigarettes, is effective in dental caries. Yamamoto in 2005 and Nishida in 2006 have also cited salivary cotinine as an appropriate marker for detecting exposure to passive smoking.^{21,22}

Goto also notes that serum nicotine has been showing passive smoker for up to 3 days back, but cotinine is a good marker for detecting the cumulative effect of passive smoking.²³

Our study also had limitations. This study is cross-sectional, and cross-sectional studies cannot necessarily show a causal relationship. Moreover, a much larger community is needed to prove such a relationship. In addition, in the higher sample size, the age categories can be examined more separately and with greater accuracy. In this study, although we have attempted to largely place or eliminate the effect of confounding factors, there are probably many other factors that may influence the rate of caries, including smoking during pregnancy, exposure to fluoride, etc. In addition, it is noteworthy that higher levels of F in DMFT in passive smoker samples in our study may not necessarily be due to decay, but it indicated more restorations due to dental traumas. Finally, more extensive studies are needed to precisely determine the relationship between cigarette smoke and caries. Overall, based on the results of regression tests, it was found that this effect of increasing caries may be related to the net effect of cigarette smoke on caries, but on the other hand, children exposed to cigarette smoke also had higher plaque-related indices. This indicates that these children have a higher plaque index for various reasons (including family culture, etc.), and this increases their caries index and tooth decay index (indirect effect).^{4,5,17}

CONCLUSION

According to the results of this study, children exposed to passive smoking have a higher rate of decay than other children. It seems that this effect is primarily due to its effect on increasing plaque accumulation, the damaging effect of passive smoker on the physiological structures of the mouth, etc.

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