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International Journal of Oral Health Dentistry

Journal homepage: www.ijohd.org

Original Research Article

Association between passive smoking and dental caries among 3-8 years children in Bangalore city

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ARTICLE INFO

Article history:

Received 23-06-2021

Accepted 22-07-2021

Available online 24-09-2021

Keywords:

Passive smoking

Dental caries

Salivary flow rate.

ABSTRACT

Background: Salivary antimicrobial peptides (AMP's) play an important role in the local defense of oral cavity and expression of these are altered by various factors. Among them cigarette smoke has known to have detrimental effects on salivary immune defense mechanisms. The effect of passive smoking on salivary AMP's and correlation to dental caries in children has not yet been reported. Thus, this study was aimed to assess the correlation between passive smoking and dental caries in exposed and unexposed children.

Materials and Methods: A randomized cross-sectional study was designed to target children aged between 3 to 8 years. Self-reported questionnaire was filled by the parents of the participants to obtain the data. Questionnaires included in the study comprised sections related to demographic part and smoking characteristics. Participants were divided into passive exposed (PE) and unexposed (UE) group based on exposure parameters. Clinical examination and Salivary flow rate were assessed and the obtained data was subjected to statistical analysis. TPE group (5.58+/-4.66) than UE group (3.15+/-3.26) where (p=0.003) and under educated parents (p=0.02). Comparison of smoking related parameters to caries prevalence have shown direct positive correlation.

Results: [K1] [M2] : The mean DMF scores were higher among children in [K3] [K4] [M5] E group (5.58+/-4.66) than UE group (3.15+/-3.26) where (p=0.003). Also it was seen that the DMF were higher in children who belonged to lower socio-economic status (p= 0.001) and under educated parents (p=0.02). Comparison of smoking related parameters to caries prevalence have shown direct positive correlation.

Conclusion: Reduction of passive smoking is important not only for the prevention of various systemic ill-effects, but also for the promotion of children's dental health.

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1. Introduction

Oral cavity is a large microbial complex ecosystem consisting of oral fluids, immunoglobulin, agglutinins, microflora, carbohydrates, proteins and certain antimicrobial enzymes. The abundance of oral microbes results in the formation of dental biofilm which on contact with the host may result in disturbances in tissue homeostasis and

subsequent disease like dental caries.¹

Global Burden of Disease 2010 Study reported that the global prevalence of untreated caries was the highest and its global burden is ranked 80th.² Carious lesion constitutes a progressive infectious process with multifactorial aetiology. Besides dietary habits, oral microorganisms, and host susceptibility, some risk factors, such as sex, age, dietary habits, socioeconomic, exposure to environmental tobacco smoke and oral hygiene status, are also associated with increased prevalence and incidence of dental caries in

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paediatric population.³

The association between second-hand smoking and dental caries have been revealed by epidemiological survey by various authors^{4–7} and several studies have also shown dose-response relationship between the extent of smoking by parents and unfavorable effects on children.^{8,9} Thus, it is not surprising that SHS would be an independent risk factor for dental caries development.

Passive smoking(PS), refers to the smoke discharged from the lit end of a burned tobacco product as well as the smoke exhaled during active smoking.¹⁰ The World Health Organization (WHO) has estimated that almost one-half of the world's children (nearly 700 million) are exposed to tobacco smoke from the 1.2 billion adults who smoke.¹¹ Furthermore, infants and children are generally not capable to manage their environment & consequently unable to perform action to escape from second hand exposure (SHS) because of low-socioeconomic status, under educated parents and small house which constrains parents to smoke inside the house.¹²

Exposure to PS can be assessed Quantitatively by determining the concentration of the most important metabolite of cotinine (by product of nicotine). It is found in blood, saliva and urine after exposure to nicotine and the levels are elevated in passive smokers. Measurement of cotinine in body fluids are sensitive technique to assess passive smoking exposure.¹³ In saliva, values between 1 ng/mL and 30 ng/mL may be associated with light smoking or passive exposure, and levels in active smokers typically reach 100 ng/mL or more.¹⁴

Children are comparatively more vulnerable to systemic ill effects of second-hand smoke effects because of higher breathing rates per body weight, immature lungs and more lung surface area compared with adults.¹⁵ SHS not only affects their general health, also few studies^{16–18} have proposed a positive association between SHS exposure and oral health. The risk of developing dental caries by exposure to secondhand smoke may be explained by three major mechanisms: (1) direct exposure of the developing teeth buds to chemicals of smoke, leading to delay in the formation and impair of mineralization;¹⁹ (2) damage to salivary glands by chemicals of smoke, resulting in a decreased salivary flow, which affects buffering capacity and cleansing mechanism;²⁰ and (3) Secondhand smoking also impairs the salivary immune system by reducing the salivary Antimicrobial Peptides (AMP) and flow rate in children and simultaneously increases the colonization of *Streptococcus mutans*, which have been attributed to the formation of dental caries.^{21,22}

It is biologically plausible that passive smoking could cause caries, particularly at childhood and there is lack of literature regarding the same. Hence, this study was designed to assess the correlation between passive smoking and dental caries in exposed and unexposed children and

possible correlation to age, gender, salivary flow rate, salivary AMP dental caries experience was determined

2. Materials and Methods

2.1. Setting and population

A randomized case-control study was conducted in Bangalore (Karnataka) among 120 children aged between 3–8 years.

2.2. Ethics study consent

The study was conducted during 2019–2020 after obtaining Ethical approval for the study from the ethical clearance committee of the institution. Written informed consent was taken from parents before implementing the study.

2.3. Study questionnaire

This study was carried out among 120 children .Self-administered close-ended questionnaire was used for parents to assess exposure to second-hand-smoking among primary caregivers of children in India (by WHO) [Table 1] which was printed both in English and regional language (Kannada). It comprised of a demographic part including age, gender, family income, and educational level of parent, and smoking characteristics part including number of smokers at home, smoking location, smoking rules or any other exposure to passive smoke in past 7 days as cotinine in biological materials is widely used and suitable for assessment of doses over short periods of time (from 1 to 10 days, in urine, plasma, or saliva).

2.4. Sample size

The sample size was calculated based on confidence level of 95%, confidence interval of 5%, and estimated population response distribution of 50%. The sample size obtained using this calculation was 120 participants. The participants were obtained from the OPD, Department of Paediatrics and Preventive Dentistry, AECS Maaruti dental college, Bangalore. However, upon scrutinizing data from questionnaire and rapid salivary cotinine test the participants were divided into PE group (case) and UE group (control).

2.5. Study procedure

The study was conducted by:

1. a) Assessing self-reports questionnaire,
2. b) Clinical dental examination &
3. c) Salivary flow rate estimation.

2.5.1. a. Assessing self-reported questionnaire

The self-reported questionnaires were given to the parents to assess smoking habits of the family members and the

subjects were then divided into two groups with 60 samples each using simple stratified sampling methods.

1. Group 1 Passive smoke exposed (PE) (n=60)
2. Group 2 Unexposed (UE) (n=60)

For further confirmation, passive smoke exposed samples underwent screening for cotinine levels estimation using *JusCheck rapid nicotine/cotinine test kits*. (Rapid, self-controlled, immunoassay for the qualitative detection of Cotinine in human saliva).

2.5.2. Inclusion & exclusion criteria

Children who were exposed and unexposed to passive smoking, aged between 3-8 years with presence of dental caries, filled or extracted teeth due to carious lesion with prior parental consent were included in the study. The exclusion criteria included children with systemic diseases, long term medication., administration of antibiotics less than one month before and Children with filled or extracted teeth due to non-carious causes.

2.5.2.1. b. Clinical dental examination. Clinical examination was performed by a single calibrated examiner using mouth mirror and straight probe under the natural light. decayed, extracted and filled surfaces (def) in deciduous teeth & Decayed, Missing & Filled surfaces in permanent teeth were counted in each subject. [according to Gruebell. A.O in 1944]. Both groups were then further divided into subgroups based on def/DMFT scores.

2.5.2.2. Salivary flow rate estimation . Children were instructed not to eat or chew anything for at least 1 hour before sample collection and were instructed to spit the unstimulated saliva into the graduated polypropylene tubes for 1 min. The unstimulated salivary flow rate was determined by measuring the saliva collected in graduated tubes.

2.6. Statistical analysis

Data were analyzed using the IBM Statistical Package for the Social Sciences (SPSS) (IBM, Armonk, New York) software, version 22.0. Statistical tests included frequency distribution: mean values and standard deviations (SDs), t test, Mann Whitney test to determine difference in mean values of DMFT & salivary flow rate among children who are exposed or not exposed to passive smoking.

3. Results

A total of 120 Participants, participated in the present case-control study after filling the given questionnaire and thorough clinical examination. Among them, the subjects were divided into PE and UE groups.

3.1. Socio demographic analysis

Among 120 participants the number of male children were 56 (PE-26 & UE-30) and the number of female children were 64 (PE-34 & UE-30) [Table 2] The mean age of the participants in the PE group was 5.18+/-1.57 and 5.55+/-1.50 in UE group, which showed no statistical difference between the two groups (p=0.19). 70% (n=20) of the parents in PE group had education status below matriculation whereas 66.7%(n=40) of the parents in UE group were better qualified. Also, majority of the parents 78.3%(n=47) in UE group belonged to high income (>1,00,000 p.a) status, unlike PE group where 48.3% belonged to medium income status (50,000 - >1,00,000 p.a) depicting statistically significant difference in the education status & income level between two groups (p=0.001).

3.2. Caries prevalence analysis

The mean DMF score was significantly higher in PE group (5.58+/-4.66) whereas (3.15+/-3.26) in UE group, having statistical difference between the two groups. The difference in means was compared using Mann Whitney test and the results were highly significant with P < 0.001. [Table 3]

3.3. Salivary flow rate analysis

Table 3 Shows the mean SFR was 1.82+/-0.77 in the PE group and 1.98+/-0.54 in the UE group which was compared using Mann Whitney test and the results showed that there was no statistically significant difference between the two groups (P=0.16)

3.4. Analysis of distribution of smoking related characteristics in PE group

Table 4 Represents the distribution of smoking related characteristics among the people at home Among 60 PE participants, 78.3% (n=47) of the participants had at least single smoker at home whereas 21.7% of the participants had two or more smokers at home. In 71.7%(n=43) of the participants the parents smoked both inside & outside. In 65% of houses there were no rules with respect to smoking. 61.7%(n=37) of the participants had history of being exposed to smoke for >3 years and 41.7% (n=25) of the parents had history of smoking 5-10 cigarettes/day.

3.5. Comparison of smoking exposure to caries

It was seen that as smoking exposure and DMFS had a positive correlation. The mean DMF score was 9.38+/-4.23 who were exposed to two or more smokers at home than one smoker with DMF score 4.53+/-4.24. Also DMF score was significantly higher (6.93+/-4.48) in children, whose parents smoked both inside & outside and (6.62+/-4.64) in families who had no rules regarding smoking habits. There was also positive correlation in DMF score & duration of smoking.

Table 1: Questionnaire to assess self-reported exposure to second-hand-smoking among primary caregivers of children <5 years of age in India (according to WHO):

1. Date	
2. Name	
3. Age	
4. Sex	
5. Address	
6. Over the past 7 days, has your child been around smoke from tobacco? Do you remember smelling cigarette, bidi, hookah smoke when your child was present?	Yes No
7. Over the past 7 days, did your child visit other people's home? If yes, did you smell cigarette, bidis or hookah smoke?	Yes No
8. Over the past 7 days, did your child visit markets, restaurants or public places? If yes, did you smell cigarette, bidis or hookah smoke?	Yes No
9. Over the past 7 days, did your child use public transportation (auto or buses)? If yes, did you smell cigarette, bidis or hookah smoke?	Yes No
10. How many people who currently live in your home smoke cigarettes or bidis?	
11. Over the past 3 months, has anyone smoke anywhere inside your home?	Yes No
12. Where do people smoke when they are at home?	a) Inside only b) Inside and outside c) Outside d) Depends on the season
13. How often does anyone, including visitors, smoke cigarettes or bidis inside your home?	a) Daily b) Weekly c) Monthly d) Sometimes/ e) Never
14. Which best describes how cigarette and bidi smoking is handled in your home?	a) No rules b) Smoking is permitted anywhere c) Smoking is permitted in some d) No one is allowed to smoke anywhere?
15. For how many years do you think your child has been exposed to tobacco smoke?	a) 0-6 months b) 6 months-3 years c) 3 years-6 years
16. Parent's education:	a) Up to matriculation b) Above matriculation
17. Parents income:	a) <50 thousand p.a b) 50 thousand-1 lakh p.a c) > 1 lakh p.a
Name and signature of the volunteer:	Signature of the investigator
Date:	
Place:	

Children who had history of exposure to smoke >3 years had mean DMF score of 8.22+/-3.17 than children exposed to smoke <6 months with absence of caries score. Frequency of smoking also have a significant correlation with respect to DMF score, where DMF was 11.91+/-2.55 in children whose parents smoke > 10 cigarettes/day. (Table 5)

3.6. Comparison of smoking exposure to educational status and income levels of the parents

From our data it was seen that, mean DMF score in the PE group had positive correlation with education status and income levels of parents with higher DMF score of

7.02+/-4.431 in parents with below matriculation education status & DMF score of 13.50+/-0.71 in parents with low income levels (<50,000 p.a). Thus, it was evident that with increase in the educational level and income levels of the parents, the level of smoking exposure decreased in household due to better awareness than under educated and low-socioeconomic status groups thereby reducing caries. (Table 6 A: & B:)

4. Discussion

Since decades, smoking is known as a potential risk factor and a major preventable cause of morbidity and

Table 2: Comparison of demographic characteristics among 2 groups

Variable	Category	Group 1		Group 2		P-Value
		Mean	SD	Mean	SD	
Age	Mean & SD	5.18	1.57	5.55	1.5	0.19a
	Range	03-Aug		03-Aug		
Sex		n	%	n	%	0.46b
	Males	26	43.30%	30	50.00%	
Income Level	Females	34	56.70%	30	50.00%	0.002*
	< 50, 000	2	3.30%	0	0.00%	
Education	> 50, 000 & < 1, 00, 000	29	48.30%	13	21.70%	0.001*
	> 1, 00, 000	29	48.30%	47	78.30%	
Up to Matriculation	Up to Matriculation	42	70.00%	20	33.30%	0.001*
	Above Matriculation	18	30.00%	40	66.70%	

Table 3: Comparison of mean values of dmf scores & sfr between 2 groups using mann whitney test

Parameters	Groups	N	Mean	SD	Mean diff	P-Value
DMFS	Group 1	60	5.58	4.66	2.43	0.003*
	Group 2	60	3.15	3.26		
Salivary flow rate	Group 1	60	1.824	0.775	-0.16	0.16
	Group 2	60	1.983	0.545		

Table 4: Distribution of smoking related characteristics among the people at home in PE group

Variable	Category	n	%
No. of smokers at house	One	47	78.3%
	Two	13	21.7%
Smoking location	Inside only	1	1.7%
	Outside only	3	5.0%
	Inside & Outside	43	71.7%
Smoking rules	Based on Season	13	21.7%
	Smoking is permitted in some places	9	15.0%
	Smoking is permitted everywhere	12	20.0%
	No rules	39	65.0%
Duration of smoking	< 6 Months	4	6.7%
	> 6 Months & < 3 years	19	31.7%
Frequency of smoking	> 3 years	37	61.7%
	< 5 nos.	24	40.0%
	> 5 & < 10 nos.	25	41.7%
	> 10 nos.	11	18.3%

mortality. Thousands of chemicals are present in complex aerosol of cigarette which contains volatile gases with suspension of particulate matter.²³

Several hypotheses support the biological plausibility of the association and explains the causal mechanism of caries due to passive tobacco exposure. Our study demonstrated that the mean DMF score was significantly higher in PE group children than those in UE group children. Similarly, Chowdhury and Bromage²⁴ and Heikkinen et al.²⁵ have also stated that exposure to tobacco use directly affects both the mineralisation of the developing tooth and the microorganisms, while cross-sectional study by Mattheus et al.²⁶ revealed that caries prevalence was 1.59 times more

in children who were exposed to smoke inside the house in comparison to smoke outside. Another study conducted by Tanaka et al.²⁷ also stated that exposure to tobacco smoke at 4 months of age was associated with an approximately two fold increased risk of caries.

Avçar et al.⁴ observed that children exposed to tobacco use had lower salivary pH, buffer capacity and saliva flow than non-exposed children however our study showed a contrasting result with no significance difference in the SFR between PE and UE groups.

The present study showed (table-7) increased DMF score of 13.1+/-0.71 in parents with low income followed by mean DMF score 7.35+/- 4.07 in parents with medium

Table 5: Comparison of mean dental caries scores based on the smoking related characteristics in group 1

Variable	Category	DMFS		P-Value
		Mean	SD	
No. of smokers at house	One	4.53	4.24	0.001*
	Two	9.38	4.23	
Smoking location	Inside only	5.00	.	0.003*
	Outside only	0.00	0.00	
	Inside & Outside	6.93	4.48	
Smoking rules	Based on Season	2.46	3.43	0.008*
	Smoking is permitted in some places	1.22	2.44	
	Smoking is permitted everywhere	5.50	4.32	
	No rules	6.62	4.64	
Duration of Smoking	< 6 Months	0.00	0.00	<0.001*
	> 6 Months & < 3 years	1.63	3.67	
	> 3 years	8.22	3.17	
Frequency of smoking	< 5 nos.	2.75	3.14	<0.001*
	> 5 & < 10 nos.	5.52	3.86	
	> 10 nos.	11.91	2.55	

Table 6: A: Comparison of mean values of DMF score based on the educational levels in PE group using mann whitney test

Parameters	Education	N	Mean	SD	Mean Diff	P-Value
DMF scores	Up to Matriculation	42	7.02	4.431	4.80	<0.001*
	Above Matriculation	18	2.22	3.318		

Table-6 B: Comparison of mean values of DMF score based on the income levels in PE group using kruskal wallis test followed by mann whitney post hoc test

Parameters	Category		Mean	SD	P-Value ^a	Sig. Diff	P-Value ^b
DMF Scores	< 50,000	2	13.50	0.71		L1 vs L2	0.02*
	> 50,000 & < 1,00,000	29	7.38	4.07	<0.001*	L1 vs L3	0.02*
	> 1,00,000	29	3.24	3.97		L2 vs L3	<0.001*

income and mean DMF score of 3.24+/-3.97 in high income parents, which indicated a negative correlation between DMF score and socioeconomic status of the parents. Jakhete and Gitterman²⁸ have also stated that exposure to tobacco use were associated with higher caries prevalence in children from a low socio-economic level. Delpisheh²⁹ confirmed that passive exposure to tobacco use in children is significantly associated with low socio-economic level. Majorama et al.³⁰ found that children who lived in families from a low socio-economic level and were exposed to tobacco use had a greater likelihood of suffering severe caries.

Our study revealed that high parental education was associated with low prevalence of dental caries, (table-8) where higher DMF score of 7.02+/-4.431 was seen in parents with below matriculation education status & DMF score of 2.22+/-3.18 in parents with above matriculation similarly, Study results by Alfred et al.³¹ and Tanka et al.²⁷ substantiate our results which showed education status of the parents were responsible for increased awareness.

The dose-response relationship between levels of exposure to tobacco use and dental caries has been studied by several authors^{22,32-34} all of whom have confirmed this relationship. Our study was found to be similar, (table -9) represents the comparison of mean DMF scores based on the smoking related characteristics. DMF score was 9.38+/-4.23 in children who were exposed to two smokers at home than one smoker with DMF score 4.53+/-4.24. Also DMF score was significantly higher (6.93+/-4.48) in children, whose parents smoked inside & outside and (6.62+/-4.64) in families who had no rules regarding smoking habits. There was also positive correlation in DMF score & duration of smoking. Children who had history of exposure to smoke >3 years had mean DMF score of 8.22+/-3.17 than children exposed to smoke <6 months with absence of caries score. Frequency of smoking also have a significant correlation with respect to DMF score, where DMF was 11.91+/-2.55 in children whose parents smoke > 10 cigarettes/day.

Exposure to tobacco smoke, which contains numerous chemical toxins, might predispose children to infection

through suppression or modulation of the immune system.³⁵

Numabae et al.³⁶ showed that the phagocytic activity of salivary PMN's intensifies after exposure to smoking, where as an another invitro study³⁷ demonstrated that nicotine inhibited phagocytic activity. Sakki and Knuutila³⁸ showed that tobacco smoking was associated with elevated levels of mutans streptococci and lactobacillus. Yet on the other hand, epidemiological studies on the association between passive smoke exposure and dental caries showed inconsistent results because of unknown factors related to passive smoking which may have confounded the observed relationship.

This study also shows long-term impact of smoking in household on their children which serves as an important motivating factor for their parents to quit smoking and the study also highlights PS as health hazard which is not known by many people in study setting and hence, serves as an important enlightening message.

Present study results showed that passive smoking was positively associated with the prevalence of dental caries, due to the suppression of Salivary AMP, which predisposes to dental caries. However, the limitation is small sample size and assessment of SHS exposure was which was obtained by questionnaire reports and was not validated by measurements of biomarkers, such as salivary cotinine levels. Using questionnaires may result in misclassification from recall bias and response bias due to parents' feelings of guilt for smoking in the presence of their children. Although the biological and behavioural plausibility for an etiological relationship between SHS exposure and dental caries is likely to be high, epidemiological evidence is still insufficient. The addition of objective markers of SHS exposure may help to clarify the role of SHS exposure on dental caries in further research. Hence the correlation between the Salivary AMP, Sal. Cotinine concentration and dental caries will be assessed in the phase 2 of the study by quantitative analysis of Salivary cotinine levels and Salivary AMP's.

5. Conclusion

Dental caries is a common public health problem among children due to multi factorial aetiological agents. Our results revealed that passive exposure to tobacco smoke had independent relationship with dental caries in Bangalore children. Although the relationship between passive smoking and dental caries is likely to be high, epidemiological evidences are insufficient. The addition of objective markers in regard to passive smoke exposure may help to clarify the role of passive smoking exposure and caries correlation by additional studies.

6. Source of Funding

None.

7. Conflict of Interest

None.

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Cite this article: Agarwal M, Rao CSS, Konde S, Peethambar P, Prasad SN. Association between passive smoking and dental caries among 3-8 years children in Bangalore city. *Int J Oral Health Dent* 2021;7(3):187-194.