Original Article

PASSIVE SMOKING AND PEDIATRIC DENTAL CARIES

Sirajam Munira[1], Md. Ziaul Islam PhD[2], Md. Towfiq Sekander[3], Badrul Alam[4]

ABSTRACT

Background: Dental caries is a chronic dental disorder of children and it can result in substantial morbidity due to pain, dysfunction, poor appearance, and problems of speech development. The study was conducted to assess the association between passive smoking and pediatric dental caries.

Methods: The cross-sectional study was conducted among 92 school children aged 5-10 years. Data on exposure to passive smoking at both indoor and outdoor were obtained through face to face interview of the parents with the help of semi-structured questionnaire and checklist. Dental caries was diagnosed by clinical examination and passive smoking exposure was assessed by estimated urinary cotinine level.

Results: The study revealed that 58.70% children were male and 41% were female respectively. The mean age of the children was 7.20 years. It was found that 59.8% father of the studied children were smoker where 68.5% children reported that some of their family members were indoor smokers. Of all the children, 62% had dental caries and 85.5% of the children had dental caries who were exposed to passive smoking and had significant association between exposure to passive smoking and dental caries ($p<0.001$). Dental caries was common among children aged 5-6years who were exposed to passive smoking. The mean urinary cotinine level was 27.81ng/ml and maximum children (65.2%) had urinary cotinine level more than 10ng/ml who was exposed to passive smoking. The study found significant positive correlation between urinary cotinine concentrations and the duration of exposure to passive smoking ($r=0.692$, $p<0.001$).

Conclusion: This study suggests reduction of exposure to passive smoking may be an effective measure for prevention of pediatric dental caries.

JOPSOM 2020; 39(1): 1-13
https://doi.org/10.3329/jopsom.v39i1.51856

Key Words: Passive smoking; Children; Dental caries; Urinary cotinine

---

[1]*Student, MPH (HP&HE) Program, NIPSOM, Mohakhali, Dhaka-1212, Bangladesh*
[2] *Professor and Head, Department of Community Medicine, NIPSOM, Mohakhali, Dhaka, Bangladesh*
[3]*Medical Officer, Dhaka Medical College Hospital, Dhaka*
[4]*Assistant Professor, Department of Nutrition and Biochemistry, NIPSOM, Mohakhali, Dhaka, Bangladesh*

Correspondence:
Prof. Dr. Md. Ziaul Islam, PhD
Head, Department of Community Medicine, NIPSOM
Mohakhali, Dhaka-1212, Bangladesh
e-mail: dr.ziaul.islam@gmail.com

INTRODUCTION

Dental caries is an infectious, communicable and multifactorial disease. The interaction of a susceptible tooth surface, fermentable carbohydrates and acid producing bacteria leads to dental caries after tooth eruption. Specific bacteria, in particular, Streptococcus mutans produce lactic acid from the fermentation of carbohydrates. Prolonged lowering of the pH in dental plaque induces demineralization of the tooth surface, resulting in the destruction of the hard structure of the tooth. Thus, the etiology of dental caries is chronic. Various factors may influence the disease process. Behavioral factors such as poor oral hygiene and the consumption of sugar-containing snacks are significant factors for caries lesion initiation [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.

Even in the developed countries the prevalence of caries in deciduous teeth remains high, 20.5% in children aged 2 to 5 years in the United States [2] and established measures for caries prevention in young children is limited to sugar restriction, oral fluoride supplementation and fluoride varnish [3].

Passive smoking, also known as "environmental tobacco smoke (ETS), refers to the smoke discharged from the lit end of a burned tobacco product as well as the smoke exhaled during active smoking. There are more than 4000 chemicals present in passive smoking, and more than 250 of these are known to be carcinogenic or toxic in some other way. [4] Passive smoke is about 4 times more toxic than mainstream smoke, although people inhale it in a more diluted form [5].

The World Health Organization classifies smoking as a chronic, progressive disease which is also contagious. Approximately 5 million people are killed annually by tobacco use. By the year 2030, according to current trends, it is assumed that this number will increase to 10 million with 70% of deaths occurring in low- and middle-income countries [6].

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, and the majority of exposures take place at home [7]. Children are frequently exposed to passive smoke and it can induce serious diseases like pneumonia, otitis media, asthma, reduction in pulmonary function, malignancies [8] and dental caries [9] in children. Children are exposed to passive smoke in many places including stores, restaurants, and other public spaces, but the most concentrated exposure is probably in the home, from both residents and visitors, and in the vehicles of smoking parents, caregivers or others.

Passive smoking may directly influence teeth and microorganisms. Passive smoking could promote the growth of cariogenic Streptococcus mutans which influence the formation of dental caries [6]. The adverse effects of passive smoking include decreased serum vitamin C levels in children and decreased levels of vitamin C are associated with growth of cariogenic bacteria. Passive smoking smoke has immunosuppressive properties that might be risk factor for caries development [6].

It is also possible that passive smoking may reduce the protective properties of saliva that can operate against caries. Saliva acts as buffering agent when acids are produced. It physically removes debris from the tooth surface and it has immunological and bacteriostatic properties [6].

Children exposed to passive smoking also have lower salivary IgA and higher levels of sialic acid with higher activity. Sialic acid enhances agglutination of Streptococcus mutans, leading to the formation of dental plaque and caries. Besides the salivary IgA antibodies can play an important role in control of dental caries. In addition to the direct effects of passive smoking, inhibition of the morphology and mineralization of dental hard tissue in the exposed to passive smoking was also reported [10].

Passive smoking is known to increase inflammation of respiratory tract, producing symptoms of various clinical condition including allergic rhinitis, which frequently cause mouth breathing and thus result in dry mouth (i.e. an effective decrease in saliva) [6]. Children also breathe faster and thus take in more harmful chemicals per kilogram of body weight than adult [7]. In addition, children often sit closer to their parents, family members or caregivers making them closer to the source of pollutants than other passive smokers [11].

The bacteria responsible for caries formation are acquired in infancy from the saliva of mothers (via kissing, etc.). Elsewhere, nicotine has been shown to promote the growth of cariogenic Streptococcus mutans bacteria in vitro; thus, mothers who smoke may be more likely than nonsmokers to transmit these germs to their children [9].

Furthermore, in early childhood, when immune system is generally less mature, the saliva is known to be different from that of adult with respect to IgA concentrations. In addition, salivary flow rate in children is lower. Young children may thus be particularly vulnerable to harmful effect of passive smoking on immune system and saliva flow. Hence, it may possible that passive smoking could cause caries, particularly in early childhood. One possible explanation suggests that ETS exposure is more likely to cause dental caries in deciduous teeth rather than permanent teeth is because enamel of deciduous teeth is much thinner compared to permanent teeth, and that enamel defect are associated with caries [6].

Quantitatively, the most important metabolite of nicotine in most mammalian species is the lactam derivative, cotinine. In humans, about 70 80% of nicotine is converted to cotinine [12]. Cotinine has a much longer half-life (about 18-20h) than nicotine (half-life about 2h), resulting in higher and more stable plasma concentrations. Cotinine is found in blood, saliva and urine after exposure to nicotine and its urinary levels are elevated in passive smokers. Cotinine can be measured with adequate sensitivity to assess passive smoking exposure[13]. The association of pediatric dental caries with serum level of cotinine

was demonstrated previously utilizing the National Health and Nutrition Examination Survey (NHANES III) database [9].

Several studies have also shown dose-response relationship between the extent of smoking by parents and the salivary, urinary or plasma cotinine level in children [14].

With the reported high prevalence of pediatric dental caries and passive smoking among Bangladeshi population, there is limited information on Cotinine level in Bangladeshi children exposed to passive smoking. The study aimed to explore the association between passive smoking, as determined by urinary cotinine level and dental caries in children.

METHODS

This was a cross sectional study to find out the association between passive smoking and pediatric dental caries. Children were selected from BEPZA (Bangladesh Export Processing Zone Authority) School and College, Chittagong and Katakhali Government Primary School, Chittagong. Both institutions are located in South Halisahar in the vicinity of Chittagong Export Processing Zone (CEPZ).

In this study, children were selected from primary school attending nursery to class 4 and aged between 5 to 10 years as a study population. All of the children (both male and female) who were exposed and not exposed to passive smoking were included in the study. A total 92 children were selected with informed consent of their parents who agreed to give urine samples for analysis of cotinine level in this study.

A semi-structured questionnaire in Bengali was used for data collection. A check list was used to note down the examined caries status and measured urinary cotinine level of the children. To measure urinary cotinine level of children exposed to passive smoking "IMMULITE 1000" machine was used. Dental caries was detected by clinical examination. Children were classified as having dental caries and not having dental caries. A history of passive smoking exposure was derived from questions. Then we compared the relationship of passive smoking between children with and without dental caries. Children were selected following systematic random sampling. Sampling interval was determined by roll number of students in each class and sample size of that respective class. Accordingly, children were selected randomly on the basis of roll number and sampling interval. The data were collected by face to face interview, questions were asked and responses were noted in the questionnaire. After taking informed consent from the respective parent, oral examination of the children was done and findings were noted in the checklist.

Urine samples were collected from all children. 15-20ml urine samples were obtained at schools under supervision in polypropylene containers. Urine samples were collected without preservation, then brought to the laboratory and were centrifuged at 4000 rpm for 15 minutes. The supernatant (clear top phase) of the sample was pipetted into Eppendorf using a micropipette and preserved in refrigerator at 2 to 8°C temperature. Samples were analyzed for cotinine, in "IMMULITE 1000" machine using chemiluminescent immunoassay method.

Data were analyzed by computer using SPSS window version 23. Descriptive statistics included mean, median, mode, SD. Inferential statistics included chi-square and Fisher'sExact test to find out any significant relationship between two qualitative variables. And to find out any significant relationship between two quantitative variables we did Pearson-correlation was done.

RESULTS

The study was carried out among 92 Bangladeshi school children aged 5 to 10 years. Data were collected through face to face interview of their parents and to diagnose caries status of children clinical examination was done and urine was collected from them to measure cotinine level, which is biomarker of nicotine. This study focused on exploring an association between passive smoking and dental caries in children. The study findings are being presented both in tabular and graphical form.

Socio-demographic characteristics of the children

The study revealed that among 92 children, 38 (41.3%) were within 5-6 years of age. The mean age of the children was found 7.20 ± 1.549 (Mean ± SD). 54 (58.70%) were male. Maximum that is 62(67.39%) families were nuclear. The mean number of family member was found 5.50 ± 1.831(Mean ± SD). Among all 62 (67.39%) lived in pucca house (Table 1).

Table 1: Socio-demographic characteristics of the children (n=92)

Category	Frequency	Percentage	Mean±SD
Age			
5-6	38	41.3	
7-8	34	37.0	7.20 ± 1.549
9-10	20	21.7	
Sex			
Male	54	58.70	
Female	38	41.30	
Type of Family			
Nuclear	62	67.39	
Combined	32	32.62	
Family member			
3-5	73	79.3	
6-8	13	14.1	5.50 ± 1.83
9-11	6	6.5	
Housing condition			
Pucca House	62	67.39	
Semi-Pucca House	30	32.62	

Distribution of children by exposure to passive smoking

Among all maximum numbers 55(59.8%) were exposed to second hand smoke due to smoking habit of their father. Maximum 63(68.48%) children had at least one family member who smoke at his/her sleeping room. The mean exposure duration of the study population to cigarette smoke inside home was 1.37 ± 0.64(Mean ± SD). The mean duration of exposure to outdoor cigarette smoke was 1.00 ± 0.46 (Mean ± SD). The mean of total (indoor and outdoor) exposure duration to cigarette smoke is 1.87 ± 1.12 (Mean ± SD) (Table 2).

Table 2: Distribution of children by exposure to passive smoking (n=92)

Category	Frequency	Percentage	Mean±SD
Relation of the children with smokers			
Father	55	59.8	
Brother	15	16.3	
Grand-father	10	10.9	
Uncle	12	13.0	
Family members smoked at room			
Yes	63	68.48	
No	29	31.52	

Inside exposure to the smoke			
<1	21	22.8	
1-2	29	31.5	1.37 ± 0.64
>2	13	14.1	
Outside exposure to the smoke			
<1	37	40.2	
1-2	4	4.3	1.00 ± 0.46
>2	4	4.3	
Both inside and outside exposure to the smoke			
<1	22	23.9	
1-2	19	20.7	
2.1-3	18	19.6	1.87± 1.12
>3	11	12.0	

Caries status of child

Upon clinical examination of 92 children, dental caries was present in maximum 62(67.39%) children, 30 children (32.65%) didn't have dental caries (Figure 1).

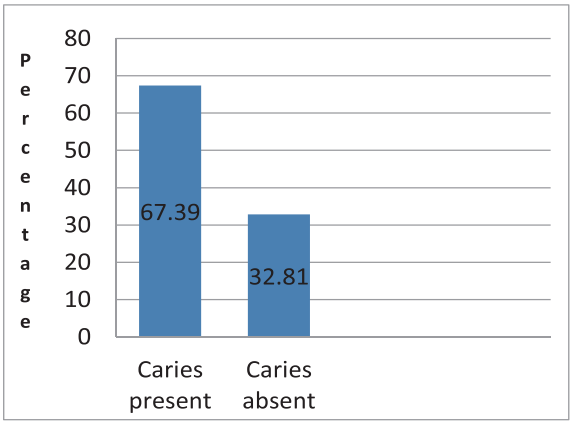

Figure: 1 Distribution of the children by dental caries

Exposure to passive smoking

Exposure to passive smoking evidenced by measuring the urinary cotinine level of the 92 children, maximum number of children 60 (65.22%) had urinary cotinine level more than 10 ng/ml and 32(34.78%) children had urinary cotinine level less than 10 ng/ml. Maximum level of urinary cotinine detected 446ng/ml, whereas minimum level was 10ng/ml (Figure 2).

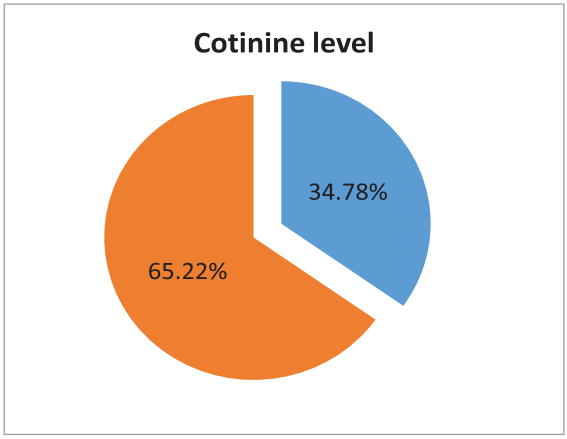

Figure: 2 Distribution of children by urinary cotinine level

Association between socio-demographic characteristics and dental caries

Among all children, caries was prevalent in 41(75.9%) male. There was statistically significant difference in the caries prevalence between male and female (χ 2 = 4.333, p< 0.04).62 came from nuclear family and maximum 40(64.5%) of them had dental caries. Children aged between 5-6 years and most caries was prevalent in this group 32(84.2%), (χ 2 = 8.34 p <

0.02). There was association between age of the children and presence of dental caries. Study showed 12 (100%) children were from Nursery and all of them had dental caries. (Fischer's exact = 11.941, p= 0.028) (Table 3).

Table-3: Association between exposure of children to passive smoking and dental caries

Category	Caries status		Total f (%)	Significance
	Present f (%)	Absent f (%)		
Sex				
Male	41(75.9)	13(24.1)	54(100)	χ^2=4.333
Female	21(55.3)	17(44.7)	38(100)	df=1
Total	62(67.4)	30(32.6)	92(100)	p-value=.04
Type of family				
Nuclear	40(64.5)	22(35.5)	62(100)	χ^2=0.715
Combined	22(73.3)	8(26.7)	30(100)	df=1
Total	62(67.4)	30(32.6)	92(100)	p-value=0.39
Age in year				
5-6	32(84.2)	6(15.8)	38(100)	χ^2=8.34
7-8	19(55.9)	15(44.1)	34(100)	df=2
9-10	11(55)	9(45.0)	20(100)	p-value=0.02
Total	62(67.4)	30(32.6)	92(100)	
Educational level of children				
Nursery	12(100)	0(0)	12(100)	
K.G	9(50)	9(50)	18(100)	
Class 1	22(71)	9(29.0)	31(100)	χ^2=11.94
Class 2	11(73.3)	4(26.7)	15(100)	df=5
Class 3	6(50)	6(50)	12(100)	p-value=0.02
Class 4	2(50)	2(50)	4(100)	
Total	62(67.4)	30(32.6)	92(100)	

Exposure of children to passive smoking and dental caries

Among 62 children with dental caries, 53(85.5%) children had urine cotinine level of >10ng/ml While >10ng/ml cotinine level detected in 7(23.3%) children without dental caries and 23(76.7%) children without dental caries had cotinine level 10ng/ml had dental caries. There was significant association between cotinine level of child and caries status (χ 2= 34.42, p=0.001) (Table 4).

Table-4: Exposure of children to passive smoking and dental caries

Dental caries	Exposure to passive smoking		Total f (%)	Significance
	Not Exposed f (%)	Exposed f (%)		
Present	9(14.5)	53(85.5)	62(100)	χ^2=34.42, df=1 p-value=0.001
Absent	23(76.7)	7(23.3)	30(100)	
Total	32(34.8)	60(65.2)	92(100)	

Association between duration of exposure to passive smoking and dental caries

Among all children, most of children 56(88.9%) was exposed to passive smoking inside home and had dental caries. There was significant association between caries status of child and exposure to passive smoking inside the home (Fisher's exact = 7.846, p = 0.01) (Table 5).

Table 5: Association between duration of exposure to passive smoking and dental caries

Duration of exposure (hour)	Caries status		Total f (%)	Significance
	Present f (%)	Absent f (%)		
Inside the home				
<1	15(71.4)	6(28.6)	21(100)	Fisher's exact =7.85 df=2 p-value=0.01
1-2	28(96.6)	1(3.4)	29(100)	
>2	13(100)	0(0)	13(100)	
Total	56(88.9)	7(11.1)	63(100)	
Outside the home				
<1	33(89.2)	4(10.8)	37(100)	Fisher's exact =0.40 df=2 p-value=1.00
1-2	4(100)	00(0)	4(100)	
>2	4(100)	00(0)	4(100)	
Total	41(91.1)	4(8.9)	45(100)	
Total				
<1	12(54.5)	10(45.5)	22(100)	Fisher's exact =17.63 df=3 p-value=0.001
1-2	18(94.7)	1(5.3)	19(100)	
2.1-3	18(100)	0(0)	18(100)	
>3	11(100)	0(0)	11(100)	
Total	59(84.3)	11(15.7)	70(100)	

Correlation between Cotinine level of child and duration of indoor exposure to passive smoking

The study showed a statistically significant positive correlation. Increased duration of exposure inside the home was associated with higher level of urinary cotinine (r=0.624, p <0.001) (Figure 3).

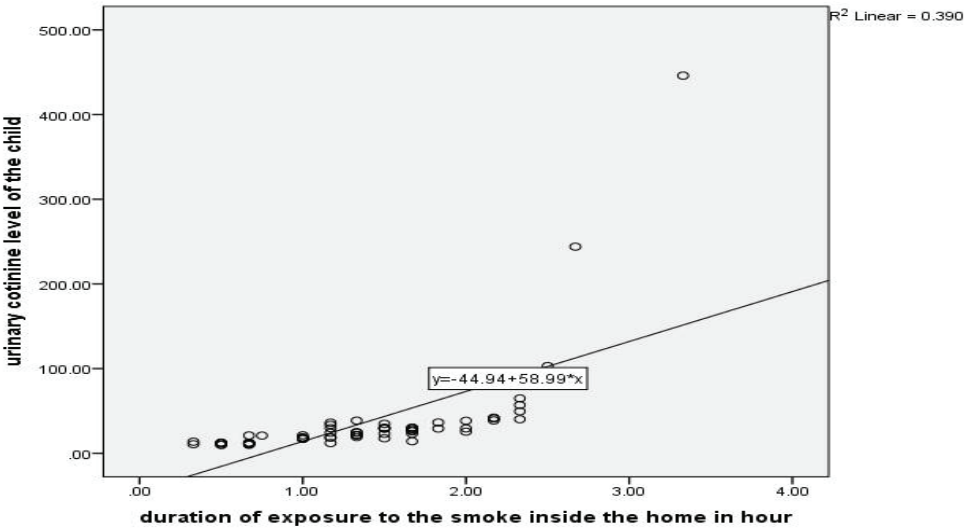

Figure-3: Correlation between Cotinine level of child and duration of indoor exposure to passive smoking.

Correlation between Cotinine level of child and duration of outdoor exposure to passive smoking

The study revealed a statistically significant positive correlation between passive smoking exposure outside home and urinary cotinine level. Increased duration of exposure outside the home was associated with higher level of urinary cotinine. (r=0.636, p <0.001) (Figure 4).

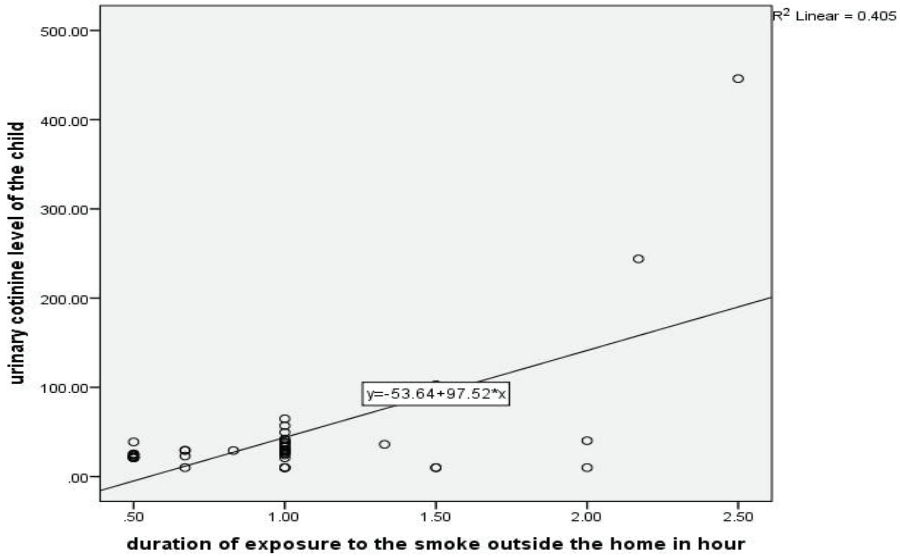

Figure-4: Correlation between Cotinine level of child and duration of outdoor exposure to passive smoking.

Correlation between Cotinine level of child and total duration of exposure to passive smoking (both inside and outside)

The study revealed total duration of exposure in a day both inside and outside home also had statistically significant (p value <0.01) positive correlation. Where increased duration of total exposure (both inside and outside the home) was associated with higher level of urinary cotinine (r=0.692, p <0.001) (Figure 5).

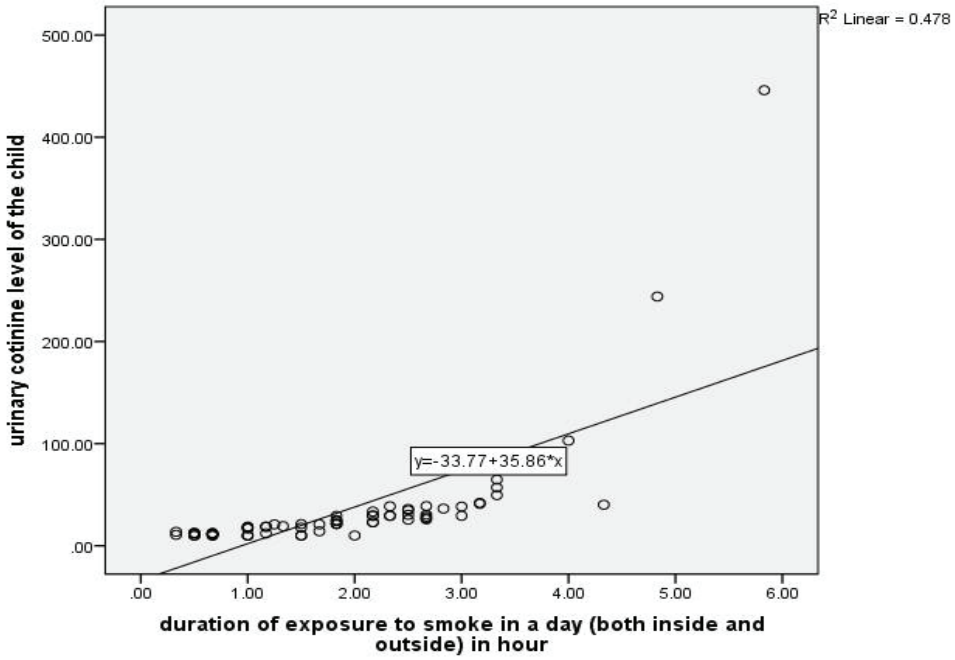

Figure-5: Correlation between Cotinine level of child and total duration of exposure to passive smoking (both inside and outside)

DISCUSSION

This cross-sectional study was conducted among 92 Bangladeshi school children aged 5-10 years to assess the association of passive smoking and pediatric dental caries. Exposure to passive smoking was evidenced by urine cotinine level of children where 10ng/ml was used as a cutoff value. Dental caries was diagnosed by clinical examination of the children. Findings provided by this study are discussed in the following headlines:

Socio-demographic characteristics:

The study revealed that 58.70% (54/92) of the study sample were male and 41% were female aged between 5-10 years. 41% children were in 5-6years age group with a mean age of 7.20 ± 1.549 (Mean age ± SD). 62 (67.39%) children were from nuclear family and most (around 80%) families had 3-5 members. Maximum families (67.39%) were living in pucca house.67.4% children of this study had dental caries. This data is significantly higher than the prevalence reported in United States where prevalence of caries was 20.5% in children aged 2 to 5 years (Dye, BA. et al., 2004). This data is also higher than the prevalence reported in Japan where prevalence of caries was 25% in children aged 3 years [16].

Passive smoking exposure:

Exposure to passive smoking was grouped into indoor exposure, outdoor exposure and double exposure (both inside and outside home). This study showed that 59.8% of father of the studied children were smoker. This figure that is somewhat higher than that reported in a previous Saudi study carried out in Taif City, where 38.4% of fathers were smokers [13] and another Saudi study carried out in Al-Khobar City, where 32% of fathers were smokers [16]. And it is lower than that reported from a Turkish study where 60.6% of fathers were smokers [17]. Another study carried out in Belgium where 30% of the parents (both father and mother) reported smoking behavior [18].

68.5% children reported that they had a family member who smokes at his/her sleeping room and were exposed to passive smoking inside their home. And cotinine levels were consistent with passive smoking exposure (>10ng/ml) in 65.2% children with any member who smokes at his/her room. This figure is higher than that reported from a study done in USA on pre-school children, where 38% of children were exposed to passive smoking at home. It is also higher that that reported from the UK (50%) and Northern European countries (57%) [19, 20]. 61.8% of children were exposed to indoor smoke, reported in a study conducted in Taif city, Saudi Arabia smokers [13]. This difference could be attributed to very high prevalence (63/92, 68.5%) of indoor smoker in the family of study children. On the other hand, 48.9% children were exposed to cigarette smoke outside their home. This figure is same as that revealed from a previous study carried out in Saudi Al- Madinah city,

where the prevalence of passive smoke exposure was 49.3% outside the home [21].

76% children were reported to be exposed to cigarette smoke both inside and outside home. And it is lower than that reported from a study carried out in Turkey where prevalence of passive smoker among elementary school children was 81.3% [7]. Whereas it is higher than that reported in a study conducted in Saudi Taif city, where 49.3% of children reported double exposure to passive smoking (both indoor and outdoor) [13]. This high percentage of double exposure (both inside and outside home) among children of this study was due to study place being an urban area in Chittagong where population density is higher. Also, children have limited option for avoiding passive smoke and they don't know the adverse effects of being exposed to smoke. Added to this is the nature of the children as they often sit closer to their parents and family members which make them closer to source of pollutants [11]. Children are also the most susceptible group for passive smoking because their bronchial tubes are smaller and their immune systems are less developed (WHO Tobacco free initiative. International consultation on environmental tobacco smoke and child health report, 1999 [22].

Caries status of children:

Oral examination of the children was performed to assess the caries status. 75.9% male children and 55.3% female children had dental caries. Majority (73.3%) of them came from combined family which may be due to significant exposure from uncle and grandfather in addition to their father compared to a nuclear family. It was previously reported that prevalence of parental smoking was significantly lower in two-parent household compared to other family structures [18].

This study also showed that dental caries was most common (84.2%) in children aged 56years. All of the children from nursery (12/12, 100%) had caries. This high percentage among young children may be due to their close proximity to family members most of the time. It was also reported in a study in United States that, if passive smoking does cause caries, there are reasons to expect the effect to be most pronounced in early childhood. Colonization with cariogenic Streptococcus mutans is thought to occur in during a window of vulnerability around age 1 year and primary teeth are particularly susceptible to caries formation. It is also known that enamel in deciduous teeth is much thinner than in permanent teeth. Also, salivary flow rate is lower in children [9]. Another study conducted in United Kingdom (UK National Diet and Nutrition Survey) also concluded parental smoking increases the caries risk in young children [23]. Also, a Turkish study concluded the association of passive smoking and dental caries in young children where mean age of children was 5.02 years [7].

Passive smoking exposure and caries prevalence:

Children exposed to indoor smoke were categorized into three groups according to their duration of exposure (<1hr, 1-2hr, >2hr). This study showed that 88.9% children who were exposed to passive smoking inside home had dental caries. The reported dental caries was 100% (13/13) in the children who were exposed to smoke inside home for more than two hours daily and 96.6% (28/29) reported in those exposed more than 1 hour daily. Passive smoking exposure evidenced with urine cotinine level >10ng/ml also revealed that, 100% children had caries in both exposure group of children mentioned above and overall 95.2% children (60/63) who had a cotinine level consistent with indoor passive smoking exposure had dental caries, which is little higher than above data presented based on history alone. This was explained partially by inadequate reporting of exposure or recall bias.

Children exposed to outdoor smoke were also categorized into three groups (<1hr, 1-2hr, >2hr) according to their duration of exposure.91.1% (41/45) children exposed to passive smoking outside home had dental caries where percentage of dental caries was 100% in both 1-2hours exposure (4/4) and more than 2hours daily exposure (4/4) outside home. While 89% children with less than 1hour exposure had caries. Passive smoking exposure outside home evidenced with urine cotinine level >10ng/ml reported 50% and 75% caries respectively in both group of children mentioned above and overall 84.4% children (38/45) who had a cotinine level consistent with passive smoking exposure had dental caries, which is little lower than above data presented based on history alone. This was also explained partially by inadequate reporting of exposure or recall bias.

Children with both indoor and outdoor were categorized into four (<1hr, 1-2hr, >2.1-3hr, >3hrs) groups based on duration of exposure. 84.3% children who were exposed to both indoor and outdoor smoke had caries. Reported dental caries was 100% in children with total (both inside and outside) exposure duration of more than 2-3hours (18/18) and more than 3hours (11/11) daily. Passive smoking exposure both inside and outside home evidenced with urine cotinine level >10ng/ml also reported 100% caries in both exposure group of children mentioned above and overall 85.7% children (60/70) who had a cotinine level consistent with passive smoking exposure had dental caries, which is almost similar to the data presented based on history alone.

Above data shows that an increased duration of exposure is associated with an increased prevalence of dental caries. Dose-response relationships were examined in Japan and the U.S. Three studies compared the relationship by three levels of exposure and two studies employed four levels of exposure. Positive associations were evident in all studies. Effect sizes of the two highest levels of exposure were similar. It is likely that there is a threshold in the increase of risk by passive smoking exposure [9, 24,25]. So, a dose-response relationship is apparent forearly childhood caries.

On the other hand, children who had not cotinine level consistent with passive smoking inside, outside, both inside and outside home had 4.8%, 15.6%, 14.3% caries respectively. This was also explained partially by inadequate reporting of exposure or recall bias and partially by parental unawareness with possible passive smoking exposure outside child's home. Another study demonstrates that 75.6% of the studied children who were classified as un-exposed to SHS had measurable cotinine levels [26] , a finding that was demonstrated also in other studies [27, 28].

In this study, 85.5% of the children (53/62) who were exposed to passive smoking and evidenced by a cotinine value of >10 ng/ml had dental caries (p value<0.01). The result of this study was somewhat higher than that was reported in a previous study conducted in USA where attributable risk from passive smoking were 27% for decayed tooth and 14% for filled tooth surfaces [9] . Study conducted in Belgium reported31% caries in children where 30% parents reported smoking behavior [18]. A recent study conducted in Japan reported risk of caries in children with exposure to tobacco smoke is 27.6% and the risk of caries to children increases by 1.5-fold with household smoking [10] . This difference could be attributed to the reported high prevalence of smoker in the area where the study was conducted. Living in a densely populated area also lead to a higher probability of passive smoking exposure. The estimate may be lower in USA and Japan because of high utilization of fluoride varnish, regular tooth brushing and dental examination.

This study shows that 65.2% (60/92) children had urine cotinine level consistent with passive smoking exposure. Among them majority (76.3%) were 5-6 years old. This result is somewhat higher than that reported in study conducted in United States where overall 53.1% children aged 4years to 11years with cotinine level consistent with passive smoking exposure and 46.7% had decayed primary teeth [9].

75.85% children who lived in pucca house were exposed to passive smoking evidenced with cotinine level above 10ng/ml. This high exposure may be attributed to poor ventilation in pucca house compared to semi-pucca house which increases the duration of exposure to passive smoke inside the home. Majority (73.3%) of children from combined family had cotinine level consistent with passive smoking compared to the nuclear family. This may be explained by added exposure from uncle, grandfather in addition to father. This is in agreement with other studies which revealed that cotinine levels in children are dose dependent and is positively correlated with the number of smokers in the home [29]. This finding is consistent with another study where urinary cotinine levels were found to increase by 5 times depending on the number of smoking parents (1 or 2 smokers) [30].

The strength of association between passive smoking exposure inside home and level of urinary cotinine was studied. Pearson correlation showed a statistically significant (p value <0.001) positive correlation. Meaning increased duration of exposure inside the home was associated higher level of urinary cotinine. The magnitude of association was strong (0.5 < | r |). Pearson correlation also showed a statistically significant (p value <0.001) positive correlation between passive smoking exposure outside home and urinary cotinine level. Increased the duration of exposure outside the home was associated higher level of urinary cotinine. The magnitude of association was strong (0.5 < | r |). Total duration of exposure in a day both inside and outside home also had statistically significant (p value <0.001) positive correlation. Where increased duration of total exposure (both inside and outside the home) was associated higher level of urinary cotinine. The magnitude of association was strong (0.5 < | r |).

The significant positive correlation found between urinary cotinine concentrations and the duration of exposure passive smoking is consistent with results revealed from previous studies. In the study conducted in Saudi Taif city, urinary cotinine level in children with double exposure to passive smoking (both indoor and outdoor) was significantly higher when compared with urinary cotinine level in children with single exposure [13]. These studies also revealed the same positive correlation between the reported amount of smoking and the levels of urinary cotinine [11, 30, 31, 32, 33].

CONCLUSION

Passive smoking is now considered more toxic than directly-inhaled firsthand smoke. Bangladesh is a high populated area and exposure of Bangladeshi children to passive smoking is very high. This cross-sectional study revealed that most of the children were exposed to passive smoking and maximum children had dental

caries. There was statistically significant association between exposure to passive smoking evidenced with urinary cotinine level and dental caries in children. With high level of urinary cotinine level most of them had caries prevalence. Fathers tend to smoke more in front of their children and alarmingly they didn't know health consequences of passive smoke exposure in their children. More than 1-hour daily exposure to passive smoke was associated with a very high prevalence of dental caries among children. Urinary cotinine level also showed a statistically significant association with duration of exposure to passive smoke in children, where an increasing duration of exposure was associated with an increasing level of cotinine in urine. Although sugar containing foods and drinks, sticky foods were some important factors related to dental caries, this study didn't show any statistical significance among those factors with the presence of dental caries in children. Despite the high prevalence passive smoking exposure, there is no evidence based high-quality epidemiological studies available in our country. So, a causal association between passive smoking and dental caries in children needs to be assessed.

REFERENCES

1. Takashi H, Miki O, Keiko T, Mito Y. Does Secondhand Smoke Affect the Development of Dental Caries in Children? A Systematic Review. Int J Environ Res Public Health 2011;8(5):1503-1519.
2. Dye BA, Tan S, Smith V. Trends in oral health status: United States, 1988-1994 and 1999-2004. Vital Health Stat 2007; 248:1-92.
3. Chou R, Cantor A, Zakher B, Mitchell JP, Pappas M. Preventing dental caries in children <5 years: systematic review updating USPSTF recommendation. Pediatrics 2013;132:332-50.
4. Sherr Z, David G, Rosenthal M, Scott S. Physical, Behavioral, and Cognitive Effects of Prenatal Tobacco and Postnatal Secondhand Smoke Exposure. CurrProblPediatrAdolesc Health Care 2014;44:219-241.
5. Cancer Research, UK 2016.
6. Sajith V, Zdenek F, JindraS,Vimal J, Pilathadka S.Influence of tobacco use in dental caries development. Cent Eur J. Public Health 2007;15(3):116-121.
7. Avşar A, Darka O, Topaloglu B, Bek Y. Association of passive smoking with caries and related salivary biomarkers in young children. Arch. Oral Biol 2008;53: 969-974.
8. Prokhorov AV, Winickoff JP, Ahluwalia JS, Ossip-Klein D, Tanski S, Lando HA, et al.. Tobacco Consortium, American Academy of Pediatrics Center for Child Health Research. Youth tobacco use: a global perspective for child health care clinicians. Pediatrics 2006;118(3): 890-903.
9. Aligne CA, Moss ME, Auinger P, Weitzman M. Association of pediatric dental caries with passive smoking. J. Am. Med. Assoc 2003;289:1258-1264.
10. Shiro T, Maki S, Hironobu T, Kahori S. Secondhand smoke and incidence of dental caries in deciduous teeth among children in Japan: population based retrospective cohort study. BMJ 2015;351.
11. Abou El-Ellaa SS, Tawfika MA, El Bassuonib MA, Yahiaa MI. Urinary cotinine level in passive smoker nondiabetic children of diabetic families. Menoufia Medical Journal 2014;27:23-27.
12. Neal L, Benowitz W, Janne H, Peyton J. Nicotine, Chemistry, Metabolism, Kinetics and Biomarkers, Handb Exp Pharmacol 2009;192:29–60.
13. Dalia E, Gamal E, Ali A, Azza A. The Relation between Exposure to Environmental Tobacco Smoke and the Quantity of Cotinine in the Urine of School Children in Taif City, Saudi Arabia, Asian Pac J Cancer Prev 2016;17 (1):139-145.
14. Goniewicz ML, Eisner MD, Eduardo L et al. Comparison of urine cotinine and the tobacco-specific nitrosamine metabolite 4-(Methylnitrosamino)-1-3-1-Butanol (NNAL) and Their Ratio to Discriminate Active From Passive Smoking. Nicotine Tob Res 2011;13:202-8.
15. Ministry of Health, Labour and Welfare. Survey of dental diseases 2011. www.mhlw.go.jp/toukei/list/62-23.html.
16. Bassiony M. Smoking in Saudi Arabia. Saudi Med J 2009; 30:876-81.
17. Boyaci H, Etiler N, Duman C, Basyigit I, Pala A. Environmental tobacco smoke exposure in school children: parent report and urine cotinine measures. Pediatrics International 2006;48:382-9.
18. Leroy R, Hoppenbrouwers K, Jara A, Declerck D. Parental smoking behavior and caries

experience in preschool children. Community Dent. Oral Epidemiol 2008;36:249-257.

19. Gergen PJ, Fowler JA, Maurer KR, Davis WW, Overpeck MD.The burden of environmental tobacco smoke exposure on the respiratory health of children 2 months through 5 years of age in the United States: Third National Health and Nutrition Examination Survey, 1988 to 1994. Pediatrics 1998;101:8.
20. Lund KE, Skrondal A, Vertio H, Helgason AR.To what extent do parents strive to protect their children from environmental tobacco smoke in the Nordic countries? A populationbased study. Tob Control 1998;7:56-60.
21. Al-Zalabani AH, Amer SM, Kasim KA, Alqabshawi RI, Abdallah AR.Secondhand smoking among intermediate and secondary school students in Madinah, Saudi Arabia. Biomed Res Int 2015;15:17-24.
22. Delpisheh A, Kelly Y, Brabin BJ. Passive cigarette smoke exposure in primary school children in Liverpool. Public Health 2006;120:65–9.
23. Williams SA, Kwan SY, Parsons S.Parental smoking practices and caries experience in pre-school children. Caries Res 2000;34:117-122.
24. Hanioka T, Nakamura E, Ojima M, Tanaka K, Aoyama H. Association of dental caries of 3-year-old children with smoking status of parents. Paediatr. Perinat. Epidemiol 2008;22:546-550.
25. Tanaka K, Miyake Y, Arakawa M, Sasaki S, Ohya Y. Household smoking and dental caries in schoolchildren: The Ryukyus Child Health Study. BMC Publ. Health 2010;10:335.
26. Mannino DM, Caraballo R, Benowitz N, Repace J.Predictors of cotinine levels in US children: data from the third national health and nutrition examination survey. Chest 2001 ;120:718-24.
27. Hecht SS, Ye M, Carmella SG, et al. Metabolites of a tobacco-specific lung carcinogen in the urine of Elementary School-aged Children. Cancer Epidemiol Biomarkers Prev 2001 ;10;1109-16.
28. Bono R, Vincenti M, Schiliro T, et al. Cotinine and N-(2-hydroxyethyl) valine as markers of passive exposure to tobacco smoke in children. J Expo Anal Environ Epidemiol 2005;15:66-73.
29. Seifert JA, Ross CA, Norris JM. Validation of a five-question survey to assess a child's exposure to environmental tobacco smoke. Ann Epidemiol 2002;12:273-7.
30. Jurado D, Muñoz C, Luna Jde D, Fernández-Crehuet M.Environmental tobacco smoke exposure in children: parental perception of smokiness at home and other factors associated with urinary cotinine in preschool children. J Expo Anal Environ Epidemiol 2004;14:330-6.
31. Kim HI, Lim Y, Lee S, et al.Relationship between environmental tobacco smoke and urinary cotinine levels in passive smokers at their residence. J Expo Anal Environ Epidemiol 2004;14:65-70.
32. Reeves S, Bernstein I. Effects of maternal tobacco-smoke exposure on fetal growth and neonatal size. Expert Rev ObstetGynecol 2008;3:719-30.
33. Schvartsman C, Farhat SCL, Schvartsman S, Saldiva PHN. Parental smoking patterns and their association with wheezing in children. Clinics (Sao Paulo) 2013;68:934-9.