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## Effect of Passive Tobacco Smoking Exposure on the Periodontal Status of Turkish Children

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## Cover Page Footnote

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**ORIGINAL ARTICLE**

**Effect of Passive Tobacco Smoking Exposure on the Periodontal Status of Turkish Children**

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**ABSTRACT**

**Objective:** This study aimed to assess the effect of passive tobacco smoking (PTS) through the comparison of the periodontal status and the C-reactive protein (CRP) and cotinine levels in the biologic fluids in children who are exposed and unexposed to PTS. **Methods:** A total of 148 participants, whom had smokers (PTS-exposed, n=82) and non-smokers (PTS-unexposed, n=66) in their families, respectively, were included in the study. Gingival index, plaque index, probing depth, and bleeding on probing were recorded. Moreover, saliva, urine, and gingival crevicular fluid samples were collected. CRP and cotinine levels in the biological fluids were determined by the enzyme-linked immunosorbent assay method. **Results:** Both groups exhibited similar values for periodontal parameters and salivary CRP levels were higher in PTS-exposed group but not significant. The mean urinary cotinine level was significantly higher in children exposed to PTS than in unexposed children. **Conclusions:** There was no evidence for causative role of PTS in periodontal disease in this study. Longitudinal studies including large populations should be conducted to provide stronger evidence for the causative role of PTS in periodontal disease. Also, further epidemiological studies on the social context of smoking should be performed to improve the quality of life and lifespan of the society.

**Key words:** children, cotinine, C-reactive protein, smoking

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**INTRODUCTION**

Periodontal disease is a condition characterized by the inflammation of the dental support tissues. The host and bacterial, behavioural, and environmental factors determine the development and progression of the disease.<sup>1</sup> Smoking is considered an important preventable risk factor for the development and progression of the periodontal disease.<sup>2,3</sup> Arbes et al.<sup>4</sup> assessed the effects of self-reported exposure to passive tobacco smoking (PTS) after controlling known risk factors for periodontal disease and reported a 1.6-fold increase in the risk of periodontal disease for people

who had never smoked and had been exposed to PTS compared with those who had not been exposed. This result suggested an association between PTS and periodontal disease, with PTS having a harmful effect. Exposure to PTS negatively affects the health of both adults and children. The exposure in childhood is associated with the onset of asthma, increase in the severity of asthma, development of allergy, nasal and sinus diseases, dental caries, behavioural problems, and childhood cancer.<sup>5-7</sup> It is estimated that 40% of children are exposed to tobacco smoking in their homes worldwide and that the smoking parents are the major cause of the exposure.<sup>8</sup>

Nicotine, cotinine, and their respective glucuronides are the major chemical derivatives detected after exposure to cigarette smoke.<sup>9</sup> Due to its relatively long half-life, which is 10 times longer than that of nicotine, and it is present in body fluids for a longer period of time, cotinine is the currently preferred marker used to measure tobacco exposure.<sup>10</sup> Measuring the cotinine levels in the human body fluids may help in estimating the last exposure to tobacco, but provide no information regarding the duration of the exposure. Moreover, cotinine can be isolated from plasma, urine, saliva, and gingival crevicular fluid (GCF).<sup>11</sup>

Inflammation is part of the immune reaction and leads to the release of C-reactive protein (CRP) into the blood stream. Serum CRP is the gold standard measure of low-grade inflammation, and provides the opportunity of predicting future problems even in healthy individuals.<sup>12</sup> However, compared with the invasive nature of the serum test, the saliva CRP test is minimally invasive, yet appears to be sensitive enough.<sup>13</sup> Azar and Richard<sup>14</sup> showed that salivary CRP, as a counterpart of the commonly used serum biomarker, has a similar relationship with the exposure to PTS.

There are restricted studies exploring the potential impacts of PTS on periodontal health and it has not been entirely elucidated whether PTS disrupts oral health.<sup>15</sup> According to a recently published systematic review, the relationship between exposure to PTS and periodontal disease is still controversial, and more research is needed on this topic.<sup>16</sup> Hence, the aim of this study was to evaluate the possible association between the saliva, urine, and GCF levels of the biochemical parameters and the periodontal conditions in Turkish children exposed and unexposed to PTS.

## **METHODS**

### **Ethics approval**

This cross-sectional study was approved by the ethics committee of the Faculty of Dentistry at Necmettin Erbakan University (app. no. 2015/006). A written informed consent was obtained from all participant parents. They participated in the study by their own wills, and they had the right to withdraw their participation at any time.

### **Study population**

Children, aged 5–13 years, attending the Department of Pediatric Dentistry, Faculty of Dentistry, University of Necmettin Erbakan, participated in the study. Children who were included had no systemic problems, were non-smokers, and had not used any drugs within the last month. A questionnaire form was designed for parents to collect demographic information such as age, gender, body mass index (BMI), oral care, smoking status, and parental education level and family

income of the participants. Moreover, the questionnaire included some questions about the parents' opinions regarding PTS. Depending on their parents' smoking status patients were divided into two groups according to their exposure to cigarettes: PTS exposed and PTS unexposed.

### **Clinical parameters**

Scores were determined using plaque index (PI)<sup>17</sup> and gingival index (GI)<sup>18</sup> prior to collecting GCF. Probing depths (PD) and bleeding on probing (BOP) scores were also recorded. Measurements were obtained from two anterior and two posterior teeth (a total of four) in the maxilla and six regions from each tooth using Williams's periodontal probe (Hu-Friedy, IL, USA).

### **GCF sampling**

GCF was sampled from four maxillary teeth. Before the sampling, the relevant tooth was gently dried with air spray and isolated with cotton tampons. Following the determination of PI, the supra-gingival plaque was removed if present, and GCF was collected with paper strips (Periopaper, NY, USA).

Extra attention was paid not to contaminate the paper strips with saliva or blood. The contaminated paper strips were not included in the study. The strips were placed gently into approximately 1 mm of the gingival crevice of each tooth from the mesial or distal midpoint of the tooth. Care was taken to avoid mechanical trauma. For standardization, the paper strips were left in the gingival crevice for 30s and then placed in a polypropylene eppendorf tube containing 250  $\mu$ l of phosphate-buffered saline. The tubes were coded, and their caps were sealed with paraffin band to prevent leakage. Each eppendorf tube containing four paper strips from a single patient was immediately transferred to  $-80^{\circ}\text{C}$ .

### **Sampling of saliva and urine**

The children were asked to accumulate their saliva in their mouths and then spit into disposable plastic containers. Urine samples were collected in the same session following the saliva sampling and immediately transferred to  $-20^{\circ}\text{C}$  after being coded.

### **Analysis**

A commercial kit was used to determine CRP levels in saliva and the cotinine levels in saliva, urine, and GCF. The measurements were expressed in ng/ml. All the analyses were performed at the Department of Biochemistry, Faculty of Veterinary, Selcuk University using the enzyme-linked immunosorbent assay method.

### **Statistical analysis**

The data were analyzed using SPSS 15.0 (SPSS Inc., IL, USA) for Windows. All data were first analyzed descriptively and were presented as mean $\pm$ SD values. Mann-Whitney U-test and Independent samples t-test

**Table 1.** Demographic characteristics of children

Characteristics	PTS-exposed (n=82) (mean±SD)	PTS-unexposed (n=66) (mean±SD)	p-value
Age	9.85±2.18	9.78±2.07	NS
Height (m)	1.42±0.16	1.44±0.16	NS
Weight (kg)	36.51±12.99	36.81±12.96	NS
BMI (kg/m <sup>2</sup> )	17.46±3.39	17.16±2.95	NS
Gender			
Female-BMI (n=87)	17.45±3.35 (n=43)	17.39±3.20 (n=44)	NS
Male-BMI (n=61)	17.46±3.47 (n=39)	16.70±2.38 (n=22)	NS
<b>p-value</b>	NS	NS	

BMI, body mass index; NS, non-significant.

**Table 2.** Demographic characteristics of parents

Questions/variables	Parents' smoking status		χ <sup>2</sup> (p-value)
	Smokers Frequency (%)	Non-smokers Frequency (%)	
How often is your child's dental examination done?			5.61 (0.132)
Every 6 months	1 (1.2%)	5 (7.6%)	
Once a year	2 (2.4%)	2 (3%)	
In the presence of any problem	74 (90.2%)	58 (87.9%)	
Never	5 (6.1%)	1 (1.5%)	
Income			12.37 (0.002)*
≤2000 *	42 (68.9%)	19 (31.1%)	
2000-3000 *	28 (56.0%)	22 (44.0%)	
≥3000 *	12 (32.4%)	25 (67.6%)	
Mother			
Age (mean±SD)	35.09±5.40	35.95±4.87	
Education			10.92 (0.012)*
Primary school	57 (69.5%)	34 (51.5%)	
High school	20 (24.4%)	16 (24.2%)	
Graduate	5(6.1%)	14 (21.2%)	
Postgraduate	-	2 (3%)	
Is the PTS harmful to your child?			0.60 (0.437)
Absolutely not	-	-	
Probably not	-	-	
Probably yes	1 (1.2%)	2 (3%)	
Absolutely yes	81 (98.8%)	64 (97%)	
Father			
Age (mean±SD)	39.19±5.62	39.76±5.70	
Education			9.57 (0.023)*
Primary school	36 (44.4%)	27 (42.2%)	
High school	30 (37%)	13 (20.3%)	
Graduate	14 (17.3%)	19 (29.7%)	
Postgraduate	1 (1.2%)	5 (7.8%)	
Is the PTS harmful to your child?			9.46 (0.024)*
Absolutely not	2 (2.5%)	-	
Probably not	8 (10%)	-	
Probably yes	3 (3.8%)	1 (1.6%)	
Absolutely yes	67 (83.8%)	63 (98.4%)	

\*statistically significant; \* Turkish Lira

**Table 3.** Clinical parameters of groups

Groups	Clinical parameters (mean±SD)			
	PI	GI	PD	BOP
PTS-exposed (n=82)	1.04±0.12	1.55±0.31	1.18±0.22	0.18±0.15
PTS-unexposed (n=66)	1.03±0.10	1.54±0.31	1.19±0.25	0.17±0.15
p-value	NS	NS	NS	NS

PI, plaque index; GI, gingival index; PD, probing depth; BOP, bleeding on probing; NS, non-significant.

**Table 4.** Measurements in body fluids of children

Groups	Body Fluids (mean±SD)			
	Saliva CRP (ng/ml)	Saliva Cotinine (ng/ml)	GCF Cotinine (ng/ml)	Urine Cotinine (ng/ml) *
PTS-exposed (n=82)	1.77±3.54	17.37±12.73	0.42±0.45	4.00±4.77
PTS-unexposed (n=66)	0.83±1.43	17.60±12.50	0.39±0.24	2.83±3.16
p-value	NS	NS	NS	0.027

GCF= gingival crevicular fluid, CRP=C reactive protein; \*statistically significant; NS= non-significant.

were used to compare the mean values. Categorical variables were expressed as frequencies and percent and compared using the  $\chi^2$  test. The significance level was set at  $p < 0.05$ .

## RESULTS

The study continued with 148 participants based on the data obtained from the biochemical analysis (no detectable range of cotinine and CRP levels, lack of some samples, etc.). Based on the smoking status of the family members, the mean age of the participants in the PTS exposed (n=82) and PTS unexposed (n=66) groups was  $9.85 \pm 2.18$  years and  $9.78 \pm 2.07$  years, respectively, with no statistically significant difference between the averages. Also, most of the participants were of normal weight, and when evaluated separately, no difference was found inter- and intra-groups in terms of BMI on comparing the genders (Table 1).

The number of the smoking fathers (53,1%) was higher than the number of the smoking mothers (10,1%). The frequency of oral care of the children included in the study and the demographic profiles of their parents were shown in Table 2. The level of education of the mothers and fathers was higher in the PTS unexposed group ( $p = 0.012$  and  $p = 0.023$ , respectively). Similarly, the incomes were higher in the PTS unexposed group ( $p = 0.002$ ). A significant difference was observed between the father's responses about the harmful effects of exposure to PTS on children ( $p = 0.024$ ). About 12.5% of the fathers in the PTS exposed group thought that PTS do not have a harmful effect on children.

The clinical characteristics of the participants regarding the exposure to PTS are presented in Table 3. The analysis yielded similar results for both groups, and no significant differences were detected between the groups.

The CRP levels in the saliva and cotinine levels in the saliva, urine, and GCF are presented in Table 4. A significant difference was found only in the urinary cotinine levels between the PTS exposed and PTS unexposed groups. The mean cotinine level in the urine was significantly higher in the PTS exposed group ( $p = 0.027$ ). When the salivary CRP levels were examined, a higher mean value was observed for the PTS exposed group, although the difference was statistically insignificant ( $p = 0.07$ ).

## DISCUSSION

Although the harmful effects of active smoking on the existence and severity of periodontal diseases are well documented, there are restricted studies exploring the potential impacts of PTS exposure on periodontal health. This study was performed to assess clinical and biochemical parameters in children exposed and unexposed to PTS. In the study, when the clinical parameters were evaluated, no significant differences were detected between the groups, while urinary cotinine levels were found to be significantly higher in children exposed to PTS.

Cotinine is the basic biomarker that distinguishes the smokers from non-smokers and reflects the extent of

the exposure.<sup>19</sup> Measuring the cotinine levels in the blood best reflects the dose of nicotine absorbed from PTS compared with other biological fluids.<sup>20</sup> Sampling from urine, saliva, and GCF, which are less invasive, was preferred over sampling from blood samples in this study to assess exposure to PTS, as the study population was children.

Because cotinine concentrations are four to six times higher in the urine than in the blood or saliva, the commonly used biomarker is urinary cotinine level.<sup>21,22</sup> It is also stated that urine is a more appropriate biological fluid to detect exposures to low levels and that it can be collected more easily than plasma or saliva.<sup>23,24</sup> The urinary cotinine levels were higher in children exposed to PTS in this study; however, no difference was observed in cotinine levels in other biological fluids. This result was congruent with the findings reported in previous studies. Matsumoto et al.<sup>9</sup> demonstrated a significant relationship between urinary cotinine levels and smoking habit, which were related to the work and home environments of the non-smokers.

Compared with active smoking, evaluation of PTS exposure creates even more difficulties. The results of studies on PTS exposure and periodontal disease should take this into account. In point of the use of biomarkers, similar restrictions perform to their use to assess PTS exposure for clinical trials. Thus, exposure to PTS is typically evaluated by self-reporting. A subject may be exposed to PTS at different times and may occur at different places. However, places may change over time and/or size and ventilation varies depending on the number and turnover of air conditioning. Lifelong differences in these factors contribute to the complexity of the problem.<sup>25,26</sup> Chen et al.<sup>27</sup> assessed the relationship between cotinine concentrations in GCF and periodontal disease status in smokers and non-smokers and found a high correlation between cotinine GCF concentrations and the mean PD and attachment loss. On the contrary similar to this study, some studies have not found a relationship between exposure to PTS and periodontal disease.<sup>28</sup> We believed that the absence of difference was not an error in children classification, because indeed children in the PTS exposed group had higher level of urinary cotinine than children in the PTS unexposed group. However, measuring biochemical compounds in GCF in all regions of the mouth, especially in healthy areas, is difficult because of the small amount of GCF.<sup>29</sup> When the study population consists of children, it is inevitable to collect smaller amounts from a smaller number of teeth because of the lack of cooperation. Similar to Erdemir et al.<sup>20</sup>, a group of children was not included in this study because the cotinine levels in their GCF were below the detectable limit.

Elevated CRP levels are associated with poor periodontal health or chronic oral infections.<sup>14</sup> Few studies have investigated the effects of exposure to PTS on the salivary CRP levels. In their study on young individuals, Azar and Richard<sup>14</sup> showed that active smokers, people exposed to PTS, and non-smokers had the highest, low, and the lowest CRP levels, respectively. In the present study, CRP salivary levels were not statistically different, and this may explained with the absent of difference between PTS exposed and unexposed groups on the clinical characteristics.

The results of the questionnaire answered by the parents showed that the smokers had lower incomes. Previously, when the population was stratified by income and occupation, cigarette consumption was found to be two to three times higher in the low-socioeconomic-status groups. It is important to understand why parents with a lower socioeconomic status consume more tobacco. Smoking can be a response to stress and challenges of living in an economically deprived environment.<sup>30</sup> Moreover, when parents were asked to express their opinions on the effects of exposure to PTS on their children's health, about 12.5% of the smokers stated that they did not have any information regarding PTS. de Carvalho Ribeiro et al.<sup>30</sup> suggested that children with a low socioeconomic status might be more vulnerable to the harmful effects of exposure to PTS because the poorest and least informed stratum of a society does not have access to the necessary information.

This study has several limitations. Firstly, a more comprehensive questionnaire can be applied. We did not explore any outdoor activities that children may be exposure to PTS, and parents time spent with their children. Also there was no data about diet type. Morzel et al.<sup>31</sup> stated that relationship between diet consumption and composition of saliva differ among people with different diets. Finally, since the study population was children, periodontal measurements were obtained from four teeth, depending on the difficulty of cooperating. This does not reflect full mouth monitorization.

## **CONCLUSION**

In conclusion, there was no evidence for a causative role of PTS in periodontal disease in this study. The results of studies related to the risk of exposure to PTS should be commented, especially when compared to studies on the impacts of active smoking. Biochemical readings do not inevitably supply the gold standards of tobacco use/non-use, and although their esteemed neutrality, are not without problems.<sup>25</sup> Therefore, longitudinal studies including large populations should be conducted to provide stronger evidence for the causative role of PTS in periodontal disease.

## CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

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