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Periodontal health in children exposed to passive smoking

Ebru Olgun Erdemir[1], Işıl Saroğlu Sönmez[2], Aylin Akbay Oba[2], Jan Bergstrom[3] and Osman Çağlayan[4]

[1]Department of Periodontology, Faculty of Dentistry, Kirikkale University, Kirikkale, Turkey; [2]Department of Pedodontics, Faculty of Dentistry, Kirikkale University, Kirikkale, Turkey; [3]Karolinska Institutet, Institute of Odontology, Stockholm, Sweden; [4]Department of Biochemistry, Faculty of Medicine, Kirikkale University, Kirikkale, Turkey

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Abstract

Aim: To determine (1) the cotinine levels of saliva, urine and gingival crevicular fluid (GCF) of children in families with and without smoking members and (2) a possible association between the periodontal health of the children and exposure to passive smoking.

Material and Methods: The study population comprised of 109 children in the age range 6–12 years. Children were classified as exposed to passive tobacco smoking (PTS-exposed, $n = 51$) and as unexposed controls (PTS-unexposed, $n = 58$). Plaque index, gingival index, bleeding on probing, probing depth and clinical attachment level (CAL) were recorded. GCF, saliva and urine samples were also collected. The levels of cotinine in these fluids were determined by enzyme-linked immunosorbent assay.

Results: The mean salivary cotinine concentration was significantly increased in PTS-exposed children compared with PTS-unexposed children ($p < 0.05$). Further, in a dose-dependent way, the mean salivary concentration was significantly higher in children whose father or mother was a smoker ($p < 0.05$) as compared, respectively, with children whose fathers and mothers were non-smokers. The mean CAL was significantly less in PTS-exposed children compared with non-PTS-exposed children (0.09 mm; $p < 0.05$) and also in children whose father was a smoker ($p < 0.05$), but not in children whose mother was a smoker as compared with non-smoker fathers and mothers, respectively. The GCF cotinine levels were below the detection limits with the assay method that was used.

Conclusions: We have observed that children who are exposed to passive smoking have elevated cotinine levels in their saliva concomitant with a lowered CAL.

Key words: children; cotinine; passive smoking; periodontal health

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Smoking is generally accepted as a major preventable risk factor in the incidence and progression of periodontal disease (Schenkein et al. 1995, Gonzalez et al. 1996, Bergstrom et al. 2000, Johnson & Hill 2004, Borrell & Papapanou 2005, Heitz-Mayfield 2005, Palmer et al. 2005, Tonetti & Claffey 2005). Recently, it was reported that among adults who had never smoked cigarettes, the odds of having periodontal disease were 1.6 times greater for persons exposed to passive smoking than for persons not exposed, after controlling for known risk factors for periodontal disease (Arbes et al. 2001). This result suggested that passive smoking may also have a harmful effect on periodontal health.

Conflict of interest and source of funding statement

None declared.
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Passive smoking has been associated with a number of negative health outcomes in children. It is causally associated with asthma induction and exacerbation, middle ear infections, chronic respiratory symptoms and acute lower respiratory tract infections such as bronchitis and pneumonia (California Environmental Protection Agency 2007).

Although self-reported smoking is the most widely used strategy to classify smokers and non-smokers, quantification of the exposure to smoking based on self-reports may at times be unreliable (Gonzalez et al. 1996). A number of biochemical markers have been used to validate claims of non-smoking, including measures based on thiocyanate, nicotine, cotinine and carbon monoxide. Levels of thiocyanate and carbon monoxide are easier to determine but may be elevated through exposures unrelated to smoking, such as traffic emissions and diet (Jarvis et al. 1987, Patrick et al. 1994).

Cotinine, a major metabolite of nicotine, is the most commonly used biochemical marker of tobacco use (Armitage et al. 1975, Benowitz 1996); its plasma half-life is longer than that of nicotine, ranging from 10 to 30 h (Benowitz et al. 1983, Curvall & Enzell 1986). A few reports have documented an association between cotinine level in

serum and saliva and periodontal parameters such as probing depth (PD) and clinical attachment loss (Gonzalez et al. 1996, Yamamoto et al. 2005).

To our knowledge, no studies have examined the possible influence of passive smoking on the periodontal health of children. Therefore, the purpose of the present study in children was to determine (1) the cotinine levels of saliva, urine and gingival crevicular fluid (GCF) of children in families with and without smoking members and (2) a possible association between the periodontal health of the children and exposure to passive smoking.

Material and Methods

The study population was a convenience sample consisting of 109 children in the age range 6–12 years (mean 9.9 years). The sample was selected from children seeking dental treatment at the Department of Pediatric Dentistry of Kirikkale University. We excluded children older than 12 years in order to reduce possible confounding from early active smoking and children who had used anti-inflammatory or anti-microbial drugs within the previous month or had any systemic disease. The parents of the children were fully informed of the nature of the study, and written consent for participation of the children in the study was obtained from all parents. The study was approved by the Medical Ethical Committee of Kirikkale University, Faculty of Dentistry.

A detailed questionnaire regarding the smoking habits of family members was administered to parents. Smokers were asked about the number of cigarettes smoked per day, and whether or not they smoked while at home. Specifically, they were asked whether they smoked in the presence of their children. The questionnaire also included questions about family income and education.

Children whose parent(s) regularly daily smoked at home were classified as exposed to passive tobacco smoking (PTS-exposed, $n = 51$; 33 boys and 18 girls) and children whose parent(s) had never smoked at home or any other place were classified as unexposed controls (PTS-unexposed, $n = 58$; 23 boys and 35 girls).

Clinical recordings

Supragingival plaque was scored before crevicular fluid collection using the plaque index (PI, Silness & Löe 1964) and gingival inflammation was scored following the collection of crevicular fluid using the gingival index (GI, Löe & Silness 1963). Gingival bleeding on probing (BOP) was dichotomously assessed according to Ainamo & Bay (1975). PD and clinical attachment level (CAL) were measured at six sites per tooth of four teeth in the maxilla (maxillary first molars and central incisors) using a Williams periodontal probe (Hu–Friedy, Chicago, IL, USA). The probe was directed parallel to the long axis of the tooth. CAL measurements were made from the cemento-enamel junction to the bottom of the gingival sulcus. The patient means were calculated from these recordings.

Crevicular fluid sampling

After supragingival plaque was removed, GCF was collected with paper strips (Periopaper, Amityville, NY, USA) from each of four maxillary teeth in patients. The individual tooth site was gently air-dried and isolated with cotton rolls and a saliva ejector was used to avoid salivary contamination of the samples. The paper strips were consecutively inserted into the crevice at the mesial or the distal midpoints until mild resistance was felt. The strips were left in situ for 30 s and then transferred for volume determination to a chair-side-located Periotron 8000 (Oraflow Inc., Plainview, NY, USA), which was calibrated using known volumes of phosphate-buffered saline (PBS). The four strips of each patient were immediately placed in a labelled tube containing 500 μl PBS and transported to the laboratory. The samples were stored at -20°C for subsequent assays. The levels of cotinine in GCF were determined using a commercial ELISA kit (DRG, Marburg, Germany) and, corrected for GCF volume, expressed as pg/μl. The total amounts of GCF cotinine were expressed as pg/4 sites.

Saliva and urine sampling

Saliva and urine samples were collected from all children between 9 and 11 hours. The children were asked to collect saliva in their mouths and to spit into a clean plastic container. The amount of unstimulated whole saliva was approximately 500 μl. Urine samples were collected on the same occasion. All samples were coded, immediately frozen and stored at -20°C until use. Cotinine levels were measured via a competitive enzyme-linked immunosorbent assay (ELISA).

For all of the cotinine analyses an ELx 50 microplate washer (BioTek, Winooski, VT, USA) was used. The results were read using a μQuant microplate reader (BioTek).

Statistical analysis

Data were expressed as means and 95% confidence intervals (CI). The statistical significance of differences between groups was primarily run with one-factor ANOVA or Kruskal–Wallis ANOVA. Further analyses were performed using general linear models with age and PI as continuous co-factors. Variables with a non-normal distribution were log-transformed to approximate normality. CAL, PD, GI, BOP, salivary cotinine and urinary cotinine were dependent variables (cotinine in GCF could not be analysed). The following served as independent variables: smoking in presence of child (two strata; exposed and unexposed), smoking of the father (four strata; non-smoker, <10 cigarettes/day, 10–20 cigarettes/day, >20 cigarettes/day) and smoking of the mother (four strata; non-smoker, <10 cigarettes/day, 10–20 cigarettes/day, >20 cigarettes/day). Simple pairwise correlations were performed according to Pearson. The null hypothesis was rejected at $p < 0.05$.

Results

PTS exposure and cotinine levels

Overall, 67.3% of the children were exposed to tobacco smoke on the basis of the number of cigarettes/day reportedly smoked by all family members inside the house. When exposure to PTS was evaluated by urinary and salivary cotinine levels, the proportion of children exposed (levels above zero) was 90.9% and 76.4%, respectively.

The levels of cotinine in saliva, urine and GCF are shown in Table 1. The GCF cotinine levels were below the detection limit in all but four children and, therefore, not further analysed.

The mean salivary cotinine concentration was significantly increased in PTS-exposed children compared with PTS-unexposed children (4.00 ng/ml, 95% CI 2.46–5.54 *versus* 1.81 ng/ml, 95% CI 0.37–3.26, $p < 0.05$). Further, in a dose-dependent way, the mean salivary concentration was significantly higher in children whose father or mother was a smoker ($p < 0.05$, Table 2). The associations remained significant when control-

ling for age and PI. The cotinine concentration in urine was also elevated in PTS-exposed children but not significantly ($p > 0.05$). The correlation between salivary and urine cotinine concentrations was $r = 0.72$ ($p < 0.05$).

PTS exposure and periodontal health

The clinical characteristics of the participants according to PTS exposure are shown in Table 3. CAL was significantly associated with PTS exposure, the mean (95% CI) distances from the cemento-enamel junction being 0.23 mm (95% CI 0.17–0.29) in PTS-exposed compared with 0.14 mm (95% CI 0.08–0.19) in PTS-unexposed children (ANOVA $F = 4.9$, $p < 0.05$). The mean CAL distance from the cemento-enamel junction was, further, significantly greater in children whose father was a smoker compared with children whose father was a non-smoker (ANOVA $F = 3.2$, $p < 0.05$, Table 4), suggesting that the length of the periodontal attachment was less in PTS-exposed children. The associations remained significant when controlling for age, gender and PI in multivariate analysis (ANOVA $F = 5.1$ and 2.5, respectively, $p < 0.05$). There were no statistically significant associations between PTS exposure and PD, PI, GI or BOP (ANOVA $F = 1.0$, 0.4, 0.1 and 0.2, respectively, $p > 0.05$).

Table 1. Cotinine levels in body fluids of children

Body fluid	PTS-exposed ($n = 51$)		PTS-unexposed ($n = 58$)	
	mean	95% CI	mean	95% CI
Saliva (ng/ml)	4.00	2.46–5.54*	1.81	0.37–3.26
Urine (ng/ml)	61.9	33.0–90.7	35.8	8.7–62.8
GCF (pg/μl)	–	–	–	–

Mean and 95% CI according to passive tobacco smoking (PTS) exposure
*$p < 0.05$.
CI, confidence intervals; GCF, gingival crevicular fluid.

Table 2. Salivary cotinine concentration (ng/ml)

Smoking consumption	Father			Mother		
	N	mean*	95% CI	*N*	mean**	95% CI
No	43	1.55	− 0.09; 3.20	77	2.03	0.80; 3.27
< 10 cigarettes/day	14	1.19	− 1.69; 4.07	23	5.73	3.47; 8.00
10–20 cigarettes/day	34	3.44	1.59; 5.29	6	1.09	− 3.33; 5.53
> 20 cigarettes/day	18	6.03	3.48; 8.57	3	4.50	− 1.77; 10.77

Mean and 95% CI according to cigarette consumption of parents.
*ANOVA $F = 3.4$, $p < 0.05$.
**ANOVA $F = 3.0$, $p < 0.05$.

Table 3. Clinical characteristics of children

Characteristic	PTS-exposed ($n = 51$)		PTS-unexposed ($n = 58$)	
	mean	95% CI	mean	95% CI
PI	1.42	1.32–1.52	1.38	1.28–1.47
GI	1.51	1.41–1.61	1.50	1.40–1.59
BOP (%)	80.4	71.6–89.2	82.8	74.5–91.0
PD (mm)	2.01	1.90–2.12	1.96	1.85–2.06
CAL (mm)	0.23	0.17–0.29*	0.14	0.08–0.19

Mean and 95% CI according to passive tobacco smoking (PTS) exposure.
*$p < 0.05$.
BOP, bleeding on probing; CAL, clinical attachment level; GI, gingival index; PD, probing depth; PI, plaque index.

Table 4. Clinical attachment level (mm)

Smoking consumption	Father			Mother		
	N	mean*	95% CI	*N*	mean**	95% CI
No	43	0.14	0.07; 0.20	77	0.19	0.14; 0.24
< 10 cigarettes/day	14	0.33	0.22; 0.44	23	0.18	0.09; 0.27
10–20 cigarettes/day	34	0.16	0.09; 0.23	6	0.10	− 0.07; 0.27
> 20 cigarettes/day	18	0.20	0.10; 0.30	3	0.01	− 0.23; 0.25

Mean and 95% CI according to cigarette consumption of parents.
*ANOVA $F = 3.2$, $p < 0.05$.
**ANOVA $F = 1.0$, $p > 0.05$.

Discussion

In the present study, which seems to be the first to report on the possible influence of passive smoking on the periodontal health of children, the clinical condition as well as the salivary, urinary and GCF levels of cotinine were explored in children of smoking and non-smoking parents. The results suggested that the salivary levels of cotinine were significantly elevated in PTS-exposed children. In addition, among the clinical variables studied, a significantly greater CAL distance from the cemento-enamel junction was observed in exposed children. In this study, we assessed the exposure to PTS using both cotinine levels and parental smoking questionnaire data in 109 children aged 6–12 years.

Exposure to passive smoking of children may be estimated from questionnaires about parents' smoking behaviour or from tests of urinary and salivary cotinine levels (Fielding & Phenow 1988, Ronchetti et al. 1992, 1994, Boyacı et al. 2006). The possibility of measuring cotinine in body fluids allows us to objectively measure the exposure to tobacco smoke (Ronchetti et al. 1994). It is known that the cotinine concentrations vary widely in different body fluids (Benowitz et al. 1983, Caraballo et al. 1998). Ronchetti et al. (1994) reported that the salivary cotinine concentration is a reliable indicator of exposure to PTS. Blood levels of cotinine most closely reflect the dose of nicotine absorbed from PTS. In the present study, because it involved children, blood sampling was not preferred; although it has been shown that saliva and blood cotinine levels are highly correlated, with a saliva to blood ratio of 1.1–1.4 (Benowitz 1996). Also, urinary cotinine might be a reliable marker

of PTS exposure (Greenberg et al. 1984, Wald et al. 1984). Jarvis et al. (1984, 1987) suggested that cotinine provides the best discrimination and, therefore, is the marker of choice where high accuracy is important. Their data showed a high degree of inter-correlation between the concentration of cotinine in urine and saliva and do not allow a recommendation of one method over the other (Jarvis et al. 1984). In the present study, the salivary cotinine concentration was significantly higher in children exposed to passive smoking compared with unexposed children. Further, salivary cotinine concentration was higher in children whose father or mother was a smoker. In addition, the urinary cotinine concentration was elevated in PTS-exposed children, but not significantly so.

In our study, the GCF cotinine level, unfortunately, was too low to be detected in most children. The method chosen for GCF collection by paper strips for 30 s was one that is generally preferred (Rossomando et al. 1990, Reinhardt et al. 1993, Atilla & Kütükçüler 1998). Therefore, the most likely reason for the low GCF cotinine detection rate was the extremely small quantities of GCF collected.

Several investigators have documented increasing cotinine levels with increasing levels of self-reported PTS exposure (Jarvis et al. 1984, Tunstall-Pedoe et al. 1991, Benowitz 1996). In our study, the correlation between self-report and salivary cotinine, although statistically significant, was rather weak (data not presented). However, Yamamoto et al. (2005) found no association between self-reported PTS exposure and salivary cotinine concentrations in adults and concluded that salivary cotinine may not be superior to self-reporting.

In the present study, the CAL distance from the cemento-enamel junction was significantly greater in PTS-exposed children and, furthermore, significantly greater in children whose father was a smoker compared with children with a non-smoker father. Our results, suggesting that the length of the periodontal attachment is reduced in children exposed to passive smoking, agree with the observation of Arbes et al. (2001) that adults exposed to passive smoking among persons in the United States who had never used tobacco were more likely to display periodontal disease than those not exposed. The present observations call attention to the possibility that passive smoke might interfere with periodontal tissue metabolism in children of a young age. It has been previously demonstrated that active smoking in adolescence causes loss of periodontal attachment (Hashim et al. 2001). Furthermore, Yamamoto et al. (2005) reported that passive smokers as defined by salivary cotinine displayed a significantly more severe periodontal status than non-smokers and argued that passive smoking should be considered as an independent risk indicator of periodontal disease.

The reason why passive smoking may negatively influence the periodontal attachment in children is unclear. It may be due to a retarded growth rate of the periodontal tissues. A negative influence of PTS exposure on the development of different organs has been described in several studies. Kieser et al. (1996) suggested that there was approximately a 4-month delay in the maturation of permanent teeth in children exposed to parental tobacco smoke as compared with unexposed children. Zavras et al. (1997) observed that exposure to environmental tobacco by-products played a significant role in determining reductions in the nasal volume of children, and argued that such an effect on the normal craniofacial development of a growing child should force parents to reconsider smoking while at home. According to the results of these studies, it seems possible that PTS exposure might affect the development of the periodontal tissues in children. The lack of an association between PTS exposure and PD measurements in the present study further supports the speculation that tobacco smoke by-products do not primarily act on the periodontium by causing deepening of pockets but rather by interfering with (re)generation properties of the connective tissues. The growth rate of the children, however, was not evaluated in the present study. Further studies regarding the relations between PTS exposure, periodontal condition and development rate in growing children, therefore, seem warranted.

In the present study, no associations were found between PTS exposure and PI, GI or BOP. Gingival inflammation and PI measures were similar in both PTS-exposed and unexposed children. Several previous reports in adults suggest a depressed clinical inflammatory response in active smokers (Preber & Bergstrom 1985, Bergstrom & Preber 1986, Bergstrom 1990, Lie et al. 1998, Bergstrom & Bostrom 2001, Dietrich et al. 2004).

In conclusion, we have observed that children who are exposed to passive smoking have elevated cotinine levels in their saliva concomitant with a greater CAL distance from the cemento-enamel junction. These observations based on parents' self-reported smoking may indicate interference of tobacco smoke by-products with the periodontal tissues in children who are exposed to passive smoking. Our observations warrant further investigation of the influence of PTS on the periodontal health of the young.

Limitations of the study

This study has several limitations. First, we did not collect data on the body mass index of the children. In the literature, it was suggested that passive tobacco smoke may affect the normal craniofacial development of a growing child (Zavras et al. 1997). The inferior CAL of PTS-exposed children might result from a reduced growth rate of the periodontal tissues. Second, although collected by an examiner masked to smoking exposure status, measurements were not performed in duplicate. Third, the clinical measurements were based on four teeth of each child, from which GCF was collected. Therefore, the representativity of these four teeth may be argued.

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Address:
Ebru Olgun Erdemir
Department of Periodontology
Faculty of Dentistry
Kirikkale University
71100 Kirikkale
Turkey
E-mail: olgun_ebru@yahoo.com

Clinical Relevance

Scientific rationale for the study: Passive smoking has been associated with a number of negative health outcomes in children. No studies have examined the possible influence of passive smoking on the periodontal health of children. Therefore, the periodontal health of the children and exposure to passive smoking were evaluated.

Principal findings: Children who are exposed to passive smoking have elevated cotinine levels in their saliva concomitant with a greater CAL distance from the cemento-enamel junction.

Practical implications: The observations based on parents' self-reported smoking suggested that the periodontal tissues in children who are exposed to passive smoking may be negatively affected.