Research Article

The effects of maternal environmental tobacco smoke exposure on periodontal health and mother-infant bonding in relation to salivary cotinine level

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Abstract: Background: Environmental tobacco smoking is produced by active smokers burning the tip of a cigarette and breathed by nonsmokers and measured by cotinine level. It has the potential to raise the risk of periodontal disease. One of the most frequent chronic diseases in adults is periodontal disease. The lower maternal-fetal attachment has been found to predict smoking status in previous studies, but no research has examined whether maternal-fetal attachment predicts environmental tobacco smoking. This study assessed the effects of maternal environmental tobacco smoke exposure on periodontal health and mother-infant bonding concerning salivary cotinine levels. Materials and methods: This is a comparative cross-sectional study comparing environmental tobacco smoke on exposed and non-exposed mothers aged between 20-35 years with their infants aged up to one year who attended primary health care centers in rural areas of AL-Karkh sector/Baghdad. Along with the essential socio-demographic data, a secondhand smoke exposure scale and postpartum bonding questionnaire were employed. Collection of unstimulated saliva from mothers was done according to Navazesh and Kumer in 2008. After that, the clinical Assessment of gingival bleeding and periodontal pockets was performed by using Community Periodontal Index according to the world health organization in 1997. Results: Out of 150 subjects,67(44.66%) were exposed to environmental tobacco whereas the non-exposed mothers were composed of 83 (55.33%). The highest mean number of CPI0(healthy gingiva) and CPI1(gingival bleeding) were among the non-exposed mothers while the highest mean number of CPI2(dental calculus), CPI3 (shallow pocket 4-5mm) and CPI4(deep pocket 6mm or more) were among the exposed mothers. The mean value of cotinine level among the non-exposed mothers was lower than exposed mothers with significant results. A higher salivary cotinine level was linked to a lower maternal-fetal bonding score. Conclusions: Mother's exposure to environmental tobacco smoke significantly negatively impacts periodontal disease. Furthermore, mothers who have a stronger sense of attachment and affiliation to their fetus have lower salivary cotinine concentrations than mothers who have a less sense of fetal attachment.

Keywords: cotinine, environmental tobacco smoke, mother-infant bonding, periodontal disease.

Introduction

Smoking is inhaling the smoke of burning tobacco encased in cigarettes, pipes and cigars. Nicotine, tar, and gases like carbon dioxide and carbon monoxide are the main components of tobacco smoke. Toxic tobacco smoke components can be detected not only in the smoke breathed by the smoker but also in environmental tobacco smoke (ETS), also referred to as secondhand smoke (SHS)—that is, the smoke

that a smoker exhales (mainstream smoke) (MS)and the smoke that rises straight from burning tobacco (sidestream smoke) (SS) [1]. Environmental tobacco smoke (ETS) exposure (also known as "passive smoking") is known to have negative health effects, including an increased risk of cardiovascular disease, lung cancer, and respiratory illness [2]. One of the most frequent chronic diseases in adults is periodontal disease [3]. It is commonly established that tobacco smokers have worse periodontal inflammatory diseases than people who do not use tobacco [4-5]. An Iraqi study concluded that smoking subjects showed worse periodontal conditions than non-smoking subjects [6]. Also, another Iraqi study done among children suggested that the mean value of plaque index and gingival index were higher among passive smokers than in the control group with a statistically significant difference [7].

Furthermore, Nonsmokers with secondhand smoking solely at home or both at home and elsewhere had a considerably greater frequency of periodontal disease than the nonsmokers without secondhand smoking and secondhand smoker had the same risk of developing periodontal disease as the current smokers; even smokers are at a higher risk for periodontal disease if they are exposed to secondhand smoke [8].

Sanders et al. [9] revealed that secondhand smoking exposure increased periodontal disease dose-dependently. Meanwhile, Benowitz et al. [10] state that periodontal disease may be influenced by both active and passive smoking via similar mechanisms. Compared to current smokers, nonsmokers exposed to secondhand smoke absorb approximately one-third of the nicotine per cigarette [8].

Cotinine is a tobacco alkaloid and the primary nicotine metabolite. It has been utilized as a reliable biomarker for tobacco exposure in the literature because of its prolonged half-life (10–30 h) compared to nicotine (30 min) [10]. Cotinine is measured in nanograms per milliliter (ng/mL). Passive smokers had values of less than five ng/mL; heavy passive smokers had values of 10 ng/mL or slightly higher; infrequent regular smokers had levels of 10 to 100 ng/mL; and regular active smokers had levels of 100 ng/mL or higher [11] . In general, nonsmokers exposed to secondhand smoke absorb nicotine and other components at the same rate as smokers, and the more passive cigarette smoke they are exposed to, the higher the amount of these components in their systems becomes [12]. Because saliva is a simple and non-invasive collection procedure, it is an appealing alternative to traditional biological matrices (such as blood and urine) for measuring cotinine concentrations [13]. Determination of cotinine levels in unstimulated whole saliva is often performed by using enzyme-linked immunosorbent assay [ELISA] [14-16].

Interaction that is both emotional and reciprocating between a mother and her baby is referred to as bonding. Its goal is to keep the baby attached to their carers while keeping the parents close by [17]. It has been discovered that maternal-fetal attachment and salivary cotinine one day after birth have a negative relationship, with the lower reported maternal-fetal attachment being linked to greater salivary cotinine at one day after delivery [18].

As far as there is no previous studyln Iraq concerning the effects of maternal environmental tobacco smoke exposure on periodontal health and mother-infant bonding concerning the salivary cotinine level, this study was conducted. It suggested the null hypothesis that there is no relation between ETS and periodontal disease and mother-infant bonding.

Materials and Methods

The study was conducted using a cross-sectional comparative design among mothers attending the primary health care centers in rural areas of AL-Karkh sector/Baghdad government for child's scheduled immunization starting from December 2021; till March 2022. They were with the age ranged from 20-35 years with their infants aged up to one year. Using G power 3.1.9.7 (Program written by Franz-Faul, Universitatit Kiel, Germany) with the power of study=80%, alpha error of probability=0.05 two-sided. A pilot study was done on ten women for each group and the CPI score was measured for them and the mean CPI score 2 ± SD for environmental tobacco smoke exposure and non-exposed smokers were (1.00±0.12) and (0.9± 0.26) making the Cohen's D effect size about 0.5 (medium) between the two groups with all these conditions the sample size was 132 adding 10 % as an error rate [19]. Thus the sample size was about 145, so 150 subjects was enough and more calculatable than G power. Cohen D were: Small =0.3, medium=0.5, large>=0.8 [20]. This study compares mothers who were exposed to environmental tobacco smoke and mothers who were not exposed to environmental tobacco smoke. Before starting the study, approval was achieved from the Ministry of Health for women's examinations. Verbal consent were obtained from all women and the ethical committee had accepted the study's protocol in the College of Dentistry, University of Baghdad. The research comprised healthy mothers without a medical problems and any medication also pregnant mothers will not be included in the study. A proforma was given to each participant to collect socio-demographic information such as age, educational level, employment, manner of delivery, feeding pattern, and baby's age and gender. The duration of the exposure (years or months), the smoking person (husband or another person), and the type of exposure (cigarettes, cigarettes, and hookah) were all factors in determining the mothers' exposure status. The number of cigarettes smoked per day was then calculated using a secondhand smoke exposure scale (SHSES) [21]

Following that, the postpartum bonding Questionnaire (PBQ) was distributed to all of the sample (mothers) who attended the health center. The PBQ is a self-report tool that evaluates a mother's feelings and attitudes toward her infant. It has 25 items scored on a six-point scale (0 to 5) [22]. Higher scores suggested that the mother has a poor attachment to the infant and she is under more psychological stress as a mother. Issa [23] had already obtained the Arabic version and translation validity of PBQ in the Ministry of higher education and scientific research; two translators translated the test items from their original language to the target language (Arabic). Subgroups of 40 mothers were taken arbitrarily from the two groups to compare the salivary cotinine level. Unstimulated salivary samples were collected by drooling into the test tube according to the University of Southern California School of Dentistry guidelines for saliva collection [24]. One hour before the test session, the mothers were told to avoid eating or drinking anything (except water). The mothers were instructed to rinse their mouths with distilled water multiple times before relaxing for five minutes. During the collection, mothers should make as few movements as possible, especially mouth movements, and lean the head forward with their lips slightly open to let saliva flow into the tube. After collecting saliva, each salivary sample was centrifuged (at 2000-3000 rpm) for approximately 20 minutes. After centrifugation, the clear samples were collected by a micropipette and stored in Eppendorf tubes at (-20 C) in a deep freeze until biochemical analysis.

After the collection of saliva, the Community Periodontal Index (CPI) was used to assess the gingival bleeding and periodontal pockets clinically as it is recommended by the WHO [25] as an indicator of early periodontal disease. Clinical examinations were carried out using a plane mouth dental mirror and CPI probe. The concentration of salivary cotinine level was detected by an enzyme-linked immune-sorbent assay (ELISA) using a salivary cotinine kit. The reagent preparation concept, technique assay, and result computation were all conducted according to the manufacturer's procedure instructions.

Statistical analysis

The Statistical Package for Social Science was used to conduct the statistical analysis (SPSS version -22, Chicago, Illionis, USA). The Frequency, percentage, mean and standard error were calculated using descriptive analysis with simple and cluster chart bars. The difference between the two groups was tested using inferential analysis as an independent sample t-test parametric test. For the linear correlation between two quantitative variables, the Pearson correlation parametric test was used. Receiver operating Characteristic Curve (ROC) for Optimal Cutoff point for differentiation between the two groups.

Results

The sample of this study consisted of 150 mothers with their infants. They were subdivided into two groups according to the ETS exposure: mothers who were exposed to ETS with their infants, which composed 67 (44.66%) mothers, and non-exposed mothers with their infants, which composed 83 (55.33%) mothers. According to SHSES, all the exposed mothers were exposed to ETS at home 67 (100%). In addition to home exposure, 23 (34.32%) and 22 (32.83%) of them were exposed to vehicles and public places respectively, while the least exposure was at work 8 (11.94%).

The results showed that the percentage of mothers in the age range 20-27 was lower than the older age group among passive smokers, while the opposite result was found concerning the non-exposed mothers, as shown in Table 1**.**

Table 1: The Distribution of the sample according to age and environmental tobacco smoke exposure groups

Age (years)	Exposed mothers		Non-Exposed mothers	
20-27	32	39.51%	49	60.49%
28-35	35	50.72%	34	49.28%

Table (2) demonstrates that the husband was the primary source of smoking at home, accounting for 55 (82.08 %), with the remaining 12 (17. 92%) coming from persons other than the husband. Concerning the exposure duration, 58 (86.57 %) had been exposed for years, whereas 9 (13.43 %) had been exposed for months.

Table 2: Smoking Exposure Characteristics of environmental tobacco smoke-exposed mothers

Variable	Category	N=67
Smoking person	Husband	55(82.08%)
	Others than husband	12(17.92%)
Duration of exposure	years	58(86.57%)
	months	9(13.43%)
Type of the exposed Smoking	Cigarette only	51(76.11%)
	Cigarette and Hookah	16(23.89%)

The results showed that the non-exposed mothers had healthy periodontium more than those who were exposed. However, the difference was not statistically significant. While the exposed mothers were found to have significantly lower gingival bleeding (score1), and more calculus (score2) in addition to having a more shallow pocket (score3) than the non-exposed mother, however, the same result was found concerning deep pockets (score4), but the difference was not statistically significant as shown in Table (3).

Table 3: Descriptive analysis and statistical difference of periodontal health status among the environmental tobacco smoke Exposed and Non-Exposed mothers

Vars.	Environmental tobacco smoke exposure					
	Exposed mothers		Non-Exposed mothers		t- test	p- value
	Mean	±SE	Mean	±SE		
CPI0	0.821	0.147	1.217	0.142	1.918	0.057
CPI1	2.224	0.166	4.096	0.143	8.588	0.000*
CPI2	2.239	0.131	0.566	0.094	10.60	0.000*
CPI3	0.627	0.104	0.145	0.052	4.400	0.000*
CPI4	0.030	0.030	0.000	0.000	1.114	0.267

df=148 *=significant at $p \leq 0.05$

Figure 1 demonstrates that the percentage of mothers with the highest CPI 0 (healthy gingiva) and CPI 1 (gingival bleeding) were among the non-exposed group. Regarding CPI 2 (dental calculus), CPI 3 (shallow pocket 4-5mm) and CPI 4 (deep pocket 6mm or more), the highest number was found among the exposed mothers.

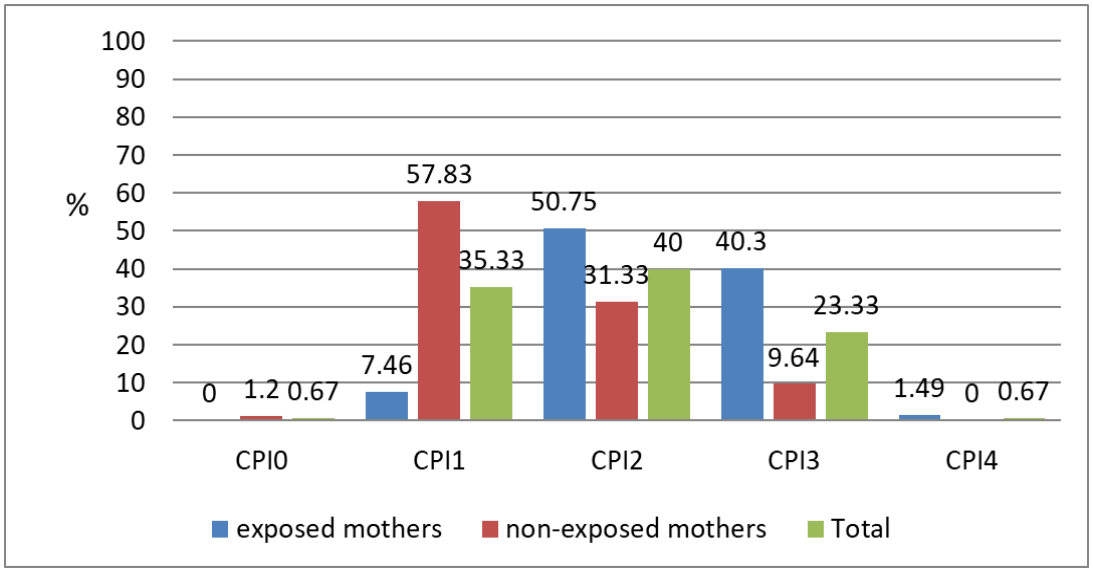

Figure 1: Distribution of mothers according to the highest score of community periodontal index

Regarding salivary cotinine level, the findings revealed that the mean values and standard error of cotinine level among the non-exposed mothers were lower than the exposed mothers and the data showed significant results as shown in Figure 2.

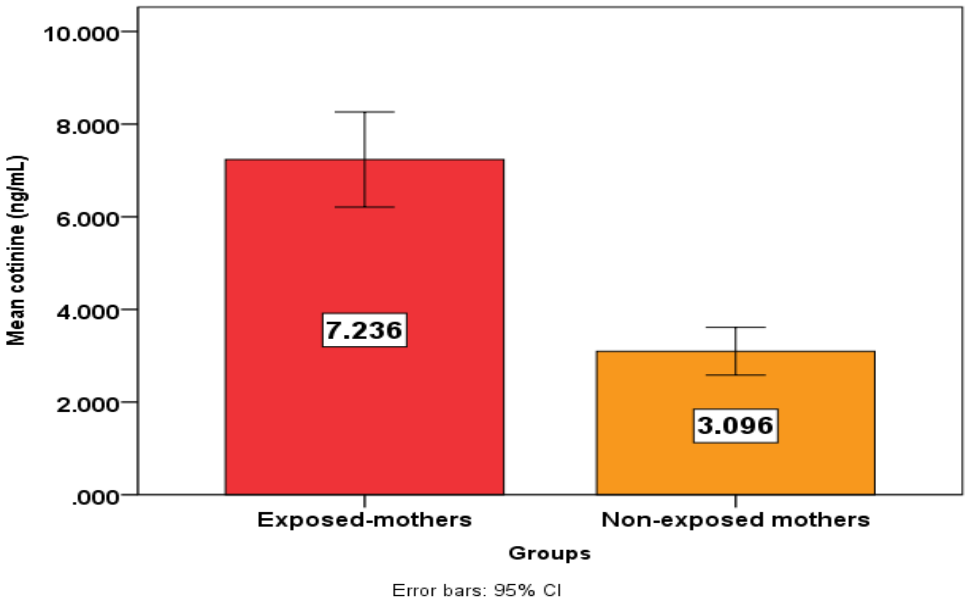

Figure 2: Descriptive and statistical tests of Salivary cotinine levels among the environmental tobacco smoke Exposed and Non-Exposed mothers

Table (4) shows that salivary cotinine was excellent in differentiation ETS exposed from non-exposed mothers with significant results and sensitivity of 95% and specificity of 85% among participants.

Table 4: Cotinine cutoff level

Area Under the Curve				
Optimal Cutoff point	**%Sensitivity**	**%Specificity**	**Area**	**P-value**
4.69350	95	85	0.946 excellent	0.000 *

*=significant at p≤ 0.05

The correlation between salivary cotinine level the periodontal health concerning environmental tobacco smoke exposure is displayed in Table (5). Salivary cotinine level had a negative correlation with CPI0 (healthy gingiva) and CPI1(gingival bleeding) among the exposed and the non-exposed mothers and the results were significant statistically (p≤0.05) concerning CPI1 among the two groups. At the same time, there were positive correlations with other CPI scores with significant correlation regarding CPI2 among the non-exposed mothers.

Table 5: Correlation Coefficient of salivary cotinine level with periodontal parameters

Environmental tobacco smoke exposure		**Total**	
		Cotinine	
		r	p
Exposed mothers	CPI0	-0.084	0.606
	CPI1	-0.446	**0.004***
	CPI2	0.524	**0.001***
	CPI3	0.056	0.731
	CPI4	0.108	0.508
Non-Exposed mothers	CPI0	-0.298	0.062
	CPI1	-0.638	**0.000***
	CPI2	0.564	**0.000***
	CPI3	0.082	0.617

*=significant at p≤ 0.05

The correlation between salivary cotinine and bonding score by groups was displayed in Table (6). The results reported that bonding score significantly correlated with cotinine levels among the exposed and the non-exposed mothers (p≤0.05).

Table 6: Correlation of salivary cotinine with bonding score by exposure groups.

Groups		**Bonding score**	
		r	**p**
Exposed mothers	Cotinine	0.544	0.000*
Non-Exposed mothers		0.388	0.013*

*=significant at p≤ 0.05

Discussion

This study was the first that evaluate the effects of maternal environmental tobacco smoke exposure on periodontal health status and mother-infant bonding based on salivary cotinine levels in Iraq. The mothers who participated in the study were housewives from rural areas, and the most prevalent location of exposure was at home, with the husband being the primary source of exposure. These results were consistent with a study by Saleh et al. in 2021 in Egypt [26]. Simple explanations for these findings include the presence of various social and cultural norms in rural communities that make them vulnerable to male dominance; additionally, living in a rural area with a lower level of education increases the risk of ETS exposure at home, as well as long periods of parental stay at home due to early day hours work and a lack of recreational activities in rural areas compared to urban areas. Another research found that nicotine and other tobacco smoke particles contaminated 88% of surfaces in living rooms and newborn bedrooms [27].

This study found that CPI 1 (gingival bleeding) was higher among non-exposed mothers with statistically significant differences, which can be explained by the fact that active and secondhand smoking may have similar mechanisms of action on periodontal disease [12]. This result agreed with Gautam et al. [28], who found statistically significant differences between cigarette smokers and nonsmokers for CPI score of 1 (nonsmokers are more likely to have gingival bleeding). They explained this result by the finding that nicotine, one of several tobacco smoke byproducts, causes local vasoconstriction, which reduces blood flow and edema, as well as inhibiting the early indications of periodontal disease by lowering gingival inflammation, redness, and bleeding. Also, this result followed that of Tjahajawati et al. [29] study who indicated that nicotine in cigarettes could cause adrenal hormones to be stimulated. It causes the vasoconstriction of peripheral blood vessels, reducing blood flow and oxygen to the gingiva. As a result, passive smokers' inflammatory and bleeding reactions to probing would be reduced.

Regarding CPI 2 (dental calculus), CPI 3 (shallow pocket 4-5mm) and CPI 4 (deep pocket 6mm or more), the highest number was among the exposed mothers. This result was in agreement with that of Ueno et al. [9] who found that ETS exposed group showed a significantly higher prevalence of periodontal disease compared to the non-exposed group and concluded that passive smokers showed a similar risk of having the periodontal disease to that of the current smokers. Also, the result agreed with the previous Iraqi study, which suggests that nicotine changes the immunological response, which may contribute to smokers' higher risk of periodontal disease [6].

Palmer *et al.* [30] illustrated the correlation between tobacco and periodontal disease by the effect of tobacco in decreasing the oxygen and other blood constituents to reach the gingiva and reducing the capacity to remove the tissue waste products leading to compromising the immune response and periodontal tissue destruction. Another explanation may be attributed to the ability of tobacco smoke and its components to change the bacterial surface and increase biofilm formation in various periodontal pathogens, including *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* [31]. Moreover, inflammatory indicators such as interleukin-1, lactoferrin, albumin, and aspartate aminotransferase have been found to elevate in the saliva of those who had been exposed to cigarette smoke [32]. Another

possible explanation is that cotinine increases the potency of toxins generated by periodontopathogenic bacteria such as *Prevotella intermedia*, *Prevotella nigrescens*, *Treponema denticola*, and *Porphyromonas gingivalis*, which may speed up the periodontal disease progression [33]. However, these results disagreed with the studies that reported no significant association between ETS exposure and periodontal disease [34-35].

The current study revealed a higher cotinine level (the reliable biomarker for environmental tobacco smoke exposure) [16] among ETS-exposed mothers. The results came in agreement with Hassanzad et al. [36] study who concluded that cotinine in saliva was significantly higher among the passive smoker group than the control group. The result also aligned with the other studies which found that the salivary cotinine level in the passive smoker subjects was higher than in the control group [37-38]. Theoretically, nonsmokers who were exposed to secondhand smoke absorb nicotine and other components in the same way as smokers did, and the more passive cigarette smoke they were exposed to, the higher the level of these components in their bodies [12].

Also, the results of the current study reported that cotinine level had a positive correlation with the bonding score. This result was in accordance with that of Magee et al. [18], who stated that women with fewer sentiments of connection to their fetus had higher salivary cotinine levels during pregnancy and postpartum. The possible explanations for this positive correlation of cotinine with bonding score were that the increase in bonding score indicated the pathology (Mother-Infant Bonding Disorder) [39] and there was a well-established connection between nicotine and depression [40]. Nicotine binds to, activates, and desensitizes nicotinic acetylcholine receptors (nAChRs), which might be a key element in nicotine's depressive symptoms effects [41] and since Radoš et al. [42] proved that impaired bonding was related to postpartum stress and depression symptoms. Hence, the highest cotinine level led to increasing in bonding score.

A meta-analysis done by Chen et al. [43] indicated that prenatal smoking had been linked to postpartum depression. While women might have postpartum depression, smokers were at a greater risk than nonsmokers [44]. Another possible explanation linked to maternal exposure to tobacco smoke in pregnancy was a risk factor for preterm birth (birth before 37 weeks gestation) [45] and preterm birth influence negatively on the bonding relationship [46].

Furthermore, there was a clear link between maternal and paternal smoking and the inability to start breastfeeding a child. A person's smoking habits reflect their attitude toward health. As a result, smoking mothers are likely to be less educated and passionate about breastfeeding than nonsmoking mothers. Similarly, fathers who smoke may be a proxy for a poorer degree of health consciousness in the home, which may influence their wives' breastfeeding decisions [47] and since breastfeeding promotes an intimate touch between the mother and infant and so creates a better relationship than bottle-feeding mothers in the early period following childbirth [48].

Conclusion

The present study's findings concluded that a mother's exposure to environmental tobacco smoke harms periodontal disease. It was found that environmental tobacco smoke exposure was associated with lesser gingival bleeding and deeper pockets as compared to the non-exposed mothers. Furthermore, the lower maternal-fetal attachment was associated with the greater exposure to environmental tobacco smoke and those who had a stronger sense of attachment to their fetus had lower salivary cotinine concentrations than the mothers who had a less sense of fetal attachment.

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Conflict of interest: None.

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العنوان: آثار التعرض لدخان التبغ البيئي للأمهات على أمراض اللثة والترابط بين الأم والرضيع فيما يتعلق بمستوى الكوتينين اللعابي

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المستخلص:

الخلفية: اثبت الدراسات السابقة ان التعلق بين الأم والجنين يتاثر بحالة التدخين، ويؤدي إلى زيادة خطر الإصابة بأمراض اللثة. أجريت هذه الدراسة لتقييم آثار التعرض لدخان التبغ البيئي للأمهات على صحة الفم والترابط بين الأم والرضيع فيما يتعلق بمستوى الكوتينين اللعابي .

المواد وطرق العمل: هذه دراسة مقارنة مقطعية تقارن بين الأمهات اللواتي تعرضن لدخان التبغ البيئي باستخدام مقياس خاص والأمهات غير المعرضات تتراوح أعمارهن بين 20-35 سنة وأطفالهن الرضع تصل أعمارهم إلى سنة واحدة بالاضافة الى استبيان للترابط بعد الولادة. تم جمع اللعاب ثم تم إجراء التقييم السريري لنزيف وجيوب اللثة باستخدام مؤشر اللثة المجتمعي وفقا لمنظمة الصحة العالمية.

النتائج: وجدت النتائج ان 44.66 %من العينة تعرضوا لدخان التبغ البيئي و منهم أكبر عدد لديهم تكلسات الاسنان وجيب ضحل و جيب عميق . وكان متوسط قيمة مستوى الكوتينين بين الأمهات غير المعرضات أقل من الأمهات المعرضات للتدخين وتظهر البيانات نتائج مهمةحيث ارتبط انخفاض درجات التعلق بين الأم والجنين بارتفاع مستوى الكوتينين اللعابي.

الاستنتاج: إن تعرض الأم لدخان التبغ البيئي له تأثير ضار مؤثر على أمراض اللثة. وقد وجد أن التعرض لدخان التبغ البيئي كان مرتبطا بالجيوب العميقة مقارنة بالأمهات غير المعرضات و كذلك ، فإن أولئك الذين لديهم مشاعر أكثر من الانتماء والتعلق بجنينهم لديهم تركيزات أقل من الكوتينين اللعابي