ORIGINAL ARTICLE

Eastern Journal of Medicine

East J Med 30(2): 183-190, 2025
DOI: 10.5505/ejm.2025.47786

Cord Blood Inflammatory Markers Due to Maternal Smoking Exposure

Sita Andarini[1*], Prasetyo Hariadi[1], Andintia Aisyah Santoso[1], Mochammad Aris Arfiansyah[1], Imam Rahadian Soleman[1], Yuyun Lisnawati[2], Tjatur Kuat Sagoro[3]

[1]Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, University of Indonesia – Persahabatan Hospital, Jakarta, Indonesia
[2]Department of Obstetric and Gynecology, Faculty of Medicine, University of Indonesia – Persahabatan Hospital, Jakarta, Indonesia
[3]Departement of Pediatrics, Faculty of Medicine, University of Indonesia – Persahabatan Hospital, Jakarta, Indonesia

ABSTRACT

Background: Maternal smoking exposure during pregnancy is correlated with negative neonatal well-being due to placental disruption. Currently, there are still conflicting findings on whether smoking affects neonatal inflammatory responses. This study aimed to evaluate the effects of maternal smoking exposure on neonatal wellbeing.
Materials and Methods: This is a cross-sectional study. Pregnant subjects were grouped into three categories: active smokers, passive smokers, and non-smokers. Smoke analyzer was used to measure levels of exhaled carbon monoxide (CO), and the levels of tumor necrosis factor-α (TNF-α), Interferon-γ (IFN-γ), and cotinine in serum umbilical cord were measured by enzyme-linked immunosorbent assay (ELISA). The newborn well-being such as birthweight, birth length, and head circumference was also measured. Statistical analysis was performed using IBM SPSS version 25.
Results: A total of 85 pregnant subjects participated in this study, distributed as 20 active smokers (23.5%), 35 passive smokers (41.2%), and 30 non-smokers (35.3%). Newborns' weight and placental weight had significant differences between each maternal group. IFN-γ and TNF-α in the groups exposed to cigarettes were lower compared to the non-smoker group. Exhaled CO and cotinine cord blood levels were associated with smoking. Serum cord blood TNF-α levels inversely correlated with exhaled CO but were not significant.
Conclusions: Maternal smoking exposure was associated with alteration of neonatal well-being and inflammatory markers.

Keywords: Cord blood, Cotinine, IFN-γ, Smoking exposure, TNF-α

Introduction

Smoking exposure during pregnancy is a crucial modifiable risk factor that remains a concern in public health. In Indonesia, the overall prevalence of smokers in adults was 37.9%, with no recent data on maternal smoking during pregnancy (1). Maternal active smoking is associated with intrauterine growth retardation, stillbirth, preterm birth, low birth weight, and congenital malformations (2,3).

Chahal et al. (4) showed that smoking during pregnancy was associated with increased interleukin-8 (IL-8) in newborns which may already be detected at birth. Furthermore, the changes in neonatal inflammation result in lifelong implications (5). Smoking-induced disruption of oxygen-related responses has been identified to play a role in placental cytotrophoblast proliferation and differentiation during critical early stages of development (6). The damaged placenta cannot transfer sufficient maternal nutrients and oxygen to the fetus, which may cause low birth weight (7).

In this study, we aimed to evaluate the effects of maternal smoking exposure on neonatal inflammatory markers using newborn umbilical cord serum and the anthropometric parameters.

Material and Methods

This is a cross-sectional study evaluating the outcome of neonates as measured by birth weight, birth length, head circumference, placental weight and Appearance, Pulse, Agrimace, Activity, and Respiration (APGAR) score, in correlation with

*Corresponding Author: Sita Andarini, Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, University of Indonesia, Persahabatan Raya Street, Number 1 Jakarta Timur, DKI Jakarta, 13230, Indonesia
E-mail: sita.laksmi@ui.ac.id, Phone: +62 818-867-189
ORCID ID: Sita Andarini: 0000-0003-0169-1109, **Prasetyo Hariadi:** 0009-0000-5790-675X, **Andintia Aisyah Santoso:** 0000-0002-1984-3388, **Mochammad Aris Arfiansyah:** 0000-0002-3820-9489, **Imam Rahadian Soleman:** 0009-0008-0546-5137, **Yuyun Lisnawati:** 0000-0002-2189-386X, **Tjatur Kuat Sagoro:** 0009-0004-5131-5608
Received: 03.06.2024, **Accepted:** 16.02.2025

cord blood inflammatory markers and status of smoking of pregnant subjects. Participants were grouped into three categories: active smokers, passive smokers, and non-smokers (non-smoking within household). We excluded pregnant subjects with: impaired systemic immunity (autoimmune disease, acquired immunodeficiency syndrome/AIDS, on immunosuppressant therapy, poor nutrition) based on history and physical examination; diseases of extra-genital organs (lung, liver, kidney, digestive, urinary, integumentary, nervous, diabetes mellitus) based on anamnesis, physical examination and basic laboratory examination; and bear more than one fetus. Participant's recruitment flow is depicted in Figure 1.

All demographic data were collected. Maternal hemoglobin levels were presented in g/dl. Measurements of anthropometric parameters of newborns such as weight (gr), birth length (cm), head circumference (cm), and placental weight (gr) were carried out immediately after the baby was born. APGAR scores were assessed at the first and fifth minutes afterward. Maternal exhaled CO levels were measured using CO analyzer (Smokerlyzer, Bedfont Scientific Ltd., Maidstone, UK). Subjects were asked to inhale fully, hold their breaths for fifteen seconds, and exhale slowly into the analyzer until the indicated time. Five milliliters of blood were collected, and umbilical cord serum was collected, centrifuged at 3,000 rpm for ten minutes, aliquoted, and stored in -20°C refrigerator for further analysis. Umbilical cord serum cotinine levels were evaluated using cotinine ELISA kit (catalog number BQ096D, Bio-Quant Inc., San Diego, CA, USA) according to manufacturer's method. Undiluted 100 μL of serum umbilical cord was used to measure IFN-γ using Quantikine Human IFN-γ immunoassay (catalog number DIF50 R&D Systems, Inc., Minneapolis, MN, USA). Also, 100 μL of undiluted serum umbilical cord was used to measure TNF-α using Quantikine ELISA Human TNF-α immunoassay (catalog number DTA00D, R&D Systems, Inc., Minneapolis, MN, USA), and the absorbance of microplate then read using iMark™ (BioRad, NJ, USA) microplate reader at 450 nm, respectively, according to manufacturer's method.

Statistical analysis was performed using IBM SPSS version 25. Prior to statistical analysis, the normality of data was tested using Kolmogorov-Smirnov test. The measurement results were presented as mean ± standard deviation (SD) for normally distributed data or median with interquartile range [(median(Q1-Q3)] for non-normally distributed data. Baseline subject characteristics were compared using Mann-Whitney U test, Student's t-test, or Chi-square test. For correlation analysis, an independent T-test was used for two-group comparisons and one-way Analysis of Variance (ANOVA) was used for multiple-group comparisons. A value of $P < 0.05$ was considered significant. This study was reviewed and approved by Ethics Committee, with the approval number: 397/PT02.FK/ETIK/2012. All subjects provided informed consent to participate in this study and to have their data published in a journal article.

Results

There were a total of 93 pregnant individuals who gave birth at the Obstetric Unit in our hospital who were willing to participate in this study, eight subjects were excluded from the analysis due to prematurity and stillbirth (2) and incomplete data (6), bringing the total subjects to 85. Pregnant subjects were divided into 3 groups, active smokers, passive smokers, and non-smokers (who were not exposed to cigarettes in household). There is no missing data of participants for each variable interest in this study.

Subject characteristics: There was no significant difference in the characteristics between groups. The mean±SD age was 29.8±6.4 years old, with the youngest subject was sixteen years old and the oldest was 42 years old. Characteristics of the subjects can be seen in Table 1.

History of smoking in pregnant subjects: This study evaluated the level of smoking exposure in pregnant subjects, especially in the active smoker group and the passive smoker group. Pregnant subjects who are active smokers are assessed for the type of cigarettes consumed, number of cigarettes consumed, length of smoking and the last time quit smoking Labor outcome

To evaluate the impact of smoking exposure during pregnancy on fetal growth and development, several anthropometric parameters of the fetus were measured immediately after delivery. The result of neonates'outcomes can be seen in Table 3.

The results of statistical calculations between maternal smoking status and birth weight showed a significant difference in newborn birth weight between maternal groups 95% CI: 2,942.15 – 3,117.85; P=0.016. There were no differences in the measurement of the APGAR score at 1 st

Table 1: Distribution of Subjects According to Characteristics and Research Groups

Characteristics	Active Smoker n=20 (23.5%)	Passive Smoker n=35 (41.2%)	Non-smoker n=30 (35.3%)
Age (years)			
Mean±SD	29.48±7.90	30.29±6.10	29.61±5.50
Hemoglobin (g/dl)			
<11	5(25)	10(28.5)	9(30)
≥11	15(75)	25(71.5)	21(70)
Education			
High (bachelor/diploma)	3(15)	2(5.7)	6(20)
Intermediate (senior high school)	9(45)	18(51.4)	15(50)
Low (elementary school-junior high school)	8(40)	15(42.9)	9(30)
Pregnancy/Gravidity			
Primary	5(25)	7(20)	14(46.6)
Secondary	5(25)	14(40)	8(26.7)
Multigravidity	10(50)	14(40)	8(26.7)
Delivery Method			
Cesarean delivery	3(15)	1(2.8)	2(6.7)
Vaginal delivery	17(85)	34(97.2)	28(93.3)
Labor Complications			
None	14(70)	29(82.8)	30(100)
Exist	6(30)	6(17.2)	0(0)
Maternal CO Exhalation (ppm)			
0-5.99	3(15)	4(11.4)	28(93.3)
6-10.99	6(30)	30(85.7)	2(6.7)
≥ 11	11(55)	1(2.9)	0(0)

minute and 5th minutes after delivery. The results of statistical calculations showed a significant difference in placental weight for each group of pregnant subjects 95% CI: 488.86 – 511.32; $P<0.01$.

Level of exhaled CO levels, umbilical cord serum of Cotinine, IFN-γ and TNF-α: We examined exhaled CO on pregnant subjects as presented in Figure 2. CO levels were 11.7 (8.6 – 13.8) ppm in active smokers, 7.8 (6.8 – 8.7) ppm in passive smokers, and

Table 2: Smoking Exposure

Subject Exposure	Active Smoker n=20 (23.5%)	Passive Smoker n=35 (41.2%)
Brinkman Index		
Light	13(48.1)	19(54.3)
Moderate	12(44.4)	16(45.7)
Heavy	2(7.5)	0(0)
Types of Cigarettes		
Kretek cigarettes with filter	6(30)	22(62.8)
Kretek cigarettes	1(5)	9(25.8)
White cigarettes	13(65)	4(11.4)
Number of cigarettes/day (active smoker)		
1 (light smoker)	8(40)	0(0)
2-9 (moderate smoker)	10(50)	0(0)
≥ 10 (heavy smoker)	2(10)	0(0)
Smoking period (years)		
< 5	5(25)	0(0)
6-10	5(25)	0(0)
≥11	10(50)	0(0)

3.5 (3.1 – 4.2) ppm in non-smokers 95% CI: 6.43 – 9.08; *P*<0.01.

We measured cotinine levels of umbilical cord serum using ELISA method as depicted in Figure 3. Cotinine levels were 13.89 (9.63 – 18.38) ng/ml in active smokers, 7.25 (2.90 – 9.25) ng/ml in passive smokers, and 0.74 (0.68 – 0.87) ng/ml in non-smokers 95% CI: 5.16 – 8.08; *P*<0.01.

There was a significant positive correlation between maternal exhaled CO levels and umbilical cord serum cotinine levels (*P*<0.01; r=0.724) which is depicted in Figure 4. There was a significant negative association between umbilical cord serum cotinine levels and birth weight (*P*=0.001; r=-0.382) as presented in Figure 5.

Measurement of inflammation markers in umbilical cord blood was carried out to assess the inflammatory changes that may occur due to maternal cigarette exposure, including IFN-γ and TNF-α. IFN-γ levels were 4.03 (1.50 – 6.84) pg/ml in active smokers, 3.46 (1.21 – 9.08) pg/ml in passive smokers, and 6.84 (2.06 – 14.42) pg/ml in non-smokers 95% CI: 5.33 – 12.52; *P*=0.384. Meanwhile, TNF-α levels were 26.94 (22.50 – 32.87) pg/ml in active smokers, 27.36 (23.55 – 30.33) pg/ml in passive smokers, and 27.36 (24.61 – 29.69) pg/ml in non-smokers 95% CI 12.68 – 71.81; *P*=0.436. The results can be seen in Figure 6.

There was inverse correlation serum cord blood TNF-α levels, however there was no significant correlation between exhaled CO levels and TNF-α levels in umbilical cord blood (*P*=0.287; r=-0.118) as presented in Figure 7.

Discussion

Differences in Birth Weight and Length Between Active Smokers, Passive Smokers, and Non-Smokers: A significant difference in average birth

Table 3: Neonates Outcome

Characteristics	Active Smoker n=20 (23.5%)	Passive Smoker n=35 (41.2%)	Non-smoker n=30 (35.3%)	P-value
Birth weight (gr)				
Mean±SD	2,883±658.90	3,03±538	3,09±285	0.016
Birth length (cm)				
Median (IQR)	48.9(48.2-50)	48.8(48-50)	49.9(49-51)	0.056
Head circumference (cm)				
Median (IQR)	31.40(30.5-33)	32(30-33)	31.50(30.2-33)	0.062
APGAR score 1st minute				
Normal (8-10)	19(95)	29(82.9)	28(93)	
Mild/moderate asphyxia (6-7)	1(5)	6(17.1)	2(7)	0.082
Severe (0-3)	0(0)	0(0)	0(0)	
APGAR score 5th minute				
Normal (8-10)	19(95)	29(82.8)	29(96.7)	
Mild/moderate asphyxia (6-7)	1(5)	6(17.2)	1(3.3)	0.071
Severe (0-3)	0(0)	0(0)	0(0)	
Placental Weight (gr)				
Mean±SD	446.7±75.2	496.5±85	519±64	0.001

weight was observed between smokers, and non-smokers pregnant women 95% CI: 2,942.15 – 3,117.85; *P*=0.016, with both active and passive smokers showing lower birth weights. This weight loss is attributed to impaired oxygenation. Another study by Kataoka et al. (8) supported this trend, revealing lower birth weights in smoking pregnant subjects. Fetal growth and development are influenced by maternal, fetal, and placental factors, including nutritional status, age, gestational age, and placental function (9,10). However, the newborns' body length measurements did not show a significant difference 95% CI: 48.85 – 49.65; *P*=0.056.

Contrasting results were found by Tatsuta et al. (11) which obtained a significant association between newborns' birth length and maternal smoking during pregnancy. Their research also demonstrated that earlier smoking cessation reduces the adverse effect of fetal growth restriction.

Differences in Placental Weight in Active Smokers, Passive Smokers, and Non-Smokers: Statistical analysis found a significant difference in placental weight for each group of pregnant subjects 95% CI: 488.86 – 511.32; *P*<0.01. Larsen et al. (12) reported lower placental weight in pregnant subjects who smoke, especially throughout the pregnancy, compared to non-smokers. This finding can be explained by the mechanism of nicotine and other components of cigarettes that lead to vasoconstriction in maternal circulation and impair trophoblast proliferation and differentiation, thereby disrupting placental development and growth. Studies also showed that smoking during pregnancy reduces fetal growth relatively more than placental growth. A high ratio of placental weight to birth weight has been linked to negative pregnancy outcomes, possibly

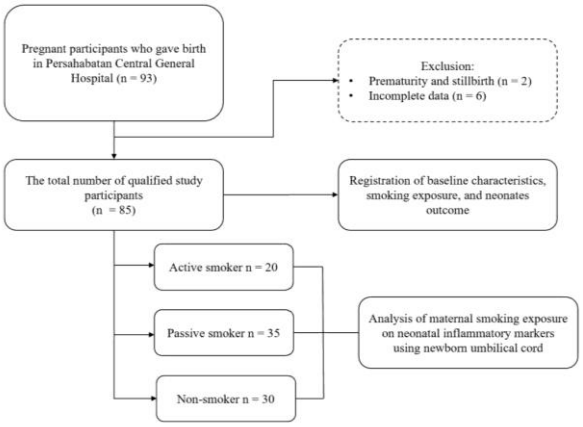

Fig.1. Participants' Recruitment Flow

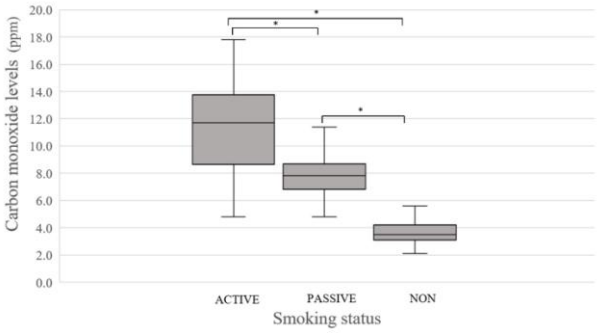

Fig. 2. Exhaled CO and maternal smoking status. (**P*<0.01)

indicating an unfavorable intrauterine environment. It is possible since hypoxia increased placental blood vessel formation as a compensatory mechanism (12,13).

Exhaled CO Levels: Exhaled CO levels can distinguish between smokers, passive smokers, and non-smokers. CO was chosen as a marker because it has a short half-life (two until six hours) and is relatively easy to measure (14,15). Statistical calculations of the relationship between smoking status and exhaled CO levels showed significant differences in exhaled CO levels between groups of active smokers, passive smokers, and pregnant subjects who were not exposed to cigarettes 95% CI: 6.43 – 9.08; *P*<0.01 (Figure 2). Delcroix-Gomez et al. (16) observed the linear relationship between maternal exhaled CO and smoking status during pregnancy. Smoking women were likely to have CO levels ≥3 ppm which significantly increased if they smoked more than ten cigarettes per day.

Umbilical Cord Blood Cotinine Levels: The results of statistical calculations obtained a 95% CI: 5.16 – 8.08; *P* <0.01 (Figure 3) so that it can be concluded that there is a significant association between serum umbilical cord cotinine levels and maternal smoking status. A study by Ramadani et al. (17) used a

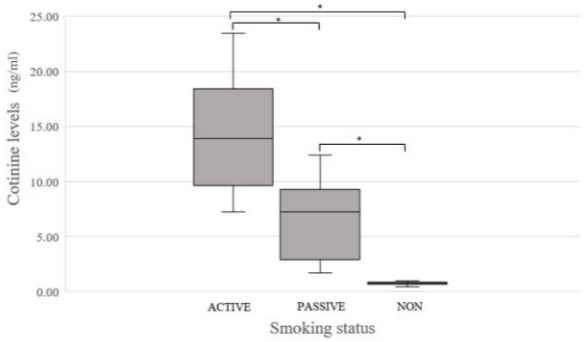

Fig. 3. Cotinine umbilical cord serum levels and maternal smoking status. (**P*<0.01)

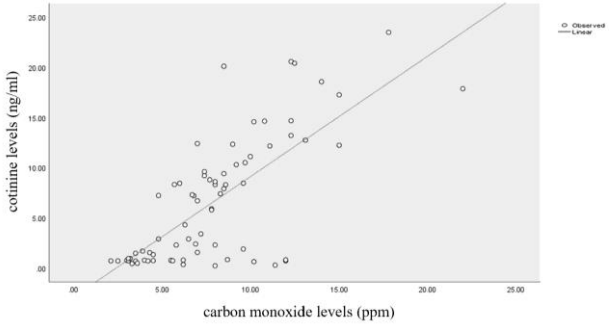

Fig. 4. Correlation between exhaled CO with cotinine levels

concentration of umbilical cord nicotine >1 ng/mL to define exposure to secondhand smoke (mean concentration was 1.3 ng/mL). During pregnancy, nicotine and its primary metabolite act as vasoconstrictors, diminishing uterine blood flow by 30–40%. This decrease in blood flow reduces the delivery of oxygen and essential nutrients necessary for fetal development. The presence of nicotine in the blood can also serve as an indicator of oxidative stress affecting both the mother and fetus.

We also found a strong correlation of maternal exhaled CO levels and umbilical cord blood cotinine levels (*P*<0.01; r=0.724) as depicted in Figure 4. A total of 11 subjects from 20 (55%) pregnant subjects who smoked had exhaled CO levels of more than 10 ppm and relatively higher than other maternal groups. This finding is in line with the theory that high levels of exhaled CO are caused by high consumption or high levels of cigarette exposure. High levels of placental blood cotinine in the active smoker group and in the passive smoker group were due to high tissue uptake and the ability to cross the placental blood barrier in response to cigarette exposure.

Inflammatory Cytokine Markers in Umbilical Cord Blood: In this study, we tried to measure TNF-α and IFN-γ in the cord blood. The median

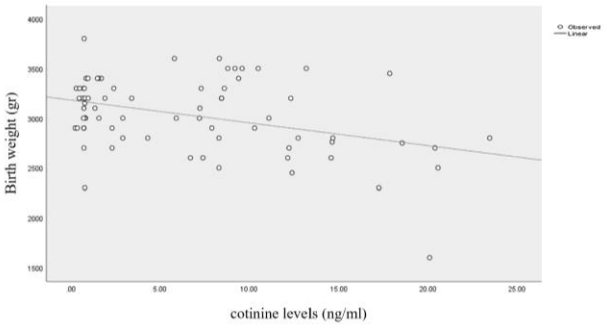

Fig. 5. Correlation between serum umbilical cord cotinines levels with birth weight

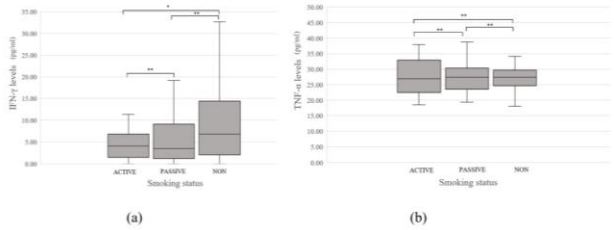

Fig. 6. Inflammation markers in cord blood levels and maternal smoking status; (a) IFN-γ, (b) TNFα. (*P<0.05; **P≥0.05)

concentration of IFN-γ and TNF-α in placental blood in the group of pregnant subjects exposed to cigarettes was lower than non-smoker group (Figure 6). Pregnancy and smoking exposure are likely to promote T-helper 2 (Th2) dominance over T-helper 1 (Th1) immune responses. This shift toward humoral immune system activation leads to the release and inhibition of various cytokines. Furthermore, smoking influences the innate immune response, affecting the regulation of crucial cytokine mediators like TNF-α (4,18). Despite most studies have shown that maternal smoking increases proinflammatory cytokines such as IL-1, IL-6, IL-8, and TNF-α, Chahal et al. (4) did not observe differences in umbilical cord blood biomarker concentrations, except IL-8. The inconsistencies between our findings and previous research could stem from variations in study samples, sample sizes, and limited control over confounding variables.

Correlation of Exhaled CO Levels with Umbilical Cord Blood TNF-α Levels: In this study, there was no correlation between exhaled CO and cord blood TNF-α levels (P=0.287; r=-0.118) (Figure 7). Prior studies had not directly linked the maternal exhaled CO and cord blood TNF-α levels. In the general population, several studies have shown that CO prevents TNF-α-induced vascular inflammation in endothelial cells (19). The same condition might also occur in passive smoking pregnant subjects which smoke exposure impaired the endothelial and placental vascular system (20-22).

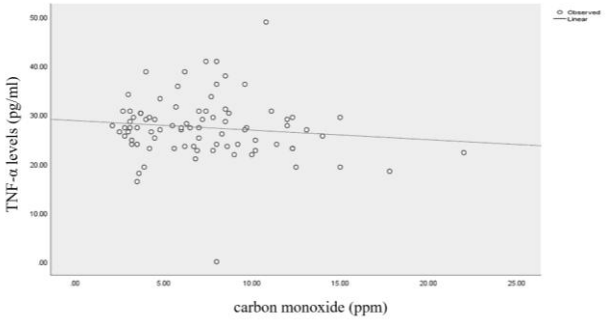

Fig. 7. Correlation between exhaled CO with TNF-α levels

Our finding highlights the importance of improving strategies to prevent exposure to cigarette smoke during early life, a critical period for growth and development. Policymakers should integrate tobacco control initiatives with maternal, newborn, and child health care services, considering the ongoing evidence of health effects. To reduce smoking exposure, strict enforcement of tobacco control laws and the promotion of smoke-free environments are essential. Additionally, it is crucial to encourage mothers to proactively avoid smoking during pregnancy. Tailored smoking cessation campaigns should also specifically target future mothers who smoke.

Study Limitations: In this study, the level of cigarette exposure for active smokers was obtained from interview, therefore, subject to interview bias. However, we managed to measure exhaled CO to minimize this bias. There was a gap of time between cord blood collection and ELISA measurement, that can compromise sample quality, even though sample storage and management were carried out according to standard.

Based on this study, newborns' birth weight was associated with smoking status, with the lowest birth weight in the active smoker group. Cotinine and CO levels were higher in the active smoker group. Meanwhile, IFN-γ and TNF-α in the groups exposed to cigarettes were lower compared to non-smoker group. Umbilical cord blood cotinine can be predictor marker since it had a strong relationship with the smoking status of pregnant subjects and negatively correlated with birth weight. In addition, in this study, exhaled CO levels and cord blood cotinine levels were not correlated with serum umbilical cord TNF-α level. Future studies must validate our findings and monitor newborns over time to determine how effectively this biomarker predicts childhood health status.

Acknowledgements: Part of this study was co-author (dr. P.H) thesis for residency program. We

received financial support from Health Professional Quality Education (HPEQ) Grant. All authors have no conflict of interst regarding this study.

References

1. World Health Organization. WHO report on the global tobacco epidemic 2021: addressing new and emerging products [Internet]. 2021 [cited 2023 Nov 10]. Available from: https://www.who.int/publications/i/item/9789240032095.
2. Ko TJ, Tsai LY, Chu LC, et al. Parental smoking during pregnancy and its association with low birth weight, small for gestational age, and preterm birth offspring: a birth cohort study. Pediatr Neonatol 2014; 55(1): 20–7.
3. Razaz N, Tomson T, Wikström AK, Cnattingius S. Association between pregnancy and perinatal outcomes among women with epilepsy. JAMA Neurol 2017; 74(8): 983–91.
4. Chahal N, McLain AC, Ghassabian A, et al. Maternal smoking and newborn cytokine and immunoglobulin levels. Nicotine Tob Res 2017; 19(7): 789–96.
5. Ji X, Yue H, Li G, Sang N. Maternal smoking-induced lung injuries in dams and offspring via inflammatory cytokines. Environ Int 2021; 156: 106618.
6. Kawashima A, Koide K, Ventura W, et al. Effects of maternal smoking on the placental expression of genes related to angiogenesis and apoptosis during the first trimester. PLoS One 2014; 9(8): 106140.
7. Niu Z, Xie C, Wen X, et al. Potential pathways by which maternal second-hand smoke exposure during pregnancy causes full-term low birth weight. Sci Rep 2016; 6: 24987.
8. Kataoka MC, Carvalheira APP, Ferrari AP, Malta MB, de Barros Leite Carvalhaes MA, de Lima Parada CMG. Smoking during pregnancy and harm reduction in birth weight: a cross-sectional study. BMC Pregnancy Childbirth 2018; 18(1): 67.
9. Roland MC, Friis CM, Godang K, Bollerslev J, Haugen G, Henriksen T. Maternal factors associated with fetal growth and birthweight are independent determinants of placental weight and exhibit differential effects by fetal sex. PLoS One 2014; 9(2): 87303.
10. Al-Enazy S, Ali S, Albekairi N, El-Tawil M, Rytting E. Placental control of drug delivery. Adv Drug Deliv Rev 2017; 116: 63–72.
11. Tatsuta N, Asato K, Anai A, et al. Japan Environment and Children's Study Group. Timing of maternal smoking cessation and newborn weight, height, and head circumference. Obstet Gynecol 2023; 141(1): 119–25.
12. Larsen S, Haavaldsen C, Bjelland EK, Dypvik J, Jukic AM, Eskild A. Placental weight and birthweight: the relations with number of daily cigarettes and smoking cessation in pregnancy. A population study. Int J Epidemiol 2018; 47(4): 1141–50.
13. Al-Shemmeri TA. Effect of smoking during pregnancy on birth weight, placental weight, and time of third stage of labor. Med J Babylon 2018; 15(4): 381–4.
14. Shahbaz S, Nandedkar M, Ara SA, Prabhu SS, Pooja DN. Evaluation of exhaled carbon monoxide levels in smokers and non-smokers-a comparative study. J Dental Sci 2023; 8(2): 000372.
15. Lawin H, Ayi Fanou L, Hinson V, et al. Exhaled carbon monoxide: a non-invasive biomarker of short-term exposure to outdoor air pollution. BMC Public Health 2017; 17(1): 320.
16. Delcroix-Gomez C, Delcroix MH, Jamee A, Gauthier T, Marquet P, Aubard Y. Fetal growth restriction, low birth weight, and preterm birth: effects of active or passive smoking evaluated by maternal expired CO at delivery, impacts of cessation at different trimesters. Tob Induc Dis 2022; 20: 70.
17. Ramadani M, Utomo B, Achadi EL, Gunardi H. Prenatal secondhand smoke exposure: correlation between nicotine in umbilical cord blood and neonatal anthropometry. Osong Public Health Res Perspect 2019; 10(4): 234–9.
18. Sabra S, Gratacós E, Gómez Roig MD. Smoking-induced changes in the maternal immune, endocrine, and metabolic pathways and their impact on fetal growth: a topical review. Fetal Diagn Ther 2017; 41(4): 241–50.
19. Choi S, Kim J, Kim JH, et al. Carbon monoxide prevents TNF-α-induced eNOS downregulation by inhibiting NF-κB-responsive miR-155-5p biogenesis. Exp Mol Med 2017; 49(11): 403.
20. Suter MA, Aagaard KM. The impact of tobacco chemicals and nicotine on placental development. Prenat Diagn 2020; 40(9): 1193–200.
21. Chełchowska M, Ambroszkiewicz J, Gajewska J, et al. Influence of active exposure to tobacco smoke on nitric oxide status of pregnant women. Int J Environ Res Public Health 2018; 15(12): 2719.
22. Argalasova L, Zitnanova I, Vondrova D, et al. Self-reported exposure to ETS (Environmental Tobacco Smoke), urinary cotinine, and oxidative stress parameters in pregnant women-the pilot study. Int J Environ Res Public Health 2019; 16(9): 1656.