

# PREVALENCE OF ACTIVE AND PASSIVE SMOKING DURING PREGNANCY: A CROSS-SECTIONAL STUDY

## Prevalência de tabagismo ativo e passivo durante a gravidez: estudo transversal

 Wallysson Costa Batista<sup>a</sup>,  Poliana Valdelice Cruz<sup>b</sup>,  
 Cristiane Baccin Bendo<sup>c</sup>,  Carolina Castro Martins<sup>c</sup>

### ABSTRACT

**Purpose:** The aim of the present study was to evaluate the prevalence of active and passive smoking during pregnancy and its association with sociodemographic indicators, as well as perinatal and postnatal outcomes in newborns. **Methods:** This cross-sectional study was comprised of 431 pairs of mothers/newborns. The study variables were: preterm birth, birth weight, oral mucosal lesions, sociodemographic indicators and smoking data. We collected data from medical records and through a self-administered questionnaire answered by mothers. A pediatric dentist examined the newborns for oral mucosal lesions. Bivariate and multivariate logistic regression models were used to evaluate the association between active and passive smoking and the other variables ( $\alpha=5\%$ ). **Results:** The prevalence of active (9.5%) and passive smoking (4.2%) during pregnancy was low. Active smoking was statistically associated with low birth weight (OR: 2.4; 95%CI:1.1-5.3), lower schooling level (OR: 0.2; 95%CI:0.1-0.5) and mothers aged  $\geq 36$  years old (OR: 4.9; 95%CI:1.2-20.0). Passive smoking was not statistically associated with the other variables. There was no association between active or passive smoking during pregnancy and premature birth and oral lesions of the newborn.

**Conclusions:** The prevalence of active and passive smoking were low. Active smoking was associated with low birth weight, maternal age and mother's schooling, suggesting a social influence of smoking behavior among a population with a lower educational level. There was no association between active and passive smoking and prematurity and oral lesions of the newborn.

**Keywords:** Pregnancy. Birth weight. Premature birth. Infant, newborn. Oral health.

### RESUMO

**Objetivo:** O objetivo do estudo foi avaliar a prevalência de tabagismo ativo e passivo durante a gestação e a associação entre indicadores sociodemográficos, bem como desfechos pré e pós-parto em recém-nascidos. **Metodologia:** Este estudo transversal foi composto por 431 pares de mães/recém-nascidos. As variáveis estudadas foram: nascimento prematuro, peso ao nascer, lesões mucosas orais, indicadores sociodemográficos e dados sobre tabagismo. Coletamos dados por meio de prontuários e por questionário auto-aplicado respondido pelas mães. Um dentista pediátrico examinou os recém-nascidos para lesões mucosas orais. A regressão logística foi utilizada para a análise bivariada e multivariada dos dados ( $\alpha=5\%$ ).

**Resultados:** Prevalência de tabagismo ativo (9,5%) e tabagismo passivo (4,2%) durante a gravidez foi baixo. O tabagismo ativo foi estatisticamente associado ao baixo peso ao nascer (OR: 2,4; 95%CI:1,1-5,3), às mães com menor escolaridade (OR: 0,2; 95%CI:0,1-0,5), e mães com idade  $\geq 36$  anos (OR: 4,9; 95%CI:1,2-20,0); enquanto o tabagismo passivo não esteve estatisticamente associado a nenhuma variável. Não houve associação entre tabagismo durante a gestação com nascimento prematuro e lesões orais de recém-nascidos. **Conclusões:** As prevalências de tabagismos passivo e ativo foram baixas. O tabagismo ativo esteve associado ao baixo peso ao nascer, à idade materna e à escolaridade da mãe, sugerindo influência social do comportamento do tabagismo entre a população com menor escolaridade. Não houve associação entre tabagismo ativo e passivo com prematuridade e lesões de mucosa oral em recém-nascido.

**Palavras-chave:** Gravidez. Peso ao nascer. Nascimento prematuro. Recém-nascido. Saúde bucal.

<sup>a</sup> Undergraduate student, Department of Pediatric Dentistry and Orthodontics, School of Dentistry, Federal University of Minas Gerais (UFMG), Belo Horizonte, MG, Brazil.

<sup>b</sup> Graduate student, Department of Pediatric Dentistry and Orthodontics, School of Dentistry, Federal University of Minas Gerais (UFMG), Belo Horizonte, MG, Brazil.

<sup>c</sup> Professor, Department of Pediatric Dentistry and Orthodontics, School of Dentistry, Federal University of Minas Gerais (UFMG), Belo Horizonte, MG, Brazil.

**Agência financiadora:** The present study was supported by the Pró-Reitoria de Pesquisa da Universidade Federal de Minas Gerais (the Dean's Office for Research of the Federal University of Minas Gerais) (PRPq-UFMG), the Coordination for the Improvement of Higher Level Education Personnel (CAPES), the National Council for Scientific and Technological Development (CNPq), and the Minas Gerais State Research Foundation (FAPEMIG), Brazil.

**Autora de correspondência:** Poliana Valdelice Cruz - E-mail: polianavacruz@gmail.com

**Número do processo:** PIBIC/CNPq 04/2018

**Data de envio:** 27/04/2020 | **Data de aceite:** 20/07/2020

## INTRODUCTION

According to the second edition of the World Health Organization (WHO) Global Report on Trends in Prevalence of Tobacco (2018)<sup>1</sup>, the global prevalence of smokers in 2015 was 20.2%, representing a reduction of 6.7% compared to 2000. It is expected that in 2025, the prevalence of smokers will decline to 17.3%. There is greater use of tobacco among males (34.1%) than females (6.4%).

Tobacco kills about 7 million people around the world, of which 6 million deaths are related to the direct use of tobacco and 890,000 are related to secondhand smoke or passive smoking<sup>2</sup>. According to the WHO (2019), passive smoking can be associated with cardiovascular diseases, serious respiratory diseases, coronary diseases and lung cancer in adults<sup>3</sup>. In young children, passive smoking can cause sudden death and respiratory diseases<sup>3,4</sup>.

Among the mechanisms involved in the association between low birth weight and smoking during pregnancy are fetal hemodynamic adaptations consistent with increased fetal arterial resistance, which can delay fetal growth and cause cardiovascular changes through the use of carbon and nicotine<sup>5</sup>. Active and passive smoking play a part in placenta vascularization, which alters blood flow, reducing gas and nutritional exchanges between the mother and the fetus. These decreased gas and nutritional exchanges can increase the risk of fetal malnutrition, and consequently lead to low birth weight and preterm birth among other maternal and fetal complications<sup>6</sup>. Therefore, low birth weight and preterm birth can be directly related to tobacco exposure. These factors can influence the development of oral structures<sup>7,8</sup>, as demonstrated by a previous study that found that oral mucosal lesions in newborns may be associated with preterm birth, low birth weight and socioeconomic level<sup>9</sup>. This hypothesis reinforces that maternal habits during pregnancy can directly impact the newborn's oral health, as passive pregnant smokers may have an increased risk of low birth weight, delayed fetal growth, premature birth and periodontitis<sup>4,10-12</sup>.

Thus, the aim of the present study was to evaluate the prevalence of active and passive smoking during pregnancy and its association with sociodemographic indicators, as well as perinatal and postnatal outcomes in newborns, such as premature birth, birth weight and oral mucosal lesions.

## MATERIALS AND METHODS

This cross-sectional study was comprised of 431 pairs of mothers and their newborns aged up to 27 days, born at the University Hospital of the Federal University of Minas Gerais, Belo Horizonte, Brazil. Data collection was carried out from August 2016 to April 2017. The present study was approved by the Human Research Ethics Committee of the Federal University of Minas Gerais (CAAE n<sup>o</sup> 57295316.3.0000.5149). All the mothers enrolled in the study volunteered to participate by signing an informed consent form after the goals of the study were explained.

The eligibility criterion included all live newborns of both sexes and their mothers present at the University Hospital during the data collection period. We excluded newborns with craniofacial anomalies, cleft lips/palates, and those with anemic conditions or heart disease.

The mothers answered a self-applied questionnaire on active and passive smoking (dependent variables). We collected data related to the independent variables: preterm birth, birth weight, oral mucosal lesions and sociodemographic indicators. Socioeconomic class was evaluated through the Brazilian Economic Classification Criteria<sup>13</sup>, and was classified as A through E according to the personal belongings and educational level of the respondent. We grouped socioeconomic class into two categories: high (classes A, B1 and B2) and low (classes C1, C2, D and E).

Information regarding preterm birth and birth weight was collected through medical records. A calibrated and trained pediatric dentist examined the oral cavities of the newborns to evaluate oral mucosal lesions. Epstein pearls, Bohn's nodules, dental lamina cysts, mucocele,

ranula, natal and neonatal teeth were evaluated. For this analysis, all the oral mucosal lesions were grouped into one variable (oral mucosal lesions: yes/no).

A calibration exercise was performed by a gold standard specialist in Pediatric Dentistry. We used photographs to analyze the lesions of the oral mucosa of newborns. The photographs were re-evaluated after a one week interval. Cohen's Kappa coefficient was used to test the intra-examiner (0.90) and inter-examiner agreement (0.81). As the Kappa values were excellent (between 0.81-1.00), the examiner was considered capable of conducting the main study<sup>14</sup>.

To obtain the sample size, a prevalence of oral lesions in children aged between 0 and 3 years of 56.4% was considered, with a confidence interval of 95.0% and an error of 5.0%<sup>15</sup>. A minimum sample size of 378 was obtained, and a sample of 53 samples was added to compensate for possible losses, representing an additional 14% in our final sample.

The SPSS software package was used to enter the data and to run a descriptive analysis of the variables. We ran a bivariate and multivariate logistic regression analysis to evaluate the association between active and passive smoking and the other variables ( $\alpha=5\%$ ). The quality of the models was tested by the Hosmer-Lemeshow test. The bivariate unadjusted model included all the variables. The multivariable model was adjusted to include variables with  $p<0.20$  in the bivariate model.

## RESULTS

The final sample size was 431 newborns (Figure 1).

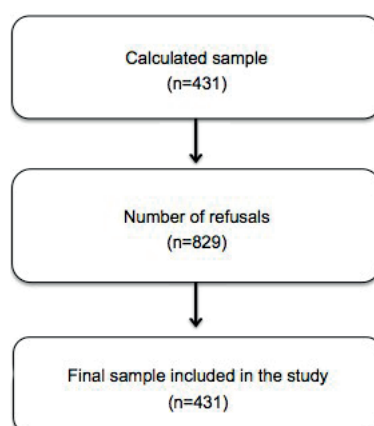


Figure 1: Flow diagram of the study design.

Table 1 shows the population characteristics. Two hundred thirty three newborns were boys (54.1%) and 198 were girls (45.9%). The prevalence of active smoking was 9.5% (n=41) and the prevalence of passive smoking was 4.2% (n=18). The prevalence of oral mucosal lesions was 61.3% (n=264).

Table 1: Demographic Characteristics and Sample Distribution (SD).

Variables	Mean $\pm$ (SD)	Minimum-maximum
Mother's age (years)	27.2 $\pm$ 7.1	15-59
Age of newborns at time of examination (days)	2.42 $\pm$ 3.49	0-27
Gestational age at birth (weeks)	38.24 $\pm$ 1.89	33-42
Birth weight (grams)	3.063 $\pm$ 524.55	1.690-4.700

Table 2 shows the bivariate (Model 1) and multivariate (Models 2) analysis, comparing active smokers and non-active smokers. Low birth weight (OR: 2.4; 95%CI:1.1-5.3), mothers aged  $\geq 36$  years old (OR: 4.9; 95%CI:1.2-20.0) and maternal education  $\geq 12$  to 18 years of study (OR: 0.2; 95%CI:0.1-0.5) were associated with active smoking.

Table 2: Bivariate and multivariate analysis of association between active smoking during pregnancy and independent variables.

Variables	Active Smoking		Bivariate model Unadjusted		Multivariable model Adjusted	
	No n (%)	Yes n (%)	OR (95% CI)	p-value	OR (95% CI)	p-value
<b>Preterm birth*</b>						
No	314 (83.1)	31 (77.5)	1	0.380	-	-
Yes	64 (16.9)	9 (22.5)	1.4 (0.6-3.1)	-	-	-
<b>Birth weight*</b>						
Normal weight	326 (84.9)	30 (73.2)	1	0.057	1	<b>0.025</b>
Low weight	58 (15.1)	11 (26.8)	2.0 (0.9-4.3)	-	2.4 (1.1-5.3)	-
<b>Oral mucosal lesions</b>						
No	155 (39.7)	16 (39.0)	1	0.299	-	-
Yes	235 (60.3)	25 (61.0)	0.7 (0.3-1.3)	-	-	-
<b>Maternal age*</b>						
up to 19 years	60 (15.5)	3 (7.3)	1	0.065	1	<b>0.023</b>
20 to 35 years	276 (71.5)	29 (70.7)	2.1 (0.6-7.1)	-	3.2 (0.9-11.4)	-
$\geq 36$ years	50 (13.0)	9 (22.0)	3.6 (0.9-14.0)	-	4.9 (1.2-20.0)	-
<b>Paternal schooling*</b>						
9 to 12 years of schooling	195 (51.7)	21 (60.0)	1	0.350	-	-
$\geq 12$ to 18 years of schooling	182 (48.3)	14 (40.0)	0.7 (0.3-1.4)	-	-	-
<b>Maternal schooling</b>						
9 to 12 years of schooling	144 (36.9)	25 (61.0)	1	0.004	1	<b>0.001</b>
$\geq 12$ to 18 years of schooling	246 (63.1)	16 (39.0)	0.3 (0.1-0.7)	-	0.2 (0.1-0.5)	-
<b>Socioeconomic class*</b>						
High <sup>†</sup>	96 (24.7)	8 (19.5)	1	0.464	-	-
Low <sup>†</sup>	293 (75.3)	33 (80.5)	1.3 (0.6-3.0)	-	-	-

Logistic regression model with robust variance for multivariate analyses.

Multivariable model: all variables with  $p < 0.20$  in the bivariate analyzes were included in this model. The Hosmer and Lemeshow test was performed ( $p = 0.692$ ).

\* Total number is lower than 431 because of incomplete questionnaires or medical records with blank responses.

† Categorization according to the Brazil economic classification criterion of ABEP (Brazilian Economic Classification Criteria).

Table 3 shows the bivariate (Model 1) and multivariate (Models 2) analysis comparing passive smoking and non-passive smoking. No statistically significant associations were found between passive smoking and the studied variables. The presence of oral mucosal lesions was not statistically significantly associated with active or passive smoking (Tables 2 and 3).

Table 3: Bivariate and multivariate analysis of association between passive smoking during pregnancy and independent variables.

Variables	Passive smoking		Bivariate Model Unadjusted		Multivariable Model Adjusted	
	No n (%)	Yes n (%)	OR (95% CI)	p-value	OR (95% CI)	p-value
<b>Preterm birth*</b>						
No	330 (82.5)	15 (83.3)	1	0.943	-	-
Yes	70 (17.5)	3 (16.7)	0.9 (0.2-3.3)	-	-	-
<b>Birth weight*</b>						
Normal weight	340 (83.5)	16 (88.9)	1	0.550	-	-
Low weight	67 (16.5)	2 (11.1)	0.6 (0.1-2.8)	-	-	-
<b>Oral mucosal lesions</b>						
No	163 (37.8)	4 (0.9)	1	0.541	-	-
Yes	250 (58)	14 (3.2)	1.3 (0.5-3.5)	-	-	-
<b>Maternal age*</b>						
Up to 19 years	57 (13.9)	5 (27.8)	1	0.190	1	0.291
20 to 35 years	295 (72.1)	10 (55.6)	0.3 (0.06-1.7)	-	0.4 (0.07-2.1)	-
Over 36 years	57 (13.9)	2 (11.1)	0.3 (0.1-0.9)	-	0.3 (0.1-1.009)	-
<b>Paternal schooling*</b>						
9 to 12 years of schooling	202 (51.3)	14 (77.8)	1	0.037	1	0.088
≥12 to 18 years of schooling	192 (48.7)	4 (22.2)	0.3 (0.09-0.9)	-	0.3 (0.1-1.1)	-
<b>Maternal schooling</b>						
9 to 12 years of schooling	161 (39.0)	8 (44.4)	1	0.643	-	-
≥12 to 18 years of schooling	252 (61.0)	10 (55.6)	0.7 (0.3-2.0)	-	-	-
<b>Socioeconomic class*</b>						
High <sup>†</sup>	102 (24.8)	2 (11.1)	1	0.202	1	0.384
Low <sup>†</sup>	310 (75.2)	16 (88.9)	2.6 (0.5-11.6)	-	1.9 (0.4-8.9)	-

Logistic regression model with robust variance for multivariate analyses.

Multivariable model: all variables with  $p < 0.20$  in the bivariate analyzes were included in this model.

The Hosmer and Lemeshow test was performed ( $p = 0.971$ ).

\* Total number is lower than 431 because of incomplete questionnaires or medical records with blank responses.

<sup>†</sup> Categorization according to the Brazil economic classification criterion of ABEP (Brazilian Economic Classification Criteria).

## DISCUSSION

Mothers that smoked had a lower level of schooling, were aged  $\geq 36$  years old and were more likely to have children with a low birth weight. Oral mucosal lesions were not associated with active or passive smoking. Passive smoking was not associated with any variable.

The results of the present study agree with those in literature, in which women with lower levels of schooling, families with more members and greater poverty increase the probability of smoking<sup>16</sup>. Indeed it seems that smoking is more common among families with lower levels of schooling, suggesting that social inequalities are related to smoking<sup>17,18</sup>. We did not find an association between socioeconomic class and active and passive smoking, although the WHO estimates that about 80% of deaths caused by smoking occur in developing countries<sup>19</sup>. Our sample was from a public hospital and the participants were mainly from a low social class, which might have contributed to the absence of an association. It may also be that pregnant women with low levels of schooling do not know about or neglect the harmful effects of active smoking.

The prevalence of active smoking was associated with mothers aged  $\geq 36$  years old. The results are in accordance with the WHO report which described a higher prevalence of smoking among older people (between 45 and 54 years)<sup>1</sup>. It can be hypothesized that younger mothers have been influenced about the dissemination of information about the harm caused by smoking.

Active and passive smoking during pregnancy can be associated with preterm birth and low birth weight<sup>20-24</sup>. In literature, a dose-response effect has been suggested between passive smoking and premature birth and low birth weight<sup>16,25</sup>. Nicotine and carbon monoxide are associated with hemodynamic adaptations during pregnancy. These adaptations can lead to an increase in fetal arterial resistance, which in turn, reduces both oxygen and the nutritional exchange between the pregnant woman and the fetus, through the placenta. These adaptations and interferences may be responsible for the reduction in birth weight, prematurity and fetal malnutrition, among other important maternal-fetal consequences<sup>5,6</sup>. However, the present study only found an association between active smoking and low birth weight, which we suggest may be due to the low prevalence of passive smokers in the sample.

Oral mucosal lesions are common in newborns and tend to disappear over time without the need for treatment<sup>26</sup>. We found a high prevalence of oral mucosal lesions, in accordance with the prevalences reported in literature, which can vary between 21%<sup>27</sup> and 91.2%<sup>28</sup>. However, we did not find an association between oral mucosal lesions and smoking habits.

Reporting bias is a limitation of the present study. Active and passive smoking can be underreported by mothers due to memory<sup>19</sup> or information bias. Mothers may consciously omit information as they know that smoking is a harmful habit. Also, mothers may be confused regarding the meaning of active or passive smoking<sup>16,20,21</sup>. The strength of the study is the sample size and the early age at which newborns were examined. Younger newborns usually have more mucosal lesions, especially those aged up to one year, in comparison with to older babies<sup>13</sup>.

The results found of the present study should be evaluated with caution, as the sample is from a single hospital. Thus, applicability to the general population is limited due to its low external validity.

While not associated with oral mucosal lesions in newborns, active and passive smoking can cause other comorbidities. The association between smoking and gestational age and birth weight needs to be further investigated in future studies. There is a need for public policies to prevent smoking, focused mainly on the most vulnerable families. Passive smoking can go unrecognized by mothers and public policies should focus on the dangers of passive smoking in pregnant women. Moreover, education can be an important aid for preventing smoking in vulnerable populations, not only through public policies but by increasing access to education and schools. Future research is needed to monitor the prevalence of smoking in pregnant women and the long term consequences for their children.



## CONCLUSION

Passive smoking during pregnancy was not associated with perinatal and postnatal outcomes in relation to newborns, while active smoking was associated with lower maternal education, maternal age and low birth weight. Oral mucosal lesions were not associated with active and passive smoking.

## OTHER INFORMATION

The present study was supported by the Pró-Reitoria de Pesquisa da Universidade Federal de Minas Gerais (the Dean's Office for Research of the Federal University of Minas Gerais) (PRPq-UFGM), the Coordination for the Improvement of Higher Level Education Personnel (CAPES), the National Council for Scientific and Technological Development (CNPq), and the Minas Gerais State Research Foundation (FAPEMIG, process #APQ-00323-17), Brazil.

## REFERENCES

1. World Health Organization. WHO global report on trends in prevalence of tobacco smoking 2000–2025, second edition. Geneva: WHO; 2018. [cited 2020 Jun 9]. Available from: <https://www.who.int/tobacco/publications/surveillance/trends-tobacco-smoking-second-edition/en/>.
2. Gakidou E, Afshin A, Abajobir AA, Abate KH, Abbafati C, Abbas KM, et al. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet*. 2017 Sept;390(10100):1345–422.
3. World Health Organization. WHO report on the global tobacco epidemic, 2019. Geneva: WHO; 2019. [cited 2020 Jun 8]. Available from: [https://www.who.int/tobacco/global\\_report/en/](https://www.who.int/tobacco/global_report/en/).
4. Coelho SA, Rocha AS, Jong LC. Consequences of passive smoking in children. *Cienc Cuid Health*. 2012 Mar;11(2):294–301.
5. Geelhoed JJM, El Marroun H, Verburg BO, Van Osch-Gevers L, Hofman A, Huizink AC, et al. Maternal smoking during pregnancy, fetal arterial resistance adaptations and cardiovascular function in childhood. *BJOG*. 2011 Mar;118(6):755–62.
6. Pintican D, Pojenar AA, Strilciuc S, Miha D. Effects of maternal smoking on human placental vascularization: a systematic review. *Taiwan J Obstet Gynecol*. 2019 Jul;58(4):454–9.
7. Paulsson L, Bondemark L, Söderfeldt B. A systematic review of the consequences of premature birth on palatal morphology, dental occlusion, tooth-crown dimensions, and tooth maturity and eruption. *The Angle Orthod*. 2004 Apr;74(2):269–79.
8. Ebrahim E, Paulsson L. The impact of premature birth on the permanent tooth size of incisors and first molars. *Eur J Orthod*. 2017 Nov;39(6):622–27.
9. Cruz PV, Bendo CB, Occhi-Alexandre IG, Paiva SM, Pordeus IA, Martins CC. Prevalence of oral inclusion cysts in a Brazilian neonatal population. *J Dent Child*. 2020 May-Jun;87(2):3–10.
10. Campos M, Bravo E, Eugén J. Respiratory dysfunctions induced by prenatal nicotine exposure. *Clin Exp Pharmacol*. 2009 May;36(12):1205–17.
11. El-Mohandes AAE, Kiely M, Blake SM, Gantz MG, El-Khorazaty MN. An intervention to reduce environmental tobacco smoke exposure improves pregnancy outcomes. *Pediatrics*. 2010 Apr;125(4):721–8.
12. Prabhu N, Smith N, Campbell D, Craig LC, Seaton A, Helms PJ, et al. First trimester maternal tobacco smoking habits and fetal growth. *Thorax*. 2010 Mar;65(3):235–40.
13. Brazilian Market Research Association. Brazilian Association of Research Companies Brazil 2015 criterion and update of class distribution for 2016. ABEP, 2016. [cited 2020 Jun 8]. Available from: <http://www.abep.org/criterio-brasil>.
14. Altman DG. *Practical statistics for medical research*. London: Chapman and Hall; 1990.
15. Padovani MCL, Santos MTBR, Sant'anna GR de, Guará RO. Prevalence of oral manifestations in soft tissues during early childhood in Brazilian children. *Braz Oral Res*. 2014 Aug;28(1):1–7.
16. Goel P, Radocha A, Singh I, Aggarwal A, Dua D. Effects of passive smoking on outcome in pregnancy. *J Postgrad Med*. 2004 Jan-Mar;50(1):12–6.

17. Vitoria PD, Nunes C, Precioso J. Parents' educational level and second-hand tobacco smoke exposure at home in a sample of Portuguese children. *PJP*. 2017 Jul-Aug;23(4):221-4.
18. Orton S, Jones LL, Cooper S, Lewis S, Coleman T. Predictors of children's secondhand smoke exposure at home: a systematic review and narrative synthesis of the evidence. *PLoS ONE*. 2014 Nov;9(11):e112690.
19. Ward C, Lewis S, Coleman T. Prevalence of maternal smoking and environmental tobacco smoke exposure during pregnancy and impact on birth weight: retrospective study using millennium cohort. *BMC Public Health*. 2007 May;7(1):81.
20. United States Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General; 2006. [cited 2019 Dec 11]. Available from: <http://www.prevention.ch/sg2006s.pdf>.
21. Abu-Baker NN, Haddad L, Savage C. The influence of secondhand smoke exposure on birth outcomes in Jordan. *Int J Environ Res Public Health*. 2010 Feb;7(2):616-34.
22. United Nations Children's Fund, World Health Organization. Low birth weight: country, regional and global estimates. UNICEF: New York; 2004. [cited 2019 Nov 12]. Available from: [https://www.unicef.org/publications/index\\_24840.html](https://www.unicef.org/publications/index_24840.html).
23. Crane J, Keough M, Murphy P, Burrage L, Hutchens D. Effects of environmental tobacco smoke on perinatal outcomes: a retrospective cohort study. *Int J Obstet Gynaecol*. 2011 Mar;118(7):865-71.
24. Pogodina C, Huber L, Racine E, Platonova E. Smoke-free homes for smoke-free babies: the role of residential environmental tobacco smoke on low birth weight. *J Commun Health*. 2009 Jun;34(5):376-82.
25. Kharrazi M, De Lorenze GN, Kaufman FL, Eskenazi B, Bernert JT, Graham S, et al. Environmental tobacco smoke and pregnancy outcome. *Epidemiology* 2004;15(6):660-70.
26. Friend GW, Harris EF, Mincer HH, Fong TL, Carruth KR. Oral anomalies in the neonate, by race and gender, in an urban setting. *Pediatr Dent*. 1990 May-Jun;12(3):157-61.
27. Baldani MH, Lopes CMDL, Scheidt WA. Prevalence of oral alterations in infants seen at the public pediatric dental clinics from Ponta Grossa - PR, Brazil. *Pesqui Odontol Bras*. 2001 Nov-Dec;15(4):302-7.
28. Perez-Aguirre B, Soto-Barreras U, Loyola-Rodriguez JP, Reyes-Macias JF, Santos-Diaz MA, Loyola-Leyva A, et al. Oral findings and its association with prenatal and perinatal factors in newborns. *Korean J Pediatr*. 2018 Sep;61(9):279-84.