Medical Journal of the
Islamic Republic of Iran

Volume 12
Number 1
Spring 1377
May 1998

EFFECT OF PASSIVE SMOKING DURING PREGNANCY ON WEIGHT, LENGTH AND CRANIAL CIRCUMFERENCE AT BIRTH IN KERMAN, IRAN

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ABSTRACT

Passive smoking (PS), a well-known health risk, is the major source of indoor pollution. There is some inconsistent evidence that PS during pregnancy may increase the risk of low birthweight. The aim of the present study was to determine the effects of PS exposure during pregnancy on weight, length and cranial circumference at birth of babies born to women who have described themselves as nonsmokers, in Kerman, Iran. A random sample of 702 admitted women aged 11 to 50 years [mean (standard deviation) 26.5 (6.1)] who delivered a live full-term singleton baby without apparent malformation during the six consecutive months from June to December 1994 were interviewed on the second day post-partum and asked about smoking in all household members. They comprised about 36.4% of total deliveries in Bahonar Kerman Medical School Hospital during this period. All women were nonsmokers, 278 (39.6%) were passive smokers while 424 (60.4%) were not exposed to tobacco smoke. Potential confounders, including fetal gender, maternal age, parity, weight gain, complications during pregnancy, maternal education, birth interval and gestation were adjusted for by multiple linear regression analysis. Infants born to passive smokers were on the average 22 gr. lighter than those born to nonsmokers, albeit this difference was not statistically significant (p = 0.56) [95% confidence interval (CI): -51, 95.7]. A mean reduction of 0.04 cm [95% CI: -0.19, 0.27] in birth length and 0.05 cm [95%CI: -0.12, 0.22) in cranial circumference was found. In multiple linear regression model, exposure to PS during pregnancy did not show any effect on weight, length and cranial circumference at birth after adjusting for confounding variables.

Keywords: Low birthweight, passive, sidestream, second-hand, environmental, smoking, epidemiology, Iran.

MJIRI, Vol. 12, No. 1, 31-36, 1998.

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INTRODUCTION

Spacious evidence has demonstrated a significant relation between maternal cigarette smoking during pregnancy and delivering a low birthweight infant.[1-3] The possible effect of passive exposure to tobacco smoke on birthweight has stimulated several epidemiological studies,[2-14] but there is inconsistency between different estimates of the magnitude of the risk. Some studies showed a significant association of low birthweight with passive smoking (PS)[5,8,9,11-14] while others showed no effect.[6,7,15-17] The explanations for this discrepancy could include difficulties in measuring the degree of passive smoke exposure and inadequate control of confounders.

The high rate of exposure to passive smoke among the general population in numerous countries, particularly in developing countries like Iran, and recognition of the public health hazard of active smoking is a constant stimulus for new research. Although there has been substantial research in developed countries, relatively little has been done in developing countries where the problem is much greater. The risk of PS is a serious problem in Iran and pregnant women are highly likely to be exposed to PS at home, work place or other areas.

This study, using household exposure to cigarette smoke as an estimate of passive smoking, attempted to clarify further the effect of passive smoking in household members on weight, length and cranial circumference at birth in Kerman, where such studies have not been carried out.

SUBJECTS AND METHODS

Using a pre-set structured questionnaire, a random sample of consecutive women were interviewed in hospital by a trained interviewer, on day one after delivery if their babies met the following criteria: a live full-term singleton baby with birthweight≥2000 gr, gestational age≥37 weeks, and no evidence of serious congenital defect or underlying illness. The study group consisted of 702 singleton live births occuring in women who have received intrapartum care in the Department of Obstetrics and Gynecology of the Bahonar Medical School Hospital, affiliated to Kerman University of Medical Sciences and Health Services, Iran, between June and December 1994 (estimated as 36.4% of total deliveries in Bahonar Hospital during the period). The questionnaire included questions on demographic information, smoking history, and the indoor cigarette smoking habit of family members, exposure to passive smoking at work, average length of passive smoke exposure during pregnancy per day at home and work place, previous pregnancy history, complications during pregnancy, as well as level of education and occupation (housewife or employee) of the mother. Each mother was asked if she smoked or used tobacco in any other form, and if there was anyone in the household who smoked tobacco. Obstetric and medical details of each mother were recorded from medical records and antenatal cards as was the newborns' birthweight, height, and head circumference which was recorded in the delivery room within 30 minutes after birth. The weighing scale used was a Seca lever type (made in Germany) which could read to the nearest 10 gr. It was calibrated at the beginning of each working day. The best estimate of gestation obtained from menstrual history or sonographic examination was recorded. In this study, nonsmokers were considered patients who never smoked any kind of tobacco regularly during their lifetime and passive smoking was defined as being exposed to someone else's cigarette smoke, either at home or at work, during pregnancy. Smoking referred only to cigarette smoke; the use of other tobacco products, such as pipe, cigar, cigarillo and snuff, was not considered since they are practically uncommon among Iranians.

Table I. The distribution of sources of passive smoke in Kerman, Iran, 1994.

Source of passive smoke	No.	%	95% CI @
Husband only	267	38	34.4-41.6
Both husband and others	5	0.7	0.2-1.7
Others only	6	0.9	0.4-2.0
Total	278	39.6	35.4-42.6

@ CI= Confidence interval.

Statistical analysis

Means and standard errors of means (SE) are presented for describing variables with continuous distribution. Mean and proportion of characteristics of passive smokers and non-passive smokers were compared using t-tests and chi-square tests. The variables reaching a P level of <0.05, and sharing a trend, even if not significant, among the two smoking categories, were further studied using stepwise multiple regression analysis, in order to adjust for the effect(s) of potential confounding variables. Covariates not found to be significant at the 0.05 level were removed from the regression model by using a stepwise elimination technique. The analysis was done on a personal computer using SPSS/PC+ version 3 and Confidence Interval Analysis Software.[18-19] All testing for statistical significance were two tailed, and performed at $p<0.05$.

RESULTS

Between June and December 1994, 702 women were interviewed. None of them used tobacco in any form regularly. Prevalence and distribution of sources of PS are given in Table I. The PS exposure was mainly from the

Table II. Group means and proportion for selected variables between non-smoking women who were and were not exposed to passive cigarette smoke.

Characteristic	Smoking exposure		Difference
	Passive (N= 278)	None (N= 424)	(95% CI@)
Continuous variables	**Mean (SE)**	**Mean (SE)**	
Birthweight (gr.)	3124 (27.8)	3146 (24.3)	22 (-51.7, 95.7)
Length at birth (cm)	49.88 (0.08)	49.92(0.08)	0.04 (-0.19, 0.27)
Cranial circumference (cm)	34.42 (0.06)	34.47 (0.06)	0.05 (-0.12, 0.22)
Gestational age (week)	38.81 (0.06)	38.81 (0.05)	0 (-0.15, 0.15)
Parity (no. of previous deliveries)	2.26 (0.14)	1.77 (0.13)	-0.49 (-0.88, -0.10)**
Maternal age (years)	26.7 (0.37)	26.4 (0.30)	-0.3 (-1.22, 0.62)
Husband's no. of cigarette/day	14.2 (0.44)	-	-
Hours of exposure/day	2.6 (0.08)	-	-
No. of cigarette exposure/day	6.2 (0.25)	-	-
Categorical variables	**No (%)**	**No (%)**	
Infant gender			
Male	157 (56.5)	226 (53.3)	-3.2 (-1.7, 4.4)
Female	121 (43.5)	198 (46.7)	-
Maternal education			
Illiteracy	46 (16.5)	57 (13.4)	-3.1 (-8.6, 2.3)
Elementary school	180 (64.7)	212 (50.0)	-14.7 (-22.1, -7.4)**
Middle school	33 (11.8)	98 (23.1)	11.2 (5.7, 16.8)**
≥ College	19 (6.8)	67 (15.8)	9.0 (4.4, 13.5)**
Maternal occupation			
Housewife	247 (88.8)	354 (83.5)	-5.4 (-10.5, -0.2)
Employee	31 (11.2)	70 (16.5)	-
Birthweight			
< 2500 gr.	25 (9.0)	51 (12.0)	3.0 (-1.5, 7.6)
≥ 2500 gr.	253 (91.0)	373 (88.0)	

*$p<0.05$, ** $p<0.01$.

@ The difference in the mean and proportion of the variables between passive and non-passive smokers.

husband. Only about 0.9% of women whose husbands never smoked were exposed at home and 272 (38.7%) of the husbands smoked during their wives' pregnancy. Passive smoking is quite common for pregnant women and 39.6% of the study subjects experienced passive smoke exposure. Almost all of the women in the study did not work outside the home (only 3 women (0.4%) worked). So the figures available for analysis of PS at work were too small for valid statistical analysis.

Table II demonstrates the results of the crude comparison among passive and non-passive smoke exposure and a set of parental and neonatal parameters. Significant differences were observed for parity and maternal educational status. The crude difference between passive and non-passive smokers was 22 gr. in mean weight, 0.03 cm in mean length, and 0.05 cm in mean cranial circumference at birth. None of these differences was significant. The number of husband's cigarettes smoked varied between 1 and 40 [mean (SE) 14.2 (0.44)] per day. The mean (SE) duration of PS among the women reporting exposure to passive smoke was 2.6 (0.08) hours (95% CI: 2.43, 2.77) per day. The two groups of infants were similar with respect to the mother's age, occupation, gestation and gender. The crude relative prevalence of delivering a low birthweight (less than 2500 gr.) baby among PS women was 0.75 (95% CI: 0.42, 1.23) compared with unexposed women, based on 76 babies less than 2500 gr. and 626 babies more than or equal to 2500 gr.

Table III shows the potential confounders or effect modifiers considered in the regression models and the linear correlation coefficient of each factor with the dependent variable's weight, length and cranial circumference at birth.

Table IV shows the regression model for weight, length and cranial circumference at birth. Multivariate analysis did not show any effect of PS on weight, length or cranial circumference at birth. The difference observed when performing bivariate analysis seems to be fully explained by other predictive factors enclosed in the model.

Table III. Potential confounders considered in regression analysis and their correlation with dependent variables (Pearson correlation coefficient).

Confounder	Birthweight	Birth length	Cranial Circumference
Birthweight	1.000	0.5452***	0.6449***
Birth length	0.5452***	1.000	0.5269***
Cranial circumference	0.6449***	0.5269***	1.000
Infant gender	-0.0766*	-0.0369	-0.0742*
Gestational age	0.1677***	0.1507***	0.1102**
Parity	0.0933*	0.0589	0.1226***
Mother's age	0.1272***	0.1041**	0.1557***
Weight gain	0.2881***	0.2083***	0.2002***
Complications during pregnancy	-0.1215***	-0.0596	-0.0451
Mother's education	0.0481*	0.0722	0.0668
Birth interval	0.0941*	0.0577	0.1122**

* $p<0.05$, ** $p<0.01$, *** $p<0.001$.

DISCUSSION

In this study the husband was usually the single most important source of PS and household exposure to cigarette smoke, from husband and other family member, was taken as an estimate of PS. The findings of this study suggest a rather small effect of maternal PS on birthweight, and both bivariate and multivariate analyses failed to reach statistical significance. The statistical power to detect small significant differences in our study was limited. In the present study, information on past exposure to PS was obtained by subject recall through interview. In an attempt to limit information bias, we used structured questionnaires and standardized interview techniques, which are often helpful in minimizing both recall and interview bias. Several possible confounders were considered in this study as suggested by several authors.[5,7,8,10] However, it is difficult to account for the results in terms of selection or recall bias. Study subjects were pregnant women who received intrapartum care from obstetricians and covered about one-third of all deliveries in Bahonar hospital (the largest maternity hospital in Kerman) during the study period. Although this may not be enough to warrant a generality from the study results, we do not contemplate that the results can be explained by selection bias. Since all of the women were interviewed after delivery, they may have been influenced in their responses by the delivery outcome. However, we would expect that where low birthweight had occurred, women would tend to overstate rather than understate their exposure to PS. This would cause an apparent increase in the effect of PS exposure and so could not explain the rather small effect observed in this study.

The crude effect of PS exposure on reduction of weight, length and head circumference at birth was not statistically significant and the effect was totally removed by adjustment for confounding factors. Multiple regression analysis of birthweight showed a significant effect on cranial circumference, length at birth, and complications during pregnancy and gestation. Although PS showed no effect on these factors in the crude associations, regression analyses were performed to determine whether such an association was obscured by confounding factors. However, no effects of PS on these pregnancy outcomes were found.

The present study excluded babies with a gestational age of less than 37 weeks, because it was difficult to be certain whether the lower birthweight of these babies was due to growth retardation, shorter gestation period, or because of pregnancy complications which may be related to premature birth.

The results of the present study suggest that maternal PS exposure has little, if any, effect on fetal growth. Several studies have investigated the effect of passive smoke exposure in pregnant women. Results are contrasting, ranging from the considerable effect detected by Rubin et al.[5] who reported a mean reduction of 120 gr. in birthweight per packet of cigarettes smoked daily by the father to the lack of effect reported by several investigators[6,15-17] and even the a passive smoking effect reported by MacArthur and Knox.[7] Previous studies on PS and birthweight have found relative risks ranging from 0.9 to 3.0.[2-4] The studies that suggest an association between PS and lower birthweight have been done in populations where smoking among women is common. As suggested by others, it may be possible that some women in these studies could have been wrongly classified as passive smokers while they were in fact active smokers. Thus, it is difficult to extricate the effects of maternal and paternal smoking when both are highly correlated. In Kerman, few young mothers smoked. The main argument used to discredit positive studies refer to the presence of serious biases in the study design, such as poor assessment of passive smoke exposure and lack of control for confounding variables. The criticisms concerning

Table IV. Multiple linear regression of effects of passive smoking exposure and other predictive factors on mean weight, length and cranial circumference at birth.

Variables	Coefficient (SE)	t-test
Birthweight (gr.)		
Cranial circumference (cm)	202.277 (13.52)	15.0***
Length at birth (cm)	83.926 (10.52)	8.0***
Weight gain (kg)	111.126 (23.43)	4.7***
Complication during pregnancy	-9.612 (3.65)	2.6**
Gestational age (weeks)	26.323 (13.08)	2.0*
Passive smoking (2 categories)	0.009 (0.003)	0.07
Passive smoke exposure (hours)	0.012 (0.017)	0.44
$f = 139.4$, $p<0.001$, r^2: 0.501		
Length at birth (cm)		
Cranial circumference (cm)	0.388 (0.05)	7.5***
Birthweight (kg)	0.001 (0.0001)	8.8***
Passive smoking (2 categories)	-0.002 (-0.003)	0.07
Passive smoke exposure (hours)	-0.017 (-0.02)	-0.55
$f=187.5$, $p<0.001$, r^2:350		
Cranial circumference (cm)		
Length at birth (cm)	0.190 (0.026)	7.4***
Birthweight (kg)	0.001 (0.00008)	15.1***
Mother's age (years)	0.012 (0.005)	2.4*
Passive smoking (2 categories)	0.008 (0.011)	0.29
Passive smoke exposure (hours)	-0.181(-0.25)	-0.65
$f = 200.8$, $p<0.001$, r^2: 0.464		

*$p<0.05$, ** $p<0.01$, ***$p<0.001$.

exposure assessment are based on the poor reliability of the quantification of PS through the father's smoking habits. On the other hand, the actual amount of passive exposure may be related not only to exposure time, but also to the concentration of residual smoke in the room, which is dependent on ventilation, room size, and smoking behaviour.

For exposure to PS at work, because few of the women worked outside the home, the numbers of subjects were too small for valid statistical analysis.

Although this study, as well as several other studies,[6,7,15-17] did not show a significant effect of passive smoke exposure on fetal growth, passive exposure to cigarette smoke is a public health hazard and may increase the risk of lung cancer and sudden infant death syndrome, as well as cause a higher frequency of otitis and respiratory ailments. Therefore, passive smoking is an important public health issue, particularly in developing countries like Iran where the prevalence of smoking among young adults is high. The overall hazard is sufficient to justify measures to restrict smoking in public places and work places, and to discourage people from smoking in their homes.

ACKNOWLEDGEMENTS

We wish to thank Dr. Ali Sadeghi-Hassanabadi for his valuable comments on the manuscript. This study was supported by a grant from the Research Center of Kerman University of Medical Sciences and Health Services.

REFERENCES

1. Johnson C: Cigarette smoking and the outcome of human pregnancy: a status report on the consequences. Clin Toxicol 18: 189-209, 1981.
2. Albel EL: Smoking during pregnancy: a review of effects on growth and development of offspring. Hum Biol 52: 593-

625, 1980.

3. US Department of Health, Education, and Welfare: Smoking and Health: A Report of the Surgeon General. Washington DC; US PHS, 1979. (DHEW publication no. (PHS) 79-500669).
4. Javis MJ, Russell MAH, Feyerabend C: Absorption of nicotine and carbon monoxide from passive smoking under natural conditions of exposure. Thorax 38: 829-33, 1983.
5. Rubin HD, Krasilinikoff PA, Leventhal JM, Berget A: Effect of passive smoking on birth weight. Lancet ii: 415-7, 1986.
6. Chen Y, Pederson LL, Letcoe NM: Passive smoking and low birthweight. Lancet ii: 54-5, 1989.
7. MacArthur C, Knox EG: Passive smoking and birthweight. Lancet i: 37-8, 1987.
8. Martin TR, Bracken MB: Association of low birthweight with passive smoke exposure in pregnancy. Am J Epidemiol 124: 633-42, 1986.
9. MacMahon B, Alpert M, Salbert BJ: Infant weight and parental smoking habits. Am J Epidemiol 82: 247-61, 1966.
10. Schwartz-Bickenbach D, Schulte-Hobein B, Abt S, Plum C, Nau H: Smoking and passive smoking during pregnancy and early infancy: effect on birthweight, lactation period and cotinine concentration in mother's milk and infant urine. Toxicol Lett 35: 73-81, 1987.
11. Yerushalmy J: Smoking habit of father and weight of infant. In: James G, Rosenthal T, (eds.), Tobacco and Health. Springfield: Charles C. Thomas, pp. 216-26, 1962.
12. Roquer JM, Figueras J, Botet F, Jimenez R: Influence on fetal growth of exposure to tobacco smoke during pregnancy. Acta Paediatr 84: 118-21, 1995.
13. Richards GA, Terblanche APS, Theron AJ, Opperman L, Crowther G, Myer MS, et al: Health effects of passive smoking in adolescent children. S Afr Med J 86: 143-147, 1996.
14. Ogawa H, Tominaga S, Hori K, Noguchi K, Kanuo I, Matsubara M: Passive smoking by pregnant women and fetal growth. J Epidemiol Community Health 45: 164-8, 1991.
15. Haddow JE, Knight GJ, Palomaki GE, McCarthy JE: Second trimester serum cotinine levels in nonsmokers in relation to birth weight. Am J Obstet Gynecol 159: 481-4, 1988.
16. Underwood PB, Kesler KF, O'Lane JM, et al: Parental smoking empirically related to pregnancy outcome. Obstet Gynecol 29: 1-8, 1967.
17. Terris M, Gold EM: An epidemiological study of prematurity. Am J Obstet Gynecol 103: 358-70, 1969.
18. Norusis MJ: Advance statistics SPSS/PC+. Chicago IL: SPSS Inc. Press 1986, P1.
19. Gardner MJ, Altman DG: Statistics with confidence. London: British Medical Association, 1989.