Int Arch Occup Environ Health (2004) 77: 47–52
DOI 10.1007/s00420-003-0475-0

ORIGINAL ARTICLE

Wojciech Hanke · Wojciech Sobala · Jarosław Kalinka

# Environmental tobacco smoke exposure among pregnant women: impact on fetal biometry at 20–24 weeks of gestation and newborn child's birth weight

Received: 12 May 2003 / Accepted: 21 August 2003 / Published online: 31 October 2003


**Abstract** ***Aim***: While there are sufficient data regarding the negative effect of exposure to the constituents of tobacco smoke on newborn infants' birth weights, it is still unclear whether this effect may originate in early pregnancy. The aim of the present study was to evaluate the impact of exposure to tobacco smoke components in early pregnancy (20–24 weeks) on fetal biometry. ***Methods***: The study population comprised 183 women consecutively enrolled at 20–24 weeks of pregnancy at the two antenatal care units. Ultrasound biometric measurements of fetal bi-parietal diameter (BPD), abdominal circumference (AC) and femur length (FL) were performed at the time of enrolment. Serum cotinine concentration was determined at 20–24 weeks of gestation by gas chromatography with mass spectrometry detector (GC/MS) to assess environmental tobacco smoke (ETS) exposure during the previous evening and the morning of the same day (blood collection at 1200–1300 h). ETS exposure (passive smoking) was assumed to occur when the level of serum cotinine ranged from 2–10 ng/ml. ***Results***: In a multiple regression model for bi-parietal diameter (BPD), after adjustment for pregnancy duration at the time of ultrasound examination, fetal gender, and maternal pre-pregnancy weight, a statistically significant negative association was found between the BPD and serum cotinine concentration. A similar association was identified for subjects with serum cotinine concentrations below 10 ng/ml (corresponding to passive smoking) ($P = 0.06$). After controlling for pregnancy duration, maternal pre-pregnancy weight and infant's gender, we found that serum cotinine levels at 20–24 weeks of gestation was inversely associated with infant birth weight ($P = 0.004$). For the subjects with serum cotinine levels below 10 ng/ml, a borderline association ($P = 0.09$) with infant birth weight was found. ***Conclusions***: Maternal exposure to tobacco smoke in early pregnancy, as measured by serum cotinine concentrations at 20–24 weeks of gestation, adversely affects fetal BPD. Preventive measures need to be undertaken to encourage pregnant women to stop smoking and avoid passive exposure to tobacco smoke from the very beginning of pregnancy.

**Keywords** Environmental tobacco smoke · Maternal smoking · Fetal biometrics · Birth weight · Fetal bi-parietal diameter

W. Hanke (✉) · W. Sobala
Department of Environmental Epidemiology, Nofer Institute of Occupational Medicine, 8 Teresy Street, 90-950 Lodz Poland
E-mail: wojt@imp.lodz.pl
Fax: +48-42-6314562

J. Kalinka
Department of Perinatology, Institute of Gynecology and Obstetrics, Medical University of Lodz, Lodz, Poland

## Introduction

Cigarette smoke inhaled by active smokers and environmental tobacco smoke (ETS) contain similar constituents. The latter is composed of exhaled smoke (MS) from smoker, sidestream smoke (SS) emitted from the smoldering tobacco puffs, contaminants emitted into the air during puffs, and contaminants that diffuse through the cigarette paper and mouth end between puffs [23] The concentrations of the chemicals found in diluted SS may exceed, even by several times, those in the MS combustion [23]. ETS is a complex mix of over 4,000 chemical compounds. Many of these substances are known to pass through the placental barrier [3, 13, 22]. Both active smokers and non-smokers exposed to ETS presented increased concentrations of the nicotine metabolite, cotinine, in amniotic fluid when compared with non-smokers with no ETS exposure [3, 13].

For a few decades it has been known that active smoking in pregnancy leads to a decrease in birth weight of approximately 150–250 g [1]. A dose–response relationship has been found between the number of cigarettes smoked by the mother per day and decreased birth weight. Recent studies have confirmed that such an

effect, although to a smaller extent, can be expected in passive smokers—with a decrement of approximately 25–40 g [14]. While there are sufficient data regarding the negative effect of exposure to the constituents of tobacco smoke or ETS on birth weight, it is still unclear whether this effect may originate from exposure in early pregnancy. To our knowledge, only the study by Newnham et al. examined the effect of tobacco smoke exposure on mid-gestation fetal biometry [17].

The main aim of our study was to evaluate the impact of exposure to tobacco smoke components on fetal biometry in early pregnancy (20–24 weeks). Specific objectives included evaluation of: (a) the proportions of active smokers and ETS exposed in the indigent population of pregnant women in Poland, (b) association between serum cotinine and fetal biometry and (c) relationship between single serum cotinine measurements and newborn children's birth weights. We used the latter outcome to validate the utilized tobacco smoke exposure estimates rather than to examine the relationship between exposure to tobacco smoke and birth weight (which has been satisfactorily evaluated in other studies).

## Methods

### Subjects

The initial study population comprised 207 consecutively enrolled women at 20–24 weeks of pregnancy who visited their obstetricians at the antenatal care units between April 1997 and April 1998. Ten units, cooperating with the Institute of Gynecology and Obstetrics, Medical University of Lodz, were covered by the study. Six women with chronic diseases (diabetes, organic heart disease, arterial hypertension and other diseases potentially hazardous to the fetus) were excluded from the analysis. For three persons, complete medical data concerning the newborn infants were not available. Fifteen women refused to have serum cotinine measurements. The final group included 183 women. The scope of the examinations to be performed and the study protocol were approved by the ethical committee of the Medical University of Lodz (RNN/212/97), and informed consent from the subjects was obtained prior to the study.

### Interview

For each subject, a detailed medical questionnaire was administered at 20–24 weeks of pregnancy. It covered socio-economic and occupational issues, obstetric background, and detailed data regarding smoking habits and passive smoking.

### Ultrasound examination

Ultrasound biometric measurements of fetal bi-parietal diameter (BPD), abdominal circumference (AC) and femur length (FL) were performed between 20 and 24 weeks of gestation. Each ultrasound measurement was made three times and the mean values were used for further analysis.

### Birth weight measurement

Neonatal morphometric measurements of birth weight, length, abdominal and thoracic circumference were performed immediately after delivery.

### Serum cotinine measurement

Serum cotinine concentration was determined at 20-24 weeks of gestation by gas chromatography with mass spectrometry detector (GC/MS) to assess ETS exposure during the previous evening and the morning of the same day (blood collection at 1200–1300 h) [4]. The limit of detection (LOD) was 0.16 ng/ml, while the limit of quantification (LOQ) was 1.25 ng/ml.

The subjects were assumed to be: non-smokers not exposed to ETS when their serum cotinine level was below 2 ng/ml; exposed to ETS (passive smokers) when their level of serum cotinine ranged from 2–10 ng/ml; active smokers when their serum cotinine levels exceeded 10 ng/ml [16].

### Statistical analysis

Differences between the mean values of the quantitative variables were analyzed by linear regression. Multiple linear regression was used to control for confounders. In our analysis of the relationship between cotinine and fetal biometrics, three confounders were considered (pregnancy duration at the time of ultrasound examination, fetal gender and maternal pre-pregnancy weight). Cotinine concentrations were log-transformed when a full-range dose–response relationship was modeled. All statistical analyses were performed with STATA 6 software.

## Results

### General characteristics

A comparison of the population of 183 women who had undergone serum cotinine measurement and ultrasound examination, with the population of 205 women considered for the study, did not reveal any significant differences with respect to gestational age at the time of examination, mean serum cotinine concentration, mean infant birth weight, and mean pregnancy duration (data not shown). Accordingly, we can assume that the study population was a fully representative sample of pregnant women covered by antenatal care in the city of Lodz.

The majority of the study population were women aged 20–29 (73.8%), married (77.6%) and primiparous (60.7%). Secondary level of education prevailed (45.3%). More than half of the subjects reported working for at least 1 month during pregnancy. No significant differences were found in the distribution of these variables in three examined cotinine groups. Tobacco smoking was reported by 13.7% of the interviewed women, and 5.5% declared smoking more than five cigarettes a day. In the non-smokers, almost half (48.7%) of the subjects reported daily exposure to ETS, mainly due to their husband's/partner's smoking and workplace exposure (46.2%).

### Smoking status and serum cotinine level

According to the results of serum cotinine determinations, 9.8% women were classified as non-smokers, while 66.1% and 24.0% were passive and active smokers, respectively (Table 1). Among women reporting a

**Table 1** Smoking status reported at interview and serum cotinine levels at 20–24 weeks' gestation

| Serum cotinine levels[a] (ng/ml) | Non-smokers | | Passive smokers | | Smokers | | Total | |
|---|---|---|---|---|---|---|---|---|
| | (*n*) | (%) | (*n*) | (%) | (*n*) | (%) | (*n*) | (%) |
| < 2.0 | 11 | 13.6 | 7 | 9.1 | 0 | 0.0 | 18 | 9.8 |
| 2.0–10 | 65 | 80.3 | 53 | 68.8 | 3 | 12.0 | 121 | 66.1 |
| > 10 | 5 | 6.2 | 17 | 22.1 | 22 | 88.0 | 44 | 24.0 |
| Total | 81 | 100 | 77 | 100 | 25 | 100 | 183 | 100 |

[a]Indicates biochemical non-smokers (< 2.0 ng/ml), passive smokers (2.0–10.0 ng/ml) and active smokers (> 10 ng/ml)

'non-smoking status', 6.2% had serum cotinine levels indicative of active smoking. These results also revealed that approximately 80% of this group were exposed to ETS, while only approximately 14% experienced no exposure to tobacco smoke (either from active or passive smoking). Passive smoking was confirmed in 68.8% of women who reported such exposure during the interview. However, in 22.1% of the subjects reporting passive smoking, the level of serum cotinine indicated their involvement in active smoking. For all the groups of non-smokers, including those who reported passive smoking, 74.6% of the subjects experienced exposure to ETS, while 13.9% were active smokers. Most of the smokers (88.0%) had serum cotinine levels that confirmed the smoking status reported in the questionnaire.

### Ultrasound biometric measurement of the fetus at 20–24 weeks of gestation, and serum cotinine level

Although mean values of BPD, AC and FL decreased with increasing values of maternal serum cotinine, none of the observed differences reached statistical significance (Table 2). However, in a multiple regression model for BPD), after adjustment for pregnancy duration at time of ultrasound examination, fetal gender, and maternal pre-pregnancy weight, a statistically significant negative coefficient was found for serum cotinine (Table 3). A similar tendency was observed for fetal FL but not for AC. In both cases the coefficients for serum cotinine were not statistically significant. In analyses performed for the subjects with serum cotinine concentrations below 10 ng/ml, BPD and FL were negatively associated with cotinine at a borderline level of significance ($P = 0.06$ and $P = 0.07$ respectively) (Table 4).

**Table 2** Mean fetal biometric parameters at 20–24 weeks' gestation and birth weight, by serum cotinine level

| Parameter | Serum cotinine levels[a] (ng/ml) | | | *P* |
|---|---|---|---|---|
| | < 2.0 (*n* = 18) | 2.0–10.0 (*n* = 121) | > 10 (*n* = 44) | |
| Mean (SD) BPD | 58.4 (12.2) | 57.5 (10.1) | 55.2 (7.4) | 0.556 |
| Mean (SD) AC | 185.8 (44.7) | 180.1(35.3) | 169.7 (28.9) | 0.304 |
| Mean (SD) FL | 40.0 (10.2) | 39.4 (7.9) | 35.5 (7.5) | 0.147 |
| Mean (SD) birth weight | 3419 (392) | 3393 (459) | 3172 (590) | 0.035 |

[a]Indicates biochemical non-smokers (< 2.0 ng/ml), passive smokers (2.0–10.0 ng/ml) and active smokers (> 10 ng/ml)

### Serum cotinine and birth weight

Mean birth weights were significantly lower for subjects with cotinine levels that indicated passive smoking and active smoking than for non-smokers not exposed to ETS ($P = 0.035$) (Table 2) After controlling for pregnancy duration, maternal pre-pregnancy weight and infant gender, we found that serum cotinine levels at 20–24 weeks of gestation was inversely associated with infant birth weight ($P = 0.004$). For the subjects with serum cotinine levels below 10 ng/ml, a borderline association ($P = 0.09$) with infant birth weight was found (Table 5). The deficit in birth weight attributed to passive smoking was 100 g.

## Discussion and conclusions

*Prevalence of environmental exposure to tobacco smoke:* the findings of our study utilizing serum cotinine measurements imply that 75% of non-smoking pregnant women are exposed to tobacco smoke in their environment. Haddow et al. found levels of serum cotinine above 0.5 ng/ml in 69% of 1,231 non-smoking pregnant women examined in their second trimesters [9]. Among German women, 83% had a detectable cotinine concentration in urine (above 2 ng/mg creatinine) and the corresponding rate for Polish women was 92% [5].

Questionnaire-based studies tend to provide lower estimates of the prevalence of ETS-exposed population than do those using biomarkers. For example, in a previous study conducted in the Lodz region (central Poland) that was based on women's reports of daily duration of passive exposure to tobacco smoke, a 47% prevalence of ETS exposure was found [10], similar to that observed in Canada [8] and USA [15]. However, it was significantly higher than in Sweden, where only approximately 24% of non-smoking pregnant women were exposed to ETS [2].

*Ultrasound biometric measurement of the fetus and serum cotinine levels:* the data obtained indicate an unfavorable effect of tobacco smoke on fetal development measured at 20–24 weeks of pregnancy. This is manifested by decreased BPD values and an increasing

**Table 3** Multiple linear regression for fetal biometric parameters at 20–24 weeks' gestation, by serum cotinine level (full concentration range), fetal gender, maternal pre-pregnancy weight and gestational age ($n = 183$)

| Variable | BPD | | | AC | | | FL | | |
|---|---|---|---|---|---|---|---|---|---|
| | Coefficient | Standard error | *P* | Coefficient | Standard error | *P* | Coefficient | Standard error | *P* |
| Gender (reference—male) | −1.680 | 0.423 | 0.001 | −4.132 | 1.515 | 0.01 | −0.183 | 0.374 | 0.625 |
| Gestational age (weeks) | 2.745 | 0.598 | 0.001 | 10.017 | 0.213 | 0.001 | 2.303 | 0.0527 | 0.001 |
| Maternal pre-pregnancy weight (kg) | 0.002 | 0.023 | 0.933 | 0.043 | 0.083 | 0.6 | 0.0222 | 0.020 | 0.278 |
| Logarithm of serum cotinine | −0.495 | 0.175 | 0.005 | 0.1153 | 0.624 | 0.8 | −0.1702 | 0.154 | 0.271 |
| Constant | −3.033 | 1.926 | 0.116 | −47.109 | 6.872 | 0.001 | −13.796 | 1.697 | 0.000 |

**Table 4** Multiple linear regression for fetal biometric parameters at 20–24 weeks' gestation, by serum cotinine level (concentrations below 10 ng/ml), fetal gender, maternal pre-pregnancy weight, and gestational age ($n = 139$)

| Variable | BPD | | | AC | | | FL | | |
|---|---|---|---|---|---|---|---|---|---|
| | Coefficient | Standard deviation | *P* | Coefficient | Standard deviation | *P* | Coefficient | Standard deviation | *P* |
| Gender (reference—male) | −1.177 | 0.430 | 0.0007 | −4.267 | 1.748 | 0.016 | −0.412 | 0.338 | 0.225 |
| Gestational age (weeks) | 2.873 | 0.067 | 0.001 | 10.346 | 0.271 | 0.001 | 2.333 | 0.052 | 0.001 |
| Maternal pre-pregnancy weight (kg) | −0.049 | 0.025 | 0,05 | −0.039 | 0.024 | 0.016 | −0.012 | 0.019 | 0.527 |
| Serum cotinine (ng/ml) | −0.172 | 0.091 | 0.06 | −0.393 | 0.024 | 0.694 | −0.069 | 0.071 | 0.335 |
| Constant | −3.427 | 2.063 | 0.1 | −47.475 | 8.382 | 0.001 | −12.247 | 1.621 | 0.001 |

**Table 5** Serum cotinine at 20–24 weeks' gestation and birth weight

| Variable | Full range of cotinine concentrations $n = 183$ | | | Serum cotinine below 10 ng/ml $n = 139$ | | |
|---|---|---|---|---|---|---|
| | Coefficient | Standard error | *P* | Coefficient | Standard error | *P* |
| Gender (reference—male) | −163.252 | 57.420 | 0.005 | −240.570 | 62.856 | 0.001 |
| Gestational age (weeks) | 148.183 | 17.959 | 0.001 | 133.835 | 19.241 | 0.001 |
| Maternal pre-pregnancy weight (kg) | 10.332 | 3.186 | 0.001 | 9.231 | 3.623 | 0.012 |
| Logarithm of serum cotinine | −70.087 | 24.191 | 0.004 | −100.486 | 60.416 | 0.09 |
| Constant | −2862.359 | 718.922 | 0.0001 | −2148.674 | 770.488 | 0.006 |

level of serum cotinine concentration. Three main confounders were considered: pregnancy duration, fetal gender, and maternal pre-pregnancy weight. The latter was used to allow for socio-economic and constitutional differences among subjects. Maternal age and education was not taken into account, as the population examined was homogeneous in that respect.

Newnham et al. performed serial ultrasound biometric measurements to assess the effect of maternal smoking in a prospective study of 535 pregnancies. Decreased fetal BPD measurements were observed in fetuses of smoking women [17]. This effect was maximal at 24 weeks' gestation but was restricted to male fetuses and was not associated with altered head circumference after birth.

*Exposure to ETS and birth weight:* in this prospective study, we demonstrated an indirect relationship between maternal serum cotinine and infant birth weight while controlling for such confounders as pregnancy duration, fetal gender, and maternal pre-pregnancy weight. The relatively small size of the population precluded us from including additional variables and building more complex models.

In our study we found a fairly large (100 g), although not statistically significant, ($P = 0.09$) decrement in birth weight that was associated with passive smoking (serum cotinine levels 2–10 ng/ml). One of the reasons might be the fact that the study population originated from an indigent community, where exposure to passive smoke could be accompanied by poor living conditions, low educational level, and, possibly, malnutrition.

In other reports, the deficit in birth weight assumed to be the result of maternal exposure to ETS (i.e., the difference in the mean birth weight between the exposed and non-exposed groups) varied from 10 g to 120 g [2, 9, 12, 15, 21]. The lowest values of 10–30 g, of no statistical significance, were found in the studies conducted in China [6, 25] and Japan [18].

Windham et al. in their analysis of five studies ascertaining ETS exposure from multiple sources, after adjusting for confounders, demonstrated that the range of decrement was 10–50 g [24]. Misra and Nguyeyen [16] reviewed the results of 11 studies on ETS and mean birth weight. The reduction in birth weight associated with ETS ranged from 25 to 90 g. Five studies used a biological marker to measure ETS exposure: cotinine in blood [7, 9, 19], cotinine in mother's saliva [20] or nicotine concentration in maternal hair [11]. Two of these studies indicated a statistically significant decrease in mean birth weight: 104 g with serum cotinine levels below 1 ng/ml [9] and 87 g with serum cotinine levels above 1.7 ng/ml [20]. Eskenazi et al. and Jaakkola et al. noted a smaller decrement (45 g and 17 g, respectively) [7, 11].

*Strengths and limitations of the study:* the major strength of this study is the assessment of fetal biometricy at early pregnancy and the evaluation of tobacco smoke exposure based on maternal serum cotinine measurements. To our knowledge, no published studies have approached the problem of health consequences of fetal exposure to tobacco smoke constituents from that point of view. The major limitations of the study are the possibility of exposure misclassification and limited control of confounders. The first one is rather unlikely, as exposure categories proved to be accurate estimators for birth weight values. In the case of severe misclassification, such an association would not have been confirmed in our population. As to uncontrolled confounders, our population did not include pregnant women with chronic diseases and was homogeneous for age, education, and other socio-economic indicators. On the other hand, three major variables that influence birth weight were covered in the analysis.

*Implications for practice:* the argument of birth weight decrease as a result of smoking is frequently used by public health authorities and primary care practitioners to persuade smoking pregnant women to cease smoking or avoid ETS exposure. However, it does have two weak points that may contribute to the generally observed low effectiveness of preventive activities focused on eliminating exposure to tobacco smoke. Firstly, the information about the average values of decrement in birth weight hardly motivates women to give up smoking or avoid exposure to tobacco smoke. Secondly, even if the woman does become motivated, she may not realize the need for prompt action. The results of our study indicate that smoking during pregnancy, as early as 20–24 weeks of gestation, seems to affect fetal biometry and it is very likely that a similar effect, although to a lower extent, may result from ETS exposure.

## Conclusions

Approximately 75% of women who reported non-smoking in pregnancy presented serum cotinine levels that indicated passive smoking, and in 24% of all the women examined cotinine concentrations implied active smoking.

Fetal BPD parameters at 20–24 weeks of pregnancy were inversely associated with levels of tobacco smoke exposure as measured by serum cotinine levels.

An inversely proportional relationship between maternal serum cotinine concentration at 20–24 weeks' gestation and infants' birth weights has been found.

Preventive measures need to be undertaken to encourage pregnant women to stop smoking and to avoid passive exposure to tobacco smoke during pregnancy. The general population should be provided with educational activities to increase social awareness of the negative effects of passive and active smoking from the very beginning of gestation.

## References


1. Abel EA (1980) Smoking during pregnancy: a review of effects on growth and development of offspring. Hum Biol 52:593–625
2. Ahlborg G, Bodin L (1991) Tobacco smoke exposure and pregnancy outcome among working women. Am J Epidemiol 133:338–347
3. Andersen BD, Ng KJ, Iams JD (1982) Cotinine in amniotic fluids from passive smokers (letter). Lancet 1:791–792
4. Bardy AH, et al. (1993) Objectively measured tobacco exposure during pregnancy: neonatal effects and relation to maternal smoking. Br J Obstet Gynaecol 100:721–726
5. Becher H, Zatonski W, Jockel KH (1992) Passive smoking in Germany and Poland: comparison of exposure levels, sources of exposure, validity, and perception. Epidemiology 3:509–514
6. Chen Y, Pederson LL, Lefcoe NM (1989) Passive smoking and low birthweight (letter). Lancet ii:54–55
7. Eskenazi B, Prehn AW, Christianson RE (1995) Passive and active maternal smoking. Am J Public Health 85:395–398
8. Fortier I, Marcoux S, Brisson J (1994) Passive smoking during pregnancy and risk of delivering a small-for-gestational-age infant. Am J Epidemiol 139:294–310
9. Haddow JE, Knight GJ, Palomaki GE, McCarthy AE (1988) Second trimester serum cotinine levels in nonsmokers in relation to birth weight. Am J Obstet Gynecol 159:481–484
10. Hanke W, Kalinka J, Florek E, Sobala W (1999) Passive smoking and pregnancy outcome in central Poland. Hum Exp Toxicol 118:265–271
11. Jaakola JK, Nastad P, Magnus P (2001) Environmental tobacco smoke, parental atopy, and childhood asthma. Environ Health Perspect 109:579–582
12. Jedrychowski W (1998) Effects of poor air quality on the health of Krakow children. Jagiellonian University Collegium Medicum, Kraków
13. Jordanov JS (1990) Cotinine concentrations in amniotic fluid and urine of smoking, passive smoking and non-smoking pregnant women at term and in the urine of their neonates on 1st day of life. Eur J Paediatr 149:734–737
14. Lindbohm ML, Salmen M, Taskinen H (2002) Effects of exposure to environmental tobacco smoke on reproductive health Scand J Environ Health 28 [Suppl 2]:84–96
15. Martin TR, Bracken MB (1986) Association of low birth weight with passive smoke exposure in pregnancy. Am J Epidemiol 124:632–642
16. Misra D, Nguyen R (1999) Environmental tobacco smoke and low birth weight: a hazard in the workplace? Environ Health Perspect 107[Suppl 6]:879–904
17. Newnham JP, Patterson L, James I, Reid SE (1990) Effects of maternal cigarette smoking on ultrasonic measurements of fetal growth and on Doppler flow velocity waveforms. Early Hum Dev 24:23–36

18. Ogawa H, Tominaga S, Hori K, Noguchi K, Kanou I, Matsubara M (1991) Passive smoking by pregnant women and fetal growth. J Epidemiol Community Health 45:164–168
19. Peacock JL, Cook DG, Carey IM, Jarvis MJ, Bryant AE, Anderson HR, et al. (1998) Maternal cotinine level during pregnancy and birthweight for gestational age. Int J Epidemiol 27:647–56
20. Rebagliato M, Bolumar F, Florey C (1995) Assessment of exposure to environmental tobacco smoke in nonsmoking pregnant women in different environments of daily living. Am J Epidemiol 142:525–530
21. Rubin DH, Krasilnikhoff PA, Leventhal JM, Weile B, Berget A (1986) Effect of passive smoking on birth weight. Lancet 2:415–417
22. Smith N, Austen J (1982) Tertiary smoking by the fetus (letter). Lancet 1:1252
23. US Environmental Protection Agency (EPA) (1993) Respiratory health effects of passive smoking: lung cancer and other disorders. EPA, Department of Health and Human Services, 1993:93–3605. NIH publication: Smoking and tobacco control, monograph 4, Washington, DC
24. Windham GC, Eaton A, Hopkins B (1999) Evidence for an association between environmental tobacco smoke exposure and birthweight: a meta-analysis and new data. Paediatr Perinat Epidemiol 13:35–57
25. Zhang J, Ratcliffe J (1993) Paternal smoking and birthweight in Shanghai. Am J Public Health 83:207–210