Does Passive Smoking in Early Pregnancy Increase the Risk of Small-for-Gestational-Age Infants?

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ABSTRACT

Objectives. This study tested the hypothesis that women who deliver small-for-gestational-age infants are more often exposed to passive smoking at home or at work.

Methods. Among a 1-year cohort of nulliparous women in the city of Malmö, Sweden, 872 (87.7%) women completed a questionnaire during their first prenatal visit. The study was carried out among women whose pregnancies resulted in a singleton live birth (n = 826); 6.7% of infants were classified as small for their gestational age.

Results. Passive smoking in early pregnancy was shown to double a woman's risk of delivering a small-for-gestational-age infant, independent of potential confounding factors such as age, height, weight, nationality, educational level, and the mother's own active smoking (odds ratio [OR] = 2.7). A stratified analysis indicated interactional effects of maternal smoking and passive smoking on relative small-for-gestational-age risk.

Conclusions. Based on an attributable risk estimate, a considerable reduction in the incidence of small-for-gestational-age births could be reached if pregnant women were not exposed to passive smoking. (*Am J Public Health.* 1998 88:1523–1527)

A child's somatic and psychomotor development is dependent on intrauterine development. Intrauterine growth retardation, resulting in low weight in relation to gestational age, is a main concern in obstetrics, and small-for-gestational-age birthweight has frequently been used to define intrauterine growth retardation at birth. The risk of major congenital malformations increases from 3.3% in appropriate-for-gestational-age infants to 8.0% in small-for-gestational-age infants.[1] Also, at 6 years of age, 16% of appropriate-for-gestational-age full-term children show minor neurological dysfunctions, in contrast to 40% of small-for-gestational-age preterm children.[2] Recently Barker et al.[3–5] described "the small-baby syndrome," showing that low birthweight and intrauterine growth retardation are related to non-insulin-dependent diabetes, hypertension, raised serum cholesterol concentrations, higher adulthood plasma concentrations of fibrinogen and factor VII, and increased mortality from cardiovascular disease.[6]

Delivery of small-for-gestational-age neonates has been found to be associated with different maternal characteristics such as age (>30 years),[7] race,[7] below-average height,[7] low prepregnancy weight,[7–9] poor maternal weight gain,[7,8] primiparous status,[7,8] and low educational level.[8] Lifestyle factors such as maternal smoking have also been linked to fetal growth retardation in numerous studies.[7–11]

Passively inhaled smoke has been identified as an important public health problem and has been associated with lung cancer, chronic respiratory disease, and ischemic heart disease.[12] Children are found to be more susceptible to passive smoking than adults because of their higher ventilation rate and metabolic differences.[13] Prior research on the association between fetal growth and exposure to passive smoking has produced conflicting results; some studies have supported an effect[14–18] and some have not.[19–21] This inconsistency could, to some extent, be explained by the fact that these studies used different measures of exposure and different designs.

The aim of this prospective study of nulliparous women was to test the hypothesis that exposure to passive smoking at home or at work during pregnancy is associated with an increased risk of small-for-gestational-age infants.

Methods

This study was based on a cohort that included all nulliparous women living in the city of Malmö (240 000 inhabitants), Sweden, who were seen for prenatal care at 4 clinics over a 1-year period. Three of the clinics were public and 1 was private, and they treated about 85% to 90% of all nulliparous women living in Malmö. Recruitment occurred between September 1991 and September 1992. A total of 872 (87.7%) of the 994 invited women agreed to participate.

Comparisons in terms of age, ethnicity, and marital status revealed only minor differences between participants and nonparticipants. The nonparticipants were somewhat younger and more likely to have been born abroad.[22] The groups did not differ, however,

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with respect to outcome variables such as infant birthweight and gestational age.

All nulliparous women were asked to complete a questionnaire during their first prenatal visit (on average, in their 12th postmenstrual week). Each woman had the opportunity to answer the questionnaire confidentially and of her own free will. The questionnaire contained background factors, such as age, nationality, educational level, and cohabiting status, and lifestyle factors such as smoking habits, physical exercise, alcohol consumption, and passive smoking.

This study was carried out among women with pregnancies resulting in a singleton live birth (n = 826). Women with multiple pregnancies (n = 12; 1.4%), those with subsequent miscarriages (n = 17; 1.9%), and, finally, those lost to follow-up at delivery (n = 17; 1.9%) were excluded. At the Department of Obstetrics and Gynecology of Malmö University Hospital and at the private Cura clinic, 806 (97.6%) of the women were routinely examined with ultrasound at 16 to 18 weeks and at 32 weeks of gestation. At the first routine examination, the pregnancy was dated and fetal anatomy examined. At the second examination, fetal weight was estimated and the deviation from the expected weight was calculated. Data on the outcome of the pregnancy, such as birthweight and gestational age, were collected from the perinatal database of the Department of Obstetrics and Gynecology at Malmö University Hospital and from medical records at other Swedish hospitals where 83 (10.0%) of the women gave birth.

Gestational Age

Determination of gestational age was based on the ultrasound examination performed before 20 weeks of gestation. The ultrasound fetometry included biparietal diameter and femur length. The reliability of ultrasound fetometry for estimating gestational age was found to be sufficient, with a standard deviation from true gestational age of 2.4 days. The following formula was used in determining gestational age: Biparietal Diameter × 1.2 + Femur Length × 1.0 + 49.[23]

In order to examine the small-for-gestational-age variable, deviations from expected birthweight were assessed according to an intrauterine growth reference curve.[24] This reference curve was based on longitudinal ultrasound estimations of fetal weights and data collected from 4 perinatal centers in Sweden and Denmark. If newborns had a birthweight more than 2 standard deviations below the age-related mean for the general population, they were classified as small for their gestational age.[24]

Background Variables

Maternal age was classified into 2 categories: 15 to 29 and 30 to 44 years.[7] Maternal weight was divided into normal and low prepregnancy weight (≤50 kg), and maternal height was classified as normal or below average (≤157 cm).[7,25] Prepregnancy weight and height were reported by the women at their first prenatal visit. Nationality was dichotomized according to whether the women were born in Sweden or not. Educational level was based on the women's years of education: 12 years or less or more than 12 years. Finally, the women were classified as cohabiting or not cohabiting with the child's father.

Lifestyle Variables

Presence of passive smoking was identified by the following question: "Have you been exposed to passive smoking during pregnancy (i.e., do other people smoke near you at home or at work)?" Two maternal smoking categories were used. Nonsmokers were women who, at their first prenatal visit, reported that they did not smoke; smokers were women who reported that they smoked regularly or irregularly. With regard to alcohol consumption, women were defined as nonconsumers (those who, at the first prenatal visit, reported that they did not drink alcohol at all) or consumers (those who were drinking).

Physical exercise was assessed via a single question. High physical exercise was defined as running, swimming, doing gymnastics, or playing tennis or badminton for at least 2 hours per week. Low physical exercise was defined as engaging in mostly sedentary activities, such as reading or watching TV, and moderate physical exercise was defined as engaging in activities such as walking or biking for at least 4 hours a week.[26] (The low and moderate categories were combined in the analyses).

Statistical Analyses

The *t* test for equality of means was used to analyze differences in weight and height between women who delivered small-for-gestational-age infants and those whose infants were not small for their gestational age. Maternal age, height, weight, nationality, educational level, and smoking were regarded as potential confounders because they are associated with both the dependent variable (small-for-gestational-age births) and the independent variable (passive smoking). Multiple logistic regression analyses were performed in order to adjust estimated odds ratios (ORs) for the influence of confounding factors. Initially, maternal age (model 1), height and weight (model 2), and nationality and educational level (model 3) were introduced into the model, followed by the lifestyle factor maternal smoking (model 4). Maternal age was used as a continuous variable in the regression analyses, and the others were used as dichotomous variables. Differences at the .05 level were considered statistically significant. Statistical analyses were performed with the SPSS program.[27]

The study was approved by the Ethics Committee of Lund University and by the Swedish Data Inspection Board.

Results

The study included 826 singleton liveborn infants. Of these, 6.7% (31 girls and 24 boys) were classified as small for gestational age, 20% (n = 11) were born preterm (less than 37 weeks of gestation), and 47.3% (n = 26) had birthweights below 2500 g. Of the 826 women examined, 65.9% (n = 530) were exposed to passive smoking at home or at work, 29.7% (n = 243) were smokers, and 27.0% (n = 214) consumed alcohol.

Associations between the various background and lifestyle variables and small-for-gestational-age births are presented in Table 1. The crude small-for-gestational-age risk was increased among women with low prepregnancy weights (≤50 kg) (OR = 1.9, 95% confidence interval [CI] = 1.0, 3.8) and among women of below-average height (≤157 cm) (OR = 2.1, 95% CI = 1.0, 4.6). The mean prepregnancy weight for women delivering small-for-gestational-age infants was 58.4 ± 9.1 kg, as compared with 61.0 ± 9.8 kg for women delivering infants of normal weight (*P* = .06). Mean heights for women with small-for-gestational-age babies and women with babies who were not small for their gestational age were 163.7 ± 7.1 cm and 166.6 ± 6.6 cm, respectively (*P* = .002). Both women of non-Swedish nationality and women with 12 or fewer years of education had an odds ratio in the univariate analysis of 1.7 (95% CI = 1.0, 3.1 and 95% CI = 0.9, 3.1, respectively). Women exposed to passive smoking at home or in the workplace had a significantly increased risk of delivering a small-for-gestational-age infant in comparison with women not exposed (OR = 2.3, 95% CI = 1.1, 4.6). Women who smoked in early pregnancy (vs nonsmokers) also had an increased risk (OR = 2.0, 95% CI = 1.1, 3.5).

Passive smoking was not significantly related to low birthweight (<2500 g) (OR = 1.3, 95% CI = 0.7, 2.5) or preterm

TABLE 1—Association Between Background and Lifestyle Characteristics and Risk for Small-for-Gestational-Age (SGA) Infants: Malmö, Sweden, 1991–1993

	SGA Infants/ All Infants	SGA, %	Crude Odds Ratio (95% Confidence Interval)
Maternal age, y			
15–29	38/600	6.3	1.0 ...
30–44	17/226	7.5	1.2 (0.7, 2.2)
Maternal weight			
>50 kg	43/697	6.2	1.0 ...
≤50 kg	12/107	11.2	1.9 (1.0, 3.8)
Missing	0/22	...	...
Maternal height			
>157 cm	46/735	6.3	1.0 ...
≤157 cm	9/72	12.5	2.1 (1.0, 4.6)
Missing	0/19	...	...
Nationality			
Swedish	37/637	5.8	1.0 ...
Non-Swedish	18/185	9.7	1.7 (1.0, 3.1)
Missing	0/4	...	...
Educational level			
>12, y	15/315	4.8	1.0 ...
≤12, y	38/496	7.7	1.7 (0.9, 3.1)
Missing	2/13	...	...
Cohabiting status			
Cohabiting	46/716	6.4	1.0 ...
Not cohabiting	7/98	7.1	1.1 (0.5, 2.6)
Missing	2/10	...	...
Passive smoking exposure			
No	10/274	3.6	1.0 ...
Yes	42/530	7.9	2.3 (1.1, 4.6)
Missing	3/19	...	...
Smoking status			
Nonsmoker	30/575	5.2	1.0 ...
Smoker	24/243	9.9	2.0 (1.1, 3.5)
Missing	1/7	...	...
Alcohol consumption			
Nonconsumer	33/578	5.7	1.0 ...
Consumer	17/214	7.9	1.4 (0.8, 2.6)
Missing	5/29	...	...
Physical exercise			
Low/moderate	40/619	6.5	1.0 ...
High	15/197	7.6	1.2 (0.6, 2.2)
Missing	0/10	...	...
Total	55/826	6.7	...

delivery (<37 gestational weeks) (OR = 1.2, 95% CI = 0.7, 2.3).

Table 2 shows the results from the multivariate analysis regarding the small-for-gestational-age risk of exposure to passive smoking. When maternal age, height, and weight were used as covariates in the logistic regression analysis, adjustment did not significantly change the small-for-gestational-age risk (OR = 2.4) (models 1 and 2). In model 3, when nationality and educational level were added, the odds ratio increased from 2.4 to 3.1. The addition of maternal smoking decreased the odds from 3.1 to 2.7, but the odds ratio was still statistically significant (model 4).

Table 3 presents the results of an analysis of the possible effect of maternal smoking and passive smoking on the risk of delivering a small-for-gestational-age infant. Nonsmoking women exposed to passive smoking had a crude odds ratio of 2.4 (95% CI = 1.02, 5.8) in comparison with nonsmoking women not exposed to passive smoking. Few women who smoked were not exposed to passive smoking (32), which could explain the finding that the higher odds ratio was not statistically significant (OR = 3.4, 95% CI = 0.8, 14.1). Women who smoked and also were exposed to passive smoking showed the highest risk (OR = 3.6, 95% CI = 1.5, 8.6). Adjustment for confounding factors (as in model 3) increased the odds of giving birth to small-for-gestational-age infants (see Table 3).

Based on the estimated odds ratios found in this study (adjusted for potential confounding factors), women exposed to passive smoking had an attributable risk of 62.9% and a population attributable risk of 53.0%.

Discussion

This study supports the hypothesis that exposure to passive smoking during pregnancy increases the risk of delivering a small-for-gestational-age infant and that the mother's exposure to both passive smoking and her own smoking might increase this risk even more. These results should, however, be discussed in terms of possible bias by selection, misclassification, or confounding.

Analysis of the nonparticipants revealed some minor differences regarding age and nationality but no differences regarding pregnancy outcomes such as birthweight and gestational age.[22] Earlier findings from this population showed that the younger women more often continued to smoke during pregnancy.[22] Since the nonparticipants comprised a greater proportion of younger women, this could indicate that the nonparticipants included more smokers than the participants. However, the effect of age, nationality, and smoking on the risk estimate concerning passive smoking and small-for-gestational-age deliveries was very marginal in our multivariate analysis. Therefore, it seems unlikely that selection bias could have affected our findings to any important degree.

The small-for-gestational-age variable was based on an estimation of birthweight in relation to gestational age. In this population, 6.7% of infants were classified as small for their gestational age, as compared with 6.3% in a Danish study[11] and 6.5% in a Scandinavian multicenter study.[9] Our somewhat higher incidence of small-for-gestational-age deliveries could be explained by the fact that such deliveries are more common in first pregnancies.[7,8] All of the women in our study were nulliparous, whereas both primigravidas and multigravidas were included in the other 2 studies. In the present study, 97.6% of all pregnancies were dated by routine ultrasound during the first half of the pregnancy. This method is regarded as the most valid measurement of gestational age.[23] Newborns were classified as small for their gestational age if their birthweight was more than 2 standard deviations below the expected age-related birthweight mean. Our small-for-gestational-age classification was based on sex-specific growth curves constructed for use in Scandinavian perinatological practice and research.[24] It was found that older standard intrauterine growth curves did not reflect fetal growth continuing in utero

TABLE 2—Association Between Exposure to Passive Smoking and Risk of a Small-for-Gestational-Age Infant: Malmö, Sweden, 1991–1993

	Crude Odds Ratio (95% Confidence Interval)	Adjusted Odds Ratio (95% Confidence Interval)			
		Model 1	Model 2	Model 3	Model 4
Passive smoking, yes vs no	2.3 (1.1, 4.6)	2.4 (1.2, 4.8)	2.4 (1.2, 4.9)	3.1 (1.4, 6.9)	2.7 (1.2, 6.0)
Maternal age, per year		1.0 (1.0, 1.1)	1.0 (1.0, 1.1)	1.1 (1.0, 1.1)	1.1 (1.0, 1.1)
Maternal height, ≤157 cm vs other			1.7 (0.7, 4.0)	1.7 (0.7, 4.2)	1.6 (0.6, 4.1)
Maternal weight, ≤50 kg vs other			1.8 (0.9, 3.9)	1.4 (0.7, 3.2)	1.4 (0.6, 3.2)
Nationality, non-Swedish vs Swedish				1.5 (0.8, 3.0)	1.7 (0.8, 3.4)
Educational level, ≤12y vs >12y				1.6 (0.8, 3.2)	1.5 (0.8, 3.0)
Maternal smoking, yes vs no					1.8 (1.0, 3.3)

TABLE 3—Association Between Different Combinations of Maternal Smoking and Exposure to Passive Smoking and Small-for-Gestational-Age (SGA) Infants: Malmö, Sweden, 1991–1993

Smoking Category	SGA Infants/All Infants	SGA, %	Crude Odds Ratio (95% Confidence Interval)	Adjusted Odds Ratio[a] (95% Confidence Interval)
Nonsmoking and no passive smoking[b]	7/240	2.9	1.0 ...	1.0 ...
Nonsmoking and passive smoking	22/323	6.8	2.4 (1.02, 5.8)	3.9 (1.4, 10.7)
Smoking and no passive smoking	3/32	9.4	3.4 (0.8, 14.1)	5.6 (1.2, 26.5)
Smoking and passive smoking	20/207	9.7	3.6 (1.5, 8.6)	6.0 (2.1, 17.5)
Total	52/802	6.5	...	...

[a]Adjusted for maternal age, weight, height, nationality, and educational level.
[b]Reference group.

and underestimated fetal growth in the preterm period.[24] Thus, we believe that misclassification was low in terms of the outcome variable.

It should be noted that environmental tobacco smoke exposure in this study was assessed with a single item, and duration of exposure and 24-hour exposure dose were not measured. Passive smoking was assessed as a dichotomous variable and defined according to whether the women were exposed to smoking from other people in their environment at home or at work. Young women may also be exposed to environmental tobacco smoke at restaurants or other public places; however, this should be a minor problem, since exposure times in such locations are relatively short in comparison with home or work exposures. Measurements of exposure to passive smoking differ considerably among earlier studies. The increasing social unacceptability of smoking could result in both over- and underreporting of passive smoking. However, self-reported exposure to passive smoking shows good agreement with measurements of serum cotinine,[18] air nicotine,[28] and saliva cotinine.[29] It could also be debated whether the association between passive smoking and reduced birthweight shown here might be explained, at least to some extent, by the fact that women underreport their own smoking. However, in a study in which parental reports of smoking were validated, it was found that only 1.5% of the infants of "nonsmoking" mothers had cotinine levels in their cord blood that could only be explained by active maternal smoking. In these cases, the fathers were nonsmokers.[18]

The results of this study suggest that passive smoking in early pregnancy approximately doubles a woman's risk of delivering a small-for-gestational-age baby. This association was not explained by the effects of confounding factors such as maternal age, weight, height, nationality, educational level, or the mother's own active smoking. However, there is always the possibility of additional residual confounding due to the existence of unmeasured variables. Several investigators have studied the effect of passive smoking among nonsmokers only, whereas our study included both smokers and nonsmokers. However, nonsmoking women exposed to passive smoking made up nearly half (42.3%) of the group delivering small-for-gestational-age infants. In the stratified analysis (Table 3), passive smoking among nonsmokers was found to be significantly associated with an increased risk of a small-for-gestational-age delivery (OR = 2.4). The combination of maternal smoking and passive smoking increased the odds to 3.6. The higher odds of small-for-gestational-age infants among smokers not exposed to passive smoking (OR = 3.4) might perhaps be explained by the fact that all smokers are also exposed to their own passive smoking (if the dose–response pattern of passive smoking and small-for-gestational-age deliveries is on a multiplicative scale). Alternatively, these higher odds could be due to low precision of the estimated odds ratio because of small numbers.

Women exposed to passive smoking had an attributable risk of 62.9% after adjustment for other risk factors, including the women's own smoking. In terms of population attributable risk percentage, we arrived at a 53.0% reduction in small-for-gestational-age deliveries. Of course, this must be regarded as a rather rough figure.

Prior research on the association between passive smoking and fetal growth has provided conflicting results.[14–21] Some studies have found, in results similar to ours, that passive smoking is significantly associated with intrauterine growth retardation when passive smoke exposure is measured during the current pregnancy.[16,17] However, no relationships have been detected when information about passive smoking is obtained after delivery.[20,21] This might be explained by recall bias. Measurements of exposure to passive smoking differ considerably between earlier studies. Many studies have investigated environmental smoke exposure attributable to fathers only,[15,28] some have estimated exposure either at home or at work,[16,20] and others have considered all environmental smoke exposures.[17,19,21]

In conclusion, our study shows that passive smoking in early pregnancy increases the odds of small-for-gestational-age deliveries, particularly in combination with the mother's own smoking. As a result of the relatively low numbers in this study, the precision of our estimate is somewhat limited, and more studies of this type are required in order to improve the odds estimate.

Identification of fetal growth retardation is a major goal of prenatal health care today, not only because it is a predictor of infant ill health but because it may affect subsequent adult disease.[3–6] Today we know more about the effect of active smoking on pregnancy than we do about the effect of passive smoke exposure. Passive smoke is an important public health problem, because many women are exposed to it. In our study population, 80.8% of the women who delivered small-for-gestational-age infants had been in smoking environments for variable periods of time early in pregnancy, compared with 65.9% of all nulliparous women. This study indicates that a considerable number of small-for-gestational-age deliveries could be prevented by removing passive smoke exposure at home and at work. Therefore, pregnant women should be strongly encouraged to avoid spending time in places where people smoke. □

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References

1. Khoury MJ, Erickson JD, Cordero JF, McCarthy BJ. Congenital malformations and intrauterine growth retardation: a population study. *Pediatrics.* 1988;82:83–90.
2. Hadders-Algra M, Touwen BCL. Body measurements, neurological and behavioural development in six-year-old children born preterm and/or small-for-gestational-age. *Early Hum Dev.* 1990;22:1–13.
3. Barker DJP, Hales CN, Fall CHD, Osmond C, Phipps K, Clark PMS. Type 2 (non-insulin-dependent) diabetes mellitus, hypertension and hyperlipidaemia (syndrome X): relation to reduced fetal growth. *Diabetologia.* 1993;36: 62–67.
4. Barker DJP, Martyn CN, Osmond C, Hales CN, Fall CHD. Growth in utero and serum cholesterol concentrations in adult life. *BMJ.* 1993;307:1524–1527.
5. Barker DJP, Meade TW, Fall CDH, et al. Relation of fetal and infant growth to plasma fibrinogen and factor VII concentrations in adult life. *BMJ.* 1992;304:148–152.
6. Osmond C, Barker DJP, Winter PD, Fall CDH, Simmonds SJ. Early growth and death from cardiovascular disease in women. *BMJ.* 1993;307:1519–1524.
7. Wen SW, Goldenberg RL, Cutter GR, Hoffman HJ, Cliver SP. Intrauterine growth retardation and preterm delivery: prenatal risk factors in an indigent population. *Am J Obstet Gynecol.* 1990;162:213–218.
8. Spinillo A, Capuzzo E, Piazzi G, Nicola S, Colonna L, Iasci A. Maternal high-risk factors and severity of growth deficit in small for gestational age infants. *Early Hum Dev.* 1994;38:35–43.
9. Bakketeig LS, Jacobsen G, Hoffman HJ, et al. Pre-pregnancy risk factors of small-for-gestational-age births among parous women in Scandinavia. *Acta Obstet Gynecol Scand.* 1993;72:273–279.
10. Brooke O, Anderson R, Bland M, Peacock J, Stewart M. Effects on birth weight of smoking, alcohol, caffeine, socioeconomic factors and psychosocial stress. *BMJ.* 1989;298:795–801.
11. Nordentoft M, Lou HC, Hansen D, et al. Intrauterine growth retardation and premature delivery: the influence of maternal smoking and psychosocial factors. *Am J Public Health.* 1996;86:347–354.
12. Law MR, Hackshaw AK. Environmental tobacco smoke. *Br Med Bull.* 1996;52:22–34.
13. Willers S, Skarping G, Dalene M, Skerfving S. Urinary cotinine in children and adults during and after semiexperimental exposure to environmental tobacco smoke. *Arch Environ Health.* 1995;50:130–138.
14. Rubin DH, Krasilnikoff PA, Leventhal JM, Weile B, Berget A. Effect of passive smoking on birth-weight. *Lancet.* 1986;23:415–417.
15. Zhang J, Ratcliffe JM. Paternal smoking and birthweight in Shanghai. *Am J Public Health.* 1993;83:207–210.
16. Martin TR, Bracken MB. Association of low birth weight with passive smoke exposure in pregnancy. *Am J Epidemiol.* 1986;124: 633–642.
17. Rebagliato M, Florey C, Bolumar F. Exposure to environmental tobacco smoke in nonsmoking pregnant women in relation to birth weight. *Am J Epidemiol.* 1995;142:531–537.
18. Martinez FD, Wright AL, Taussig LM. The effect of paternal smoking on the birthweight of newborns whose mothers did not smoke. *Am J Public Health.* 1994;84:1489–1491.
19. Ogawa H, Tominaga S, Hori K, Noguchi K, Kanou I, Matsubara M. Passive smoking by pregnant women and fetal growth. *J Epidemiol Community Health.* 1991;45:164–168.
20. Fortier I, Marcoux S, Brisson J. Passive smoking during pregnancy and the risk of delivering a small-for-gestational-age infant. *Am J Epidemiol.* 1994;139:294–310.
21. Chen LH, Petitti DB. Case-control study of passive smoking and the risk of small-for- gestational-age at term. *Am J Epidemiol.* 1995; 142:158–165.
22. Dejin-Karlsson E, Hanson BS, Östergren P-O, Ranstam J, Isacsson S-O, Sjöberg N-O. Psychosocial resources and persistent smoking in early pregnancy—a population study of women in their first pregnancy in Sweden. *J Epidemiol Community Health.* 1996;50:33–39.
23. Persson P-H, Weldner B-M. Reliability of ultrasound fetometry in estimating gestational age in the second trimester. *Acta Obstet Gynecol Scand.* 1986;65:481–483.
24. Marsal K, Persson P-H, Larsen T, Lilja H, Selbing A, Sultan B. Intrauterine growth curves based on ultrasonically estimated foetal weights. *Acta Paediatr.* 1996;85:843–848.
25. Bergsjö P, Hoffman HJ, Davies RO, et al. Preliminary results from the collaborative Alabama and Scandinavian study of successive small-for-gestational age births. *Acta Obstet Gynecol Scand.* 1989;68:19–25.
26. Grimby G, Wilhelmsen L, Björnstorp P, Saltin B, Tibblin G. Habitual physical activity. Aerobic power and blood lipids. In: Pernow B, Saltin B, eds. *Muscle Metabolism During Exercise.* New York, NY: Plenum Press; 1971:469–481.
27. Norusis MJ. *The SPSS Guide to Data Analysis for Release 4.* Chicago, Ill: SPSS Inc; 1990.
28. O'Connor TZ, Holford TR, Leaderer BP, Hammond SK, Bracken MB. Measurement of exposure to environmental tobacco smoke in pregnant women. *Am J Epidemiol.* 1995;142: 1315–1321.
29. Rebagliato M, Bolumar F, Florey C. Assessment of exposure to environmental tobacco smoke in nonsmoking pregnant women in different environments of daily living. *Am J Epidemiol.* 1995;142:525–530.