behavior (in never smokers, smokers who stopped before the sixth month, and persistent smokers) showed very particular dietary behaviors, inversely correlated to smoking: nonsmokers did not change their eating habits during the first trimester, those who stopped smoking increased their caloric intake by 95 kcal/day, and persistent smokers increased their caloric intake by 200 kcal/day. Caloric intake then decreased in all groups from 3 months to delivery, but significantly less so in persistent smokers than in the others. Consequently, weight gain was higher in smokers than in nonsmokers, which, in the multiple regression analysis, appeared to be a key factor in infant birth weight. In the whole sample, weight gain ranked second after controlling for duration of gestation; among smokers, it was ranked first, two positions before number of cigarettes smoked. As a whole, the difference in birth weight between infants of smokers and those of nonsmokers was only 70 g. Finally, the fetal growth of infants of mothers who smoked was better than expected because of the higher weight gain observed in women in this category.

The results of this study (5) may have been missed by the authors (1) or considered not applicable to the US population. Subsequent studies did not find such a clear interaction between anthropometry and smoking on birth weight (6, 7). Hence, the concern is to assess whether the behaviors we observed—which seem, up to now, specific to French women—cannot be considered as a basis for counseling mothers in order to balance the risk of small babies linked to thinness, especially in persistent smokers.

REFERENCES


Laure Papoz
INSERM U500, 34093 Montpellier, Cedex 5, France

THE AUTHORS REPLY

We thank Dr. Papoz for his observations (1) on our paper (2). His comments underscore the uncertainty that remains regarding the mechanism through which smoking affects fetal growth. As Dr. Papoz pointed out, maternal nutrition is hypothesized to mediate intrauterine growth restriction in infants born to smokers. While the extent of the role played by nutrition in fetal growth among smokers remains controversial, studies have shown that factors other than nutritional status are involved (3, 4).

In our study (2), we described the nature of the dose-response relation between tobacco exposure and birth weight as well as the relation between urine cotinine concentration and number of cigarettes smoked per day. We did not attempt to determine the mechanism through which smoking affects birth weight. It is true that we did not have information on dietary factors; however, it is unclear how these data would have contributed to our study. Because maternal nutrition status may be part of the causal pathway between smoking and fetal growth restriction, controlling for dietary factors in our analysis would have been inappropriate (5). Even controlling for prepregnancy body mass index (as we did) can potentially result in attenuation of the relation between smoking and infant birth weight. Therefore, we do not believe that the lack of data on dietary factors in our study prevented us from addressing our stated research questions.

We disagree with Dr. Papoz’s suggestion (1) that cigarette smoking and/or urine cotinine concentration in late pregnancy may not be useful measures of tobacco exposure for estimating the variation in birth weight due to smoking. Studies suggest that women who quit smoking during pregnancy deliver infants weighing as much as infants of never smokers (6, 7). This finding implies that tobacco exposure in late pregnancy is a more important determinant of birth weight than exposure in early pregnancy. Our data (2) also support this concept. We directly addressed the effects of changing tobacco exposure during pregnancy on infant birth weight in a later paper (8).

We agree with Dr. Papoz’s suggestion (1) that, in our study (2), the variability in birth weight explained by urine cotinine concentration and by cigarettes smoked per day may have been low because our study population consisted of only smokers and former smokers. Therefore, our findings are most relevant for populations of self-reported smokers. Cotinine concentration, for example, would undoubtedly be more useful in a population of women that included self-reported nonsmokers, where it would serve to both accurately categorize women as smokers or nonsmokers and quantify exposure.

Dr. Papoz (1) suggests that calorie supplementation may be an effective strategy to minimize fetal growth restriction in smokers. However, as Dr. Papoz acknowledges, studies addressing this issue have produced conflicting results (3, 4, 9, 10). In addition, reduced birth weight should be considered a surrogate for other adverse birth outcomes. While it is possible that calorie supplementation may offset some of the effects of smoking on fetal growth, the fetus is still exposed to toxins as long as the mother continues to smoke. Therefore, health care providers should continue to focus their efforts on helping pregnant women to quit smoking, because quitting is the most effective strategy for improving birth outcomes.
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Lucinda J. England1, Juliette S. Kendrick2, and Paul M. Gargiullo3
1National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD
2Division of Reproductive Health, Centers for Disease Control and Prevention, Atlanta, GA 30341
3Cancer Surveillance Branch, Centers for Disease Control and Prevention, Atlanta, GA 30341