Cotinine levels in women who smoke during pregnancy

To the Editors: I read with interest the article by Rebagliato et al (Rebagliato M, Bolúmar F, Florey C du V, Jarvis MJ, Pérez-Hoyos S, Hernández-Aguado I, et al. Variations in cotinine levels in smokers during and after pregnancy. Am J Obstet Gynecol 1998;178:568-71) in which they reported that women who smoked cigarettes during pregnancy had a lower cotinine per cigarette ratio during the last trimester than in the postnatal period. They suggested that this difference persisted after allowing for differences in cigarette consumption. I measured nicotine metabolites during pregnancy but used a new point-of-care urine test (performed in front of the patient) for nicotine metabolites[1] adapted from the method of Ellard et al.[2] During a randomized case-control study we measured the nicotine intake of >100 women who were smoking during pregnancy with the 5-minute colorimetric test and provided them with feedback about level of smoking. We measured nicotine intakes at recruitment and at 36 weeks' gestation and coupled these measurements with advice about smoking reduction. This led to an overall fall in nicotine intake and an improvement in smoking cessation among the intervention group.

The study also highlighted the fact that nicotine metabolite levels increased in 40% of control subjects (as opposed to 17.4% of women in the intervention group). Information collected by interview from the control women supported this increase. Control subjects reported an increase in smoking during the last trimester because of increased anxiety about the forthcoming birth and increased immobility and boredom. This increase largely manifested itself through a greater efficiency in smoking each cigarette, rather than through an increase in the number of cigarettes smoked. Members of the intervention group, in contrast, were more aware of the smoking habit and its effects on pregnancy and so resisted this tendency to increase nicotine intake. This elevation in cotinine levels during pregnancy had been reported previously in an uncontrolled study that did not witness the relative drop in levels among women who received counseling and feedback about their smoking levels.[3] Large-scale return to smoking during the postpartum period is well documented. Those women who continue to smoke throughout pregnancy will also return to smoking at their previous efficiency and thus increase their nicotine intake. From these data we suggest that the manner of smoking is as important as the number of cigarettes smoked.

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Reply

To the Editors: We agree with Cope's suggestion that the manner of smoking may in part explain the differences found between antenatal and postnatal cotinine levels. This was discussed in the Comment section of our article. Unfortunately we did not get information about the length and depth of inhalation or the number of puffs, and so this factor could not be accounted for in the analyses.

In our study, in contrast to the study referred to by Cope, each woman acted as her own control and there was no intervention that would be expected to affect the manner of smoking. Furthermore, it is difficult to ascertain from the available information whether there was a general tendency to increase efficiency of smoking during the first months after giving birth, a period during which women tend to be busy breast-feeding or taking care of their babies. Although the manner of smoking could account for part of the variation between the 2 cotinine measurements, we do not think that such a large difference can be explained simply by the manner of smoking.

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Peripheral blood leukocyte antigen expression in pregnancy and preeclampsia

To the Editors: Sacks et al (Sacks GP, Studena K, Sargent K, Redman CW. Normal pregnancy and preeclampsia both produce inflammatory changes in peripheral blood leukocytes akin to those of sepsis. Am J Obstet Gynecol 1998;179:80-6) reported on the profiles of leukocyte surface antigens in normal pregnancy and in preeclampsia. They noted similarities to antigens expressed on leukocytes from patients with sepsis and consequently suggested that the cells in the circulation are activated and that pregnancy involves a generalized inflammatory response that is more marked in preeclampsia. There is an alternative explanation for their data.

CD64 and CD14 are never expressed when normal neutrophils are activated in vitro and are not activation markers. Both of these antigens are expressed by circulating neutrophils after treatment of healthy subjects with granulocyte colony-stimulating factor, indicating mobilization of a different population of cells from the bone marrow.[1] Consequently, the report by Sacks et al that these antigens are present on neutrophils from both