



Editorial

Voluntary and involuntary smoking during pregnancy



Both voluntary and involuntary smoking exposure are main routes to intentional or unintentional environmental exposure to toxicants, and associated with an aggravated health status of the general population.¹ The adverse effects of these exposures threaten to undermine recent improvements in maternal and child health, and will result in increased rates of chronic diseases, disability and premature death in both newborns and women. Therefore, it is encouraging to learn that Dr. Huang's study published in this issue of the *Journal of the Chinese Medical Association* investigated this topic – the effects of maternal smoking exposure during pregnancy on postnatal outcomes: a cross sectional study.²

The authors enrolled 278 pregnant women in the third trimester at Fooyin University Hospital between March 2009 and February 2010, to study maternal exposure to smoking and postnatal outcomes.² The authors found that only 7% of the study subjects smoked; however, it was quite surprising to find that up to two-fifths of the study subjects were exposed to an involuntary smoking environment.² As expected, there was significantly higher birth weight, larger chest size, and higher serum bilirubin levels on postnatal day 3 as well as lower maternal urinary cotinine level in patients with smoking-free status, compared to those in the voluntary or involuntary smoking environments. This supportive evidence indicates a strong link between the occurrence of low birth weight babies and exposure to smoking while pregnant, either voluntary or involuntary.² This article is important, and worthy of additional discussion.

First, it is well-known that voluntary (active smoking) and involuntary (second-hand smoking or passive smoking) smoke exposure during pregnancy is associated with poor pregnancy and fetal outcomes, including lower birth weight babies who may generate much higher hospital costs than normal birth weight babies.^{3,4} The current study conducted by Huang and colleagues² clearly demonstrated that exposure to smoke is associated with low birth weight. Of further importance is that nearly half of pregnant women (47.8%) in southern Taiwan were exposed to environmental tobacco (toxicants), although the majority of them were exposed to involuntary smoke (85%, 7.2%/47.8%). In addition, the rate of paternal smoking was 59%, but the rate of passive smoking was more than two-fifths in the current study, suggesting that passive smoking for

pregnant women often occurred in the home. In fact, it is very difficult to cease involuntary smoke exposure at home. A recent study showed that the majority of husbands had never attempted to stop smoking at home, and most women had repeatedly asked their husbands to smoke outside, with little success.³ That is why the current study by Huang et al. emphasized that further studies are required to evaluate useful interventions in enhancing a smoking-free environment during pregnancy.²

Second, the use of DRI[®] cotinine enzyme immunoassay (Microgenics Corp., Fremont, CA, USA) in Huang's study² to establish a relationship between smoke and postnatal outcome requires further discussion.¹ In fact, based on the manufacturer's introduction (<https://tools.thermofisher.com/content/sfs/manuals/0228-DRI-Cotinine-Assay-EN.pdf>), the central limitations of the current test included: 1) a positive result from this assay indicated only the presence of cotinine, and does not necessarily correlate with the extent of physiological and psychological effects; and 2) a positive result by this assay should be confirmed by another non-immunological method such as the liquid chromatography-tandem mass spectrometry method. In addition, Dixon and Dasgupta found that the DRI cotinine assay consistently overestimated cotinine values obtained by the LC/MS/MS method, indicating that semi-quantitative values obtained using the DRI assay may be unreliable. They concluded that the DRI cotinine assay is suitable only for screening cotinine in urine specimens.⁵ Furthermore, Kim found that the selection of an optimal cotinine cutoff value for distinguishing true smokers from true nonsmokers manifested a lack of standardization.⁶ For example, a urine cotinine cutoff value range of 50–200 ng/mL has been commonly used to validate self-reported smoking status using a 2 × 2 table or a receiver operating characteristics (ROC) curve. The current study by Huang et al. showed an apparent overlap of maternal urinary cotinine level between smoking-free and passive smoking groups.

In conclusion, although the above-mentioned questions are raised, the value of Huang's article should be underscored, since smoke-free environment should be given considerably greater attention. Focus on this article should not be limited to smoking in public areas, but should include the in-house (home) environment, a locale which is often neglected.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the subject matter or materials discussed in this article.

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