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Exposure to environmental tobacco smoke for fetal lipid peroxidation

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Objectives: Environmental Tobacco Smoking (ETS) has been known to be associated with the adverse pregnancy outcomes and prenatal exposure to oxidative stress may explain fetal origin of adult disease. The purpose of this study was to investigate the association between maternal exposure to ETS and oxidative stress for neonates

Method : We measured the urinary concentration of cotinine in 266 pregnant women who denied smoking cigarettes during pregnancy and their singleton baby using radioimmunoassay. The urinary concentration of MDA is also assessed by HPLC.

Result: Maternal cotinine concentration was significantly associated with fetal cotinine concentration. Each increase of 10 μg/g cr in maternal urine cotinine concentration was associated with a 4 μg/g cr increase in fetal urine cotinine concentration ($P = 0.002$). For the subgroup of mothers whose urine cotinine concentrations remained below 120 μg/g cr, the maternal urine cotinine had no association with the fetal urine cotinine concentration. In contrast, for mothers above 120 μg/g cr, each increase of 10 μg/g cr in maternal urine cotinine concentration was associated with a 18 μg/g cr increase in fetal urine cotinine concentration ($P = 0.001$), and each increase of 10 μg/g cr in fetal urine cotinine concentration was associated with a 2 μg/g cr increase in fetal urine MDA concentration ($P < 0.001$)

Conclusions: These results suggest that maternal exposure to ETS affects fetal urine cotinine concentration and prenatal cotinine exposure affects fetal lipid peroxidation, particularly for maternal urine cotinine concentration above 120 μg/g cr. Therefore, smoking cessation is required among household members during pregnancy to prevent oxidative damage to fetus.