번호 III-15

제 목	국문	간접흡연에 의한 모성의 유전적 감수성과 모성과 태아의 산소성 손상				
	영문	Maternal and neonatal genetic susceptibility to oxidative damage from environmental tobacco smoke				
저자 및 소속	국문	홍윤철[1], 김현[2], 임문환[3], 이관희[4], 우복희[5], David C. Christiani[6] 인하대학교 의과대학 산업의학교실[1,4], 산부인과학 교실[3], 충북대학교 의과대학 예방의학교실[2], 이화여자대학교 의과대학 산부인과학교실[5], 하바드 보건대학원 환경보건학과[6]				
	영문	Yun-Chul Hong, Heon Kim, Mun-Whan Leem, Kwan-Hee Lee, Bock-Hi Woo, David C. Christiani Department of Occupational Medicine and Department of Obstetrics and Gynecology, Inha University College of Medicine, Department of Preventive Medicine, Chungbuk National University College of Medicine, Department of Obstetrics and Gynecology, Ewha Womans' University College of Medicine, Department of Environmental Health, Harvard School of Public Health				
분 야	보건관리 ( ) 역 학 ( ) 환 경 (○)		발 표 자	일반회원 (○) 전 공 의 ( )	발표 형식	구 연 ( ) 포스터 (○)
진행 상황	연구완료(○), 연구중( ) → 완료 예정 시기 : 년 월					

1. Background

Pregnant women and developing fetuses are reported to be vulnerable to environmental exposure to polycyclic aromatic hydrocarbons (PAH) including environmental tobacco smoke (ETS) (1). We conducted a study to examine whether exposure to PAH and ETS is associated with maternal and fetal oxidative damage.

2. Methods

We investigated 81 mothers and 20 newborns from Inchon, Korea. In addition to a questionnaire survey, we measured urinary cotinine, 1-hydroxypyrene, and 2-naphthol as exposure biomarkers and 8-hydroxydeoxyguanosine and malondialdehyde as oxidative damage biomarkers. CYP1A1 (MspI and Ile-Val), CYP2E1, GSTM1 and GSTT1 polymorphisms were also analyzed to assess genetic modification of any observed association.

3. Results

Maternal Urinary level of 1-OHP is· significantly correlated to urinary level of 2-naphthol (P<0.01). A significant correlation is also found between urinary concentrations of 8-OH-dG and MDA (P<0.01). In addition, urinary concentrations of 1-OHP and 2-naphthol are significantly correlated with concentration of MDA in urine (P<0.01), which indicates a close relationship between exposure to PAH and oxidative injury. However, urinary cotinine level was not related significantly with other concentrations of exposure and oxidative injury biomarkers.

The effect of genetic polymorphisms on the relationship of exposure biomarkers to oxidative biomarkers in maternal samples was evaluated while adjusting for age, height, body weight and diet. There was a significant interaction of CYP2E1 and 1-OHP on the level of 8-OH-dG (P=0.02). The regression coefficient of 1-OHP changed greatly from 0.072 to 0.787 when CYP2E1 gene had variant alleles. Variant alleles of CYP1A1 MspI polymorphism and the GSTM1 null genotype also markedly changed the regression coefficient of 1-OHP and 2-naphthol on the level of 8-OH-dG, even though there were not significant interactions between these polymorphisms and exposure biomarkers. The relationships between exposure biomarkers and MDA were not changed significantly by the genetic subtypes.

Neonatal urinary 8-OH-dG concentration was significantly associated with maternal exposure to ETS (P<0.05), even though the relationship of maternal exposure to ETS and maternal urinary 8-OH-dG was not significant (P=0.89). Neonatal urinary 8-OH-dG levels were also significantly elevated with maternal GSTM1 null genotype (P<0.01). These statistically significant relationships were unchanged in the two-predictor model and the multiple regression model which adjusted for other potential confounders. When neonatal urinary 8-OHdG was grouped according to maternal ETS exposure status and GSTM1 genotype, there were significant differences among subgroups. The concentration was highest in ETS exposure with maternal GSTM1 null and lowest in ETS non-exposure with maternal GSTM1 wild type.

4. Conclusions

Biomarkers of exposure and oxidative damage are correlated with each other in term pregnant women at labor. In addition, fetuses are very sensitive to oxidative damage from maternal exposure to ETS. For the relationship of exposure and oxidative damage of mother and neonates, there was modification of the toxic effects by some genotypes, including polymorphisms in metabolizing genes such as CYP1A1, CYP2E1, and GSTM1.