DIETARY BENZO(A)PYRENE AND FETAL GROWTH: EFFECT MODIFICATION BY VITAMIN C INTAKE AND MATERNAL *GSTP1* POLYMORPHISM

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Background and aims: Previous studies report reductions in fetal growth associated with maternal exposure to airborne polycyclic aromatic hydrocarbons (PAH), and with higher levels of DNA adducts reflecting total PAH exposure. The potential role of dietary PAHs, the main source of exposure to these compounds among non-smokers, remains uncertain. This study assesses associations between fetal growth indicators and maternal dietary intakes of PAH benzo(a)pyrene [B(a)P] during pregnancy, exploring the potential effect modification by dietary vitamin C and maternal *glutathione S-transferase pi1* (*GSTP1*) polymorphism, both hypothesized to influence PAH detoxification/metabolism.
Methods: 657 women in the INMA (Environment and Childhood) Project from Sabadell (Barcelona) were recruited during the first trimester of pregnancy. Dietary B(a)P intakes were estimated from food consumption data. Genotyping was conducted for the Ile105Val variant of *GSTP1*. Multivariable linear or logistic models were used to assess associations between size at birth and dietary B(a)P, stratified by vitamin C intakes and *GSTP1* polymorphism.
Results: There were significant interactions between elevated vitamin C intakes (above the mean, 189.41mg/day) and dietary B(a)P in models for birth weight and length ($P<0.05$), as well as small size for gestational age (SGA) ($P<0.10$). B(a)P intakes were associated with significant reductions in birth weight and length (coefficient±SE for a 1-SD increase in B(a)P: -101.63±34.62 g and -0.38±0.16 cm, respectively), and increased odds of SGA (OR for the top tertile of B(a)P: 3.51; 95%CI: 1.16-10.59) among women with low vitamin C intakes. Among these women, associations were strongest in those carrying the *GSTP1* Val allele, associated with lower contaminant detoxification activity.
Conclusions: Increasing intake of vitamin C in pregnancy could help to reduce the adverse effect of B(a)P exposure on fetal growth, particularly in genetically susceptible populations.