ARCHIVES OF PUBLIC HEALTH

POSTER PRESENTATION Open Access

Placental DNA methylation as a proxy for fetal neurodevelopment and sex-specific associations with in utero particulate air pollution

Nelly Saenen[1*], Bram Janssen[1], Karen Vrijens[1], Harry Roels[1], Wim Vanden Berghe[2], Wilfried Gyselaers[3], Charlotte Vanpoucke[4], Patrick De Boever[5], Tim Nawrot[1]

From Methods in Epidemiology Symposium
Leuven, Belgium. 17 September 2015

Background and aims

Exposure to particulate matter (PM) air pollution during pregnancy may affect human fetal development. Epigenetic mechanisms are believed to play an essential role in the developmental changes during early life. Within the ENVIRONAGE birth cohort, we investigated whether *in utero* exposure to PM is associated with differences in placental DNA methylation of genes involved in early neurodevelopment, i.e., Brain-Derived Neurotrophic Factor (*BDNF*), Leptin (*LEP*) and 5-Hydroxytryptamine (serotonin) receptor 2A (*HTR2A*).

Methods

Using highly quantitative bisulfite-PCR pyrosequencing, DNA promoter methylation was assessed in placental tissue of 385 newborns from the ENVIRONAGE birth cohort. Daily $PM_{2.5}$ exposure levels were estimated for each participant's home address using a spatiotemporal interpolation model in combination with a dispersion model. We fitted mixed-effect models, stratified for newborn's sex, to evaluate the associations between DNA promoter methylation of the selected genes and $PM_{2.5}$ exposure during pregnancy.

Results

Methylation of placental *BDNF* in male infants rose by 0.46% (p = 0.02) for an interquartile range (IQR) increment in $PM_{2.5}$ during the second trimester of pregnancy. For placental *HTR2A*, methylation in male infants rose by 4.8% (p = 0.02) for an IQR increment in first trimester $PM_{2.5}$ exposure. These associations were independent of maternal age, maternal education, maternal smoking status, gestational age, CpG site, first trimester temperature, and season at birth. No associations were observed between $PM_{2.5}$ exposure and placental *LEP methylation.* In girls no significant associations were noted.

Conclusions

Placental promoter methylation of *BDNF* and *HTR2A*, two genes implicated in early neurodevelopmental trajectories, are influenced by *in utero* exposure to $PM_{2.5}$ in a sex-specific way. Future studies should elucidate the significance of the sex-specific $PM_{2.5}$ impact on placental promoter methylation with respect to neurodevelopment later in life.

Authors' details
[1]Hasselt University, Diepenbeek, Belgium. [2]Department of Biomedical Sciences, PPES Epigenetic Signaling Lab, University of Antwerp, Antwerp, Belgium. [3]Department of Obstetrics, East-Limburg Hospital, Genk, Belgium. [4]Belgian Interregional Environment Agency, Brussels, Belgium. [5]Flemish Institute for Technological Research (VITO), Mol, Belgium.

Published: 17 September 2015

doi:10.1186/2049-3258-73-S1-P35
Cite this article as: Saenen *et al.*: Placental DNA methylation as a proxy for fetal neurodevelopment and sex-specific associations with in utero particulate air pollution. *Archives of Public Health* 2015 **73**(Suppl 1):P35.

[1]Hasselt University, Diepenbeek, Belgium
Full list of author information is available at the end of the article