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Elevated Cardiovascular Endothelial Inflammatory Biomarkers Linked to Environmental Pollutant Exposure in E-Waste-Exposed Children

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Background: Several environmental pollutant such as lead (Pb) and polycyclic aromatic hydrocarbon (PAH) exposure are positively associated with cardiovascular disease and the possible potential mechanism may be caused by damage to the endotheliumby modulation of inflammatory processes. S100A8/A9 is a useful biomarker of cardiovascular endothelial inflammation and has been implicated in endothelial cell apoptosis, endothelial dysfunction, leukocyte recruitment and adhesion and intimal hyperplasia. The objective of this study is to discuss the potential immune mechanism of children cardiovascular endothelial inflammation by exposure to environmental pollutants in the e-waste recycling area.

Methods: A total of 203 preschool children (3–7 years) were recruited from an e-waste exposed area and a reference area. We measured blood Pb levels and urinary PAH metabolites bygraphite furnace atomic absorption spectrometry and gas chromatography/mass spectrometry, respectively. Inflammatory cytokine (IL-6, IL-12p70 and IP-10) concentrations were detected by Luminex 200 analyzer. Endothelial inflammatory biomarkers S100A8/A9 levels were evaluated by ELISA.

Results: The exposed group exhibited higher blood Pb, total urinary hydroxylated PAH **(ΣOHPAH**), total hydroxynaphthalene (**ΣOHNap**) and total hydroxyfluorene (**Σ**OHFlu) levels in children compared with the reference group. Serum IL-6, IL-12p70, IP-10 and S100A8/A9 concentrations of children were significantly higher in the exposed group compared with the reference group. After adjustment for various confounders, linear regression analysis showed that both blood Pb levels and urinary **ΣOHFlu** levels are positively associated with IL-6, IL-12p70 and IP-10. Joint exposure of Pb × PAH is also positively associated with IL-6, IL-12p70 and IP-10. Mediator models indicated that neutrophils exert the significant mediation effect on the relationship between blood Pb levels and S100A8/A9.

Conclusions: E-waste-exposed children with elevated exposure levels of Pb and PAH exacerbate cardiovascular endothelial inflammation, ultimately increasing the risk for cardiovascular disease.

Keyword: E-waste; Environmental pollutants; Children; S100A8/A9; Cardiovascular endothelial inflammation

Biography:

Mr. Xiangbin Zheng a Ph.D. student of the Laboratory of Environmental Medicine and Developmental Toxicology, Shantou University Medical College, China. His primary research interest is in environmental toxicology and epidemiology, studying environmental toxicant exposures in relation to fetal and child development of various physiological systems. He investigated a variety of environmental exposures (e.g., Pb, Cd, Cr, Mn, PM2.5 and PAHs) for human developmental toxicity, including thyroid hormone disruption, fetal and child growth, child respiratory system and cardiovascular system development.