Chemosphere
Volume 183, September 2017, Pages 589-598

Chemosphere. 2017 Sep;183:589-598. doi: 10.1016/j.chemosphere.2017.05.153. Epub 2017 May 27.

Gene expression profiles and bioinformatics analysis of human umbilical vein endothelial cells exposed to $PM_{2.5}$.

Hu H[1], Asweto CO[1], Wu J[1], Shi Y[1], Feng L[1], Yang X[1], Liang S[1], Cao L[1], Duan J[2], Sun Z[3].

Author information

1. Department of Toxicology and Sanitary Chemistry, School of Public Health, Capital Medical University, Beijing 100069, PR China; Beijing Key Laboratory of Environmental Toxicology, Capital Medical University, Beijing 100069, PR China.
2. Department of Toxicology and Sanitary Chemistry, School of Public Health, Capital Medical University, Beijing 100069, PR China; Beijing Key Laboratory of Environmental Toxicology, Capital Medical University, Beijing 100069, PR China. Electronic address: jcduan@ccmu.edu.cn.
3. Department of Toxicology and Sanitary Chemistry, School of Public Health, Capital Medical University, Beijing 100069, PR China; Beijing Key Laboratory of Environmental Toxicology, Capital Medical University, Beijing 100069, PR China. Electronic address: zwsun@ccmu.edu.cn.

Abstract

Cardiovascular system is demonstrated the main target of $PM_{2.5}$ and the objective of this study was to explore the toxic effect and molecular mechanisms caused by $PM_{2.5}$ in primary human umbilical vein endothelial cells (HUVECs) using microarray and bioinformatics analysis. The results showed that 591 genes were differentially expressed triggered by $PM_{2.5}$, of which 174 genes were down-regulated, while 417 genes were up-regulated. Gene ontology analysis revealed that $PM_{2.5}$ caused significant changes in gene expression patterns, including response to stimuli, immune response, and cellular processes. Pathway analysis and Signal-net analysis suggested that endocytosis, chemokine signaling pathway, RNA transport, protein processing in endoplasmic reticulum (ER) and autophagy regulation were the most critical pathways in $PM_{2.5}$-induced toxicity in HUVECs. Moreover, gene expression confirmation of LIF, BCL2L1, CSF3, HMOX1, RPS6, PFKFB, CAPN1, HSPBP1, MOGS, PREB, TUBB2A, GABARAP by qRT-PCR indicated that endocytosis might be involved in the cellular uptake of $PM_{2.5}$ by forming phagosomes, and subsequently inflammation, hypoxia and ER stress was occurred, which finally activated autophagy after $PM_{2.5}$ exposure in HUVECs. In summary, our data can serve as fundamental research clues for further studies of $PM_{2.5}$-induced toxicity in HUVECs.

KEYWORDS:

Autophagy; Bioinformatics analysis; ER stress; HUVECs; Inflammation; PM(2.5)