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Co-exposure to amorphous silica nanoparticles and benzo[a]pyrene at low level in human bronchial epithelial BEAS-2B cells

- Authors
Jing Wu, Yanfeng Shi, Collins Otieno Asweto, Lin Feng, Xiaozhe Yang, Yannan Zhang, Hejing Hu, Junchao Duan, Zhiwei Sun

Abstract

Both ultrafine particles (UFP) and polycyclic aromatic hydrocarbons (PAHs) are widely present in the environment, thus increasing their chances of exposure to human in the daily life. However, the study on the combined toxicity of UFP and PAHs on respiratory system is still limited. In this study, we examined the potential interactive effects of silica nanoparticles (SiNPs) and benzo[a]pyrene (B[a]P) in bronchial epithelial cells (BEAS-2B). Cells were exposed to SiNPs and B[a]P alone or in combination for 24 h. Co-exposure to SiNPs and B[a]P enhanced the malondialdehyde (MDA) contents and reduced superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) activities significantly, while the reactive oxygen species (ROS) generation had a slight increase in the exposed groups compared to the control but not statistically significant. Cell cycle arrest induced by the co-exposure showed a significant percentage increase in G2/M phase cells and a decrease in G0/G1 phase cells. In addition, there was a significant increase in BEAS-2B cells multinucleation as well as DNA damage. Cellular apoptosis was markedly increased even at the low-level co-exposure. Our results suggest that co-exposure to SiNPs and B[a]P exerts synergistic and additive cytotoxic and genotoxic effects.

Keywords

Silica nanoparticle; Benzo[a]pyrene; Co-exposure; Cytotoxicity; Genotoxicity