

Low-dose combined exposure of nanoparticles and heavy metal compared with PM_{2.5} in human myocardial AC16 cells

- [Authors](#)

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Abstract

The co-exposure toxicity mechanism of ultrafine particles and pollutants on human cardiovascular system are still unclear. In this study, the combined effects of silica nanoparticles (SiNPs) and/or carbon black nanoparticles (CBNPs) with Pb(AC)₂ compared with particulate matter (PM)_{2.5} were investigated in human myocardial cells (AC16). Our study detected three different combinations of SiNPs and Pb(AC)₂, CBNPs and Pb(AC)₂, and SiNPs and CBNPs compared with PM_{2.5} at low-dose exposure. Using PM_{2.5} as positive control, our results suggested that the combination of SiNPs and Pb(AC)₂/CBNPs could increase the production of reactive oxygen species (ROS), lactate dehydrogenase leakage (LDH), and malondialdehyde (MDA) and decrease the activities of superoxide dismutase (SOD) and glutathione (GSH); induce inflammation by the upregulation of protein CRP and TNF- α , and apoptosis by the upregulation of protein caspase-3, caspase-9, and Bax while the downregulation of protein Bcl-2; and trigger G2/M phase arrest by the upregulation of protein Chk2 and downregulation of protein Cdc2 and cyclin B1. In addition, the combination of CBNPs and Pb(AC)₂ induced a significant increase in MDA and reduced the activities of ROS, LDH, SOD, and GSH, with G1/S phase arrest via upregulation of Chk1 and downregulation of CDK6 and cyclin D1. Our data suggested that the additive interaction and synergistic interaction are the major interaction in co-exposure system, and PM_{2.5} could trigger more severe oxidative stress, G2/M arrest, and apoptosis than either co-exposure or single exposure.

Keywords

Silica nanoparticles Carbon black nanoparticles Pb(AC)₂ Combined cardiovascular toxicity Human myocardial cells