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COMBINED EFFECT OF SILICA NANOPARTICLES AND BENZO[A]PYRENE ON CELL CYCLE ARREST INDUCTION AND APOPTOSIS IN HUMAN UMBILICAL VEIN ENDOTHELIAL CELLS

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Particulate matter (PM) such as ultrafine particulate matter (UFP) and the organic compound pollutants such as polycyclic aromatic hydrocarbon (PAH) are widespread in the environment. UFP and PAH are present in the air, and their presence may enhance their individual adverse effects on human health. However, the mechanism and effect of their combined interactions on human cells are not well understood. We investigated the combined toxicity of silica nanoparticles (SiNPs) (UFP) and Benzo[a]pyrene (B[a]P) (PAH) on human endothelial cells. Human umbilical vascular endothelial cells (HUVECs) were exposed to SiNPs or B[a]P, or a combination

of SiNPs and B[a]P. The toxicity was investigated by assessing cellular oxidative stress, DNA damage, cell cycle arrest, and apoptosis. Our results show that SiNPs were able to induce reactive oxygen species generation (ROS). B[a]P, when acting alone, had no toxicity effect. However, a co-exposure of SiNPs and B[a]P synergistically induced DNA damage, oxidative stress, cell cycle arrest at the G2/M check point, and apoptosis. The co-exposure induced G2/M arrest through the upregulation of Chk1 and downregulation of Cdc25C, cyclin B1. The co-exposure also upregulated bax, caspase-3, and caspase-9, the proapoptotic proteins, while down-regulating bcl-2, which is an antiapoptotic protein. These results show that interactions between SiNPs and B[a]P synergistically potentiated toxicological effects on HUVECs. This information should help further our understanding of the combined toxicity of PAH and UFP.

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