

The Effect of Particulate Matter on Cardiomyocyte Apoptosis Through Mitochondrial Fission

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Abstract : There is much evidence that exposure to fine particulate matter (PM) from air pollution increases the risk of cardiovascular morbidity and mortality. According to previous reports, PM in the air enters the respiratory tract, contacts the alveoli, and enters the blood circulation, leading to the progression of cardiovascular disease. PM pollution may also lead to cardiometabolic disturbances, increasing the risk of cardiovascular disease. The effects of PM on cardiac function and mitochondrial damage are currently unknown. We used mice and rat cardiomyocytes (H9c2) as animal and in vitro cell models, respectively, to simulate an air pollution environment using PM. These results indicate that the apoptosis-related factor PUMA, a regulator of apoptosis upregulated by p53, is increased in mice treated with PM. Apoptosis was aggravated in cardiomyocytes treated with PM, as measured by TUNEL assay and Annexin V/PI. Western blot results showed that CASPASE3 was significantly increased and BCL2 (B-cell lymphoid 2) was significantly decreased under PM treatment. Concurrent exposure to PM increases mitochondrial reactive oxygen species (ROS) production by MitoSOX Red staining. Furthermore, using Mitotracker staining, PM treatment significantly shortened mitochondrial length, indicating mitochondrial fission. The expression of mitochondrial fission-related proteins p-DRP1 (phosphodynamics-related protein 1) and FIS1 (mitochondrial fission 1 protein) was significantly increased. Based on these results, the exposure to PM worsens mitochondrial function and leads to cardiomyocyte apoptosis.

Keywords : particulate matter, cardiomyocyte, apoptosis, mitochondria

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