THE BIOLOGICAL EFFECTS OF AMBIENT FINE PARTICLES ON CARDIOVASCULAR RISK IN CITY RESIDENTS

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Background and Aims: Several studies have found that long-term exposure to particulate matter is an important environmental risk factor for cardiopulmonary disease morbidity and mortality, especially for particles with aerodynamic diameters under 2.5 micron fine particles. It is a challenge to elucidate the underlying pathophysiological mechanisms linking specific characteristics of fine particles and cardiovascular toxicity. The study aims to explore the association between long-term exposure to traffic-related fine particles air pollution and biological effects in cardiovascular system.

Methods: The participants included 420 men and women aged from 45-75. The distances between participants’ residences and major roads were categorized as • 50 m, 51–100 m, 101–200, and > 200 m. Concentration of fine particles were measured using SIDEPAK™ AM510 (TSI, USA) in 2010. Personal exposure to traffic particle was measured using AM510. Aortic augmentation index (AI) and heart rate variability (HRV) were determined using Model SKY-A4 Bioelectric Signals Processing System. Meanwhile, heart rate and blood pressure were measured. The association between long-term exposure to fine particles and AI, HRV, heart rate and blood pressure were analyzed with sex-stratified multiple linear regression analyses.

Results: The results showed that the concentrations of fine particles were 125, 94, 57 and 67 g/m^3 in January, April, July and October, respectively. Meanwhile, fine particles concentrations were correlated with residential proximity to major road. The concentrations were 108, 99, 71 and 64 g/m^3, respectively. In the adjusted analysis, fine particle exposure was associated with the decrease of HRV and increase of AI and blood pressure.

Conclusions: Traffic-related fine particles air pollution was associated with the cardiovascular risk. These results added to the evidence that long-term exposure to ambient fine particles associated with increased cardiovascular injury.