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Combined action of ultrafine particles and polycyclic aromatic hydrocarbon on human body

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Abstract
There is increased cardiovascular disease incidence attributed to ambient air pollution. It is estimated that ischaemic heart disease and stroke account for 80% of ambient air pollution-related early deaths. The most deleterious are the ultrafine particles (UFPs) which have more prominent potential health threat, since they can act as carriers of other strong air pollutant such as polycyclic aromatic hydrocarbons (PAHs). UFPs are small in size, hence can easily penetrate and reach cardiovascular tissues and organs. Endothelial dysfunction is associated with cardiovascular diseases such as hypertension, coronary artery disease, chronic heart failure, and peripheral vascular disease.

Keywords: Ultrafine particles, cardiovascular, endothelial dysfunction

Introduction
Cardiac diseases such as ischaemic heart disease and strokes account for 80% of ambient air pollution-related early deaths. Ambient air pollution has been on the rapid increase in most part of the world due to industrialization, urbanization, and motorization Ambient air pollutants comprise of particulate matter (PM), gases, organic compounds, and toxic metals. Particulate matter (PM) is a blend of liquid droplets (aerosols) and solid particles like dust, soot, smoke, and dirt. PM is found in smoke, diesel exhaust, and haze that either come specifically from combustion or is a result of response to gases and sunlight or air. In any case, the inhalable particles are of 10 μm in aerodynamic diameter (PM10), fine particles 2.5 μm (PM2.5), and ultra-fine particles <0.1 μm (UFP). PM2.5, most generally utilized as a representative marker of exposure to air pollution. polycyclic aromatic hydrocarbons (PAHs) result from incomplete combustion of organic materials and are ever present in the environment. Human exposure to PAHs can occur via ingestion of PAHs contaminated soil, food, and water, inhalation of PAHs contaminated soil dust and air and dermal contact with PAHs contaminated soil After PAHs entering human body via various exposure pathways, complex metabolism and unknown factors would also make PAHs reach cardiovascular tissues and organs. Due to widespread sources and persistent UFPs and PAHs in the environment. Human beings are exposed to UFP and PAH mixtures in particulate phases in ambient air. Long-term exposure to high concentrations of the mixture is associated with adverse health problems. Thus, studies on PAHs in particulate matter (PM), such as UFP in ambient air, have become attention greater focus of research in recent years.

Ultra-fine Particles and Polycyclic Aromatic Hydrocarbons
Ultra-fine Particles (UFPs) have been found to have an atmospheric concentration ten times higher in the urban air than in rural air and are considered the most detrimental of all PM fractions. UFPs act as carriers of other strong air pollutant such as Polycyclic Aromatic Hydrocarbons (PAHs) because of their adsorption capabilities. UFP are incidentally generated in the environment, often as by-products of fossil fuel combustion, condensation of semi-volatile substances or industrial emissions

Besides, elevated PAH metabolites in urine has been associated with increased CVD events Moreover, an increased risk for fatal ischemic heart disease in relation to occupational exposure to benzo(a)pyrene Polycyclic aromatic hydrocarbons (PAHs) are organic compound pollutants, which are ubiquitous in ambient air and exist as gases or joined to the Particulate Matter (PM). The existing interaction between UFPs and PAH from traffic related air pollutants might be the reason for progression of atherosclerosis observed in low level

pollution below the existing regulatory standards.

Mechanisms

Upon entering the bloodstream, the UFPs bound with PAH get into direct contact with blood vessels and the heart endothelial cells lining. Endothelial cells are the biological barriers which mediate clearance of nanoparticles, maintain vascular function and homeostasis. Moreover, *in vivo* and human studies have indicated damage to endothelial cells as an important mechanistic event by which inhalation of particles is associated with cardiovascular diseases. The initial events in atherogenesis include the expression of cell adhesion molecules on the surface of the endothelium, inflammation, and endothelial dysfunction. Both UFPs and PAH have also been shown to individually induce endothelial cell toxicity resulting in endothelial dysfunction Tithof *et al*. (2002) investigate the effects of polycyclic aromatic hydrocarbons contained in cigarette smoke on phospholipase A2 (PLA2) activity and apoptosis of human coronary artery endothelial cells. They found that B(a)P induce apoptosis of endothelial cells by a mechanism that involves activation of phospholipase A2 (PLA2), leading to endothelium dysfunction.

Joint Effects of UFPs and PAHs

Ultra-fine particles have been shown to have a greater content of redox active compounds, such as prooxidative polycyclic aromatic hydrocarbons (PAHs) that could provide them with a greater prooxidative potential In addition, their smaller size and greater surface-to-mass ratio may enable them to have greater bioavailability for the PAHs on their large surface area, making them more accessible to the contact sites of cells. PAHs adsorb onto particles play a toxicological role in generating ROS, oxidative stress, and inflammation once inhaled. The SiNPs and B[a]P coexposure of induced excessive oxidative stress, subsequently resulting to DNA damage, cell cycle arrest, and apoptosis of endothelial cells. Moreover, enhanced expression of proinflammatory and procoagulant genes have also been previously observed in SiNPs and B[a]P co-exposure, which is an indication of inflammation-coagulation cascade involvement in the co-exposure toxicity mechanism. Furthermore, oxidant injury plays an important role in UFP induced adverse health effects including exacerbation and promotion of atherosclerosis.

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