CARDIOVASCULAR EFFECTS OF AIR POLLUTION – POSSIBLE PATHWAYS

Particulate air pollution

Particulate matter (PM) is composed of solid and liquid particles from various sources, for example vehicle exhaust, road dust, forest fires or wind-blown soil. Combustion particles consist of an elemental carbon core surrounded by a layer of chemicals such as organic hydrocarbons, metals, nitrates and sulphates. The carbon core as well as the enclosing chemicals determine the toxicity of the particle. Ultrafine particles (UFP, particles with less than 100 nm in aerodynamic diameter), the major component in vehicle emission, have a large surface area and a high content of potentially harmful hydrocarbons which can promote oxidative stress. Traditionally, PM is measured as mass per space, such as PM_{10} and $PM_{2.5}$ (particle mass of particles with less than 10 µm and 2.5 µm in aerodynamic diameter, respectively); Importantly, particles below 100 nm contribute little to the overall mass, but represent more than 85% of the total $PM_{2.5}$ particle number.

Health effects of air pollution

Epidemiological research has shown that ambient air pollution is associated with an increase in mortality and morbidity of respiratory and cardiovascular diseases. Although it may intuitively seem that PM would pose a health risk to the lungs, the highest attributable risk of adverse health effects of PM is upon the cardiovascular system. In 2010, the American Heart Association concluded in an updated Scientific Statement, that "the overall evidence is consistent with a causal relationship between $PM_{2.5}$ exposure and cardiovascular morbidity and mortality".

Moreover, several studies have identified subgroups which are more susceptible to the adverse effects of particulate air pollution than the general population. They show that patients with pre-existing diseases such as chronic obstructive pulmonary disease, previous myocardial infarction or type 2 diabetes are at an increased risk of experiencing acute exacerbation of their disease on days with high concentrations of air pollution.

Hypothesized biological pathways

Air pollution can have chronic (long-term) and acute (short-term) effects on the cardiovascular system and potential mechanisms might differ. While chronic risk factors determine a vulnerability to acute coronary events, e.g. through particle-induced progression of atherosclerosis, acute effects are mediated by transient risk factors that might trigger acute events in susceptible populations. The major cause of coronary heart disease and cardiovascular death is a disruption of an atherosclerotic plaque and thrombus formation. The association between ambient air pollution and acute cardiovascular events could therefore be due to alterations in thrombus formation and/or behaviour of the vessel wall. Today, three main pathways linking ambient air pollution to cardiovascular health are being discussed, although the complex combination and interaction of mechanisms are still not fully understood.

1. Particles deposited in the pulmonary tree can alter systematic autonomic balance leading to parasympathetic nervous system withdrawal and/or sympathetic nervous system activation. These effects can be either triggered directly, by stimulating pulmonary neural reflexes or indirectly, by provoking oxidative stress and inflammation in the lung, or a combination of both. Alterations in autonomic tone can contribute to the instability of a vascular plaque or initiate cardiac arrhythmias.
2. Circulating pro-oxidative and/or pro-inflammatory mediators released from the lungs may induce a systemic chain reaction. Such mediators include cytokines (e.g. interleukin-6), acute-phase reactants (e.g. C-reactive protein and fibrinogen) and vasoactive hormones (e.g. endothelins) which may lead to endothelial dysfunction and a pro-coagulatory state with thrombus formation and promotion of atherosclerotic lesions.
3. After inhalation, UFP or soluble particle constituents may rapidly translocate from the pulmonary epithelium into the circulation and interact directly with the cardiovascular system. These small particles might not only affect the vascular endothelium and atherosclerotic plaques, but also provoke local inflammation and oxidative stress. Once in the circulation, UFP might also have direct effects on the heart and other organs.