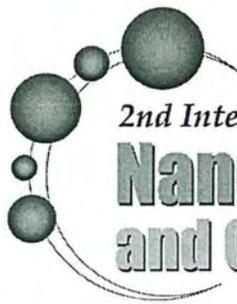


## Carbon Nanotube Exposure and Cardiovascular Outcomes

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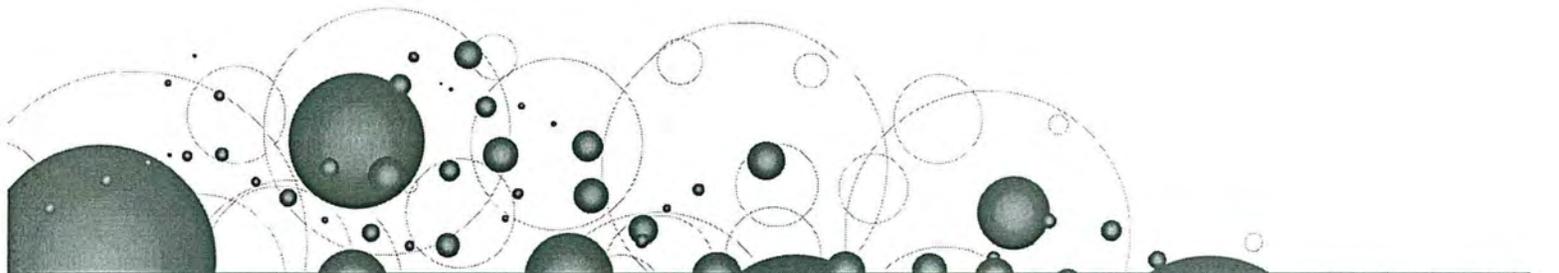
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During the past few years many epidemiological and experimental studies have found positive associations between particulate matter in air pollution and adverse cardiovascular outcomes. Although exposure to particles occurs in certain work environments, the role of these exposures in cardiovascular diseases is minimally understood. One hypothesis is that occupational exposure to inorganic ultrafine particles in addition to its pulmonary effects, may induce systemic effects of oxidative stress and inflammation which modify the atherosclerotic processes. Engineered nanosized particles, such as carbon nanotubes (CNT), are new materials of emerging technological importance in different industries. The unique physical characteristics of these particles raise concerns that they may have not only pulmonary toxicity but also systemic toxicity. In the present study, we hypothesized that CNT pulmonary exposure is associated with oxidative and inflammatory responses in the vascular system, which might be a prerequisite of atherogenesis. C57BL/6 mice were exposed to CNT in doses (0.5; 1; 2 mg/kg) by single intra-pharyngeal installation and the mice were sacrificed at different time points (1; 7; 28; 56; and 180 days) after the exposure (the experimental settings have been related to pulmonary toxicity including development of granulomas). By extra long quantitative PCR of mitochondrial (mt) DNA, we found that CNT exposure induced a dose-dependent aortic mtDNA damage, an oxidative stress dependable parameter, at day 7, 28, 56, and 180 days after exposure. Consistently, these responses were accompanied by increase production of reactive oxygen species in the aortic tissue, measured by an electron spin resonance (ESR) assay. Similar oxidative modifications were observed in the aortic tissue of ApoE<sup>-/-</sup> mice, a model of human atherosclerosis. The serum levels of IL-6, an inflammatory cytokine, involved in induction of acute phase response proteins, were increased in the CNT-exposed mice. In conclusion, CNT induces direct or indirect toxic effects which might be predisposing factors for atherogenesis.



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