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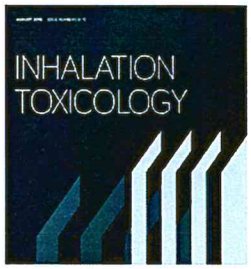

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REVIEW ARTICLE

Engineered nanoparticle exposure and cardiovascular effects: the role of a neuronal-regulated pathway

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ABSTRACT

Human and animal studies have confirmed that inhalation of particles from ambient air or occupational settings not only causes pathophysiological changes in the respiratory system, but causes cardiovascular effects as well. At an equal mass lung burden, nanoparticles are more potent in causing systemic microvascular dysfunction than fine particles of similar composition. Thus, accumulated evidence from animal studies has led to heightened concerns about the potential short- and long-term deleterious effects of inhalation of engineered nanoparticles on the cardiovascular system. This review highlights the new observations from animal studies, which document the adverse effects of pulmonary exposure to engineered nanoparticles on the cardiovascular system and elucidate the potential mechanisms involved in regulation of cardiovascular function, in particular, how the neuronal system plays a role and reacts to pulmonary nanoparticle exposure based on both *in vivo* and *in vitro* studies. In addition, this review also discusses the possible influence of altered autonomic nervous activity on preexisting cardiovascular conditions. Whether engineered nanoparticle exposure serves as a risk factor in the development of cardiovascular diseases warrants further investigation.

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KEY WORDS
Engineered nanoparticles; Occupation exposure; Autonomic activity; Cardiovascular diseases

Introduction

Incidental nanoparticles, existing on a nanoscale (with diameters of <100 nm), are generated during combustion and can agglomerate to form fine particles (with diameters of 0.1–2.5 μm) or remain as nanoparticles in ambient air. Adverse health effects linking ambient particle exposure to cardiovascular diseases have been proposed. In past decades, epidemiological studies suggest that there is a correlation between an increased level of particles in ambient air and increased incidences of cardiovascular diseases such as angina, arrhythmia, ischemic heart failure, and sudden death (Pope et al., 2004; Schwartz and Dockery, 1992). This association is stronger for $PM_{2.5}$ than for PM_{10} (particulate matter <2.5 μm or <10 μm, respectively). *In vivo* and *in vitro* animal studies also confirmed that either systemic administration or perfusion of an isolated rat heart with particles prepared from ambient air or diesel engine exhaust could alter cardiac contractility, increase left-ventricular end-diastolic pressure (LVEDP), decrease left-ventricular systolic pressure (LVSP), and trigger ventricular premature beats, depending on the type of particle (Wold et al. 2006).

Recently, the potential adverse cardiovascular effects of exposure to engineered nanoparticles have been receiving a great deal of attention from investigators. This is because engineered nanoparticles have various applications in the fields of electronics, mechanical design, environmental remediation, and biomedicine (Bar et al., 2008; Bunger, 2007; Campbell, 2013; Mazzola, 2003; Samadishadlou et al., 2018; Yan et al., 2013), which has resulted in dramatic increases in their production. Due to this increasing production, occupational respiratory exposures may, in all likelihood, be increased. Additionally, with a rapidly growing manufacturing industry, release of such materials into the air, water, and soil may also be expected to increase in the coming years (Marquis et al., 2009; Simonet and Valcarcel, 2009).

Engineered nanoparticles are similar in size to incidental nanoparticles in ambient air, being <100 nm in diameter. However, unlike incidental ambient nanoparticles, the composition and size of engineered nanoparticles are tightly controlled. Thus, engineered nanoparticles exhibit unique physical and chemical properties, which allow unique applications (Mazzola, 2003; Paull et al., 2003; Weiss, 2008). Compared to inhaled ambient nanoparticles, those unique properties of engineered nanoparticles may be more likely to cause significant adverse health impacts on the cardiovascular system. Evidence accumulated from animal studies have already indicated that exposure to engineered nanoparticles can cause pathophysiological changes not only in the respiratory system but also in the cardiovascular system (Li et al. 2016; Stone et al., 2016). For example, Li et al. (2007) reported that pharyngeal aspiration of single-walled carbon nanotubes (SWCNT) stimulate the progression of

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Engineered nanoparticle exposure and cardiovascular effects: the role of a neuronal-regulated pathway

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Engineered nanoparticle exposure and cardiovascular effects : the role of a neuronal-regulated pathway (Review)

Kan, H.[a,b], Pan, D.[a], Castranova, V.[b]

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Abstract

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Author keywords

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Engineered nanoparticle exposure and cardiovascular effects: the role of a neuronal-regulated pathway.

Kan H[1,2], Pan D[1], Castranova V[2].

Author information

Abstract

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KEYWORDS: Autonomic activity; **Cardiovascular** diseases; **Engineered** nanoparticles; Occupation **exposure**

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