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Oxidative stress risk analysis for exposure to diesel exhaust particle-induced reactive oxygen species

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Abstract

We constructed a probabilistic risk-based framework to assess the human oxidative stress (OS) risk from diesel exhaust particle (DEP)-induced reactive oxygen species. A human respiratory tract model was used to estimate DEP concentration and cumulative doses in lung regions for three occupational groupings (*driver*, *homeworker* and *student*) in northern, central, and southern Taiwan. Dose–response profiles were reconstructed in terms of the specific ratio of the reduced and oxidized forms of glutathione (GSH/GSSG ratio) for the human macrophage cell (THP-1) and human bronchial epithelial cell (BEAS-2B). The highest estimated median daily cumulative dose of DEP with 95% CI was for *driver* in northern Taiwan ($DEP_{2.5}$: 0.716 (0.443−1.197) mg and $DEP_{0.18}$: 0.584 (0.417−0.822) mg), significantly higher than that of the other settings. The *driver* in northern Taiwan setting had the highest cumulative dose–response calculated over a 2-year exposure period: 0.57 (0.41–0.76) and 0.70 (0.53–0.87) for $DEP_{2.5}$ and 0.40 (0.25−0.70) and 0.47 (0.34−0.80) for $DEP_{0.18}$, respectively, in THP-1 and BEAS-2B cells. Our results implicate that potential risks of OS from above-critical exposure to $DEP_{2.5}$ in all settings are found, whereas from $DEP_{0.18}$ in the *driver* in northern Taiwan setting is also alarming.

Keywords: Diesel exhaust particle; Reactive oxygen species; Oxidative stress; Human respiratory tract; Probabilistic risk assessment

1. Introduction

Epidemiological studies show a strong correlation between ambient particulate matter (PM) concentrations and adverse health (Donaldson et al., 1998; Cho et al., 2005; Nel, 2005; Reliene et al., 2005; Chen et al., 2006; Tovalin et al., 2006). Chio et al. (2004) identified that the vehicle emissions as the largest source of airborne PM in Taiwan, and evidence also shows that diesel vehicle emissions contribute more to the ambient PM load than gasoline vehicle emissions (Gertler, 2005). Furthermore, diesel vehicle emissions are the major source of ultrafine particulate matter (PM of diameter <0.1 μm) (Lin et al., 2005), which is more readily respirable than larger diameter particulate. Knaapen et al. (2004) found that inhalation of diesel exhaust particle (DEP) can induce epithelial cells in the lung to generate cell-damaging intracellular reactive oxygen species (ROS), and the USEPA (2002) has listed DEP as an atmospheric toxin due to the cancer and other disease effects associated with whole diesel exhaust exposure.

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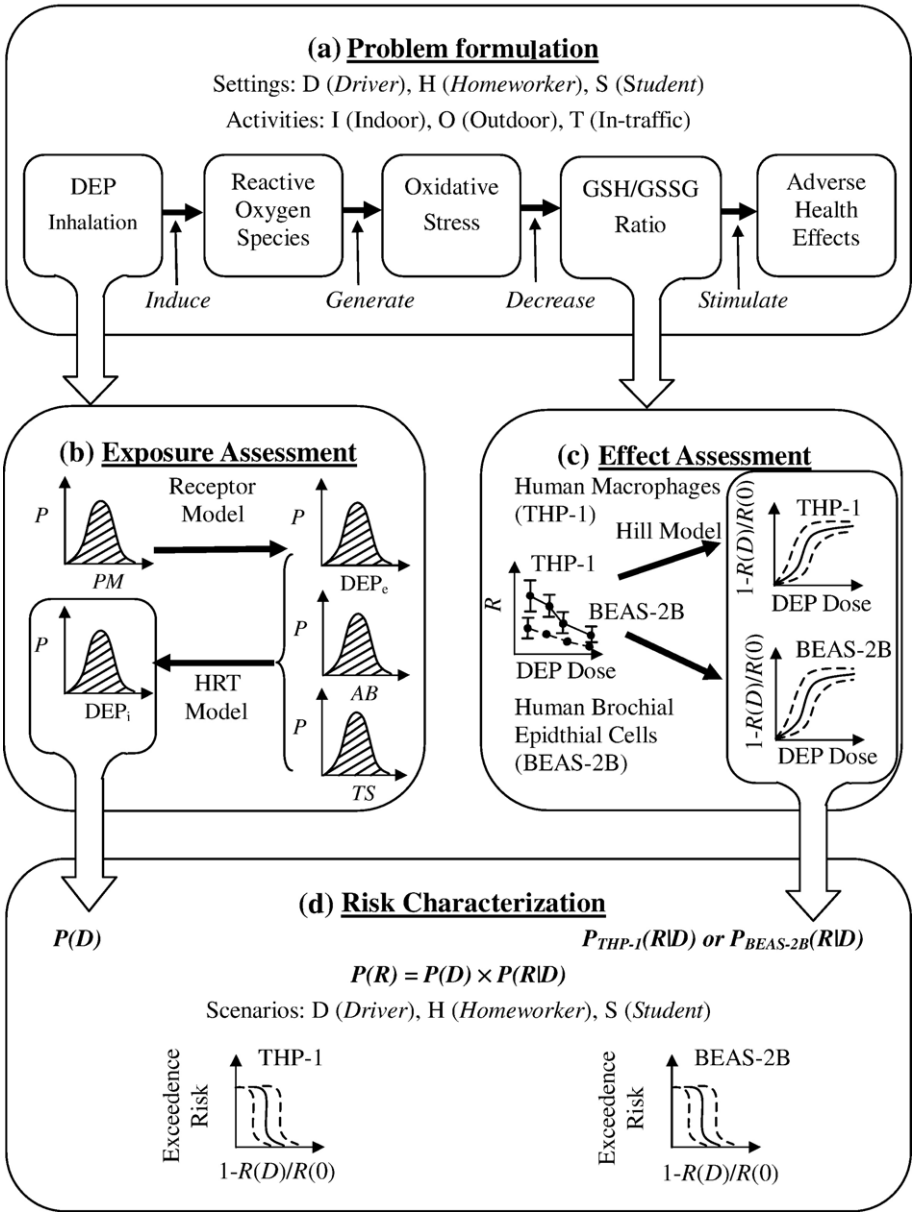

Fig. 1. Schematic diagram showing the proposed probabilistic assessment framework for oxidative stress risk for human exposed to diesel exhaust particle-induced reactive oxygen species.

ROS are broadly defined as oxygen-containing chemical species with reactive chemical properties, and include free radicals, superoxide ($O_2^{\bullet -}$), and hydroxyl radicals ($HO^{\bullet}$) (Pelicano et al., 2004). Hung and Wang (2001) measured H_2O_2 concentration as a surrogate for ROS concentration in Taipei and found it ranged from 0.016 to 0.146 and 0.026 to 0.592 nM m^{-3} in fine ($PM_{2.5}$, PM of diameter <2.5 μm) and ultrafine PM, respectively. Kao and Wang (2002) also indicated that the mean ROS concentrations in PM_1 (PM of diameter <1.0 μm) and $PM_{2.5}$ from incense smoke were 15.6±1.0 and 13.5±1.3 nmol H_2O_2 mg^{-1} of particle, respectively. ROS are also constantly generated in the cell via a variety of pathways, including enzyme-catalyzed and non-enzyme-catalyzed reactions (Pelicano et al., 2004), and can damage cellular proteins, lipid, membranes, and DNA (Iwai et al., 2000; Nel, 2005; Reliene et al., 2005; Tovalin et al., 2006). Ordinarily ROS are cleared from the cell by the action of antioxidants such as glutathione (GSH). However, Nel (2005) reported that the generation