

RESEARCH ARTICLE

Diesel exhaust, thrombus formation, and Kounis syndrome: A potential association

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Abstract

Diesel exhaust (DE) is associated with triggering acute myocardial infarction. Furthermore, DE can induce and exacerbate hypersensitivity responses, amplify the production of a variety of chemokines and cytokines, facilitate interactions between inflammatory cells and act as allergen transporter. Therefore Kounis syndrome Type I which includes patients with normal coronary arteries, and Type II variant which applies to individuals with quiescent preexisting atheromatous disease can be the result of these hypersensitivity processes.

Keywords: Allergy; diesel exhaust; myocardial infarction; Kounis syndrome

In their excellent paper Carlsen et al. (2008) did not demonstrate any prothrombotic effect of diesel exhaust (DE) in metabolic syndrome subjects, they have omitted to refer to coronary hypersensitivity syndrome (Kounis syndrome) (Kounis, 2006) as a potential cause of thrombus formation.

DE can both induce and exacerbate in vivo hypersensitivity responses and modify the immune system's handling of allergens (Bonay & Aubier, 2007). DE can act in synergy with antigens to selectively amplify the production of immunoglobulin E (IgE), Th2 cells, cytokines, and chemokines and to increase the expression of intracellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) (Riedl & Diaz-Sanchez, 2005).

Diesel exhaust particles (DEP) modify antigen presentation by the macrophages and facilitate its interaction with T-cells (Devouassoux & Brambilla, 2002) and can act as allergen "transporters" (Bartra et al., 2007), and when they measure <2.5 µm they can penetrate the airways. In vitro studies have shown that Lol p 1 and Bet v 1 allergens of *Lolium*, which is a gramineous species, and birch, respectively, bind to DEP due to their absorptive properties (Armentia et al., 2002). Such affinity has been demonstrated for other allergens such as Der p 1, Fel d, and Can f 1 (Ormstad et al., 1998). Furthermore, emissions of engines without catalytic converters and diesel engines increase the allergenic potential of *Parietaria* pollen compared with emissions with catalytic converters (D'Amato, 2000).

Other experimental studies have shown a direct link between DE-induced histamine release and peripheral thrombotic events (Nemmar et al., 2004). According to these studies, pharmacological stabilization of mast cells with dexamethasone or sodium cromoglycate managed to abrogate DE-induced late thrombotic events, thus confirming hypersensitivity involvement in these processes.

Declaration of interest: The authors report no financial conflicts of interest.

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