

Air Pollution from Diesel Particles and Chronic Obstruction Pulmonary Disease - CT Scan Study

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Abstract

Health problems have been observed in rabbits at low DEP concentration. The subject (animal) showed signs of chronic obstruction pulmonary disease (COPD). CT scan of the biopsy reveals significant emphysematous lesions. DEP grain causes inflammation of lung tissues around sites of deposition. Observations of tissue after formalin fixation reveal brown spots around the points of impact. It also reveals the double organic and inorganic aspect of DEPs. The organic compounds dissolve through slow diffusion in the lung physiological fluid while the inorganic part is partly handled by macrophages (phagocytosis).

Keywords: Diesel Particulate, air pollution, Lung, CT Scan, COPD

Introduction

Diesel exhaust gases have been shown to be carcinogenic (OMS. 2012, Guo et al. 2004; Boffetta et al.2001). The air polluting particles (INC. 2013; Wargner and Rossi 2006, Black. 2005) induce, exacerbate and lead to invalidating diseases (Loomos et al. 2013). The induced chronic obstructive

pulmonary disease (COPD) is the leading cause of death worldwide (Atsou et al. 2012). Epidemiologic data show that the prevalence of this disease suggests, to a large extent, exposure to risk factors (Halbert et al. 2006). And because of its high prevalence and disabling potential, it results in huge economic and social costs (Wouters. 2003). COPD is a progressive-onset disease (GOLD. 2011). It is characterized by an obstruction, not completely reversible and often progressive. The disease is associated with an abnormal inflammatory response of bronchi to particles, harmful substances or gas (Gordon et al. 1992). The high levels of air pollution affect lung function; there is evidence that air pollution by diesel engine particles (DEP) is associated with a decline in respiratory function (Abbey et al. 1998).

After inhalation of air polluted by DEP, particles were observed in the lungs, liver, spleen and kidney of laboratory animals. In alveoli, insoluble particles are engulfed by macrophages whereas ultrafine particles can hardly be phagocytized. The accumulation is proportional to exposure. The reaction with the alveolar epithelial cells is demonstrated; the ultrafine particle passes into the bloodstream. These physicochemical mechanisms may be responsible for cardiovascular and nervous diseases (Oberdorster et al. 1990).

Recent studies (INRS. 2005) based on electron microscopic analysis of biopsies of human lung exposed to both high particulate pollution (PM10: 66 g/m³) and low particulate pollution (PM10: 14 mg/m³) showed that the former contained about 10 times more particles than the latter. And 96% of all the particulate matter was found to be fine carbon particles probably related to combustion processes (road traffic). The fine and ultrafine particles cross biological barriers. The issue concerning the translocation in humans of ultrafine particles through biological barriers has been raised by studies with 20 nm carbon nanoparticles, marked with Technetium 99. One of those studies shows a rapid translocation into the blood and a significant accumulation in the liver (Kimber. 1998). But, if taken into account the studies carried out in animals (Lafon. 1993), it seems highly probable that the ultrafine particles can pass through the epithelial barrier into the pulmonary interstitium. They would also be able to cross the vessel wall, enter the bloodstream and thereby distributed in the body. Their accumulation in target organs was investigated using nanoparticles. The liver is the major accumulation site while smaller amounts were found in the heart and the kidney (Wolff et al. 1980). Moreover, the crossing of the hematoencephalic and placental barriers is suspected but not sufficiently supported by evidence (INRS. 2005). Therefore, the issue of such a transfer arises for ultrafine atmospheric particles. Disturbing observations of chronic inflammation of the brain and accelerated process of neurodegenerative diseases in dogs have been associated with high particulate pollution in cities (Oberdorster et al. 1995). In mice, the intra-tracheal administration of 200 micrograms of black carbon (14 nm) induced

inflammation associated with a vascular growth factor production, which increases the permeability of the alveolar capillary membrane (Dill et al. 2004). Furthermore, an increased risk of the cervix (Chang et al. 2005), bladder (Olsson et al. 2011; Tyler et al. 1988), ovary (Nerrière et al. 2005; Turner et al. 2006), esophagus (Afsset. 2006), gastric (Ji et al. 2005) and kidney cancers (Nerrière et al. 2005; Laden et al. 2006; Man et al. 1988; Carvalheiro et al. 1995; Tyler et al. 1988) has been reported in the literature. The nature and intensity of the reaction to an irritant agent depend on the physical properties of the gas or aerosol, its concentration, exposure duration and other variables such as temperature or ambient humidity and the presence of pathogens or other gases (McMillan et al. 1982). Host specific factors; such as age (Lyng et al. 1997), exposure history (Kogevinas et al. 2003), the concentration of antioxidants (Sjodahl et al. 2007) and the presence of infection; can all have an influence on the observed pathological disorders. This multiplicity of factors makes it difficult to carry out a systematic study of pathogenic respiratory irritants.

II. Materials and methods

Laboratory studies have played an important role in recent years in understanding the biological effects of atmospheric particulate pollutants.

Controlled filtered air chamber S30 LB TSA.

Bred rabbit from battery cage No. V5 / 2015 TSA.

Dissection: In the present study, a rabbit (Fig.1) is an ideal animal model for experimentation given his thick, spongy and voluminous lungs which offer adequate possibilities to perform biopsies and core samplings. This allows assessing the evolution of DEPs in the lung tissue.

Physiological water, ethanol, methanol, formaldehyde, and experimental accessories (wind tunnel, medical air) provided by Technologie de la santé Algérie (TSA)



Figure 1. Bred rabbit

III. Results and discussion

Diesel exhaust particulates (DEPs) represent up to 80% of road traffic related particulates. Their composition consists of elemental carbon particles (10 - 100 nm) onto which is adsorbed a large number of organic compounds through a nucleation phenomenon (Gordon et al. 1992) (Fig. 2).

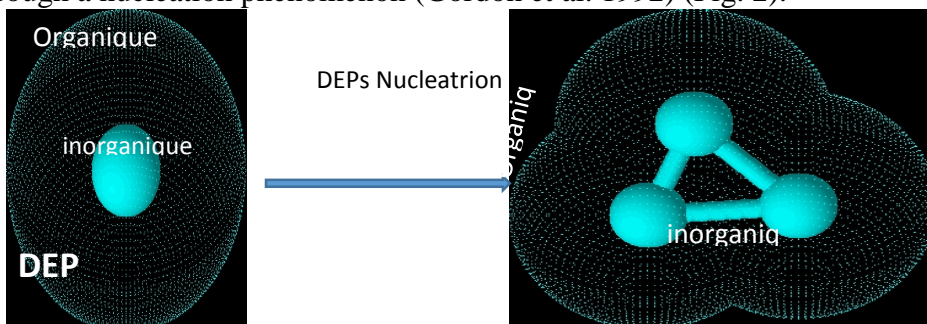


Figure 2. Schematic structure of DEP nucleation

Collected and condensed diesel exhaust gases precipitate as a black powder. The dislocation of the grain by a strong acid solution shows different aspects: Ultrafine 10-100 nm; fine 100-1000; and > 1 µm (Dill et al. 2004).



Figure 3. Control lung



Figure 4. Emphysematous lung (DEP emphysema)



Figure 5. Cross-section of the emphysematous lung (post-DEP emphysema)

A completely unstressed rabbit, living in normal psychological and physiological conditions was used. The pollutant gases containing DEPs were injected in the controlled atmosphere chamber. The penetration of particles in the respiratory system at normal speed induces deposition of the particle along the respiratory tract in agreement with studies reported by Oberdorster G et al. (1990).

Figure 3 shows the aspect of the control organ "Lung". The biopsy from the post DEP lung is performed just after the first signs of chronic pulmonary obstructive disease (appearance of cough and breathing difficulties after eight weeks, 10 mg/m³, 12 hours/day). The microscopic pictures (Fig.

5) represent structures that are an accumulation of black spots. Fixation of cells in formalin for 24 hours reveals DEP grain surrounded by a necrotic area in the form of dark brown spots, which are commonly seen during inflammatory diseases (Hervé-Bazin. 2007) accompanied by cell lysis (embedment of grains inside cells).

Figure 6 shows a scan of the bronchi and bronchioles (Oberdorster et al. 2005) while, figure 7 shows polluted bronchi and bronchioles; the distribution of DEPs in the lungs can clearly be seen. The scanner (Fig. 8) reveals neatly the DEP grain type Pi10 stopped by the junction of two bronchioles. The DEP partly disintegrates and dissolves into the physiological fluid of the lungs (Fig. 9). The remaining solid part is phagocytized as reported by the Bordeaux team. Similar results have been obtained in our laboratory by biopsy (Fig. 10).

The aspect of polluted cells of alveoli is observed (Fig. 11). The deterioration is clearly caused by DEPs and the host of chemical compounds enveloping them.



Figure 6. Visiographie of bronchi and bronchioles

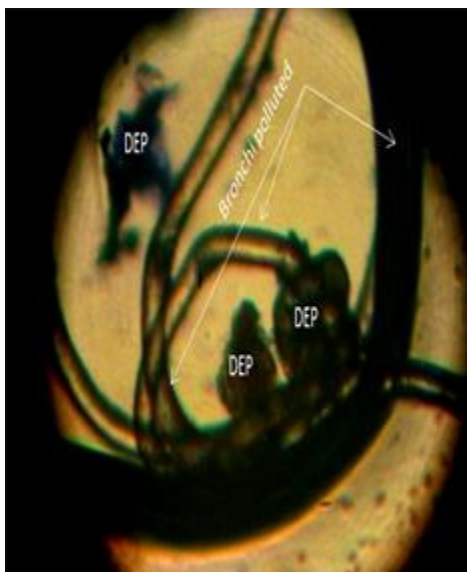


Figure 7. Visiographie of Bronchi polluted by DEPs

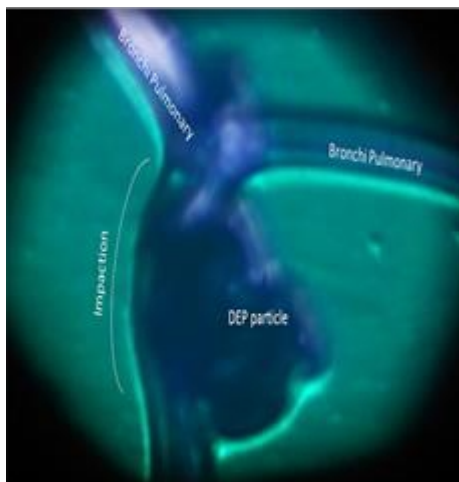


Figure 8. DEPs impaction of the bronchi and bronchioles

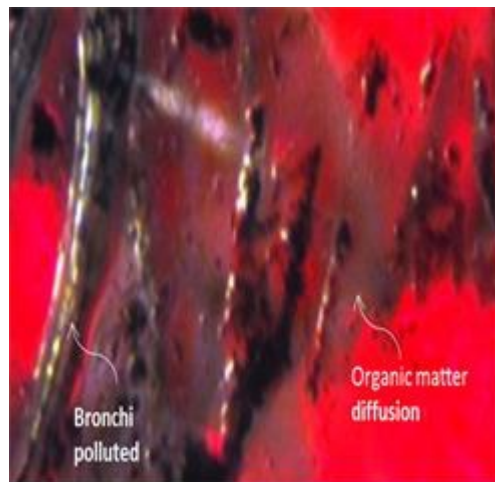


Figure 9. Visiographie of organic materials diffused in the physiological medium of the lungs

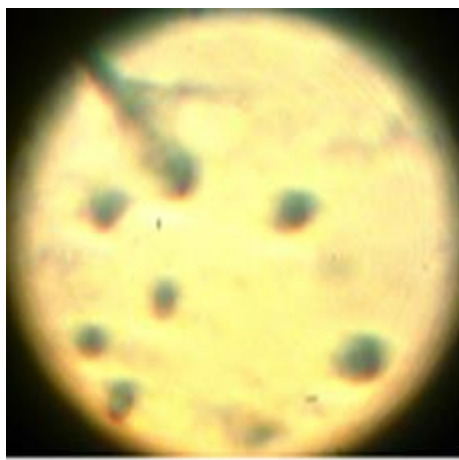


Figure 10. CT scan of DEPs polluted lung samples

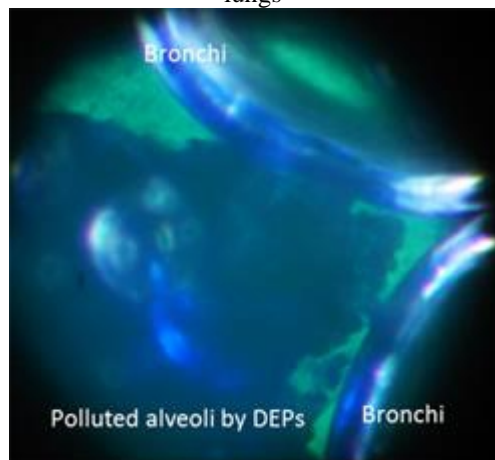


Figure 11. Appearance of polluted cells x 400

IV. Conclusion

Respiratory symptoms such as productive cough is a frequent consequence of exposure to DEP dusts and most studies show that their frequency increased compared to unexposed control groups. The incidence of respiratory symptoms rises with successive exposure to DEPs. Symptoms seem to be associated with impaired lung function. The exposure to dusts contributes to disease processes; it has been underlined that mortality from emphysema increases with increasing exposure to DEPs. Anatomic pathology investigations point to increased emphysematous lesions in polluted rabbits compared to control groups. Additionally, it was observed that the significance

of emphysema was related to the amount of particles in the lungs. It is currently considered that emphysema results from tissue destruction rather than from distension or expansion. It points to the likelihood of a relationship with the duration of exposure. This assessment is confirmed by recent findings that established an association between mortality from bronchitis, emphysema and dusts exposure. These agents and substances may be harmful in many ways, and the extent of damage they provoke is dependent on the level of exposure and on the biochemical properties of the inhaled agent.

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