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# Exposure to polycyclic aromatic hydrocarbons in atmospheric $PM_{1.0}$ of urban environments: Carcinogenic and mutagenic respiratory health risk by age groups☆

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ABSTRACT

We investigated the carcinogenic and mutagenic respiratory health risks related to the exposure to atmospheric PAHs in an urban area. Our study focused in the association of these pollutants and their possible effect in human health, principally respiratory and circulatory diseases. Also, we determined a relationship between the inhalation risk of PAHs and meteorological conditions. We validated the hypothesis that in winter PAHs with high molecular weight associated to submicron particles ($PM_1$) may increase exposure risk, especially for respiratory diseases, bronchitis and pneumonia diseases. Moreover, in our study we verified the relationship between diseases and several carcinogenic PAHs (Ind, BbkF, DahA, BaP, and BghiP). These individual PAHs contributed the most to the potential risk of exposure for inhalation of $PM_{1.0}$. Even at lower ambient concentrations of BaP and DahA in comparison with individual concentrations of other PAHs associated to $PM_{1.0}$. Mainly, research suggests to include carcinogenic and mutagenic PAHs in future studies of environmental health risk due to their capacity to associate to $PM_{10}$. Such carcinogenic and mutagenic PAHs are likely to provide the majority of the human exposure, since they originate from dense traffic urban areas were humans congregate.



## 1. Introduction

Particulate matter (PM) is one of the major pollutants affecting Human's health, especially, PM's fine and ultrafine fraction that may absorb greater concentrations of toxic compounds like the polycyclic aromatic hydrocarbons (PAHs) (Agudelo-Castañeda et al., 2015). PAHs are one of the most important atmospheric pollutants that are of human health concern, owing to their high carcinogenic potential and the ubiquity of their sources (Wang et al., 2013; Dabestani and Ivanov, 1999). Majorly, PAHs found in the environment come from incomplete combustion of carbonaceous materials. PAHs sources include: biomass burning, coal and petroleum combustion, coke and metal production (Zhang and Tao, 2009). Nowadays, the emissions of these pollutants to the atmosphere has increased with the urban and industrial development of cities. These pollutants are widely distributed in the atmosphere. PAHs have received a particular attention for the last decades attributed to their toxic, mutagenic and carcinogenic properties, and their persistence in the environment (Biache et al., 2014; Kim et al., 2013). As a result, 16 PAHs are listed as priority pollutants by the United States Environmental Protection Agency (USEPA) and the World Health Organization (WHO) established an air quality standard of 1 ng/m$^3$ of Benzo[*a*]pyrene.

PM1.0's pollution source in urban ambient air is largely low−mass aerosol from motor vehicle combustion exhausts (Verma et al., 2014). These emissions contain particles were over 90% of

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the number concentration, belongs to the fraction <1.0 μm, which can be inhaled deeply into the lung. Moreover, typically, highly carcinogenic PAHs in exhaust are mainly associated with particles emitted from mobile sources (Li et al., 2014). Thus, the adverse health effects may be enhanced by the physical and chemical characteristics of these submicron atmospheric particles (<1.0 μm). Therefore, PAHs from vehicle exhaust in urban areas and their related adverse health effects have become an issue of concern. In Brazil, despite advances in air pollution control, it remains difficult to obtain data regarding the environmental concentrations of atmospheric PAHs for environmental risk assessments (Franco et al., 2008).

Several authors have linked the effects of short- and long-term particle exposure with allergies, respiratory and cardiovascular diseases, and premature mortality (Sun et al., 2015; Bozlaker et al., 2014; WHO, 2013). Braga et al. (2001) suggested that atmospheric particles are facilitators and propagators of infectious diseases, especially pneumonia. Other authors stated associations between PM exposure and decreased lung function, worsening of respiratory diseases like asthma, premature death and increased hospital admissions in the elderly and individuals with cardiopulmonary diseases (Buczyńska et al., 2014). Several authors from the US and Europe assessed the relationship between asthma and air pollution (Atkinson et al., 2001; Medina-Ramón et al., 2006; Wilson et al., 2005; Tao et al., 2014). Moreover, other authors have associated high prenatal exposure to PAHs with childhood asthma (Perera and Herbstman, 2011; Perera et al., 2006). Evidence suggests that the chemical composition and physical properties of PM may contribute significantly to these adverse health effects. Experts worldwide have seen a relationship between hospital admissions attributable to asthma and fossil-fuel-generated particulate matter, independently of the size of the particles and the population's age (Arbex et al., 2012). Children, elders and patients suffering from asthma and/or with cardio-respiratory diseases are more susceptible to the effects of pollution. Moreover, emerging epidemiological studies evidenced an association between cardiorespiratory health and short-term exposures to ultrafine particles. Another evident association is between short- and long-term $PM_{2.5}$ (<2.5 μm) exposure with mortality and morbidity (WHO, 2013). Such effects are due to the deep deposit of submicron particulate matter into the lower respiratory tract (Kreyling et al., 2006). This deep deposit facilitates the submicron particular matter to avoid the tissues' natural mechanisms of clearance, including the subfraction $PM_{1.0}$ that consists of inhalable particles that form active oxides into the lungs (Kok et al., 2006). These are the most cytotoxic ambient particles (Massolo et al., 2002). As a result, there is a long–term retention of $PM_{1.0}$ (<1.0 μm) and their absorbed chemicals causing oxidative damage and an increase in the risk of toxicity.

Additionally, researches have recognized atmospheric PAHs inhalation exposure risk and ambient air pollution as a major cause of lung cancer (Li et al., 2014). The impact of PAHs on human health depend mainly on the length and route of exposure, the amount or concentration of PAHs one is exposed to, as well as the relative toxicity of the PAHs. Health effects from short- and long-term or chronic exposure to PAHs may include: nausea, inflammation, decreased immune function, cataracts, kidney and liver damage (e.g. jaundice), breathing problems, asthma-like symptoms, and lung function abnormalities, DNA and liver damage, gene mutation cell damaging and cardiopulmonary mortality (Franco et al., 2008; Kim et al., 2013). Furthermore, epidemiological studies have utilized PAH associated with PM as their molecular basis in populations exposed to environment (Farmer et al., 2003). However, their mechanisms of action and the health effects of individual PAHs are not exactly alike; there are limitations to quantify the exposure risk (Chen and Liao, 2006). Although atmospheric PAHs exist at low concentrations, epidemiologic studies have linked long-term low-level PAH exposure with various health outcomes, including cancers among adult populations (Liu et al., 2016; Pieterse et al., 2013; Tao et al., 2014). In addition, PAHs mixtures may be even more carcinogenic to humans than individual PAHs (Amarillo et al., 2014). Thus, the International Agency of Research on Cancer (IARC) and the WHO have raised environmental concerns about atmospheric particles affecting air quality and humans health (IARC, 2010).

In order to obtain an accurate assessment of the potential risk of exposure to a complex mixture of PAHs, several approaches were developed using toxic equivalency factors (TEF) based on BaP (Jung et al., 2010). Approaches included the carcinogenic equivalency factors and the individual mutagenicity of PAH relative to BaP. Durant et al. (1999) proposed to use the mutagenic equivalency factor (MEF); however, Young et al. (2010) showed that the Mutagenic activity was not uniformly associated with cancer. They found that the mutagenic activity might be implied to non-cancerous adverse health effects, like pulmonary diseases. Other researchers have combined TEF and MEF values with measured air concentrations to calculate carcinogenic equivalents (TEQ, expressed in ng/$m^3$) and mutagenic equivalents (MEQ, expressed in ng/$m^3$) in environmental samples. Although, there is a lack of studies exploring the health risks associated with exposure to atmospheric PAHs on the population level (Liu et al., 2016). In Brazil, particularly in the studied area, there is limited published data about the associations between PAHs in $PM_{1.0}$ and diseases. Despite the importance, the available data on ambient PAH concentrations in many regions and potential exposure are not sufficient to fully assess the PAH effects on human health (Rogula-Kozłowska et al., 2013). Probably, due to the insufficient mass collection in their sampling from urban ambient aerosol (Verma et al., 2014), costly measurements of ambient air PAHs, sparse information, between others.

Given that, this study aims to contribute to this growing area of research by exploring the PAHs associated to $PM_{1.0}$ in urban areas and their probably human health effects, principally respiratory and circulatory diseases. Thus, the objective of this research was to determine the inhalation risk of PAHs and the relation between atmospheric pollutants (PAHs and $PM_{1.0}$) exposure with human health (respiratory and cardiovascular diseases) in the urban area of the metropolitan area of Porto Alegre, RS, Brazil.

## 2. Study area

We selected for this study the Metropolitan Area of Porto Alegre (MAPA) located at 29°30′S–30°30′S/50°25′W–51°55′W in the east of the state of Rio Grande do Sul, Brazil. According to the Brazilian Institute of Geography and Statistics (IBGE, 2016), this region comprises an area of 9800 $km^2$, representing 3.76% of the total area of the state, and it has a population of 3,979,561 inhabitants. The MAPA is the most urbanized area of the state. The study area is characterized by different types of stationary sources, however, is estimated that the most significant contribution are mobile sources due to the large number of vehicles in circulation in the region. More details on the study area can be found in studies reported by Teixeira et al. (2012a) and Agudelo-Castañeda and Teixeira (2014).

Although gasoline is the predominant fuel used by the fleet in the region (84.98%) in comparison to diesel (6.67%), the latter one has a level of 500 ppm of sulfur and with just 5% of biodiesel (Mattiuzi et al., 2012). Thus, the environmental impact caused by this source in the air is elevated. Teixeira et al. (2012b) reported the influence of mobile sources, by showing that from the total emissions originated from mobile sources within the MAPA, diesel

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