Atmospheric Environment 60 (2012) 375–382

Contents lists available at SciVerse ScienceDirect

Atmospheric Environment

journal homepage: www.elsevier.com/locate/atmosenv

Carcinogenic and mutagenic risk associated to airborne particle-phase polycyclic aromatic hydrocarbons: A source apportionment

Mauro Masiol, Angelika Hofer, Stefania Squizzato, Rossano Piazza, Giancarlo Rampazzo, Bruno Pavoni*

Dipartimento di Scienze Ambientali, Informatica e Statistica, Università Ca' Foscari Venezia, Dorsoduro 2137, 30123 Venice, Italy

HIGHLIGHTS

- Eleven particle-bound PAHs were monitored for one year in an urban site.
- A receptor model was used to detect the main PM emission sources.
- Sources of carcinogenic and mutagenic effects were also identified.
- A risk assessment was made for each identified source.
- Applicable to other areas, the approach provides information for pollution control.

ARTICLE INFO

Article history:
Received 23 April 2012
Received in revised form
12 June 2012
Accepted 25 June 2012

Keywords:
PAHs
Aerosol
Cancer risk assessment
Toxic equivalent factors
Mutagenic equivalent factors

ABSTRACT

Conventional risk assessment studies provide no detailed information about the role of specific sources determining the total carcinogenic and mutagenic potencies of PAH mixtures on humans health. In this study, the main emission sources of 11 particle-phase PAHs listed as carcinogenic and mutagenic agents by the IARC were identified by a risk apportionment method. The contribution of sources to the total concentration of PAHs in the study area was also quantified. A receptor model based on factor and multiple linear regression analyses was applied to estimate the source-specific risk associated to PAH inhalation in an urban background area of a large city (Venice-Mestre, Northern Italy). The proposed approach has discriminated the sources of mutagenic and carcinogenic congeners and their role in determining a serious hazard for human health. Results, interpreted on the basis of seasonal variations and atmospheric conditions, have shown that even though domestic heating is the main source of total PAHs in winter, a background pollution including traffic mainly accounts for the carcinogenic and mutagenic risk during the whole year. The findings of this work and the approach used can be easily applied to other geographic areas and provide useful information for local and regional air pollution control strategies.

1. Introduction

Several epidemiological studies (Pope et al., 2002; Pope and Dockery, 2006) have shown a significant correlation between some adverse health effects on humans and the exposure to airborne fine particulate matter ($PM_{2.5}$). However, some chemical, physical and biological properties enhance these effects (Künzli et al., 2005). In particular, particles carried into the respiratory tract can be composed of, or can supply absorption surface for, some hazardous substances associated with toxic, carcinogenic and/or mutagenic effects. Among these, Polycyclic Aromatic Hydrocarbons (PAHs) have received particular care (WHO, 2000; Armstrong et al., 2004; IARC, 2010) because of their wide distribution in the atmosphere (Ravindra et al., 2008; Zhang and Tao, 2009).

PAHs are mainly produced by incomplete combustion and pyrolysis of organic material (Manahan, 2009) and their occurrence in urban atmospheres is largely the result of anthropogenic emissions, such as mobile emissions (vehicular, shipping and flying), domestic heating, oil refining, waste incineration, industrial activities, asphalt production, agricultural burning of biomass, etc. (Marchand et al., 2004; Ravindra et al., 2008). Generally, PAHs occur in the atmosphere as complex mixtures of congeners with different molecular weights: lighter PAHs (2–3 aromatic rings) are present almost exclusively in the vapor-phase, whereas PAHs with higher molecular weights (≥4 rings) are almost totally adsorbed on the

* Corresponding author. Tel.: +39 041 234 8522; fax: +39 041 234 8582.
E-mail address: brown@unive.it (B. Pavoni).

1352-2310/$ – see front matter © 2012 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.atmosenv.2012.06.073

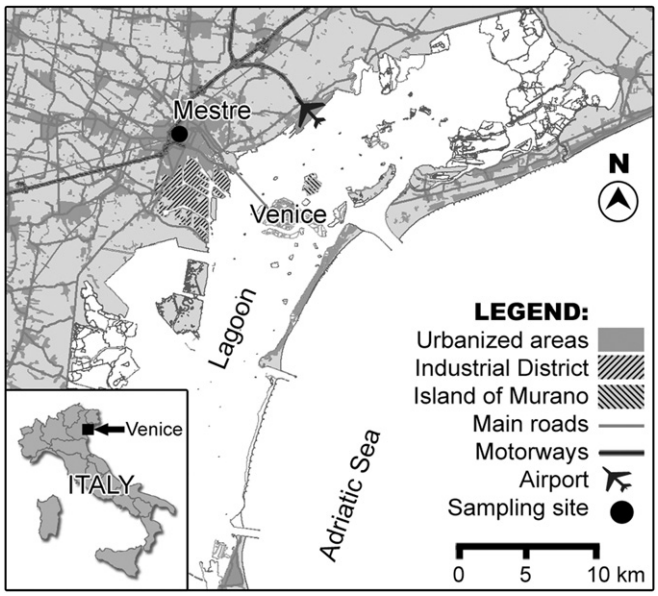

Fig. 1. Study area.

particulate matter. As many of the heaviest PAHs are carcinogenic (IARC, 2010) and were, in particular, categorized as human carcinogens (class 1), probable (class 2A) or possible human carcinogens (class 2B) by the International Agency for Research on Cancer (IARC, 2012), the quantification of particle-phase PAHs is a key tool to assess health risks for humans exposed to airborne pollutants.

As every congener has its individual toxicity, several approaches have been developed to discriminate individual contributions in a PAH mixture. In conventional risk assessments, the carcinogenic potency of a given congener was estimated on the basis of its Benzo(*a*)pyrene (BaP) carcinogenic equivalent concentration (BaP_{TEQ}), assessed by multiplying the ambient concentration of a congener by its Toxic Equivalency Factor (TEF) (Petry et al., 1996; Boström et al., 2002; USEPA, 2005). On the basis of these TEF-adjusted concentrations, the total carcinogenic potency (ΣBaP_{TEQ}) was estimated as the sum of BaP_{TEQ} for all the congeners in a mixture, assuming that the carcinogenic effects of different congeners are additive (WHO, 2000; Vu et al., 2011). Similarly, just with the replacement of TEF with MEF (Mutagenic Equivalency Factors), the mutagenicity related to BaP (BaP_{MEQ}) was calculated as well. Despite the mutagenic activities are associated to some adverse health effects, i.e. pulmonary diseases (DeMarini et al., 2004), they are not uniformly related to cancer (Zeiger, 2001). Many studies have attempted to estimate the carcinogenic potency of PAHs (Bari et al., 2010; Delgado-Saborit et al., 2011; Jia et al., 2011), but less attention was given to mutagenicity.

Though these studies have highlighted the hazard associated to the exposure to PAH mixtures, risk assessments give still limited information about the carcinogenic and mutagenic potencies related to specific emission sources. However, in view of the mitigation of air pollution, the knowledge of emission processes and sources that mainly contribute to the carcinogenic and mutagenic potencies represents a key objective. Moreover, as most of the specific sources have unsteady emissions in time and atmospheric conditions vary seasonally, the impact of some sources should be assessed and interpreted on a seasonal basis.

This study proposes the use of a receptor model to identify and quantify the source-specific mass concentrations of emitted PAHs. Source-specific carcinogenic and mutagenic potencies were also assessed to estimate their potential impact on human health. The interpretation of the results on a seasonal basis gives further information about the processes that may contribute to the carcinogenic and mutagenic hazard of particle-phase PAHs. In addition, a lifetime lung cancer assessment in relation to the different emission scenarios was carried out.

2. Materials and methods

2.1. Experimental data collection: study area

$PM_{2.5}$ samples were collected in Venice-Mestre (Fig. 1), a city located in the Eastern part of the Po Valley (Northern Italy), with a population exceeding 270,000 inhabitants. The nearby industrial district is one of the most important in Italy, including chemical and power plants, waste incinerators, an oil-refinery, and other industrial activities. Important atmospheric emissions originate from several heavy traffic roads and an expressway, the artistic glass factories on the island of Murano, an international airport, commercial, industrial and cruise terminals (Rampazzo et al., 2008a,b; Masiol et al., 2010).

2.2. Sampling strategy

A one year sampling campaign (2009–2010) was conducted in different sites of the Venice-Mestre area with the aim of characterizing the fine particulate matter and the relationship with both local and regional sources. To study the effects of some well known carcinogenic and mutagenic compounds, the most representative sampling site was an Urban BackGround site (UBG), with a high density residential zone at a distance of ~50 m from frequently congested roads and an expressway (Fig. 1). In this site, a total of 119 $PM_{2.5}$ samples were collected during four sampling periods selected to be representative of all seasons: spring (March 2009), summer (June–July 2009), autumn (October 2009) and winter (December 2009–January 2010). Their meteorological conditions are summarized in Table 1. Glass fiber filters (PALL Corp., type A/E, Ø 47 mm) were exposed for 24 h in low volume sequential automatic samplers (PM TCR TECORA and HYDRA FAI, Italy) set according to EN 14907 standard (2.3 $m^3\ h^{-1}$), and afterwards stored at −20 °C until chemical processing to avoid PAH losses and sample degradation. The concentrations of gaseous pollutants (SO_2, NO_2 and CO) were measured by ARPAV (Environmental Protection Agency of Veneto Region), meteorological conditions were recorded by Ente della Zona Industriale di Porto Marghera.

2.3. Sample analyses

A half filter was ultrasonically extracted for three times in dichloromethane (>99.9% SpS Romil, UK) for 15 min. The extracts were reduced to few milliliters under a gentle stream of nitrogen (≥99.9% v, SIAD, Italy) and cleaned up by column chromatography using 4 g silica gel 60 (Macherey–Nagel, Germany) and 0.5 g anhydrous sodium sulfate (ACS ≥99.0%, Sigma–Aldrich, USA). Samples were eluted by a mixture of *n*-hexane (>95% SpS Romil, UK) and dichloromethane 3:2 (v/v). The final extracts were reduced to a volume of ~0.5 mL under a nitrogen flow and finally transferred in amber vials kept at −20 °C until analysis. To attain quantifiable concentrations of PAH congeners for summer samples, five filters were combined and therefore an average value was obtained.

Eleven PAHs listed by the IARC as class 1, class 2A and 2B carcinogens (Benz(*a*)anthracene (BaA), Chrysene (Chry), Benzo(*b*)fluoranthene (BbF), Benzo(*k*)fluoranthene (BkF), Benzo(*e*)pyrene (BeP), BaP, Indeno(1,2,3-*c*,*d*)pyrene (IP), Dibenzo(*a*,*h*)anthracene (DBahA), Benzo(*g*,*h*,*i*)perylene (BghiP)), mainly associated to the