Air Qual Atmos Health (2010) 3:225–234
DOI 10.1007/s11869-010-0074-7

PAHs sources contribution to the air quality of an office environment: experimental results and receptor model (PMF) application

Dikaia E. Saraga · Thomas E. Maggos · Athanasios Sfetsos · Evangelos I. Tolis · Spyros Andronopoulos · John G. Bartzis · Christos Vasilakos

Received: 16 October 2009 / Accepted: 4 May 2010 / Published online: 4 June 2010

Abstract The objectives of this study were to measure the concentrations of PAHs (polycyclic aromatic hydrocarbons) in three particle fractions [particulate matter (PM) <1 μm in diameter (PM1), PM <2.5 μm in diameter (PM2.5) and the respirable fraction (<4 μm in diameter)] in the air of offices in which smoking was allowed and forbidden, respectively, and to identify the potential sources by applying the Positive Matrix Factorization (PMF) model. We sampled the indoor air of both environments during 24-h periods for PM1 and PM2.5 and conducted personal exposure measurements for the respirable PM fraction during working hours. The measurements indicated a clear difference in the levels of carcinogenic PAHs (PAHcancer) in the two office environments. In the smokers' office, PAHcancer levels fell by >70% during the weekend relative to working days, implying the importance of smoking and dust resuspension sources. The PMF model identified four contributory factors–sources: smoking, dust resuspension and two different vehicle-related sources. For 15 PAHs, the same factor made the largest contribution to both the PM1 and PM2.5 fractions, implying a common origin and further supporting the validity of the proposed approach. For the majority of the carcinogenic PAHs, smoking and dust resuspension made the strongest contribution (>90%) to both the PM1 and PM2.5 fractions. Although our PMF analysis confirmed the well-known contribution of smoking and traffic-related sources to levels of PAHs, the identification of dust resuspension as a separate source of PAHs is of great interest and requires further study.

Keywords Office environment · PAHs · Particulate matter · PMF · Smoking

D. E. Saraga · T. E. Maggos · A. Sfetsos · S. Andronopoulos · C. Vasilakos
Environmental Research Laboratory Institute of Nuclear Technology-Radiation Protection, NCRS "Demokritos", 15310 Aghia Paraskevi, Athens, Attica, Greece

D. E. Saraga (✉) · E. I. Tolis · J. G. Bartzis
Department of Mechanical Engineering, University of West Macedonia, Sialvera & Bakola Street, 50100 Kozani, Greece
e-mail: dsaraga@ipta.demokritos.gr

Introduction

Numerous studies have underlined the role of good indoor air quality, since an average person spends more than 80% of the day in an indoor environment (Robinson and Nelson 1995). Indoor air quality depends on several factors, including outdoor area, indoor sources, ventilation, building design, among others (Hameri et al. 2003). Common indoor particle sources in an occupied building include smoking, combustion sources and resuspension from carpets, clothes, etc., while outdoor particles originate from both mobile and stationary (natural or anthropogenic) sources.

Polycyclic aromatic hydrocarbons (PAHs) are ubiquitous environmental pollutants that are formed during combustion processes of carbonaceous materials at high temperatures. The combustion sources include emissions from automobiles, industrial processes, domestic heating systems, waste incineration facilities and several natural sources, such as forest fires. Sources of PAHs have also been identified in indoor air (natural gas heating and cooking, emissions from wood or electric stoves, smoking). Human exposure to PAHs in the indoor environment has received increasing attention during the last decades (Rogge et al. 1993; Chao et al. 2002; Naumova et al. 2002; Ohura et al. 2004; Li et al. 2005; Lu and Zhu 2007), particularly in

terms of smoking, and many studies have focused on the environmental tobacco smoke (ETS) fingerprint and its impact on occupants' health (Sheldon et al. 1993;U.S. Department of Health and Human Services 2006; Lu and Zhu 2007). Mannino and Orecchio (2008) reported levels of PAHs in indoor dust sampled from smoking-permitted households that were 2- to 16-fold higher than these in homes where smoking was forbidden, particularly the PAHs acenaphthylene, fluorine, fluoranthene and pyrene. Lu and Zhu (2007) found that tobacco smoke was the source for more than 80% of the benzo(a)pyrene present in the indoor air of the residences in their study.

Elevated levels of PAHs have also been found in dust that accumulated in a building (Chuang et al. 1995; Mannino and Orecchio 2008). As concluded by Mannino and Orecchio (2008), PAH concentrations in dust collected from indoor environments are dependent on both the type of anthropogenic outdoor sources of PAHs and the intensity of activities carried out in the indoor environments. Another source of PAHs source—especially in an office environment—is the dust from computers (Ren et al. 2006), which accumulates on the cooling fans of computer boxes and then re-emitted to the indoor environment. Ren et al. (2006) suggested that the heated plastic material of PC boards is a primary emission source of PAH deposited in dust samples. It would appear, therefore, that although much has been learned, the current state of our knowledge on the behavior of indoor PAHs is still limited, largely due to the complex nature of the sources and the large number of potentially influencing factors.

The objectives of the study reported here were (1) to measure the concentrations of PAHs in particulate matter <1 μm in diameter (PM1), PM2.5 (<2.5 μm in diameter) and the respirable fraction (<4 μm in diameter) in smokers' and non-smokers' office environments, respectively, and (2) to apply a receptor model (Positive Matrix Factorization) using the chemical data collected for identifying potential PAH sources and estimating their respective contribution.

Materials and methods

Sampling site

Two adjacent offices served as the sites for this study during the period 16–27 July 2007. These two offices are located in the administration building of the National Center for Scientific Research "Demokritos", which is situated in Aghia Paraskevi, a suburb of Athens located to the north-east of city center. During the study, the two offices were identical in terms of building type, furniture and equipment/apparatus, with smoking activity of the occupants being the major factor differentiating the two environments. Smoking was permitted in one office, and during the study there were two occupants, namely, a smoker and a passive smoker; in contrast, two non-smokers occupied the second office and smoking was not allowed. The equipment/apparatus in both offices included two computers, a printer and a photocopy machine. A graphic description of the layouts of the offices are provided in Fig. 1. The volume of the smokers' and non-smokers' offices was 63 and 75.6 m^3, respectively. The average indoor air temperature and relative humidity for the period of the study were 28±3°C and 57±5%, respectively; in comparison, the outdoor air temperature and relative humidity were 32±5°C and 55±6%, respectively.

The offices were ventilated daily using natural ventilation, i.e. open windows. The average air exchange rate during the study, which was estimated using Nordtest method NT VVS 118 [Nordtest 1994], was 0.46±4 and 0.41±7% h^{-1} for the non-smoking and smoking office, respectively. The offices are open to the public weekdays and closed during the weekend. All occupants in the offices followed the same daily working schedule, as recorded in questionnaires.

The outdoor area in the immediate vicinity of the building is extensively covered by evergreen trees and other plants, while the Hemittos mountain peripheral highway is situated at a distance of 300 m. A parking area

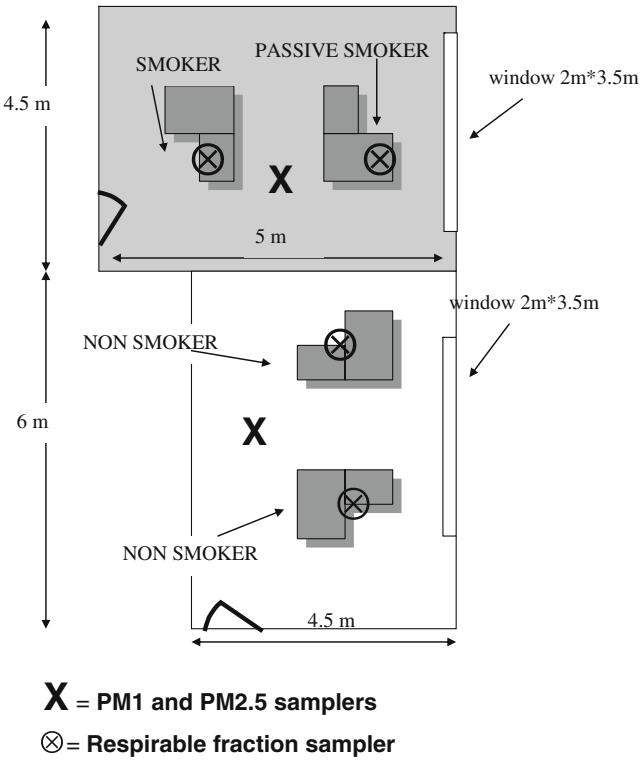

Fig. 1 Graphic description of the layout of the two offices and the position of the samplers. *PM1*, *PM2.5* Particulate matter <1 and <2.5 μm in diameter, respectively

is also situated nearby the building (at a distance of approximately 50 m).

Experimental methodology

Twenty-four-hour samplings of indoor PM1 and PM2.5 and personal exposure measurements of the respirable fraction were carried out during working hours (0800–1600 hours) using two Deranda LVS3.1 samplers (Norbert Derenda, Stahnsdorf, Germany) and a TSI SP530 sampler (TSI, Shoreview, MN), respectively. A total of seven and five samples of PM1 and PM2.5 fractions were collected in the smokers' and non-smokers' offices, respectively, and four samples of the respirable fractions from each volunteer were collected. Particulate matter was collected on 47-mm pre-conditioned tissue quartz filters, and particle mass concentration was determined gravimetrically using an electronic microbalance (model MX-5; Mettler-Toledo, Beaumont Leys, UK) at a resolution of 10^{-6} g, (scale was placed in a "weighing room". In order to achieve the appropriate conditions in the weighing room (temperature=20±1°C, relative humidity=50±5%), the temperature and relative humidity were automatically regulated by means of a continuously operating air-conditioner. The conditioning period of the filters before and after weighing was at least 48 h.

The concentrations of particle-bound PAHs were determined by gas chromatography–mass spectrometric (GC-MS) analysis (ISO 12884:2000; Tolis et al 2008). PAHs were extracted from the filters by ultrasonication as follows. The filters were placed in a flask to which 40 ml of dichloromethane was added, and the samples were ultrasonically extracted for 30 min in an ultrasonic bath. The procedure was repeated using a second aliquot of 40 ml of dichloromethane, following which the two extracted volumes were combined (80 ml) and then evaporated down to a concentrated volume of 5 ml. *n*-Hexane (10 ml) was then added to the concentrated extract, and the extract was rotary evaporated until a volume of 2–3 ml volume. The *n*-hexane extract was transferred quantitatively to a pre-wash, with 10 ml dichloromethane, column (25×0.9 cm I internal diameter) filled with approximately 2 g of activated silica gel and topped with 0.5 g of anhydrous sodium sulphate. The aliphatic hydrocarbons were eluted first by passing 10 ml of*n*-hexane through the column, and the PAH fractions were collected by eluting the column with 10 ml of a *n*-hexane–ethylacetate (9:1) mixture. The solution containing the PAHs was first concentrated down to 2 ml by rotary evaporation and then further concentrated down to 100 µl by a gentle steam of pure nitrogen. This procedure was repeated twice, each time adding 2 ml of isooctane. The extracts were analyzed for PAHs using an Agilent Technologies 6890 N gas chromatography system (Agilent Technologies, Santa Clara, CA) equipped with a 5973 mass selective detector (GC–MSD) and a DB-5MS capillary column (30 m, internal diameter 0.25 mm, film thickness 0.25 µm), operated in the selected ion-monitoring mode. The final determination of the two- to six-ring PAHs was carried out at the following parameters: (1) carrier gas: helium (1 ml/min, constant flow); (2) temperature program: 60°C (1 min), 60–290°C (15°C/min) 290°C (15 min); (3) injection volume: 1 µl (splitless); (4) injector temperature: 285°C. The transfer line was held at 280°C, and the identification–quantification procedure was based on calibration with standard PAH solutions on the basis of an internal standard solution of deuterated PAHs (d8-Nap, d10-A, d10-Phe, d10-Chr, d10-Pyr, d12-BP and d12-perylene). The standard PAH solutions were purchased from Dr. Ehrenstorfer (Augsburg, Germany) and added prior to the analysis.

As a quality control measure of the chemical analysis, we analyzed field blank and laboratory blank samples to determine potential contamination. Instrument solvent blanks were run to check the status of the analytical system. Twenty-six native PAHs and seven surrogates were analyzed. The limit of detection (LOD) and limit of quantitation (LOQ) of the method were around 0.001 and 0.003 ng/m^3, respectively, for each PAH. The recoveries of the native and deuterated PAHs were in the range of 50–120%, and the relative standard deviation (SD) for replicates ranged from 10 to 20%. Seven surrogates were used to monitor the behavior of the PAHs during sample pretreatment and instrument analysis. The recoveries of the various surrogates were 50–80% of naphthalene-d8, 60–100% of acenaphthene-d10 and 70–120% of phenanthrene-d10, pyrene-d10, chrysene-d12, perylene-d12 and benzo (ghi)perylene; thus all surrogate recoveries fell into the acceptable range of 50–150% (goal 75–125, acceptable 50–150) according to the international standard ISO 12884 (ISO 12884:2000). The uncertainties for the PAHs measured ranged from 5.5% (fluorene and phenanthrene) to 18.6% [indeno(1,2,3-c,d)pyrene].

Finally, the measurement procedure included the daily recording of all necessary details of the occupants' activities. In the smokers' office, the number of cigarettes smoked ranged from 18 to 24 per day.

PMF model background

Positive matrix factorization is a new variant factor analysis method developed by Dr. Paatero at the University of Helsinki (Finland) in the mid-1990s. In our study, we used the PMF1.1 version, in the robust mode. The mathematical background of the model has been described in detail by Paatero 1997, and we only provide a brief description of the technique here. PMF uses a weighted least-squares fit with the known error

estimates of the elements of the data matrix used to derive the weights. The factor model can be written as

$$X = GF + E \tag{1}$$

where X is the known $n \times m$ matrix of the m measured chemical species in n samples. G is an $n \times p$ matrix of the contributions of p sources to the samples (time variations). F is a $p \times m$ matrix of source compositions (source profiles). Both G and F are factor matrices to be determined. E is defined as a residual matrix, i.e., the difference between the measurement X and the model Y as a function of factors G and F.

$$e_{ij} = x_{ij} - y_{ij} = x_{ij} - \sum_{k=1}^{p} g_{ik} f_{kj} (I = 1, ..., n; \; j = 1, ..., m; k = 1, ..., p) \tag{2}$$

The objective of PMF is to minimize the sum of the squares of the residuals weighted inversely with error estimates of the data points. Furthermore, PMF constrains all of the elements of G and F to be non-negative; this means that sources cannot have a negative species' concentration ($f_{kj} \geq 0$), and samples cannot have a negative source contribution ($g_{ik} \geq 0$). Therefore, the task of PMF analysis can be described to be that of minimizing $Q(E)$, which is defined as

$$Q(E) = \sum_{i=1}^{n} \sum_{j=1}^{m} (e_{ij} / s_{ij})^2 \tag{3}$$

with $f_{kj} \geq 0$, $g_{ik} \geq 0$ and s_{ij} is the error estimate for x_{ij}.

In the robust analysis, Eq. (3) is transformed to

$$Q(E) = \sum_{i=1}^{n} \sum_{j=1}^{m} \left(e_{ij} / h_{ij} \, s_{ij}\right)^2 \tag{4}$$

where $h_{ij}^2 = 1$ if $\left|\frac{e_{ij}}{s_{ij}}\right| \leq a$ or $h_{ij}^2 = \left|\frac{e_{ij}}{s_{ij}}\right| / a$ otherwise.

Here, h_{ij} denotes the outlier distancem and the value 4 has been selected for the parameter α (Huber 1981).

PMF uses a non-zero rotational parameter, FPEAK (Paatero et al 2002), to impose rotations to the emerging solutions throughout the iteration sequence. The appropriate value of FPEAK is that value which corresponds to the global minimum value of Q(E).

A critical step in PMF analysis is the determination of the number of factors. It is an established truth that choosing too few factors may lead to sources that are not well separated, whereas too many factors may essentially lead to a split up of a true source into two or more non-existing sources. In practice, one usually examines the frequency distributions of the scaled residuals (e_{ij}/s_{ij}) as well as changes in the Q-value given by Eq. 4. The value of $Q(E)$ must be a minimum or stable. This method of determining the 'correct' number of factors has been described by Lee et al. 1999 and Yakovleva et al. 1999. After the appropriate number of factors have been included in the model fit, additional factors will not result in significant further improvements in the Q-value. In this study, the number of factors that was examined ranged from three to seven, with the optimal number of factors determined from the slope of the Q-value versus the number of factors. On the basis of this analysis, a four-factor solution was chosen for both data sets (PM1 and PM2.5 fractions).

The data set for each sample, including concentrations of 21 PAHs (which were over the detection limit), was used as input in the PMF analysis. A data set that included unique uncertainty values of each data point was created and inserted into the model. The calculation of the uncertainty for each concentration value was based on Eq. 5:

$$\text{uncertainty} = \sqrt{(\text{error fraction} \times \text{concentration})^2 + (\text{Method Detenction Limit})^2} \tag{5}$$

Results and discussion

Experimental results

Figure 2 shows the diurnal variation patterns of PM1 and PM2.5 in both offices. We found a clear decrease in mass and ΣPAH concentration in the smokers' office during weekend, with the mass concentration for PM1 and PM2.5 being 138 and 62% lower, respectively, during the weekend than on weekdays. The ΣPAH levels showed a similar trend, with both fractions being 70% lower during the weekend. The maximum PM and ΣPAH levels in the smokers' office were observed on the day that the most cigarettes were smoked (day 5). In addition, opening of the windows led to a further increase in the concentration of PAHs, implying a contribution from the outdoor environment. In particular, on day 4 in the non-smokers' office (window was open during all working hours), ΣPAH levels (for both PM1 and PM2.5) were threefold higher than the office's background. In the smokers' office, however, the contribution of the outdoor environment is not so distinct because of the presence of smoking, which is expected to be the dominant source.

Among the PAHs measured, fluorene, pyrene, benzo (a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k) fluoranthene, benzo(e)pyrene, benzo(a)pyrene, perylene, indeno(1,2,3-cd)pyrene, dibenzo(a,h)anthracene and benzo(ghi)perylene are carcinogenic (IARC 2004; Okuda et al 2006) (carcinogens are indicated with an asterisk in the following text). The average total concentration of

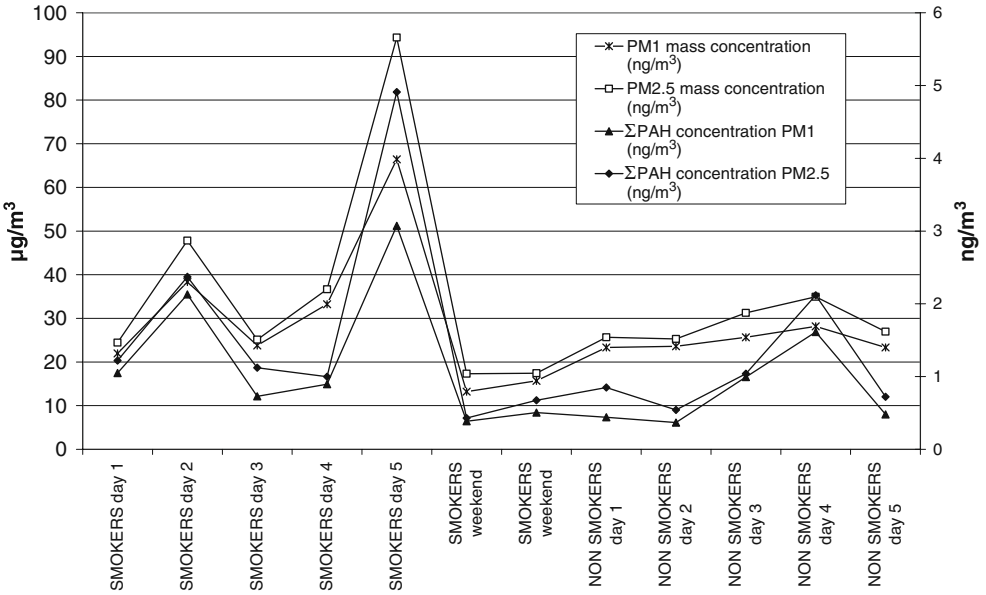

Fig. 2 The diurnal variation patterns of PM1 and PM2.5 in both the smokers' and non-smokers' offices. *PAH* Polycyclic aromatic hydrocarbons

these PAHs, ΣPAH_{cancer}, was 0.813 (for PM1) and 1.128 ng/m^3 (for PM2.5) in the smokers' office, and 0.424 (for PM1) and 0.624 ng/m^3 (for PM2.5) in the non-smokers' office. The average concentrations of carcinogenic PAHs in the PM1 and PM2.5 fractions for both offices and the respirable fraction measured at volunteer's breathing height are presented Figs. 3 and 4. In the smokers' office, the levels of all carcinogenic PAHs fell by more than 70% during the weekend, implying that smoking was a major indoor source of PAHs. In the respirable PM fraction, the average ΣPAH_{cancer} concentration, determined from measurements at volunteer's breathing height, was 22.37, 31.58 and 4.140 ng/m^3 for the smoker, passive smoker and non-smoker, respectively. It is quite remarkable that the average ΣPAH_{cancer} for the passive smoker was 1.4-fold higher than that for the smoker. This increased value for the passive smoker could be attributed to the smoke's plume formation, which seems to affect mostly the passive smoker who works at a distance of 2.5 m from the smoker. It has been found that additional compounds may be generated by sidestream smoke alone, which is the emission from the burning end of a cigarette (EPA 1994; IARC 2004; U.S. Department of Health and Human Services 2006). Environmental tobacco smoke consists of both sidestream and exhaled mainstream smoke and is often termed as "secondhand smoke" or "passive smoke".

Table 1 presents the average concentration of PAHs in the PM1, PM2.5 and respirable fraction in both offices. The results show that in the smokers' office, the most abundant PAHs in both the PM1 and PM2.5 fractions were 2-methyl phenanthrene and benzo(b)fluoranthene*. In the non-smokers' office, the most abundant PAHs in the PM1 and PM2.5 fractions were 2-methyl phenanthrene and benzo(ghi)perylene*, and 2-methyl phenanthrene and indeno(1,23-cd)pyrene*, respectively. PAH levels in the respirable fraction were significantly higher, with the most abundant PAHs in the respirable fraction of the smoker being benzo(a)anthracene* and chrysene*, in the respirable fraction of the passive smoker (i.e. non-smoking volunteer working in the smokers' office), benzo(e)pyrene* and benzo(b)fluoranthene* and in that of the non-smoker, pyrene* and naphthalene. Our measurements clearly indicate that the PAHs mostly detected from measurements in the smoker's office and at volunteers' breathing height are associated with smoking activity (U.S. Department of Health and Human Services 2006; Risk Assessment Information 2009; Lu and Zhu 2007).

Among the PAHs, benzo[a]pyrene (BaP) has been studied extensively because of its strong and direct carcinogenicity in the particulate phase (EIONET 2009). Chuang et al. reported that the indoor BaP concentration in a heavy smoker's house was tenfold higher than that in a non-smoker's home. In our study, the BaP concentration in the PM1 fraction in the smokers' office was 3.8-fold higher than that in non-smokers' office; the corresponding value for the PM2.5 fraction was 3.9-fold.

PMF results

Table 2 presents the contribution of the four factors to each species (PAHs), and Figs. 5 and 6 present the variations in the factors during the study period. A brief analysis of the factors follows.

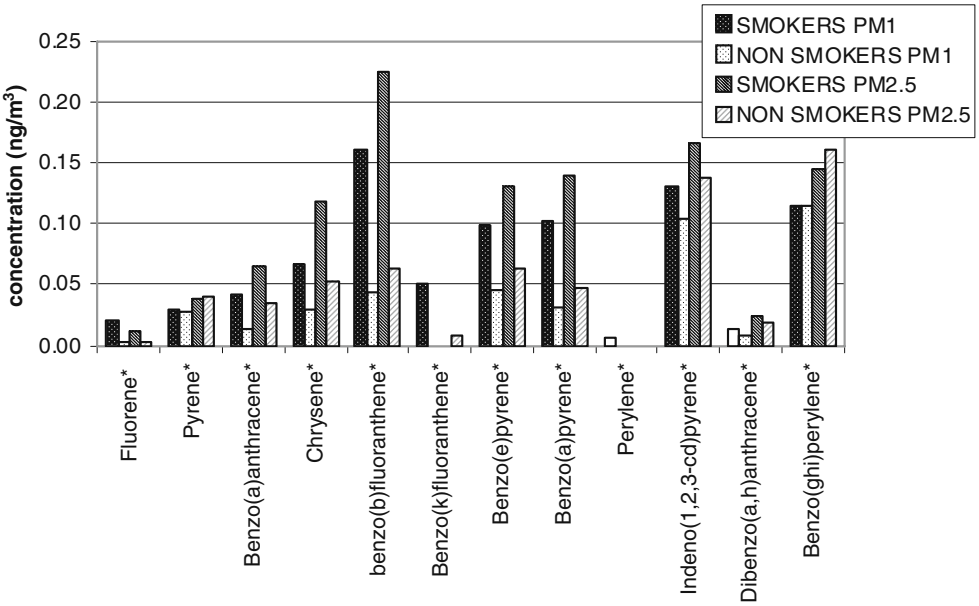

Fig. 3 Levels of carcinogenic PAHs (PAHcancer) in the PM1 and PM2.5 fractions in the smokers' and non-smokers' office. *Asterisk* indicates the PAH is carcinogenic

Factor 1 was present mainly in the smokers' office during weekdays, but is absent during the weekend. When windows were open during all working hours (4th day, smokers' office), factor 1 decreased, especially for in the PM2.5 fraction, while PM1 fraction levels remained low. Factor 1 was also slightly present in non-smokers' office, especially in the PM1 fraction which has an intense infiltration capability. These results suggest that this factor is strongly associated with smoking activity. Furthermore, the most abundant PAHs were related to this factor, namely, 2,6 dimethylnaphthalene, 2,3,5-trimethylnaphthalene, benzoanthracene, as expected (Lu and Zhu, 2007; Risk Assessment Information System 2009).

Factor 2 was present in both offices during working days, while being absent during the weekend (Figs. 5 and 6). This factor seems to fluctuate for the PM1 fraction in particular, significantly decreasing during the days when the windows remained open (4th day, smokers' office and 4th day, non-smokers' office). Chrysene, benzo(a)pyrene and dibenzo(a,h)perylene, which were strongly associated with this factor, have been found to be related to the dust accumulating in computers (Ren et al. 2006). Taking all these results into consideration, we suggest that factor 2 is associated with the dust that accumulates indoors and is influenced by resuspension and ventilation. The fact that this dust component may be reflective of absorbed tobacco smoke components cannot be ignored.

Factor 3 fluctuated during the entire week. PAHs associated with this factor originate from combustion sources, such as vehicle exhausts, coal burning without excluding tobacco smoking (Menichini et al. 2007; Risk Assessment Information System 2009). Specifically, fluorene, which is the dominant compound in Factor 3 (Table 3),

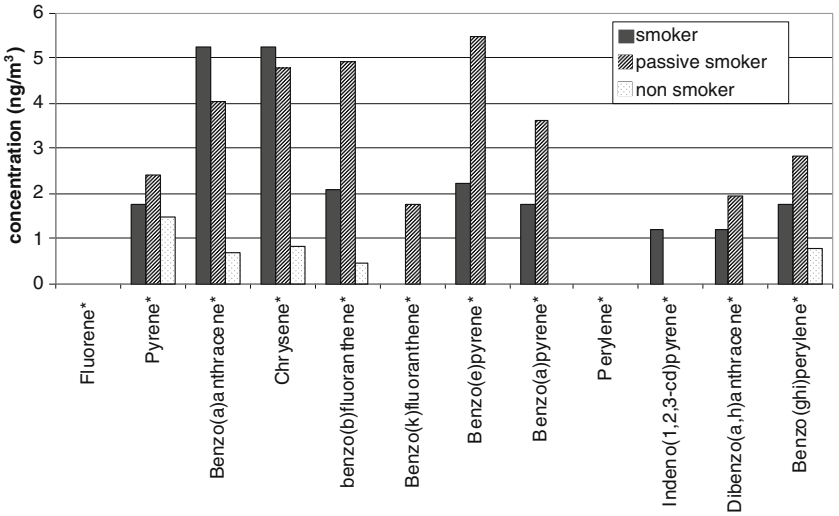

Fig. 4 PAHcancer levels in the respirable fraction. *Asterisk* indicates the PAH is carcinogenic

Table 1 Average concentration of polycyclic aromatic hydrocarbons (ng/m^3) for PM1, PM2.5 and respirable fractions in both offices

Polycyclic aromatic hydrocarbon[a]	Smoker' office PM1		Non-smokers' office PM1		Smokers' office PM2.5		Non-smokers' office PM2.5		Smoker (respirable fraction)		Passive smoker (respirable fraction)		Non-smoker (respirable fraction)	
	AVG	STDEV	AVG	STDEV	AVG	STDEV	AVG	STDEV	AVG	STDEV	AVG	STDEV	AVG	STDEV
Naphthalene	0.014	0.018	0.012	0.009	0.023	0.029	0.030	0.030	2.158	2.393	3.281	0.207	1.290	1.464
2-Methynaphthalene	0.008	0.016	0.004	0.006	0.005	0.005	0.014	0.016	< D.L		0.099	0.140	< D.L	
1-Methynaphthalene	0.004	0.009	0.001	0.002	0.006	0.012	0.095	0.133	< D.L		0.210	0.144	< D.L	
2.6-Dimethylnaphthale ne	0.003	0.007	0.001	0.002	0.004	0.009	0.004	0.005	< D.L		< D.L		< D.L	
Acenaphthylene	0.013	0.012	0.014	0.008	0.023	0.012	0.015	0.011	< D.L		< D.L		< D.L	
1.2-Dimethyl naphthalene	< D.L[b]		< D.L		< D.L		< D.L		< D.L		< D.L		< D.L	
Acenaphthene	0.001	0.002	< D.L		< D.L		0.002	0.005	< D.L		< D.L		< D.L	
2.3.5-Trimethyl naphthalene	< D.L		< D.L		< D.L		< D.L		< D.L		< D.L		< D.L	
Fluorene*	0.020	0.018	0.003	0.004	0.032	0.014	0.004	0.006	< D.L		< D.L		< D.L	
Phenanthrene	0.025	0.011	0.023	0.018	0.029	0.021	0.028	0.010	0.516	0.093	1.123	1.015	0.255	0.360
Anthracene	< D.L		< D.L		< D.L		< D.L		< D.L		0.581	0.821	< D.L	
2-Methyl phenanthrene	0.319	0.201	0.172	0.126	0.468	0.334	0.200	0.121	< D.L		< D.L		< D.L	
1-Methy phenanthrene	< D.L		< D.L		< D.L		< D.L		< D.L		< D.L		< D.L	
3.6-Dimethyl phenanthrene	0.026	0.044	< D.L		0.035	0.029	< D.L		< D.L		< D.L		< D.L	
Fluoranthene	0.030	0.011	0.027	0.020	0.040	0.024	0.038	0.013	0.644	0.362	1.238	1.254	0.732	1.036
Pyrene*	0.029	0.010	0.028	0.021	0.037	0.026	0.040	0.016	1.745	0.714	2.385	1.996	1.481	2.094
Benzo(a)anthracen e*	0.042	0.038	0.013	0.015	0.064	0.066	0.034	0.005	5.243	3.687	4.008	5.669	0.653	0.923
Chrysene*	0.067	0.085	0.029	0.034	0.117	0.190	0.053	0.021	5.244	0.291	4.773	6.751	0.812	1.148
Benzo(b)fluoranthe ne*	0.160	0.196	0.043	0.044	0.225	0.317	0.063	0.033	2.054	2.905	4.887	6.911	0.446	0.631
Benzo(k)fluoranthe ne*	0.050	0.123	0.006	0.018	0.070	0.063	0.008	0.018	< D.L	< D.L	1.728	2.444	< D.L	
Benzo(e)pyrene*	0.098	0.087	0.046	0.041	0.130	0.137	0.063	0.034	2.205	0.488	5.482	7.752	< D.L	
Benzo(a)pyrene*	0.102	0.105	0.030	0.036	0.140	0.158	0.048	0.029	1.730	0.184	3.584	5.068	< D.L	
Perylene*	0.006	0.016	< D.L		0.009	0.011	< D.L		< D.L		< D.L		< D.L	
Indeno(1,2,3-cd)pyrene*	0.130	0.097	0.103	0.121	0.167	0.146	0.138	0.092	1.202	1.699	< D.L	< D.L	< D.L	
Dibenzo(a,h)anthra cene*	0.013	0.026	0.009	0.017	0.024	0.038	0.018	0.025	1.200	1.697	1.912	2.704	< D.L	
Benzo(ghi)perylene *	0.115	0.070	0.114	0.129	0.144	0.112	0.161	0.127	1.748	0.992	2.820	1.580	0.748	1.058

PAH, Polycyclic aromatic hydrocarbon; PM1, PM2.5, particulate matter <1 and <2.5 μm in diameter, respectively. AVG, average; STDEV, standard deviation

[a] Asterisk indicates the PAH is carcinogenic

[b] <D.L., Under the detection limit

Table 2 Factors contribution (%) to each species

Polycyclic aromatic hydrocarbon	PM1				PM2.5			
	Factor 1	Factor 2	Factor 3	Factor 4	Factor 1	Factor 2	Factor 3	Factor 4
Naphthalene	0.0	3.1	92.9	3.9	7.1	5.2	87.7	0.0
2-Methylnaphthalene	0.0	0.0	92.9	7.1	0.0	0.0	92.5	7.5
1-Methylnaphthalene	6.7	0.0	93.3	0.0	0.0	4.8	95.2	0.0
2,6-Dimethylnaphthalene	0.0	100	0.0	0.0	75.0	0.0	25.0	0.0
Acenaphthene	0.0	90.0	10.0	0.0	30.5	1.1	31.1	37.4
2,3,5-Trimethylnaphthalene	100	0.0	0.0	0.0	13.0	39.0	23.4	24.7
Fluorene	2.4	0.0	0.0	97.6	0.0	0.0	0.0	100
Phenanthrene	2.1	56.4	12.4	29.1	13.6	51.1	18.9	16.4
2-Methylphenanthrene	50.3	8.0	12.2	29.5	58.7	0.0	0.0	41.3
3,6-Dimethylphenanthre	0.0	0.0	0.0	100	13.7	41.1	27.4	17.8
Fluoranthene	1.0	62.5	0.0	36.5	3.9	70.4	13.4	12.4
Pyrene	0.0	63.0	3.1	33.9	0.0	74.0	16.5	9.4
Benzoaanthracene	92.5	0.0	0.9	6.5	30.7	68.3	0.0	1.0
Chrysene	6.1	92.9	0.9	0.0	20.6	79.4	0.0	0.0
Benzo(b)fluoranthene	75.7	14.7	9.6	0.0	53.3	42.5	0.2	3.9
Benzo(k)fluoranthene	53.5	23.3	23.3	0.0	0.0	5.4	90.1	4.5
Benzo(e)pyrene	52.9	29.4	1.7	15.9	44.7	39.6	12.7	3.1
Benzo(a)pyrene	21.3	77.8	0.7	0.3	36.7	59.7	0.9	2.8
Indeno(1,2,3-c,d)pyrene	40.9	40.2	0.0	19.0	25.4	50.2	22.0	2.3
Dibenzo(a,h)anthracene	0.0	33.3	0.0	66.7	0.0	100	0.0	0.0
Benzo(ghi)perylene	27.7	48.6	0.0	23.7	13.9	54.7	27.8	3.6

has been reported to be associated with oil combustion (Ravindra et al. 2006a; Harrison et al. 1996). It can be concluded that this factor corresponds to outdoor oil-combustion sources and mainly to oil-using cars and motorbikes, as other combustion sources do not exist in the area during summer.

Factor 4 follows a different variation than that of factor 3. During the days that factor 4 is dominant, the diagnostic ratio benzo(a)anthracene/benzapyrene was around 0.5, which indicates gasoline-source origin (Li and Kamens 1993; Esen et al. 2008). During the same days, the fluorene/(fluorene+pyrene) ratio was <0.5, which also indicates a

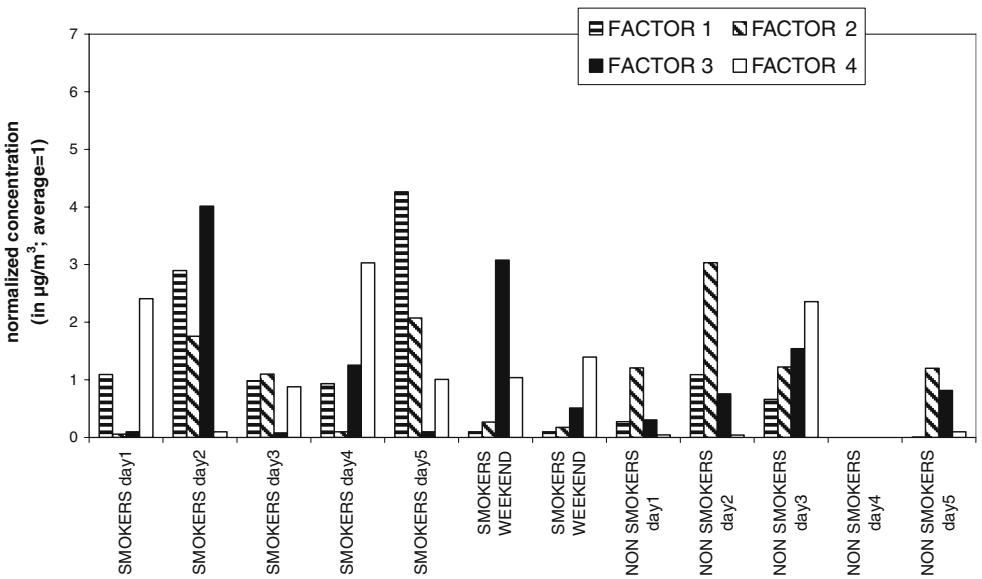

Fig. 5 Variation in the four factors for the PM1 fraction. *Factor 1* Smoking activity; *Factor 2* dust accumulation/resuspension, *Factor 3* outdoor oil-combustion sources, *Factor 4* gasoline-burning vehicles

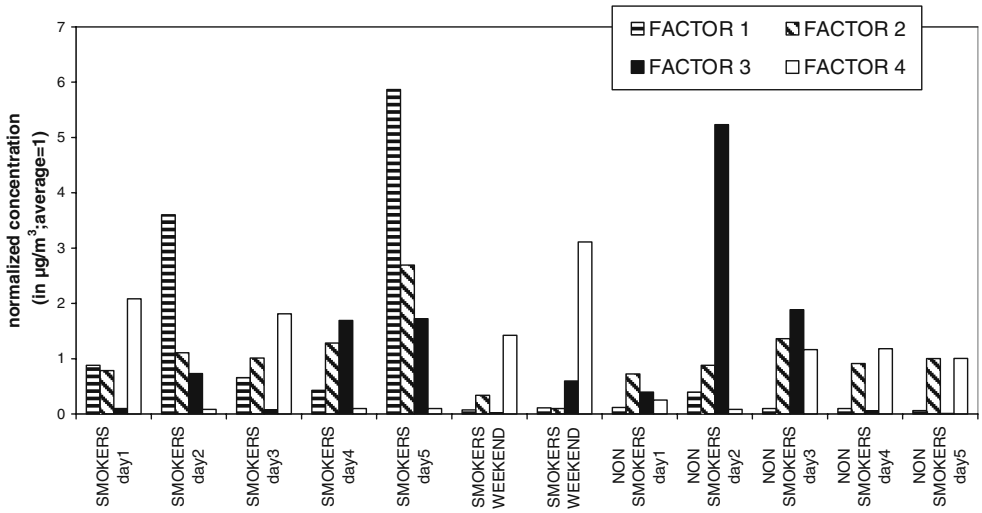

Fig. 6 Variation in the four factors for the PM2.5 fraction

gasoline-source origin (Fang et al. 2004; Ravindra et al. 2006a, b). It can therefore be concluded that this factor is associated with gasoline-burning vehicles.

Table 3 shows that in 15 PAHs, the factor which contributes the most is the same in both fractions (PM1 and PM2.5), implying a common origin and further supporting the validity of the proposed approach. Smoking and dust resuspension contribute the most (>90%) to benzo (a)pyrene in both the PM1 and PM2.5 fractions. The correlation between the PM1 and PM2.5 fractions for each factor was also examined (Table 3), and a significant correlation was found only for variations in Factor 1 (smoking activity) between the PM1 and PM2.5 fractions. The other factors exhibited a lower correlation, which is statistically non-significant at the 95% confidence level. This result is an indication of significant variation in the origin and composition of the sources that form each factor.

Finally, a point for consideration is the manner in which information on PAH levels can be used for an indoor source apportionment study. Most PAHs are related to combustion sources, and it is quite complicated to distinguish between such PAHs when different indoor and outdoor sources are to be identified. The a priori knowledge of source profiles could be helpful in the application of a receptor model (i.e. Chemical Mass Balance), but such knowledge is to a large extent inapplicable when indoor PAHs sources profiles are used.

Table 3 Correlation coefficient of factors between PM1 and PM2.5

Factors	*r*
Factor 1 (PM1)–Factor 1 (PM2.5)	0.97
Factor 2 (PM1)–Factor 2 (PM2.5)	0.44
Factor 3 (PM1)–Factor 3 (PM2.5)	−0.02
Factor 4 (PM1)–Factor 4 (PM2.5)	0.25

Factor 1, smoking; Factor 2, dust resuspension; Factor 3, outdoor oil-combustion sources; Factor 4, gasoline-burning vehicles

Conclusions

The objectives of our study were to estimate the levels of PAHs in the PM1, PM2.5 and respirable fractions in a smokers' and non-smokers' office environment and, by applying a receptor model, to identify the main PAH sources and their contribution.

Our measurements indicate a clear difference in PAH-cancer levels (in PM1 and PM2.5) between the two office environments. Measurements made during the weekend in the smokers' office revealed that PAHcancer levels were more than 70% lower than those on weekdays. This result emphasizes the importance of smoking and dust resuspension sources, as outdoor sources did not present a clear differentiation. In terms of the respirable fraction measurements, it is remarkable that the passive smoker's PAHcancer average levels were 1.4-fold higher than those of the smoker. Thus, according to the measurements, in the absence of indoor heating sources, smoking is the dominant indoor source influencing all particle fractions. However, a contribution by the outdoor environment cannot be excluded. In our study, emissions from vehicle exhausts are assumed to be the main outdoor sources, as other combustion sources (industries, coal burning) do not exist in the area.

The PMF model identified four factors–sources: smoking, dust resuspension and two different vehicle-related sources. It is remarkable that in terms of benzo(a)pyrene, smoking and dust resuspension had the strongest contribution (>90%) for both the PM1 and PM2.5 fractions. Although the PMF analysis confirmed the well-known contribution of smoking and traffic-related sources on the levels of PAHs, the identification of dust resuspension as a

separate PAH source is of great interest and requires further study.

Acknowledgements Authors would like to thank all volunteers (employees in NSCR "Demokritos") who took part in this study.

References

Chao CYH, Yeung LL, Choi PSH (2002) Quantification of polycyclic aromatic hydrocarbons and aliphatic hydrocarbons in air particulate samples in homes. Indoor Built Environ 11:123–133

Chuang JC, Mark GA, Koetz JR, Petersen BA (1995) A pilot study of sampling and analysis for polynuclear aromatic compound in indoor air. EPA/600/4-86/036. US Environmental Protection Agency Office of Research and Development, Environmental Monitoring System, Washington D.C.

EIONET (2009) Review of the Structural Indicator, the Urban Audit Indicators and EEA's Core Set Indicator (CSI004)—Recommendations. In: 14th EIONET Workshop Air Quality Management and Assessment. Warsaw, Poland

Environmental Protection Agency (EPA) (1994) Indoor air pollution: an introduction for health professionals. Available at: www.epa.gov/iaq/pubs/hpguide.html

Esen F, Tasdemir Y, Vardar N (2008) Atmospheric concentrations of PAHs, their possible sources and gas-to-particle partitioning at a residential site of Bursa, Turkey. Atmos Res 88(3–4):243–255

Fang GC, Wu YS, Chen MH, Ho TT, Huang SH, Rau JY (2004) Polycyclic aromatic hydrocarbons study in Taichung, Taiwan, during 2002–2003. Atmos Environ 38:3385–3391

Hameri K, Lahde T, Niemela R, Korhonen P (2003) Fine aerosols indoors and outdoors in downtown Helsinki. In: Abstracts European Aerosol Conf 2003.

Harrison RM, Smith DJT, Luhana L (1996) Sourceapportionment of atmospheric polycyclic aromatic hydrocarbonscollected from an urban location in Birmingham,UK. Environ Sci Technol 30:825–832

Huber PJ (1981) Robust statistics. Wiley, New York

International Agency for Research on Cancer (IARC) (2004) Monographs on the evaluation of carcinogenic risks to humans vol. 83. IARC Press, Lyon

International Organization for Standardization (ISO) (2000) ISO 12884. Ambient air—determination of total (gas and particle-phase) polycyclic aromatic hydrocarbons—collection on sorbent-backed filters with gas chromatographic/mass spectrometric analyses. ISO, Geneve

Lee E, Chun CK, Paatero P (1999) Application of positive matrix factorization in source apportionment of particulate pollutants in Hong Kong. Atmos Environ 33:3201–3212

Li A, Schoonover TM, Zou Q, Norlock F, Conroy LM, Scheff PA, Wadden RA (2005) Polycyclic aromatic hydrocarbons in residential air of ten Chicago area homes: Concentrations and influencing factors. Atmos Environ 39(19):3491–3501

Li CK, Kamens RM (1993) The use of polycyclic aromatic hydrocarbons as sources signatures in receptor modeling. Atmos Environ 27A:523–532

Lu H, Zhu L (2007) Pollution patterns of polycyclic aromatic hydrocarbons in tobacco smoke. J Haz Mater 139(2):193–198

Mannino MR, Orecchio S (2008) Polycyclic aromatic hydrocarbons (PAHs) in indoor dust matter of Palermo (Italy) area: Extraction, GC-MS analysis, distribution and sources. Atmos Environ 42 (8):1801–1817

Menichini E, Iacovella N, Monfredini F, Turrio-Baldassari L (2007) Relationships between indoor and outdoor air pollution by carcinogenic PAHs and PCBs. Atmos Environ 41:9518–9529

Naumova YY, Eisenreich SJ, Turpin BJ, Weisel CP, Morandi MT, Colome SD, Totten LA, Stock TH, Winer AM, Alimokhtari S, Kwon J, Shendell D, Jones J, Maberti S, Wall SJ (2002) Polycyclic aromatic hydrocarbons in the indoor and outdoor air of three cities in the US. Environ Sci Technol 36:2552–2559

Nordtest (1994) Nordtest Method NT VVS 105. Ventilation: flow rate, total effective-by single zone approximation. Nordtest, Espoo

Ohura T, Amagai T, Fusaya M, Matsushita H (2004) Polycyclic aromatic hydrocarbons in indoor and outdoor environments and factors affecting their concentrations. Environ Sci Technol 38:49–55

Okuda T, Naoi D, Tenmoku M, Tanaka S, He K, Ma Y, Yang F, Lei Y, Jia Y, Zhang D (2006) Polycyclic aromatic hydrocarbons (PAHs) in the aerosol in Beijing, China, measured by aminopropylsilane chemically-bonded stationary-phase column chromatography and HPLC/fluorescence detection. Chemosphere 65(3):427–435

Paatero P (1997) Least squares formulation of robust non-negative factor analysis. Chemometr Intelligent Lab Syst 6037:23–35

Paatero P, Hopke PK, Song XH, Ramadan Z (2002) Understanding and controlling rotations in factor analytic models. Chemometr Intelligent Lab Syst 60(1–2):253–264

Ravindra K, Bencs L, Wauters E, de Hoog J, Deutsch F, Roekens E, Bleux N, Bergmans P, Van Grieken R (2006a) Seasonal and site specific variation in vapor and aerosol phase PAHs over Flanders (Belgium) and their relation with anthropogenic activities. Atmos Environ 40:771–785

Ravindra K, Wauters E, Taygi SK, Mor S, Van Grieken R (2006b) Assessment of air quality after the implementation of CNG as fuel in public transport in Delhi, India. Environ Monit Assess 115:405–417

Ren Y, Cheng T, Chen J (2006) Polycyclic aromatic hydrocarbons in dust from computers: one possible indoor source of human exposure. Atmos Environ 40:6956–6965

Risk Assessment Information System (2009). Available at: www.rais.oml.gov

Robinson J, Nelson WC (1995) National human activity pattern survey data base. United States Environmental Protection Agency, Research Triangle Park

Rogge WF, Hildermaqnn LM, Maurec MA, Cass GR, Simoneit BRT (1993) Sources of fine organic aerosol. 5. Natural gas home appliances. Environ Sci Technol 35:2736–2744

Sheldon L, Clayton A, Keever J, Perritt R, Whitaker D (1993) Indoor concentrations of polycyclic aromatic hydrocarbons in California residences. Final report. Air Resources Board, Sacramento

Tolis EI, Amarantidis S, Missia DA, Saraga DE, Koziakis NE, Bartzis JG (2008) Preliminary results on PM1 and particle-Bound polycyclic aromatic hydrocarbons (PAHs) in Kozani, Greece. Fresenius Environ Bull 17 (10 A):1634–1639

U.S. Department of Health and Human Services (2006) The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. ISBN 0-16-076152-2. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. Washington D.C.

Yakovleva E, Hopke P, Wallace L (1999) Receptor modelling assessment of particle total exposure assessment methodology data. Environ Sci Technol 33:3645–3652