

# Exposure to dust-bound PAHs and associated carcinogenic risk in primitive and traditional cooking practices in Pakistan

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**Abstract** The aim of this study was to determine the abundance and distribution of polycyclic aromatic hydrocarbons (PAHs) in dust samples collected from the selected professional cooking workplaces (WCs) and residential household cooking areas (WRs), where traditional and primitive cooking practices are still prevalent. Another aim of this study was to investigate the carcinogenic risk for Pakistani human exposure to dust-bound PAHs via the routes of inhalation, ingestion, and dermal contact. Generally, the concentration of individual congeners of PAHs in surface dust samples of WC sites was higher than those measured in WR sites ( $p < 0.05$ ). The benzo(*a*)pyrene (B(*a*)P), a very high carcinogenic compound, was present in the dust samples from WC sites in the highest mean concentration (630 ng g<sup>-1</sup> dry weight (d.w.)). The BaP mean concentration in WC workplaces was almost eight times higher than the mean value found in WR exposure sites. Moreover, the average concentration of  $\Sigma$ PAHs, combustion origin PAHs ( $\Sigma$ COMB) and sum total of 7-carcinogenic PAHs

( $\Sigma$ 7-carcinogens) were also significantly higher in WC dusts samples than that in WR workplaces. Principal component analysis (PCA) and diagnostic ratios suggested coal/wood combustion as major PAH emission sources in both exposure sites. The average incremental lifetime cancer risk (ILCR) suggested a moderate to potential high cancer risk for adults and children exposed to dust-bound PAHs in both exposure sites, in particular via both dermal and ingestion contact pathways.

**Keywords** Biomass fuel combustion · Pollution · ILCR · Dust-bound PAHs · Pakistan

## Abbreviations

$\Sigma$ COMB	Combustion origin PAHs
ILCR	Incremental lifetime cancer risk
MW	Molecular weight
PAHs	Polycyclic aromatic hydrocarbons
TEF	Toxic equivalency factor
U.S. EPA	United States Environmental Protection Agency
WC	Dust sample from occupational cooking workplaces
WHO	World Health Organization
WR	Dust samples from residential cooking places

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## Introduction

The simultaneous occurrence of rapid population growth and environmental pollution both in urban and rural areas are common and the principal eco-health problems in developing countries as Pakistan (Kamal et al. 2014a, 2014b). The use of solid fuels (coals and biomass in the form of wood, bushes, tree leaves, cow dung, and crop residues) continues to be a major source of household energy for cooking in several underdeveloped areas of Pakistan. However, biomass fuel, when burnt in raw form, results in the inefficient combustion and

produces chemicals such as particulate matter (PM), carbon monoxide, sulfur dioxide, and several carcinogenic compounds including polycyclic aromatic hydrocarbons (PAHs).

The use of biomass fuel is very common in Pakistan with 66 % of households (22 % in urban areas and 90 % in rural areas). In general, these fuels are burnt indoor, using poorly functioning stoves and in scarcely ventilated ambience. As combustion is incomplete in most of these stoves, it can also serve as a key route to transfer high levels of indoor pollutants to those who are responsible for cooking and their family members. In particular, women (who do most of the cooking), children, and professional cooks are the population mainly exposed to the harmful effects of these combustion products. According to estimates of World Health Organization (WHO), there is a strong evidence that acute respiratory infections in children and obstructive pulmonary diseases in women are associated with indoor biomass smoke.

PAHs, primarily formed by incomplete combustion of organic matter and fossil fuels, are related with several human morbidities including mutagenesis, carcinogenesis, potential toxicity, and hemolytic properties (Kamal et al. 2014a; Chen et al. 2013). The carcinogenic properties of PAHs are of great concern for exposed urban population (Szabova et al. 2008). The human exposure to PAHs is mostly due to inhalation and ingestion, while dermal contact exposure may affect DNA and lead to cancer or mutation in humans (Ngo Viet et al. 2014). PAH deposition is facilitated by both dry and wet deposition in rural and urban environments, in particular in soil and dust (Cincinelli et al. 2004; Wild and Jones 1995; Murakami et al. 2005; Yang et al. 2014).

The aim of this study was to determine the abundance and distribution of PAHs in dust samples from selected professional cooking workplaces and residential household cooking areas, where traditional and primitive cooking practices are still used, in order to investigate the carcinogenic risk for Pakistani human exposure to dust-bound PAHs via inhalation, dermal contact, and ingestion routes.

## Materials and methods

### Reagents

All solvents were pesticide grade and purchased from Supelco (Bellefonte, PA, USA) and tested for contaminants before use. Standard PAH mixture EPA was purchased from Supelco (Bellefonte, PA, USA). Benzo(*e*)pyrene and coronene were purchased from Alltech (Deerfield, USA). Silica (100–200 mesh) and sodium sulfate ( $\text{Na}_2\text{SO}_4$ ) were purchased from the Merck (Darmstadt, Germany). Sodium sulfate was heated for 12 h at 450 °C to remove any organic matter and kept at 120 °C until use.

### Sample collection and processing

Dust samples were collected from residential household cooking areas in the Gujranwala and Sohdra villages (WRs) and professional cooking workplaces (semi-outdoor cooking places) (WCs) in the city of Rawalpindi (Fig. S1; EMS). In general, three to four adjacent cooking shops were considered one unit, and dust samples collected from all these shops were combined into one representative/composite sample. Each dust sample (15–20 g) was collected using a new set of dust-pan and sterile plastic brush to avoid cross-contamination. Dust samples were wrapped in pre-rinsed aluminum foil and stored in a pre-labeled zip-lock polyethylene bag for transportation to the analytical laboratory. The samples were sieved in laboratory to remove fibers, large dust particles, and other possible debris.

### Sample preparation, extraction, cleanup, and analysis

Sample preparation and instrumental analysis are described in detail in previous publications (Martellini et al. 2012; Kamal et al. 2014a). Briefly, 3 g of dust from each sample was homogenized, spiked with a mixture of deuterated standards (d4p-terphenyl, d10-fluoranthene-, d12-benzo(*a*)anthracene, and d12-benzo(*a*)pyrene) and extracted in an ultrasonic bath for 15 min (in crushed ice), using a mixture of hexane and dichloromethane (1:1 *v/v*). Then the extracts were vortex stirred (1 min) and centrifuged for 5 min at 2500 rpm. The supernatants were reduced approximately to 1 ml under a gentle stream of ultra pure nitrogen, and were further subjected to cleanup using silica gel column chromatography (9 g; Fisher Scientific 100–200 mesh silica gel). The column was first eluted with 25 ml of hexane and then with 50 ml solution of a hexane and dichloromethane (4:1 *v/v*) (Martellini et al. 2012; Kamal et al. 2015b). The resulting extracts were concentrated to 1 ml volume under gentle stream of high-purity nitrogen and transferred into a vial for storage. Prior to instrumental analyses, the extracts were spiked with an internal standard mixture containing d8-naphthalene, d10-phenanthrene, d12-chrysene, and d12-indeno(1,2,3-*c*, *d*)pyrene. The samples were injected into a Hewlett–Packard 6890 gas chromatography-mass spectrometer (GC-MS), equipped with a 5973 MSD and a HP-5MS capillary column (J&W Scientific, Folsom, CA, USA, 30 m, 0.25 mm I.D., 0.25 mm film thickness). Compound identification was based on the MSD database (National Institute of Standards and Technology (NIST), 98) and GC retaining time of authentic PAH standards. The MSD was operated in selected ion monitoring mode (SIM).

### Quality assurance and quality control (QA/QC)

Average PAHs recoveries and relative standard deviations (RSDs) were measured to evaluate the method performance

by multiple analyses of clean sand samples spiked with PAH standard mixture containing 18 PAHs (acenaphthene (Ace), acenaphthylene (Acy), anthracene (Ant), benzo(a)anthracene (BaA), benzo(a)pyrene (BaP), benzo(b)fluoranthene (BbF), benzo(e)pyrene (BeP), benzo(ghi)perylene (BghiP), benzo(k)fluoranthene (BkF), chrysene (Chry), coronene (Cor), dibenzo(a,h)anthracene (DBA), fluoranthene (Fla), fluorene (Fl), indeno(1,2,3-c,d)pyrene (IP), naphthalene (Naph), phenanthrene (Phe), and pyrene (Pyr). BbF and BkF were reported together as BbkF. In order to determine any potential laboratory contamination, the procedural blanks were also periodically performed and evaluated. The percentage of recovery (spike) of individual PAHs were between 84.1 and 122.4 %, while that of surrogate PAHs were  $93 \pm 13$  % for d12-benzo(a)pyrene-,  $97 \pm 12$  % for d10-fluoranthene,  $95 \pm 7$  % for d12-benzo(a)-anthracene, and  $94 \pm 11$  % for d4-*p*-terphenyl. The urban dust standard reference material (SRM1649a) from the National Institute of Standards and Technology (NIST) was used to evaluate the PAH mean recovery efficiency, which

varied from 86 (Acy) to 95.2 % (BghiP) in good agreement with the certified concentrations. The diluted standards used were between the range of 0.16 and  $10 \text{ ng } \mu\text{L}^{-1}$ , and a six-point calibration curve was drawn for quantification of PAHs in the dust samples. The limits of detection individual PAHs were in the range of 20–60  $\text{pg g}^{-1}$  (dry weight basis).

### Incremental lifetime carcinogenic risk model and parameters

The probabilistic incremental lifetime cancer risk (ILCR) was applied in this study to assess the potential lifetime carcinogenic risk of the exposure of people to ambient dust in their cooking workplaces/kitchens areas.

The ILCR model can be used in estimating the incremental probability of an individual developing a tumor over an average 70 years of lifetime (Meiners and Yandle 1995). The ILCR was calculated by summing the individual cancer risk calculated for each route of exposure (direct ingestion, dermal contact and inhalation) using the following EPA models:

$$\text{ILCR (Ingestion)} = \frac{\sum \text{TEQ} \times \epsilon \left\{ \text{CSF}_{(\text{ingestion})} \times (\text{BW}/70)^{1/3} \right\} \times \text{ED} \times \text{EF} \times \text{IngR}}{\text{BW} \times \text{AT} \times \text{CF}}$$

$$\text{ILCR (Inhalation)} = \frac{\sum \text{TEQ} \times \epsilon \left\{ \text{CSF}_{(\text{inhalation})} \times (\text{BW}/70)^{1/3} \right\} \times \text{ED} \times \text{EF} \times \text{InhR}}{\text{BW} \times \text{AT} \times \text{PEF}}$$

$$\text{ILCR (Dermal contact)} = \frac{\sum \text{TEQ} \times \epsilon \left\{ \text{CSF}_{(\text{dermal contact})} \times (\text{BW}/70)^{1/3} \right\} \times \text{ABS} \times \text{AF} \times \text{SA} \times \text{ED} \times \text{EF}}{\text{BW} \times \text{AT} \times \text{CF}}$$

The details of values and units of parameters used in these formulae and the terminology have been explained in Table S1 (see ESM1), which were based on the risk assessment guidance of United States Environmental Protection Agency (U.S. EPA) and related publications.

### Source contribution using multiple linear regressions

The percentage contribution of individual sources was also assessed using principal component analysis (PCA) following multiple linear regression (MLR) as described by Larsen and Baker (2003). The multiple linear regression was performed on statistical package for social sciences (SPSS), using step-wise method of variable entering, which allows independent variable to enter the regression analysis, only if can significantly increase the correlation. To perform MLR (based on

basic Eq. (1)), PCA factor scores were taken as independent variables  $X_i$ , while the sum total of PAHs ( $\sum \text{PAHs}$ ) was taken as dependent variable ( $Y$ ). The dependent variable (i.e., PAHs) was first normally standardized using Eq. (2) and the influence of dependent variable on the independent variables was compared using regression coefficients. In the equation,  $X$  represent (any) variable, and  $x$  is the mean of  $X$ ,  $\sigma_x$  is the standard deviation of variable  $X$  and SND stands for standard normal deviation of  $X$ , which represents the distance in the units of standard deviations.

$$Y = \sum m_i X_i + b \quad (1)$$

$$\text{SND}(X) = (X - M_x) / \sigma_x \quad (2)$$

Application of Eq. (3), do not change the  $X_i$ , because the  $z$  (Eq. (4)) represents the standardized normal deviate of  $\sum$ PAHs, with the factor score having a mean of 0 and standard deviation of 1.

$$z = \sum Bi X_i \tag{3}$$

$$R^2 = \epsilon \sum Bi^2 \tag{4}$$

$$\text{Contribution (\%)} = 100 \times \left( \frac{Bi}{\sum Bi} \right) \tag{5}$$

$R^2$  is the squared multiple correlation and  $Bi$  is partial correlation coefficient. The average contribution of the  $i$ th source is calculated using the Eq. (5).

**Statistical analysis**

All the concentrations of individual and total PAHs detected in dust samples and diagnostic ratios calculated for source identification have been presented in mean±standard deviation, median, minimum, and maximum values; all the PAH results were presented on the basis of dry weight (d.w.). Normality of data was tested with the Kolmogorov-Smirnov (K-S) normality test. Principal component analysis (PCA) was also used for source identification using varimax rotation and extracting principal components having eigenvalues >1. Differences between sites were assessed using the Mann-Whitney’s  $U$  test. The concentration of B(a)P equivalent (BaP.Eq) were determined using the TEF provided by Nisbet and LaGoy (1992); U.S. EPA (1989; 2005; 2009), and Malcom and Dobson (1994). All statistical analyses were performed using SPSS software (version 20) for Windows.

**Results and discussion**

**Profile and abundance of PAHs in the exposure sites (WC and WR)**

The average concentrations of individual and total PAHs measured in surface dust samples together with the comparative analyses between sampling sites have been reported in Table 1. Generally, the concentrations of individual congeners of PAHs in surface dust samples of WC sites were higher than those measured in WR sites. B(a)P, a very high carcinogenic compound, was present in the dust samples from WC sites in the highest mean concentration (630 ng g<sup>-1</sup> d.w.) followed by Chry (617 ng g<sup>-1</sup> d.w.)>IP (564 ng g<sup>-1</sup> d.w.)>BghiP (525 ng g<sup>-1</sup> d.w.)>BaA (511 ng g<sup>-1</sup> d.w.)=Phe (511 ng g<sup>-1</sup> d.w.)>BbkF (498 ng g<sup>-1</sup> d.w.)>BeP (360 ng g<sup>-1</sup> d.w.)>Ant (277 ng g<sup>-1</sup> d.w.)>Naph (196 ng g<sup>-1</sup> d.w.)>Cor (164 ng g<sup>-1</sup>

d.w.)>Acy (144 ng g<sup>-1</sup> d.w.)>DBA (58 ng g<sup>-1</sup> d.w.). Significant differences in the concentrations of BaA, Fla, BeP, Cor, and IP were observed ( $p<0.05$ ). The BaP mean concentration in WC workplaces was almost eight times higher than the mean value found in WR exposure sites. In fact, it should be taken into account that BaP is known to be a tracer of high-temperature cooking and/or heating combustion (Yang 2003). Moreover, the average concentrations of  $\sum$ PAHs, sum of combustion origin PAHs ( $\sum$ COMB) and total sum of 7-carcinogenic PAHs ( $\sum$ 7-carcinogens) ranged from 1108 to 36710 ng g<sup>-1</sup> d.w., from 388 to 20394 ng g<sup>-1</sup> d.w., and from 823 to 30265 ng g<sup>-1</sup> d.w., respectively, and these values were 3.5, 4.3, and 4.1 times higher in WC-dust samples than in WR workplaces, respectively. In addition, the total sum of high molecular weight PAHs, ranging between 852 and 30455 ng g<sup>-1</sup> d.w., was significantly higher (almost 5.5 times higher,  $p<0.01$ ) in WC workplaces than in dust samples from WR exposure sites (Fig. 1).

The PAH concentrations showed differences between the two workplaces in term of levels and potential cancer risk associated with exposure to dust-bound PAHs. In particular, the WC workplaces represent unique exposure sites in some developing Asian countries such as India, Bangladesh, and Pakistan. In fact, the primitive technologies of cooking large amount of food (especially rice dishes) utilizing wood and coal biomass fuel makes this type of employment (also a family business) one of the most hazardous occupations in the Asian countries. The analytical results, which showed the high concentration of carcinogenic PAHs (in particular BaP), evidenced a high probability for subjects exposed in WC-exposure sites to develop cancer and/or respiratory diseases during routine exposure. In the case of residential cooking practices, the results showed slightly lower concentrations of carcinogenic PAHs. The difference in the PAH profile in the two exposure sites may be a reflection of choice of fuel selection by these two groups.

The diagnostic ratios of PAH isomeric compounds can be used for qualitative investigation of the source contribution (Kamal et al. 2015b). The specific ratios applied in this study to identify the sources are reported in Fig. 2.

The sum of the concentrations of the PAHs originating from the process of combustion ( $\sum$ COMB) (Fl, Pyr, BaA, BbkF, BaP, BeP, IP, and BghiP) divided by the total concentration of the PAHs ( $\sum$ PAHs) has often been used as a characteristic diagnostic parameter for PAHs produced by stationary combustion sources. The values of 0.73±0.09 and 0.63±0.12 for WCs and WRs, respectively, denoted that a considerable portion of PAHs mass could have originated from the combustion emission of the mobile sources.

The mean values of the ratio Fla/(Pyr+Fla) were 0.53±0.2 and 0.52±0.02 for WC and WR dust samples, respectively, indicating the contribute of coal wood combustion (De La Torre-Roche et al. 2009).

**Table 1** PAHs profile in dust samples from WC and WR sites and summary of diagnostic ratios used for source apportionment

Sites	WC (13)		WR (13)		$p^a$
	Mean±S.dev Med	Min–Max	Mean±S.dev Med	Min–Max	
Acenaphthene (Ace) (3) 0.001	21±8 20.6	4.4–37.3	47±17 33	11–213	0.26
Acenaphthylene (Acy) (3) 0.001	144±41 68.9	11.7–804	115±19.6 36	10–834.3	0.78
Anthracene (Ant) (3) 0.01	277±62 80.5	16.4–1433	62.8±26 39	12–229.6	0.16
Benzo( <i>ghi</i> )perylene (BghiP) (6) 0.01	525±101 327	30–1956	216±85 194	119–335	0.16
Benzo( <i>a</i> )anthracene (BaA) (4) 0.1	511±129 158	102–2847	84.7±34 95	19–177	<0.01
Benzo( <i>a</i> )pyrene (BaP) (5) 1	630±141 148	13.2–4148	78±26 61.6	13–199.9	0.56
Benzo( <i>b+k</i> )fluoranthene (BbkF) (5) 0.1	498±171 128	16.8–4022	206±56 219	10–387	0.16
Benzo( <i>e</i> )pyrene (BeP) (5) 0.01	360±89 92	12–2030	75±20 49	11.4–174	0.037
Chrysene (Chry) (4) 0.01	617±156 318.9	23.8–3048	160±45 149	17–383	0.19
Coronene (Cor) (6) 0.001	164±58 27.7	10.6–713	22±6.9 17.7	0.32–61	0.05
Dibenzo( <i>a,h</i> )anthracene (DBA) (5) 1	58±14 29	12.6–191	42±17 39	22.6–83	0.92
Fluoranthene (Fla) (4) 0.001	596±195 460	38.2–4791	83±28 63.6	11–244.6	0.007
Fluorene (Fl) (3) 0.001	106±65 63.6	21.2–358	71.5±32 35.99	13.6–318	0.26
Indeno(1,2,3- <i>c,d</i> )pyrene (IP) (6) 0.1	564±86 358.6	44.4–2147	158.5±85 121	48–365	0.02
Naphthalene (Naph) (3) 0.001	196±61 138	65.4–639	201±44 125	106–609	0.61
Phenanthrene (Phe) (3) 0.001	511±190 130.95	40.5–2283	113±34 46	10.9–781	0.11
Pyrene (Pyr) (4) 0.001	615±147 203.3	35–5274	72.8±18 54	11–223.9	0.13
$\Sigma$ COMB/ $\Sigma$ PAHs	0.73±0.09 0.7	0.50–0.84	0.63±0.12 0.72	0.37–0.84	0.28
$\Sigma$ 4,5,6 rings- PAHs/ $\Sigma$ PAHs	0.75±0.1 0.77	0.51–0.86	0.69±0.13 0.73	0.38–0.84	0.26
$\Sigma$ 7-carcinogens	3344±582 1107	388–20394	815±135 855	422–1276	0.01
$\Sigma$ COMB	4917±485 1736.5	823–30265	1133±383 1053	593–2078	0.011
$\Sigma$ HMWPAHs	4976±833 1760	852–30455	903±185 738	388–2112	<0.01
$\Sigma$ LMWPAHs	1255±187 648	209–5541	610±98 395	220–2232	0.37
$\Sigma$ LMW-PAHs/ $\Sigma$ HMW-PAHs	0.33±0.22 0.26	0.14–0.95	0.7±0.5 0.62	0.18–1.96	<0.01
$\Sigma$ PAHs	6395±552 2508	1108–36710	1808±849 1506	1067–3624	0.04
Ant/(Phe+Ant)	0.32±0.1 0.31	0.22–0.39	0.44±0.24 0.33	0.23–0.86	0.65
BaA/(BaA+Chry)	0.45±0.14 0.42	0.32–0.84	0.37±0.12 0.35	0.18–0.63	0.06
BaP/BghiP	0.85±0.7 0.94	0.03–2.1	0.47±0.5 0.3	0.04–1.67	0.52
BeP/(BeP+BaP)	0.44±0.16 0.40	0.33–0.9	0.49±0.23 0.43	0.09–0.91	0.28
Fla/(Pyr+Fla)	0.53±0.2	0.48–0.6	0.52±0.02	0.48–0.56	0.88

**Table 1** (continued)

Sites	WC (13)		WR (13)		<i>p</i> <sup>a</sup>
	Mean±S.dev Med	Min–Max	Mean±S.dev Med	Min–Max	
PAH congeners (aromatic rings #)					
TEF					
IP/(IP+BghiP)	0.52 0.52±0.03 0.51	0.50–0.6	0.52 0.42±0.09 0.44	0.22–0.53	<0.01
Phe/(Phe+Ant)	0.68±0.1 0.69	0.61–0.78	0.56±0.24 0.67	0.14–0.77	0.65

Toxic equivalency factor (TEF) for individual PAHs relative to BaP as reported by Nisbet and LaGoy (1992), except TEF values for Cor and BeP which were adopted from Malcom and Dobson (1994)

∑COMB=PAHs of combustion origin, i.e., BaA, BaP, BbkF, BeP, BghiP, Chry, Fla, IP, Pyr (Prah and Carpenter 1983)

∑7-carcinogenic PAHs=BaA, BaP, Bb+kF, BghiP, IP, Pyr

*S.dev* standard deviation, *Min-Max* minimum and maximum values, *Med* median value, *p* probability value, significant when <0.05, ∑PAHs sum of non-alkylated total PAHs, *LMW-PAHs* low molecular weight PAHs, *HMW-PAHs* high molecular weight PAHs

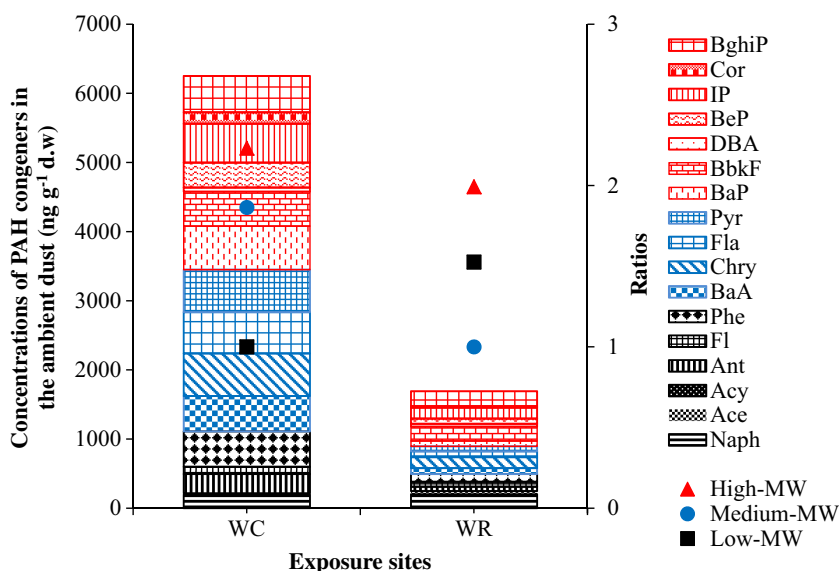
The average values of IP/(IP+BghiP) ratios obtained in this work were 0.52±0.03 for WCs and 0.42±0.09 for WRs. Values previously documented of 0.18 for gasoline emissions (Kavouras et al. 1999; Pio et al. 2001, Alves et al. 2001), 0.37–0.7 for diesel emissions (Kavouras et al. 1999, 2001; Pio et al. 2001; Cerededa-Balic et al. 2002), 0.56 for coal powder (Pio et al. 2001), and 0.62 for wood combustion emissions (Gogou et al. 1996) denote the importance of diesel and coal combustion emissions in the study sites. A petrogenic source was also suggested for both sites from the Ant/(Phe+Ant) >0.1 ratios (Pies et al. 2008). The BaA/(BaA+Chry) ratio is generally used to evaluate the contribution of vehicular emissions. The mean value of this ratio in both scenarios indicated the importance of gasoline and diesel engine emissions to the atmosphere.

Moreover, ∑4,5,6-rings PAHs/∑PAHs ratios >0.5 also suggested coal and soft wood combustion (Biache et al. 2014) origins. However the BaP/BghiP ratios showed different results for WCs and WRs, with a more evident traffic signature in the former and a probable mix signature of gasoline and coal combustion processes in the later (Hanedar et al. 2013; Han et al. 2009; Allen et al. 2008), as expected taking into account that most of the WC exposure sites are mainly located on the road, while WR exposure sites are located in residential and enclosed areas.

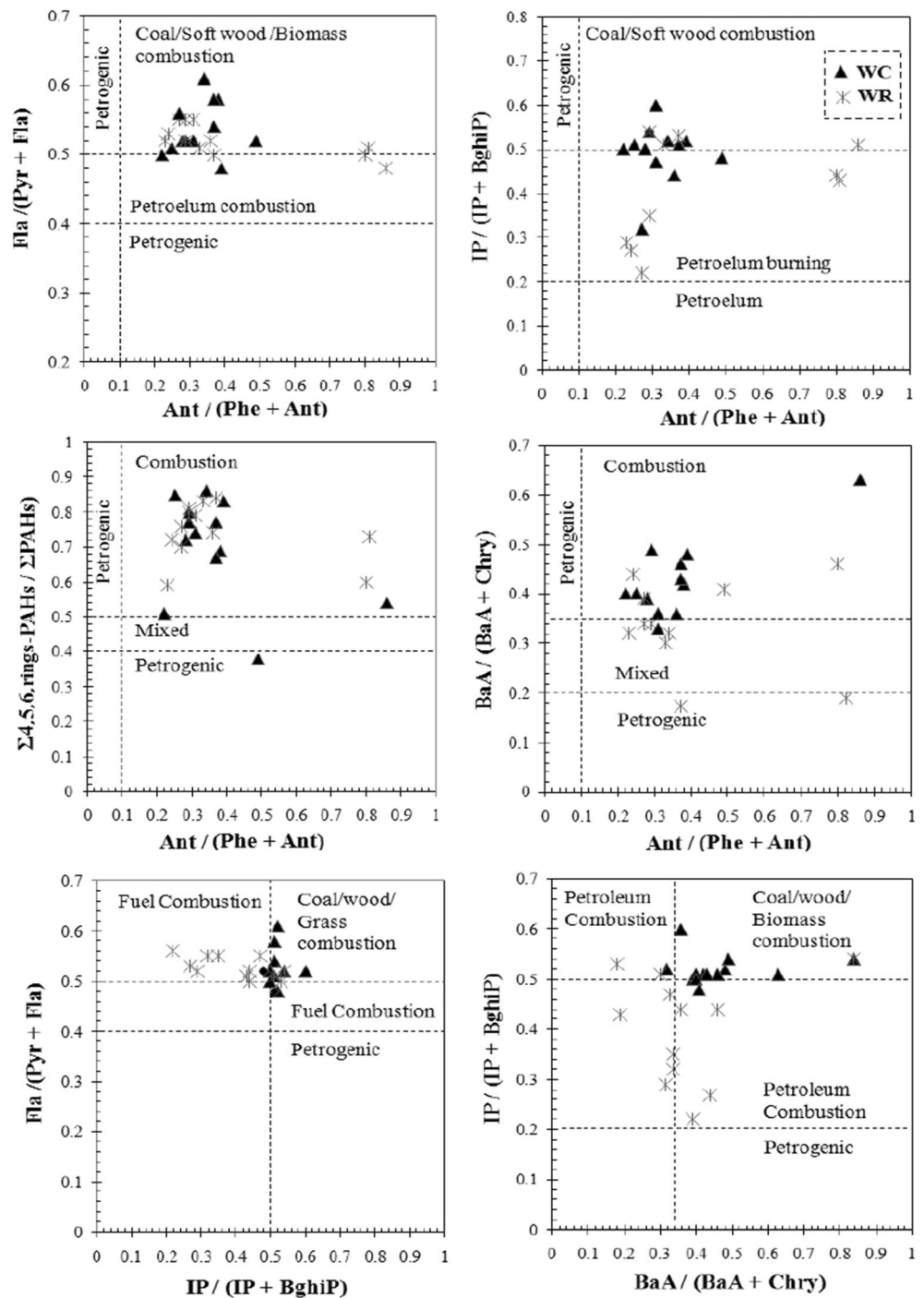
**Source apportionment using PCA-MLR**

Considering the suitability of PAH data for principal component analysis (PCA) and multiple linear regressions (MLR) analysis of surface dust samples of WC and WR exposure sites,

**Fig. 1** PAHs profile based on low, middle and high molecular weight (MW) distribution in residential household (WR) and professional (WC) workplace-dust samples



**Fig. 2** Cross plots of diagnostic ratios for source apportionment of PAHs in WR and WC exposure sites

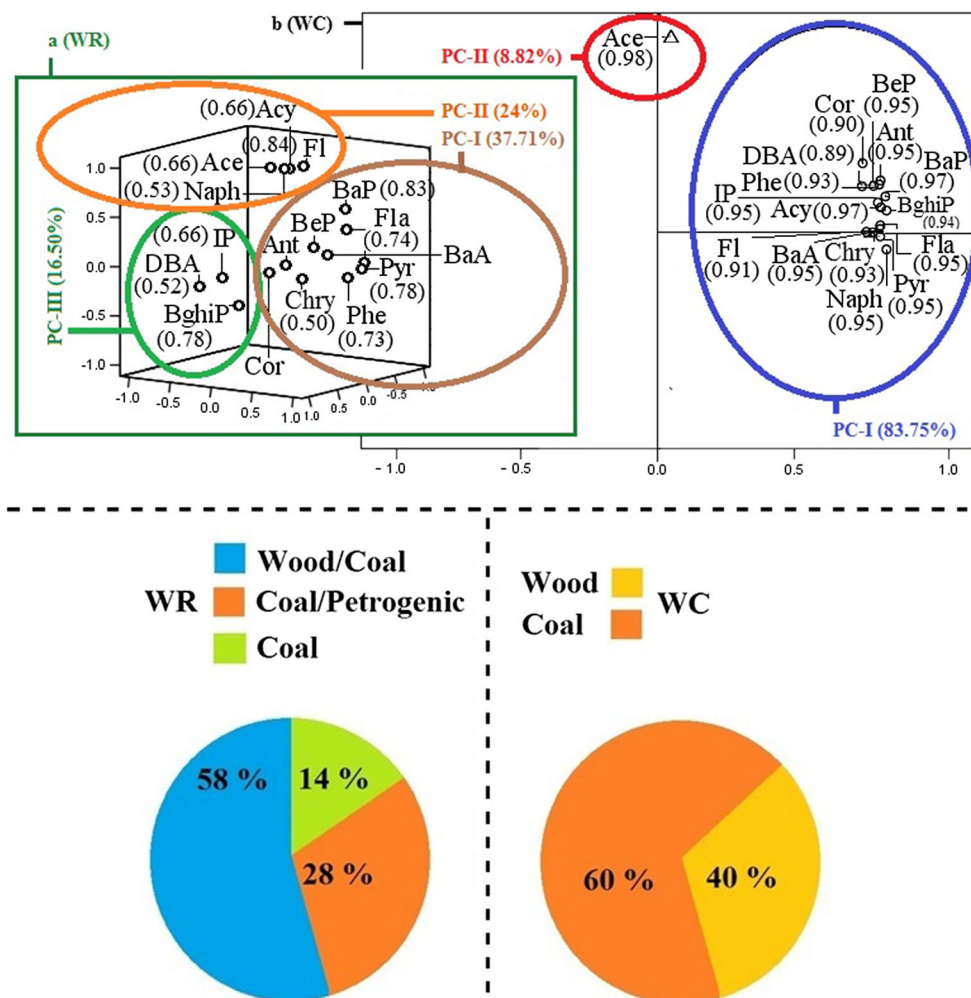


it was employed to unveil independent source tracers within each group separately (Field 2009). Based on the PCA-MLR, two factors and three factors were extracted for the surface dust data from the WC and WR exposure sites, respectively.

For WC dust (Fig. 2b), factor 1 (PC-I) explained approximately 83.75 % of total variance with high loading of all investigated PAHs, in particular B(a)P, except the Ace. These compounds could be attributed to the mixture of combustion source with high temperature, such as fossil fuel

combustion (coal or gasoline combustion). The DBA is often known as indication of large scale combustion of coal (Fang et al. 2004). Factor 2 (PC-II), representing 8.82 % of the total variance, is characterized by Ace which is representative of wood combustion source. According the results of MLR, wood and coal contributed to the ~40 and ~60 % of total PAHs, respectively, in WC-exposures sites (Fig. 2).

For WR dust samples (Fig. 3a), three factors were extracted. Factor 1(PC-I), accounted for 37.71 % of total variance



**Fig. 3** PCA-MLR analysis of dust-bound PAHs from WR (a) and WC (b) exposure sites

with high loading of 5- to 6-rings PAHs (BaP, BeP, Fla, Ant, Pyr, BaA, Chry, Phe), which could have originated from fossil fuel combustion, coal combustion, or gasoline vehicle emission. Factor 2 (PC-II), which explains 24 % of the total variance, displayed a high abundance of Acy, Ace, Fl, and Naph, which could be associated with straw combustion (Zhang et al. 2008) or wood burning (Fang et al. 2004). Thus, factor 2 could be considered as a biomass combustion source. Factor 3 (i.e., PC-III, 16.50 % of total variance) is characterized by IP, DBA, and BghiP, indicating a possible source of coal combustion (Harrison et al. 1996; Simcik et al. 1999; Mastral et al. 1996). In general, high molecular weight PAHs may arise from the pyrogenic emission sources (Bouloubassi et al. 2012; Zakaria et al. 2002).

The MLR further revealed that overall ~14 % PAHs were contributed by coal, and 28 % by coal/petrogenic mixed sources and large proportion (58 %) PAHs in WR exposure sites were contributed by wood and coal mixed sources (Figs. 3 and 4). The prevalence of ΣCOMB-PAHs was also confirmed from Fig. 4, which indicated that in both sites (>70–80 %), PAHs were originated from the combustion of biomass fuels.

**Health risk associated with exposure to dust-bound PAHs**

The incremental lifetime cancer risk (ILCR) model was used as a tool to evaluate human health risk from the exposure to dust particle-bound PAHs. In particular, there are few studies regarding the characterization of PAHs in soil/dust samples from different occupational or residential settings in Pakistan.

Probabilistic risk assessment for personal exposure to carcinogenic PAHs via dermal contact, ingestion, and inhalation pathways showed that an ILCR between 10<sup>-6</sup> and 10<sup>-4</sup> indicates potential risk, whereas ILCR greater than 10<sup>-4</sup> suggests high potential health risk. The acceptable level is equal or lower than 10<sup>-6</sup> (see Table S1, in ESM1).

Our estimated values, which are reported in Table 2, are higher than our previously reported ILCR values for exposure of brick kiln workers to dust-bound PAHs (Kamal et al. 2014).

The ILCR calculations are based on the model parameters specified for outdoor and indoor working classes exposed to dust-bound contaminant according to U.S. EPA (Table 1). Both adults and children (child labor) could be exposed to dust-bound PAHs in WC cooking sites, and similarly in WR



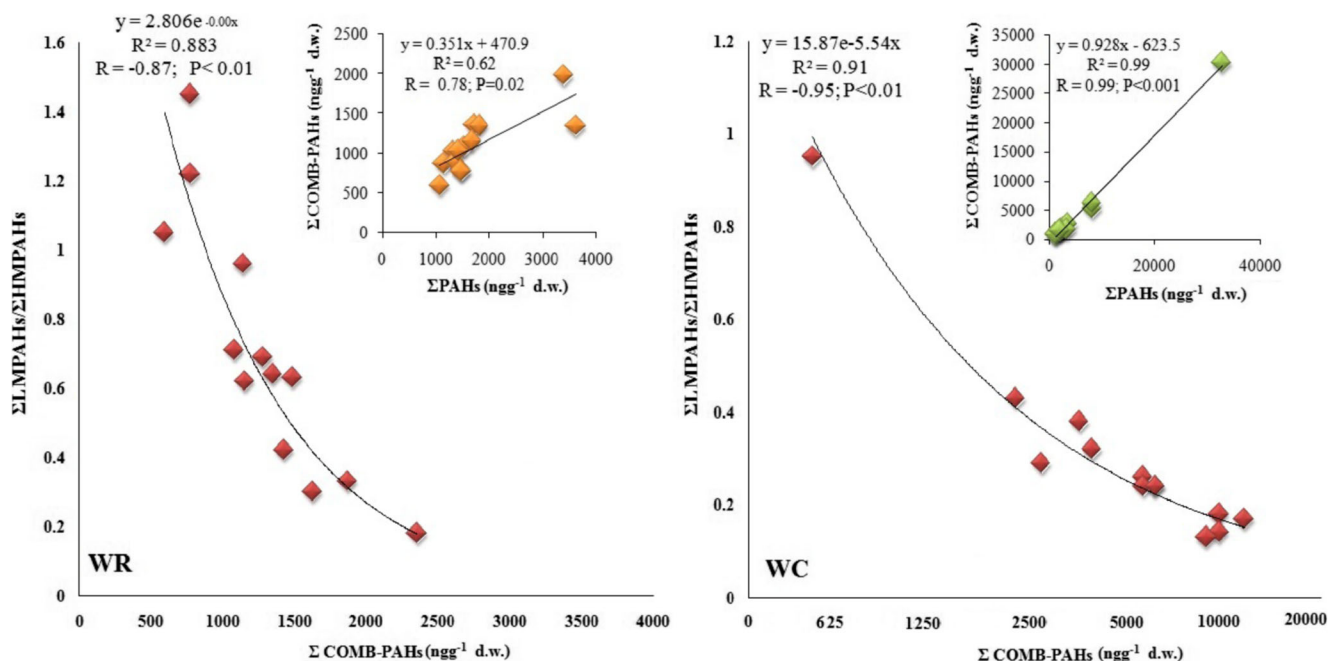


Fig. 4 Association between the ratios of  $\Sigma$ LMW-PAHs,  $\Sigma$ COMB-PAHs, and PAHs and  $\Sigma$ COMB-PAHs in the WR and WC exposure sites

residential households, as a routine exposure scenario. Thus, the ILCR values were calculated for both groups to compare the risk to both children and adult subjects. Children in particular, represents a sub-group which is the most vulnerable to the hazards associated with the exposure to pollutants (Martí-Cid et al. 2008). Considering the ambience of these cooking places, both WC and WR workplaces can be categorized into semi-outdoor exposure sites, since no single description of workplace exists in all place, rather the workplaces are designed according to the economical conditions and special variability. However, regardless of the economical differences, the workers are highly exposed to the combustion emission of biomass fuel.

The results of ILCR were almost similar for children and adults; however, the risk for children could be relatively high in some cases.

The ILCR values for different exposure pathways can be summarized as follows: WC (dermal>ingestion>inhalation)>WR (dermal>ingestion>inhalation). Cancer risk levels via dermal contact were ranging from  $4.58 \times 10^{-4}$  to  $2.70 \times 10^{-2}$  with an average of  $4.45 \times 10^{-3}$  and between  $3.36 \times 10^{-4}$  and  $1.98 \times 10^{-2}$  with an average of  $3.26 \times 10^{-3}$  for adults and children in WC sampling sites, respectively. The risk levels were slightly higher in WC-exposure sites than in those in the WR-exposure sites (ranging between  $4.59 \times 10^{-4}$  and  $1.42 \times 10^{-3}$  for adults and between  $3.36 \times 10^{-4}$  and  $1.04 \times$

Table 2 Incremental lifetime cancers from individual and combined dermal, inhalation, and ingestion routes of exposure, calculated for adults and children

		Dermal		Inhalation		Ingestion		Total	
		Adult	Children	Adults	Children	Adults	Children	Adults	Children
WC	Mean	4.45E-03	3.26E-03	1.86E-07	5.11E-08	2.40E-03	1.76E-03	6.85E-03	5.02E-03
	St. dev	4.92E-03	4.81E-03	1.32E-07	4.11E-08	2.27E-03	1.13E-03	1.22E-02	4.94E-03
	Median	1.13E-03	1.29E-04	4.73E-08	1.30E-08	6.10E-04	4.47E-04	1.74E-03	1.28E-03
	Min	4.58E-04	3.36E-04	1.92E-08	5.27E-09	2.47E-04	1.81E-04	7.05E-04	5.17E-04
	Max	2.70E-02	1.98E-02	1.13E-06	3.10E-07	1.45E-02	1.07E-02	4.15E-02	3.05E-02
WR	Mean	8.18E-04	6.00E-04	3.42E-08	9.41E-09	4.41E-04	3.23E-04	1.26E-03	9.23E-04
	St. dev	3.24E-04	2.38E-04	1.36E-08	3.73E-09	1.75E-04	1.28E-04	4.99E-04	3.66E-04
	Median	7.20E-04	5.28E-04	3.01E-08	8.28E-09	3.89E-04	2.85E-04	1.11E-03	8.13E-04
	Min	4.59E-04	3.36E-04	1.92E-08	5.28E-09	2.48E-04	1.81E-04	7.07E-04	5.17E-04
	Max	1.42E-03	1.04E-03	5.95E-08	1.64E-08	7.68E-04	5.62E-04	2.19E-03	1.60E-03

$10^{-3}$  for children). Similarly, cancer risk levels via ingestion pathway in WC sites (ranging from  $2.47 \times 10^{-4}$  to  $1.45 \times 10^{-2}$  for adults and from  $1.81 \times 10^{-4}$  to  $1.07 \times 10^{-2}$  for children) were also somewhat higher than those in WR-exposure sites ranging between  $2.48 \times 10^{-4}$  and  $7.68 \times 10^{-4}$  and between  $1.81 \times 10^{-4}$  and  $5.62 \times 10^{-4}$  for adults and children, respectively.

Minimum or negligible (i.e.,  $>10^{-6}$ ) cancer risk levels were observed for inhalation pathways, ranging between  $1.92 \times 10^{-8}$  and  $1.13 \times 10^{-6}$  for adults and between  $5.27 \times 10^{-9}$  and  $3.10 \times 10^{-7}$  for children exposed in WC-exposure sites, and from  $1.92 \times 10^{-8}$  to  $5.95 \times 10^{-8}$  for adults and from  $5.28 \times 10^{-9}$  to  $1.64 \times 10^{-8}$  for children exposed in WR-exposure sites. The total cancer risk due to PAH exposure from all the routes was  $6.85 \times 10^{-3}$  and  $5.02 \times 10^{-3}$  for adults and children exposed in WC-exposure sites, and  $1.26 \times 10^{-3}$  and  $9.23 \times 10^{-4}$  for adults and children exposed in WR-exposure sites.

The total ILCR values were compared with U.S. EPA's acceptable risk range of  $1 \times 10^{-6}$ – $1 \times 10^{-4}$ . For both exposed population, the ILCR values exceeded the acceptable safe values proposed by U.S. EPA. The results of cancer risk assessment obtained in this study also signify the implication of lung cancer/respiratory disorders in exposed population.

## Conclusions

In Pakistan, traditional and primitive household and professional cooking practices are widespread in the country, which is attributable both to poverty and severe energy crisis, present in the country for many years. Analysis of surface dust from residential and occupational areas showed high concentrations of PAHs in both the exposure sites. In particular, B(a)P, which is a potent carcinogenic PAH, was found in highest concentrations. Comparatively, the professional cooking sites present high concentrations of PAHs and consequent health risks compared to residential sites; however, both sites pose high potential lifetime cancer risk to humans from exposure to dust-bound PAHs in particular via both ingestion and dermal contact pathways. The issue needs control/remediation or preventive measures on urgent basis, by introducing pollution abatement policies and improving the conditions of household and occupational cooking environments.

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## References

Allen AG, da Rocha GO, Cardoso AA, Paterlini WC, Machado CMD, de Andrade JB (2008) Atmospheric particulate polycyclic aromatic

hydrocarbons from road transport in southeast Brazil. *Transp Res D* 13:483–490

Biache C, Mansuy-Huault L, Faurea P (2014) Impact of oxidation and biodegradation on the most commonly used polycyclic aromatic hydrocarbon (PAH) diagnostic ratios: implications for the source identifications. *J Hazard Mater* 267:31–39

Bouloubassi I, Roussiez V, Azzoug M, Lorre A (2012) Sources, dispersal pathways and mass budget of sedimentary polycyclic aromatic hydrocarbons (PAH) in the NW Mediterranean margin, Gulf of Lions. *Mar Chem* 142–144:18–28

Cereceda-Balic F, Kleist E, Prast H, Schlimper H, Engel H, Gunther K (2002) Description and evaluation of a sampling system for long-time monitoring of PAHs wet deposition. *Chemosphere* 49(3):331–340

Chen H-Y et al (2013) Source apportionment for sediment PAHs from the Daliao River (China) using an extended fit measurement mode of chemical mass balance model. *Ecotoxicol Environ Saf* 88:148–154

Cincinelli A, Stefani S, Costantini S, Lepri L (2004) Characterization of n-alkanes and PAHs in PM10 samples in Prato (Italy). *Ann Chim* 94:281–293

De La Torre-Roche RJ, Lee WY, Campos-Diaz SI (2009) Soil-borne polycyclic aromatic hydrocarbons in El Paso, Texas: analysis of a potential problem in the United States/Mexico border region. *J Hazard Mater* 163:946–958

Fang G, Chang KF, Lu C, Bai H (2004) Estimation of PAHs dry deposition and BaP toxic equivalency factors (TEFs) study at Urban, Industry Park and rural sampling sites in central Taiwan, Taichung. *Chemosphere* 55:787–796

Field A. *Discovery statistic using SPSS* (2009); 3rd Edition.

Gogou AI, Stratigakis N, Kanakidou M, Stephanou EG (1996) Organic aerosols in Eastern Mediterranean: components source reconciliation by using molecular markers and atmospheric back trajectories. *Organic Geochemistry* 25:79–96

Han B, Bai Z, Guo G, Wang F, Li F, Liu Q, Ji Y, Li X, Hu Y (2009) Characterization of PM10 fraction of road dust for polycyclic aromatic hydrocarbons (PAHs) from Anshan. *China J Hazard Mater* 170:934–940

Hanedar A, Alp K, Kaynak B, Avşar E (2013) Toxicity evaluation and source apportionment of polycyclic aromatic hydrocarbons (PAHs) at three stations in Istanbul, Turkey. *Scie Total Environ* 488–489: 437–446. doi:10.1016/j.scitotenv.2013.11.123

Harrison RM, Smith DJT, Luhana L (1996) Source apportionment of atmospheric polycyclic aromatic hydrocarbons collected from an urban location in Birmingham, UK. *Environ Sci Technol* 30(3): 825–832

Kamal A, Malik RN, Martellini T, Cincinelli A (2014a) Cancer risk evaluation of brick kiln workers exposed to dust bound PAHs in Punjab province, Pakistan. *Sci Total Environ* 493:562–570

Kamal A, Malik RN, Martellini T, Cincinelli A (2014b) PAHs exposure biomarkers are associated with clinico-chemical changes in the brick kiln workers in Pakistan. *Sci Total Environ* 490:521–527

Kamal A, Qamar K, Anwar M, Gulfranz M, Malik RN (2015a) PAHs exposure and oxidative stress indicators of humans exposed to traffic pollution in Lahore city (Pakistan). *Chemosphere* 120:59–67. doi:10.1016/j.chemosphere.2014.05.021

Kamal A, Cincinelli A, Martellini T, Malik RN (2015b) Source, profile and carcinogenic risk assessment for cohorts occupationally exposed to dust-bound PAHs in Lahore and Rawalpindi cities (Punjab province, Pakistan). *Environ Sci Pollut Res*. doi:10.1007/s11356-015-4215-2

Kavouras IG, Lawrence J, Koutrakis P, Stephanou EG, Oyola P (1999) Measurement of particulate aliphatic and polynuclear aromatic hydrocarbons in Santiago de Chile: source reconciliation and evaluation of sampling artifacts. *Atmos Environ* 33(30):4977–4986

Kavouras IG, Koutrakis P, Tsapakis M, Lagoudaki E, Stephanou EG, Baer DV et al (2001) Source apportionment of urban particulate

- aliphatic and polynuclear aromatic hydrocarbons (PAHs) using multivariate methods. *Environ Sci Technol* 35:2288–2294
- Larsen RK III, Baker JE (2003) Source apportionment of polycyclic aromatic hydrocarbons in the urban atmosphere: a comparison of three methods. *Environ Sci Technol* 37:1873–1881
- Malcom HM, Dobson S (1994) The calculation of an environmental assessment level (EAL) for atmospheric PAHs using relative potencies. London, UK7 Department of the Environment 34–46.
- Martellini T, Giannoni M, Lepri L, Katsoyiannis A, Cincinelli A (2012) One year intensive PM 2.5 bound polycyclic aromatic hydrocarbons monitoring in the area of Tuscany, Italy. Concentrations, source understanding and implications. *Environ Pollut* 164:252–258
- Martí-Cid R, Llobet JM, Castell V, Domingo JL (2008) Evolution of the dietary exposure to polycyclic aromatic hydrocarbons in Catalonia, Spain. *Food Chem Toxicol* 46:3163–3171
- Mastral AM, Callen M, Murillo R (1996) Assessment of PAH emissions as a function of coal combustion variables. *Fuel* 75:1533–1536
- Meiners RE and Yandle B (1995) Rowman & Littlefield Publishers, Taking the environment seriously. Inc. USA. 3 Henrietta Street, London WC2E 8 LU, England.
- Murakami M, Nakajima F, Furumai H (2005) Size- and density-distributions and sources of polycyclic aromatic hydrocarbons in urban road dust. *Chemosphere* 61:783–791
- Ngo Viet V, Michel J, Gujsaite V, Latifi A, Simonnot M-O (2014) Parameters describing nonequilibrium transport of polycyclic aromatic hydrocarbons through contaminated soil columns: estimability analysis, correlation, and optimization. *J Contam Hydrol* 158:93–109
- Nisbet ICT, LaGoy PK (1992) Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs). *Regul Toxicol Pharmacol* 16:290–300
- Pies C, Hoffmann B, Petrowsky J, Yang Y, Ternes TA, Hofmann T (2008) Characterization and source identification of polycyclic aromatic hydrocarbons (PAHs) in river bank soils. *Chemosphere* 72:1594–1601
- Pio CA, Alves CA, Duarte AC (2001) Identification, abundance and origin of atmospheric organic particulate matter in a Portuguese rural area. *Atmos Environ* 35:1365–1375
- Prahl FG, Carpenter R (1983) Polycyclic aromatic hydrocarbon (PAH)-phase associations in Washington coastal sediment. *Geochim Cosmochim Acta* 47:1013–1023
- Simcik MF, Eisenreich SJ, Liou PJ (1999) Source apportionment and source/sink relationships of PAHs in the coastal atmosphere of Chicago and Lake Michigan. *Atmos Environ* 33:5071–5079
- Szabova E, Zeljenkova D, Nescákova E, Simko M, Turecky L (2008) Polycyclic aromatic hydrocarbons and occupational risk factor. *Reprod Toxicol* 26:74
- U.S. EPA (1989) (United States Environmental Protection Agency) Risk assessment guidance for Superfund. Vol. I: Human health evaluation manual. EPA/540/1-89/002. Office of Solid Waste and Emergency Response, Washington, DC
- U.S. EPA (1991) Human health evaluation manual, supplemental guidance: “Standard default exposure factors”. OSWER Directive 9285: 6–03
- U.S. EPA (2005) Guidance on selecting age groups for monitoring and assessing childhood exposures to environ contaminants; EPA/630/P-03/003F.
- U.S. EPA (2009) (United States Environmental Protection Agency) Risk assessment guidance for Superfund. Vol. I: human health evaluation manual (F, Supplemental guidance for inhalation risk assessment) EPA/540/R/070/002, Office of Superfund Remediation and Technology Innovation, Washington, DC, [http://www.epa.gov/oswer/riskassessment/ragsf/pdf/partf\\_200901\\_final.pdf](http://www.epa.gov/oswer/riskassessment/ragsf/pdf/partf_200901_final.pdf) Accessed 12-1-2013.
- Wild SR, Jones KC (1995) Polynuclear aromatic hydrocarbons in the United Kingdom environment: a preliminary source inventory and budget. *Environ Pollut* 88:91–108
- Yang XS (2003) Study on the source apportionment of polycyclic aromatic hydrocarbons on airborne particulate at representative areas in Nanjing City Paper
- Yang Y, Woodward LA, Li QX, Wang J (2014) Concentrations, source and risk assessment of polycyclic aromatic hydrocarbons in soils from Midway Atoll, North Pacific Ocean. *PLoS One* 9(1):86441
- Zakaria MP, Takada H, Tsutsumi S, Ohno K, Yamada J, Kouno E, Kumata H (2002) Distribution of polycyclic aromatic hydrocarbons (PAHs) in rivers and estuaries in Malaysia: a widespread input of petrogenic PAHs. *Environ Sci Technol* 36:1907–1918
- Zhang S, Zhang W, ShenY WK, Hu L, Wang X (2008) Dry deposition of atmospheric polycyclic aromatic hydrocarbons (PAHs) in the south east suburb of Beijing, China. *Atmos Res* 89:138–148