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Source apportionment of the carcinogenic potential of polycyclic aromatic hydrocarbons (PAH) associated to airborne PM10 by a PMF model

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Abstract

In order to perform a study of the carcinogenic potential of PAH, benzo(a)pyrene equivalent (BaP-eq) concentration was calculated and modelled by a receptor model based on Positive Matrix Factorization (PMF). 19 polycyclic aromatic hydrocarbons (PAH) associated to airborne PM10 of Zaragoza, Spain, were quantified during the sampling period 2001-2009 and used as potential variables by the PMF model. Afterwards, multiple linear regression analysis (MLR) was used to quantify the potential sources of BaP-eq. Five sources were obtained as the optimal solution and vehicular emission was identified as the main carcinogenic source (35%) followed by heavy-duty vehicles (28%), light-oil combustion (18%), natural gas (10%) and coal combustion (9%). Two of the most prevailing directions contributing to this carcinogenic character were the NE and N directions associated with a highway, industrial parks and a paper factory. The lifetime lung cancer risk exceeded the unit risk of 8.7 x 10^{-5} per ng/m^3 BaP in both, winter and autumn seasons and the most contributing source was the vehicular emission factor becoming an important issue in control strategies.

Keywords PM10· PAH· BaP equivalent· PMF· Receptor modelling· Lifetime lung cancer risk

Introduction

Polycyclic aromatic hydrocarbons (PAH) are produced by incomplete combustion of organic material. Their origin can be natural (forest fires, volcanic eruption) or anthropogenic, mainly from fossil fuel emissions, which can be classified as stationary and mobile sources (Ravindra et al. 2008).

PAH are distributed in the atmosphere between gas and particulate phases, depending on the volatility of the PAH species. Higher condensed molecules with four and more rings are particle bound, whereas smaller PAH mainly remain in the gas phase (Westerholm et al. 1988; Beak et al. 1991). In the atmosphere, they can undergo degradation by photochemical reactions and they can deposit on the ground, both by dry and/or wet deposition. Due to their relative volatility and their physiochemical properties, PAH can re-volatilize, undergoing long-range transport when adsorbed and/or absorbed in atmospheric particles (Zelenyuk et al. 2012).

One of the main concerns of the human exposure at PAH is due to their carcinogenic and mutagenic properties (Boström et al. 2002; Bourotte et al. 2005; Ravindra et al. 2006; Callén et al. 2011). Most of the probable human carcinogenic PAH are found to be associated with particulate matter (Callén et al. 2008a), especially in fine mode particles in ambient air (Ravindra et al. 2008). For this reason, humans are exposed to PAH mainly through respiratory tract, although PAH can enter in our organism through digestive tract following intake of food containing PAH (e.g. grilled meat or vegetables grown near areas with intense traffic…) or through the skin by contact with derivate of petroleum (tar, pitch…). Once inside, PAH can be transformed into reactive electrophilic intermediates that can form adducts with DNA, RNA and proteins, and induce mutations which can cause tumors in several organs (the lungs, the esophagus, the colon, the pancreas, the skin, the bladder…) (Boström et al. 2002).

The IARC (International Agency for Research on Cancer) has classified several PAH with respect to their carcinogenicity to humans, inclusive of: BaP (group 1 toxic, carcinogenic); DahA (group 2A, probably carcinogenic); BbF, BkF, BjF, BaA, Chry, IcdP (group 2B toxic, possibly carcinogenic to humans); Flt, Cor, Py and

BghiP (group 3, carcinogenicity not classifiable) (IARC, 2012). In order to establish a metric that estimates the total carcinogenic potential of atmospheric PAH, the concept of BaP-equivalent toxicity (BaP-eq) was established, whereby the toxicity of several PAH species has been quantified with respect to BaP, a reference specie with well characterized toxicity (Nisbet and LaGoy 1992; Larsen and Larsen 1998). Since PAH health risk comes from exposure to a number of species, the aggregate BaP-eq metric, calculated from an appropriately large suite of PAH species, allows a more accurate risk assessment of ambient PAH concentrations compared with Directive 2004/107/EC, which establishes a target value of exposure only for BaP (annual average of 1 ng/m^3 sampled in the PM10 size fraction).

In the present work, the main anthropogenic sources contributing to PM10-associated PAH in Zaragoza, Spain were identified and apportioned with respect to their carcinogenic potential (BaP-eq) during 2001-2009. According to our knowledge, this is the first time that source contributions to BaP-eq are determined using a combined PMF/MLR model. The locations of source types identified by PMF/MLR as impacting the sampling site were estimated with respect to wind directions by using the Conditional Probability Function (CPF) method (Kim and Hopke 2004; Vestenius et al. 2011). An assessment of the lifetime lung cancer risk of PAH exposure by inhalation in Zaragoza was carried out for each one of the sources obtained by the PMF/MLR model and for the BaP-eq modeled.

Experimental

Study area and sampling description

The study was performed in the Rio Ebro campus of a medium-size city, Zaragoza (population 679,624) (INE, 2012) located in the North-East of Spain, (41°39′49.38″N; 0°53′16.68″W) close to a heavy traffic motorway (approximately 50 m) and influenced by several industrial parks, four paper factories and two waste water treatment plants located in the surroundings of the city. The sampling site has been already described in previous articles (Callén et al. 2008a; Callén et al. 2012) (Fig. 1) and the prevailing winds were from the northwest direction associated with a cold northerly wind called “cierzo”.

Samples were collected by using a Graseby Andersen high-volume air sampler (1.13 m^3/min) provided with a PM10 cut off inlet to collect particulate phase on a teflon-coated, glass fibre filter (0.6 µm pore size; 20.5 cm × 25.5 cm) (Callén et al. 2008a; Callén et al. 2008b). The PM10 was determined by gravimetric method after conditioning the filter according to EN12341:1998.

A total of 111 integrated 24-hr samples were collected over several sampling periods from 2001 to 2009 (bi-weekly from 15 November 2001 to 16 July, 2002; 7 April 2003 to 5 July 2004; daily from 23 May – 8 June 2008 and 13 – 27 January 2009). An ANOVA-test was performed in order to infer whether the sources contributing to the sampling site were likely to have remained constant over the 2001-2009 sampling time period and no statistically significant change in total sampled PAH and BaP-eq were found (Table S1, Supporting Information), demonstrating the suitability of using the entire sampling time series in the PMF receptor model.

Extraction and analysis

A total of 19 PAH species, listed in Table 1, were quantified by gas chromatography mass-spectrometry mass-spectrometry detection (GC-MS-MS) according to a previous publication (Callén et al. 2008b). Briefly, samples were extracted by Soxhlet for 24 hours with dichloromethane (DCM) after the addition of a surrogate deuterated solution (An-d_{10}, BaP-d_{12}, BghiP-d_{12}) to account for any losses of analytes during sample treatment. Samples were eluted through a silica gel column with DCM to finally exchange the solvent to hexane. p-terphenyl native was added as recovery internal standard previous to GC-MS-MS quantification (Callén et al. 2007; Callén et al. 2008a; Callén et al. 2008b). For quantitative determinations, a set of PAH standard mixtures were spiked with the same surrogate deuterated solution as the one used for samples and average response factors were calculated for all analytes with respect to the closest deuterated internal standard. A summary of the mean and standard deviation for each analysed PAH is shown in Fig. 2.

Quality control and quality assurance

Field blank determinations were used for background correction on the sampled filters and the detection and quantification limits were determined according to three and ten times the blank standard deviation (with detection limits ranging from 1 pg m^{-3} to 156 pgm^{-3}). Analyses of standard reference materials, SRM1944 and SRM 1649a provided by the National Institute of Standards and Technology (NIST), were carried out in order to check the analytical accuracy and precision of PAH quantification. Measured values were satisfactorily comparable to certified values with deviations lower than 20% for PAH (with the exception of Chry, probably due to the interference of triphenylene (40%)).

Data analysis

Benzo(a)pyrene equivalent concentration

BaP-eq is a useful metric to quantitatively assess the carcinogenic health risk of PAH (Boström et al, 2002; Callén et al, 2011). BaP-eq was calculated for each sample by multiplying the concentration of each species by its Toxic Equivalency Factor (TEF, see Table 1; Larsen and Larsen, 1998) and summing across the species suite (equation 1), adapting the method of Jung et al. (2010).

[BaP-eq] = [An]*0.0005 + [Phe]*0.0005 + [2+2/4MePhe]*0.0005+ [9MePhe]*0.0005 + [1MePhe]*0.0005+ [DiMePhe]*0.0005 [BaA]*0.005 + [Chry]*0.03 + [Flt]*0.05+ [Py]*0.001 + [BaP]*1 + [BeP]*0.002 + [B(b+k+j)F]*0.067 + [IcdP+DahA]*0.55 + [BghiP]*0.02 + [Cor]*0.01 (1)

TEF values for B(b+j+k)F and IcdP+DahA were calculated as the average TEF values between BbF, BjF and BkF, and IcdP and DahA, respectively. Although Larsen and Larsen (1998) were not able to provide TEF for MePhe and DiMePhe that were analyzed in this study, it was assumed to assign their TEF according to Phe.

PMF model

Anthropogenic source contributions to modelled BaP-eq were determined by use of a combined PMF/MLR receptor model. Firstly, an identification of the main sources contributing to atmospheric PAH at the sampling site were

determined by applying the US EPA PMF 3.0 model (Paatero and Tapper 1994; Paatero 1997) to the 2001-2009 data set (111 samples, 16 individual sampled PAH used as predictors, no total variable used). Afterwards, with the sampled BaP-eq known for each sample, MLR was performed to regress the BaP-eq against the factor scores obtained by the PMF model (dependent variable=total sampled BaP-eq, from equation 1: independent variables= modelled contribution of each source from PMF model output). In addition to resolving factor profiles that could be interpreted as source types, the regression coefficients were also used to yield the contribution among the resolved sources to BaP-eq over the sampled time series.

PMF model is a multivariate factor analysis software that decomposes a matrix X (n x m) into factor contribution matrix G (n x p), factor profiles matrix F (p x m) and residual matrix E (n x m), where n is the number of samples, m is the number of species and p is the number of sources (equation 2).

$$X = GF + E \quad (2)$$

The PMF solution minimizes the object function Q (equation 3) where the value σ_{ij} is the uncertainty in the j^{th} species for the sample i.

$$Q(E) = \sum_{i=1}^{m} \sum_{j=1}^{n} \left(\frac{E_{ij}}{\sigma_{ij}}\right)^2 \quad (3)$$

One of the main factors contributing to the success of the PMF model is related to uncertainty matrix. In this work, the uncertainties for each sample were calculated using an equation based uncertainty procedure from Polissar et al. (1998). Where the sample species concentration was less than or equal to the method detection limit (MDL), the uncertainty was calculated by equation 4:

$$Unc = \frac{5}{6} x\ MDL \quad (4)$$

Where the sample species concentration was greater than MDL, the uncertainty was calculated by equation 5, with error fraction estimated as precision in the error obtained after comparing with standard reference material SRM 1649 (n=4 samples)(Table S2, Supplementary Information).

$$Unc = \sqrt{(Error\ Fraction\ x\ concentration)^2 + (MDL)^2} \quad (5)$$

Sample species concentrations below the detection limit were substituted with one-half MDL. Data treatment for missing data was not required as the sample dataset had no missing sample species concentrations. A critical step in PMF analysis is considering if number of factors is correct. In order to claim the optimal number of factors was achieved, several parameters were evaluated. The first was the closeness of Qrobust, Qtrue and Qtheoretical, which was calculated as follows: n*m-p*(n+m), where n is the number of samples, m is the number of species and p is the number of factors. Other parameters evaluated were the major of standardized residuals between -3 and +3, and the good correlation between the sampled and the modelled data. Solution stability was also checked by bootstrap run analysis and F-peak runs (Norris et al. 2008).

In this work, the data set used was a 111x 16 matrix. Firstly, all the individual PAH were considered as strong variables and solutions between 4 and 7 factors were considered by running the data with 20 initial starting points and a random seed value that later was changed to a seed value=23 in order to reproduce results. No total variable was used. To improve their model fit, three species (Phe, An, 2+2/4 MePhe) were down weighted to weak, tripling their uncertainty. The other 13 variables were fitted as strong variables (9MePhe, 1MePhe, DiMePhe, Flt, Py, BaA,

Chry, B(b+j+k)F, BeP, BaP, IcdP+DahA, BghiP, Cor). Table 2 presents the minimum, 25th percentile, median, 75th percentile and maximum concentration values for each compound in PMF. No total variable was used. The optimal solution found had 5 factors with 10% of extra modelling uncertainty. The Q theoretical obtained was 1141, the Q robust 1258 and the Q true 1266 (Table S3, Supplementary Information). The scaled residuals were lower than +3 for the 87% of the data. The optimal solution was bootstrapped 100 times with a minimum correlation r value of 0.6 as threshold level (Table S4, Supplementary Information). The effect of rotation on the PMF results revealed minimum variations by varying the parameter FPEAK (+0.2,-0.2) (Table S5, Supplementary Information) so that non rotated profiles were illustrated in this work.

PMF/MLR

The daily contribution of each source obtained by the PMF model was quantitatively assessed by MLR analysis considering as dependent variable the sampled BaP-eq concentrations and as independent variables the five identified sources obtained by the PMF model output. The regression diagnostics for every PAH and for the modelled BaP-eq according to the PMF and PMF/MLR models are shown in Table 3.

Conditional probability function

The Conditional Probability Function (CPF) was used to analyse the impacts from varying wind directions by using the source contribution estimates from PMF coupled with the wind direction values measured on site (Vestenius et al. 2011). CPF was calculated using the equation 6:

$$CPF_{\Delta\theta} = \frac{m_{\Delta\theta}}{n_{\Delta\theta}} \quad (6)$$

where $m_{\Delta\theta}$ is the number of occurrence from wind sector $\Delta\theta$ that exceeded the threshold criterion, and $n_{\Delta\theta}$ is the total number of data from the same wind sector.

A total of 12 sectors were considered in this study ($\Delta\theta$=30°) and a threshold criterion of the upper 25th percentile was chosen to define the directionality of the sources. No episodes of calm wind conditions ($\leq$1.0 m/s) were included in the analysis because of the isotropic behaviour of wind direction at low wind speeds.

Lifetime lung cancer risk of PAH

The concern of studying PAH is related to their potential carcinogenic risk. Based on World Health Organization (WHO) data, the unit risk (UR) for PAH, indicating the estimated lifetime lung cancer risk from exposure to atmospheric PAH, is 8.7×10^{-5} (i.e., incidence of 8.7 cases per 100,000 people with chronic inhalation exposure to 1 ng/m^3 BaP over a lifetime of 70 years)(WHO 2000). In this work, the lifetime lung cancer risk was calculated by multiplying this UR for the modelled BaP-eq concentration, enabling more direct comparison with the WHO lifetime lung cancer risk based on exposure to BaP in the PM10 size fraction (equation 7).

Lifetime lung cancer risk = BaP-eq (ng/m^3) × UR (7)

Results and discussion

BaP-eq concentration

Rather than considering only BaP exposure, BaP-eq concentration data aggregated from a larger suite of species provides a better assessment of the total health risk from exposure to ambient PAH (i.e., exposure to BaP plus additional coincident toxic species). However, a limitation of this work was that sampled BaP-eq concentrations were calculated only from particle phase concentrations and ambient PAH and BaP-eq will be slightly higher, since lighter molecular weight species are found largely in vapour phase and they have not been sampled in this work.

The average BaP-eq concentration calculated for the sampling period (2001-2009) in Zaragoza was 0.99±1.07 ng/m^3, which is close to the target value of BaP=1.0 ng/m^3 established by Directive 2004/107/EC. In our study, the largest contributing species to BaP-eq were IcdP+DahA and BaP which contributed 53% and 34%, respectively to the average BaP-eq. This compared with sampling study published for several New Zealand sites, which reported BaP contributing no more than 50% of BaP-eq for a similar group of sampled species (Brown et al. 2005). Our data confirmed the importance of including PAH species in addition to BaP when assessing PAH toxicity.

In the literature, different BaP-eq concentrations have been published for different sampling sites and countries. However, a comparison between BaP-eq related only to PM10-bound PAH and data reported in this paper is shown in Table 4.

Similar annual mean BaP-eq concentrations of 1.4 and 1.7 ng/m^3 were found for the PAH associated to the PM10 in Christchurch, Alexandra two urban locations in New Zealand whereas lower BaP-eq concentration (0.06 ng/m^3) was obtained in Dunedin (Brown et al. 2005). In this case, the Nisbet and LaGoy (1992) toxic equivalency factors were used. In UK, BaP-eq concentrations of 0.62 ng/m^3 were obtained in outdoors environments (Delgado-Saborit et al. 2011) in 2006-2007. In Mexico city, BaP-eq concentration of 0.352 ng/m^3 in May-October 1999 and 1.150 ng/m^3 in November-December 2002 were reported (Amador-Muñoz et al. 2013). In Italy, Amodio et al. (2009) reported concentrations of BaP-eq reaching 16 ng/m^3 in Taranto, a large industrial area where metallurgical, chemical, petrochemical and cement-producing plants are located. In Algiers (Algeria), sub-urban and urban areas were sampled and BaP-eq concentrations of 0.41-0.63 and 0.41-0.99 ng/m^3 were reported (Ladji et al. 2009).

Also, higher BaP-eq concentrations than the reported ones in this paper have been published for different Asian countries. In Ulsan (Korea), the BaP-eq concentration varied between 3.01, 3.75 and 6.30 ng/m^3 in late spring in 2009 (Vu et al. 2011) and 3.66 ng/m^3 in Taiwan for an urban area (Chang et al. 2006). In Chiang Mai (Thailand), BaP-eq concentrations of 3.70, 0.18 and 0.25 ng/m^3 were reported in different periods between 2010 and 2011 (Wiriya et al. 2013).

Regarding the seasonal behaviour of BaP-eq, it was found that higher concentrations of BaP-eq were obtained in Zaragoza during the cold season (winter and autumn) versus the warm season (summer and spring). In particular, the dates corresponding to 27/12/2001, 14/03/2002, 12/01/2004, 09/02/2004 and 15/03/2004 showed BaP-eq concentrations of 3.22, 3.06, 4.78, 5.37 and 3.52 ng/m^3, respectively. This could be attributed to different factors: increase of use of fossil fuels for domestic heating, increase of PAH in the particulate phase due to low temperature, lower photochemical degradation and chemical reaction, lower mixing height, stagnant meteorological conditions in the winter that favour the pollutants accumulation (Ravindra et al. 2008). These results were also reported by Jung et

al. (2010) for outdoors in New York city corroborating that cold season is an important contributor to the potential risk of PAH exposure.

Source identification by PMF model

Firstly, the PMF model was applied in order to know the main anthropogenic PAH pollution sources by considering the individual PAH as potential variables. Five sources were identified as optimal solution and their profiles were shown in Fig. 3. Results were interpreted by use of indicator species from source sampling studies, seasonal variation and emission inventory for Zaragoza city (DGA, 2003, 2006).

The heaviest analysed compounds, IcdP, BghiP and Cor, were the major PAH contributing to factor 1, (Fig. 3). High molecular weight PAH are reported as dominant in vehicle emissions (Boström et al. 2002; Ravindra et al. 2008). These high molecular weight PAH have also been attributed to the combustion of heavy oils in agreement with Lee et al. (2004). Several industrial activities related to chemical, metal, transport, paper and cardboard could corroborate the heavy-oil combustion in Zaragoza (DGA, 2006). Moreover, previous studies carried out in Zaragoza showed high concentrations of Cor in areas using diesel as main fuel and with high density of traffic (buses, trucks)(López et al. 2003). The influence of the airport and diesel vehicles mainly associated with buses, trucks close to the sampling site could also contribute to these emissions. Authors like Riddle et al. (2007) also reported emissions even of Cor in heavy-duty diesel vehicles depending on the driving cycle and vehicle technology. This factor could be considered as a mixture also associated with heavy-duty vehicles. No seasonal behaviour was observed and 100% of its bootstrapped factor mapped to its base factor.

Factor 2 was characterized by high loadings of BeP, BaP and BbjkF (Fig. 3). In the literature, BaP and BeP are considered as light oil burning markers (Bari et al. 2009) and it was identified as light oil combustion. The t test ($p<0.05$) indicated significant differences between cold and warm seasons, showing higher concentration in cold season that could be attributed to the contribution of residential heating systems in addition to industrial emissions. 96% of its bootstrapped factor mapped to its base factor.

Factor 3 was mainly composed of Phe, An, 2+2/4MePhe, 9MePhe, 1MePhe, DiMePhe, Flt and Py (Fig. 3). These compounds have been described by different authors as coal combustion source markers (Harrison et al. 1996; Simcik et al. 1999; Sofowote et al. 2011). 98% of the bootstrapped factor mapped to the base factor. Factor 3 showed seasonality ($p< 0.05$), as a consequence of higher use of domestic heating systems in colder months (Sofowote et al. 2011) and the trend of heavier PAH to remain on the particle phase at lower temperatures (Ravindra et al. 2008). Although in Zaragoza, natural gas is one of the main fuels used for domestic heating, there are still central heating systems using coal and petrol-derived fuels. Moreover, the influence of power stations using coal as fuel in Teruel province could be also reflected on this factor.

Factor 4 presented a profile with high factor loadings for BaA, BbkjF, BaP and IcdP+DahA (Fig. 3) showing a seasonal behaviour ($p<0.05$). On the literature high factor loadings for BbF, BkF were attributed to diesel motor vehicle emissions (Lee et al. 2004; Harrison et al. 1996). However, IcdP, BaP and BaA were attributed to gasoline and diesel markers (Lee et al. 2004; Zhang et al. 2012; Sofowote et al. 2008; Harrison et al. 1996). This factor could be related with vehicular emissions and 78% bootstrapped factors mapped to base factor.

Factor 5 was attributed to natural gas emissions due to high factor loadings of BaA and Chry (Fig. 3) (Simcik et al. 1999; Motelay-Massei et al. 2007; Esen et al. 2008). 88% bootstrapped factors mapped to base factor. The seasonal behaviour ($p<0.05$) agreed with the increasing use of domestic heating systems during winter season (Lee et al. 2004).

Source apportionment of BaP-eq by PMF/MLR

Once the pollution sources were identified by the PMF model, the contribution of each source to the daily carcinogenic potential was quantitatively assessed by using the sampled BaP-eq concentrations as the dependent variable and the contribution of each source obtained by the PMF model as independent variables. The model explained 98% of the carcinogenic risk (R^2=0.98; slope=0.98; constant=0.033) (Fig. S1, Supplementary Information) and results were statistically significant at a confidence level of 99% (ANOVA) corroborating the adequacy of the PMF/MLR to model the BaP-eq concentration.

The modelled BaP-eq concentration was obtained by equation 8:

BaP-eq=0.179*Light-oil comb+0.088*Coal comb+ 0.102*Gas natural+ 0.355*Vehicular emissions+0.285*Heavy-duty vehicles (8)

Results from equation 8 showed that vehicular emissions mainly influenced the BaP-eq concentration (35%) followed by the heavy-duty vehicles (28%), the light-oil combustion (18%), the gas natural (10%) and the coal combustion (9%). The time series of modelled BaP-eq concentrations according to the PMF/MLR model are shown in Fig. 4 as a function of the sampling period. It was observed the major contribution of the vehicular emissions during the 2001-2002 and 2008-2009 periods whereas during 2003-2004, the heavy-duty vehicles dominated.

Conditional Probability Function

CPF were also useful in order to determine the influence of local and regional pollution sources affecting the sampling site (Fig. 5a-b). CPF was studied for the five pollution sources attributed by the PMF model. The light-oil combustion (Fig. 5a) and the vehicular emissions factors (Fig. 5b) showed the NE direction as the prevailing direction, reflecting the impact of the A-23 highway (direction to Huesca) and common fuels used for heating and industrial activities located in that direction. In fact, a paper factory and different industrial parks, in particular the one known as “ciudad del transporte” (6 km from Zaragoza), where heavy-duty diesel vehicles can be parked, are located in that direction. The NW direction was also reflected, especially in the vehicular emissions factor, indicating the potential contribution of the AP-68 highway (direction to Logroño) as well as different industrial parks located in that direction, which involve a lot of traffic especially during week days. In the case of the vehicular emissions, the SW and SE also reflected the impact of two national highways direction to Madrid (A-2) and Barcelona (A-2). The influence of the ring roads Z-40 and Z-30, which allow easy access into the city could be also contributing.

The coal combustion factor (Fig. 5a) also pointed out the NE and N as dominant directions. Both directions were likely to be influenced by residential heating and the potential contribution of power stations located in the north of Spain and in Cataluña. Industrial activities located in the A-23 “mudéjar” motorway, direction to Huesca could be

also contributing. The coal combustion factor (Fig. 5a) also showed the impact of the S and SE directions, indicating on the one hand, the use of coal as domestic heating fuel in the city centre and on the other hand, two thermal power stations located in Teruel using coal and natural gas for power generation. In the case of the natural gas factor (Fig. 5a), the S direction would be also one of the prevailing directions, indicating the impact of residential heating in the city centre of Zaragoza. Industrial parks located along the E-90 highway, direction to Madrid and a logistic platform where different companies are located could impact on the SW direction.
In addition, the CPF for heavy-duty vehicles factor also reflected the N, NW, SE and SW directions as possible source regions (Fig. 5b). This last direction, SW could also show the influence of the railway and bus station, industrial parks located in the E-90 (direction to Madrid) and A-21 (direction to Valencia) highways, both with high density of traffic.

Lifetime lung cancer risk

Due to the toxicity of some PAH, a study of the lifetime lung cancer risk for the atmosphere of Zaragoza was estimated by considering the UR=8.7×10^{-5} per ng/m^3 BaP according to WHO (1987, 2000). Fig. 6 showed the lifetime lung cancer risk calculated with the modelled BaP-eq by the PMF/MLR model along the different sampling years in Zaragoza as a function of the four seasons: winter, autumn, spring, summer. In this case, it was provided a more accurate risk assessment from environmental exposure to PAH than only considering BaP. It was observed that independently of the sampling year, winter was the season reaching the highest risk for human health exceeding the UR. This risk was also exceeded during autumn season. In addition, it was also interesting to reflect the average contributions to this cancer risk for each one of the five sources obtained by the PMF/MLR model as a function of the seasons (Fig. 7). It is reflected that although each individual source did not reach the UR, the vehicular emissions was the factor with a major contribution. Authors like Masiol et al. (2012) also highlighted the strong risk for human health due to mobile sources associated with vehicular emissions. The heavy-duty vehicles source also showed a remarkable contribution, which was not affected by seasonality. In fact, the lifetime cancer risk for the BaP-eq concentration followed the decreasing order of risk: winter, autumn, spring and summer reaching a lung cancer risk of 1.5 cases per 10,000 people in winter, to 1.2 cases per 10,000 people in autumn. Both seasons, winter and autumn, exceeded the UR of the WHO, 8.7 x 10^{-5} per 1ng/m^3. In spring season, the lung cancer risk decreased to 5 cases per 100,000 people and 4 cases per 100,000 people in summer.
This assessment reflected, not only the impact that season can have on the lifetime lung cancer risk, but also demonstrated the need to focus risk reduction efforts on mobile source emissions, a common problem affecting many large urban centres.

Conclusions

An assessment of the lifetime lung cancer risk in the atmosphere of Zaragoza was performed by using BaP-eq concentrations, which were calculated by applying toxic equivalent factors and later, modelled by a receptor model based on PMF/MLR. A total of five sources were discerned as responsible of this carcinogenic toxicity, where the major contributing source type was the vehicular emissions followed by the heavy-duty vehicles, the light-oil

combustion, the natural gas and the coal combustion. The PMF/MLR model was able to predict the sampled BaP-eq concentrations very well (R^2=0.98).

Using CPF, directionality of the PMF-derived source profile contributions identified the NE direction as one of the prevailing directions for light-oil combustion and vehicular emissions source contributions; this confirmed the impact of several local sources (E-7 highway, a paper factory, industrial parks) as well as regionally transported sources (industrial activity).

By season, winter and autumn were important contributors to the potential risk of PAH exposure with higher lifetime lung cancer risk, exceeding the WHO-established unit risk of 8.7 x 10^{-5} per 1 ng/m^3 of BaP (2000). Further investigations are still needed because this risk could be underestimated as only the particle phase PAH were considered in this work.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at:

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Table 1. PAH analyzed, short name and toxic equivalency factors (TEF) according to Larsen and Larsen (1998).

Species name	Short form	TEF Larsen&Larsen
Phenanthrene	Phe	0.0005
Anthracene	An	0.0005
2 +2/4-methylphenanthrene	2+2,4MePhe	0.0005
9-Methyl Phenanthrene	9 MePhe	0.0005
1-Methyl Phenanthrene	1 MePhe	0.0005
2,5-/2,7-/4,5 Dimethyl Phenanthrene	DiMePhe	0.0005
Fluoranthene	Flt	0.05
Pyrene	Py	0.001
Benzo[a]anthracene	BaA	0.005
Chrysene	Chry	0.03
Benzo[b]fluoranthene	BbF	0.1
Benzo[k]fluoranthene	BkF	0.05
Benzo[j]fluoranthene	BjF	0.05
Benzo[e]pyrene	BeP	0.002
Benzo[a]pyrene	BaP	1
Indeno[c,d]pyrene	IcdP	0.1
Dibenz[a,h]anthracene	DahA	1.1
Benzo[g,h,i]perylene	BghiP	0.02
Coronene	Cor	0.01

BbF+BjF+BkF= B(b+j+k)F

Table 2 Input data statistics for the individual PAH (ng/m^3) considered by the PMF model.

Species	Category	% BDL	S/N	Min.	25th	Median	75th	Max.
Phe	Weak	13	1.2	0.0300	0.03	0.07	0.2	1.27
An	Weak	53	4.4	0.0055	0.005	0.005	0.02	0.32
2+2,4MePhe	Weak	21	5.4	0.0030	0.01	0.01	0.06	0.38
9 MePhe	Strong	11	3.4	0.0050	0.02	0.03	0.05	0.3
1 MePhe	Strong	15	5.7	0.0035	0.01	0.01	0.06	0.19
DiMePhe	Strong	20	1.7	0.0001	0.01	0.06	0.13	0.43
Flt	Strong	1	10.0	0.0088	0.10	0.19	0.33	1.57
Py	Strong	1	2.6	0.0200	0.13	0.24	0.46	1.64
BaA	Strong	2	19.8	0.0001	0.07	0.16	0.5	3.03
Chry	Strong	2	31.6	0.0027	0.14	0.24	0.58	3.32
B(b+j+k)F	Strong	1	26.5	0.0130	0.21	0.46	0.83	5.87
BeP	Strong	1	47.1	0.0016	0.10	0.19	0.38	2.71
BaP	Strong	1	42.5	0.0005	0.08	0.19	0.36	1.94
IcdP+DahA	Strong	1	8.1	0.0007	0.29	0.59	1.17	6.49
BghiP	Strong	1	45.9	0.0036	0.29	0.53	1.00	4.61
Cor	Strong	3	11.5	0.0001	0.27	0.59	1.22	7.57

BDL= below detection limit

Table 3 Regression diagnostics for every PAH and modeled BaP-eq obtained by the PMF and the PMF/MLR models (slope, intercept, R^2 (coefficient of determination) and standard error (SE)).

PAH	Slope	Intercept	R^2	SE
Phe	0.41	0.05	0.35	0.09
An	0.37	0.01	0.57	0.01
2+2/4 MePhe	0.45	0.02	0.50	0.03
9MePhe	0.79	0.00	0.80	0.02
1MePhe	0.88	0.00	0.94	0.01
DiMePhe	0.79	0.01	0.79	0.03
Flt	0.76	0.04	0.87	0.10
Py	0.90	0.02	0.91	0.10
BaA	0.82	0.04	0.96	0.09
Chry	0.98	0.01	0.97	0.11
B(b+j+k)F	0.87	0.06	0.95	0.21
BeP	1.06	-0.02	0.98	0.07
BaP	0.89	0.01	0.94	0.09
IcdP+DahA	0.88	0.01	0.92	0.30
BghiP	1.04	-0.03	0.98	0.13
Cor	0.89	0.04	0.95	0.26
BaP-eq[a]	0.98	0.033	0.98	0.15

a= PMF/MLR model

Table 4. BaP-eq concentrations (ng/m^3) related to PM10-bound PAH for different sampling sites around the world.

Reference	Brown et al. 2005	Brown et al. 2005	Brown et al. 2005	Delgado-Saborit et al. 2011	Ladji et al. 2009	Amodio et al. 2009	Amador-Muñoz et al. 2013	Vu et al. 2011	Wiriya et al. 2013	This study
Sampling site	Christchurch, New Zealand	Alexandra, New Zealand	Dunedin, New Zealand	UK	Algiers, Algeria	Bari, Taranto (Italy)	Mexico city	Ulsan (Korea)	Chiang Mai (Thailand)	Zaragoza
Sampling Period	2001-2002	2001-2002	2001-2002	2006-2007	July 2005-June 2006	Jan2000-July 2005; Oct 2005; Febr 2006	May-October 1999 Nov-Dec 2002	2009 warm season	Apr 2010 Aug–Nov 2010 Jan–Mar 2011	2001-2009
Site characteristics	City	Provincial town	City	Outdoor	Sub-urban and urban area	Cities			Sub-urban	Sub-urban area
BaP-eq (ng/m^3)	1.4	1.7	0.006	0.62	0.41-0.63; 0.41-0.99	16 (max) Taranto	0.352 1.150	4.15	3.70 0.18 0.25	1.01
Included species	Nap, Ace, Ac, Flu, Phe, Flt, Py, An, BghiP, Chry, IcdP, BbF, BkF, BaA, BaP, DahA	Nap, Ace, Ac, Flu, Phe, Flt, Py, An, BghiP, Chry, IcdP, BbF, BkF, BaA, BaP, DahA	Nap, Ace, Ac, Flu, Phe, Flt, Py, An, BghiP, Chry, IcdP, BbF, BkF, BaA, BaP, DahA	BaA, Chry, BbF, BkF, BaP, IcdP, DahA,	BaA, BbF, BaP, IcdP, DahA	BaA,BbF,BkF, BaP,IcdP,DahA, BghiP	BaA, Chry, BbF, BkF, BaP, IcdP, DahA	Nap, Ace, Ac, Flu, Phe, Flt, Py,An, Chry, BaA, BbF, BkF, IcdP, BaP, BghiP,DahA	Nap, Ace, Ac, Flu, Phe, Flt, Py,An, Chry, BaA, BbF, BkF, IcdP, BaP, BghiP,DahA	Phe, An, MePhe, DiMephe, Flt, Py, BaA, Chry, B(b+k+j), BeP, BaP, IcdP+Daha,BghiP, Cor
TEF	Nisbet and LaGoy, 1992	Nisbet and LaGoy, 1992	Nisbet and LaGoy, 1992	Nisbet and LaGoy[a]	Cecinato, 1997	Nisbet and LaGoy 1992	Nisbet and LaGoy, 1992	Nisbet and LaGoy[b]	Nisbet and LaGoy (1992)	Larsen and Larsen (1998)

Ac= Acenaphtylene Ace=Acenapthene, Fl= Fluorene
S=summer; W= winter; A= annual
[a] Modified by Malcolm and Dobson
[b] Modified by Muller
B(e)P and DBahA adopted from Malcolm and Dobson (1994).

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B(e)P and DBahA adopted from Malcolm and Dobson (1994).

Table 4. BaP-eq concentrations (ng/m^3) related to PM10-bound PAH for different sampling sites around the world.

Sampling site	Sampling period	Site characteristics	BaP-eq (ng/m^3)	Included species	TEF	Reference
Christchurch, New Zealand	2001-2002	City	1.4	Nap, Ace, Ac, Flu, Phe, Flt, Py, An, BghiP, Chry, IcdP, BbF, BkF, BaA, BaP, DahA	Nisbet and LaGoy, 1992	Brown et al. 2005
Alexandra, New Zealand	2001-2002	Provincial town	1.7	Nap, Ace, Ac, Flu, Phe, Flt, Py, An, BghiP, Chry, IcdP, BbF, BkF, BaA, BaP, DahA	Nisbet and LaGoy, 1992	Brown et al. 2005
Dunedin, New Zealand	2001-2002	City	0.006	Nap, Ace, Ac, Flu, Phe, Flt, Py, An, BghiP, Chry, IcdP, BbF, BkF, BaA, BaP, DahA	Nisbet and LaGoy, 1992	Brown et al. 2005
UK	2006-2007	Outdoor	0.62	BaA, Chry, BbF, BkF, BaP, IcdP, DahA,	Nisbet and LaGoya, 1992	Delgado-Saborit et al. 2011
Algiers, Algeria	July 2005-June 2006	Sub-urban and urban area	0.41-0.63; 0.41-0.99	BaA, BbF, BaP, IcdP, DahA	Cecinato, 1997	Ladji et al. 2009
Bari, Taranto (Italy)	Jan2000-July 2005; Oct 2005; Febr 2006	Cities	16 (max) Taranto	BaA,BbF,BkF, BaP,IcdP,DahA, BghiP	Nisbet and LaGoy, 1992	Amodio et al. 2009
Mexico city	May-Oct 1999 Nov-Dec 2002	University of Mexico	0.35 1.15	BaA, Chry, BbF, BkF, BaP, IcdP, DahA	Nisbet and LaGoy, 1992	Amador-Muñoz et al. 2013
Ulsan (Korea)	May-June 2009	Residential area Downtown area Industrial complex	3.01 6.30 3.75	Nap, Ace, Ac, Flu, Phe, Flt, Py,An, Chry, BaA, BbF, BkF, IcdP, BaP, BghiP,DahA	Nisbet and LaGoyb, 1992	Vu et al. 2011
Chiang Mai (Thailand)	Apr 2010 Aug–Nov 2010 Jan–Mar 2011	Sub-urban	3.70 0.18 0.25	Nap, Ace, Ac, Flu, Phe, Flt, Py,An, Chry, BaA, BbF, BkF, IcdP, BaP, BghiP,DahA	Nisbet and LaGoy, 1992	Wiriya et al. 2013
Zaragoza	2001-2009	Sub-urban area	1.01	Phe, An, 9MePhe, 1MePhe,2+2/4MePhe, DiMephe, Flt, Py, BaA, Chry, B(b+k+j), BeP, BaP, IcdP+Daha, BghiP, Cor	Larsen and Larsen, 1998	This study

Nap= Napthalene, Ac= Acenaphtylene Ace=Acenapthene, Fl= Fluorene,
[a] Modified by Malcolm and Dobson
[b] Modified by Muller

Fig. 1 Location of the sampling site in Zaragoza.

Fig. 2 Box plot of PAH sampling data, 2001-2009. (Box is interquartile range, line inside box is median, whiskers are 95% of data, outlier vaues indicated by asterisk.)

Fig. 3 Source profiles resolved by the PMF model for the BaP-eq (concentration and percentage).

Fig. 4 Time series plots of source contribution for the BaP-eq resolved by the PMF model.

Fig. 5 Conditional probability function plots for the average five source contributions obtained by the PMF model.

Fig. 6 Lifetime lung cancer risk as a function of the sampling dates in Zaragoza. The WHO unit risk is also shown.

Fig. 7 Lifetime lung cancer risk for the five sources apportioned by the PMF model and for the BaP-eq modeled and experimental as a function of the season. The WHO unit risk is also shown.

Train and bus station

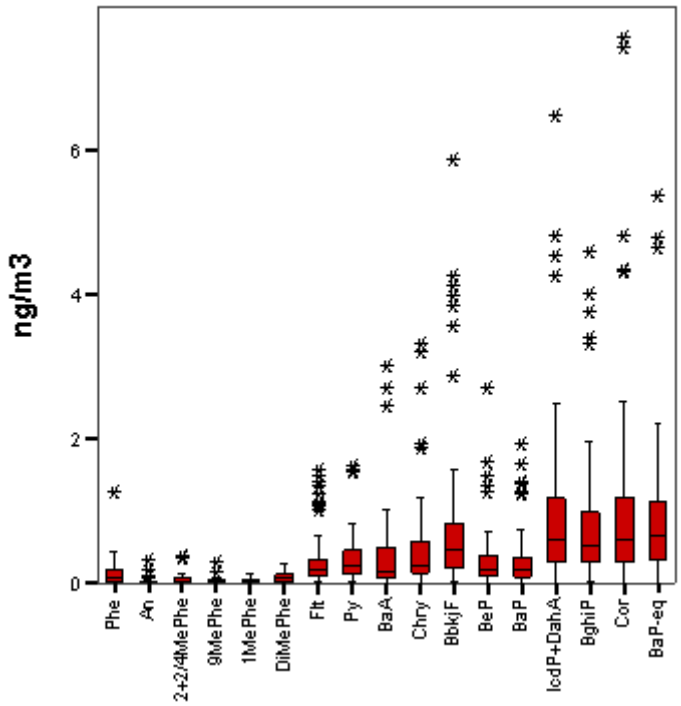
Airport

Industrial park

Thermal power station

Ciudad del transporte

Fig. 1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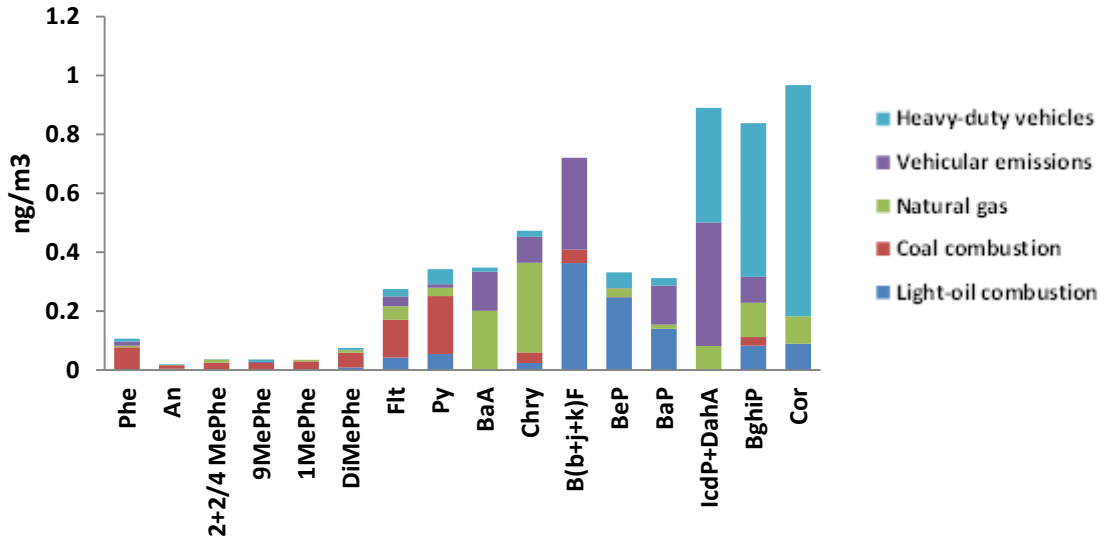
BaP-eq

Fig. 2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Heavy-duty vehicles
Vehicular emissions
Natural gas
Coal combustion
Light-oil combustion

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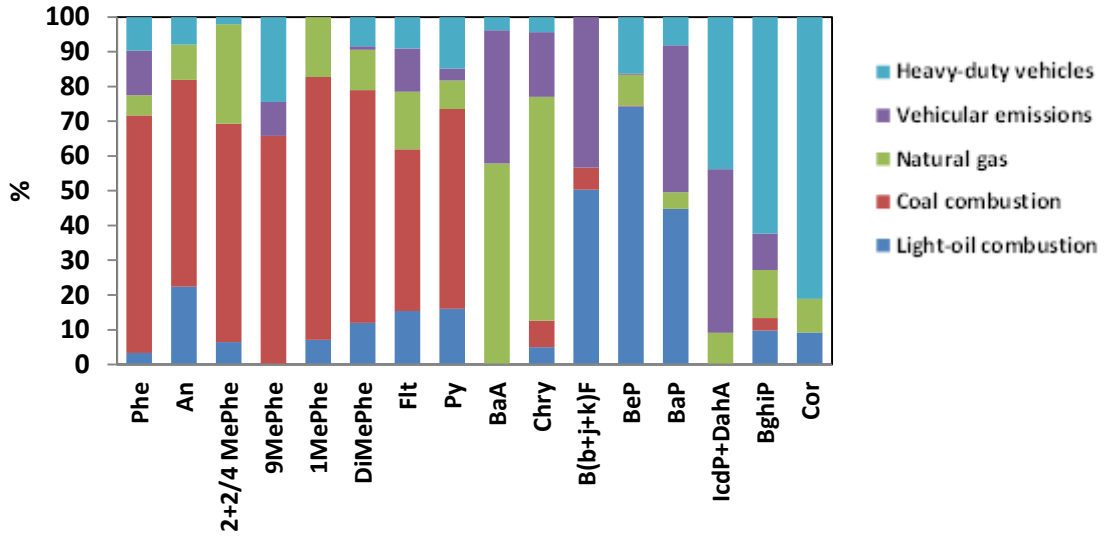
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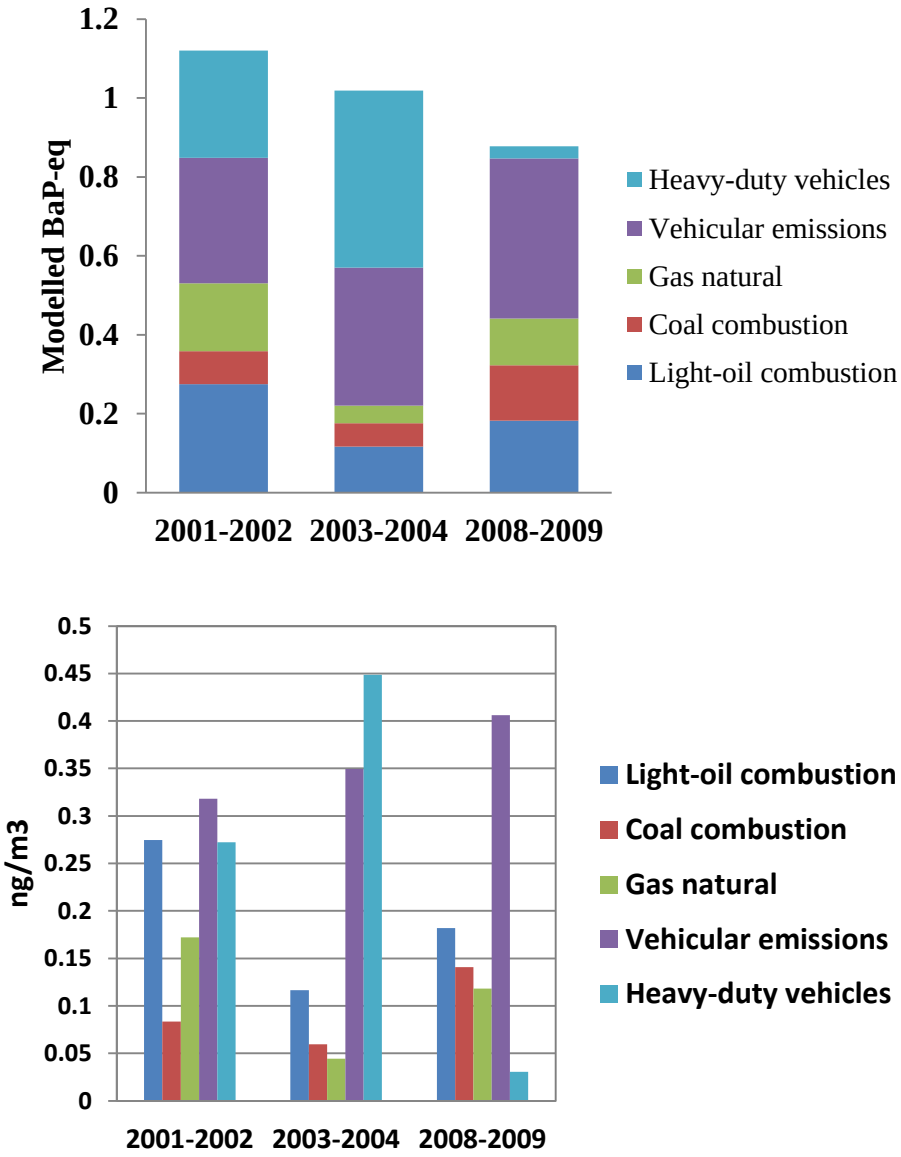
Heavy-duty vehicles
Vehicular emissions
Natural gas
Coal combustion
Light-oil combustion

b)

Fig. 3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Coal combustion
Light-oil combustion
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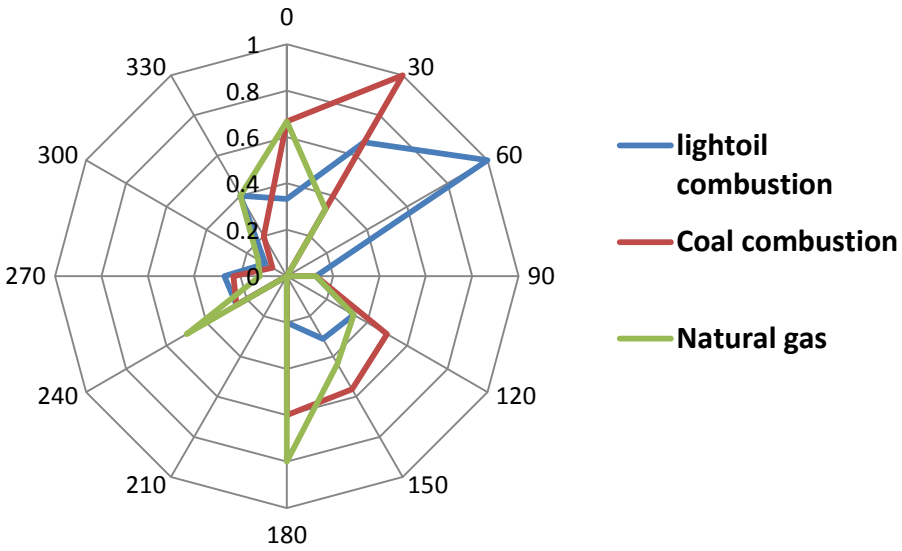
Gas natural
Vehicular emissions
Heavy-duty vehicles

Fig. 4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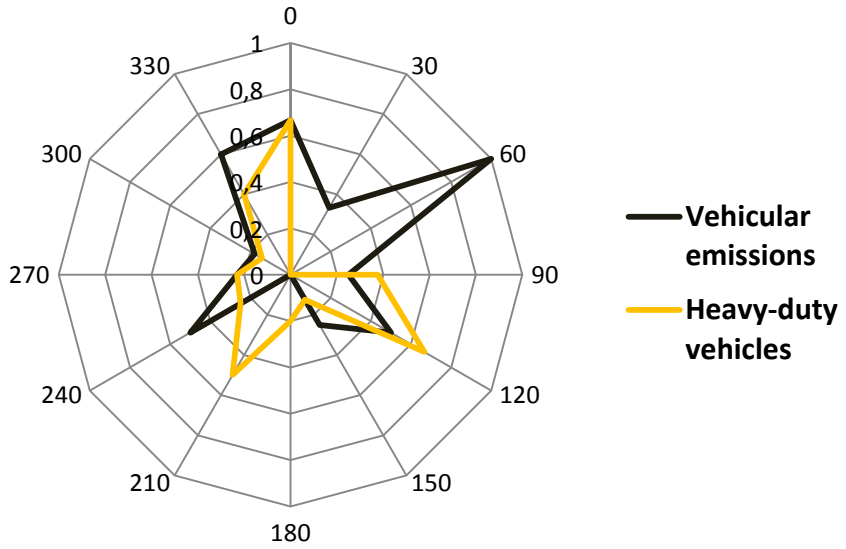
Natural gas

a)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Vehicular emissions
Heavy-duty vehicles

b)

Fig. 5

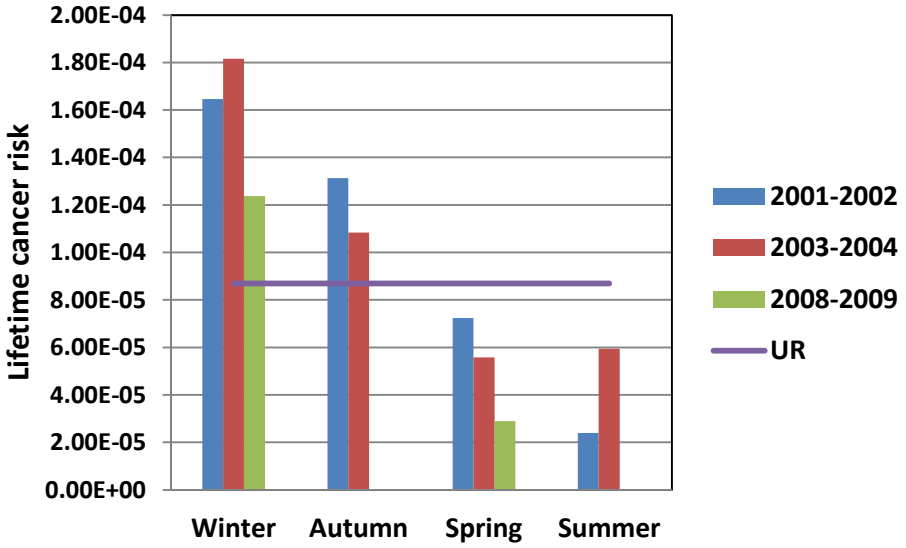
2.00E-04
1.80E-04
1.60E-04
1.40E-04
1.20E-04
1.00E-04
8.00E-05
6.00E-05
4.00E-05
2.00E-05
0.00E+00
Lifetime cancer risk
Winter
Autumn
Spring
Summer
2001-2002
2003-2004
2008-2009
UR

Fig. 6

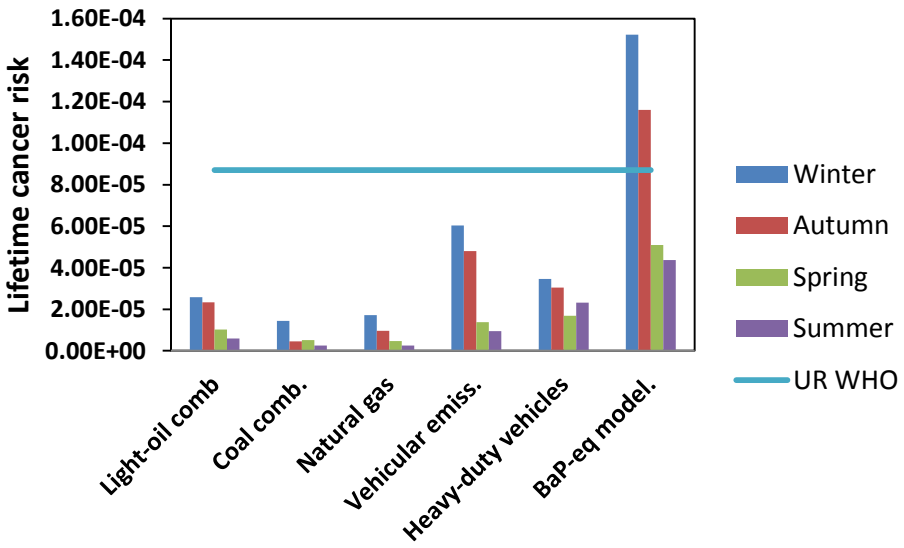
1.60E-04
1.40E-04
1.20E-04
1.00E-04
8.00E-05
6.00E-05
4.00E-05
2.00E-05
0.00E+00
Lifetime cancer risk
Light-oil comb
Coal comb.
Natural gas
Vehicular emiss.
Heavy-duty vehicles
BaP-eq model.
Winter
Autumn
Spring
Summer
UR WHO

Fig. 7