This article was published in Environmental Science and Pollution Research 21 (6), 4492-4501, 2014
http://dx.doi.org/10.1007/s11356-013-2391-5

Levels and risks of particulate-bound PAHs in indoor air influenced by tobacco smoke: A field measurement

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Abstract

Considering tobacco smoke as one of the most health relevant indoor sources, the aim of this work was to further understand its negative impacts on human health. The specific objectives of this work were to evaluate the levels of particulate–bound PAHs in smoking and non-smoking homes and to assess the risks associated with inhalation exposure to these compounds. The developed work concerned the application of the toxicity equivalency factors (TEF) approach (including the estimation of the lifetime lung cancer risks, WHO) and the methodology established by USEPA (considering 3 different age categories) to 18 PAHs detected in inhalable (PM_{10}) and fine ($PM_{2.5}$) particles at two homes. The total concentrations of 18 PAHs (Σ_{PAHs}) was 17.1 and 16.6 ng m^{-3} in PM_{10} and fine $PM_{2.5}$ at smoking home and 7.60 and 7.16 ng m^{-3} in PM_{10} and fine $PM_{2.5}$ at non-smoking one. Compounds with 5 and 6 rings composed the majority of the particulate PAHs content (i.e. 73% and 78% of Σ_{PAHs} at the smoking and non-smoking home, respectively). Target carcinogenic risks exceeded USEPA health–based guideline at smoking home for 2 different age categories. Estimated values of lifetime lung cancer risks largely exceeded (68–200 times) the health–based guideline levels at both homes thus demonstrating that long–term exposure to PAHs at the respective levels would eventually cause risk of developing cancer. The high determined values of cancer risks in the absence of smoking were probably caused by contribution of PAHs from outdoor sources.

Keywords: Polycyclic aromatic hydrocarbons (PAHs); indoor air; tobacco smoke; PM_{10}; $PM_{2.5}$; risk assessment;

1. **Introduction**

It is well known that tobacco smoking is associated with various diseases of lung and heart as well as with cancers of various organ systems (IARC 2004, 2010). Tobacco smoke is certainly one of the greatest sources of the indoor pollution not only for the smokers but also for all those who are somehow exposed to it. The scientific research has shown that exposure to second-hand smoke, also referred as environmental tobacco smoke (ETS), is associated with various adverse health outcomes including increased risk of lung cancer, acute coronary syndromes and stroke, an increased prevalence of respiratory symptoms and inflammatory reactions (Adlkofer 2001; Barnoya and Glantz 2005; Bhalla et al. 2009; Cesaroni et al. 2008; Dransfield et al. 2007; Hoffmann and Hoffmann 1997; Madureira et al. 2012; Raupach et al. 2008). Thus US Environmental Protection Agency (USEPA) and International Agency for Research on Cancer (IARC) have classified exposure to tobacco smoke as Class A and 1 human carcinogen, respectively (IARC 2004; USEPA 1993). Considering the negative health impacts, USA and a number of European countries have ban smoking in public places (McNabola and Gill 2009) in order to ensure smoke-free environments and to protect public health (Madureira et al. 2012; Pacheco et al. 2012; WHO 2009). However for individuals that spent large amount of their time at homes (seniors, infants) the exposure to tobacco smoke in the respective ambiences are relevant. Young children in particular are in great risks; it was estimated that four out of ten children (approximately 700 million children globally) have at least one parent who currently smokes (IARC 2012), thus predisposing them to exposure to second-hand tobacco smoke at their homes. In Europe, the prevalence of children exposure to tobacco smoke at homes is particularly high (78%) being the highest of all geographical regions (WHO 2009). In order to

protect the public health, it is thus necessary to continue with scientific and regulatory efforts to reduce tobacco-related pollution.

From the chemical point of view, tobacco smoke is a complex mixture of gaseous components and particles of different sizes. Up to 5200 components, including heavy metals, aromatic amines and N-nitrosamines have been identified in tobacco smoke (Rodgman and Perfetti 2008), as well as 549 polycyclic aromatic hydrocarbons (PAHs; Thielen et al. 2008). PAHs are a large group of organic compounds with two or more fused aromatic rings that are produced during incomplete combustion of organic matter. Indoors tobacco smoke is considered among their most significant source (Ball and Truskewycz 2013). PAHs are cytotoxic and mutagenic compounds, some of them being considered as carcinogens to humans (WHO 1998). In air PAHs are distributed between gas phase and particles, but the especially harmful compounds (with 5-6 aromatic rings) are predominantly found in particulates, mostly due to their high molecular weights and low volatility (Liu et al. 2001; Lu and Chen 2008; Slezakova et al. 2011). Because of their hazardous properties there have been efforts to regulate PAHs in air. Current European legislation on ambient air (Directive 2004/107/EC) sets annual target value of 1 ng m^{-3} for carcinogenic PAHs in PM_{10} (particulate matter with aerodynamic diameter below 10 μm) using benzo[a]pyrene as indicator of carcinogenic PAHs. Benzo[a]pyrene is probably the most studied carcinogenic compound and is often used as a surrogate for other carcinogenic PAHs in studies estimating human cancer risks. However, the suitability of this approach started to be questioned (Pufulete et al. 2004) by new findings on the presence of more potent PAHs, such as dibenzo[a,l]pyrene or dibenz[a,h]anthracene (Okona–Mensah et al. 2005).

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Considering tobacco smoke among the most health relevant indoor emission sources, this work aims to evaluate the associated health risks regarding particulate–bound PAHs. The developed work concerns the application of the toxicity equivalency factors (Boström et al. 2002) approach (including the estimation of the lifetime lung cancer risks; WHO 1987, 2000) and the methodology established by USEPA (USEPA 2013) to 18 PAHs detected in inhalable (PM_{10}) and fine ($PM_{2.5}$) particles at one home influenced by smoking and one non-smoking home. The determined compounds were the 16 PAHs considered by USEPA as priority pollutants, dibenzo[a,l]pyrene, and benzo[j]fluoranthene (the latter recommended by EU Directive 2004/107/EC).

2. Materials and methods

2.1 Sample Collection

Particulate–bound PAHs were collected for a period of 19 consecutive days in January 2009 at two homes situated in Oporto, Portugal: one influenced by smoking and one non-smoking home accordingly with Castro et al. (2011). Both homes were located in Paranhos district. To avoid dissimilar influence of outdoor air both homes were located in the same block of flats and on the same floor (4^{th}). The characteristics of both homes were similar (i.e. area, cleaning and cooking activities, number of inhabitants) and are shown in Table 1S of the Supplementary materials. During the whole period the occupants of both homes kept detailed reports of the performed activities including frequency of ventilation that was provided by opened windows (as occupants thought necessary).

The samples were collected daily for a period of 24 hours by constant flow samplers (Bravo H2, TCR TECORA, Italy) that were combined with PM EN LVS

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sampling heads (in compliance with norm EN12341 for PM_{10} and EN14907 for $PM_{2.5}$). The air flow rate was 2.3 $m^3 h^{-1}$ which corresponded to less than 5% of the room volume sampled per 1 hour (in agreement with available guidelines on indoor air sampling; ISO 16000-1:2004). Inlets were placed 1.5 m above the floor (in order to simulate human breathing zone) and minimally 1 m from the walls, without obstructing the normal usage of the rooms. Different fractions of particles, i.e. PM_{10} and $PM_{2.5}$, were collected on polytetrafluoroethylene (PTFE) membrane filters with polymethylpentene support ring (2 μm porosity, Ø47 mm, SKC Ltd., UK).

During the sampling period, the levels of outdoor air pollutants (PM_{10}, NO_2, NO_X, O_3, SO_2 and CO) were registered (Table 2S of the Supplementary material) as well as meteorological conditions (Table 3S); the respective station was situated approximately 350 m east from the homes.

2.2 Gravimetric mass determination

PM_{10} and $PM_{2.5}$ masses were determined gravimetrically as described previously in detail by Slezakova et al. (2010, 2013). The $PM_{2.5-10}$ fraction (i.e. coarse fraction with particles of aerodynamic diameter between 2.5 and 10 μm) was determined as difference (by subtraction) between PM_{10} and $PM_{2.5}$.

For the gravimetric mass determination, the filters (76 samples) were stored in Petri dishes and the same analytical balance (Mettler Toledo AG245 analytical balance weighing with accuracy of 10 μg) was always used. The steps of gravimetric mass determinations were the following: 24 hours to equilibrate filters (temperature 22.5 ± 1.0 °C, relative humidity 42 ± 6%) in a desiccator before weighing, followed by weighing during the following 24–48 hours. After sampling, filters were

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immediately weighed, stored in Petri dishes covered in parafilm, and kept in freezer (–18 °C) until they were further analysed.

2.3 Extraction and quantification of PAHs

Dibenzo[a,l]pyrene (D[a,l]P), benzo[j]fluoranthene (B[j]F) and more 16 PAHs identified as priority pollutants by USEPA were determined in the collected particulate samples: naphthalene (Nap), acenaphthylene (Acy), acenaphthene (Acp), fluorene (Flr), phenanthrene (Phe), anthracene (Ant), fluoranthene (Flt), pyrene (Pyr), benz[a]anthracene (B[a]A), chrysene (Chr), benzo[b]fluoranthene (B[b]F), benzo[k]fluoranthene (B[k]F), benzo[a]pyrene (B[a]P), dibenz[a,h]anthracene (D[a,h]A), benzo[ghi]perylene (B[ghi]P), indeno[1,2,3–cd]pyrene (InP); B[j]F and B[b]F (determined as sum B[j+b]F). The extraction of PAHs from particles (i.e. from PM_{10} and $PM_{2.5}$) was performed by MAE (MARS–X 1500 W Microwave Accelerated Reaction System for Extraction and Digestion, CEM, Mathews, NC, USA) for 20 min at 110 ºC using 30 mL of acetonitrile (Sigmal–Aldrich) (Castro et al. 2009a, 2009b; Ramalhosa et al. 2012). Extracts were carefully filtered through a PTFE membrane filter (0.45 µm) and reduced to a small volume using a rotary evaporator (Buchi Rotavapor, R–200) at 20 ºC. A gentle stream of nitrogen was used to dry the extracts under low temperature; the residue was then re–dissolved in 1000 µL of acetonitrile immediately before analysis.

Extracts were analysed using a Shimadzu LC system (Shimadzu Corporation, Kyoto, Japan) equipped with a LC–20AD pump, DGU–20AS degasser and photodiode array SPD–M20A (PAD) and fluorescence RF–10AXL (FLD) detectors on line. Separation of the compounds was performed in a C18 column (CC 150/4 Nucleosil 100-5 C18 PAH, 150 × 4.0 mm; 5 µm particle size; Macherey-Nagel,

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Duren, Germany); the injected volume was 15.0 μL. A mixture of water (ultra–pure grade; prepared by a Milli–Q simplicity 185 system, Millipore, Molsheim, France) and acetonitrile (Lichrosol for gradient elution, Carlo Erba, Rodano, Italy, purity > 99.9%) was used as the mobile phase. The initial composition of the mobile phase was 50% of acetonitrile and 50% ultra-pure water, and a linear gradient to 100% of acetonitrile was programmed in 15 min, with a final hold of 13 min. Initial conditions were reached in 1 min and maintained for 6 min before next run. The total run time was 40 min with a flow rate of 0.8 mL min^{-1}. Fluorescence wavelength programming was used to perform better sensitivity and minimal interference. Each compound was detected at its optimum excitation/emission wavelength pair: 260/315 nm (naphthalene, acenaphthene and fluorene), 260/366 nm (phenanthrene), 260/430 nm (anthracene, fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b+j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, benzo[ghi]perylene and dibenzo[a,l]pyrene), and 290/505 nm (indeno[1,2,3-cd]pyrene). Acenaphthylene, which does not show fluorescence, was analysed at 254 nm in PAD. Each analysis was performed at least in triplicate.

Calibration curves obtained using six mixed matrix matched standards containing all the PAHs showed good linearity over the entire range of concentrations with correlation coefficients always higher than 0.999 for all PAHs. Limits of detection (LODs) and limits of quantification (LOQs) were calculated and expressed as PAH concentration in air samples (Castro et al. 2009b). LODs between 0.0016 ng m^{-3} for benz[a]anthracene and 0.027 ng m^{-3} for naphthalene were obtained, with corresponding LOQs in the range 0.0054–0.089 ng m^{-3}.

2.4 Health risk analysis

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The risks associated with inhalation exposure to all 18 PAHs were assessed by toxicity equivalency factors (TEF) using values estimated by Muller, 1997 (Boström et al. 2002). Consequently, the lifetime lung cancer risks were estimated (WHO 1987, 2000).

The carcinogenic risks were assessed according to the methodology provided by USEPA Region III Risk–based Concentration Table (USEPA 2013). The risks were estimated as the incremental probability of an individual to develop cancer, over a lifetime, as a result of inhalation exposure to that potential carcinogen (i.e. incremental or excess individual lifetime cancer risk; USEPA 1989). Acceptable risk levels for carcinogens range from 10^{-4} (risk of developing cancer over a human lifetime is 1 in 10 000) to 10^{-6} (risk of developing cancer over a human lifetime is 1 in 1 000 000). The carcinogenic risks were calculated using the following equation:

$$TR = [(EFr \times ED \times ET \times IUR \times C) / AT] \quad (1)$$

where TR is target carcinogenic risk (dimensionless); EFr is the exposure frequency (350 days per year); ED is the exposure duration (years); ET is indoor air exposure time (0.80, i.e. 19.2 h per day); IUR is the chronic inhalation unit risk $(\mu g\ m^{-3})^{-1}$ (USEPA 2013); C is the concentration of PAH ($\mu g\ m^{-3}$); and AT is the number of days over which the exposure is averaged (25 500 days, i.e. 70 years × 365 days per year). The carcinogenic risks were estimated only for PAHs for which IUR values are available (USEPA 2013), namely: naphthalene (IUR of 3.4×10^{-5} $(\mu g\ m^{-3})^{-1}$); chrysene (1.1×10^{-5} $(\mu g\ m^{-3})^{-1}$); benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene and indeno[1,2,3–cd]pyrene (IUR of 1.1×10^{-4} $(\mu g\ m^{-3})^{-1}$); benzo[a]pyrene (IUR of 1.1×10^{-3} $(\mu g\ m^{-3})^{-1}$); and dibenz[a,h]anthracene (1.2×10^{-3} $(\mu g\ m^{-3})^{-1}$). In this work three different age–categories (USEPA 2008; Vieira et al. 2011) were used for the estimation of the target risks using the following ED values

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(in brackets): children 1–3 years (1 year), adults 25–54 years (25 years), and seniors >65 years (65 years). The lowest possible ED was chosen for each age category in order not to over–estimate the respective cancer risks. The detailed examples of TR calculations are shown in Table 4S of the Supplementary material.

2.5 Statistical analysis

For the data treatment, the Student's t-test was applied to determine the statistical significance ($p<0.05$, two tailed) of the differences between the determined means.

3. Results

3.1 PM_{10} and $PM_{2.5}$ masses

The means and statistical parameters of PM_{10} and $PM_{2.5}$ concentrations measured at two homes are summarized in Fig. 1. The statistical analysis of the results indicated that at smoking home PM_{10} and $PM_{2.5}$ were significantly higher ($p<0.05$) than at non-smoking one. In addition, at both homes PM_{10} means were not significantly different ($p<0.05$) from $PM_{2.5}$. No significant differences ($p<0.05$) were observed between PM levels during weekdays and weekends. PM_{10} concentrations were well correlated with $PM_{2.5}$ with correlation coefficients of 0.976 and 0.954 at smoking and non-smoking home, respectively.

In order to study the relationship between different PM fractions, mass concentration ratios were also analyzed. The $PM_{2.5}/PM_{10}$ ratios were calculated from each measurement. The mean of $PM_{2.5}/PM_{10}$ ratios was significantly higher at smoking home (0.86) where values ranged from 0.81 to 0.97 whereas it was between 0.70 and 0.97 at non-smoking home (mean of 0.79).

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3.2 PAHs

The means and concentration range of PAHs in PM_{10} and $PM_{2.5}$ at two homes are summarised in Table 1, with concentrations presented as sums of individual compounds (according to the number of rings, i.e. groups with 2, 3, 4, 5 and 6 rings, respectively). An increase in PAH molecular weight globally corresponds to an increase of compound toxicity; PAHs with 5 and 6 rings are among the most harmful ones. At both homes compounds with five rings that comprised of benzo[b+j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, and dibenz[a,h]anthracene were the most abundant groups of PAHs in both PM_{10} and $PM_{2.5}$. These PAHs accounted for 54% and 59% of total PAH content (i.e. Σ_{PAHs}) at smoking and non-smoking home, respectively. The highest concentrations were observed for dibenz[a,h]antracene that reached at the non-smoking home means of 2.25 and 2.11 ng m^{-3} in PM_{10} and $PM_{2.5}$, respectively; at smoking home its obtained levels were approximately twice higher. Compounds with 6 rings were the second most abundant group of PAHs and accounted for 19% and 20% of Σ_{PAHs} at smoking and non-smoking home, respectively. This group comprised of benzo[g,h,i]perylene, indeno[1,2,3-cd]pyrene, and dibenzo[a,l]pyrene that was the least abundant compound of all PAHs. At non-smoking home, dibenzo[a,l]pyrene reached mean concentration of 68.9×10^{-3} and 61.7×10^{-3} ng m^{-3} in PM_{10} and $PM_{2.5}$, respectively being approximately 3.5 times lower than at the smoking home. Compounds with 4 rings (fluoranthene, pyrene, benz[a]anthracene, and chrysene) accounted for 16 % of Σ_{PAHs} in both PM at both homes, whereas it was 11% and 6–7% of Σ_{PAHs} for compounds with 3 rings (fluorene, phenanthrene, anthracene, acenaphthylene, and acenaphthene)

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at smoking and non-smoking home, respectively. Finally, PAHs with 2 rings that included naphthalene accounted at smoking home for 1% of Σ_{PAHs} in both PM; at non-smoking home these compounds were not found.

3.3 Health risks assessment

In order to estimate the carcinogenic risks for humans, the benzo[a]pyrene equivalent carcinogenicity were evaluated by multiplying concentration of each PAH with their TEF value. The results of TEF adjusted concentration for 18 PAHs at smoking and non-smoking home are presented in Table 2. As expected the higher risks were found for smoking home, where total TEF–adjusted concentration of all PAHs ($\Sigma_{TEF\text{-}PAHs}$) was 29.0×10^3 pg m^{-3} in PM_{10} and 27.4×10^3 pg m^{-3} in $PM_{2.5}$, being approximately 200% higher than at non-smoking home. The values of $\Sigma_{TEF\text{-}PAHs}$ at both homes were then used to estimate the corresponding lifetime lung cancer risks.

The means and range of target carcinogenic risks associated with inhalation exposure to PAHs that were estimated by USEPA methodology are presented in Table 3. The obtained results demonstrate that: i) for both PM fractions significantly higher risks were observed at the smoking home than at the non-smoking one (Fig. 2); ii) at both homes higher risks (2–11%) were found for PM_{10} than $PM_{2.5}$; iii) for all compounds the highest carcinogenic risks were observed for the age group of seniors (>65 years); and iv) for all three age–groups the highest risk were found for dibenz[a,h]anthracene. Considering the above mentioned, the highest cancer risks were observed for dibenz[a,h]anthracene which in PM_{10} at smoking home reached for seniors a value of 3.86×10^{-6}.

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4. Discussion

The health risks associated with PAHs bound in PM_{10} and $PM_{2.5}$ were evaluated at two homes with and without smoking. The obtained results showed significantly (p<0.05) higher levels of both PM_{10} and $PM_{2.5}$ at the home with smoking. Previously, Wallace et al. (2003) estimated an increase of 37 μg m^{-3} for indoor particles (range 0.6 - 5 μm) due to smoking in a study conducted in USA. BéruBé et al. (2004) reported PM_{10} concentration in UK homes with smokers approximately 10 to 44 μg m^{-3} greater (depending upon the number of smokers) than in those without smokers. Data obtained within this study were similar. Specifically, the increase between smoking and non-smoking home was 47 μg m^{-3} for PM_{10} and 42 μg m^{-3} for $PM_{2.5}$. The average indoor/outdoor PM_{10} ratio was 0.65 at the non-smoking home, which suggests that outdoor air was the major contributor to indoor PM levels (outdoor $PM_{2.5}$ data were not available for comparison). At smoking home the average value of PM_{10} indoor/outdoor ratio was 2.31 thus indicating the contribution from indoor sources (i.e. tobacco smoke). It is however necessary to point that both homes were situated in the multiunit building. Therefore, infiltration of the second-hand smoke emissions from other units might potentially contribute to indoor PM measured at both (smoking and smoke-free) homes (Dacunto et al. 2013a, King et al. 2013). Finally, cooking activities were performed at both homes (Table 1S) and could account for some of the measured PM (Dacunto et al. 2013b).

The ratios between indoor $PM_{2.5}$ and PM_{10} concentrations that were obtained within this work were in general similar to those previously reported for indoor environments in Portugal (0.87 for smoking home and 0.74 non-smoking one; Slezakova et al. 2009), being considerably higher than those found outdoors in the

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same district area (0.68–0.72; Slezakova et al. 2010, 2011, 2013). High values of $PM_{2.5}/PM_{10}$ ratios indicate that indoor PM_{10} were mostly composed of fine particles. These findings are health-relevant because fine particles especially represent a serious risk to human health (Pope et al. 2002; WHO 2006). In addition, at the smoking home $PM_{2.5}$ concentrations were very high (increase of 280%) but $PM_{2.5-10}$ concentrations were rather low. The significantly higher $PM_{2.5}/PM_{10}$ ratio and the much higher $PM_{2.5}$ concentrations at smoking home thus corroborate the previous findings that indoor combustion sources, namely tobacco smoke had the determinant influence on the presence of fine particles (Klepeis et al. 2003; Dacunto et al. 2013b).

Considering the protection of public health, it is important to enhance that at both homes compounds with 5 and 6 rings composed the majority of the particulate PAH content (i.e. 73% and 78% of Σ_{PAHs} at the smoking and non-smoking home, respectively). With exception to benzo[ghi]perylene, all studied 5 and 6–rings PAHs are probable and possible human carcinogens (IARC 2010) and include also benzo[a]pyrene, a class 1 human carcinogen. The total concentration of ten (out of 18) carcinogenic PAHs (i.e. $\Sigma_{carcPAHs}$) was approximately 120% higher when influenced by tobacco smoke; the carcinogenic PAHs were predominantly associated with fine particles (93–96% of $\Sigma_{carcPAHs}$). Thus in order to protect public health it is necessary to develop strategies to reduce exposure to $PM_{2.5}$, mainly related to carcinogenic PAHs.

When the health risks associated with inhalation exposure are evaluated by TEF method, typically TEF values estimated by Nisbet and La Goy (1992) are used (Bari et al. 2010; Halek et al. 2008; Mugica et al. 2010; Ohura et al. 2004). However, Nisbet and La Goy did not refer TEF value for dibenzo[a,l]pyrene. As this PAH is considered relevant for the respective evaluation, in this work TEF reported by Muller

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1997 (that included TEF for dibenzo[a,l]pyrene; Boström et al. 2002) were used to calculate the TEF–adjusted concentrations. Dibenzo[a,l]pyrene was previously the least abundant PAH in both PM at both homes. Due to its TEF of 100, dibenzo[a,l]pyrene became the largest contributor to $\Sigma_{TEF-PAHs}$ (80% and 70% of $\Sigma_{TEF-PAHs}$ at smoking and non-smoking home, respectively) and its TEF-adjusted concentration was 240% higher when influenced by tobacco smoke. This PAH is not commonly assessed when evaluating particulate–bound PAHs, although its relative contribution to carcinogenic potential is very strong, even at very low concentrations as demonstrated within this study. Dibenz[a,h]anthracene (the most abundant PAH in both PM at both homes) was the second largest contributor to $\Sigma_{TEF-PAHs}$ with 14% and 21% of $\Sigma_{TEF-PAHs}$ at smoking and non-smoking home, respectively. In general these results confirm and emphasize the importance of the analysis and evaluation of these two potent carcinogens that are being currently discussed as possible surrogate compounds for PAH mixtures from various environments (Okona–Mensah et al. 2005). Finally, benzo[a]pyrene was the third most abundant PAH (5% and 6% of $\Sigma_{TEF-PAHs}$ at smoking and non-smoking home, respectively) with levels 130% higher when influenced by smoking. Overall the obtained results allow concluding that analysis of all three PAHs is relevant in relation to tobacco smoke; the common approach of using benzo[a]pyrene as an indicator might lead to underestimating the potential carcinogenic potency of PAHs in air.

Regarding the lung cancer risk via the inhalation route, the World Health Organization suggested the unit risk of 8.7×10^{-5} (ng m^{-3})$^{-1}$ for lifetime (70 years) PAH exposure (Ohura et al. 2004). Taking into the consideration that people spend indoors approximately 80% of their time, the estimated lifetime lung cancer risks at

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the smoking home were 2.0×10^{-3} for PAHs in $PM_{2.5}$, being about 18 times lower for coarse fraction (i.e. 1.1×10^{-4}). At the non-smoking home, the corresponding values of lifetime lung cancer risks were lower, with figures of 6.8×10^{-4} for PAHs in $PM_{2.5}$ and 6.2×10^{-5} for $PM_{2.5\text{-}10}$; PAHs in fine fraction exhibited risks about 11 times higher than in $PM_{2.5–10}$. It is important to point out that at both homes risks estimated for both fine and coarse fractions exceeded the health–based guideline level of 10^{-5} (Boström et al. 2002). The exceedances were especially considerable for $PM_{2.5}$ with values 200 and 68 times higher than health–based guideline at smoking home and non-smoking home, respectively. These results thus demonstrate that particulate–bound PAHs, and especially those from tobacco smoke represent a serious health risk.

When evaluating carcinogenic risks associated with inhalation exposure to PAHs by USEPA methodology, dibenzo[a,l]pyrene was not considered as its chronic inhalation unit risk value is not available; therefore settling IUR value for dibenzo[a,l]pyrene is important for the respective risk analysis once this compound is a potent carcinogen (Okona–Mensah et al. 2005). For carcinogens, USEPA set a risk level of 10^{-6} for individual compounds and pathways with the understanding that it will generally cause negligible cancer risks. However, caution is recommended to ensure that cumulative cancer risks of all potential carcinogenic components do not have residual cancer risk exceeding 10^{-4}. At smoking home (Fig. 2) target carcinogenic risks exceeded in both PM the USEPA health–based guideline level for 2 different age categories: adults with 25–54 years and seniors (> 65 years). These results confirm that tobacco smoke considerably increases the carcinogenic risks. Furthermore, the exposure to tobacco smoke combined with certain life style (such as diet or regimen) may result in even increased cancer risks related to these pollutants (Slezakova et al. 2011). At non-smoking home, target carcinogenic risks were

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approximately twice lower than at smoking home. USEPA health–based guideline was exceeded for one age category, namely seniors, which suggest that long-term exposure to PAHs at levels found at non-smoking home can eventually cause risks for developing cancer. The high values of cancer risks in the absence of smoking indicate a significant contribution of PAHs from another source. The values of PM indoor/outdoor ratios (lower than 1), indicate that outdoor emissions can be a significant contributor to indoor levels (Castro et al. 2010). Therefore, Fig. 3 shows comparison between compositional profiles of particulate-bound PAHs collected indoors at non-smoking home and in ambient (outdoor) air. The abundances of PAHs in ambient air were retrieved from Slezakova et al. (2011) and corresponded to sampling period of 40 days during November – December 2008 (i.e. previous months to the indoor measurements). Despite the existing limitations between both studies (different sampling period, outdoor data collected at ground level, distance between both sampling sites) composition profiles of PAHs at non-smoking home were rather similar to those of ambient air. In addition, Slezakova et al. (2013) has shown that vehicular road emissions are the major source of ambient PAHs in the respective area of Oporto. In a view of these findings, traffic emissions thus can be a significant source of indoor PAHs. This is especially relevant for homes in close vicinity to major roads where vehicular emissions can be the major contributor of indoor health–relevant pollutants; the risks associated with the elevated concentrations in those indoor environments could be significantly higher than those calculated in this work.

Conclusions

At the smoking home, the mean total concentrations of 18 PAHs was 17.1 and 16.6 ng m^{-3} in PM_{10} and $PM_{2.5}$, respectively. The corresponding concentrations were

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2.3 times lower at non-smoking home, with means of 7.60 ng m^{-3} in PM_{10} and 7.16 ng m^{-3} in $PM_{2.5}$. PAHs with 5 and 6 rings composed the majority of the particulate PAH content (i.e. 73% and 78% of Σ_{PAHs} at the smoking and non-smoking home, respectively). Target carcinogenic risks exceeded USEPA health–based guideline at smoking home for 2 different age categories demonstrating that tobacco smoke considerably increases the carcinogenic risks. The estimated values of lifetime lung cancer risks largely exceeded (68–200 times) the health–based guideline levels at both homes thus demonstrating that long–term exposure to PAHs at the respective levels would eventually cause risk of developing cancer. In the absence of smoking the high achieved values of cancer risks suggests a significant contribution of PAHs from outdoors.

Acknowledgments

This work was supported by Fundação para Ciência e Tecnologia through grants number PEst-C/EQB/LA0006/2011 and PEst-C/EQB/UI0511/2011, and fellowship SFRH/BPD/65722/2009.

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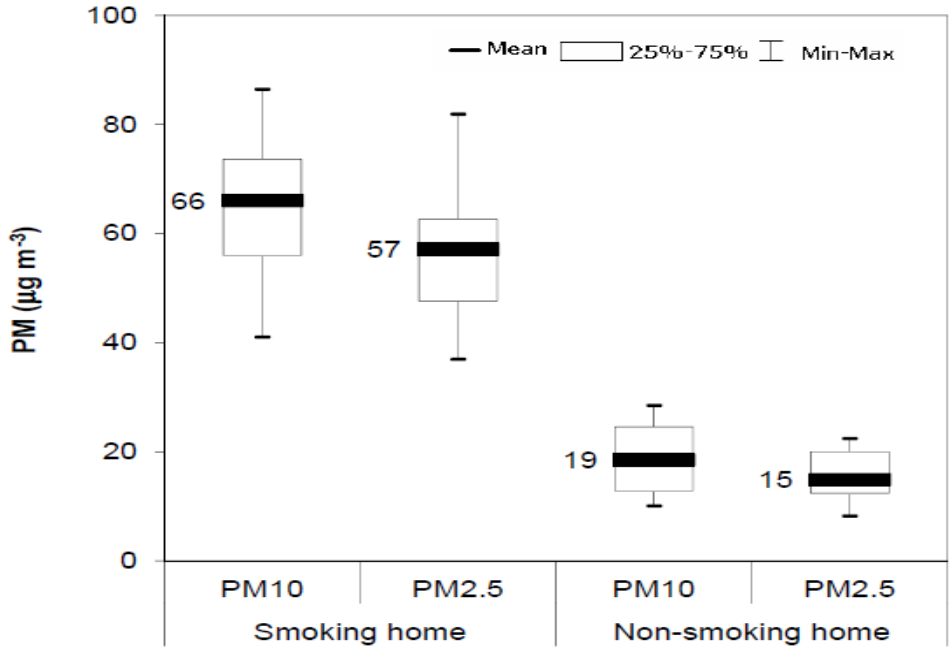

Fig 1 PM_{10} and $PM_{2.5}$ concentrations at two homes: means, minima and maxima values, and 25th and 75th percentiles.

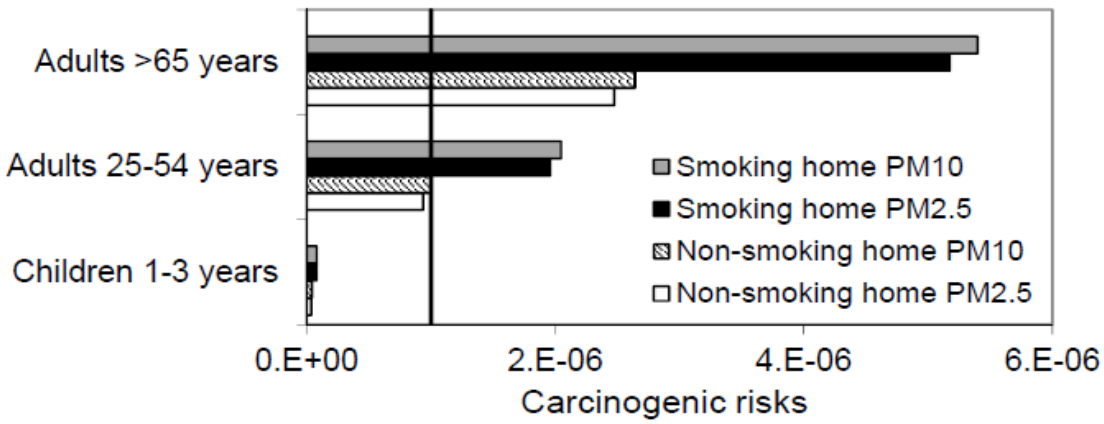

Fig 2 Carcinogenic risks of PAHs in PM_{10} and $PM_{2.5}$ at two homes. The values represents sum of target carcinogenic risks of eight individual PAHs (naphthalene chrysene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[1,2,3–cd]pyrene, benzo[a]pyrene, and dibenz[a,h]anthracene); the horizontal black line represents USEPA health–based guideline level (10^{-6}).

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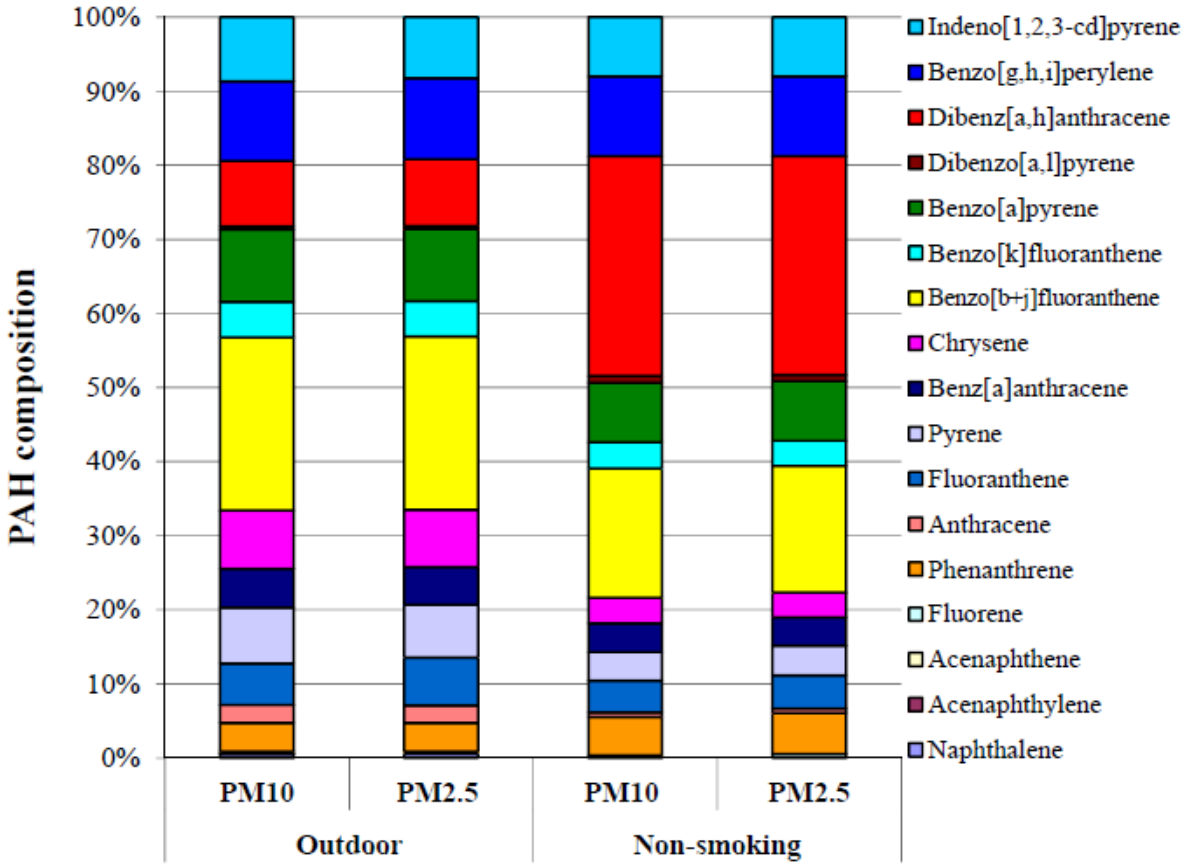

Fig 3 Compositional profiles of 18 PAH in PM_{10} and $PM_{2.5}$ obtained at indoor none-smoking home and in ambient air. The abundances of PAHs in ambient air were retrieved from Slezakova et al. (2011).

Table 1

Mean concentrations of PAHs in PM_{10} and $PM_{2.5}$ at smoking and non-smoking homes (ng m^{-3})

PAHs	Smoking home				Non-smoking home			
	PM_{10} (n=19)		$PM_{2.5}$ (n=19)		PM_{10} (n=19)		$PM_{2.5}$ (n=19)	
Number of rings	mean	range	mean	range	mean	range	mean	range
2-rings	0.126	n.d.– 0.402	0.115	n.d.–0.373	n.d.	n.d.	n.d.	n.d.
3-rings	1.81	0.506–4.76	1.77	0.441–4.63	0.467	0.168–0.788	0.478	0.167–0.738
4-rings	2.66	1.57–4.84	2.64	1.53–4.40	1.18	0.355–2.64	1.12	0.365–2.24
5-rings	9.30	2.21–15.6	8.94	2.12–14.5	4.45	1.03–12.0	4.15	1.01–10.5
6-rings	3.21	0.570–5.4	3.13	0.515–5.20	1.50	0.361–3.98	1.41	0.327–3.49
Σ_{PAHs}	17.1	5.07–27.8	16.6	5.05–26.5	7.60	1.91–19.4	7.16	1.87–17.0

n.d. – not detected

Table 2

TEF–adjusted mean concentrations of PAHs in PM_{10} and $PM_{2.5}$ at two homes (pg m^{-3})

		Smoking home		Non-smoking home	
	TEF[a]	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Naphthalene	n.a	–	–	–	–
Fluorene	n.a	–	–	–	–
Acenapthylene	n.a	–	–	–	–
Acenapthene	n.a	–	–	–	–
Phenanthrene	0.00064	0.365	0.336	0.251	0.253
Anthracene	n.a	–	–	–	–
Fluoranthene	n.a	–	–	–	–
Pyrene	0	–	–	–	–
Benz[a]anthracene	0.014	5.26	4.83	4.09	3.84
Chrysene	0.026	22.7	23.9	6.92	6.23
Benzo[b]fluoranthene[b]	0.11	319	309	146	134
Benzo[k]fluoranthene	0.037	20.3	20.0	9.77	9.10
Benzo[a]pyrene	1	1 400	1 330	610	573
Dibenz[a,h]anthracene	0.89	3 960	3 800	2 010	1 880
Dibenzo[a,l]pyrene	100	23 200	21 800	6 890	6 170
Benzo[ghi]perylene	0.012	19.9	19.2	9.84	9.23
Indeno[1,2,3–cd]pyrene	0.067	89.8	88.1	40.7	38.5
$\sum_{PAHs}$	–	29 000	27 400	9 720	8 830

[a] TEF estimated by Muller, 1997 (Boström et al., 2002)

[b]Quantified as benzo[b+j]fluoranthene

n.a. – not available

Table 3

Estimated target carcinogenic risks of PAHs in PM_{10} and $PM_{2.5}$ at two homes

Age group[a]	Target carcinogenic risk							
	Smoking home							
	PM_{10}							
	Nap	B[a]A	Chr	B[b]F	B[k]F	B[a]P	D[a,h]A	InP
Children	4.71×10^{-11}	4.53×10^{-10}	1.05×10^{-10}	3.50×10^{-9}	6.61×10^{-10}	1.69×10^{-8}	5.85×10^{-8}	1.62×10^{-9}
1–3 years	(n.a.–1.50×10^{-10})	($2.85–7.32\times10^{-10}$)	($0.47–2.54\times10^{-10}$)	($0.92–5.59\times10^{-9}$)	($1.50–12.4\times10^{-10}$)	($0.57–2.67\times10^{-9}$)	($2.32–10.5\times10^{-8}$)	($0.38–2.67\times10^{-9}$)
Adults	1.18×10^{-9}	1.13×10^{-8}	2.63×10^{-9}	8.75×10^{-8}	1.65×10^{-8}	4.22×10^{-7}	1.46×10^{-6}	4.04×10^{-8}
25–54 years	(n.a.–3.74×10^{-9})	($0.64–1.83\times10^{-8}$)	($1.18–6.36\times10^{-9}$)	($2.29–14.0\times10^{-8}$)	($0.38–3.09\times10^{-8}$)	($1.43–6.68\times10^{-7}$)	($0.58–2.61\times10^{-6}$)	($0.95–6.68\times10^{-8}$)
Seniors	3.11×10^{-9}	2.99×10^{-8}	6.93×10^{-9}	2.31×10^{-7}	4.36×10^{-8}	1.11×10^{-6}	3.86×10^{-6}	1.07×10^{-7}
>65 years	(n.a.–9.88×10^{-9})	($1.70–4.83\times10^{-8}$)	($3.12–16.8\times10^{-9}$)	($0.61–3.69\times10^{-7}$)	($0.99–8.17\times10^{-8}$)	($0.38–1.76\times10^{-6}$)	($1.53–6.90\times10^{-6}$)	($0.25–1.76\times10^{-7}$)
	$PM_{2.5}$							
	Nap	B[a]A	Chr	B[b]F	B[k]F	B[a]P	D[a,h]A	InP
Children	4.28×10^{-11}	4.16×10^{-10}	1.11×10^{-10}	3.38×10^{-9}	6.50×10^{-10}	1.61×10^{-8}	5.61×10^{-8}	1.59×10^{-9}
1 –3 years	(n.a.–1.39×10^{-10})	($2.58–6.94\times10^{-10}$)	($0.47–1.91\times10^{-10}$)	($0.92–5.16\times10^{-9}$)	($1.50–10.9\times10^{-10}$)	($0.57–2.62\times10^{-8}$)	($2.32–9.86\times10^{-8}$)	($0.38–2.53\times10^{-9}$)
Adults	1.07×10^{-9}	1.04×10^{-8}	2.77×10^{-9}	8.45×10^{-8}	1.63×10^{-8}	4.02×10^{-7}	1.40×10^{-6}	3.96×10^{-8}
25–54 years	(n.a.–3.48×10^{-9})	($0.64–1.73\times10^{-8}$)	($1.18–4.77\times10^{-9}$)	($2.29–12.9\times10^{-8}$)	($0.38–2.73\times10^{-8}$)	($1.43–6.56\times10^{-7}$)	($0.58–2.47\times10^{-6}$)	($0.95–6.32\times10^{-8}$)
Seniors	2.83×10^{-9}	2.75×10^{-8}	7.30×10^{-9}	2.23×10^{-7}	4.29×10^{-8}	1.06×10^{-6}	3.70×10^{-6}	1.05×10^{-7}
>65 years	(n.a.–9.18×10^{-9})	($1.70–4.58\times10^{-8}$)	($3.12–12.6\times10^{-9}$)	($0.61–3.40\times10^{-7}$)	($0.99–7.21\times10^{-8}$)	($0.38–1.73\times10^{-6}$)	($1.53–6.5\times10^{-6}$)	($0.25–1.67\times10^{-7}$)
	Non-smoking home							
	PM_{10}							
	Nap	B[a]A	Chr	B[b]F	B[k]F	B[a]P	D[a,h]A	InP
Children	–	3.52×10^{-10}	3.21×10^{-11}	1.60×10^{-9}	3.18×10^{-10}	7.35×10^{-9}	2.96×10^{-8}	7.32×10^{-10}
1–3 years		($0.81–8.29\times10^{-10}$)	($0.88–9.23\times10^{-11}$)	($0.38–4.22\times10^{-9}$)	($0.71–8.73\times10^{-10}$)	($1.72–20.7\times10^{-8}$)	($0.67–7.92\times10^{-8}$)	($1.51–21.0\times10^{-10}$)
Adults	–	8.80×10^{-9}	8.02×10^{-10}	4.00×10^{-8}	7.96×10^{-9}	1.84×10^{-7}	7.41×10^{-7}	1.83×10^{-8}
25–54 years		($2.03–2.07\times10^{-9}$)	($2.21–2.31\times10^{-10}$)	($0.95–10.6\times10^{-8}$)	($1.76–21.8\times10^{-9}$)	($0.43–5.18\times10^{-7}$)	($1.68–19.8\times10^{-7}$)	($0.38–5.25\times10^{-8}$)
Seniors	–	2.32×10^{-8}	2.12×10^{-9}	1.06×10^{-7}	2.10×10^{-8}	4.85×10^{-7}	1.96×10^{-6}	4.83×10^{-8}

>65 years		(0.54–5.47×10^{-8})	(0.58–6.09×10^{-9})	(0.25–2.89×10^{-7})	(0.47–5.76×10^{-8})	(1.13–13.7×10^{-7})	(0.44–5.23×10^{-6})	(1.01–13.9×10^{-8})
	$PM_{2.5}$							
	Nap	B[a]A	Chr	B[b]F	B[k]F	B[a]P	D[a,h]A	InP
Children	–	3.31 × 10^{-10}	2.89 × 10^{-11}	1.47 × 10^{-9}	2.96 × 10^{-10}	6.91 × 10^{-9}	2.78 × 10^{-8}	6.92 × 10^{-10}
1–3 years		(0.92–7.14×10^{-10})	(1.09–7.17×10^{-11})	(0.32–3.67×10^{-9})	(0.71–7.66×10^{-10})	(1.57–18.2×10^{-9})	(0.72–6.98×10^{-8})	(1.43–18.6×10^{-10})
Adults	–	8.27 × 10^{-9}	7.23 × 10^{-10}	3.68 × 10^{-8}	7.41 × 10^{-9}	1.73 × 10^{-7}	6.94 × 10^{-7}	1.73 × 10^{-8}
25–54 years		(2.29–17.8×10^{-9})	(2.71–17.9×10^{-10})	(0.81–9.18×10^{-8})	(1.78–19.2×10^{-9})	(0.39–4.56×10^{-7})	(1.81–17.5×10^{-7})	(0.36–4.66×10^{-8})
Seniors	–	2.18 × 10^{-8}	1.91 × 10^{-9}	9.72 × 10^{-8}	1.96 × 10^{-8}	4.56 × 10^{-7}	1.83 × 10^{-6}	4.57 × 10^{-8}
>65 years		(0.60–4.71×10^{-8})	(0.72–4.73×10^{-9})	(2.14–24.2×10^{-8})	(0.47–5.06×10^{-8})	(1.04–12.0×10^{-7})	(0.48–4.61×10^{-6})	(0.95–12.3×10^{-8})

[a] According to the USEPA document EPA/600/R-06/096F (USEPA, 2008)

n.a. – not available