This article was published in Environmental Science and Pollution Research, 22(18), 13892-13902, 2015
http://dx.doi.org/10.1007/s11356-015-4588-2

Exposure to polycyclic aromatic hydrocarbons and assessment of potential risks in preschool children

Marta Oliveira[a], Klara Slezakova[a,b], Cristina Delerue-Matos[a], Maria do Carmo Pereira[b]

Simone Morais[a,*]

[a]REQUIMTE, Instituto Superior de Engenharia do Porto, Instituto Politécnico do Porto, R. Dr. António Bernardino de Almeida 431, 4200-072 Porto, Portugal

[b]LEPABE, Departamento de Engenharia Química, Faculdade de Engenharia, Universidade do Porto, R. Dr. Roberto Frias, 4200-465 Porto, Portugal

*Corresponding author: Tel.: +351 22 834 0500 - ext 1918, Fax: +351 22 834 0500

E-mail: sbm@isep.ipp.pt

Abstract

As children represent one of the most vulnerable groups in society, more information concerning their exposure to health hazardous air pollutants in school environments is necessary. Polycyclic aromatic hydrocarbons (PAHs) have been identified as priority air pollutants due to their mutagenic and carcinogenic properties that strongly affect human health. Thus this work aims to characterize levels of 18 selected PAHs in preschool environment, and to estimate exposure and assess the respective risks for 3–5–years old children (in comparison with adults). Gaseous PAHs (mean of 44.5±12.3 ng m^{-3}) accounted for 87% of the total concentration (ΣPAHs) with 3–ringed compounds being the most abundant (66% of gaseous ΣPAHs). PAHs with 5 rings were the most abundant ones in particulate phase (PM; mean of 6.89± 2.85 ng m^{-3}) being predominantly found in PM_1 (76% particulate ΣPAHs). Overall child exposures to PAHs were not significantly different between older children (4–5–years old) and younger ones (3–years old). Total carcinogenic risks due to particulate–bound PAHs indoors were higher than outdoor ones. The estimated cancer risks of both preschool children and the staff were lower than USEPA threshold of 10^{-6} but slightly higher than WHO–based guideline.

Keywords: polycyclic aromatic hydrocarbons (PAHs), particles, gas phase, preschools, risk assessment

1. **Introduction**

Much of the scientific attention was originally focused on ambient air pollution and its impacts on public health. Due to the need to reduce high levels of air pollutants, policy makers in the air pollution field mostly focused on outdoor air whereas the concern of epidemiologists laid in defining coefficients linking outdoor concentrations of air pollutants with effects on health. As a result, the knowledge of indoor air pollution and its health impacts has been somewhat hindered. Understanding the complexity of indoor air exposure and the respective health impacts, and development of the protective guidelines are among the priorities of the World Health Organization (WHO; WHO, 2010). In addition, WHO has defined relevant indoor air pollutants, polycyclic aromatic hydrocarbons (PAHs) being one of them (WHO, 2010).

PAHs are a large group of organic pollutants that are ubiquitously found in environment. They are released from various sources during incomplete combustion: vehicular road transport, power plants, coal burning, and waste treatment (Hanedar et al., 2014; Ravindra et al., 2008; Slezakova et al., 2013a,b); second–hand cigarette smoke, cooking, and infiltration of outdoor PAHs (both gaseous and particles) through windows, doors, building cracks, and ventilation system to indoors are considered as the relevant indoor sources (Chen et al., 2012; Qi et al., 2014; Shen et al., 2012; Slezakova et al., 2014). PAHs have cytotoxic and mutagenic properties (Annesi-Maesano et al., 2007; Tuntawiroon et al., 2007), some of them being recognized as endocrine disrupting chemicals (WHO, 2013). Benzo[a]pyrene, one of the most well–known marker of PAHs, is categorised by International Agency for Research on Cancer (IARC) as carcinogens to humans (group 1) (IARC, 2010); in studies estimating human cancer risks it is often used as a surrogate for the carcinogenic PAHs.

Children represent one of the most vulnerable groups of a society with regard to potentially harmful effects induced by air pollution (Schüepp and Sly, 2012; WHO, 2010). In their earliest years, children stay mostly indoors with significant amount of time spent at preschools (Schwab et al., 1992). Nevertheless, there is only little information concerning PAHs in schools. The available data comes mostly from European studies (Alves et al., 2014; Cirillo et al., 2006; Gatto et al., 2014; Krugly et al., 2014; Moshammer and Neuberger, 2003), Asia (Jyethi et al., 2014; Ruchirawat et al., 2006, 2007; Tuntawiroon et al., 2007) and USA (Wilson et al., 2003). It is necessary to point that majority of the conducted studies focused mostly on particulate PAHs. Furthermore, the existent studies focused on evaluating PAH levels but the respective information concerning the doses or risks assessment due to exposure to PAHs in school environments is scarce (Bae et al., 2010; Wilson et al., 2003). Understanding child exposure is vital to healthy child development (Burtscher and Schüepp, 2012). As throughout the day children move between different preschool microenvironments (with different levels of pollution) where they conduct various activities for a different duration of time and with different physical intensity, time–activity patterns of children are significant parameters to determine their exposure (Cohen Hubal et al., 2000; Edwards et al., 2006).

The aim of this study was to characterize levels of PAHs (16 considered by USEPA as priority pollutants, and dibenzo[a,l]pyrene and benzo[j]fluoranthene; the latter recommended by EU Directive 2004/107/EC) at preschool environment and to estimate the exposure for 3–5 years old children. Furthermore, the health risks of 3–5 years old children to all 18 PAHs were assessed by toxicity equivalency factors (TEF) and according to the methodology recommended by USEPA (USEPA, 2014) and compared with those of the adult staff.

2. Material and methods

2.1 Sampling site

During 15 days of six consecutive weeks (February – April 2012), 16 PAHs considered by USEPA as priority pollutants, plus dibenzo[a,l]pyrene and benzo[j]fluoranthene were sampled in gas and particulate phases ($PM_{2.5}$ and $PM_{1,}$ i.e. particulate matter with a 50% efficiency cut-off at 2.5 and 1 μm, respectively) at one preschool situated in a urban zone of Oporto, Portugal. The selected preschool was considered as the representative of the respective area. The preschool was placed on a moderately trafficked street (mean traffic density of 16 vehicles min^{-1}) and consisted of one two–floor building that was constructed in 1940. The total number of enrolled students (3–5 years old) was 173, the preschool being one of the largest ones in Oporto city.

2.2 Sample collection

Gas and particulate samples were collected for a period of 9 h (between 8 a.m. and 5 p.m.) when children were present at the school. During the sampling campaign a total of 60 samples of gas and particulate phases were obtained. The samplers were placed indoors in a common room that was used during the whole day for educational and entertaining activities as well as for physical exercises and eating (snacks and etc.). The selected room was used due to the various activities that children conducted in there. The room (area 102 m^2 and height 2.9 m) was equipped with wood furniture, PVC floor coating and white wall paint; construction/material characteristics were similar as in other classrooms. It contained four doors (wood panels) and 10 windows (single glass layer). Throughout the day the room was naturally ventilated by opening windows as occupants thought necessary. In addition, in the morning (approximately for 15–20 min before children arrivals) and at the end of day (after 6:0 p.m. when everybody left and room was empty) all windows were approximately opened for 15-20 minutes. The doors were always maintained closed during the educational activities

(classes) and physical activities; during recesses and periods with recreational activities they were opened.

Sampling was conducted using constant flow samplers (model Bravo H2; TCR TECORA, Italy) that were combined with PM LVS (low volume system) sampling heads for gas and particulate samples (in compliance with norm EN14907:2011 for $PM_{2.5}$, and PM_1) with an air flow rate of 38.3 L min^{-1}. In order to minimize direct influence of outdoor sources, the samplers were placed as far as possible from windows or doors. Due to the safety precautions, the inlets of the samplers were positioned at height of approximately 1.5 m and minimally 1 m from the walls, without obstructing the normal usage of the rooms. All requirements to maintain child safety were fulfilled. In order to better understand the impacts of outdoor PAH emissions to indoor preschool environment, the levels of PAHs were concurrently measured in ambient air (i.e. outdoors). $PM_{2.5}$–bound PAHs were measured at preschool yard in a safe distance from areas with children intense activity. The samplers were placed in open area avoiding any obstacles and barriers (trees, bushes walls, and fences) that could interfere with data collection. The distance from the main street was 8 m.

$PM_{2.5}$ and PM_1 were collected on polytetrafluoroethylene (PTFE) membrane filters with polymethylpentene support ring (2 μm porosity, Ø47 mm, SKC Ltd., United Kingdom). Gas samples were collected on polyurethane foam (PUF) plugs (75 mm, SKC Ltd., United Kingdom; in compliance with USEPA TO-13A, and ASTM D6209 method specifications) that were pre-cleaned according to Castro et al. (2011).

During sample collection a researcher was present in preschool area who kept a record of room occupancy, ventilations (door and window positions), and potential sources and activities was kept. Teachers and staff were daily questioned concerning the additional occurrence of any potential source (such use of printers, computers, use of additional heating systems, candle burning) and/or indoor activities (students' art activities, cleaning, etc.). In

order to better characterize the sampling conditions, meteorological parameters were registered both indoors and outdoors (Table 1S of the Supplementary material).

2.3 $PM_{2.5}$ and PM_1 masses

$PM_{2.5}$ and PM_1 masses were determined gravimetrically as described previously in detail by Slezakova et al. (2013a, 2014). Briefly, the initial mean mass of the blank filter was subtracted from the final mean mass of the exposed filter; the difference was then divided by the total volume of air that passed through filter (at 25 °C and 101.3 kPa). After the sampling, both filters and PUF plugs were stored in a freezer (– 20 °C) before consequent chemical analysis.

2.4 Extraction and chromatographic analysis of PAHs

The extractions of PAHs from particles ($PM_{2.5}$ and PM_1) and PUF plugs were performed by previously validated analytical procedure (Castro et al. 2009, 2011). Filters and PUF plugs were microwave-assisted extracted with 30 and 45 mL of acetonitrile, respectively for 20 min at 110 ºC. Extracts were reduced to a small volume using a rotary evaporator (Buchi Rotavapor, R–200) at 20 ºC and carefully filtered through a PTFE membrane filter (0.45 μm). 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.

To quantify PAHs, 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 according to a validated study, conducted by Castro et al. (2009, 2011). 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, Duren, Germany) maintained at room temperature

(20 ± 1 ºC). The injected volume was 15.0 μL. A mixture of water and acetonitrile 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 shows limited fluorescence, was analysed at 254 nm in PAD. Each analysis was performed at least in triplicate.

2.5 Quality control

The overall MAE-LC procedure for analysis of PAHs in atmospheric particulate samples was previously validated by systematic recovery experiments and analysing the certified reference material SRM 1650b "Diesel particulate matter" (Castro et al., 2009). PAHs were extracted from particles with recoveries ranging from 81.4 ± 8.8% to 112.0 ± 1.1%, for all the compounds except for naphthalene (62.3 ± 18.0%) and anthracene (67.3 ± 5.7%). The validation of MAE procedure for extracting PAHs from PUFs was performed according to Castro et al. (2011). The extraction efficiency was consistent over the entire range of concentrations and the results ranged from 50.2 ± 3.5% (acenaphthylene) to 107.9 ± 1.5% (fluoranthene) for all PAHs. The presented concentrations were not corrected by the recovery values. External calibrations with PAHs mixed standards, using at least 6 calibration

points, were performed. Calibration curves were linearly fitted with correlation coefficients always higher than 0.9997 for all PAHs. Limits of detection (LODs) (Miller and Miller, 2000) between 1.0 pg m^{-3} (for anthracene, benzo[k]fluoranthene, chrysene, benz[a]anthracene, phenanthrene and indeno[1,2,3-cd]pyrene) and 148 pg m^{-3} (for acenaphthylene) were obtained, with corresponding limits of quantification (LOQs) in the range 3.4–492 pg m^{-3}. During each set of MAE extractions, a filter blank or a PUF plug blank was included. The repeatability was evaluated by the relative standard deviations (RSD) of triplicate samples. RSD values ranged from 1.8 (dibenzo[a,l]pyrene) to 9.1% (naphthalene) and 0.9 (chrysene) to 9.8% (naphthalene) for PAHs extraction from filters and PUFs, respectively. Standards used for calibration were analysed daily and regularly, as well as blank MAE extracts (from filter blank or PUF plug blank), between samples to check instrument performance during PAHs analysis. Each analysis was run at least in triplicate.

2.6 Exposure assessment

The dose rates from inhalation exposure to PAHs in preschool environment were calculated using Equation 1 (Kalaiarasan et al., 2009):

$$\text{Dose rate (D)} = (BR_{WA}/BW) \times C_{WA} \times OF \times N \quad (1)$$

where D is the age-specific dose rate (ng kg^{-1} day^{-1}); BR_{WA} is the age–specific weighted average breathing rate (L min^{-1}); BW is age-specific body weight (kg); C_{WA} is the time-weighted average concentration (ng L^{-1}); OF is the occupancy factor (considered 1, as children kept their schedules and associated locations tightly); N is the total time per day spent by age-specific children in the preschool (min day^{-1}). The dose rates were estimated for 3–5 years old children. The daily activity patterns of these children were analysed throughout each day. During the sampling period children spent approximately 8 hours at the preschool during each day. The daily residence time of children indoors and outdoors and the types of

activities performed were registered. Each activity was characterized in terms of intensity level in order to assess the corresponding BR. An example of children timetable and activity patterns is shown in Table 2S of the Supplementary material. As the information concerning the Portuguese population is not available, the age–specific factors were retrieved from USEPA data (USEPA, 2011) considering the mixed population (both male and females). BW of 18.6 kg for 3–5 years old children was used. The values of BR were selected as the followings: 4.3 L min^{-1} for rest or sleep; 4.5 L min^{-1} for sedentary or passive activities; 11.0 L min^{-1} for light intense activity, and 37.0 L min^{-1} for highly intense activities (running, etc.). BR_{WA} was estimated then as weighted average, i.e. considering the intensity of each performed activities and the amount of time. The exposure doses were then estimated using the average indoor and outdoor concentrations (weighted by the real time that children spent in each place).

2.7 Health risk analysis

The carcinogenic risks of PAHs were assessed according to the methodology provided by USEPA Region III Risk-based Concentration Table (USEPA, 2014). The risks were estimated as the incremental probability of an individual to develop cancer, over a lifetime, as a result of 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 (2):

$$TR = [(EFr \times ED \times ET \times IUR \times C) / AT] \quad (2)$$

where TR is target carcinogenic risk (dimensionless); EFr is the exposure frequency (250 days $year^{-1}$); ED is the exposure duration (years); ET is indoor air exposure time (h day^{-1});

IUR is the chronic inhalation unit risk (μg m^{-3})$^{-1}$ (USEPA, 2014); C is the concentration of PAH (μg m^{-3}); and AT is the number of days over which the exposure is averaged (25 500 days, i.e. 70 years × 365 days $year^{-1}$; USEPA, 2014). The carcinogenic risks were estimated only for PAHs for which IUR values are available (USEPA, 2014), namely: naphthalene (IUR of 3.4×10^{-5} (μg m^{-3})$^{-1}$); chrysene (1.1×10^{-5} (μg 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} (μg m^{-3})$^{-1}$); benzo[a]pyrene (IUR of 1.1×10^{-3} (μg m^{-3})$^{-1}$); and dibenz[a,h]anthracene (1.2×10^{-3} (μg m^{-3})$^{-1}$). The target risks for 3– and 4–5–years old children were estimated using the lowest possible ED (i.e. 1 and 2 years of exposure at the preschool, respectively) in order to not over–estimate the respective cancer risks. Table 3S of the Supplementary material shows an example of TR calculation. For comparison purposes, the health risks for two adult age categories of the school staff were also evaluated: 25–54–years and 55–64–years old (ED of 15 and 40 years of employment, respectively).

2.8 Statistical analysis

Statistical analysis was performed using the SPSS (IBM SPSS Statistics 20) and Statistica software (v. 7, StatSoft Inc., USA). Mean values were compared through the nonparametric Mann−Whitney U test, since normal distribution was not observed by Shapiro−Wilk's test. Statistical significance was defined as $p < 0.05$.

3. Results and discussion

3.1 Indoor PAHs

Over the sampling period, the 9 h concentrations of $PM_{2.5}$ indoors ranged from 9 to 106 μg m^{-3} (mean of 37 μg m^{-3}). The corresponding indoor levels of PM_1 were between 7 and 81 μg m^{-3}, with a mean of 33 μg m^{-3}. The statistical analysis of these results indicated that

indoor $PM_{2.5}$ concentrations were not significantly higher (p = 0.734) than PM_1. Furthermore, the results showed that $PM_{2.5}$ was composed mostly by PM_1 as this fraction accounted for 89% of $PM_{2.5}$.

The levels of PAHs in indoor air of the studied preschool are summarised in Table 1, which shows the 9 h mean concentrations (as well as median and ranges) of all collected samples presented as sums of individual compounds according to the number of aromatic rings (i.e. groups with 2, 3, 4, 5 and 6 rings) in particulate ($PM_{2.5}$, PM_1) and the gas phases. In agreement with the previous studies on indoor air quality in Oporto Metropolitan Area (Castro et al., 2011; Slezakova et al., 2014) compounds with 5 and 6 aromatic rings were the most abundant groups of PM-bound PAHs, accounting, respectively, for 47% and 21% of Σ_{PAHs} in $PM_{2.5}$ and 51% and 22% in PM_1. These results were in general agreement with the previous indoor studies conducted in this area (Castro et al., 2011; Slezakova et al., 2014). Specifically, dibenz[a,h]anthracene (5 rings) was the most abundant PAH in indoor air of the studied preschool (mean of 1.74 ng m^{-3} and 1.52 ng m^{-3} in $PM_{2.5}$ and PM_1, respectively, i.e. 25 and 28% of Σ_{PAHs}); the dominance of this compound indicates emissions from motor light-duty gasoline vehicles (Ravindra et al., 2008). The other most abundant PAHs in $PM_{2.5}$ and PM_1 were, by descending order, benzo[b+j]fluoranthene (5 rings; 0.934 and 0.757 ng m^{-3} in $PM_{2.5}$ and PM_1, respectively, i.e. approximately 14% of Σ_{PAHs}), indeno[1,2,3–cd]pyrene (6 rings; 13–14% in PM_1 and $PM_{2.5}$, respectively) and benzo[ghi]perylene (6 rings; 7% in $PM_{2.5}$, 9% in PM_1). Compounds with 3 rings, namely fluorene, phenanthrene, and anthracene were the least abundant particulate–bound PAHs (approximately 3% and 8% of Σ_{PAHs} in PM_1 and $PM_{2.5}$) but they accounted for 66% of gaseous Σ_{PAHs}, being the most abundant ones (means of 1.77 ng m^{-3}, 8.87 ng m^{-3}, and 14.0 ng m^{-3}, respectively). Finally, PAH with 2 rings, namely naphthalene, was the second most abundant gaseous compounds (mean of 12.8 ng m^{-3}) and accounted for 29% of gaseous Σ_{PAHs}. Specifically, the mean of naphthalene in indoor air of

the studied preschool reached a value of 13.6 ng m^{-3} which was below the WHO recommend annual guideline value of 10 μg m^{-3} (WHO, 2010).

As demonstrated in Table 1, gaseous PAHs accounted in total for 87% of Σ_{PAHs}. The distribution of PAHs between particles and gas phase predominantly depends on the physical characteristics of the compounds and on the physical conditions of the studied environments such as temperature and relative humidity (Ravindra et al., 2008). Whereas PAHs with higher molecular weight (5 and more aromatic rings) are typically associated with particles, compounds with 2 and 3 rings are mostly found in gas phase (Guo et al., 2011). As demonstrated in Figure 1, phase–distribution of PAHs in indoor air of the studied preschool was in agreement with these findings (Krugly et al., 2014). Compounds with 5 aromatic rings (benzo[b+j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, and dibenz[a,h]anthracene were predominantly bound to particles (i.e. more than 92%) whereas PAHs with 6 rings (dibenzo[a,l]pyrene, benzo[ghi]perylene, and indeno[1,2,3–cd]pyrene) were entirely present in the PM (i.e. 100%). PAHs with 2 and 3 aromatic rings were almost entirely present in the gaseous phase (i.e. 98%) Finally, PAHs with 4 rings (fluoranthene, pyrene, benz[a]anthracene, and chrysene) were distributed between both phases with mean proportion of 60% in gas phase.

On average, PM_1–bound PAHs accounted for 78% particulate Σ_{PAHs} whereas PAHs on particles with aerodynamic diameter between 1.0 and 2.5 μm (i.e. $PM_{1\text{-}2.5}$) accounted for 22% of PAH particulate content. These results thus confirmed the previously reported findings that particulate-bound PAHs are predominantly found in smaller fractions of PM (Klejnowski et al., 2010; Ladji et al., 2014).

Out of 18 PAHs, ten compounds were reported as carcinogenic ones (possible, probable) (IARC, 2002, 2010): naphthalene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]luoranthene, benzo[a]pyrene,

dibenzo[a,l]pyrene, dibenz[a,h]anthracene and indeno[1,2,3–cd]pyrene. The mean concentrations of these carcinogenic PAHs (i.e. $\Sigma_{PAHscarc}$) in indoor PM, gas phase, and in indoor air are reported in Table 1. Dibenz[a,h]anthracene (strong carcinogen with TEF 5 times higher than benzo[a]pyrene; Okona-Mensah et al., 2005) was the most abundant carcinogen and accounted for approximately 35% of $\Sigma_{PAHscarc}$ in both PM. In a view of protection of public health, it is important to point out that this compound exhibited in indoor air of the studied preschool the highest concentrations of all 18 PAHs in both PM, being followed by other carcinogens: benzo[b+j]fluoranthene (19 and 18% of $\Sigma_{PAHscarc}$ in $PM_{2.5}$ and PM_1, respectively) and indeno[1,2,3–cd]pyrene (19 and 16% of $\Sigma_{PAHscarc}$ in $PM_{2.5}$ and PM_1, respectively). Benzo[a]pyrene, the most characterized carcinogen (IARC, 2010), was the fourth most abundant carcinogenic PAH, contributing 7 and 8% of $\Sigma_{PAHscarc}$ in $PM_{2.5}$ and PM_1, respectively. Concerning the gas phase, naphthalene was the predominant carcinogenic PAH (96% of gaseous $\Sigma_{PAHscarc}$); the content of other carcinogenic PAHs was much less significant (i.e. 0.1–1.5% of gas $\Sigma_{PAHscarc}$). In addition, the obtained results showed that 72% of $\Sigma_{PAHscarc}$ existed in the gas phase whereas 28% was particulate–bound. Despite this distribution, carcinogenic PAHs accounted for 30% of the gaseous PAH content whereas it was 73 and 76% of Σ_{PAHs} in $PM_{2.5}$ and PM_1, respectively; being in similar to other studies (Jyethi et al., 2014). Finally, in agreement with the obtained results carcinogenic particulate PAHs were predominantly associated with PM_1 (82%).

3.2 Outdoor PAHs

The 9 h outdoor $PM_{2.5}$ concentrations exhibited similar ranges as indoor ones, with values ranging between 9 and 113 $\mu g\ m^{-3}$ (mean of 32 $\mu g\ m^{-3}$); the estimated indoor and outdoor means were not significantly different ($p = 0.347$).

The levels of $PM_{2.5}$–bound PAHs measured in ambient air (i.e. in preschool yard) are presented in Table 2. In general, the total levels of outdoor $PM_{2.5}$–bound PAHs were not statistically different (p = 0.880) from the indoors one, with concentrations being slightly higher outdoors than indoors. PAHs with 5 rings were the most abundant ones accounting for 39% of Σ_{PAHs}, whereas the proportions of compounds with 3 and 4 rings was lower (21% of Σ_{PAHs}). Specifically, the most abundant individual compounds were (in descending order): dibenz[a,h]anthracene (22% of Σ_{PAHs}), benzo[b+j]fluoranthene (approximately 11%), and indeno[1,2,3–cd]pyrene (8%). Ten carcinogenic PAHs accounted for 60% of the particulate PAHs. This work principally focused on assessment of outdoor particulate PAHs due to fact that majority of individual carcinogenic compounds (i.e. with 4–6 aromatic rings) are of high molecular weights and hence predominantly found in particulate-phase (Slezakova et al., 2011).

3.3. Exposure estimation

The estimated dose rates of PAHs (indoor, outdoor and total) for different age–groups of children at the studied preschool are presented in Table 3. At the preschool, 3–5 years old children were divided into the classes according to their age. These age–classes had different daily schedules and activities which could have influenced the overall child dose rates of PAHs. For example the 3–years old children slept after lunch for 2–2.5 h whereas older children spent daily more times outdoors (0.75–1.75 h). The results in Table 3 show that total dose rates were not significantly different being approximately up to 1.4 (5-6 ringed PAHs) times higher for 4–5–years old children than for younger ones. Older children spent approximately twice more time outdoors (22% of their school time) than younger ones (9% of school time). In addition, older children performed more frequently physical activities such as exercising, running, and playing (both indoors and outdoors) which were associated with the

highest breathing rates. In agreement with these findings, dose rates due to outdoor PAHs were approximately twice higher for older children than for younger ones. Specifically, the dose rates due PAHs outdoors contributed for older children between 7% (PAHs with 3 rings) till 56% (compounds with 5 rings) of the total PAHs school doses whereas for 3–years old children it was between 3% (3–ringed PAHs) and 34% (compounds with 5 rings).

3.4 Risk assessment

Several approaches have been developed to evaluate the potencies of the components of a complex mixture of PAHs. Typically, most of the authors (and in this work) use TEF values estimated by Nisbeth and La Goy, 1992 (Boström et al., 2002). However, these authors did not report TEF value for dibenzo[a,l]pyrene, which is a relevant compound from the health point of view (Okona-Mensah et al., 2005). Therefore, in this work, a TEF value estimated by Muller (Boström et al., 2002) was used in order to calculate the TEF–adjusted concentration of this PAH. The results of TEF–adjusted concentrations for 18 PAHs at the studied preschool are presented in Table 4. These results demonstrate that total TEF-adjusted concentrations of 18 PAHs ($\Sigma_{TEF\text{-}PAHs}$) in $PM_{2.5}$ outdoors (14.1 ng m^{-3}) were very similar (p = 1) to the indoors. Mean $\Sigma_{TEF\text{-}PAHs}$ in indoor air reached a value of 14.4 ng m^{-3} (in $PM_{2.5}$ and 0.270 ng m^{-3} in gas phase) and was in the similar range as values reported in other studies (Krugly et al., 2014). Dibenz[a,h]anthracene with TEF of 5 was the largest contributor (59%) to $\Sigma_{TEF\text{-}PAHs}$ in indoor air. The concentration of dibenzo[a,l]pyrene were low in indoor air (7.43×10^{-2} ng m^{-3}, i.e. less than 0.2%) but due to its high TEF (100) it was the second largest contributor to $\Sigma_{TEF\text{-}PAHs}$ (35%) in indoor air of the studied preschool. These results emphasize the importance of the analysis and evaluation of these two potent carcinogens that are being discussed as possible surrogate compounds for PAH mixtures from various environments (Okona-Mensah et al., 2005). Concerning the naphthalene, this PAH made the largest

contribution to total PAH content in indoor air of the preschool (26% of Σ_{PAHs}; Table 1), but its contribution to TEF–adjusted concentrations was less than 1% (Table 4). Nevertheless, in a view of WHO guidelines (WHO, 2010) this gaseous PAH should be considered when assessing the health risks of PAHs. Furthermore, the high abundance of naphthalene in the gas phase indicates that that this compound should be routinely monitored in indoor air.

The values of $\Sigma_{TEF\text{-}PAHs}$ were used to estimate the corresponding lifetime lung cancer risks for the exposed populations. Concerning the lung cancer risk for PAH mixtures, for indoor air WHO suggests the unit risk of 8.7×10^{-5} (ng m^{-3})$^{-1}$ of benzo[a]pyrene for lifetime (70 years) exposure (WHO, 2010). Considering that children and teaching staff spent approximately 7 h of their school daily time indoors and 1 h outdoors, the corresponding lung cancer risks were 3.71×10^{-4} in indoor air (3.66×10^{-4} in $PM_{2.5}$ and 5.2×10^{-6} in gaseous phase) and 5.11×10^{-5} for outdoors (i.e. in $PM_{2.5}$). Both estimated values exceeded WHO health–based guideline level of 10^{-5}, approximately 37 and 5 times higher for indoor and outdoor air, respectively (Boström et al., 2002). It is necessary to point out that unit risk guideline of benzo[a]pyrene recommended for indoor air that was used for calculation is based on epidemiological data from studies on coke-oven workers. It implies that benzo[a]pyrene represents the same proportion of carcinogenic activity of the PAH mixture as in the occupational exposure used to derive the unit risk (WHO, 2010). Although this assumption is probably not correct, the associated uncertainties in risk estimates are unlikely to be large (WHO, 2010).

The means of target carcinogenic risks associated with inhalation exposure to PAHs for two children age–groups (3 years and 4–5–years old) were estimated by USEPA methodology (Table 5). USEPA set a risk level of 10^{-6} for carcinogenic 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. The results in Table 5 show that total and individual carcinogenic target risks of all PAHs, both in indoor air and outdoors, were below 10^{-6} and thus can be considered as negligible. Total carcinogenic risks due to indoor particulate–bound PAHs were higher than outdoor ones (13 and 4 times for 3– and 4–5–years old children, respectively), which was mostly due to the prolonged periods that children spent indoors (7.25 and 6.25 h indoors versus 0.75 and 1.75 h outdoors, respectively). Furthermore, indoor risk values were approximately 4 times higher for particulate–bound PAHs than for gaseous compounds. Overall, indoor exposure contributed 93% and 78% of the overall (i.e. both indoor and outdoor) school risks (ΣTR_{school}) for 3– and 4–5–years old children, respectively. Finally, considering different age groups, the total cancer risks of overall (i.e. both indoors and outdoors) school exposure (ΣTR_{school}) were approximately 2.1 times higher (but still negligible) for 4-5–years old children than for 3–years old. These differences were firstly caused by different exposure time (1 versus 2 years) and secondly, by different daily schedules. For comparison purposes, the health risks for two adult age categories of the school staff (25–54 and 55–64 years old) were also evaluated (Table 4S). The values of total cancer risks of overall school exposure (ΣTR_{school}) ranged from 1.60×10^{-7} to 4.26×10^{-7} for 25–54– and 55–64–years old adults, respectively. Whereas these cancer risks were negligible (lower than 10^{-6}), they were 7–40 times higher than of children, mostly due to longer period (i.e. ED of 15 and 40 years versus 1–2 years) of the respective exposures.

It is necessary to point out that dibenzo[a,l]pyrene was not considered for the evaluation of carcinogenic risks by USEPA methodology, because 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.

Finally, it is necessary to point out that apart from the PAH carcinogenicity, other health effects are relevant. Short-term exposure to PAHs has been reported to cause impaired lung function in asthmatics and thrombotic effects in people affected by coronary heart disease (Kim et al., 2013). Especially for children, Annesi-Maesano et al. (2007) reported an increased risk for flexural dermatitis in subjects exposed to high levels of traffic-related air pollution. The authors pointed out that particles may enhance inflammatory reactions, which could be due to the intervention of PAHs contained in PM. Bae et al. (2010) also found evidence of a synergistic effect of exposure to high levels of PM, PAH and oxidative stress in schoolchildren. In order to fully understand the health implications, more studies concerning schoolchildren exposure to PAHs are needed.

4. **Conclusions**

This study provides new data concerning levels, exposure dose rates and risk assessment of PAHs in indoor air of a preschool environment as there is little information available in literature (in regard to gaseous compounds or PAHs bound to finest PM fraction). Overall, it was possible to conclude that obtained levels of particulate and gaseous PAHs (Σ_{PAHS} of 0.721–15.9 ng m^{-3}, and 27.1–66.1 ng m^{-3}) were similar to those reported for some other European schools (Gatto et el., 2014; Krugly et al., 2014).

Particulate PAHs were predominantly associated with PM_1 (76% of particulate ΣPAHs) with 5–ringed PAHs being the most abundant compounds. These smaller classes of particles such as PM_1 are more apt to cause respiratory toxicity and dysfunction due to their ability to deposit deep in the lower airways. Health hazardous compounds such as PAHs that are bound to these small particles, may eventually enhance (or at least contribute to) the PM–induced adverse health effects (Kim et al., 2013; Saravia et al., 2013).

Gaseous PAHs accounted for 87% of Σ_{PAHs}. The high abundance of PAHs in the gas phase indicates that adequate assessment of PAHs exposure requires considerations of the gaseous compounds indoors and outdoors, which are often neglected.

Total carcinogenic risks due indoor particulate–bound PAHs were higher than for outdoor ones (10 and 3 times for 3– and 4–5–years old children, respectively), which was mostly due to the prolonged periods that children spent indoors. Health risks assessment based on USEPA methodology revealed that cancer risks of both preschool children and the respective staff were negligible, however WHO health–based guideline level of 10^{-5} was (37 and 5 times) exceeded.

As children represent one of the most vulnerable groups in society, more information concerning their exposure in the preschool environments is necessary. In order to provide a correct representation of child's overall preschool exposure, the future study need to account

for PAH exposures occurring in different preschool microenvironments (i.e. classroom, canteens, gym or libraries) as no information on topic exists. A more comprehensive assessment should include higher number of schools, preferentially from different geographical areas, various indoor microenvironments, and exposure assessment *via* the use of biomonitoring coupled with personal air sampling.

Acknowledgments

This work was supported by Fundação para Ciência e Tecnologia through fellowships SFRH/BD/80113/2011, SFRH/BPD/65722/2009. It also received financial support from the European Union (FEDER funds through COMPETE) and National Funds (Fundação para a Ciência e Tecnologia) through projects Pest-C/EQB/LA0006/2013 and PEst-C/EQB/UI0511/2013.

References

Alves CA, Urban RC, Pegas PN, Nunes T (2014) Indoor/Outdoor relationships between PM10 and associated organic compounds in a primary school. Aerosol Air Qual Res 14:86–98.

Annesi-Maesano I, Moreau D, Caillaud D, Lavaud F, Le Moullec Y, Taytard A, Pauli G, Charpin D (2007) Residential proximity fine particles related to allergic sensitisation and asthma in primary school children. Resp Med 101:1721–1729.

Bae S, Pan X-C, Kim S-Y, Park K, Kim Y-H, Kim H, Hong, Y-C (2010) Exposures to particulate matter and polycyclic aromatic hydrocarbons and oxidative stress in schoolchildren. Environ Health Persp 118:579–583.

Boström CE, Gerde P, Hanberg A, Jernstrom B, Johansson C, Kyrklund T, Rannug A, Tornqvist M, Victorin K, Westerholm R (2002) Cancer risk assessments, indicators, and guidelines for polycyclic aromatic hydrocarbons in the ambient air. Environ Health Perspect 10:451–488.

Burtscher H, Schüepp K (2012) The occurrence of ultrafine particles in the specific environment of children. Paediatr Respir Rev 13(2):89–94.

Castro D, Slezakova K, Delerue–Matos C, Alvim–Ferraz M, Morais S, Pereira MC (2011) Polycyclic aromatic hydrocarbons in gas and particulate phases of indoor environment influenced by tobacco smoke: levels, phase distribution and health risks. Atmos Environ 45:1799–1808.

Castro D, Slezakova K, Oliva–Teles MT, Delerue–Matos C, Alvim–Ferraz MC, Morais, S, Pereira MC (2009) Analysis of polycyclic aromatic hydrocarbons in atmospheric particulate samples by microwave–assisted extraction and liquid chromatography. J Sep Sci 32:501–510.

Chen C, Zhao B, Zhou WT, Jiang XY, Tan ZC (2012) A methodology for predicting particle penetration factor through cracks of windows and doors for actual engineering application. Build Environ 47:339–348.

Cirillo T, Montuori P, Mainardi P, Russo I, Triassi M, Amodio-Cocchieri R (2006) Multipathway polycyclic aromatic hydrocarbon and pyrene exposure among children living in Campania (Italy). J Environ Sci Health A Tox Hazard Subst Environ Eng 41:2089–2107.

Cohen Hubal EA, Sheldon LS, Burke JM, McCurdy TR, Berry MR, Rigas ML, Zartarian VG, Freeman NC (2000) Children's exposure assessment: a review of factors influencing Children's exposure, and the data available to characterize and assess that exposure. Environ Health Perspect 108(6):475–486.

Edwards RD, Schweize C, Llacqua V, Lai HK, Jantunen M, Bayer-Oglesby L, Künzli N (2006) Time–activity relationships to VOC personal exposure factors. Atmos Environ 40: 5685–5700.

Gatto MP, Gariazzo C, Gordian A, L'Episcopo N, Gherardi M (2014) Children and elders exposure assessment to particle-bound polycyclic aromatic hydrocarbons (PAHs) in the city of Rome, Italy. Environ Sci Pollut Res 21:13152–13159.

Guo Y, Wu K, Huo X, Xu X (2011) Sources, distribution, and toxicity of polycyclic aromatic hydrocarbons. J Environ Health 73: 2–25.

Hanedar A, Alp K, Kaynak B, Avşar E (2014) Toxicity evaluation and source apportionment of Polycyclic Aromatic Hydrocarbons (PAHs) at three stations in Istanbul, Turkey. Sci Total Environ 488–489:437–446.

IARC Working Group on the Evaluation of Carcinogenic Risks to Humans (2002) Some traditional herbal medicines, some mycotoxins, naphthalene and styrene. IARC Monogr Eval Carcinog Risks Hum 82:1–556.

IARC Working Group on the Evaluation of Carcinogenic Risks to Humans (2010) Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. IARC Monogr Eval Carcinog Risks Hum 92:1–853.

Jyethi DS, Khillare PS, Sarkar S (2014) Risk assessment of inhalation exposure to polycyclic aromatic hydrocarbons in school children. Environ Sci Pollut Res 21:366–378.

Kalaiarasan M, Balasubramanian R, Cheong KWD, Tham KW (2009) Traffic-generated airborne particles in naturally ventilated multi-storey residential buildings of Singapore: Vertical distribution and potential health risks. Build Environ 44(7):1493–1500.

Kim KH, Jahan SA, Kabir E, Brown RJ (2013) A review of airborne polycyclic aromatic hydrocarbons (PAHs) and their human health effects. Environ Int 60:71–80.

Klejnowski K, Kozielska B, Krasa A, Rogula-Kozlowska W (2010) Polycyclic aromatic hydrocarbons in PM1, PM2.5, PM10 and TSP in the upper Silesian agglomeration. Poland Archives of Environmental Protection 36:65–72.

Krugly E, Martuzevicius D, Sidaraviciute R, Ciuzas D, Prasauskas T, Kauneliene V, Stasiulaitiene I, Kliucininkas L (2014) Characterization of particulate and vapor phase polycyclic aromatic hydrocarbons in indoor and outdoor air of primary schools. Atmos Environ 82:298–306.

Ladji R, Yassaa N, Balducci C, Cecinato A (2014) Particle size distribution of n-alkanes and polycyclic aromatic hydrocarbons (PAHS) in urban and industrial aerosol of Algiers, Algeria. Environ Sci Pollut Res 21:1819–1832.

Miller JN, Miller JC (2000) Statistics for analytical chemistry. Pearson Education Ltd., Harlow.

Moshammer H, Neuberger M (2003) The active surface of suspended particles as a predictor of lung function and pulmonary symptoms in Austrian school children. Atmos Environ 37:1737–1744.

Okona-Mensah KW, Battershill J, Boobis A, Fielder R (2005) An approach to investigating the importance of high potency polycyclic aromatic hydrocarbons (PAHs) in the induction of lung cancer by air pollution. Food Chem Toxicol 43:1103–1116.

Qi H, Li W-L, Zhu N-Z, Ma W-L, Liu L-Y, Zhang F, Li Y-F (2014) Concentrations and sources of polycyclic aromatic hydrocarbons in indoor dust in China. Sci Total Environ 491–492:100–107.

Ravindra K, Sokh R, Grieken RV (2008) Atmospheric polycyclic aromatic hydrocarbons: source attribution, emission factors and regulation. Atmos Environ 42:2895–2921.

Ruchirawat M, Navasumrit P, Settachan D, Autrup H (2006) Environmental impacts on children's health in Southeast Asia: genotoxic compounds in urban air. Ann N Y Acad Sci 1076:678–690.

Ruchirawat M, Settachan D, Navasumrit P, Tuntawiroon J, Autrup H (2007) Assessment of potential cancer risk in children exposed to urban air pollution in Bangkok, Thailand. Toxicol Lett 168:200–209.

Saravia J, Lee GI, Lomnicki S, Dellinger B, Cormier SA (2013) Particulate matter containing environmentally persistent free radicals and adverse infant respiratory health effects: a review. J Biochem Mol Toxicol 27:56–68.

Schüepp K, Sly PD (2012) The developing respiratory tract and its specific needs in regard to ultrafine particulate matter exposure. Paediatr Respir Rev 13(2):95–99.

Schwab M, McDermot A, Spengler JD (1992) Using longitudinal data to understand children's activity patterns in an exposure context: Data from the Kanawha County Health Study. Environ Int 18(2):173–189.

Shen G, Wei S, Zhang Y, Wang R, Wang B, Li W, Shen H, Huang Y, Chen Y, Chen H, Wei W, Tao S (2012) Emission of oxygenated polycyclic aromatic hydrocarbons from biomass pellet burning in a modern burner for cooking in China. Atmos Environ 60:234–237.

Slezakova K, Castro D, Delerue-Matos C, Morais S, Pereira MC (2014) Levels and risks of particulate-bound PAHs in indoor air influenced by tobacco smoke: A field measurement. Environ Sci Pollut Res 21:4492–4501.

Slezakova K, Pires JCM, Castro D, Alvim-Ferraz MC, Delerue-Matos C, Morais S, Pereira MC (2013a) PAH air pollution at a Portuguese urban area: Carcinogenic risks and sources identification. Environ Sci Pollut Res 20:3932–3945.

Slezakova K, Castro D, Delerue-Matos C, Alvim-Ferraz MC, Morais S, Pereira MC (2013b) Impact of vehicular traffic emissions on particulate-bound PAHs: Levels and associated health risks. Atmos Res 127:141–147.

Slezakova K, Castro D, Begonha A, Delerue-Matos C, Alvim-Ferraz MC, Morais S, Pereira MC (2011) Air pollution from traffic emissions in Oporto, Portugal: Health and environmental implications. Microchem J 99 51–59.

Tuntawiroon J, Mahidol C, Navasumrit P, Autrup H, Ruchirawat M (2007) Increased health risk in Bangkok children exposed to polycyclic aromatic hydrocarbons from traffic-related sources. Carcinogenesis 28:816–822.

United States Environmental Protection Agency (USEPA) (1989) Risk assessment guidance for superfund, vol. I: Human health evaluation manual. EPA/540/1–89/002, Office of Emergency and Remedial Response, Washington, DC.

United States Environmental Protection Agency (USEPA) (2011) Exposure factors handbook 2011 Edition (Final). United States Environmental Protection Agency, Washington, DC.

United States Environmental Protection Agency (USEPA) (2014) Risk–Based Concentration Table. http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/usersguide.htm. Accessed 26 October 2014.

Wilson NK, Chuang JC, Lyu C, Menton R, Morgan MK (2003) Aggregate exposures of nine preschool children to persistent organic pollutants at day care and at home. J Expo Anal Env Epid 13:187–202.

World Health Organization (2010) WHO Guidelines for indoor air quality: selected pollutants. Regional Office for Europe of the World Health Organization, Copenhagen, Denmark.

World Health Organization (2013) State of the science of endocrine disrupting chemicals 2012. United Nations Environment Programme and the World Health Organization, Geneva.

Figure Captions

Figure 1 Distribution of PAHs between particulates and gas phase in indoor air of the studied preschool. PAHs are presented as sums of individual compounds according to the number of aromatic rings, i.e. groups with 2, 3, 4, 5 and 6 rings, respectively. Particulate phase is further divided into PM_1 and $PM_{1–2.5}$ (i.e. particles with aerodynamic diameter between 1.0 and 2.5 μm) fractions.

Table 1

Indoor levels of PAHs in $PM_{2.5}$, PM_1, gas phase, and in air (i.e. total) in the studied preschool (ng m^{-3}). Concentrations are presented as sums of individual compounds according to the number of aromatic rings, i.e. groups with 2, 3, 4, 5 and 6 rings, respectively.

	Particulate phase (n=30)				Gas phase (n=15)		Air	
Compounds	$PM_{2.5}$		PM_1					
	Mean (Min–Max)	Median	Mean (Min–Max)	Median	Mean (Min–Max)	Median	Mean (Min–Max)	Median
2 rings	0.317 (n.d.– 0.867)	0.229	0.311 (n.d.7.58– 0.602)	0.220	12.8 (7.58–17.3)	12.3	13.1 (7.58–18.1)	12.6
3 rings	0.582 (8.51×10^{-2}–0.724)	0.224	0.179 (2.01×10^{-2}–0.428)	9.66×10^{-2}	29.5 (17.7–50.8)	29.8	30.1 (17.7–51.5)	29.3
4 rings	1.31 (0.230–2.68)	1.12	0.933 (0.141–2.32)	0.787	1.93 (1.46–2.02)	1.68	3.23 (1.69–4.69)	2.84
5 rings	3.22 (0.354–9.32)	2.26	2.76 (0.158–9.05)	1.54	0.281 (0.192–0.299)	0.201	3.50 (0.546–9.35)	3.32
6 rings	1.46 (9.65×10^{-2}–3.30)	1.02	1.18 (0.102–3.05)	0.748	n.d. (n.d.–n.d.)	n.d.	1.46 (9.65×10^{-2}–3.30)	1.43
Σ_{PAHs}	6.89 (0.721–15.9)	4.99	5.40 (0.534–14.7)	3.63	44.5 (27.1–66.1)	35.4	51.4 (27.8–82.0)	43.8
$\Sigma_{PAHscarc.}$	5.05 (0.426–12.3)	3.62	4.12 (0.158–11.5)	2.52	13.3 (12.0–17.6)	13.6	18.4 (12.4–25.9)	15.4

n.d.– not detected

Table 2

Levels of $PM_{2.5}$–bound PAHs (presented as sums of individual compounds according to the number of aromatic rings, i.e. groups with 2, 3, 4, 5 and 6 rings, respectively) in outdoor air of the studied preschool (ng m^{-3})

Compound	Mean (n = 15)	Min–Max	Median
2 rings	0.304	n.d. – 0.696	0.156
3 rings	1.63	0.134 – 2.37	0.298
4 rings	1.65	0.701 – 4.08	1.21
5 rings	3.01	0.804 – 9.88	1.88
6 rings	1.14	0.239 – 3.36	0.622
Σ_{PAHs}	7.73	2.22 – 9.88	8.40
$\Sigma_{PAHscarc}$	4.54	1.01 – 13.3	3.62

n.d.– not detected

Table 3

Mean dose rates of PAHs for 3– and 4–5 years old children at the studied preschool (ng kg^{-1} day^{-1})

3–years old children	Dose rate (ng kg^{-1} day^{-1})					
	Indoor				Outdoor	Total
	Particulate phase		Gas phase	Σ_{indoor}	$PM_{2.5}$	
Compounds	$PM_{2.5}$	PM_1				
2 rings	4.79×10^4	4.69×10^4	1.94×10^6	1.99×10^6	2.74×10^4	2.01×10^6
3 rings	8.06×10^4	2.14×10^4	4.42×10^6	4.50×10^6	1.40×10^5	4.64×10^6
4 rings	1.28×10^5	9.46×10^4	2.96×10^5	4.24×10^5	1.01×10^5	5.25×10^5
5 rings	4.86×10^5	4.17×10^5	4.25×10^4	5.29×10^5	2.68×10^5	7.97×10^5
6 rings	2.21×10^5	1.78×10^5	–	2.21×10^5	1.02×10^5	3.23×10^5
$\sum_{\text{TEF-PAHs}}$	1.04×10^6	8.10×10^5	6.73×10^6	7.77×10^6	6.92×10^5	8.46×10^6
4–5 years old children						
Compounds						
2 rings	4.37×10^4	4.28×10^4	1.77×10^6	1.81×10^6	6.38×10^4	1.87×10^6
3 rings	7.35×10^4	1.96×10^4	4.03×10^6	4.10×10^6	3.28×10^5	4.43×10^6
4 rings	1.17×10^5	8.63×10^4	2.70×10^5	3.87×10^5	2.35×10^5	6.22×10^5
5 rings	4.44×10^5	3.81×10^5	3.88×10^4	4.83×10^5	6.26×10^5	1.11×10^6
6 rings	2.02×10^5	1.62×10^5	–	2.02×10^6	2.38×10^5	4.40×10^5
$\sum_{\text{TEF-PAHs}}$	9.50×10^5	7.39×10^5	6.14×10^6	7.09×10^6	1.62×10^6	8.71×10^6

Table 4

TEF–adjusted mean concentrations of PAHs in indoor and outdoor air of the studied preschool (pg m^{-3})

		Indoor				Outdoor
	TEF[a]	Particulate phase (n=30)		Gas phase	Air	$PM_{2.5}$ (n=15)
Compound		$PM_{2.5}$	PM_1	(n=15)		
Naphthalene	0.001	0.317	0.3106	12.8	13.1	0.306
Acenaphthylene	0.001	n.d.	n.d.	4.55	4.55	1.15
Acenaphthene	0.001	0.348	n.d.	1.77	2.12	n.d.
Fluorene	0.001	3.76×10^{-2}	2.57×10^{-2}	8.88	8.91	8.74×10^{-2}
Phenanthrene	0.001	0.148	0.116	14.0	14.2	0.336
Anthracene	0.01	0.488	0.372	2.41	2.90	0.639
Fluoranthene	0.001	0.487	0.371	0.572	1.06	0.553
Pyrene	0.001	0.310	0.217	1.15	1.46	0.506
Benz[a]anthracene	0.1	11.9	9.49	1.70	13.6	13.3
Chrysene	0.01	3.92	2.49	1.89	5.81	4.61
Benzo[b+j]fluoranthene	0.1	93.4	75.7	12.2	106	82.9
Benzo[k]fluoranthene	0.1	20.3	16.4	1.51	21.9	18.3
Benzo[a]pyrene	1	341	322	144	485	286
Dibenzo[a,l]pyrene	100[b]	5 140	2 100	n.d	5 140	5 130
Dibenz[a,h]anthracene	5	8 670	7 580	n.d	8 670	8 500
Benzo[ghi]perylene	0.01	4.61	4.85	n.d	4.61	5.01
Indeno[1,2,3-cd]pyrene	0.1	94.9	67.1	n.d	94.9	59.0
$\sum_{\text{TEF-PAHs}}$	–	14 410	10 180	207	14 620	14 110

[a]TEF values estimated by Nisbeth and LaGoy 1992 (Boström et al., 2002)

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

Table 5

Estimated target carcinogenic risks (TR) of PAHs for different age categories children

Children 3–years old	TR_{indoor}			ΣTR_{indoor}	$TR_{outdoor}$	ΣTR_{school}
	$PM_{2.5}$	PM_1	Gas phase		$PM_{2.5}$	
Naphthalene	3.18×10^{-11}	3.12×10^{-11}	1.29×10^{-9}	1.32×10^{-9}	3.18×10^{-12}	1.32×10^{-9}
Benz[a]anthracene	3.87×10^{-11}	3.09×10^{-11}	5.53×10^{-12}	4.42×10^{-11}	4.46×10^{-12}	4.87×10^{-11}
Chrysene	1.28×10^{-11}	8.11×10^{-12}	6.15×10^{-12}	1.89×10^{-11}	1.55×10^{-12}	2.05×10^{-11}
Benzo[b+j]fluoranthene	3.04×10^{-10}	2.46×10^{-10}	3.97×10^{-11}	3.44×10^{-10}	2.79×10^{-11}	3.71×10^{-10}
Benzo[k]fluoranthene	6.61×10^{-11}	5.32×10^{-11}	4.24×10^{-12}	7.04×10^{-11}	6.17×10^{-12}	7.65×10^{-11}
Benzo[a]pyrene	1.11×10^{-9}	1.05×10^{-9}	4.67×10^{-10}	1.58×10^{-9}	9.63×10^{-11}	1.67×10^{-9}
Dibenz[a,h]anthracene	6.17×10^{-9}	1.85×10^{-10}	n.d	6.17×10^{-9}	6.24×10^{-10}	6.80×10^{-9}
Indeno[1,2,3-cd]pyrene	3.09×10^{-10}	2.18×10^{-10}	n.d.	3.09×10^{-10}	1.98×10^{-11}	3.29×10^{-10}
ΣPAHs	8.04×10^{-9}	1.82×10^{-9}	1.81×10^{-9}	9.85×10^{-9}	7.83×10^{-10}	1.06×10^{-8}

Children 4–5–years old	TR_{indoor}			$\Sigma\ TR_{indoor}$	$TR_{outdoor}$	$\Sigma\ TR_{school}$
	$PM_{2.5}$	PM_1	Gas phase		$PM_{2.5}$	
Naphthalene	5.49×10^{-11}	5.38×10^{-11}	2.22×10^{-9}	2.28×10^{-9}	1.48×10^{-11}	2.29×10^{-9}
Benz[a]anthracene	6.67×10^{-11}	5.32×10^{-11}	9.53×10^{-12}	7.63×10^{-11}	2.08×10^{-11}	9.71×10^{-11}
Chrysene	2.20×10^{-11}	1.40×10^{-11}	1.06×10^{-11}	3.26×10^{-11}	7.24×10^{-12}	3.98×10^{-11}
Benzo[b+j]fluoranthene	5.24×10^{-10}	4.25×10^{-10}	6.85×10^{-11}	5.92×10^{-10}	1.30×10^{-9}	1.89×10^{-9}
Benzo[k]fluoranthene	1.14×10^{-10}	9.15×10^{-11}	8.49×10^{-12}	1.23×10^{-10}	2.88×10^{-11}	1.51×10^{-10}
Benzo[a]pyrene	1.91×10^{-9}	1.80×10^{-09}	8.05×10^{-10}	2.72×10^{-9}	4.49×10^{-10}	3.17×10^{-9}
Dibenz[a,h]anthracene	1.06×10^{-8}	9.27×10^{-9}	n.d	1.06×10^{-8}	2.91×10^{-9}	1.36×10^{-8}
Indeno[1,2,3-cd]pyrene	5.32×10^{-10}	3.76×10^{-10}	n.d.	5.32×10^{-10}	9.26×10^{-11}	6.25×10^{-10}
ΣPAHs	1.39×10^{-8}	1.21×10^{-8}	3.13×10^{-9}	1.70×10^{-8}	4.83×10^{-9}	2.18×10^{-8}

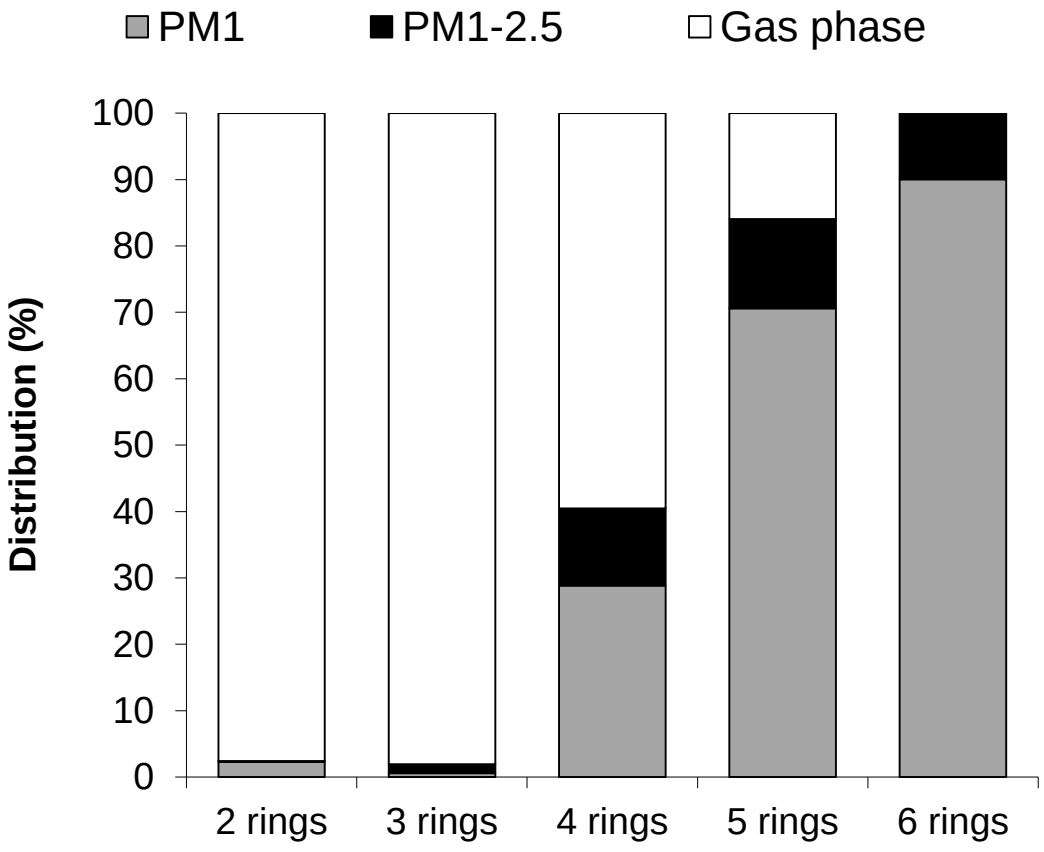
PM1
PM1-2.5
Gas phase
Distribution (%)
100
90
80
70
60
50
40
30
20
10
0
2 rings
3 rings
4 rings
5 rings
6 rings

Figure 1

Click here to download Supplementary Material: Suplementary materials_revised.docx