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ORIGINAL PAPER

Concentration levels and carcinogenic and mutagenic risks of $PM_{2.5}$-bound polycyclic aromatic hydrocarbons in an urban–industrial area in South Africa

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Abstract Concerns over the health effects of exposure to particulate matter of aerodynamic diameter of less than 2.5 μm ($PM_{2.5}$) led the South African Government to establish the national standard for $PM_{2.5}$ in the year 2012. However, there is currently no exposure limit for polycyclic aromatic hydrocarbons (PAHs) and $PM_{2.5}$-bound PAHs. The understanding of the concentration levels and potential health risks of exposure to $PM_{2.5}$-bound PAHs is important in ensuring a suitable risk assessment and risk management plans. This study, therefore, determined the concentration levels and carcinogenic and mutagenic health risks of $PM_{2.5}$-bound PAHs. A hundred and forty-four $PM_{2.5}$ samples were collected over 4 months during the winter and summer seasons of 2016 in an industrial area. The concentrations of 16 PAHs were analysed by gas chromatography–mass spectrometry, and their carcinogenic and mutagenic risks were determined using the Human Health Risk Assessment model. The mean winter (38.20 ± 8.4 μg/m^3) and summer (22.3 ± 4.1 μg/m^3) concentrations of $PM_{2.5}$ levels were lower than the stipulated 40 μg/m^3 daily limit. The daily inhalation and ingestion exposure to PAHs for all age groups were higher than the daily exposure through the dermal contact. Children and adults are more likely to inhale and ingest PAHs in $PM_{2.5}$ than infants. The excess cancer risk and excess mutagenic risk values were below the priority risk level (10^{-4}). There is a potential risk of 1–8 per million persons developing cancer from exposure to benzo[a]anthracene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, and dibenz[a,h]anthracene over a lifetime of 70 years.

Keywords Particulate matter · Polycyclic aromatic hydrocarbons · Health risk assessment · Diagnostic ratio · South Africa

Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a toxic component of particulate matter of aerodynamic diameter of less than 2.5 μm ($PM_{2.5}$). PAHs are widely dispersed in the environment as persistent organic pollutants (Slezakova et al. 2013; Sram et al. 2013). PAHs represent a group of organic substances that are composed of carbon and hydrogen atoms fused into 2–8 aromatic rings and are of great concern to public health (Bortey-Sam et al. 2015).

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PAHs originate mainly from natural processes (Pongpiachan 2015) and anthropogenic sources (Gupta et al. 2011). The major anthropogenic emission sources are formed during incomplete combustion of coal, oil, biofuel, and biomass for domestic and industrial purposes (Shen et al. 2013). PAHs are also released from vehicular emissions, petroleum refining, chemical manufacturing, burning of organic substances, and oil spills (Kang et al. 2017).

The United States Environmental Protection Agency (US EPA) recognised 16 PAHs as priority pollutants due to their toxicity (US EPA 1993). Among the priority PAHs, benzo(a)pyrene (BaP) is the recognised indicator for assessing PAH-related carcinogenicity (Han et al. 2011; Kong et al. 2012). Other carcinogenic PAH species include benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-cd]pyrene, and dibenz[a,h]anthracene, and these have high carcinogenic potential (US EPA 1993).

Human exposure to PAHs can result in adverse health outcomes. PAHs have been tagged carcinogenic and mutagenic by different organisations (Abdel-Shafy and Mansour 2016) due to their association with increased lung, skin, and bladder cancer risks (Mahler et al. 2012). Exposure to PAHs may modify the replication and transcription nature of deoxyribonucleic acid and induce the development of cancerous cells (Armstrong et al. 1994; Boström et al. 2002).

Moreover, exposure to PAHs can also present as endocrine-disrupting chemicals, thereby causing aberrations in the functioning of the reproductive system, neurological disorders, hormonal imbalances, respiratory disorders, and premature births (Kim et al. 2013; Yang et al. 2015). Positive associations between long-term human exposure to small concentrations of PAHs and incidences of cancer (Kim et al. 2013; Mordukhovich et al. 2010), reduced weight at birth (Wilhelm et al. 2012), poor cognitive development (Edwards et al. 2010), oxidative stress (Bae et al. 2010), and obesity (Scinicariello and Buser 2014) have been reported in epidemiological studies.

Though the South African National Ambient Air Quality Standard for $PM_{2.5}$ was established on the 29th of June 2012 in terms of section 9(1) of the National Environmental Management: Air Quality Act, 2004 (DEAT 2012), there are no regulatory standards for PAHs and $PM_{2.5}$-bound PAHs. This is at variance with what is obtainable in most developed countries of the world. In most developed nations, regulation of ambient air quality to protect human health through the formulation of air quality standards is controlled by the government. For instance, in the European Union, BaP in outdoor PM_{10} is set at 1 ng/m^3 (Jose et al. 2013), and at 10 ng/m^3 for the daily mean concentration in the Chinese air (Ding et al. 2012). However, there are no regulatory standards for PAHs in South Africa.

This study provides an ideal avenue to assess the concentration levels of $PM_{2.5}$-bound PAHs in the Pretoria West industrial area vis-a-vis recommended regulatory standards for enforcement action. There is a need for evidence-based research that will determine the levels of PAHs in $PM_{2.5}$ and its associated cancer and mutagenic health risks. This information will be useful for policy formulation for the establishment of a regulatory guideline for PAHs in South Africa. Therefore, the study sought to determine the concentration levels of PAHs in $PM_{2.5}$ during the winter and summer seasons and to establish the health risks (carcinogenic and mutagenic) of $PM_{2.5}$-bound PAHs in different exposure groups.

Methods

Description of the sampling site

The study was carried out in an industrial area located at 25° 44′ 46″ S 28° 11′ 17″ E in the Tshwane Metro known as Pretoria West (Fig. 1). In the Pretoria West industrial area, there are 13 industrial facilities with approved air emission licences and 37 other industrial facilities with small boilers (Environmental Management Services Department 2015). These are in addition to two power plants and metallurgical industries with high stack emission sources, which are recognised emitters of PM. The full description of the study area has been reported in our previous works (Morakinyo et al. 2017a, b, 2019).

Sampling of $PM_{2.5}$

The sampling equipment used for the monitoring of $PM_{2.5}$ was the Beta^{PLUS} Particle measurement system—model 602. The sampling equipment was part of an existing ambient air quality monitoring network sited at the Pretoria West industrial area and managed

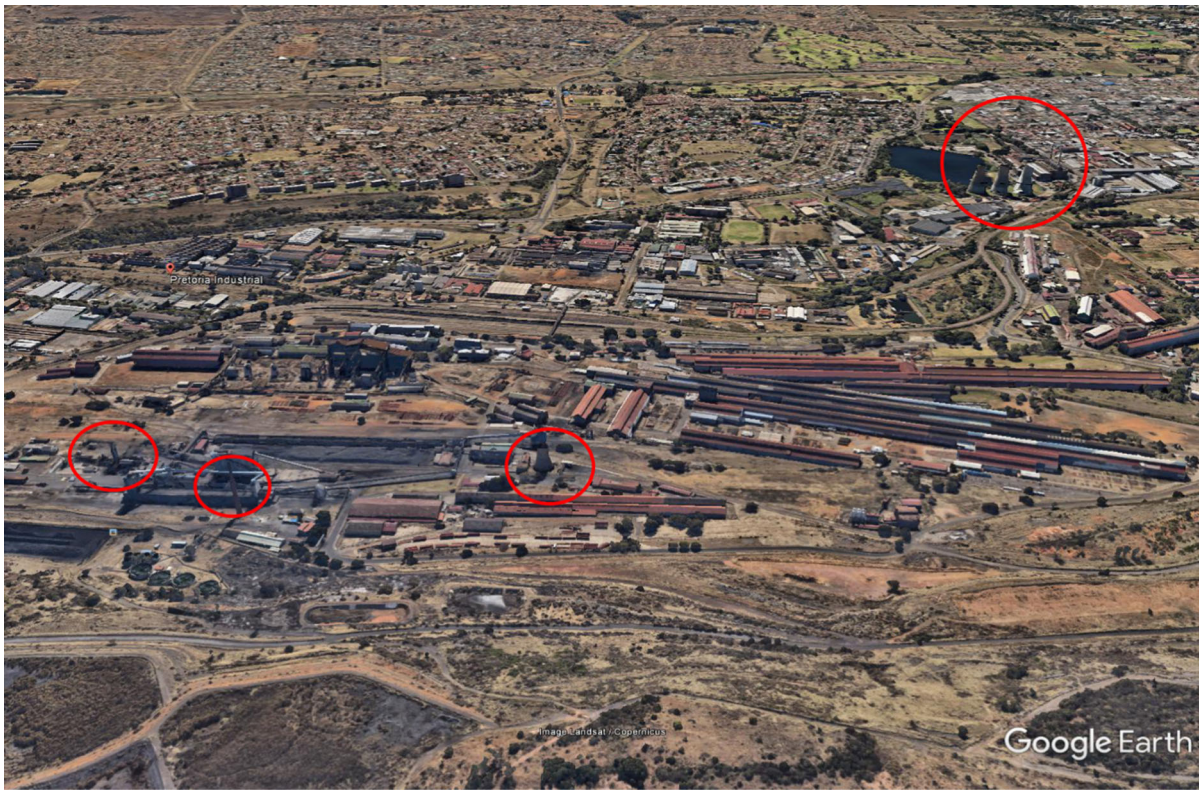

Fig. 1 Google Earth image depicting the Pretoria West industrial area (Morakinyo et al. 2019)

by the Environmental Management Services Department City of Tshwane. This equipment, designed by Teledyne Advanced Pollution Instrumentation, Inc. (Teledyne API), provides the continuous automated monitoring and sampling of $PM_{2.5}$, PM_{10} and $PM_{10-2.5}$ mass concentration present in ambient air. The system provides a completely representative sampling period, actively sampling the air for > 57 min in an hourly mode (Teledyne 2012).

The sampled $PM_{2.5}$ was collected on a 47-mm quartz fibre filter with a porosity of 2 µm by the Beta^{PLUS} Particle measurement system operating at a constant flow rate of 1 m^3/h for 24 h. The instrument was designed in such a way that filters move in sequence from a supply magazine to a sample position and thereafter to the measurement positions and ultimately to a storage magazine for retrieval. In 24 h, the instrument sampled three filters (an average of 8 h on each filter). The $PM_{2.5}$ samples were collected from 1 January 2016 to 29 February 2016 (summer) and from 1 June 2016 to 31 July 2016 (winter). Filters corresponding to Mondays, Wednesdays, and Saturdays for 16 weeks spanning the summer and winter months were retrieved for gravimetric analysis. The selection of the days was based on the preliminary findings from the analysis of secondary ambient pollution data obtained from the South African Weather Service through the South African Air Quality Information System. In all, 144 filters were analysed for $PM_{2.5}$ chemical speciation.

Gravimetric analysis

Filters were preconditioned for 48 h in a desiccator before and after sampling in a temperature and relative humidity controlled room ($T = 20 \pm 1$ °C, RH = 50 $\pm$ 5%) at the Council for Scientific and Industrial Research in Pretoria, South Africa. After weighing, all filters were placed in tin foil packages and stored in a freezer at $-$ 20 °C within 24 h of sampling to prevent loss of volatile components. The sampling scheme also included several duplicate samples and field blank samples. The analysis of the sample took place 3 days after the sample collection.

The gravimetric analysis of $PM_{2.5}$ was conducted following three consecutive weight measurements on a Sartorius ME5-OCE analytical microbalance according to the European Standard EN 14907 (CEN 2005) during the winter and summer seasons. The concentration of $PM_{2.5}$ was calculated from the difference in filter weight before and after measurement, divided by the volume of sampled air.

$$PM_{2.5} = M_{2.5}/V_a$$
$$M_{2.5} = \left(M_{\text{post}} - M_{\text{pre}}\right)$$
$$V_a = \left(Q_{\text{avg}}\right)(T)\left(10^3\right),$$

where $M_{2.5}$ = total mass gain (μg), M_{post} = post-sample filter weight (mg), M_{pre} = pre-sample filter weight (mg), V_a = total sample volume (m^3), Q_{avg} = average sample flow rate (L/min), T = total sample time (min), and 10^3 = units conversion (m^3/L).

Extraction and analysis of PAHs in $PM_{2.5}$

The extraction technique was modified from the procedure used by Chen et al. (2017), Jamhari et al. (2014) and Kong et al. (2011). Measures of 400 ng of aromatic internal standards containing a mixture of acenaphthene $d\text{-}_{10}$, naphthalene-d8, perylene $d\text{-}_{12}$, phenanthrene $d\text{-}_{10}$, and chrysene $d\text{-}_{12}$ were spiked into the quartz filters containing the $PM_{2.5}$ samples for recovery purposes before extraction. Extraction was done three times ultrasonically, each with 50 mL dichloromethane for 20 min in a Soxhlet extractor. This was followed by the purification of extracts through the addition of a sodium sulphate–silica gel column. Thereafter, the extracts were concentrated on a rotary evaporator to approximately 2 mL and subsequently exchanged to hexane by the addition of 20 mL of a dichloromethane and hexane mixture (1:1 v/v). The extracts were re-concentrated on a rotary evaporator to a volume of 250 μL using a gentle stream of nitrogen. To each concentrated sample solution was added 240 ng of hexamethylbenzene as an internal standard, and the solution was stored at 4 °C until analysis (Xu et al. 2012; Liu et al. 2014).

The analysis of the 16 PAHs listed as priority pollutants (Table 1) by the US EPA was performed on an Agilent 7890 gas chromatography–mass spectrometry (GC–MS) fitted with a DB-5MS capillary column of length 30 m, inner diameter 0.25 mm, and thickness 0.25 μm. The injection of samples (1 μL) was done in a designated ion monitoring mode, with 99.99% purity helium gas used as a carrier gas at a flow rate of 1.2 mL/min at a temperature of 280 °C. The temperature of the GC column was set as follows: 50 °C isothermal for 1 min, 5 °C/min from 50 to 140 °C, 4 °C/min from 140 to 300 °C, and 300 °C isothermal for 15 min. The concentrations of the sampled PAHs were calculated from the response factors of the PAHs of the standard solution used (Chen et al. 2007).

Quality control

Quality control was ensured using the US EPA Method TO-13A (US EPA 1999). Field blanks, laboratory blanks, standard spike, and species-dependent recovery analyses were conducted to minimise measurement errors. In achieving quality assurance and determining the detection limits, a total of 72 field blank filters were also analysed.

Blank filters were quantified with each batch of samples and subsequently deducted from the sample concentrations. The detection limit was estimated twice the standard deviation of the field blanks. Target PAHs were not detected in any of the procedural blank samples. The recoveries of used internal standards ranged from 70.9 to 115.4%, and the coefficient of variance was from 2.7 to 12.6%. The stability of the equipment was tested daily using internal standards.

A quarterly quality control (calibration verification) of the monitoring station at three intermediate point checks is routinely done. In addition, the South African National Accreditation System manages the annual calibration of the monitoring station. This complements the weekly routine maintenance carried out by the Environmental Management Services Department of the City of Tshwane to ensure the proper functioning of the monitoring station.

Determination of source of $PM_{2.5}$-bound PAHs

In this study, the likely sources of the PAHs in $PM_{2.5}$ were determined using the isomeric ratio, which has been recognised as an essential tool for determining the characteristics of the specified source of emission (Tobiszewski and Namiesnik 2012; Wiriya et al. 2013) (Table 2). The relative molecular concentration ratios of PAHs are assumed to be a reflection of the given emission source (Mackay et al. 2006). Isomer ratios

Table 1 Toxic equivalent factor, mutagenic potency factor, and cancer slope factor values of PAH compounds

PAHs	Abbreviation	Chemical formula	No. of rings	TEF	MEF	CSF
Acenaphthylene	Acy	$C_{12}H_8$	3	0.001		
Acenaphthene	Ace	$C_{12}H_{10}$	3	0.001		
Anthracene	Ant	$C_{14}H_{10}$	3	0.001		
Benzo[a]anthracene[a]	BaA	$C_{18}H_{12}$	4	0.1	0.082	0.61
Benzo[a]pyrene[a]	BaP	$C_{20}H_{12}$	5	1.0	1	6.1
Benzo[b]fluoranthene[a]	BbF	$C_{20}H_{12}$	5	0.1	0.25	0.061
Benzo[k]fluoranthene[a]	BkF	$C_{20}H_{12}$	5	0.1	0.11	0.061
Benzo[g,h,i]perylene	BPer	$C_{22}H_{12}$	6	0.01	0.19	
Chrysene[a]	Chr	$C_{18}H_{12}$	4	0.1	0.017	0.006
Dibenz[a,h]anthracene[a]	DbA	$C_{22}H_{14}$	5	1.0	0.29	6.1
Fluoranthene	Fla	$C_{16}H_{10}$	4	0.001		
Fluorene	Flu	$C_{13}H_{10}$	3	0.001		
Indeno[1,2,3-cd]pyrene[a]	Ind	$C_{22}H_{12}$	6	0.1	0.31	0.61
Naphthalene	Nap	$C_{10}H_8$	2	0.001		
Phenanthrene	Phe	$C_{14}H_{10}$	3	0.01		
Pyrene	Pyr	$C_{16}H_{10}$	4	0.001		

TEF toxic equivalency factor for carcinogenicity relative to BaP (Nisbet and LaGoy 1992), *MEF* mutagenic potency factor of PAHs relative to BaP (Durant et al. 1999), *CSF* cancer slope factor (mg^{-1} kg day) (US EPA 1992)

[a]Carcinogenic PAHs

Table 2 Diagnostic ratios of polycyclic aromatic hydrocarbons associated with $PM_{2.5}$. *Source*: Wang et al. (2017)

Ratio	Specific value	Source
Ant/(Ant + Phe)	< 0.10	Petroleum
	> 0.10	Combustion
BaA/(BaA + Chr)	< 0.20	Petroleum
	0.20–0.35	Mixed sources
	> 0.35	Combustion
Fla/(Fla + Pyr)	< 0.40	Petroleum
	0.40–0.50	Petroleum combustion
	> 0.50	Grass, wood and coal combustion
Ind/(Ind + Bper)	< 0.5	Petroleum combustion
	> 0.50	Grass, wood, and coal combustion
IcdP/(IcdP + BghiP)	< 0.20	Petroleum
	0.20–0.50	Petroleum combustion
	> 0.50	Grass, wood, and coal combustion
BaP/BghiP	< 0.60	Non-traffic exhaust
	> 0.60	Traffic exhaust
Flo/(Flo + Pyr)	< 0.50	Gasoline vehicle emissions
	> 0.50	Diesel vehicle emissions

such as Fla/(Fla + Pyr), Ant/(Ant + Phe), BaA/(BaA + Chr), and Ind/(Ind + Bper) have been widely used for the identification of PAHs sources (Bootdee et al. 2016; Wiriya et al. 2013).

Estimation of carcinogenic and mutagenic potential of PAHs

The World Health Organization recommended three methods by which the estimation of carcinogenic and mutagenic risks of PAHs can be done, namely (1) benzo[a]pyrene as a surrogate marker, which is based on the assumption that B[a]P is an indicator of all PAHs in a given compound; (2) comparative potency approach, which does not identify or quantify the individual compounds; and (3) toxic equivalent factor (TEF) approach, which is based on the potency of individual PAHs relative to B[a]P in order to obtain a benzo(a)pyrene equivalent (WHO/IPCS 1998).

The first method is based on the assumption of the stability of the composition of PAH mixtures in different exposure scenarios, which is impossible in a natural setting. The second method is dependent on carrying out a bioassay, which was not carried out in this study. The third approach that was used in this study is the use of toxic equivalent factors (TEFs) (Ma et al. 2017). The Environmental Protection Agency proposed that the TEF be used in the conversion of carcinogenic PAHs to B[a]P when estimating the potential risks of exposure to these substances (Sarigiannis et al. 2015). The TEF method uses benzo[a]-pyrene (B[a]P) as a reference owing to its carcinogenic ability and allots potency factors relative to B[a]P for the other compounds in the mixture (Jyethi et al. 2014; Ma et al. 2017). The TEQ has been widely used to assess the risk of carcinogenic potency of each individual PAH (Bootdee et al. 2016; Sarigiannis et al. 2015).

The carcinogenic and mutagenic risks of $PM_{2.5}$-bound PAHs were computed using the carcinogenic toxicity equivalent (BaP-TEQ) and the mutagenic toxicity equivalent (BaP-MEQ) concentrations. These were achieved by multiplying the concentrations of individual PAHs in $PM_{2.5}$ by their respective toxic equivalent factors (TEFs) and the mutagenic potency factors (MEFs) (Callen et al. 2014; Sarkar and Khillare 2012) as shown in Eqs. (1) and (2). Table 1 shows the TEF and MEF values for individual PAHs as recommended by Nisbet and LaGoy (1992) and US EPA (1993).

$$\text{BaP-TEQ} = \text{B[a]P}_{\text{eq}} = \sum_{i}^{n=1} [\text{C}_i \times \text{TEF}_i] \tag{1}$$

$$\text{BaP-MEQ} = \text{B[a]P}_{\text{eq}} = \sum_{i}^{n=1} [\text{C}_i \times \text{MEF}_i] \tag{2}$$

where BaP-TEQ is the carcinogenic toxicity equivalent, BaP-MEQ is the mutagenic toxicity equivalent, and B[a]P_{eq} is the carcinogenic potency of a congener evaluated based on benzo[a]pyrene equivalent concentration. The B[a]P_{eq} has been used as an indicator of toxicity risks associated with exposure to PAHs (WHO 1987). TEFi is the toxic equivalent factors of the ith target PAH, MEFi is the mutagenic potency factors of the ith target compound, and Ci is the concentration of the ith target compound.

The B[a]P equivalency is derived from the multiplication of the concentrations and individual TEF values of each PAH. Equations (1) and (2) are further expanded as Eqs. (1.1) and (1.2), respectively (Bootdee et al. 2016; Pongpiachan et al. 2015). The abbreviations of the PAHs represent their concentrations in $PM_{2.5}$, as presented in Table 1.

$$\begin{aligned}\text{BaP-TEQ} = {} & 0.001(\text{Nap} + \text{Acy} + \text{Ace} + \text{Flu} + \text{Phe} \\ & + \text{Fla} + \text{Pyr}) + 0.01(\text{Ant} + \text{BPer} + \text{Chr}) \\ & + 0.1(\text{BaA} + \text{BbF} + \text{BkF} + \text{Ind}) \\ & + \text{BaP} + \text{DbA}\end{aligned} \tag{1.1}$$

$$\begin{aligned}\text{BaP-MEQ} = {} & 0.082(\text{BaA}) + 0.017(\text{Chr}) \\ & + 0.25(\text{BbF}) + 0.11(\text{BkF}) + 0.31(\text{Ind}) \\ & + 0.29(\text{DbA}) + 0.19(\text{BPer}) + \text{BaP}.\end{aligned} \tag{1.2}$$

The BaP-TEQ and BaP-MEQ values computed in this study were compared with 0.25 ng/m^3, 0.1 ng/m^3, and 1 ng/m^3 for the UK, Swedish, and European standards, respectively, since South Africa does not have a BaP-TEQ reference value for PAHs in PM (particulate matter) (Directive E.C. 2004).

Excess cancer and mutagenic risks (ECR) from inhalation of the 16 priority PAHs in $PM_{2.5}$ were computed from the product of BaP-TEQ and the inhalation unit risk ($UR_{B[a]P}$) using Eqs. (3) and (4).

These equations have been previously used in different studies (Bootdee et al. 2016; Wiriya et al. 2013).

$$\text{ECR-BaPTEQ} = \text{BaP - TEQ} \times \text{UR}_{\text{B[a]P}} \quad (3)$$

$$\text{ECR-BaPMEQ} = \text{BaP-MEQ} \times \text{UR}_{\text{B[a]P}}, \quad (4)$$

where BaP-TEQ and BaP-MEQ are the carcinogenic and mutagenic toxicity equivalent concentrations that were calculated from the product of each PAH component in $PM_{2.5}$ and its corresponding TEFs and MEFs as shown in Eq. (1.1); and $UR_{B[a]P}$ is the inhalation cancer unit risk of BaP which signifies the number of people who will likely contract cancer from inhalation of 1 ng/m^3 of B[a]P equivalent concentration within a lifetime of 70 years (Bandowe et al. 2014). The inhalation cancer unit risk of BaP was used because it depicts the overall health risks of PAHs (Jamhari et al. 2014). The WHO (2000) stipulates a URB[a]P value of 8.7×10^{-5}.

Exposure assessment and risk characterisation of PAHs

Human exposure to PAHs can occur through inhalation, ingestion, and dermal contact (Ma et al. 2017; Urbancok et al. 2017). In line with the US EPA guidelines (US EPA 2013), the incremental lifetime cancer risk (ILCR) of human exposure to carcinogenic PAH-bound (BaA, Chr, BkF, BbF, BaP, Ind, and DbA) $PM_{2.5}$ was estimated by the product of the lifetime average daily dose (LADD) and the cancer slope factor (CSF). The LADD is the intake quantity of a known pollutant with a potential to cause adverse health effects when absorbed into the body over a period of time (Jamhari et al. 2014). In this study, the LADD and the ILCR were computed for infants (0–1 year), children (2–5 years), children (6–12 years), and adults (19–75 years). The LADDs through the inhalation ($LADD_{inh}$), ingestion ($LADD_{ing}$), and dermal ($LADD_{derm}$) pathways were estimated using Eqs. (5–8) as follows:

$$\text{LADD}_{\text{inh}} = \frac{C * \text{InhR} * \text{ET} * \text{EF} * \text{ED} * \text{CF}}{\text{BW} * \text{AT}} \quad (5)$$

$$\text{LADD}_{\text{ing}} = \frac{C * \text{InhR} * \text{ET} * \text{EF} * \text{ED} * \text{CF}}{\text{BW} * \text{AT}} \quad (6)$$

$$\text{LADD}_{\text{derm}} = \frac{C * \text{SA} * \text{AF} * \text{ABS} * \text{ET} * \text{EF} * \text{ED} * \text{CF}}{\text{BW} * \text{AT}} \quad (7)$$

$$\text{ILCR} = \text{LADD} \times \text{CSF}, \quad (8)$$

where C is the concentration of PAHs in $PM_{2.5}$ (ng/m^3); ED is the exposure duration (days); BW is the body weight of the exposed group (kg); AT is the averaging time (days), ET is the exposure time (h/day); IngR is the ingestion rate (mg/day); InhR is the inhalation rate (m^3/day); SA is the surface area of the skin exposed to pollutants (cm^2); AF is the skin adherence factor (mg/cm^2/day); ABS is the dermal absorption factor; EF is the exposure frequency (days/year); CSF is the cancer slope factor (mg^{-1} kg day) (Table 1); and CF is the unit conversion factor ($C = 10^{-6}$). The values of these parameters are presented in Table 3.

Results and discussion

PAH concentration in $PM_{2.5}$

The mean concentrations of individual PAHs ranged from 0.07 to 0.92 ng/m^3 in winter and from 0.04 to 0.88 ng/m^3 in summer. The total PAHs (10.97 ng/m^3) recorded in the study area were lower than the total recorded in urban environments by Liu et al. (2015) for Guangzhou, China (33.89 ng/m^3), by Fang et al. (2006) for Taichung Harbor (56.12 ng/m^3), and by Zhou et al. (2005) for Beijing (116 ng/m^3). Nonetheless, the total PAHs recorded in this study were higher than those reported by Fraser et al. (2002) for Houston, USA (0.78 ng/m^3), by Khan et al. (2015) for Lumpur, Malaysia (2.79 ng/m^3), by Oliveira et al. (2014) for Rio de Janeiro, Brazil (3.80 ng/m^3), and by Li et al. (2010) for Mount Taishan, China (6.88 ng/m^3).

Although the total concentration of PAHs measured in this study was low, epidemiologic studies have associated exposure to long-term low-level PAHs with different health outcomes, including cancers (Liu et al. 2016; Tao et al. 2014).

The foremost PAH species in winter were determined as Phe (0.92 ng/m^3), Ace (0.91 ng/m^3), Fla (0.77 ng/m^3), and Acy (0.76 ng/m^3), while in summer, Phe (0.88 ng/m^3), Acy (0.65 ng/m^3), Flu (0.43 ng/m^3), and Fla (0.29 ng/m^3) were demonstrated

Table 3 Recommended values in equations of the daily exposure dose of $PM_{2.5}$

Parameter	Definition	Value for age categories				References
		Infant (0–1 year)	Child (2–5 years)	Child (6–12 years)	Adult (19–75 years)	
C	Mean concentration of $PM_{2.5}$ in ambient air ($\mu g/m^3$)					
IngR	Ingestion rate (mg/day)	60	60	60	30	US EPA (2007)
EF	Exposure frequency (days/year)	350	350	350	350	Morakinyo et al. (2017a), US EPA (1997)
ED	Exposure duration (years)	1	6	12	30	Matooane and Diab (2003), US EPA (1997)
ET	Exposure time (h)	1	8	6	3	Matooane and Diab (2003) US EPA (1997)
AT	Averaging time (days); AT = ED × 365 days	365	2190	4380	10,950	Matooane and Diab (2003), US EPA (1997)
BW	Body weight (kg)	11.3	22.6	45.3	71.8	Matooane and Diab (2003)
SA	Skin surface area (cm^2)	2800	2800	2800	5700	US EPA (2004)
AF	Adherence factor of soil to skin (mg/cm^2/event)	0.2	0.2	0.2	0.07	US EPA (2004)
ABS	Dermal absorption fraction	0.001	0.001	0.001	0.001	US EPA (2004)
InhR	Inhalation rate (m^3/day)	9.2	16.74	21.02	21.4	US EPA (1997)

Table 4 Average concentration of 16 priority polycyclic aromatic hydrocarbons in $PM_{2.5}$

PAHs	Winter					Summer				DL
	Mean (ng/m^3)	SD	Min	Max	DL	Mean (ng/m^3)	SD	Min	Max	
Acy	0.76	0.18	0.003	0.063	0.05	0.65	0.32	0.001	0.038	0.003
Ace	0.91	0.31	0.001	0.006	0.02	0.11	0.08	0.001	0.020	0.001
Ant	0.56	0.01	0.007	0.072	0.07	0.28	0.04	0.001	0.013	0.02
BaA	0.21	0.16	0.017	0.596	0.03	0.15	0.21	0.001	0.039	BDL
BaP	0.19	0.17	0.007	0.114	0.01	0.08	0.14	0.002	0.087	BDL
BbF	0.32	0.22	0.020	0.220	0.03	0.16	0.21	0.001	0.158	0.004
BkF	0.07	0.06	0.025	1.340	0.01	0.05	0.02	0.001	0.717	0.01
BPer	0.28	0.14	0.017	0.596	0.54	0.16	0.14	0.001	0.133	0.54
Chr	0.33	0.24	0.030	0.424	0.21	0.25	0.17	0.008	0.162	0.01
DbA	0.11	0.10	0.001	1.340	0.04	0.04	0.01	0.001	0.612	0.02
Fla	0.77	0.47	0.006	0.643	0.05	0.29	0.13	0.002	0.243	0.01
Flu	0.68	0.17	0.001	0.050	0.22	0.43	0.12	0.001	0.023	0.22
Ind	0.49	0.18	0.001	0.321	0.50	0.14	0.10	0.004	0.221	0.50
Nap	0.18	0.13	0.030	0.424	0.21	0.25	0.17	0.008	0.162	0.01
Phe	0.92	0.05	0.031	0.502	0.12	0.88	0.26	0.005	0.034	0.09
Pyr	0.39	0.25	0.056	1.345	0.16	0.09	0.10	0.015	0.322	0.11

SD standard deviation, *Min* minimum, *Max* maximum, *BDL* below detection limit

(Table 4). The 16 PAHs were clustered into lower molecular weight (two- and three-ringed PAHs—Acy, Flu, Phe, Ant), middle molecular weight (four-ringed PAHs), and higher molecular weight (five-, and six-ringed PAHs). The prime PAHs in $PM_{2.5}$ were the lower molecular weight compounds accounting for 59.0% of the total concentration compared with 21.9% and 19.1% for middle and higher molecular weight, respectively. This is consistent with findings reported for Guangzhou atmosphere where lower and middle molecular weights have the highest concentration in $PM_{2.5}$ (Liu et al. 2015). Lower and middle molecular weight PAHs are split between particulate matter and the gas phase since they are more volatile than high molecular weight PAHs. Moreover, the high lower molecular PAHs can be attributed to three sources: (i) coal combustion processes, (ii) unburned petroleum, and (iii) industrial emissions (Zhao et al. 2014). These findings are at variance with the studies of Chen et al. (2017) in which PAHs of higher molecular weights dominated the composition of $PM_{2.5}$.

The measured PAHs presented a seasonal variation with higher concentrations observed in winter than in summer. A similar pattern has been reported in other studies (Alves et al. 2017; Kang et al. 2017). Changes in meteorological conditions such as reduced precipitation, calm winds, poor dispersion conditions, reduced temperature, and boundary layer height and anthropogenic factors are arguments presented by researchers as the possible reasons for the increased PAHs observed in the winter season (Alves et al. 2017; Kang et al. 2017).

Bandowe et al. (2014) reported the role of ambient temperature in the gas-particle partitioning of PAH. An increase in ambient temperature facilitates the conversion of particle-phase PAH to the gas phase, whereas condensation of the gas-phase PAH into airborne particulates occurs when there is a decrease in atmospheric temperature. In summer, the breakdown or degradation of PAH from chemical and photochemical reactions in the presence of elevated temperatures and solar radiation explains the observed reduction in PAH concentrations (Callen et al. 2014).

Source of $PM_{2.5}$-bound PAHs

In this study, the values of Fla/(Fla + Pyr) and Ind/(Ind + Bper) in winter (0.7 vs 0.6) and summer (0.6 vs 0.5) were > 0.5, which signified grass, wood, and coal combustion sources. The ratio of BaA/(BaA + Chr) was 0.4 (combustion source) in winter and 0.2 (petroleum or combustion sources), while the ratio of Ant/(Ant + Phe) in winter (0.4) and summer (0.2) implied high-temperature (combustion) processes. The emission profile of the PAHs at the source is a reflection of the processes generating it (Mostert et al. 2010). These authors state that PAHs of lower molecular weight are produced in low-temperature processes, while PAHs of higher molecular weight are generated during high-temperature processes (Mostert et al. 2010).

Findings from this study are consistent with that of Ma et al. (2010), who reported variation in the emission rates and profiles of PAHs across seasons. Instances of higher values of Fla/(Fla + Pyr), BaA/(BaA + Chr), and Ant/(Ant + Phe) in winter than in summer have been reported (Dvorská et al. 2011). A plausible explanation for the reduction observed in summer was that faster photodegradation of PAHs occurs in summer. Tobiszewski and Namiesnik (2012) reported faster photodegradation of Ant, BaA, and Pyr than their isomers in summer. However, Yang et al. (2010) reported lower Ant/(Ant + Phe) and BaA/(BaA + Chr) values in winter than in summer. The strong effect of peripheral sources and ageing of air masses could explain the lower values recorded in the winter months.

Carcinogenic and mutagenic potential of PAHs

Both BaP-TEQ and BaP-MEQ presented a similar trend, with higher concentrations in winter (0.43 ng/m^3 vs 0.54 ng/m^3) than in summer (0.17 ng/m^3 vs 0.18 ng/m^3). The BaP-MEQ values were higher than the corresponding BaP-TEQ values. This finding is consistent with that of Bootdee et al. (2016) who reported higher values of BaP-TEQ and BaP-MEQ in winter than in summer. These values were lower than the value of 1 ng/m^3 recommended by the European Union (European Commission 2001) but higher than the values of 0.25 ng/m^3 and 0.1 ng/m^3 recommended by governments of the UK and Sweden, respectively (Directive E.C. 2004).

An assessment of the carcinogenic and mutagenic potential of PAHs in $PM_{2.5}$ showed values of 3.70×10^{-5} and 4.70×10^{-5} for ECR-BaPTEQ and ECR-BaPMEQ, respectively, in winter, and 1.49×10^{-5} and 1.57×10^{-5} for ECR-BaPTEQ

and ECR-BaPMEQ, respectively, in summer. Overall, the ECR-BaPTEQ and ECR-BaPMEQ values were below the priority risk level (10^{-4}), indicating no obvious cancer and mutagenic risks for the people in the study area (Sarkar and Khillare 2012).

Health risk assessment through inhalation, ingestion, and dermal pathways

The estimated LADD values of carcinogenic PAHs in $PM_{2.5}$ for specific age groups are presented in Table 5. In the winter and summer seasons, the values for daily inhalation and ingestion exposure to PAHs for all age groups were higher than the values for daily exposure through dermal contact. In addition, children and adults are more likely to inhale and ingest PAHs in $PM_{2.5}$ than infants.

The estimated ILCR values of carcinogenic PAHs for specific age groups are presented in Table 6. The cancer risk levels via the ingestion, inhalation, and dermal pathways ranged from 10^{-10} to 10^{-5}, 10^{-9} to 10^{-5}, and 10^{-11} to 10^{-7}, respectively. In health risk assessment, an ILCR of $< 10^{-6}$ represents a negligible cancer risk, while a value between 10^{-6} and 10^{-4} is defined as a potential cancer risk. An ILCR that exceeds 10^{-4} is a significant cancer risk (Wang et al. 2011).

In this study, the ILCRs for all PAHs across all age groups through the dermal route and also the ILCRs for BbF, BbK, and Chr through the inhalation and ingestion routes were lower than the acceptable limits. This indicated that the probabilistic cancer risk is very low. Akyüz and Çabuk (2008) reported that predominant carcinogenic PAHs such as BbF play an inconsequential role in the carcinogenic activity in an urban environment.

However, the findings of the current study demonstrated that for some children, toddlers, and adults, the upper-bound ILCRs (10^{-6}) from exposure to BaA, BaP, Ind, and DbA exceeded the acceptable level. This implies that there is a potential risk of 1–8 per million persons developing cancer from exposure to BaA, BaP, Ind, and DbA over a lifetime of 70 years. However, these values are lower than the level of one in ten thousand (10^{-4}) that is termed serious or of high potential by the US EPA (2001).

For all age groups, both the $ILCR_{inh}$ and the $ILCR_{ing}$ values were higher than the $ILCR_{derm}$ value, underscoring that the dermal risk of $PM_{2.5}$-bound PAH was negligible when compared with the inhalation and ingestion exposure pathways. The ILCR of exposure to $PM_{2.5}$-bound PAH was higher in winter than in summer. High concentrations of PAHs and the increased ADD of PAHs recorded in winter could be a possible reason for the seasonal difference of ILCR.

Also, the highest potential cancer risks were observed in adults and the lowest in infants. Previous studies have mentioned that adults tend to have higher cancer risks than infants (Sulong et al. 2017; Taner et al. 2013). Adults are believed to be more at risk than infants because of their greater inhalation rate over a specified period of time. It is believed that adults engage in more physically demanding activities that require a higher rate of inhalation than children (Hu et al. 2012). However, some researchers have reported higher lifetime risks for children than adults regarding exposure to ambient air pollutants (Morakinyo et al. 2017b; Thabethe et al. 2014). Moreover, the integrated carcinogenic risks through the exposure routes were lower than the 10^{-4} level that would constitute a high potential risk for the residents of the study area.

Conclusion

The seasonal concentrations of $PM_{2.5}$- and $PM_{2.5}$-bound PAHs were measured in an industrial area in Pretoria West, South Africa. Moreover, the sources of $PM_{2.5}$-bound PAHs and their potential carcinogenic and mutagenic risks were also determined. There was a variation in the emission sources and the profiles of the PAHs across seasons. The leading PAHs in $PM_{2.5}$ were the lower molecular weight compounds. The measured PAHs presented a seasonal variation, with higher concentrations observed in winter.

Exposure to PAHs in $PM_{2.5}$ through the dermal route was negligible when compared with the inhalation and ingestion exposure pathways. Also, there are no obvious cancer and mutagenic risks to the residents of the Pretoria West industrial area following the results obtained from the study area. Overall, the incremental cancer risk induced by all the sixteen PAHs in $PM_{2.5}$ was below the priority risk level and was therefore negligible. This signifies a low carcinogenic risk for the population residing in the study area. These findings can equip relevant stakeholders and policymakers with the knowledge of the concentration and risk of exposure to $PM_{2.5}$-bound PAHs and

Table 5 Lifetime average daily dose of $PM_{2.5}$-bound PAHs

PAHs	Season	$LADD_{Inh}$ (mg/kg/day)				$LADD_{Ing}$ (mg/kg/day)				$LADD_{Derm}$ (mg/kg/day)			
		Infant (0–1 year)	Child (2–5 years)	Child (6–12 years)	Adult (19–75 years)	Infant (0–1 year)	Child (2–5 years)	Child (6–12 years)	Adult (19–75 years)	Infant (0–1 year)	Child (2–5 years)	Child (6–12 years)	Adult (19–75 years)
BaA	Winter	2.65E−08	2.90E−07	7.26E−07	1.85E−06	1.73E−07	1.04E−06	2.07E−06	2.59E−07	1.61E−09	9.67E−09	1.93E−08	3.44E−08
	Summer	8.82E−09	9.63E−08	2.42E−08	6.16E−07	5.75E−08	3.45E−07	6.90E−07	8.63E−07	5.37E−10	3.22E−09	6.44E−09	1.15E−08
BaP	Winter	2.40E−08	2.61E−07	6.57E−07	1.67E−06	1.56E−07	9.37E−07	1.87E−06	2.34E−06	1.46E−09	8.75E−09	1.75E−08	3.12E−08
	Summer	1.01E−08	1.10E−07	2.76E−07	7.04E−07	6.58E−08	3.95E−07	7.89E−07	9.86E−07	6.14E−10	3.68E−09	7.36E−09	1.31E−08
BbF	Winter	4.03E−08	4.40E−07	1.11E−06	2.81E−06	2.63E−07	1.58E−06	3.16E−06	3.95E−06	2.46E−09	1.47E−08	2.95E−08	5.25E−08
	Summer	2.02E−08	2.20E−07	5.53E−07	1.41E−06	1.32E−07	7.89E−07	1.58E−06	1.97E−06	1.23E−09	7.36E−09	1.47E−08	2.62E−08
BkF	Winter	8.82E−09	9.63E−08	2.42E−08	6.16E−07	5.75E−08	3.45E−07	6.90E−07	8.63E−07	5.37E−10	3.22E−09	6.44E−09	1.15E−08
	Summer	6.30E−09	6.88E−08	1.73E−07	4.40E−07	4.11E−08	2.47E−07	4.93E−07	6.16E−07	3.84E−10	2.30E−09	4.60E−09	8.20E−09
Chr	Winter	4.16E−08	4.54E−07	1.14E−06	2.90E−06	2.71E−07	1.63E−06	3.26E−06	4.07E−06	2.53E−09	1.52E−08	3.04E−08	5.41E−08
	Summer	3.15E−08	3.44E−07	8.64E−07	2.20E−06	2.06E−07	1.23E−06	2.47E−06	3.08E−06	1.92E−09	1.15E−08	2.30E−08	4.10E−08
Ind	Winter	6.18E−08	6.74E−07	1.69E−06	4.31E−06	4.03E−07	2.42E−06	4.83E−06	6.04E−06	3.76E−09	2.26E−08	4.51E−08	8.04E−08
	Summer	1.76E−08	9.26E−07	4.84E−07	1.23E−06	1.15E−07	6.90E−07	1.38E−06	1.73E−06	1.07E−09	6.44E−09	1.29E−08	2.30E−08
DbA	Winter	1.39E−08	1.51E−07	3.80E−07	9.67E−07	9.04E−08	5.42E−07	1.09E−06	1.36E−06	8.44E−10	5.06E−09	1.01E−08	1.80E−08
	Summer	5.04E−09	5.50E−08	1.38E−07	3.52E−07	3.29E−08	1.97E−07	3.93E−07	4.93E−07	3.07E−10	1.84E−09	3.68E−09	6.56E−09

Table 6 Lifetime cancer risk of $PM_{2.5}$-bound PAHs

PAHs	Season	$ILCR_{Inh}$				$ILCR_{Ing}$				$ILCR_{Ing}$			
		Infant (0–1 year)	Child (2–5 years)	Child (6–12 years)	Adult (19–75 years)	Infant (0–1 year)	Child (2–5 years)	Child (6–12 years)	Adult (19–75 years)	Infant (0–1 year)	Child (2–5 years)	Child (6–12 years)	Adult (19–75 years)
BaA	Winter	1.62E−08	1.77E−09	4.43E−07	**1.13E−06**	1.06E−07	6.34E−07	**1.26E−06**	1.58E−07	9.82E−10	5.90E−10	1.18E−08	2.10E−08
	Summer	5.38E−09	5.87E−08	1.48E−08	3.76E−07	3.51E−08	4.21E−07	5.26E−07	5.26E−07	3.28E−10	1.96E−09	3.93E−09	7.02E−09
BaP	Winter	1.46E−07	**1.59E−06**	**4.01E−06**	**1.02E−05**	9.52E−07	**5.72E−06**	**1.14E−05**	**1.43E−05**	8.91E−09	5.34E−08	1.07E−07	1.90E−07
	Summer	6.16E−08	6.71E−07	**1.68E−06**	**4.29E−06**	4.01E−07	**2.41E−06**	**4.81E−06**	**6.02E−06**	3.75E−09	2.25E−08	4.49E−08	7.99E−08
BbF	Winter	2.46E−09	2.68E−08	6.77E−08	1.71E−07	1.60E−08	9.64E−08	1.93E−07	2.41E−07	1.50E−10	8.97E−10	1.80E−09	3.20E−09
	Summer	1.23E−09	1.34E−08	3.37E−08	8.60E−08	8.05E−09	4.81E−08	9.64E−08	1.20E−07	7.50E−11	4.49E−10	8.97E−10	1.60E−09
BkF	Winter	5.38E−10	5.87E−09	1.48E−09	3.76E−07	3.51E−08	4.21E−07	5.26E−07	5.26E−07	3.28E−10	1.96E−09	3.93E−09	7.02E−09
	Summer	3.84E−10	4.20E−09	1.06E−08	2.68E−08	2.51E−09	2.57E−08	3.01E−08	3.76E−08	2.34E−11	2.81E−10	2.81E−10	5.00E−10
Chr	Winter	2.54E−10	4.11E−09	6.95E−09	1.77E−08	1.65E−09	9.94E−09	1.99E−08	2.48E−08	1.54E−11	9.27E−11	1.85E−10	3.30E−10
	Summer	1.92E−10	2.10E−09	5.27E−09	1.34E−08	1.26E−09	7.50E−09	1.51E−08	1.88E−08	1.17E−11	7.02E−11	1.40E−10	2.50E−10
Ind	Winter	3.77E−08	4.11E−07	**1.03E−06**	**2.63E−06**	2.46E−07	**1.48E−06**	**2.95E−06**	**3.68E−06**	2.29E−09	2.75E−08	2.75E−08	4.90E−08
	Summer	1.07E−08	5.65E−07	2.95E−07	7.50E−07	7.02E−08	4.21E−07	8.42E−07	**1.06E−06**	6.53E−10	3.93E−09	7.87E−09	1.40E−08
DbA	Winter	8.48E−08	9.21E−07	**2.32E−06**	**5.90E−06**	5.51E−07	**3.31E−06**	**6.65E−06**	**8.30E−06**	5.15E−09	3.09E−08	6.16E−08	1.10E−07
	Summer	3.07E−08	3.36E−07	8.42E−07	**2.15E−06**	2.01E−07	**1.20E−06**	**2.40E−06**	**3.01E−06**	1.87E−09	1.12E−08	2.25E−08	4.00E−08
∑7PAHS		3.98E−07	4.61E−06	1.08E−05	2.81E−05	1.13E−06	1.62E−05	3.17E−05	3.80E−05	2.45E−08	1.56E−07	2.94E−07	5.24E−07

The values in bold depict that the upper-bound acceptable level (10^{-6}) of the $PM_{2.5}$-bound PAH through the inhalation and ingestion pathways exceeded

therefore institute strategies and plans for further emissions control.

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