# Levels, potential sources and human health risk of polycyclic aromatic hydrocarbons (PAHs) in particulate matter ($PM_{10}$) in Kumasi–Ghana

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**Abstract**

Airborne particulate samples were collected on quartz filters to determine the concentrations, sources and health risks of polycyclic aromatic hydrocarbons (PAHs) in air in Kumasi, Ghana. A total of thirty two air samples were collected in Kwame Nkrumah University of Science and Technology campus (KNUST) (pristine site) and city centre (CC). Samples were extracted with 1:2 v/v acetone:hexane mixture prior to GC–MS analyses. The sum of concentrations of 17 PAHs in air ranged from 0.51–16 (KNUST) and 19–38 ng/m$^3$ (CC). The concentration of Benzo[a]Pyrene, BaP, ranged from below detection limit to 0.08 ng/m$^3$ (KNUST) and 1.6 to 5.6 ng/m$^3$ (CC). Chemical mass balance model showed that PAHs in air in Kumasi were mainly from fuel combustion. The total BaP toxic equivalent concentration (BaPeq) in CC was 18 times higher compared to KNUST; based on the European Legislation, Swedish and United Kingdom Standards for BaP in air, CC could be classified as highly polluted. Estimated carcinogenicity of PAHs in terms of BaPeq indicated that BaP was the principal PAHs contributor in CC (70%). Health risk of adults and children associated to PAHs inhalation was assessed by taking into account the lifetime average daily dose and corresponding incremental lifetime cancer risk (ILCR). The ILCR was within the acceptable range ($10^{-6}$ to $10^{-4}$) indicating low health risk to residents.

**Keywords** Airborne particulate; PAHs; Kumasi; BaP toxic equivalent; Incremental lifetime cancer risk; Chemical mass balance

## Introduction

Polycyclic aromatic hydrocarbons (PAHs) are compounds whose structure consist of two or more fused benzene rings in linear, angular or cluster arrangements. PAHs are ubiquitous environmental pollutants affecting the air, soil, stream, sediment, water and food. They are found naturally in coal, crude oil and in emissions from forest fires and volcanoes. Nevertheless, most PAHs entering the environment are formed unintentionally during burning of fossil fuel, biomass, wood etc. (Manoli et al. 2000).

Exposure to PAHs takes place mainly by inhalation of contaminated air or ingestion of soil, food and drinking contaminated water (Barranco et al. 2004; Dissanayake et al. 2004). PAH exposure to particular work or areas has been explored. These studies have provided many valuable insights on the potential threat of PAHs to human health. Cases include on–duty traffic policemen (Ruchirawat et al. 2002; Liu et al. 2007), incense smoke in vehicle (Kuo et al. 2003), fixed site with heavy traffic (Ho and Lee 2002), urban site, vegetation area, forest area (Vasconcellos et al. 2003), bus station and traffic tunnel (Pereira et al. 2002), outdoor air (Velasco et al. 2004), roadside air (Chetwittayachan et al. 2002; Marr et al. 2004), and ambient traffic site (Lodovici et al. 2003).

Due to their ubiquitous occurrence, persistence, suspected carcinogenicity and mutagenicity, PAHs are included in the US Environmental Protection Agency (EPA), Environmental Monitoring Assessment and the European Union priority lists of pollutants. The US EPA fixed 16 parent PAHs as priority pollutants (Mastral and Callén 2000; Magi et al. 2002; Szolar et al. 2002; Schubert et al. 2003).

As one of the most industrialized and economically significant cities in Ghana, Kumasi has been subjected to heavy anthropogenic impacts due to rapid economic development and urbanization. The population and number of vehicles has drastically increased, during the past decade and many fuel filling stations are located in this region leading to greater fuel combustion rate. All of these have led to fuel leakages, smoke production from exhaust of automobiles and as a result high levels of PAHs are released into the environment. Other pollution sources in the Kumasi metropolis, Ghana, are the garbage, wood, and paper burning (Bortey–Sam et al. 2014). According to available literature, no studies on PAHs have been made before in this city.

The objectives of this study were: i) to determine the concentrations of 21 PAHs in particulate matter ($PM_{10}$) in the Kumasi metropolis; ii) to identify the possible sources of these PAHs; and iii) to determine the toxic potential and possible health risk implications associated with them.

## Materials and methods

*Study area and sampling*

Two sampling sites were selected for this study. They were located at Kwame Nkrumah University of Science and Technology campus, KNUST, (pristine site) and city centre, CC, (which is densely populated and host a number of vehicles, fuel stations and small scale industries). Airborne particulate samples ($PM_{10}$) were collected on quartz filters using a Sibata Low Volume Pump air sampler (SL–30; Shibata; Saitama, Japan). The sampling sites were selected using Global Positioning System (GPS). The GPS coordinates at KNUST site were N06$^{o}$40'24.7", W001$^{o}$34'04.8" and that of CC were N06$^{o}$41'51.6", W001$^{o}$37’23.6’’. A total of 20 samples were collected in KNUST (5 samples were collected each month) whilst 12 samples were collected in CC (3 samples were collected each month).

Air sampling was carried out for 8 h each day at a flow rate of 30 L/min. Thirty two air samples were collected in total from June to September, 2011. The air inlet was located 1.5 m above ground level to simulate the breathing zone. Samples/filters were wrapped in aluminum foil, double packed in zip–lock bags and stored at –20 $^{0}$C in the Department of Chemistry, KNUST, Ghana. Finally, samples were transported to the Laboratory of Toxicology, Hokkaido University, Japan where they stored at –30 $^{0}$C prior to analysis.

From June to September, the northern and central regions of Ghana receive 150–250 mm of rain per month; this period is the coldest and wettest in the year (McSweeney et al. 2013). Variations in temperature both annually and daily are quite small and the minimum temperature is around 23 $^{0}$C (73 $^{0}$F).

*Sample extraction and clean–up*

Extraction of PAHs in air samples (quartz filters) was done by soxhlet extractor (Soxtherm Gerhardt Variostat; Soxtherm V7.5, Germany) using 170 mL acetone/hexane (1:2; v/v) for 6 h after spiking with 25 µL of 1 mg/L PAH internal standard (acenaphthene_$d_{10}$, phenanthrene_$d_{10}$, chrysene_$d_{12}$, and perylene_$d_{12}$). The extracts were dehydrated by filtering through anhydrous sodium sulfate (Kanto Chemical Co., Inc, Tokyo, Japan). The dehydrated extracts were collected into a round bottom flask. It was then concentrated to approximately 1.5 mL on a rotary evaporator and transferred into a test tube. The dehydrated extract in the test tube was further concentrated to 0.3 mL under a gentle $N_2$ stream.

The extracts were cleaned–up to remove other contaminants that may have been extracted with PAHs. Clean–up was done using column packed with 5% water content silica gel (Kanto Chemical Co., Inc, Tokyo, Japan). It was then eluted with 100 mL of diethyl ether/hexane mixture in the ratio 1:4, v/v. The eluate was further concentrated to 2 mL using a rotary evaporator. 300 µL of n–decane was added to the extract and was further concentrated to 0.3 mL using a gentle $N_2$ stream. All solvents used (Kanto Chemical Co., Inc, Tokyo, Japan) were of analytical grade.

*PAHs analysis*

PAHs analyses were carried out using Auto Sampler 3000 Gas Chromatograph (Focus GC) coupled with a Thermo scientific Mass Selective Detector (DSQ II MS, Kanagawa, Japan) operating in the electron impact mode (GC–MS). The selective ion monitoring mode was used for quantification. A FactorFour capillary column, VF–Xms, 30 m, 0.25 mm inner diameter, 0.25

126 μm film thickness (Varian Inc., Lake Forest, CA, USA) was used for separation. Helium gas was
127 used as the carrier gas at a constant flow rate of 1.2 mL/min. Injector and mass transfer line
128 temperatures were 260 $^{o}$C and 280 $^{o}$C, respectively. Temperature programming for the column
129 were as follows, initial temperature of 90 $^{o}$C held for 1 min, ramped to 280 $^{o}$C at 10 $^{o}$C/min and
130 finally to 320 $^{o}$C at a ramp rate of 5 $^{o}$C/min and held for 10 min. 1 μL sample was injected in the
131 splitless mode for analysis.
132 Concentrations of 21 individual PAHs (AccuStandard, New Haven, USA) including 16
133 USEPA priority pollutants (naphthalene (Nap), acenaphthylene (Acl) acenaphthene (Ace),
134 fluorene (Fle), phenanthrene (Phe), anthracene (Ant), fluoranthene (Flu), pyrene (Pyr),
135 benz[a]anthracene (BaA), chrysene (Chr), benzo[b]fluoranthene (BbF), benzo[k]fluoranthene
136 (BkF), benzo[a]pyrene (BaP), indeno[1,2,3–cd]pyrene (IDP), dibenz[a,h]anthracene (DBahA),
137 benzo[g,h,i]perylene (BghiP), perylene (Peryl), benzo[e]pyrene (BeP), methylene phenanthrene
138 (Methy–Phe), 1–methyl phenanthrene (Me–Phe) and retene (Ret)) were measured in each sample.
139 Difficulties were often associated with the GC separation of BbF and BkF and since
140 benzo[j]fluoranthene (BjF) co–eluted with BbF (Pietrogrande et al. 2014), the sum of these
141 isomers was used as an abbreviation, BbF + BjF + BkF. All results were expressed in ng/m$^3$.
142
143 *Quality control and quality assurance*
144 Quantitation was performed using internal standard calibration method (five–point calibration),
145 the correlation coefficients ($r^2$) for the calibration curves were all greater than 0.995. Analytical
146 methods were checked for precision and accuracy. Limits of detection (LODs) were calculated
147 based on 3SD/S (SD is the standard deviation of the response of seven replicate standard solution

148 measurements and S is the slope of the calibration graph). LODs of PAHs were in the range of
149 0.02–0.67 ng/m$^3$.

150 Prior to extraction, four internal standards (acenaphthene_$d_{10}$, phenanthrene_$d_{10}$,
151 chrysene_$d_{12}$, and perylene_$d_{12}$) were added to air samples for recovery assessment. Recoveries
152 in spiked air samples were as follows, acenaphthene_d10, 90 ± 1.6%, phenanthrene_d10, 89 ±
153 2.1%, chrysene_d12, 93 ± 0.88% and perylene_d12, 91 ± 1.0%. The final PAH concentrations
154 were not corrected from the recoveries of the internal standards.

155 For each batch of 6 samples, a method blank (solvent), a spiked blank (internal standards
156 spiked into solvent), and a matrix spike (internal standards spiked into pre–extracted air sample)
157 were analyzed. The average recoveries in spiked blanks and matrix spikes varied from 85–102%
158 for PAHs (21 components). Blanks that were run periodically contained no detectable amount of
159 target analyte. The coefficients of variation of PAHs concentration in duplicate samples were
160 less than 15%.

161

162 *Health Risk Characterization*

163 Health risk estimation can be calculated using PAH exposure through one of the following
164 exposure pathways: ingestion, inhalation, or dermal exposure (USEPA 2005). In this study,
165 inhalation of air particles contaminated with PAHs was considered. Health risk assessment of
166 carcinogenic PAHs cannot be related to the sole total concentration, rather each PAH has a
167 different carcinogenic potential. For this purpose, toxicity equivalency factors (TEFs) are used,
168 which allow to quantify the carcinogenic potential of other PAHs, relative to BaP, and estimate
169 BaP–equivalent concentration (BaPeq) (Nadal et al. 2004). Nonetheless, usually the health risk
170 associated with inhalatory PAH uptake is estimated on the basis of the sole BaP, which was the

principal contributor in every studied site (Halek et al. 2008). In our study, the list of TEFs compiled by Tsai et al. (2004) was adopted (see Table 3), and the total PAH–associated carcinogenicity was calculated through the formula:

$$BaPeq = \sum(C \times TEF)$$

The Increamental Lifetime Cancer Risk, ILCR, in humans can be determined by calculating the Lifetime average daily dose (LADD) of PAHs according to the USEPA guidelines (USEPA, 2013). The equation for estimating LADD and ILCR were as follows:

$$\text{LADD (mg/kg/d)} = \frac{Cs \times IR \times ET \times EF \times ED}{BW \times AT} \times CF$$

Cancer risk (ILCR) = LADD × cancer slope factor (CSF)

Where LADD is the amount of intake per kg of body weight per day of a chemical suspected of having adverse health effects when absorbed into the body over a long period of time. Cs represents the average concentration of particular PAH (ng/$m^3$); IR is the intake rate ($IR_A$ = 0.83 $m^3$/h for adults and $IR_c$ = 0.5 $m^3$/h for children up to the age of six); ET is the exposure time (21 hrs/day), EF is the exposure frequency (350 days/year), ED represents the exposure duration, the value of which is $ED_A$ = 70 years (adults) and $ED_C$ = 6 years (children); CF is the unit conversion factor (CF = $10^{-6}$). BW is the average body weight (it was assumed that $BW_A$ = 70 kg for adults and $BW_C$ = 15 kg for children), AT is the average timing, which was $AT_A$ = 25550 days (70 × 365) for adults and $AT_C$ = 2190 days (6 × 365) for children (Bozek et al. 2009). The USEPA (1992) and WDNR (1997) CSF values for carcinogenic PAHs were adopted for this study (Table 4). Estimated LADD values of the measured PAHs for human adults and children were calculated and listed in Table 4, and the ILCR values for (7 carcinogenic PAHs by IARC

192 2006) human adults and children were also listed alongside. It was assumed that the
193 concentration of pollutant would remain approximately constant in that period of time.

194 *Data analysis*

195 Data were analysed using SPSS version 20.0 for windows (IBM SPSS Statistics., 2011). The
196 PAHs concentrations in KNUST and CC were tested for normality and distribution was
197 considered statistically significant if *p* value was less than 0.05. Statistical difference of PAHs
198 between the two sample sites was tested using ANOVA. Results were considered significant
199 with *p* values less than 0.05. Principal component analysis (PCA), was done to determine the
200 distribution pattern of PAHs in air (except for Nap; because concentrations were not detected in
201 most samples from both sites), and was performed using JMP statistical software v. 10 (SAS
202 Institute). The principal components were extracted with eigenvalues > 1 through varimax
203 rotation.

204 Chemical mass balance version 8 (CMB8) was applied for source identification from both
205 sample sites and source profiles/input were classified as either coal related (power plant,
206 residential or coke oven) or traffic related (gasoline engine, diesel engine or traffic tunnel) (Li et
207 al. 2003). Study by Bortey–Sam et al. (2014) indicated that coke ovens and traffic were the two
208 major sources of PAHs in the area and these have also been considered to be the two most
209 important source categories in many metropolitan areas in the 20th century (Li et al. 2003).

210 The basic idea of the CMB model is that the measured chemical pollutants in a sample are the
211 sums of the contributions from several sources (Li et al. 2001; Wang et al. 2010). However, only
212 relative compound concentrations in a source or a sample profile (e.g., normalized with BeP, or
213 any other PAH compound) are important (Christensen et al. 1999). The inputs required by the
214 EPA CMB8 include the ambient data and source profiles. In this study, the “ambient” data are

the experimentally measured concentrations, in ng/m$^3$, of PAHs in $PM_{10}$. The key to a successful CMB application is to obtain a set of source fingerprints which is consistent with the measurements at the receptor location. For the purpose of this work, 11 and 17 published PAH source profiles were collected from literature for coal and traffic related sources respectively (Li et al. 2003).

Nap was not included in the modeling, because of the high uncertainties in its source fingerprints and possible evaporative losses during chemical analysis of the samples. Due to the lack of sufficient data for DBahA in many source profiles, this compound was not selected as fitting species in most model runs (Li et al. 2003). Similarly, Methy–Phe, Me–Phe, Ret, and Peryl were not included. The CMB8 model results were evaluated by using several fit indices such as coefficient of determination, $R^2$ ($\geq 0.85$), chi–square distribution, $\chi^2$ ($\leq 3.0$), percent mass accounted for (72–95%), T-statistics ($\geq 3.0$), and the residuals and uncertainty ratios (–1.8 to 4.2). The values in parentheses were the statistical parameters obtained from the modelling results.

## Results and discussion

*PAHs in ambient air in KNUST and CC*

From the 21 PAHs analyzed, the more volatile compounds (Nap, Acl, Ace and Fle) were not discussed at each sampling location because these lower ring molecules are present in the gas phase, especially at high ambient temperatures (20–38 $^{0}$C), which was observed throughout the sampling period. Moreover, their (Nap, Acl, Ace and Fle) concentrations were either undetected or low, which was also observed in studies by Oanh et al. (2000) and Salam et al. (2011).

The sum of concentrations of 17 PAHs ($\Sigma_{17}$ PAHs) in air samples ($PM_{10}$) in KNUST ranged from 0.51–16 ng/m$^3$ with mean and median values of 3.3 ± 3.3 and 2.6 ng/m$^3$ respectively. Meanwhile, $\Sigma_{17}$ PAHs in air samples in CC ranged from 19–38 ng/m$^3$ with mean and median concentrations of 30 ± 4.9 and 30 ng/m$^3$ respectively. The mean concentration of $\Sigma_{17}$ PAHs in CC was 9 times greater than in KNUST. This may be attributed to the fact that the sampling in Kejetia was performed in a traffic area suffering high exhaust emission and oil seep from vehicles, and hosting handicraft and small industrial activities (e.g., corn milling).

$PM_{10}$–bound PAH concentrations were compared with recent measurements in other urban areas around the world, including Turkey, Spain and the comparative information is illustrated in Table 1. The concentrations observed in CC was comparable with that of cities in Thailand (Wiriya et al. 2013) (Table 1), but they were lower than those detected in Zonguldak, Turkey (Akyuz and Cabuk 2009) (Table 1). However, Callen et al. (2008) and Pengchai et al. (2009) reported low PAHs concentrations in air ($PM_{10}$) in Zaragoza (8.8 ± 7.9 ng/m$^3$) and Thailand (0.05–22 ng/m$^3$), respectively (Table 1).

*Most abundant PAHs in air in KNUST and CC*

The most abundant PAHs in air ($PM_{10}$) in KNUST were Phe (21%), BghiP (16%), Ret (15%), and Pyr (11%) (Table 2). Chr was the least abundant with concentration below detection limit in almost all samples (Table 2). Ret (one of the most abundant PAHs in air in KNUST) is a molecular marker of wood combustion (Shen et al. 2012). PAHs profile developed by Harrison et al. (1996) and Ho et al. (2002b) on the types of emission sources suggested that Phe and Pyr in KNUST were typical diesel vehicle markers whereas BghiP, the $2^{nd}$ most abundant, was indicative of gasoline vehicle emission.

As shown in Table 2, air samples in KNUST was dominated by 3–(44%), 6–(24%), 4–(21%), and 5–ring PAHs (11%). Because only PAHs in the particulate phase were collected, it is expected that some of these 3–ring PAHs will also be in the gas phase and concentrations could be underestimated, especially in KNUST, where 3–ring PAHs showed high contribution. During combustion at low temperatures, the low molecular weight (LMW) PAH compounds (< 4 rings) are abundant (Lake et al. 1979). Whilst at high temperature combustion the high molecular weight (HMW) PAH compounds (≥ 4 rings) are dominant (Laflamme and Hites 1978). According to that, ~55% of PAHs in KNUST were released by high temperature combustion sources.

The most abundant PAHs in air in CC were BghiP, IDP, BeP and BaP (HMW PAHs) (Table 2, $p < 0.001$). Their abundances were 29%, 19%, 14%, 12% respectively (Table 2). Me–Phe were the least abundant PAHs there ($0.01 \pm 0.04$ ng/m$^3$). This pathway has been reported as indicative of vehicle emission (Harrison et al. 1996; Oanh et al. 2000; Jamhari et al. 2014).

Moreover, in CC the PAHs profile was dominated by 6–(48%) ($p < 0.001$), 5–(42%) ($p < 0.001$), 3–(5%) ($p > 0.001$), and 4–ring PAHs (4.5%) ($p < 0.001$) (Table 2). The high percentage

277 of HMW PAHs (95%) confirmed high temperature processes, such as combustion of fuels in
278 engines, as prevailing sources (Mostert et al. 2010; Tobiszewski and Namiesnik 2012).

279 PCA was applied to PAHs profiles in KNUST and CC. The PCA revealed that the first
280 principal component (component 1) accounted for 47% of the variation while component 2
281 accounted for 19% (Fig. 1). As observed from the score plot (Fig. 1a), there was a clear
282 separation between the two sampling sites (K and C). That could depend on the high levels of
283 PAHs in CC compared to KNUST. From the loading plot (Fig. 1b), most of the PAHs (both
284 LMW and HMW) were highly associated/clustered on one side of the component. Another
285 interesting feature observed along the component (Fig. 1) was the clear separation between
286 LMW and HMW PAHs, (Fig. 1b) with the HMW PAHs highly distributed in the CC of Kumasi
287 (Fig. 1).

288

289 *Levels of BaP in air in KNUST and CC*

290 Concentrations of BaP in air in KNUST ranged from below detection (from most sampling
291 dates) to 0.08 ng/m$^3$. The mean and median concentrations of BaP in KNUST were 0.02 ± 0.03
292 and 0.01 ng/m$^3$ respectively (Table 3). Since no environmental standard is available for Ghana,
293 the United Kingdom (UK), Swedish Standards and European Legislation were adopted to assess
294 the quality of air. The UK air quality standards, Swedish guideline and European Legislation,
295 values for BaP are equal 0.25, 0.1 and 1 ng/m$^3$ (for the total content in the $PM_{10}$ fraction
296 averaged over a calendar year), respectively (Dimashki et al. 2001; Bostrom et al. 2002;
297 Directive 2004/107/EC).

The mean concentration of BaP in air in KNUST (0.02 ± 0.03 ng/m$^3$) was 5, 12, and 50 times below the BaP standard set by the Swedish, UK, and European Legislation respectively. Similarly, individual concentrations of BaP in air in KNUST were all below the recommended guideline values. Low concentrations of BaP and $\Sigma_{17}$ PAHs were recorded in air in KNUST because the sampling site was located within an area with low vehicular movement, low industrial and human activities and therefore PAHs from point sources were negligible.

BaP was the fourth most abundant PAH in air in CC ($p < 0.001$) and concentration ranged from 1.6–5.6 ng/m$^3$. The mean concentration of BaP in CC (3.7 ± 1.1 ng/m$^3$) was 187 times higher than in KNUST (0.02 ± 0.03 ng/m$^3$) and exceeded 5 times that found in Taichung Industrial Park, TIP, Taiwan (particles–bound) (0.7 ng/m$^3$; Guor–Cheng et al. 2004). Similarly, the mean concentrations of BaP in CC was higher than Zaragoza, Spain (Callen et al. 2013) and in Amritsar, India (Kaur et al. 2013). On the other hand, the BaP values were comparable with those of Naples, Italy (2.9 ng/m$^3$) and Seoul, Korea (2.6 ± 3.3 ng/m$^3$) (Caricchia et al. 1999; Park et al. 2002).

Anyway, the BaP concentrations in CC were 37, 15, and 4 times higher, respectively, than the air quality standards of Sweden, UK, and European Legislation. Therefore, the CC air was classified as heavily polluted.

Determination of sources of PAHs in air

*Chemical mass balance (CMB) model*

In general, the contributions appeared similar in both cases. Fuel contributions from diesel and gasoline engines were higher from both sites. From Fig. 2, 15.3% of PAHs in KNUST was contribution from coal related sources i.e. coke oven and 84.7% from traffic related sources (Fig.

2). Of this 84.7%, 42.5% were emissions from diesel engines and the other 42.2% from roadway traffic (Fig. 2). Similarly 85% of PAHs in CC were from traffic related sources out of which 78.4% was contribution from gasoline engines and 6.3% from roadway traffic (Fig. 3).

The overall CMB results clearly showed that gasoline and diesel engines were the two major sources of PAHs in air samples ($PM_{10}$) in the Kumasi metropolis.

*Air toxicity assessment (CC)*

In Kumasi (CC), PAHs including human carcinogenic compounds (BaA, BbF, BkF, BaP, Chr, DBahA, and IDP) (IARC, 2006) were at high concentrations (Tables 2 and 3). The mean concentration of these compounds were IDP (5.8 ± 1.2), BaP (3.7 ± 1.1), BbF + BjF + BkF (3.2 ± 0.98), DBahA (0.5 ±0.16) BaA (0.29 ± 0.08) and Chr (0.15 ± 0.06) ng/$m^3$ (Table 3).

WHO (2000) stated that the BaP equivalent concentration producing an excess lifetime cancer risk of 1/10,000 is approximately 1.2 ng/$m^3$. The mean concentrations of BaP alone in CC (3.7 ± 1.1 ng/$m^3$) exceeded this threshold. The toxic equivalent concentration (BaPeq) in air of carcinogenic PAHs was 0.3 ngBaPeq/$m^3$ in KNUST and 5.2 ngBaPeq/$m^3$ in CC (Table 3). Thus BaPeqs in CC were, on the average, 18 times higher than in KNUST.

According to BaPeqs of individual PAHs, BaP was the predominant contributor to toxicity in CC. In comparison of the BaPeq's of PAHs in air with other conducted studies in $PM_{10}$, the BaPeq values of 16 USEPA PAHs in CC (5.3 ng/$m^3$; Table 3) was found to be higher than similar work done in Christchurch, New Zealand (1.4 ng/$m^3$; city) (Brown et al. 2005; assigned same TEF values as this study except for DBahA, which was assigned a value of 5); Dunedin, New Zealand (0.006 ng/$m^3$; city) (Brown et al. 2005); Taiwan (3.7 ng/$m^3$; urban), (Chang et al. 2006); Chiang Mai, Thailand (sub–urban, 0.18–3.7) (Wiriya et al. 2013). The BaPeqs from this

344 study were however lower than similar study from traffic site (particle phase; 13 ng/m$^3$) in Japan
345 (Chang et al. 2006).
346 To assess the health risk for humans, adults and children were assumed to be exposed to
347 airborne PAHs through inhaling $PM_{10}$. The estimates were made by considering LADD and the
348 corresponding ILCR.
349 The LADD PAH values ranged from 2.5E–9 to 2.1E–6 for adults (average 4.4E–7) and 6.9E–
350 8 to 5.8E–6 for children (average 1.3E–6). According to that, ILCR associated to carcinogenic
351 PAHs (IARC 2006) ranged from 2.2E–10 to 5.4E–6 (adults) and 6.1E–10 to 1.5E–5 (children).
352 The mean ILCR for adults (1.2E–6) and children (3.3E–6) were within the acceptable limit of
353 $10^{-6}$ to $10^{-4}$ as acknowledged by regulatory agencies (USEPA 2005). The acceptable risk
354 distribution is expressed as a constraints–based percentile and must be $\leq 10^{-6}$ to $10^{-4}$ using
355 upper–bound factors, which represents the increase of cancer by one in the group of million of
356 people (USEPA 2005).

357

358 **Conclusions**

359 BaP concentrations in air samples ($PM_{10}$) in KNUST varied from below detection to 0.08 ng/m$^3$
360 and 1.69 to 5.66 ng/m$^3$ in CC. The mean concentrations of BaP in air was 0.02 ± 0.03 (KNUST)
361 and 3.7 ± 1.1 ng/m$^3$ (CC) respectively. By the Swedish, UK air quality standards and European
362 Legislation value for BaP in air, CC is polluted with BaP. CMB model showed that PAHs in air
363 in KNUST and CC were emissions from diesel and gasoline engines respectively. 78% of PAHs
364 in CC originated from gasoline combustion. The estimated carcinogenicity of PAHs in terms of
365 BaPeq confirmed that BaP was the dominant PAHs contributor (70%). Estimated probabilistic

366 health risk for human adults and children was within the acceptable levels ($10^{-6}$ to $10^{-4}$),
367 suggesting low health risk to residents in Kumasi.

368

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558 **Figure captions:**

559 **Fig. 1** Distribution patterns of PAHs in $PM_{10}$ in KNUST and CC characterized by PCA; (a) score

560 plot (b) loading plot (K: pristine site; C: city centre; LMW: low molecular weight PAHs; HMW:

561 high molecular weight PAHs)

562 **Fig. 2** Chemical mass balance source apportionment of PAHs in air ($PM_{10}$) in KNUST

563 **Fig. 3** Chemical mass balance source apportionment of PAHs in air ($PM_{10}$) in CC

564

565 **Table 1** Concentrations of PAHs in air (ng/m$^3$) ($PM_{10}$) in different sites/countries

| City/Country | Σ PAHs | | | Reference |
|---|---|---|---|---|
| | Nos. | Range | Average | |
| KNUST, Ghana | 17 | 0.51–16 | 3.3 ± 3.3 | This study |
| City centre, Kumasi, Ghana | 17 | 19–38 | 30 ± 4.9 | This study |
| Chiang Mai, Thailand[#] | 16 | | 25 ± 10 | Wiriya et al. (2013) |
| Zonguldak, Turkey[##] | 14 | – | 94 ± 122 | Akyuz and Cabuk (2009) |
| Thailand[#] | 16 | 0.05–22 | – | Pengchai et al. (2009) |
| Zaragoza, Spain[###] | 18 | 0.4–30 | 8.8 ± 7.9 | Callen et al. (2008) |

566 Nos.: indicates, number of PAHs measured

567 [#]: indicates, Nap, Acl, Ace, Fle, Phe, Ant, Flu, Pyr, BaA, Chr, BbF + BkF, BaP, DBahA, IDP,
568 BghiP

569 [##]: indicates, Ace, Fle, Phe, Ant, Pyr, Flu, BaA, Chr, BbF, BkF, BaP, DBahA, IDP and BghiP

570 [###]: indicates, Phe, Ant, 2+2/4–methyl phe, 9-methyl phe, 1–methyl phe, 5–dimethyl phe, Flu,
571 Pyr, BaA, Chr, BbF, BkF, BeP, BaP, IDP, DBahA, BghiP and Coronene

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574 **Table 2** Abundance of PAHs in air samples in KNUST and CC

| | | | KNUST | CC | |
|---|---|---|---|---|---|
| PAHs | Abbreviation | Ring No. | % Abundance | % Abundance | *p* value |
| Phenanthrene | Phe | 3 | 21 | 2.6 | 0.56 |
| Anthracene | Ant | 3 | 3.6 | 0.91 | 0.15 |
| Methylene phenanthrene | Methy–Phe | 3 | 3.3 | 0.20 | 0.67 |
| 1–methyl phenanthrene | Me–Phe | 3 | 1.2 | 0.03 | 0.29 |
| Fluoranthene | Flu | 4 | 8.1 | 1.3 | 0.07 |
| Pyrene | Pyr | 4 | 11 | 1.7 | 0.30 |
| Retene | Ret | 3 | 15 | 1.2 | 0.64 |
| Benz[a]anthracene | BaA | 4 | 1.5 | 0.96 | **0.00** |
| Chrysene | Chr | 4 | 0.0 | 0.49 | **0.00** |
| Benzo[b+j+k]fluoranthene | BbF + BjF + BkF | 5 + 5 | 0.9 | 11 | **0.00** |
| Benzo[e]pyrene | BeP | 5 | 1.5 | 14 | **0.00** |
| Benzo[a]pyrene | BaP | 5 | 0.6 | 12 | **0.00** |
| Perylene | Peryl | 5 | 1.2 | 3.1 | **0.00** |
| Dibenz[a,h]anthracene | DBahA | 5 | 6.9 | 1.6 | **0.00** |
| Indeno[1,2,3–cd]pyrene | IDP | 6 | 7.8 | 19 | **0.00** |
| Benzo[g,h,i]perylene | BghiP | 6 | 16 | 29 | **0.00** |

575 Bold values: means data are statistically significant (ANOVA)

576

577 **Table 3** Mean Concentrations, standard deviations of PAHs in air (ng/m$^3$), toxic equivalence
578 factor (TEF), BaP equivalence (BaPeq) in Kumasi metropolis

| PAHs | TEF | Sampling Sites | | BaPeq | |
|---|---|---|---|---|---|
| | | KNUST (n = 20) | CC (n = 12) | KNUST | CC |
| | | Mean ± SD | Mean ± SD | TEF × Mean conc. | TEF × Mean conc. |
| **Phe** | 0.001 | 0.70 ± 0.49 | 0.79 ± 0.38 | 7.0E–04 | 7.9E–04 |
| **Ant** | 0.01 | 0.12 ± 0.27* | 0.27 ± 0.31* | 1.2E–03 | 2.7E–03 |
| **Flu** | 0.001 | 0.27 ± 0.19* | 0.39 ± 0.14* | 2.7E–04 | 3.9E–04 |
| **Pyr** | 0.001 | 0.39 ± 0.35* | 0.50 ± 0.16 | 3.9E–04 | 5.0E–04 |
| **BaA**[a] | 0.1 | 0.05 ± 0.02 | 0.29 ± 0.08 | 5.0E–03 | 3.0E–02 |
| **Chr**[a] | 0.01 | 0.01 ± 0.01 | 0.15 ± 0.06 | 5.0E–05 | 1.5E–03 |
| **BbF**[a] **+** BjF +**BkF**[a] | 0.1 | 0.03 ± 0.02 | 3.2 ± 0.98 | 3.0E–03 | 3.2E–01 |
| BeP | 0.01 | 0.05 ± 0.05* | 4.1 ± 1.1 | 5.0E–04 | 4.0E–02 |
| **BaP**[a] | 1 | 0.02 ± 0.03* | 3.7 ± 1.1 | 2.0E–02 | 3.7E+00 |
| Peryl | 0.001 | 0.04 ± 0.05* | 0.94 ± 0.32 | 4.0E–05 | 9.4E–04 |
| **DBahA**[a] | 1 | 0.23 ± 0.12* | 0.50 ± 0.16 | 2.3E–01 | 5.0E–01 |
| **IDP**[a] | 0.1 | 0.26 ± 0.35* | 5.8 ± 1.2* | 3.0E–02 | 5.8E–01 |
| **BghiP** | 0.01 | 0.53 ± 1.1* | 8.6 ± 2.3 | 1.0E–02 | 9.0E–02 |
| $\sum_{carc}$ PAHs | | 0.6 ± 0.5 | 14 ± 3.6 | 0.28 | 5.2 |
| $\sum_{16}$ USEPA PAHs | | 3.0 ± 3.5 | 25 ± 7.9 | 0.30 | 5.3 |
| $\sum_{14}$ PAHs | | 2.7 ± 3.0 | 29 ± 8.7 | 0.30 | 5.3 |

579 PAHs Carc[a]: BaA, Chr, BkF, BbF, BaP, DBahA and IDP according to IARC (2006)
580 Bold: indicates the 16 USEPA priority PAHs
581 *: indicates statistical significance with *p* value less than 0.05 (Shapiro–Wilks test for normality)
582 n: number of samples
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586 **Table 4** LADD and ILCR of PAHs in air from in CC

| | LADD | | | ILCR | |
|---|---|---|---|---|---|
| PAHs | Adult | Child | CSF | Adult | Child |
| **Phe** | 1.9E–07 | 5.3E–07 | | | |
| **Ant** | 6.6E–08 | 1.8E–07 | | | |
| Methy–Phe | 1.5E–08 | 4.2E–08 | | | |
| Me–Phe | 2.5E–09 | 6.9E–09 | | | |
| **Flu** | 9.4E–08 | 2.6E–07 | | | |
| **Pyr** | 1.2E–07 | 3.4E–07 | | | |
| Ret | 8.6E–08 | 2.4E–07 | | | |
| **BaA** | 6.9E–08 | 1.9E–07 | 0.61[a] | 4.2E–08 | 1.2E–07 |
| **Chr** | 3.5E–08 | 9.9E–08 | 0.0061[a] | 2.2E–10 | 6.1E–10 |
| **BbF** + BjF +**BkF** | 7.6E–07 | 2.1E–06 | 0.061[a] | 4.6E–08 | 1.3E–07 |
| BeP | 9.8E–07 | 2.8E–06 | | | |
| **BaP** | 8.9E–07 | 2.5E–06 | 6.1[a] | 5.4E–06 | 1.5E–05 |
| Peryl | 2.3E–07 | 6.3E–07 | | | |
| **DBahA** | 1.2E–07 | 3.3E–07 | 6.1[a] | 7.2E–07 | 2.0E–06 |
| **IDP** | 1.4E–06 | 3.9E–06 | 0.61[a] | 8.4E–07 | 2.4E–06 |
| **BghiP** | 2.1E–06 | 5.8E–06 | | | |
| ∑ PAHs | 7.1E–06 | 2.0E–05 | | | |
| Minimum | 2.5E–09 | 6.9E–09 | | | |
| Maximum | 2.1E–06 | 5.8E–06 | | | |
| Average | 4.4E–07 | 1.3E–06 | | 1.2E–06 | 3.3E–06 |

587 [a] indicates CSF values (WDNR 1997; USEPA 1992)
588 Bold: indicates 16 USEPA priority PAHs
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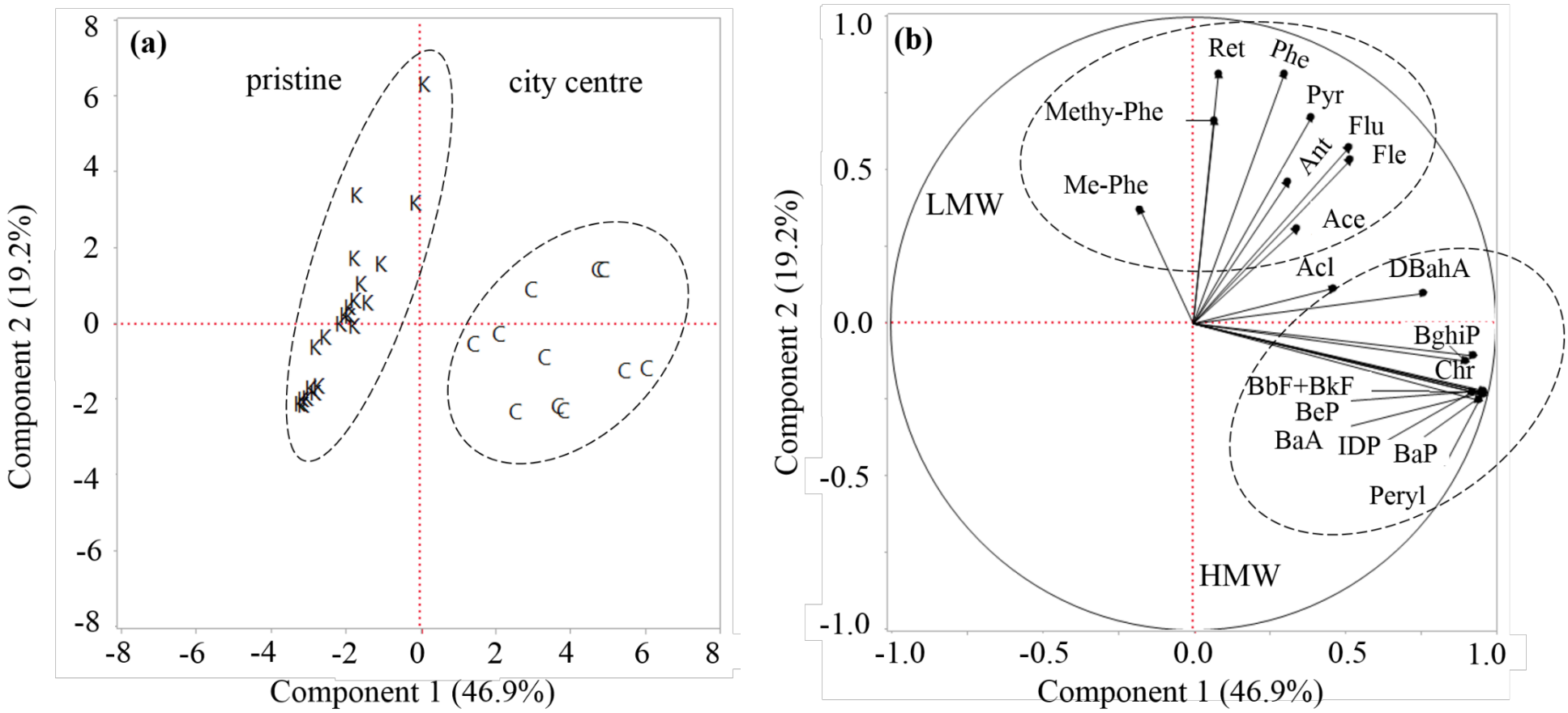
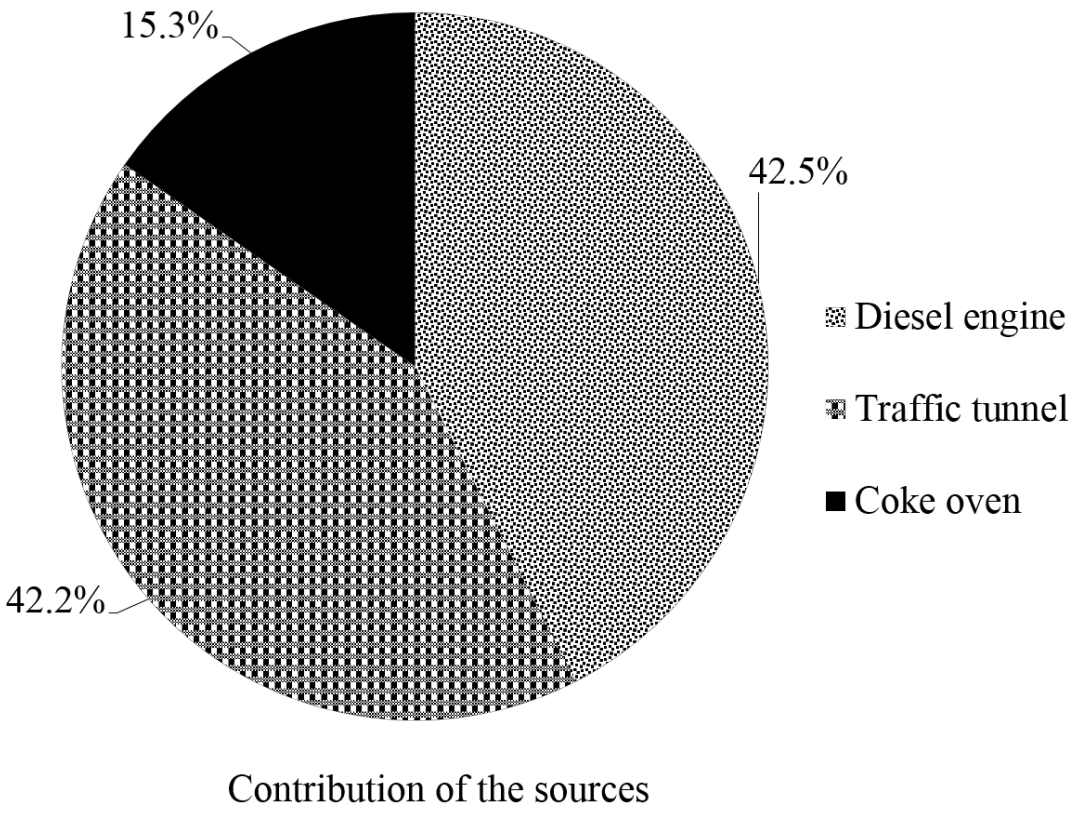

(a)
pristine
city centre
Component 2 (19.2%)
Component 1 (46.9%)
(b)
Ret
Phe
Pyr
Flu
Fle
Ant
Methy-Phe
Me-Phe
Ace
Acl
DBahA
BghiP
Chr
BbF+BkF
BeP
BaA
IDP
BaP
Peryl
LMW
HMW
Component 2 (19.2%)
Component 1 (46.9%)

**Fig. 1**

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599 **Fig. 2**

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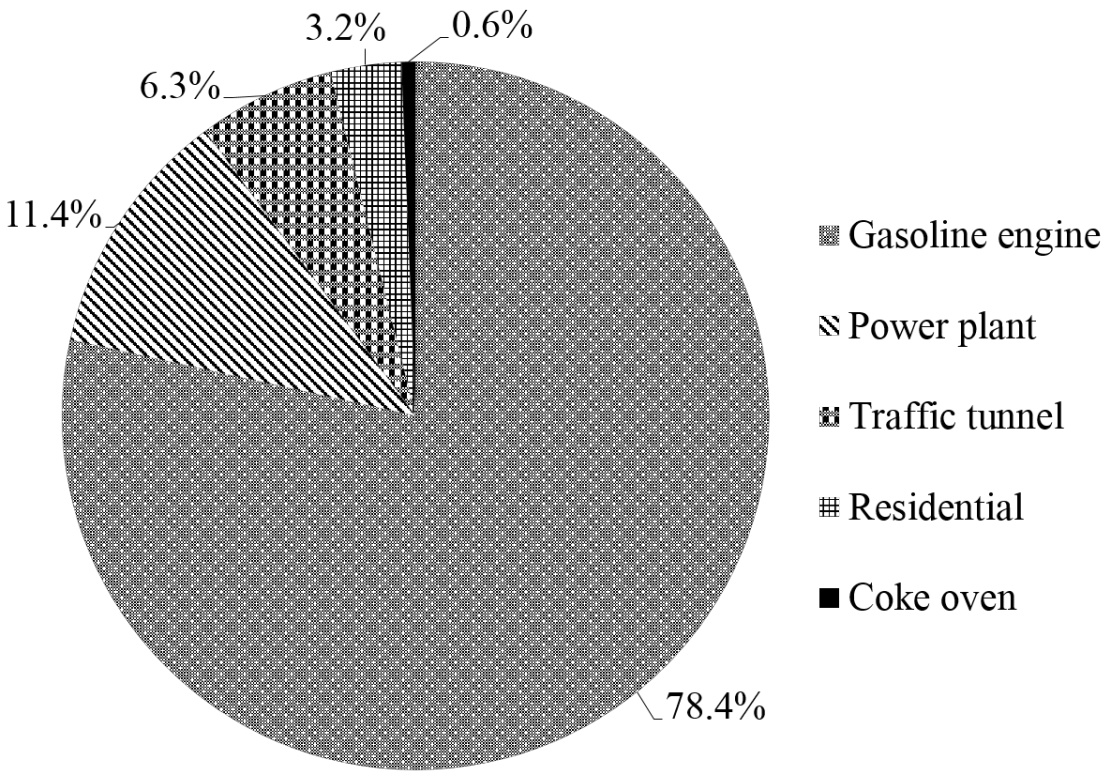
3.2%
0.6%
6.3%
11.4%
78.4%
Gasoline engine
Power plant
Traffic tunnel
Residential
Coke oven
Contribution of the sources

**Fig. 3**