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Cancer risk from polycyclic aromatic hydrocarbons in fine particulate matter generated from household coal combustion in Xuanwei, China

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Abstract

Polycyclic aromatic hydrocarbons (PAHs) and their polar derivatives (oxygenated PAHs: OPAHs and azaarenes: AZAs) were characterized in fine particulates ($PM_{2.5}$) emitted from indoor coal combustion. Samples were collected in Xuanwei (Yunnan Province), a region in China with a high rate of lung cancer. A sample from the community with the highest mortality contained the highest total concentration of PAHs (at least 1.42 times higher) and posed the highest excess risk for cancer from a lifetime of inhaling fine particulates. Positive correlations between total carbonyl-OPAHs/AZAs and PAHs implied that the emissions were from the same source, regardless of sample location and type. The calculated cancer risk ranged from $5.23–10.7 \times 10^{-3}$, which is 3.61 times higher than the national average. This appears to be because inhabitants in the area tend to spend more time cooking, and more days per year indoors than the national average. The risk in each sample was ∼1–2 orders of magnitude higher than that deemed high risk, suggesting that the safety of these households is in jeopardy.

Keywords:

Indoor Air; PAHs; Oxygenated PAHs; Azaarenes; Coal; Cancer Risk

Practical Implications:

Emissions from household coal combustion were sampled in Xuanwei, Yunnan Province, China. From the most harmful kind (fine = $PM_{2.5}$) of particulate matter, a number of hazardous polycyclic aromatic hydrocarbons (PAHs), and their polar derivatives (oxygenated PAHs and azaarenes), were quantified at unusually high concentrations. This seems highly significant given that the cancer-related mortality of this region is among the highest in China. The evaluation of local and personal exposure factors resulted in estimates of very high cancer risk

from inhalation of fine particulates while cooking indoors. Samples in different communities all indicated cooking risk higher than that deemed high risk (> 10^{-4}), suggesting the need to revise current regulations on coal usage.

1. **Introduction**

Coal is a fossil fuel containing various types of solids (e.g., combustible, sedimentary, and organic rock) that are commonly present in rock strata. China is the largest coal producer and consumer in the world: more than 75% of its electricity supply comes from coal combustion (Liu et al., 2008). Over 400 million residents in China still use coal as their domestic energy source (i.e., for heating and cooking) because of the country's massive coal reserves (~1000 billion tons). These reserves provide relatively economical energy with wide access (Bin, 2000; Xu et al., 2000; Tang and Wang, 2000; Liu et al., 2004). Carbonaceous aerosol emissions due to coal combustion (~70% of total) severely deteriorate environmental conditions in China (Cooke et al., 1999). Concerns about coal burning in the past have usually targeted gaseous pollutants (e.g., CO, CO_2, NO_x, and SO_2); however, recent studies have shown that particulate matter (PM) emitted during combustion contains large amounts of polycyclic aromatic hydrocarbons (PAHs) and other hazardous compounds (e.g., elemental carbon, organic carbon, and silicon) that could impact environmental conditions and human health (Stracher and Taylor, 2004; Borm, 1997; Ando et al., 1998; Schins and Borm, 1999).

The type of toxic substances emitted from indoor coal combustion depend on the quality (chemical and morphological) of the coal, the efficiency of the combustion process, and the ventilation used indoors (Simoneit et al., 2007; Tian et al., 2009; Downward et al., 2014b). Polycyclic aromatic compounds (PACs) bound to fine particulate matter are of prime concern because fine particulates are capable of traveling further into human respiratory passages, and the smallest of these can enter the blood stream after inhalation. Past studies showed that a high proportion of PACs are bound to these finer particles (Ringuet et al., 2012; Cao et al., 2013; Hu et al., 2014). PACs are known to have mutagenic, genotoxic, carcinogenic, and estrogenic properties (Boström et al., 2002; Machala et al., 2001). Previous studies on the composition of PAC emissions from coal combustion were mainly focused on the parent PAHs (Mumford et al., 1995; Downward et al., 2014a).

Recent studies, however, demonstrated that coal combustion emissions contain other constituents (e.g., alkyl-PAHs, oxygenated PAHs (OPAHs), azaarenes (AZAs), and nitro-PAHs) that are also of concern (Bi et al., 2008; Simoneit et al., 2007; Shen et al., 2013). Azaarenes can be formed by conversion of parent PAHs via photochemical, thermal, and photolytic reactions (Lundstedt et al., 2014a). Ding et al. (2012) detected higher concentrations of oxygenated PAHs than parent PAHs in Chinese kitchens. Toxicological tests confirmed that the more polar derivatives are even more toxic than the parent PAHs (Umbuzeiro et al., 2008; Andrysík et al., 2011; Wang et al., 2011; Machala et al., 2001). These newly recognized polar PACs can therefore be assumed to be more environmentally threatening than "regular" PACs and potentially linked to higher lung cancer rates.

Xuanwei City-County is located in Yunnan Province (China) and has a population of approximately 1.2 million people in an area of 6257 km^2. Xuanwei is renowned for its exceptionally high rate of mortality from lung cancer (i.e., in 2004–2005, the lung cancer mortality rate in Xuanwei was 91 per 100,000 people, whereas the national average was 31 per 100,000 people) (Lin et al., 2015). Mumford et al. (1995) proposed that epidemic diseases might be related to PAHs released to interior air from coal burning. An attempt was made to reduce indoor air pollution by installing chimneys; however, this was not completely successful (Tian et al., 2009).

The aims of this study are to: 1) characterize $PM_{2.5}$-bound PAHs and polar PACs (OPAHs and AZAs) from four types of coal used in Xuanwei; 2) determine the PAC composition and characterize the relationships between the components; 3) estimate the potential health implications of interior coal emissions for local inhabitants.

2. Materials and methods

2.1 Experimental procedures

2.1.1 Indoor environmental conditions mimic "Pre-1990" kitchen design

Four types of coal were tested for emissions. The samples were labeled A–D and were collected from different locations, Dongshan (K9), Zhaojiachong (B3), Dongshan (K3), and Laibin (B2), in Xuanwei. The coal seams from which the samples were collected are denoted in parentheses. The coal combustion experiment was conducted between November 2012 and January 2013, in a separate kitchen area opposite a one story building in a village called Shangzuosuo (Xuanwei). The volume of the kitchen was ~42.6 m^3 (5.9 m long × 3.8 m wide × 1.9 m high). The air exchange rate in the kitchen was continuously monitored by measuring the first-order decay of carbon dioxide using a Q-Trak™ indoor air quality monitor (model 8550; TSI, Inc., Shoreview, MN, USA). The air change rate was set as 6.9 h^{-1}.

2.1.2 Preparation of fuels

A laboratory stove (internal diameter of 15 cm; shown in Figure S1 of Supplementary Material) was used to simulate a fire pit for routine daily burning of coal. Larger coal pieces were sieved to retain only samples <5 cm in diameter, to facilitate combustion performance. The stove mass (~7 kg) and coal masses sampled (0.8 ± 0.7 to 1.6 ± 0.1 kg) were monitored throughout the experiment. The coal masses were a random factor and epitomized the usual mass range used for domestic cooking activities.

2.1.3 Burning cycle

Four types of coal samples were used in the combustion tests to collect air particulate samples. Analysis of samples of each type of coal was done in triplicate. The fire was set and kindling was ensured. The air was then purged through a stove inlet to provide oxygen for combustion and a chimney was installed over the stove to optimize the chimney effect. When full kindling had occurred (~5 min after initial ignition), ~2 kg of the coal sample was immediately added to the stove. After 10 min from initial ignition, the remainder of the coal sample was used to fill up the stove. The stove was immediately positioned above the burning coal and remained in place until completion of the experiment. The weight of the stove and coal samples were recorded. All biomass materials were completely removed outdoors, prior to setting the fire. A water pot containing 2 kg of water at room temperature was placed above the stove. Coal lumps could melt and coagulate during combustion, which could extinguish the fire. To simulate cooking in the best possible manner, the fire was stoked and poked at the beginning and at 20 min intervals during the combustion cycle to assure favorable air ventilation through the coal lumps. Additional coal was added to the stove at 20 min intervals, and the weights were recorded throughout the cycle. The water was heated to a boil during the heating process. The complete heating process required 30–60 min depending on the different types of coal. The remaining ashes were weighed after each combustion cycle. The combustion cycle was on par with household coal burning activity in Xuanwei (~1 h). The fire was either re-used (for another burning cycle with the same type of coal) or extinguished using a water sprayer. The weight of the coal and water was recorded at 10 min intervals during the experiment.

2.1.4 $PM_{2.5}$ sampling

All Teflon membranes and quartz microfiber filters were equilibrated at 21.5 ± 2 °C and relative humidity of 35 ± 5%, for no less than 24 h before weighing. Sample weighing was performed on a microbalance (Mettler Toledo MT5 with ±0.001 mg sensitivity). The charge on each filter was neutralized by exposure to a ^{210}Po ionizing source for no less than 30 s before the filter was transferred to the balance pan. The balance was calibrated to 200 mg and 100 mg Class 1 weights, and tare was set before weighing the filters. The 200 mg Class 1 weight was used for re-calibration after weighing every 10 filters. The balance performance was ensured not to deviate more than ±0.003 mg by specification. Two Mini-volume portable air samplers (Mini-vol, Airmetrics, Eugene, OR, USA) were used to collect air samples at uniform flow rates of 5 L/min during the combustion cycle. The samples were collected separately on 47 mm diameter Teflon membranes and on quartz microfiber filters inside a $PM_{2.5}$ inlet cassette on the Mini-vol samplers. The air was purged through a PM_{10} size cut impactor, and coarse particles were filtered out. The remaining particles went through the $PM_{2.5}$ impactor and particles with aerodynamic diameters larger than 2.5 µm were filtered out. Care was taken that the filters were not overloaded with particles. The $PM_{2.5}$ particles were deposited on the filters for further analysis.

2.2 Analytical methods

2.2.1 Analysis of PAHs, OPAHs, and AZAs

The concentrations of PAHs, OPAHs, and AZAs were determined for each filter sample. Filters were cut into smaller pieces and transferred to a 33 mL accelerated solvent extractor (ASE) extraction cell and spiked with deuterated PAHs (100 µL of a mixture of naphthalene-D8, acenaphthene-D8, phenanthrene-D10, pyrene-D10, chrysene-D12, perylene-D12, and benzo[ghi]perylene-D12, each with a concentration of 10 µg mL^{-1}); deuterated carbonyl-

OPAH (50 µL Benzophenone-D5, 9,10-Anthraquinone-D8: 20 µg mL^{-1}); 50 µL Carbazole-D8 (20 µg mL^{-1}); and 2-Napthol-D7 (20 µg mL^{-1}), as internal standards for PAHs, carbonyl-OPAHs, AZAs, and hydroxyl and carboxyl-OPAHs, respectively. The remaining spaces within each ASE cell was filled with inert bulk sorbent (Isolute HMN, Biotage, Uppsala, Sweden). Each sample was then extracted twice by pressurized liquid extraction (ASE 200, Dionex, and Sunnyvale, CA, USA). The first extraction of each sample was done using a dichloromethane solution, and the second extraction was done using a mixed solution of acetone and dichloromethane (2:1 v/v). The conditions of the ASE instrument were the same as specified elsewhere (Bandowe and Wilcke, 2010; Bandowe et al., 2010; Bandowe et al., 2011). The two extracts from each sample were combined, and 15 ml of hexane was added. This was concentrated to a volume of <1 mL in a TurboVap II Concentrator Workstation (Biotage, Charlotte, NC, USA) operated at a 35 °C bath temperature and an N_2 gas pressure of 15 Psi. Each concentrated sample was then quantitatively transferred onto 3 g of silica gel (10, deactivated) in a glass column for clean-up and fractionation. The column was sequentially eluted with [1] 15 mL hexane:dichloromethane (5:1 v/v), and [2] 8 mL dichloromethane and 5 mL acetone. Fractions [1] and [2] were collected in separate 50 mL pointed flasks. Each fraction was spiked with toluene (0.75 mL), concentrated to <1 mL, then spiked with 25 µL of fluoranthene-D_{10} (22 µg mL^{-1}) before being transferred to a 2 mL vial for PAC analysis. PAHs, carbonyl-OPAHs, AZAs, and hydroxyl and carboxyl OPAHs were measured in separate runs using a gas chromatograph (Agilent, 7890 N) coupled to a mass spectrometer (Agilent, 5975 C). Hydroxyl and carboxyl-OPAHs were measured after derivatization with BSTFA+TMCS (99:1 v/v) as previously described (Bandowe and Wilcke, 2010). All GC-MS measurements were run in split-less mode, with MS operating in the electron-impact-ionization, selected-ion-monitoring mode. Further details of the instrumental conditions of the GC-MS system, the target, and the qualifier ions for each compound were the same as specified in previous papers

(Bandowe and Wilcke, 2010; Bandowe et al., 2011; Bandowe et al., 2014a; Bandowe et al., 2014b; Lundstedt et al., 2014). Target compounds were quantified using internal standard quantification calibration curves set up with the Agilent ChemStation Software. Further details of the quality control procedures can be found in Text S1 (Supplementary Material).

2.3 Calculation of excess cancer risk

The lifetime excess cancer risk due to inhalation of $PM_{2.5}$-bound PAHs in the emissions was calculated based on Eq. (1):

$$\text{Lifetime Cancer Risk} = \sum_{i=1}^{n}\left(\frac{(C_i \times PEF_i) \times IR \times EF \times SF}{BW \times AT \times CV}\right) \quad (1)$$

Where C_i is the average concentration of the i^{th} target compound (ng m^{-3}) and PEF_i is the potency equivalency factor (PEF) of the i^{th} target compound. PEF_i values (only fluoranthene, pyrene, benzo[a]anthracene, chrysene and triphenylene, benzo[b+j+k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, dibenzo[a,h]anthracene, and benzo[ghi]perylene were used) were set according to the literature (Collins et al., 1998; Nisbet and LaGoy, 1992). Other PEF values (PAHs, OPAHs and AZAs) were not available. IR is the average inhalation rate (m^3 hr^{-1}). EF is the exposure factor and is determined by average duration indoors (h d^{-1}), average indoor exposure frequency (days), and average life expectancy (years). SF is the inhalation slope factor (OEHHA, 2002 and 2005). BW is the average body weight (kg). AT (days) is the average exposure duration for carcinogenic effects. An estimated average exposure duration of 25,550 days (70 years) for carcinogenic effects was applied in the calculation (Hoddinott and Lee, 2000). CV is a conversion factor (from ng to mg). The IR, EF, and BW were calculated based on the information given in the Chinese exposure factors handbook, and the average duration indoors was assumed based on time–activity patterns of cooks in kitchens from a relevant study in China (Duan, 2015; Jiang and Bell, 2008). Information extracted from the handbook and relevant studies can be found in Table 3.

3. Results and discussion

3.1 Concentrations of PAHs

Table 1 shows concentrations of PAHs in the samples. The PAHs are either unsubstituted or methyl- and dimethyl-substituted homologs. All of the Xuanwei $PM_{2.5}$ samples were identified with the same number of PAHs compounds; however, the absolute concentrations of individual components varied by ~1–2 orders of magnitude. Sample D shows the highest concentrations of PAHs, ~1.06–1.19 times higher than the other samples (A–C). Because Sample D was collected from "Labin", the location that has been, since 1990, and remains, the community with the highest mortality from lung cancer. The age-standardized mortality rate reached ~160 per 100,000 in 2011–2013. Lung cancer in the research area accounted for ~57–63% of all deaths from cancers compared to ~21–26% in rural areas of China (i.e., in the periods 1990–1992, 2004–2005, and 2011–2013). It was also noted that in the communities with the highest mortality rates (e.g., Labin), the difference could be >20 times the rates in communities with the lowest mortality (Chen et al., 2015a). In the samples, phenanthrene (975.5–2020.1 μg/g $PM_{2.5}$) and benzo[a]anthracene (1057.3–1982.9 μg/g $PM_{2.5}$) were the two most abundant components and accounted for ~9–18% of the total in all the samples. The substances 1-methylphenanthrene (970.2–1095.8 μg/g $PM_{2.5}$), 3,6-dimethylphenanthrene (456.5-–708.9 μg/g $PM_{2.5}$), pyrene (785.3–1189.7 μg/g $PM_{2.5}$), benzo[b+j+k]fluoranthene (842.9–1262.6 μg/g $PM_{2.5}$), benzo[e]pyrene (786.3–884.3 μg/g $PM_{2.5}$), and benzo[ghi]perylene (573.0–644.0 μg/g $PM_{2.5}$) are the components that contributed in the range 5–11%. All of the remaining components made up less than 5% of the total. For the PAHs, compounds >1% by composition were three-ring (or more) PAHs. Five- to seven-ring PAHs showed similar compositions among samples A–D (Figure S2, Supplementary Material). Benzo[a]pyrene concentrations ranged from 294.9 to 508.1 μg/g in $PM_{2.5}$ and were ~3–5% of the PAH concentrations. This is consistent with a similar residential coal combustion study in China (Chen et al., 2004). A previous study showed that concentrations of benzo[a]pyrene in various indoor air conditions in Britain (pubs with environmental tobacco smoke, households with wood burning, or gas-fire activity) were all less than 5 ng/m^3 (Delgado-Saborit et al., 2011). Benzo[a]pyrene is a probable human carcinogen and, along with other multi-ringed PAHs (five rings or more), is mostly bound to particles (Pankow, 1987; Wang et al., 2002). According to the World Health Organization (WHO), a unit risk of lung cancer for PAH mixtures is estimated to be 8.7×10^{-5} per ng/m^3 of benzo[a]pyrene for PAH in indoor air (W.H.O., 2010). Armstrong et al. (1994) reported that exposure to a mixture of PAH compounds equivalent to 0.25–2.5 μg/m^3 benzo[a]pyrene over 40 years could be associated with a 50% increase of lung cancer risk in an aluminum smelter study. The concentrations of benzo[a]pyrene in samples A–D nonetheless exceeded safety exposure levels. Sample D further shows the highest total concentration of U.S. EPA Group B2 PAHs (141.5 μg/m^3) in indoor air, ~1.06–2.00 times higher than the other samples (A–C).

The concentrations of benzo[a]pyrene (788 μg/g PM_{10}) and other five- to seven-ring PAHs (indeno[1,2,3-cd]pyrene (435 μg/g PM_{10}), benzo[ghi]perylene (999 μg/g PM_{10}), and coronene (263 μg/g PM_{10})) in a previous smoky coal combustion study in Xuanwei homes (Mumford et al., 1995), were all higher than in the present study. This shows that the concentrations of individual PAHs in PM_{10} are higher than in $PM_{2.5,}$ which is in agreement with other studies (Wu et al., 2014; Dong et al., 2012; Yang et al., 2012). Past studies used PAH diagnostic ratios to characterize different PAH sources (Ravindra et al., 2008). The PAH diagnostic ratios of indeno[1,2,3-cd]pyrene/indeno[1,2,3-cd]pyrene+benzo[ghi]perylene and fluoranthene/fluoranthene+pyrene from combustion of five types of coal were 0.14–0.32 and 0.36–0.53, respectively (Huang et al., 2014). A similar residential coal combustion study in China reported the two ratios were on average of 0.57 ± 0.05 and 0.60 ± 0.06, respectively (Wang et al., 2016). The present study shows that the two ratios were in the range 0.24–0.32 and 0.29–0.32, respectively, which is on par and lower than, respectively, the above values. This study only reported $PM_{2.5}$-bound PAHs and their derivatives; therefore, differences may arise compared to other values in the literature, which usually include gas phase measurements (Huang et al., 2014). The coal rank (indicator of quality) determines the abundance of the coal combustion particulate constituents (Simoneit et al., 2007). Particulate-phase PAHs could change significantly because of partitioning and deposition of particulates after emission to the atmosphere (Ding et al., 2012). PAH formation could be influenced by combustion conditions such as temperature, moisture, and oxygen supply (Jenkins et al., 1996). These might all lead to different PAH diagnostic ratios. Comparison between data in the literature requires careful attention.

3.2 Concentrations of OPAHs and AZAs

Table 2 shows the concentrations of all the polar PACs in the coal samples. Individual carbonyl-OPAH, hydroxyl and carboxyl-OPAH, and AZA components are in the same order of magnitude. Sample D shows the highest concentrations of AZAs, hydroxyl and carboxyl-OPAHs, and carbonyl-OPAHs (which were 1.42–1.57, 1.65–1.74, and 1.57–1.71 times, respectively, higher than the other samples (A–C).

Carbazole (492.5–663.0 μg/g $PM_{2.5}$), and 2-Naphthol (267.2–497.2 μg/g $PM_{2.5}$), were the most abundant components of AZA and hydroxyl and carboxyl-OPAHs in each sample, accounting for ~50–60% and ~57–66%, respectively, of the total concentrations (Figure S3, Supplementary Material). The carbonyl-OPAHs were, on the other hand, dominated by 7H-benz[de]anthracene-7-one, 9-fluorenone, and 9,10-anthraquinone (Figure S3). Several of these OPAHs are known to actively induce toxicological processes that are responsible for mutagenic, carcinogenic, and cytotoxic effects (Wilson et al., 1996; Misaki et al., 2016; Wincent et al., 2016). The IARC has also recently classified 9, 10-anthraquinone as a possible Group 2B human carcinogen (IARC, 2013a).

Carbazole and the other AZAs found in the emitted $PM_{2.5}$ are known to exert a wide range of (eco)toxicological effects and are inducers of toxicological mechanisms that can cause cancerous mutations and developmental effects (Peddinghaus et al., 2012). AZAs showed cytochrome P450 (CYP)1A induction potencies similar to those of related PAHs, despite usually lower concentrations in the environment (Jung et al., 2001). The IARC has also classified carbazole as a possible human carcinogen (IARC, 2013b).

The presence of 2-naphthol in the coal emissions is consistent with a similar coal combustion study (Bi et al., 2008). Hydroxyl-OPAHs have also been shown to be cytotoxic (Wilson et al., 1996), and possess an aryl hydrocarbon receptor binding property (Ohura et al., 2010), a process known to be responsible for the carcinogenic effects.

Urinary 2-naphthol is used as a biomarker for inhalation exposure to airborne PAHs (e.g., naphthalene). Most of the urinary biomarkers provide only a rough estimate of the carcinogenic dose because of limited understanding of the human metabolism of the parent compound (Nan et al., 2001). Direct exposure to 2-naphthol smoke could possibly alter the concentration of urinary 2-naphthol because making the distinction between non-metabolic and metabolic 2-naphthol remains difficult. Hence, the accuracy of estimations of the carcinogen dose could possibly be affected.

The concentration ratios of OPAHs to their parent-PAHs shows that the 9,10-anthraquinone/anthracene and 9-fluorenone/fluorene ratio were in the range 1.2–2.0 and 24.3–136.1, respectively, confirming the characteristics of OPAHs, which are low volatile compounds that favor association with atmospheric particles. Possible secondary formation of OPAHs in warm kitchens could occur during winter (Ding et al., 2012), and the present study was conducted in winter. The high ratios could be due to the conditions mentioned above.

Figure 1 shows correlations between the log-transformed concentrations of OPAH or AZA compounds and their corresponding parent PAH compounds. Six pairs of OPAHs or AZAs were investigated in relation to the parent PAH compounds. Three exhibit positive values, the rest are negative, and two have significant correlations (9, 10-anthraquinone/anthraquinone and benzo[h]quinoline/phenanthrene) ($p < 0.05$). The regression analysis shows that variation of the OPAH or AZA (and parental PAH) concentrations in the two pairs depend on the same combustion conditions (e.g., moisture content, amount of oxygen supply, and temperature). The linear relationships suggest quantitative dependence of the OPAHs or AZAs on their parental PAH counterparts, in a positive manner. However, the limitation of the correlations is that, how individual combustion parameters (e.g., temperature) correlate to the concentrations cannot be determined because of insufficient information about the combustion conditions. Figure 2 reveals the correlations between the log-transformed concentrations of total polar PACs against concentrations of PAHs. The dependence of carbonyl-OPAHs or AZAs on PAHs is positively correlated ($R > 0.60$, $p < 0.05$), which implies that the emissions were from the same source, regardless of sample locations and types. No correlation was identified between total hydroxyl and carbonyl-OPAHs and PAHs, which suggests that the total concentrations of the two are dependent on the source and sample type.

3.3 Lifetime excess cancer risk assessment from PAHs

Figure 3 shows the estimated lifetime excess cancer risk per million people due to exposure to PAH inhalation in the kitchens of Xuanwei, Yunnan Province. The non-dietary exposure in this study is defined as human exposure to $PM_{2.5}$-bonded PAHs via inhalation of household air. The total cancer risk value ($>10^{-4}$) is considered high in common regulatory programs (Chen and Liao, 2006). Under the same conditions of PAH exposure (as in Table 1), the mean estimated excess cancer risk associated with inhalation exposure is in the range of 5231–10,712 cancer cases per million people ($\sim 5.23–10.7 \times 10^{-3}$) in the area near the experimental kitchen in Yunnan. Dibenzo[a,h]anthracene is the component that contributed at least 55% of the total risk. The highest inhalation risk was observed in sample D and showed 1.05–2.05 times higher risk than the other samples. This suggests that different types of coal could contribute to the variation of inhalation risk.

Under the same set of PAC emissions, the inhabitants of Yunnan show ~3.61 times higher risk compared to the national average due to different exposure conditions (Table 3) (Duan, 2015; Jiang and Bell, 2008).

All of the above results show the inhalation cancer risk is ~1–2 orders of magnitude higher than the risk value classified as high risk, which is clearly not safe. The levels of PAC emissions (Table 1) are the determinants of estimated inhalation risk regardless of the type of coal, and the exposure conditions at the calculated risk levels (A–D) are all above the values rated high risk. The PAC levels in the kitchen could also have implications for risk in other living areas in the house, especially in winter, when all the house windows are usually closed and ventilation is limited.

These risk levels are alarming, even without taking into consideration the emissions of polar PACs (limited by absence of TEF values). OPAHs and AZAs were demonstrated to be highly cytotoxic, mutagenic, carcinogenic, and estrogenic (Walgraeve et al., 2010; Sidhu et al., 2005). Therefore, the risk could be potentially even higher than the current levels. Future study should include the polar PAC components in the assessment, provided the TEF values are available.

A household air pollution study investigated various household energy sources (e.g., honeycomb briquettes) in Shanxi, China, and showed that the 95th percentile of PAHs exposure of cancer risk in the area was 1.7×10^{-5} (Chen et al., 2015b). The 95th percentile values of inhalation cancer risk during combustion of firewood and dung cakes were 2.1 and 3.0×10^{-4}, respectively, in a domestic environment in India (Tiwari et al., 2015).

The above risk findings summarize variable indoor inhalation cancer risks, targeting risk in different combustion conditions. Nonetheless, the risk levels in Yunnan are higher than the outcomes above. These differences could be attributed to high concentrations of particulate PAHs in the coal smoke samples in Xuanwei. The factor of longer exposure in Yunnan, compared to the national average, could also have contributed to the higher risk levels. Winter heating was an important factor increasing the $PM_{2.5}$ concentration in China (Xiao et al., 2015) and inhabitants of Xuanwei spend an average of >75% of their time per day indoors (Duan, 2015). Many of the studies on household indoor air pollution have concentrated only on indoor air concentrations without considering personal exposure factors. This results in an incomplete understanding of the correlation between the two (Clark et al., 2013). The present cancer risk calculation is completed with an attempt to use relevant and accessible information, because the exposure factors were specific for Yunnan province and only recently revealed (Duan, 2015). The results suggest that the characteristics of coal usage by Xuanwei households can pose a bigger threat to humans than all of the aforementioned conditions.

However, the overall cancer risk assessment contains uncertainties such as inadequate personal exposure information regarding the age and sex of the inhabitants in Yunnan (i.e., lack of exposure information specific for Xuanwei). Insufficient characterization of the sampling households, for example, the number of windows and number of stoves in each household, as well as seasonal variation, could have affected the final cancer risk outcome. Additional studies should focus on quantifying and harmonizing these uncertainties (e.g., using personal air monitoring devices to collect personal exposure data in Xuanwei households) to improve future cancer risk analyses.

4. Conclusions

The characteristics of fine particulate matter ($PM_{2.5}$) emitted during indoor coal combustion in Xuanwei were investigated. A sample from a community with the highest lung-cancer-related mortality contained the highest concentrations of total PAHs. This was at least 1.42 times higher in total polar PAHs concentration than found in samples outside the communities with the highest cancer mortality. Positive correlations were identified in a statistical regression analysis, which showed that total carbonyl-OPAH or AZA (in relation to PAHs) emissions were from the same source regardless of sample location and type. The lifetime excess cancer risk from inhalation of fine particulates suggests that the sample from one of the communities with the highest cancer mortality poses the highest risk. Xuanwei inhabitants in general bear ~3.61 times higher risk compared to the national average, and the risk is ~1–2 orders of magnitude higher than the value deemed high risk.

These findings support claims that household coal combustion is associated with negative human health conditions. The results suggest the need to revise current regulations affecting ordinary coal usage.

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Table 1 PAH Concentrations*

Sample (µg/g $PM_{2.5}$)[a]/(ng/m^3 air)[b]	A	B	C	D
1,2,3,4-Tetrahydronaphthalene	(1.5±0.6) / (48.8±4.9)	(3.5±3.5) / (59.7±41.4)	(8.2±3.3) / (103.6±4.5)	(4.3±0.6) / (130.2±32.1)
Naphthalene	(11.9±2.5) / (416.8±60.5)	(25.3±18.5) / (460.3±178.2)	(51.4±18.7) / (658.4±40.8)	(29.8±3.6) / (898.5±130.5)
2-Methylnaphthalene	(1.6±0.5) / (57.8±25.1)	(2.7±2.1) / (50.6±23.7)	(6.1±2.2) / (78.4±6.4)	(5.0±0.8) / (154.3±45.8)
1-Methylnaphthalene	(3.1±0.6) / (108.9±19.6)	(3.1±2.7) / (75.4±66.4)	(B.D.) / (B.D.)	(B.D.) / (B.D.)
Biphenyl	(3.7±1.1) / (127.9±5.1)	(9.3±8.1) / (163.6±88.5)	(21.0±5.3) / (281.3±60.0)	(9.9±3.0) / (298.2±83.6)
1,3-Dimethylnaphthalene	(5.2±5.3) / (170.8±148.2)	(11.6±10.1) / (257.3±261.8)	(20.3±6.5) / (272.5±73.7)	(13.2±12.1) / (415.5±414.0)
Acenaphthylene	(B.D.) / (B.D.)[c]	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)
Acenaphthene	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)
Fluorene	(19.4±1.9) / (703.1±215.3)	(19.8±6.3) / (445.1±233.6)	(12.6±0.7) / (182.6±91.4)	(7.1±3.3) / (212.8±94.3)
Phenanthrene	(1699.8±437.1) / (65911.4±35876.3)	(1435.2±567.7) / (32337.6±17982.6)	(975.5±346.1) / (15901.1±13377.7)	(2020.1±271.3) / (61822.7±15733.6)
Anthracene	(349.8±107.7) / (13625.2±7,631.2)	(398.2±141.0 / (8901.4±4678.8)	(252.1±57.0) / (3934.3±2806.4)	(502.6±76.9) / (15402.3±4173.1)
1-Methylphenanthrene	(1095.8±48.9) / (40248.1±13287.1)	(1038.0±178.5) / (22303.7±8211.7)	(970.2±90.7) / (14112.2±6992.4)	(1087.6±26.2) / (32875.5±3803.3)
3,6-Dimethylphenanthrene	(708.9±45.9) / (25943.7±8685.8)	(586.3±136.0) / (12845.7±5665.2)	(456.5±101.0) / (6754.2±3778.0)	(582.2±27.3) / (17636.7±2525.5)
Fluoranthene	(322.3±164.7) / (12098.4±6738.0)	(405.3±173.8) / (8762.6±4332.0)	(500.7±160.1) / (6538.2±886.2)	(529.5±93.0) / (15910.8±2545.5)
Pyrene	(785.3±229.3) / (29020.5±11823.9)	(1050.5±357.5) / (22360.5±9264.8)	(1189.7±368.2) / (15597.2±2325.1)	(1125.4±204.1) / (33839.3±5767.8)
Retene	(362.1±10.4) / (13360.1±4829.7)	(344.0±38.1) / (7358.0±2806.7)	(218.5±48.2) / (3378.5±2320.1)	(319.4±18.9) / (9724.0±1878.1)
Benzo[a]anthracene[d]	(1057.3±334.2) / (39064.0±16356.0)	(1559.6±516.0) / (33126.0±13390.3)	(1982.9±744.8) / (25283.0±1074.8)	(1611.6±318.2) / (48527.6±9462.5)
Chrysene+Triphenylene[d]	(480.9±65.1) / (17683.1±6007.2)	(489.7±138.3) / (10443.3±4036.9)	(576.9±206.6) / (7405.6±499.1)	(483.4±76.7) / (14552.8±2276.6)

Benzo[b+j+k]fluoranthene[d]	(842.9±286.1) / (31294.3±13800.0)	(1005.2±379.5) / (21435.3±9447.4)	(1262.6±479.9) / (16134.2±1197.3)	(1082.7±230.2) / (32505.3±6279.3)
Benzo[e]pyrene	(806.3±96.9) / (29518.2±9463.6)	(807.0±209.6) / (17211.1±6467.3)	(884.3±278.9) / (11557.3±1565.2)	(786.3±146.4) / (23656.9±4232.9)
Benzo[a]pyrene[d]	(294.9±81.7) / (10924.4±4441.6)	(432.8±158.6) / (9278.6±4116.1)	(508.1±168.5) / (6602.5±767.9)	(430.2±91.9) / (12960.0±2761.1)
Perylene	(58.8±19.5) / (2194.5±982.1)	(75.2±24.7) / (1608.8±674.1)	(92.7±24.2) / (1244.8±299.8)	(79.0±17.6) / (2343.3±262.8)
Indeno[1,2,3-cd]pyrene[d]	(181.7±82.2) / (6858.7±3639.0)	(242.5±97.3) / (5222.8±2463.5)	(297.8±123.7) / (3777.0±375.7)	(275.7±60.4) / (8289.3±1716.1)
Dibenzo[a,h]anthracene[d]	(177.3±44.9) / (6620.5±2778.2)	(213.9±73.8) / (4614.6±2049.4)	(236.2±75.1) / (3079.8±381.6)	(214.8±48.3) / (6482.3±1506.7)
Benzo[ghi]perylene[d]	(573.0±81.1) / (21227.3±7810.0)	(601.6±164.3) / (12900.1±5105.3)	(644.0±223.4) / (8337.2±939.3)	(601.5±112.2) / (18145.8±3563.7)
Coronene	(225.9±44.1) / (8495.9±3640.1)	(196.7±44.8) / (4240.8±1671.7)	(178.3±40.7) / (2516.2±1090.6)	(177.3±29.8) / (5311.0±670.7)
Summation of total U.S. EPA Group B2 PAHs (Σ_7PAHs)[d]	(3608.0±352.1) / (133672.2±14089.2)	(4545.2±507.3) / (97020.7±11276.0)	(5508.5±666.3) / (70619.4±7568.3)	(4699.9±496.2) / (141463.3±14894.1)
Total	(10069.2±1856.5) / (375722.3±150132.2)	(10956.8±3196.3) / (236462.7±99275.1)	(11346.5±2459.4) / (153730.1±39827.7)	(11978.5±1546.2) / (362095.3±59473.2)

[a] Particle-bound PAH concentrations (µg/g) were defined as PAH mass (µg) of $PM_{2.5}$ and were normalized by the unit mass (g) of $PM_{2.5}$ for samples A to D.

[b] Particle-bound PAH concentrations (µg/g) were defined as PAH mass (µg) of $PM_{2.5}$ and were normalized by the unit cubic meter of air (m^3) for samples A to D.

[c] B.D. = below detection limit.

[d] Indicated by U.S. EPA as probable human carcinogen.

*n = 3 for each type of coal.

Table 2 OPAH and AZA Concentrations*

Sample (µg/g $PM_{2.5}$)[a] / (ng/m^3 air)[b]		A	B	C	D
Azaarenes	Benzo[h]quinoline	(230.8±81.1) / (8951.6±4912.6)	(284.8±102.1) / (6400.3±3452.4)	(235.8±42.4) / (3619.2±2401.8)	(443.9±86.8) / (13638.6±4154.0)
	Acridine	(167.2±62.4) / (6569.7±3897.7)	(109.0±11.2) / (2346.7±861.5)	(134.3±24.0) / (1939.4±968.1)	(210.5±101.0) / (6436.8±3138.2)
	Carbazole	(492.5±191.8) / (18680.0±9474.4)	(443.9±147.2) / (9704.3±4523.8)	(558.4±157.1) / (7501.4±1907.8)	(663.0±115.5) / (20187.8±4846.3)
	Total	(890.5±326.5) / (34201.4±18019.0)	(837.7±256.7) / (18451.3±8764.9)	(928.5±136.5) / (13060.1±5062.2)	(1317.4±290.9) / (40263.2±11610.5)
Hydroxyl and Carboxyl-OPAHs	2-Naphthol	(297.6±163.1) / (12345.1±10082.7)	(274.6±66.5) / (6226.8±3511.5)	(267.2±293.5) / (5397.8±7591.5)	(497.2±199.1) / (15465.9±8166.0)
	1-Acenaphthenol	(44.5±3.4) / (1614.4±443.7)	(56.1±34.4) / (1059.2±305.9)	(81.5±9.8) / (1176.6±573.9)	(75.7±13.1) / (2310.0±631.5)
	2-Naphthoic acid	(31.9±6.9) / (1120.8±185.0)	(57.4±32.4) / (1105.9±566.0)	(32.3±5.8) / (495.3±335.5)	(51.8±5.1) / (1572.0±298.7)
	9-Hydroxyfluorene	(74.1±13.9) / (2651.7±658.5)	(84.8±34.3) / (1669.3±239.8)	(87.7±9.0) / (1311.4±777.8)	(153.1±44.2) / (4595.2±1215.7)
	Total	(448.2±146.7) / (17732.1±11150.8)	(472.9±20.5) / (10061.3±3258.2)	(468.7±301.0) / (8381.1±9272.6)	(777.8±238.8) / (23943.1±10000.4)
Carbonyl-OPAHs	1-Indanone	(2.5±1.9) / (99.7±74.5)	(4.5±1.9) / (87.9±9.6)	(12.0±5.8) / (147.6±19.3)	(10.8±2.4) / (332.2±102.3)
	1-Naphthaldehyde	(8.6±4.2) / (351.2±282.9)	(12.2±5.5) / (243.8±72.0)	(18.1±4.0) / (246.6±71.5)	(27.1±0.6) / (821.3±128.0)
	2-Biphenylcarboxaldehyde	(109.7±53.7) / (4493.5±3600.4)	(81.4±25.8) / (1860.3±1089.1)	(53.0±27.1) / (913.6±916.0)	(132.3±46.1) / (4137.5±1995.8)
	9-Fluorenone	(471.5±200.0) / (18165.8±10056.9)	(423.5±137.8) / (9487.4±4966.7)	(387.8±28.2) / (5809.2±3422.6)	(965.3±121.5) / (29496.3±7557.6)
	1,2-Acenaphthenequinone	(B.D.) / (B.D.)[c]	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)

9,10-Anthraquinone	(516.2±49.4) / (18953.6±6184.1)	(480.5±116.7) / (10622.3±4995.2)	(496.6±71.3) / (7104.8±3162.8)	(727.1±50.1) / (22035.8±3594.9)
4H-Cyclopenta[def]phenanthrene-4-one	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)
2-Methyl-9,10-anthraquinone	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)
Benzo[a]fluorenone	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)
7H-Benz[de]anthracene-7-one	(452.2±218.2) / (16984.3±9160.8)	(475.4±161.0) / (10420.5±4945.1)	(606.9±213.6) / (7807.8±650.7)	(710.7±90.6) / (21321.3±1788.9)
Benzo[a]anthracene-7,12-dione	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)
5,12-Naphthacenequinone	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)	(B.D.) / (B.D.)
6H-Benzo[cd]pyren-6-one	(139.6±87.5) / (5390.7±3527.4)	(193.5±88.8) / (4138.0±2064.7)	(254.2±108.1) / (3251.0±528.1)	(291.2±51.6) / (8682.1±671.3)
Total	(1700.4±557.7) / (64438.8±31014.0)	(1671.1±506.9) / (36860.2±17604.0)	(1828.6±297.3) / (25280.7±8279.4)	(2864.5±59.4) / (86826.4±12958.0)

[a] Particle-bound OPAH/AZA concentrations (μg/g) were defined as OPAH/AZA mass (μg) of $PM_{2.5}$ and normalized by the unit mass (g) of $PM_{2.5}$ for samples A to D.

[b] Particle-bound OPAH/AZA concentrations (μg/g) were defined as OPAH/AZA mass (μg) of $PM_{2.5}$ and normalized by the unit cubic meter of air (m^3) for samples A to D.

[c] B.D. = below detection limit.

*n = 3 for each type of coal.

Table 3 Exposure Conditions

Exposure Factors	Yunnan	National Average by Provinces
Average inhalation rate (IR) ($m^3 h^{-1}$)	0.645	0.654
Average duration in indoor ($h\ d^{-1}$)	5.0	2.0
Average indoor exposure frequency (d)	320	221
Average life expectancy (yr)	69.54	74.83
Average body weight (BW) (kg)	55.9	60.6
Average exposure duration (AT) (d)	25550	25550

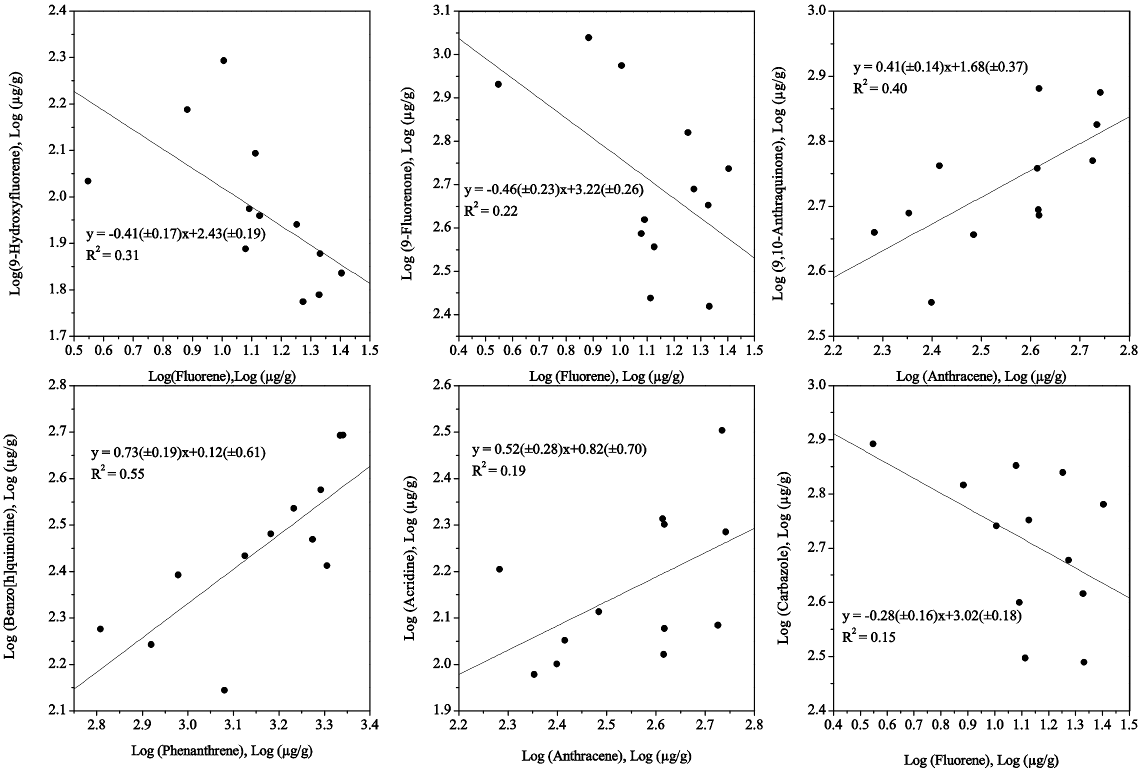

Figure 1 Relationships between log-transformed concentrations of OPAH or AZA compounds and their PAHs counterparts. The six pairs are 9-hydroxyfluorene/fluorene, 9-fluorenone/fluorene, 9,10-anthraquinone/anthracene, benzo[h]quinolone/phenanthrene, acridine/anthracene, and carbazole/fluorene. There were four types of coal with three replicates each (n = 12). Coefficients and standard errors were included in the regression equations.

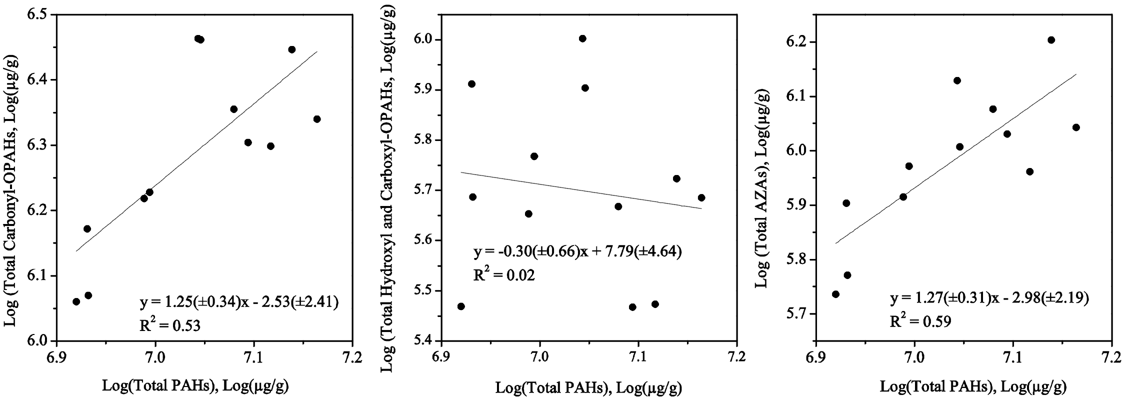

Figure 2 Relationships between log-transformed concentrations of Total Carbonyl-OPAHs, Hydroxyl and Carboxyl-OPAHs, or AZAs in relation to PAHs. There were four types of coal with 3 replicates each (n = 12). Coefficients and standard errors were included in the regression equations.

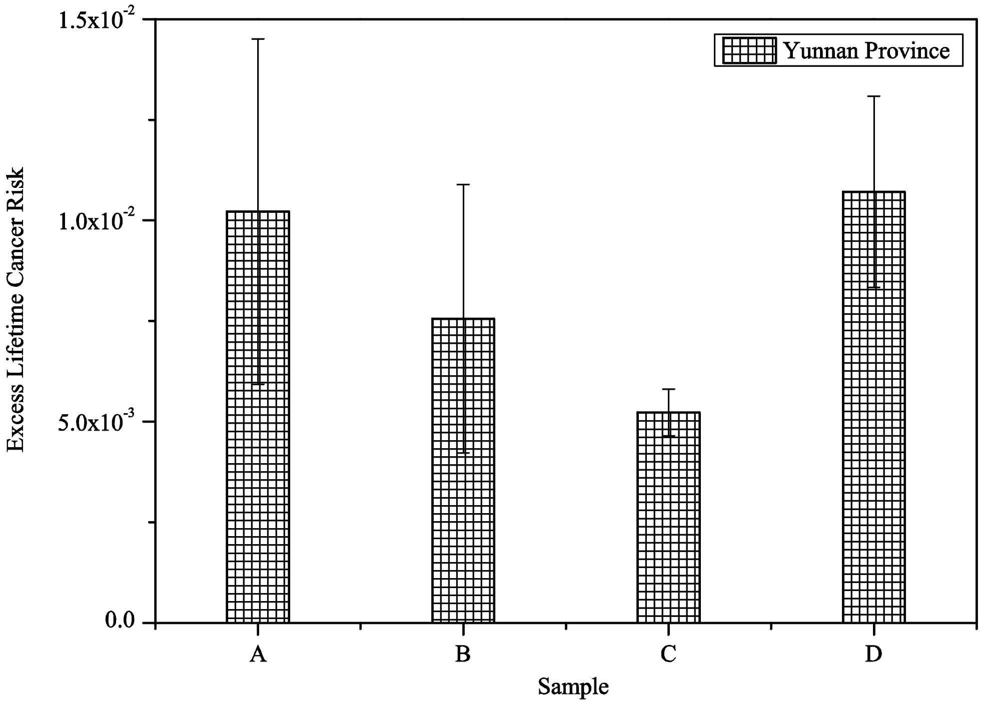

Figure 3 Excess cancer risk associated with inhalation of selected PAHs in coal emissions. Risk error bars represent minimum and maximum values calculated using the lowest and highest PEF values for PAHs, respectively.