



## Inhalation Risk Assessment of PAH Exposure Due to Combustion Aerosols Generated from Household Fuels

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### ABSTRACT

The typical mixtures of polycyclic aromatic hydrocarbons are established as lung carcinogens, but their exposure and associated risk from different household fuels are less known. Five commonly used household fuels namely firewood, coal, dung cake, kerosene, and liquefied petroleum gas (LPG) were tested for their size-fractionated polycyclic aromatic hydrocarbon (PAHs) emission. A total of sixteen PAHs were analyzed in size fractionated combustion aerosols using HPLC-UV technique. Single box modeling approach is applied to estimate time average B[a]P<sub>eq</sub> concentration of PAHs in indoor air during cooking time. Incremental lifetime cancer risk (ILCR) of PAH exposure during cooking time was calculated, which was found to have a positively skewed distribution for all types of fuels combustion. The uncertainty and variability of the predicted exposure risk were evaluated using Monte Carlo simulation. The 50<sup>th</sup> percentile of risk due to exposure of PAHs emissions during household use of fuels were found to be  $6.25 \times 10^{-5}$ ,  $2.99 \times 10^{-5}$ ,  $9.11 \times 10^{-5}$ ,  $1.14 \times 10^{-5}$ , and  $3.84 \times 10^{-6}$  for firewood, coal, dung cake, kerosene and LPG stove, respectively. The 50<sup>th</sup> percentile value of risk indicates the hazard associated with solid biomass combustion is higher than non-solid fuels; the risk associated with LPG stove use is found to be one order of magnitude less compared to other fuels.

**Keywords:** Box modeling; Carcinogens; HPLC-UV; ILCR; Monte Carlo simulation.

### INTRODUCTION

The indoor environment is an important determinant of health as humans typically spend 90% of their time indoors. Indoor air pollution attributed to solid fuel combustion products from heating and cooking is a major public health challenge mostly throughout the developing world. Solid fuel consists of coal and various forms of biomass, such as wood, crops, and dung. Biomass is most frequently utilized as domestic fuel in India and Africa, and coal fuel in China are typically used. The use of these fuels in poor and no-ventilation situations is worrisome as the attributed smoke has been associated with a variety of cancers, the most notable being lung cancer. Indoor emissions from household combustion of coal have been classified as a human carcinogen, whereas biomass combustion emissions have been classified as probably carcinogenic to humans (Hosgood and Lane, 2011; Kang *et al.*, 2011; Chen *et al.*, 2014).

PAHs are a large group of organic compounds comprised of two or more fused benzene rings arranged in different configurations. PAHs are widespread environmental

contaminants formed as a result of incomplete combustion of organic materials such as fossil fuels and biomass, are ubiquitous in the occupational and general environment. The occurrence of PAHs in ambient and indoor air is an increasing concern because of their carcinogenicity and mutagenicity. Although emissions and allowable concentrations of PAHs in air are now regulated, the health risk posed by PAH exposure suggests a continuing need for their control through air quality management. It has well known that several PAHs can produce cancers in experimental animals, and epidemiologic studies of exposed workers, especially in coke ovens and aluminum smelters, have shown clear excesses of lung cancer and highly suggestive excesses of bladder cancer (IARC 1985; Boffetta *et al.*, 1997; Liu *et al.*, 2001; Armstrong *et al.*, 2004; Kima *et al.*, 2013). PAHs such as benzo[a]pyrene (B[a]P), chrysene, indeno[1,2,3-c,d] pyrene, and benzo[b]fluoranthene have produced carcinogenic, mutagenic, and genotoxic effects in animal experiments (Thyssen *et al.*, 1981; Somers *et al.*, 2002; Chen and Liao 2006; Wei *et al.*, 2010). Humans are exposed to PAHs by inhalation, ingestion, and skin contact. The primary sources of exposure to PAHs for most of the population are inhalation of the compounds in tobacco smoke, wood smoke, and ambient air. No studies were located regarding death in humans following inhalation exposure of priority PAHs. However, a dose-related decrease in survival was noted in hamsters after 60 weeks of inhalation exposure to 46.5

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mg/m<sup>3</sup> benzo[a]pyrene for 109 weeks (Thyssen *et al.*, 1981). The interpretation of the studies on cancer risk from PAH exposure in humans is complicated by several factors. Skin, lungs, and bladder cancer in human have been associated with PAHs (IARC, 1983; Boffetta *et al.*, 1997).

A study of inhalation of ambient PAHs and lung cancer in china reveals, the average B[a]P equivalent concentration as 2.43 (1.29–4.50 as interquartile range) ng/m<sup>3</sup> (Zhang *et al.*, 2009). The population weighted B[a]P equivalent was 7.64 (interquartile range; 4.05–14.1) ng/m<sup>3</sup> because of the spatial overlap of the emission and population density. Taking into consideration the variation in exposure concentration, respiration rate, and susceptibility, the overall population attributable fraction (PAF) for lung cancer caused by inhalation exposure to PAHs was 1.6% (interquartile range, 0.91–2.6%), corresponding to an excess annual lung cancer incidence rate of  $0.65 \times 10^{-5}$  (Zhang *et al.*, 2009). PAH compositions are varies with particle size fraction (Evans *et al.*, 1990; Maruya *et al.*, 1996, Lee *et al.*, 2005) so it is important to characterize size fractionated PAHs in combustion aerosols generated from household fuels. In this study lifetime incremental cancer inhalation risk is calculated for PAHs exposure during cooking for different household fuels. Present work consists of experimental results, modeling of various parameter regarding emission, dilution and removal of PAHs from indoor environment due to indoor combustion, and calculation of risk associated with their exposure. Monte Carlo based approach is adopted for risk calculation.

## METHODS

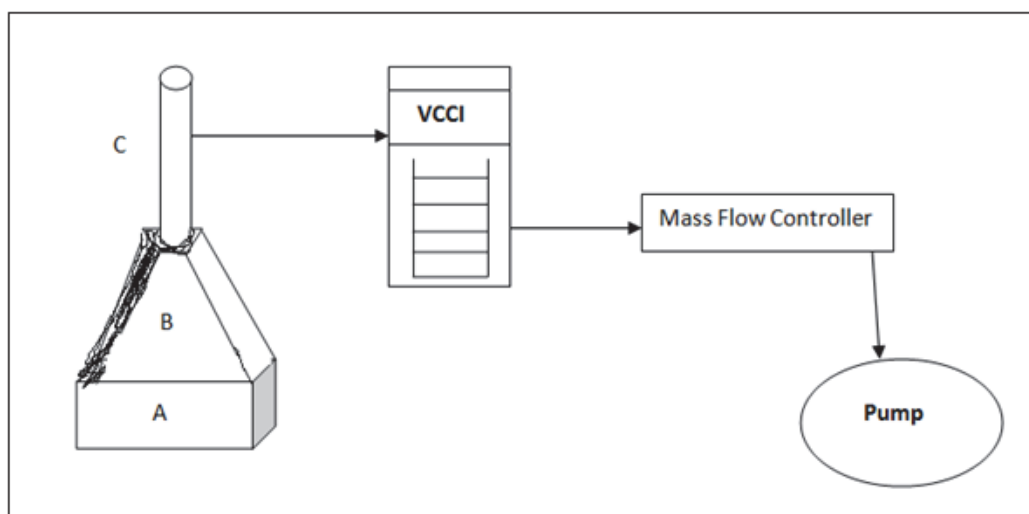
### Experimental Setup

Firewood, Coal, dung cake, LPG and kerosene (fuel and stove) fuels were purchased locally from Trombay, Mumbai. All solid samples were oven dried to reduce their moisture content at 60°C for one hour prior to experiment. The

stoves used for dung cake and firewood were traditional stoves locally named as "chulha" which were made of clay with a U shaped front opening and cylindrical pot hole. The analysis of coal smoke was carried out after emission of visible smoke ceased. Wick or pressure stoves for kerosene fuel and stoves with two gas burners for LPG fuel were used for experimental combustion. The experimental setup for determination of particle size distributions of combustion aerosols is shown in Fig. 1, it consists of a combustion chamber, mass flow controller, impaction stage and pump. The combustion chamber was designed in three parts A, B, and C. Sampling point was drawn from part C at a height of 0.90 m which is the breathing height in squatting posture. Sampling was forced through part C, for actual emission wall loss factor and sample collection fractions were evaluated. Wall loss factor come to picture due to deposition of combustion aerosol on inner walls of set up, while sample collection fraction was applied because chamber was open from upper side.

### Sample Collection and Analysis

Size fractionated combustion aerosols were collected in glass fiber filter paper using a variable configuration cascade impactor (VCCI). The size range ( $\mu\text{m}$ ) collected from the different stage of cascade impactor were ranges from  $> 21.3$  to  $< 0.10$   $\mu\text{m}$  aerodynamic diameter in total eleven bin. Aerosols consists of three modes: ultrafine mode, accumulation mode (which together with the ultrafine mode form the fine mode), and coarse mode. Ultrafine particles have an aerodynamic diameter ( $D_p$ ) of less than ca. 0.1  $\mu\text{m}$ ; they are emitted from combustion processes and are formed in the atmosphere by homogeneous nucleation. Accumulation mode particles are in the approximate range  $0.1 < D_p < 2$   $\mu\text{m}$  and are formed by coagulation and condensation. Coarse particles, those larger than ca. 2  $\mu\text{m}$ , are formed through mechanical attrition and disintegration processes, such as the wind-blown suspension of land surface dust and soil.



**Fig. 1.** Experimental setup for particle size distributions of combustion aerosols part (A) of setup design to contains stove, part (B) was provide a space to combustion aerosol get cool and dilute prior to analysis while part (C) contain sampling point. VCCI is stand for variable configuration cascade impactor.

Coarse particles are mainly deposited in the extrathoracic region, while some inhaled fine particles reach the alveolar region of the lung. In particular, ultrafine particles are deposited at much higher efficiency in the alveolar region than accumulation mode particles (Brown *et al.*, 2000; Kawanaka *et al.*, 2011). Then size fractionated combustion aerosols were analyzed using HPLC-UV technique for PAHs contents following the ultrasonic extraction. The identification and quantification of PAHs in size fractionated combustion aerosol was performed on a high performance liquid chromatography (HPLC) system (Shimadzu LC-10 AD) with UV-visible detector. The detector wavelength set at 254 nm. The analysis is carried out reverse phase and isocratic mode (Acetonitrile: H<sub>2</sub>O, 85:15), C-18 column (5 µm totally porous Octa decyl silane packing, Merck Germany), 250 mm × 4.6 mm i.d. with a C-18 guard column. Synthetic standard of PAHs were purchased from Supelco Belle-fonte USA. The method was optimized using synthetic standards. The recovery efficiencies were determined by spiking filter paper samples with PAH standard mixture. The mean recovery varied from 82.6% to 93.4%. The efficacy of the extraction process has been evaluated for NIST SRM-1649 urban dust sample and the results obtained were in good agreement with the certified values. For more details on the experiments and analysis were reported elsewhere (Tiwari *et al.*, 2013).

#### Calculation of B[a]P Equivalent Concentration

Potency equivalency factors (PEFs) for cancer induction relative to B[a]P were used to convert PAHs concentration in same scale of toxicity. PEFs have been derived only for PAHs with demonstrated carcinogenicity in bioassays. A much larger number of PAHs and PAH derivatives are considered mutagenic or genotoxic and may have limited evidence for carcinogenicity. New cancer bioassay data and possibly structure-activity analysis may indicate that additional PAHs are carcinogenic. Thus, additional PAHs may be identified as potential human carcinogens when such data become available. However, until that time the PEFs proposed for use in risk assessment were estimated only for PAHs currently classified as carcinogens (Collines *et al.*, 1998). Several hundred PAHs have been characterized;

the best known is Benzo[a] Pyrene, which is often, used as a marker of PAH exposure. The carcinogenic risk of a PAHs mixture is often expressed by its B[a]P equivalent concentration (B[a]P<sub>eq</sub>). The B[a]P<sub>eq</sub> of ambient PAHs (BEC) was calculated according to Eq. (1).

$$BEC = \sum_{i=1}^n TEF_i \times C_i \quad (1)$$

where  $C_i$  concentration of PAH congener  $i$ ;  $TEF_i$  the toxicity equivalency factor (TEF) of PAH congener (Table 1) (Nisbet and LaGoy, 1992; Petry *et al.*, 1996).

#### Time Average B[a]P<sub>eq</sub> Concentration and Exposure during Cooking Hours

Single compartment mass balance model was used to estimate the concentration of PAHs as a function of time during cooking in indoor environment. The change in PAHs concentration with time during cooking can be represented as Eq. (2). In the Eq. (2) right hand side first term represent the source term of PAHs by emission of household combustion and second term the loss of PAHs in indoor air due to air exchange.

$$\frac{dC}{dt} = \frac{Q}{V} - \alpha C \quad (2)$$

where  $V$  is volume of kitchen room,  $\alpha$  is air exchange rate ( $\text{min}^{-1}$ ).  $Q = E_f/E_d\beta$ , is emission rate ( $\mu\text{g}/\text{min}$ ), and  $\beta$  is stove energy output (MJ/min) of PAHs and different for each fuel.  $E_f$  is emission factor (mg of pollutant per kg of fuel),  $E_d$  is energy density of fuel (MJ per kg of fuel). Distributions of kitchen Volumes ( $V$ ) were based on measurement in Indian homes, which is a lognormal distribution with geometrical mean 30 m<sup>3</sup> and coefficient of variance (COV) 0.5 (Habib *et al.*, 2004). The air exchange rate  $\alpha$  ( $\text{min}^{-1}$ ) is also distributed log normally with a geometrical mean of 0.4  $\text{min}^{-1}$  and COV 0.6 (Johnson *et al.*, 2011). For volume of kitchen (m<sup>3</sup>) and air exchange rate ( $\text{min}^{-1}$ ) random numbers were generated for their use in calculation of  $C$ . Five hundred random numbers were generated using STATGRAPHICS Plus 4.0

**Table 1.** Mass concentration ( $\mu \pm 1\sigma$ ,  $n = 30$ ) of combustion aerosols generated in different size bins from tested household fuels.

size range (µm)	Firewood (µg/m <sup>3</sup> )	Dung cake (µg/m <sup>3</sup> )	Coal (µg/m <sup>3</sup> )	Kerosene (µg/m <sup>3</sup> )	LPG (µg/m <sup>3</sup> )
< 0.10	16.9 ± 1.5	14.33 ± 1.96	1.5 ± 0.2	1.32 ± 0.05	0.88 ± 0.11
0.30–0.10	8.22 ± 0.81	4.67 ± 0.34	1 ± 0.09	1.33 ± 0.12	0.95 ± 0.09
0.50–0.30	4 ± 0.31	3.17 ± 0.11	1.5 ± 0.13	1 ± 0.07	0.58 ± 0.13
0.71–0.50	2.89 ± 0.14	1.83 ± 0.18	1.17 ± 0.31	1.34 ± 0.17	0.72 ± 0.07
1.13–0.71	38.67 ± 1.67	81.5 ± 5.45	0.82 ± 0.07	1.33 ± 0.21	1.2 ± 0.12
2.23–1.13	32. ± 3.1	91.5 ± 4.96	0.83 ± 0.11	0.67 ± 0.14	1.23 ± 0.21
5.47–2.23	7.78 ± 0.68	2.33 ± 0.91	2.33 ± 0.23	1 ± 0.09	1.27 ± 0.12
7.38–5.47	3.56 ± 0.17	1.67 ± 0.17	7.5 ± 0.62	1.83 ± 0.12	1.6 ± 0.17
11.2–7.38	5.33 ± 0.95	2.65 ± 0.2	1.33 ± 0.09	0.51 ± 0.06	1.17 ± 0.19
15.1–11.2	9.56 ± 1.01	2.68 ± 0.25	1.17 ± 0.12	0.49 ± 0.09	1.42 ± 0.21
21.3–15.1	5.33 ± 0.76	2.5 ± 0.27	1.5 ± 0.17	0.48 ± 0.08	1.37 ± 0.31
> 21.3	21.33 ± 1.23	15.83 ± 1.77	9.17 ± 0.45	0.83 ± 0.12	0.65 ± 0.11

for both  $V$  and  $\alpha$ . By taking initial condition if  $t = 0$ ,  $C = 0$  the solution for Eq. (1) can give as following:

$$C_t = \frac{Q}{V\alpha}(1 - e^{-\alpha t}) \quad (3)$$

Above solution is based upon the following assumptions (i) there are no other sources of PAHs than stove; (ii) only ventilation is responsible for the decrease in concentration of PAHs with time; (iii) the room air is well mixed; (iv)  $E_F$  is constant during the course of combustion; (v) the fuel burn rate is constant; (vi) All the PAHs emitted is directly released into room (which is the case when the fuel is burnt in a cook stove without use of any flue, hood, or other venting device). Cooking time is another important parameter for calculating exposure and consequent risk of PAHs during cooking hours. The cooking duration ( $T_c$ ) can be given as following.

$$T_c = \frac{E_{DC} / 3}{\beta(\eta)} \quad (4)$$

where  $E_{DC}$  total daily energy required (MJ), and  $\eta$  is stove's thermal efficiency (Johnson et al., 2011).  $E_{dc}$  average energy required for cooking is 11 MJ (Habib et al., 2004),  $\beta$  is stove power calculated earlier and  $\eta$  is stove's thermal efficiency. Values of thermal efficiency ( $\eta$ ) for firewood, coal, dung cake, kerosene, and LPG stoves were taken as 0.25, 0.25, 0.22, 0.55, and 0.6 respectively (Sullivan and Barnes 2006). The time average PAHs concentration  $\bar{C}$  during combustion can be calculated from the equation:

$$\bar{C} = \frac{\int_0^{T_c} C_t dt}{T_c} = \frac{Q}{V\alpha} \left[ 1 + \frac{1}{\alpha T_c} (e^{-\alpha T_c} - 1) \right] \quad (5)$$

Daily inhalation of PAHs bounds with combustion aerosols during cooking was calculated using the time average concentration of PAHs.

$$I = IR \times T_c \times \bar{C} \quad (6)$$

$I$  is daily intake of PAHs (mg/day) through inhalation of  $B[a]P_{eq}$ ,  $IR$  inhalation rate ( $m^3/min$ ),  $T_c$  is cooking time (min/day),  $\bar{C}$  is average  $B[a]P_{eq}$  concentration in kitchen ( $mg/m^3$ ). A Monte Carlo approach was used to incorporate the variability in model parameters, resulting in a predicted distribution of PAHs concentration for different fuels emissions. Multiple simulations of cooking were run, with the inputs randomly selected from their respective probability distribution.

### Inhalation Risk Assessment

Monte Carlo Analysis (MCA) is the most widely used probabilistic method of risk assessment. The MCA technique treats any uncertain parameter as random variable that obeys a given probability distribution. This technique is

widely used for probabilistic uncertainty. In MCA computer simulations are used to combine multiple probability distributions associated with the risk equation. Thus we get a probabilistic distribution of risk. Inhalation risk due to exposure PAH emitted from different type of household cooking fuels is calculated using following equation.

$$Risk = \frac{I \times CSF \times EF \times ED}{BW \times AT} \quad (7)$$

CSF is cancer slop factor for  $B[a]P$  in  $(mg/kg/day)^{-1}$ , EF exposure frequency (day/year), ED exposure duration (year), BW is average body weight (kg), and AT is averaging time (day) (Chen et al., 2006; Xia et al., 2013). Cancer slop factor (CSF) for  $B[a]P$  is taken as log normally distributed with a geometrical mean  $3.14 (mg/kg/day)^{-1}$  and a geometrical standard deviation 1.80 (Collins et al., 1991). Exposure frequency (EF) was taken as lognormal distribution LN (252, 1.01) day/year, and the exposure duration (ED) was considered 50 year. Body weight was considered as lognormal distribution LN (49.78, 1.07) and considered for women, because of their high probability of exposure.

## RESULTS AND DISCUSSION

### Mass size Distribution of Aerosols Generated Household Fuels

The mass percentages emitted in different size fraction (respirable, fine and ultrafine) by the all tested fuels were shown in Fig. 2. Mass concentrations of combustion aerosols in individual size bin for all tested fuels represented in Table 1. A general trend was found that most of the mass accumulate in fine fraction size range 0.71 to 1.13  $\mu m$ . LPG and kerosene stove generated aerosols were found equally distributed in measured size fraction. For firewood, coal, dung cake, kerosene and LPG mass percentages of particles having aerodynamic diameter  $< 11.2 \mu m$  (respirable size) of the total suspended particulate were found to be 76%, 86%, 91%, 84% and 79% respectively which indicates that a major part of smoke mass emitted from the all fuels burning is capable of getting into respiratory tract. The mass percentages of fine particle ( $< 2.23 \mu m$  aerodynamic diameter), which are capable of deep pulmonary infiltration and alveolar deposition, for firewood, coal, dung cake, kerosene and LPG were found to be 66%, 77%, 88%, 57% and 48% of TSP respectively. The contribution of ultrafine particle i.e. particle having aerodynamic diameter  $< 0.1 \mu m$  to the total mass emitted from firewood, coal, dung cake, kerosene and LPG as 10%, 30%, 6%, 10% and 7.7% of the total smoke.

### PAHs Emission from Different Kinds of Stoves

Order of total PAHs emission in tested fuels was found as dung cake  $>$  firewood  $>$  coal  $>$  LPG  $>$  kerosene. Results are slightly different from a previous study by Raiyani et al. (1993b) found that PAH contents in total suspended particles (TSP) were the highest in wood smoke followed by cattle dung, kerosene, coal "sigri" smoke, and were the lowest in the TSP collected from the LPG-using houses. In ultrafine

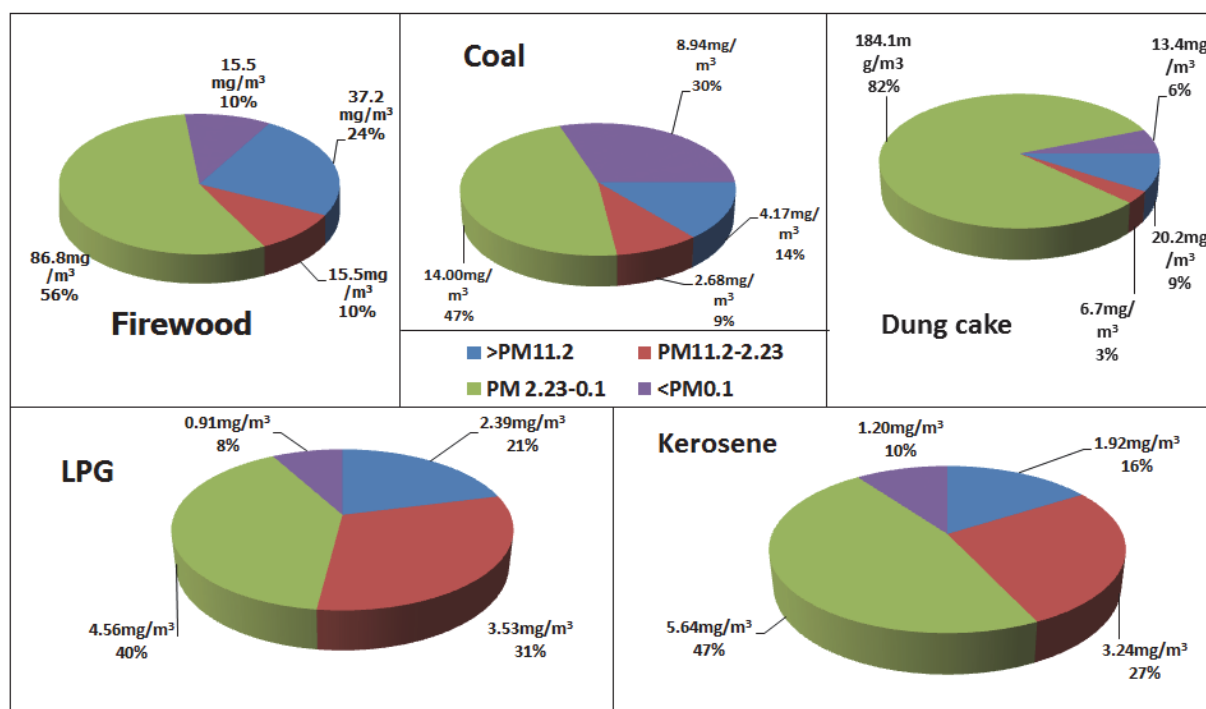


Fig. 2. Percentage distribution of particulate matter in different size fractions for commonly used household fuels.

fraction of the combustion aerosols, firewood generated a large PAHs mass percentage compared to other tested fuels in current study. LPG stove generated minimum PAHs in fine fraction as compared to other tested fuels. Firewood, coal and kerosene emitted most PAHs associated with fine fraction, while PAH content for LPG was found in all size fractions. Solid biomass was found to generate higher molecular weight (Five or more ring) PAHs more compared to non-solid fuels like Kerosene and LPG, while the later are mainly emitting two and three ring member PAHs. The formation of multiple rings PAHs was a function of combustion temperature which may vary with fuel type and methods of combustion. PAH markers for tested fuel can be determined from the data of PAHs contents in different size fraction. PAH emission factor ( $E_p$ ) were found highest (964  $\mu\text{g}/\text{kg}$ ) for dung cake, and lowest for LPG (484  $\mu\text{g}/\text{kg}$ ). For firewood, coal and kerosene burners emission factor mean values were found be 912  $\mu\text{g}/\text{kg}$ , 739  $\mu\text{g}/\text{kg}$  and 647  $\mu\text{g}/\text{kg}$  respectively.

#### ***B[a]P Equivalent Concentrations of PAHs***

Average B[a]P equivalent concentration at the sampling point of combustion chamber for firewood, coal, dung cake, kerosene and LPG stove were 327.15  $\mu\text{g}/\text{m}^3$ , 233.42  $\mu\text{g}/\text{m}^3$ , 306.83  $\mu\text{g}/\text{m}^3$ , 97.95  $\mu\text{g}/\text{m}^3$  and 69.99  $\mu\text{g}/\text{m}^3$  respectively. These values are calculated using potency equivalent factor (PEF) of PAHs relative to B[a]P by multiplying with their concentration. Table 2 shows the B[a]P equivalent concentrations of individual PAHs emitted during combustion of different type fuels at the sampling point.

#### ***Average B[a]P Equivalent Concentration***

Time average B[a]P equivalent concentration in kitchen

room was estimated using Eq. (5). The value of Q for Firewood, coal, dung cake, kerosene and LPG stove were found as 7.243  $\mu\text{g}/\text{min}$ , 3.976  $\mu\text{g}/\text{min}$ , 7.647  $\mu\text{g}/\text{min}$ , 2.697  $\mu\text{g}/\text{min}$  and 1.36  $\mu\text{g}/\text{min}$  respectively. The cooking times for firewood, coal, dung cake, kerosene stove and LPG stove were found 107 min, 96 min, 152 min, 54 min and 36min respectively. The 50<sup>th</sup> percentile value of B(a)P<sub>eq</sub> PAHs concentration for LPG was found to be lowest and for dung cake it was found maximum amongst all the tested domestic fuels. The order of 50<sup>th</sup> percentile B[a]P equivalent concentration was found as Dung Cake > Firewood > Coal > Kerosene > LPG.

#### ***Excess Life Time Inhalation Risk Due to Exposure of Combustion Generated PAHs***

Time average B[a]P equivalent PAHs concentration in different fueled kitchen during combustion hour as determined earlier was used for probabilistic inhalation risk assessment. Exposure of B[a]P equivalent PAHs per day (mg/d) it was found to be log normally distributed and its value in domestic environment for different fuel type are as discussed in earlier section of this manuscript. The 5<sup>th</sup>, 50<sup>th</sup>, and 95<sup>th</sup> percentile values of lifetime excess cancer risk to inhalation of combustion generated PAHs are represented in Table 3. The values of 50<sup>th</sup> percentile of risk distribution were  $6.25 \times 10^{-5}$ ,  $2.99 \times 10^{-5}$ ,  $9.11 \times 10^{-5}$ ,  $1.14 \times 10^{-5}$ , and  $3.84 \times 10^{-6}$  respectively for PAHs exposure during combustion for firewood, coal, dung cake, kerosene and LPG stove in Indian domestic environment. Box and whisker plots for inhalation incremental life time cancer risk due to exposure of PAHs emits from combustion of different household fuels as shown in Fig. 3. Predicted probability distributions of risk were found positively

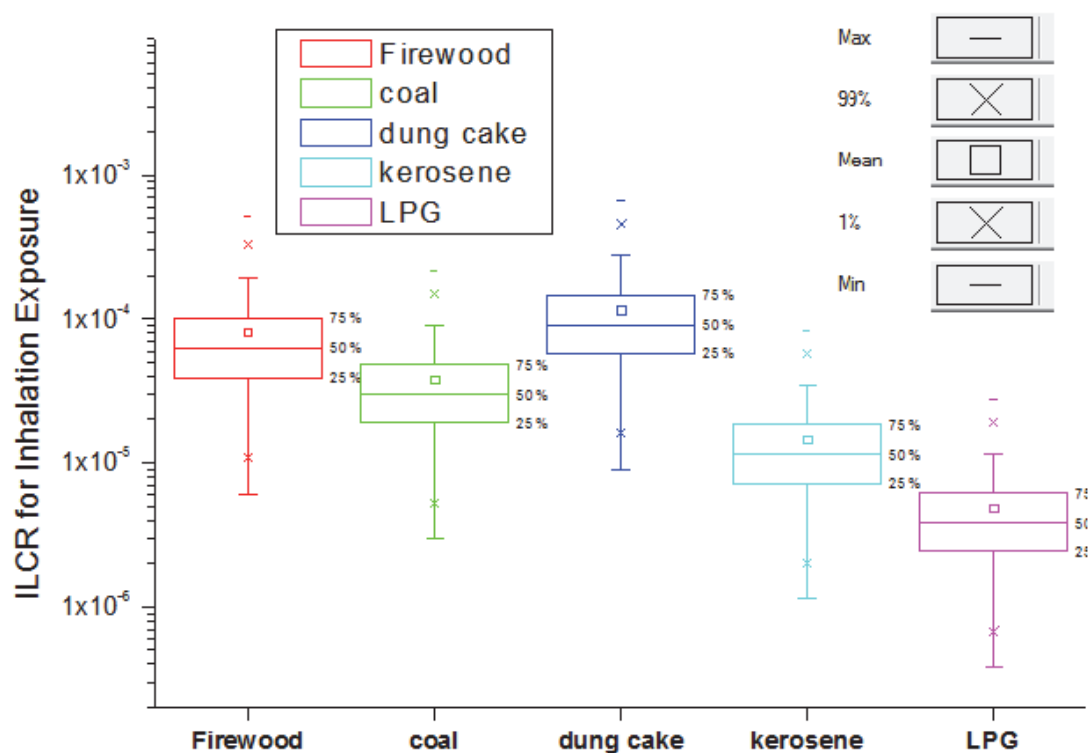
**Table 2.** Average B[a]P equivalent concentration ( $\mu\text{g}/\text{m}^3$ ) for individual PAHs emitted by combustion of different kind of fuels.

PAHs	TEF* (B(a)P <sub>eq</sub> )	Firewood (B(a)P <sub>eq</sub> ) ( $\mu\text{g}/\text{m}^3$ )	Coal (B(a)P <sub>eq</sub> ) ( $\mu\text{g}/\text{m}^3$ )	Dung cake (B(a)P <sub>eq</sub> ) ( $\mu\text{g}/\text{m}^3$ )	Kerosene (B(a)P <sub>eq</sub> ) ( $\mu\text{g}/\text{m}^3$ )	LPG (B(a)P <sub>eq</sub> ) ( $\mu\text{g}/\text{m}^3$ )
Naphthalene	0.001	0.33	0.91	0.34	0.15	0.14
Acenaphthylene	0.001	0.29	0.04	0.17	0.03	0.15
Fluorine	0.001	0.04	0.00	0.18	0.56	0.54
Acenaphthene	0.001	0.61	0.06	0.61	0.63	0.12
Phenanthrene	0.001	0.03	0.07	1.09	0.50	0.05
Anthracene	0.010	1.21	2.54	6.46	0.60	0.63
Fluoranthene	0.001	0.13	0.01	0.22	0.07	0.05
Pyrene	0.001	0.33	0.39	0.14	0.08	0.05
Chrysene + Benzo (a) Anthracene	0.100	2.36	1.80	15.98	5.78	6.37
Benzo (b) Fluoranthene	0.100	0.70	9.20	24.23	12.13	2.34
Benzo (k) Fluoranthene + Perylene	0.100	1.31	0.94	15.93	5.87	4.06
Benzo (a) Pyrene	1.000	299.70	172.09	231.61	70.76	52.59
Indeno (123,cd) Pyrene	0.100	2.14	34.86	5.57	0.33	1.74
Benzo (ghi) Perylene	0.010	17.98	10.52	4.29	0.46	1.16
SUM		327.15	233.42	306.83	97.95	69.99

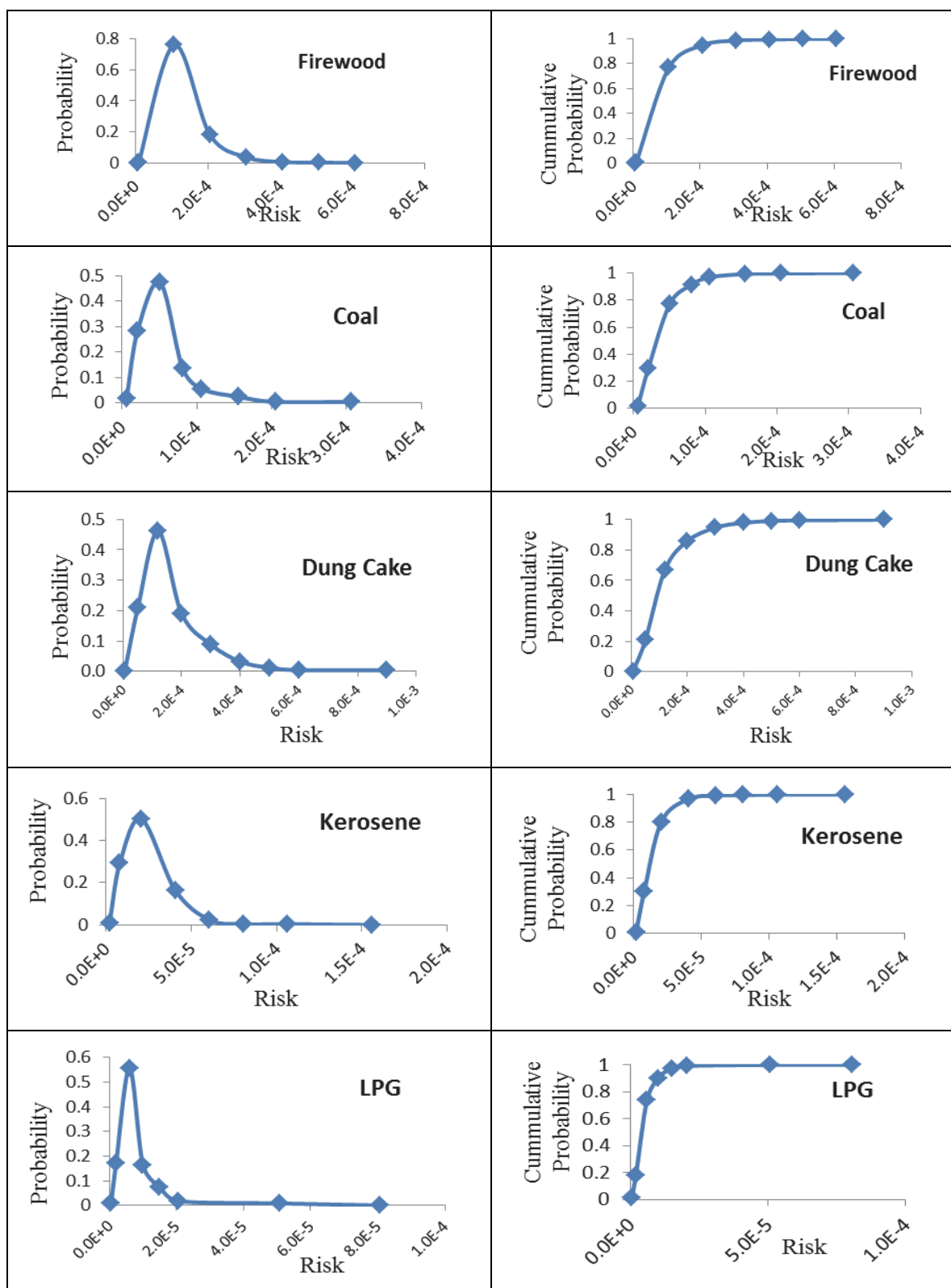
\* Toxicity equivalency factor (TEF).

**Table 3.** 5<sup>th</sup>, 50<sup>th</sup>, 95<sup>th</sup> percentile values of lifetime cancer risk due inhalation of combustion generated PAHs.

Fuel Type	5 <sup>th</sup> Percentile	50 <sup>th</sup> Percentile	95 <sup>th</sup> Percentile
Firewood	$1.85 \times 10^{-5}$	$6.26 \times 10^{-5}$	$2.10 \times 10^{-4}$
Coal	$9.05 \times 10^{-6}$	$2.99 \times 10^{-5}$	$9.86 \times 10^{-5}$
Dung Cake	$2.76 \times 10^{-5}$	$9.11 \times 10^{-5}$	$3.00 \times 10^{-4}$
Kerosene	$3.45 \times 10^{-6}$	$1.14 \times 10^{-5}$	$3.76 \times 10^{-5}$
LPG	$1.16 \times 10^{-6}$	$3.84 \times 10^{-6}$	$1.27 \times 10^{-5}$



**Fig. 3.** Box and whisker plots for incremental life time cancer risk due to inhalation exposure of PAHs emits from combustion of different household fuels.



**Fig. 4.** Predicted probability density functions and respective cumulative density function of total life time risk due to exposure of PAHs generated from combustion of different fuel types in Indian domestic environment.

skewed for all tested fuels. The probability density functions and cumulative density function of risk due to exposure of PAHs for all tested fuels were shown in Fig. 4.

## CONCLUSION

The major portion of aerosols emitted from household

combustion are likely to get deposited in human respiratory tract as mass percentages of particles having aerodynamic diameter < 11.2 µm (respirable size) of the total suspended particulate were found to be 76% to 91% for tested fuels. For coal generated combustion aerosol the contribution of ultrafine particle i.e. particle having aerodynamic diameter < 0.1 µm to the total mass emitted as a smoke was found 30% which is highest among all tested fuel, for others fuels this value below 10% of the total TSP. Order of total PAHs emission from tested fuels was found as Dung cake > Firewood > coal > LPG > kerosene. Firewood, coal and kerosene emitted maximum PAHs associated with fine fraction, while PAH content for LPG was found more or less same in all size fractions. Solid biomass generating higher molecular weight (Five or more ring) PAHs compared to non-solid fuel as Kerosene and LPG, while the later are mainly emitting two and three ring member PAHs. For solid biomass fuels cooking time values are low as compare to non-solid fuel because having less stove power and thermal efficiency. The 50<sup>th</sup> percentile value of risk indicate hazard associated with solid biomass combustion is higher than non-solid fuels, LPG stove risk is one order of magnitude less compare to other fuels. The results of risk assessment reveal solid biomass fuel as dung cake and fire wood combustion pose a great risk as compare to non-solid fuel as LPG and kerosene stove for in house cooking. The hazard associated with that may increase when the living and kitchen room is same and ventilation is very low, which was commonly observed in family of low income strata of society.

## REFERENCES

- Armstrong, B., Hutchinson, E., Unwin, J. and Fletcher T. (2004). Lung Cancer Risk after Exposure to Polycyclic Aromatic Hydrocarbons: A Review and Meta-Analysis. *Environ. Health Perspect.* 112: 970–978.
- Boffetta, P., Jourenkova, N. and Gustavsson, P. (1997). Cancer Risk from Occupational and Environmental Exposure to Polycyclic Aromatic Hydrocarbons. *Cancer Causes Control* 8: 444–472.
- Brown, L.M., Collings, N., Harrison, R.M., Maynard, A.D. and Maynard, R.L. (2000). *Ultrafine Particles in the Atmosphere*, Imperial College Press, London.
- Chen, S.C. and Liao, C.M. (2006). Health Risk Assessment on Human Exposed to Environmental Polycyclic Aromatic Hydrocarbons Pollution Sources. *Sci. Total Environ.* 366: 112–123.
- Chen, W., Han J, Qin, L., Furuuchi, M. and Mitsuhiko, H. (2014). The Emission Characteristics of PAHs during Coal and Sewage Sludge Co-Combustion in a Drop Tube Furnace. *Aerosol Air Qual. Res.* 14: 1160–1167, doi: 10.4209/aaqr.2013.06.0192.
- Collins, J.F., Brown, J.P., Dowson, S.V. and Marty, M.A. (1991). Risk Assessment for Benzo[a]pyrene. *Regul. Toxicol. Pharm.* 13: 170–184.
- Collins, J.F., Brown, J.P., Alexeeff, G.V. and Salmon, A.G. (1998). Potency Equivalency Factors for Some Polycyclic Aromatic Hydrocarbons and Polycyclic Aromatic Hydrocarbon Derivatives. *Regul. Toxicol. Pharm.* 28: 45–54.
- Evans, K.M., Gill, R.A. and Robotham, P.W.J. (1990). The PAH and Organic Content of Sediment Particle Size Fractions. *Water Air Soil Pollut.* 51: 13–31.
- Habib, G., Venkataraman, C., Shrivastava, M., Banerjee, R., Stehr, J.W. and Dickerson, R.R. (2004). New Methodology for Estimating Biofuel Consumption for Cooking: Atmospheric Emissions of Black Carbon and Sulfur Dioxide from India. *Global Biogeochem. Cycles* 18, doi: 10.1029/2003GB002157.
- Hosgood, H.D. and Lan, Q. (2011) Indoor Air Pollution Attributed to Solid Fuel Use for Heating and Cooking and Cancer Risk, In *Encyclopedia of Environmental Health* p. 198–200.
- IARC (1983). International Agency for Research on Cancer. Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data. Lyon, France: *Monogr Eval Carcinog Risk Chem Humans*, Vol. 32.
- IARC (1985). Polynuclear Aromatic Compounds. Part 4: Bitumens, Coal-Tars and Derived Products, Shale Oils and Soots. *IARC Monogr Eval Carcinog Risks Hum* 35: 1–271.
- Johnson, M., Lam, N., Brant, S., Gray, C. and Pennise, D. (2011). Modeling Indoor Air Pollution from Cookstove Emissions in Developing Countries Using a Monte Carlo Single-box Model. *Atmos. Environ.* 45: 3237–3243.
- Kang, Y., Cheung, K.C. and Wong, M.H. (2011). Mutagenicity, Genotoxicity and Carcinogenic Risk Assessment of Indoor Dust from Three Major Cities around the Pearl River Delta. *Environ. Int.* 37: 637–643.
- Kawanaka, Y., Matsumoto, E., Sakamoto, K. and Yun, S.J. (2011). Estimation of the Contribution of Ultrafine Particles to Lung Deposition of Particle-bound Mutagens in the Atmosphere. *Sci. Total Environ.* 409: 1033–1038.
- Kima, K.H., Jahan, S.A., Kabir, E. and Brown, R.J.C. (2013). A Review of Airborne Polycyclic Aromatic Hydrocarbons (PAHs) and Their Human Health Effects. *Environ. Int.* 60: 71–80.
- Lee, B.C., Shimizu, Y., Matsuda, T. and Matsui S. (2005). Characterization of Polycyclic Aromatic Hydrocarbons (PAHs) in Different Size Fractions in Deposited Road Particles (DRPs) from Lake Biwa Area, Japan. *Environ. Sci. Technol.* 39: 7402–7409.
- Liu, Y., Zhu, L. and Shen X. (2001). Polycyclic Aromatic Hydrocarbons (PAHs) in Indoor and Outdoor Air of Hangzhou, China. *Environ. Sci. Technol.* 35: 840–844.
- Maruya, K., Risebrough, R.W. and Horne, A.J. (1996). Partitioning of Polynuclear Aromatic Hydrocarbons between Sediments from San Francisco Bay and Their Porewaters. *Environ. Sci. Technol.* 30: 2942–2949.
- Nisbet, C. and LaGoy, P. (1992). Toxic Equivalency Factors (TEFs) for Polycyclic Aromatic Hydrocarbons (PAHs). *Regul. Toxicol. Pharm.* 16: 290–300.
- Petry, T., Schmid, P. and Schlatter, C. (1996). The Use of Toxic Equivalency Factors in Assessing Occupational and Environmental Health Risk Associated with Exposure to Airborne Mixtures of Polycyclic Aromatic Hydrocarbons (Pahs). *Chemosphere* 32: 639–648.



- Raiyani, C.V., Shah, S.H., Desai, N.M., Venkaiah, K., Patel, J.S., Parikh, D.J. and Kashyap, S.K. (1993b). Characterization and Problems of Indoor Air Pollution Due to Cooking Stove Smoke. *Atmos. Environ.* 27A: 1643–1655.
- Somers, C.M., Yauk, C.L., White, P.A., Parfett, C.L.J. and Quinn, J.S. (2002). Air Pollution Induces Heritable DNA Mutations. *Proc. Nat. Acad. Sci. U.S.A.* 99: 15904–15907.
- Sullivan, K. and Barnes, D. (2006). Energy Policies and Multitopic Household Surveys: Guidelines for Questionnaire Design in Living Standards Measurement Studies (Energy and Mining Sector Board Paper No. 17), Washington DC, World Bank.
- Thyssen, J., Althoff, J., Kimmerle, G. and Mohr, U. (1981). Inhalation Studies with Benzo[a]pyrene in Syrian Golden Hamsters. *J. Natl. Cancer Inst.* 66: 575–577.
- Tiwari, M., Sahu, S.K., Bhangare, R.C., Ajmal, P.Y. and Pandit, G.G. (2013). Estimation of Polycyclic Aromatic Hydrocarbons Associated with Size Segregated Combustion Aerosols Generated from Household Fuels. *Microchem. J.* 106: 79–86.
- Wei, Y., Han, I., Hu, M., Shao, M., Zhang, J.F. and Tang, X. (2010). Personal Exposure to Particulate PAHs and Anthraquinone and Oxidative DNA Damages in Humans. *Chemosphere* 81:1280–1285.
- Xia, Z., Duan, X., Tao, S., Qiu, W., Liu, D., Wang, Y., Wei, S., Wang, B., Jiang, Q., Lu, B., Song, Y., Hu, X. (2013). Pollution Level, Inhalation Exposure and Lung Cancer Risk of Ambient Atmospheric Polycyclic Aromatic Hydrocarbons (PAHs) in Taiyuan, China. *Environ. Pollut.* 173: 150–156.
- Zhang, Y., Tao, S., Huizhong, S. and Ma, J. (2009). Inhalation Exposure to Ambient Polycyclic Aromatic Hydrocarbons and Lung Cancer Risk of Chinese Population. *PNAS* 50: 21063–21067.

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