IARJSET
ISSN (Online) 2393-8021
ISSN (Print) 2394-1588
International Advanced Research Journal in Science, Engineering and Technology
ISO 3297:2007 Certified
Vol. 3, Issue 9, September 2016

Ambient Polycyclic Aromatic Hydrocarbons and its Carcinogenic and Mutagenic Risk – A Review

Papiya Mandal[1, 2],R. Sarkar[2], A. Mandal[2]

CSIR-National Environmental Engineering Research Institute, Zonal Laboratory, Delhi, India[1]

Department of Civil Engineering, Delhi Technological University, Delhi, India[2]

Abstract: Emission of atmospheric polycyclic aromatic hydrocarbons is gaining considerable importance in present-day scenario, as they are ubiquitous and some of them are having carcinogenic and mutagenic potency to human's health. Though PAH have few natural sources like forest fires, volcanic eruption etc. but major emission of PAHs are directly linked with urbanization, industrialization and vehicular emission. Both rural and urban environment, PAHs has significant contribution to contaminate the ambient air. Recent interest has centered to identify and quantify of PAHs in soil, water and air environment, identify their emission sources through various approaches and to evaluate their carcinogenicity and mutagenicity to human's health. The approaches distinguish anthropogenic multiple emission sources of PAHs like from petroleum combustion, diesel combustion, coal combustion industrial emission and biomass burning. This paper deals with concentrations of ambient PAHs, their emission sources and total carcinogenic and mutagenic potential risk to human's health.

Keywords: PAHs diagnostic ratios; Carcinogenicity; Mutagenicity; Air toxic.

I.INTRODUCTION

Polycyclic aromatic hydrocarbon (PAHs) are the ubiquitous pollutants in both rural and urban atmosphere, containing atleast two or more fused aromatic (benzene) rings with different arrangements and present in various environments such as soil, water, sediment and air. The term "PAH" refers to class of organic compounds consisting only carbon and hydrogen atoms.

In general PAHs are formed through pyrolysis process and subsequently recombined with other organic molecules and formed the complex compound. PAHs emissions in the atmosphere are in the form of either gaseous or particulate phases. They are highly lipophilic and mostly soluble in organic solvents. They also manifest various functions in the environment such as light sensitivity, heat resistance, conductivity, emittability, corrosion resistance, and physiological action. United States of Protection Agency (USEPA) has classified sixteen PAHs as carcinogenic. Incomplete combustion and pyrolysis of organic material influences the emission of PAHs in the gaseous form and settles on either in particulates or in soils or sediments after condensation [1&2]. Though PAH has natural and anthropogenic sources but emission of PAHs are almost anthropogenic in origin [3].

PAHs reacts with atmospheric trace gases eg., ozone, hydroxyl radicals, nitrogen dioxide and sulphur dioxide, nitro and dinitro-PAHs, and sulfonic acids [4]. The emission sources of individual PAHs are mostly from industrial emission, vehicular emission and biomass burning. The multiple emission sources of PAHs are broadly categorized into five major emission sources eg., natural, domestic, mobile, industrial and agricultural sources. The occurrence of PAHs in the atmosphere and their carcinogenicity and mutagenicity potential is of greatconcern, especially in the urban atmosphere as people are exposed to PAHs by various means. Mostly people are exposed to PAHs in both ways either inhalation of contaminated toxic air or consumption of contaminated food and water. The contaminated foods have more route of exposure of PAHs as compared to inhalation of contaminated air. Several epidemiological studies reported that complex mixture of PAHs with different molecular weights is associated with adverse health risks and potential carcinogens and mutagens [5]. It was reported by WHO, 2002, [6] reported that approximately 75% people in China, India and South East Asia, and 50–75% people in various parts of South America and Africa use solid fuels (wood, dried animal-dung-cake and crop waste) for daily cooking. Air quality guidelines for Europe country estimated that the unit risk is $9x10^{-5}$ would theoretically lead to one extra cancer case in 1 lakh exposed individuals [7].

It was estimated that Indo-Gangetic Plains alone contributes 30.21% PAHs emissions of total PAH emissions from India [8]. The annual PAHs emissions of India are estimated to be 90 Gg per year [9]. The congested traffic movement at urban areas promotes the emissions of PAHs in the urban air. Other major sources of PAH emission are from vehicle tire abrasion, asphalted surfaces and brake lining (Rogge et al. 1993a) [10].

II.FORMATION OF ATMOSPHERIC PAHs

Atmospheric PAHs are formed either due to incomplete combustion (pyrolysis) or high temperature pyrolytic process during anthropogenic activities like combustion of fossil fuels/ combustion of natural gas/ processing of coal

DOI 10.17148/IARJSET.2016.3937

and crude oil/ combustion of refuse material /cooking and tobacco smoking/ biomass burning etc. It may also produce as a result of natural processes such as carbonisation (pyrosynthesis). PAHs are likely to form in the atmosphere by two main mechanisms like pyrolysis and pyro-synthesis. Low hydrocarbons form PAHs by pyro-synthesis process. When temperature increased 5000C or greater than 5000C, carbon-hydrogen and carbon-carbon bonds are broken and free radicals are released in the atmosphere. These free radicals are further combined with acetylene, which condenses into aromatic ring [11]. These rings have the resistance to thermal degradation. The tendency of the hydrocarbon is to form PAH structure by the process of pyro-synthesis in the following order: aromatics > cycloolefins > Olefins >paraffins [12]. The reaction of PAHs with other atmospheric trace gases viz., NOx, SO_2, O_2 etc. may form hetero-PAHs and nitro-PAHs. benzo[a]pyrene (BaP) is recognized as most carcinogenic PAHs. Sometimes,carcinogenicity and mutagenicity of many of these hetero-PAHs compounds is greater than their parent compounds, recommended by Central Pollution Control Board [13]. Fig.1illustrates the carbonisation process of PAHs starting with ethane.

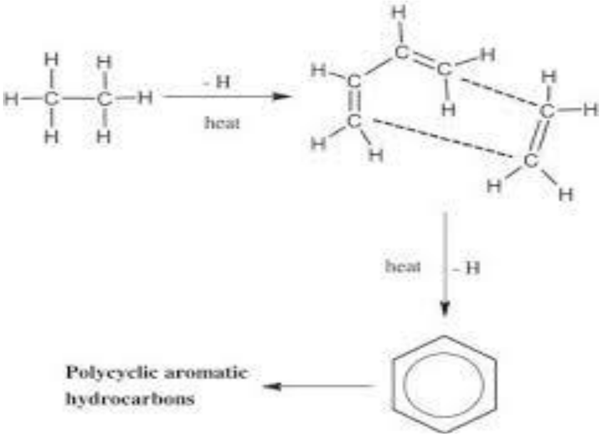

Fig. 1: Carbonisation process of PAHs starting with ethane

Atmospheric PAHs are formed during the combustion process by three possible mechanisms like slow Diels-Alder condensations, rapid radical reactions and ionic reaction mechanism [14]. For inter combustion engine, rapid radical reactions is the most favorable reaction. In this reaction process, gaseous hydrocarbon radicals are rearranged quickly to form of PAHs and lower molecular PAHs are in a state, to form higher molecular PAHs [15]. It was also reported by the researcher, that type of fuel, atmospheric temperature and presence of oxygen are also plays the significant role for formation of various types of PAHs during combustion activity [16].

III.COMMON ATMOSPHERIC PAHs & THEIR CHARACTERISTICS

The individual PAHs,physical and chemical propertiesdepend on their molecular weight. In general, PAHs have maximum melting and boiling temperature for solid, low vapor pressure, and low aqueous solubility (mg/l at 25^{0}C). The solubility of PAHs decreases with increasing molecular weight, but oxidation and reduction resistance increases. So, PAHs has different behavior and properties in the different environment, and they are mostly effective on biological systems. Low-molecular-weight PAHs (two and three rings) are weaker carcinogenic and mutagenic as compared to multi-ringed PAHs (four rings or more) and potential to humans health risk. Only two benzene rings are fused together inNaphthalene ($C_{10}H_8$), has the lowest molecular weight(MW=128g)among all PAHs. Whereas the maximum seven benzene rings are fused together in Coronene ($C_{24}H_{12}$), has the highest molecular weight (MW=300g) of all PAHs. The benzene rings of the environmentally significant PAHs vary from two to seven. The common atmospheric PAHs and their characteristics molecular formula, molecular weight, presence of benzene rings, water solubility, classification as per International Agency for Research on Cancer (IARC) and their phase distribution in the ambient air is shown in Table 1.

TABLE 1Common atmospheric PAHs and their characteristics

Particulate PAHs	Molecular Formula	Molecular weight (g)	Benzene Rings	Water Solubility mg/l at 25^{0}C	Classification as per IARC* (1983,1987)	Particle/ Gas Phase Distribution
Naphthalene (NP)	$C_{10}H_8$	128	2	31	NE	Gas Phase
Acenaphthylene (ACY)	$C_{10}H_8$	128	2	16	NE	Gas Phase
Acenaphthene (ACE)	$C_{10}H_8$	128	2	3.8	NE	Gas Phase
Phenanthrene (PHE)	$C_{14}H_{10}$	178	3	1.1	Group 3	Particle Gas Phase
Anthracene (ANT)	$C_{14}H_{10}$	178	3	0.04	Group3	Particle Gas Phase
Fluoranthene (FLA)	$C_{16}H_{10}$	202	4	0.2	Group3	Particle Gas Phase
Pyrene (PYR)	$C_{16}H_{10}$	202	4	0.13	Group3	Particle Gas Phase
Benzo(a)anthracene (BaA)	$C_{18}H_{12}$	228	4	0.011	Group2B	Particle Phase

Chrysene (CHR)	$C_{18}H_{12}$	228	4	0.0019	Group2B	Particle Phase
Benzo(b)fluoranthene (BbF)	$C_{20}H_{12}$	252	5	0.0015	Group2B	Particle Phase
Benzo(k)fluoranthene (BkF)	$C_{20}H_{12}$	252	5	0.0008	Group2B	Particle Phase
Benzo(j)fluoranthene (BkF)	$C_{20}H_{12}$	252	5	0.0008	Group2B	Particle Phase
Benzo(e)pyrene (BaP)	$C_{20}H_{12}$	252	5	0.0015	Group1	Particle Phase
Benzo(a)pyrene (BaP)	$C_{20}H_{12}$	252	5	0.0015	Group1	Particle Phase
Benzo(ghi)pyrelene (BghiP)	$C_{22}H_{12}$	276	6	0.00015	Group3	Particle Phase
Dibenz(ah)anthracene (DahA)	$C_{22}H_{14}$	278	5	0.0005	Group2A	Particle Phase
Indo(123-cd)pyrene (IcdP)	$C_{22}H_{12}$	276	6	0.00019	Group2B	Particle Phase
Coronene	$C_{24}H_{12}$	300	7	0.00014	Group3	Particle Phase

Group 1	Carcinogenic to humans
Group 2A	Probably carcinogenic to humans
Group 2B	Possibly carcinogenic to humans
Group 3	Not classifiable as its carcinogenicity to humans
Group 4	Probably not carcinogenic to humans
NE	Not Evaluated

IARC*International Agency for Research on Cancer

IV.PREDOMINANT PAH SOURCE PROFILE/ MARKERS

PAHs are well-known air toxic and mostly identified as indicator for various emission sources in both rural and urban environment. They are originated from anthropogenic sources like mobile sources (e.g., cars, trucks, buses) and stationary sources (e.g., refineries, petrochemical industries, power plants, steel plants etc.), biomass burning as well as from indoor environment (e.g., cooking, smoking, construction materials, cleaning products). The natural sources include only volcanic eruptions and forest fires.

The made global emission inventory of 16 USEPA approved PAHs and estimated that total emission of PAHs is 520000 tonnes per year [9]. The annual emission of PAHs per year,only from Asian countries is 290000 tonnes, which is approximately 50% of the total global emission of ambient PAHs. The emission of PAHs in the Asian country like China and India are 144000 and 90000 tonnes per year respectively. The emission of PAHs from USA is 32000 tonnes per year, the third largest emitter of PAHs. The major source emission sources of USEPA identified PAHs in global, China, India and USA is shown in Table 2.

TABLE 2Major emission sources of USEPA identified PAHs in global, China, India and USA

Source	Global	China	India	USA
Biofuel	56.70%	66.4%	92.5%	9.1%
Wild fire	17.00%	0.0%	0.0%	3.3%
Consumer product use	6.90%	0.9%	0.6%	35.1%
Traffic oil	4.80%	2.0%	Insignificant	23.0%
Domestic coal	3.70%	10.7%	1.30%	Insignificant
Coke production	3.60%	14.4%	Insignificant	Insignificant
Petroleum refining	2.40%	1.0%	Insignificant	8.70%
Waste incineration	1.90%	Insignificant	Insignificant	9.50%
Aluminum electrolysis	1.40%	Insignificant	Insignificant	1.90%
Open straw burning	Insignificant	2.0%	3.20%	Insignificant
Gasoline distribution	Insignificant	Insignificant	Insignificant	3.00%
Aerospace industry	Insignificant	Insignificant	Insignificant	2.50%
Other	1.50%	-	2.70%	3.90%
Tonnes in thousands	**530**	**114**	**90**	**32**

Source: Zang and Tao et al.2009

Benzo(a)pyrene (BaP) is considered as most carcinogenic PAHs and many countries have included BaP in their hazardous pollution list, as well in their regulatory standards. European Union Directive (EUD), 2004 has proposed average target value of BaP is one ng/m^{3}in PM$_{10}$ (diameter of less than 10µg/m^3) size of particulate matter [17]. For Germany, annual average target value of BaP is 10 ng/m^3. Country like Australia, and India, annual average target value of BaP is one ng/m^3 only. Country like Belgium, and Netherland, annual average target value of BaP is reduced by 50% ie 0.5 ng/m^3. For country Croatia, France and Sweden the set target value is more stringent (0.1 ng/m^3) only.

V. IDENTIFICATION OF SOURCES OF PAHs

The emission of PAHs from a particular source depends on the processes involved for production of materials [18]. During low temperature activities like biomass burning, PAHs having low molecular weight are formed whereas PAHs having maximum molecular weight are formed in PAHs having maximum molecular weight are formed in theatmosphere during high temperature activities like combustion of fuels in engines, incinerations etc. [19]. PAHs diagnostic ratios are widely used to identify anthropogenic sources of PAHs emission. The ambient PAH concentrations and their diagnostic ratios indicate the different sources of PAHs in the environment [20]. The diagnostic PAHs ratios calculated for each hypothetical source are not definitive:for example [18] reported that BaA/ (BaA+CHR)= 0.3 to 0.6 indicates the emission of PAHs from cement industry. However similar many sources like FLA/(FLA+PYR)= 0.4 to 0.5 indicates the emission of PAHs from several industries like cement, metal manufacturing, fertilizer production, diesel combustion and sometimes from road dusts also [21] BaA/(BaA+ CHR) ratio greater than 0.35 is widely accepted to indicate the contribution of biomass burning [22] and vehicular emissions [23]. The identification of ambient PAH an emission sources using diagnostic ratiois shown in Table 3.

TABLE 3Identification of ambient PAH emission sources using diagnostic ratios

Diagnostic ratio	Value range	Sources	References
FL/(FL + PYR)	<0.5	Petrol emissions	[24]
	>0.5	Diesel emissions	
FL/PYR	0.6	Vehicular emissions	[25]
FL/BeP	3.5±0.5	Automobile exhaust	[26]
ANT/(ANT+PHE)	<0.1	Petrogenic	[27]
	>0.1	Pyrogenic	
FLA/(FLA+PYR)	<0.4	Petrogenic	[28]
	0.4 - 0.5	Fossil fuel combustion	
	>0.5	Grass, wood, coal combustion	
BaA/(BaA+ CHR)	0.2 - 0.35	Coal combustion	[23]
	>0.35	Vehicular emissions	
	<0.2	Petrogenic	
BaP/(BaP + BeP)	>0.35	Combustion	[29]
	0.5	Fresh particles	
	<0.5	Photolysis (ageing of particles)	
BaP/(BaP+CHR)	0.5	Diesel emission	[30] ,[20]
IcdP/(IcdP+BghiP)	<0.2	Petrogenic	[31]
	0.2 - 0.5	Petroleum combustion	
	>0.5	Grass, wood andcoal combustion	
IcdP/BghiP	<0.4	Gasoline	[32]
BbF/BkF	>0.5	Diesel emissions	[33], [3]
BaP/BghiP	<0.6	Non-traffic emissions	[34]
	>0.6	Traffic emissions	

It was suggested that the ratio if FL/(FL+PYR) ratio is less than 0.5, indicates the petrol emission, whereas if the ratio is greater than 0.5, indicates the emission of PAHs from diesel sources[11]. Similarly the ratio of IcdP/ (IcdP+BghiP) equal to 0.62, indicates the emission of PAHs from wood combustion [35] and theratio within the range of 0.35 to 0.70, indicating diesel emission [36]. It was also indicated that if the ratio of IcdP/ (IcdP+BghiP) is less than 0.2, indicates petrogenic sources, and the ratio varies between 0.2-0.5, indicates combustion of petroleum products and if the ratio greater than 0.5 indicates combustion of biomass burning like grass wood and coal [31]. The ratios are very similar, but the researchers hadreported the different types of source [33]. If the ratio of BbF/BkF is greater than 0.5, indicates the emission from diesel source[3]. The ratio of BaP/(BaP+CHR) is equal to 0.5, indicates the diesel emission [30] and [20]. The ratio ANT/(ANT+PHE) are the indicator of

DOI 10.17148/IARJSET.2016.3937

petrogenic sources. If the ratio is higher than 0.10 indicates petrogenic sources with lubricant oils and fossil fuels and the ratio less than 0.10 are typically associated pyrogenic sources ie., combustion of wood, coal, coal tar etc. [27]. ANT and PHE have low molecular weight (MW= 178) and present in the atmosphere mostly in the gas phase. They are most suitable for understanding of photolysis process in the gaseous phase [23]. Similarly the ratios of few particulate PAHs like BaP and BbF, BbK and BkF are widely used to understand the influence of photo processes of the particulate phase [21]. BaP is photo degraded faster as compared to its isomer BeP and adsorbed on PM_{10} size of particulate matter was reported by [36]. The ratio of BaP/(BaP+BeP) is photosensitive, and often used as the indicator of atmospheric particle ageing and the photodegradation of gaseous and particulate PAHs [21]. During the large number of daylight hours, BaP degradation is comparatively faster in the ambient air [37] and it shows the distinct diurnal variations [38]. Sometimes, it is difficult to differentiate the individual PAHs emission sources, according to the values of diagnostics ratios of PAHs.

VI.THE SOURCE OF INDIVIDUAL AMBIENT PAHs (RELATIVE TO BaP = 1.0)

The major twelve types of ambient PAHs and their emissions from various sources like point source, near mobile source, home heating and transport source (relative to benzo[a]pyrene BaP = 1.0) was reported by WHO 1998 is shown in Table 4.

TABLE 4Thecommon source of individual ambient PAHs (Relative to BaP = 1.0)

Types of PAHs	Point source	Near mobile source	Home heating	Transport	Geometric mean
Anthracene	5.5	7.6	1	1.8	2.9
Phenanthrene	38	200	39	43	60
Fluoranthene	14	48	12	13	18
Pyrene	9.3	28	11	7.1	12
Benz[a]anthracene	1.4	0.82	1	0.78	0.97
Perylene	0.33	0.25	0.22	0.24	0.26
Benzo[e]pyrene	1.5	1.3	1.6	1.4	1.4
Benzo[g,h,i]perylene	1.4	1.5	2.4	1.3	1.6
Indeno[1,2,3-cd]pyrene	1.5	1.3	1.5	1.4	1.4
Anthanthrene	0.19	0.15	0.13	0.2	0.17
Chrysene and triphenylene	3	2.7	3.5	2.9	3
Benzofluoranthene	3.6	2.9	3.6	4.4	3.6

Source WHO, 1998

WHO report,1998 [39] suggested that from all type of selected sources, emission of phenanthrene (relative to BaP) is maximum and emission of anthanthrene (relative to BaP) is minimum in the ambient air. The other major emission sources of ambient PAHs compound relative to BaP are pyrene, followed by anthracene and benzofluoranthene.

VII. MODE OF EXPOSURE AND DAILY INHALATION OF PAHS

Humans are usually exposed to PAHs by different pathways, mostly due to inhalation of polluted air. Other pathways may be due to consumption of food, water, and dermal contact with soil and dust, CPCB, 2003 [13]. The comparative potential carcinogenic PAHs risk to non-smokers and smokers due to inhalation is shown in Table 5.

TABLE 5The comparative potential carcinogenic PAHs risk to non-smokers and smokers due to inhalation

Source of PAHs	**Inhalation of carcinogenic PAHs**			
	Non Smoker		**Smoker**	
	µg/day	**Total %**	**µg/day**	**Total %**
Food	3	9.3	3	44.6
Air	0.16	4.9	0.16	2.4
Water	0.006	0.2	0.006	<0.01
Soil (Accidental Injection)	0.06	1.9	0.06	1
Cigarette	-	-	3.5	52
Total	**3.22**	**100**	**6.72**	**100**

Source CPCB, 2003

People are exposed to soil contact directly at the outdoor environment, while food and water consumption are usually in the indoor environment. The exposure of PAH to humans due to inhalation may be in both gaseous and particulate form, at indoor and outdoor environment. Smokers are directly in contact with PAHs due to tobacco smoking. It is estimated that the carcinogenic PAHs potential doses due to inhalation have a wide range (0.02 to 3µg/day). The inhalation of average PAHs is approximately 0.16 µg/day. The average intake of PAH concentration through air inhalation is approximately 20 times less than food consumption and approximately 25 times more than the potential intake of drinking water [13].The table indicates that intake of carcinogenic PAHs by the smokers (6.72 µg/day) which is double as compared to the intake by non-smokers (3.22 µg/day). Smokers are directly in contact with carcinogenic PAHs about to be total 52%. Researchers estimated that inhalation of air contributes 3 to 20% of total humans exposure to PAH, which is next to PAHs exposure through food contamination. It is estimated that smokers may intake additional 1-5 µg/day of PAHs through consumption of one pack of non-filtered cigarettes.[13]. The exposure of PAHs through consumptions of food is highest (for smoker 44.6% and non-smoker 9.3%) while exposure of PAHs through consumptions of water is lowest (for smoker <0.01% and non-smoker 0.2%). The exposure of PAH through contaminated air inhalation is highest for non-smoker (4.9%) and lowest for smoking people (2.4%).

VIII. SOURCEWISE INHALATION DAILY DOSE OF BENZO[a] PYRENE

The inhalation of potential carcinogenic PAHs in terms of BaP was estimated as per recommendation of EPA by using the individual's respiration rate.General public average inhalation rate is approximately 11.3 m^3/ day for women and 15.2m^3/day for men recommended by USEPA 1997[40]. The reported BaP concentrations at indoor environment and taking consideration of adult male inhalation rate as a worst-case scenario, the estimated daily intake dose ranged from 0.15–26 ng/day. In general, the maximum inhalation of BaP dose per day occurs in the Asian indoor environment. The cooking activity with different types of fuels like kerosene, wood, coal, cattle dung etc., the minimum inhalation of BaP per day is 93 ng/day, whereas the inhalation per day can be extended more than 25 times higher ie 2523 ng/day also. Fordomestic heating purposes, the use non-airtight stove also emits high concentrations of BaP (30–7448 ng/day). The most relevant source of exposure of BaP for individual is at the indoor environment, as people spent approximately 80–93% of their time at indoors environment. Source wise inhalation daily dose of Benzo[a]pyrene (BaP) at indoor environment is shown in Table 6.

TABLE 6Source wiseinhalation daily dose of BaP at indoor environment

Type of source	**Dose of BaP (ng/day)**	**Comment**	**Reference**
General	0.15–21	Homes located industrialized countries	[40]
	3–26	Asian homes	
	6–21	Indoor spaces for public in the United Kingdom and the United States	
Cooking	91–365	Chinese kitchens	[41]
	105	Cooking with kerosene as fuel	
	502	Cooking with wood as fuel	
	2523	Cooking with cattle dung as fuel	
Heating	30–7448	Indoors using non-airtight stoves	
Environmental Tobacco Smoke (ETS)	4–15	ETS-polluted indoors	[42]
	1.3–6.7	Non-ETS-polluted indoors	
	26–62	Pubs and discotheques	

Source WHO, 2010

ETS (Environmental tobacco smoke) is a complex mixture of toxic chemicals, and mostly emits due to combustion of tobacco substances like cigarettes, cigars, pipes etc. ETS is a well-known human carcinogen and has the better penetrability in terms of the airways to humans. The breathing of ETS has the serious health riskto adultsas compared to young people through diseases like, heart disease, lung cancer etc. ETS is considered as one of the major inhalation sources of PAHs in the indoor environment. At indoor environment, the daily inhalation of BaP from ETSmay range from 4 to15 ng/day, whereas Non-ETS-pollutedenvironments, BaP inhalation may vary from 1.3–6.7 ng/day only. In pubs and discotheques,24 hourly basis, the average inhalation of BaP from indoor environment can be as high as 26–62 ng/day. Children are havingpassive exposure to tobacco smoke in the indoor environmentand significantly, susceptibleto morbidity and mortality [43]. The concentrations of PAHs in the indoor

DOI 10.17148/IARJSET.2016.3937

environmentmay vary from 1 ng/m^{3}to 50 ng/m^3. The variation depends on types of fuel combustion like wood, coal, kerosene and other materials used for residential cooking purposes and tobacco smoke [39].

IX. CARCINOGENICITY & MUTAGENICITY

A substance that has the ability or tendency to cause cancer is known as carcinogen. It may damage the genome material (mostly DNA) of an organism or can disrupt the cellular metabolic processes. Genetics science, the definition of mutagen is a physical or chemical factor that often changes the material of genetic, mostly DNA, of aliving organism. IARC classified few substances (60 in number) which are probably or positively carcinogenic and mutagenic to humans health. These substances are classified according to agents and groups of agents, mixtures and exposure circumstances. Few PAHs are probably or positively carcinogen to humans. Among them, BaP is widely accepted as the indicator for measurement of carcinogenicity. Thus BaP-equivalent toxicity for other carcinogenic PAHs was mostly evaluated for cancer risk assessment. This approach may provide overestimation of cancer and mutagen potency of individual particulate PAH as each PAH are comparatively less carcinogen than BaP. BaP is the most carcinogenic PAHs as per the recommendation of WHO, 2010 [42]. The toxic equivalent factor (TEF) and mutagenic equivalent factor (MEF) relative to BaP are widely used for risk assessment analysis to humans.

BaP-TEQ (carcinogenic equivalents, ng/m^3) and BaP-MEQ (mutagenic equivalents, ng/m^3) are measure for sum of total 8 number of particulate PAHs (∑8PAH), having molecular weight greater than 228 gram. ∑8PAH includes benzo(a)pyrene (BaP), benz(a)anthracene (BaA), chrysene/iso-chrysene (CHR), benzo(b)fluoranthene (BbF), benzo(k)fluoranthene (BkF), indo(123-cd)pyrene (IcdP),Dibenz(a,h)anthracene(DahA),and benzo(ghi)pyrelene(BghiP). BaP-TEQ: Carcinogenic equivalents calculated from the cancer potency relative to BaP (TEF) multiplied by PAH concentration. BaP-MEQ: Mutagenic equivalents calculated from the mutagenic potency relative to BaP (MEF) multiplied by PAH concentration. TEF: toxic equivalency factors for cancer potency relative to BaP [44] and MEF: mutagenic potency factor relative to BaP [45 a,b]. The equations for measurement of BaP-TEQ and BaP-MEQ are given below.

- $(\text{BaP-TEQ})\Sigma 8\text{PAH} = [\text{BaA}] \times 0.1 + [\text{CHR}] \times 0.01 + [\text{BbF}] \times 0.1 + [\text{BkF}] \times 0.1 + [\text{BaP}] \times 1 + [\text{IcdP}] \times 0.1 + [\text{DahA}] \times 5 + [\text{BghiP}] \times 0.01$.
- $(\text{BaP-MEQ})\Sigma 8\text{PAH} = [\text{BaA}] \times 0.082 + [\text{CHR}] \times 0.017 + [\text{BbFA}] \times 0.25 + [\text{BkFA}] \times 0.11 + [\text{BaP}] \times 1 + [\text{IcdP}] \times 0.31 + [\text{DahA}] \times 0.29 + [\text{BghiP}] \times 0.19$.

TABLE 7BaP-TEQ and BaP-MEQ for children at residential indoor and outdoor air environment

	Type of PAHs	Number of samples	TEF*	BaP-TEQ (ng/m^3)	MEF*	BaP-MEQ (ng/m^3)
Residential Indoor Air Environment	Σ8PAH	255	NA	0.098–8.348	NA	0.069–19.72
	BaP	255	1	0.015–4.494	1	0.015–4.494
	BaA	255	0.1	0.002–0.132	0.082	0.001–0.108
	CHR	255	0.01	0.000–0.021	0.017	0.000–0.036
	BbF	255	0.1	0.004–0.346	0.25	0.010–0.865
	BkF	255	0.1	0.002–0.140	0.11	0.002–0.154
	IcdP	255	0.1	0.002–1.400	0.31	0.006–4.340
	DahA	255	5	0.055–1.741	0.29	0.003–0.101
	BghiP	255	0.01	0.001–0.517	0.19	0.016–9.828
Outdoor Air Environment	**Type of PAHs**	**Number of samples**	**TEF***	**BaP-TEQ (ng/m^3)**	**MEF***	**BaP-MEQ (ng/m^3)**
	Σ8PAH	82	NA	0.109–1.932	NA	0.062–2.394
	BaP	82	1	0.016–0.748	1	0.016–0.748
	BaA	82	0.1	0.001–0.059	0.082	0.001–0.048
	CHR	82	0.01	0.0003–0.008	0.017	0.001–0.013
	BbF	82	0.1	0.006–0.237	0.25	0.015–0.592
	BkF	82	0.1	0.002–0.114	0.11	0.002–0.125
	IcdP	82	0.1	0.003–0.197	0.31	0.010–0.611
	DahA	82	5	0.075–1.021	0.29	0.004–0.059
	BghiP	82	0.01	0.001–0.039	0.19	0.010–0.739

Source: Miller et al. 2010

TEF*: toxic equivalency factors for cancer potency relative to BaP (Nisbet and LaGoy et al. 1992) [44]

MEF*: mutagenic potency factor relative to BaP (Durant et al. 1996 and 1999) [45 a,b]

The exposure of BaP-equivalent (BaP-TEQ and BaP-MEQ) concentrations for sum of 8 type of particulate PAHs as well as individual PAH to young children in New York City in both residential indoor and outdoor environment is presented in Table 7 [46]. The indoor (BaP-TEQ)Σ8PAH and (BaP-MEQ)Σ8PAH ranged from 0.098–8.348 ng/m^3 and 0.069–19.72 ng/m^3, respectively which were alarmingly high as compared to outdoor (BaP-TEQ)Σ8PAH (0.109–1.932 ng/m^3) and (BaP-MEQ)Σ8PAH (0.062–2.394 ng/m^3) respectively. The contributions of BaP to (BaP-TEQ)Σ8PAH and (BaP-MEQ)Σ8PAH were higher in both the residential indoor and outdoor environment as compared to other carcinogenic particulate PAHs. In the outdoor environment, the contribution of BbF to (BaP-MEQ)Σ8PAH was comparatively higher than indoor environment, might be due to traffic related emissions of PAHs. BaP is the most carcinogenic and mutagenic contributor, followed by DahA and BbF [47]. The toxicity of DahA is almost equal to BaP,though DahA is a new surrogate compound of particle phase-PAHs. Thus in present day, BaP and DahA both are considered as most air toxic, carcinogenic and mutagenic particulate PAHs in the atmospheric environment.

IX. CONCLUSION

PAHs are generated in the atmosphere due to incomplete combustion of organic compounds and toxic to air environment. The emissions of PAHs are either from pyrogenic source (natural) or petrogenic source (anthropogenic). They may present in the atmosphere either in gas phase or particulate phase or mixture of both the phases. Though there are many petrogenic sources of PAHs in the ambient air, but the in global air environment, but the maximum emission of PAHs is from biofuels (57.6%) only. India is the highest contributor of PAHs from biofuels (92.5%) followed by China (66.4%) and USA (9.1%).

PAHs are one of the major contributors of airborne inhalable particles. Hence it is necessary to understand the importance to measure the individual PAHs and identify their emission sources, so that management control plan to reduce emission of PAHs may be prepared. The emission of atmospheric PAHs occur significantlyfrom multiple emission sources like from vehicular emission, combustion of biofuels (wood, coal etc.) and industrial activities. This review article also highlighted the importance of BaP and BaP-equivalent cancer risk potential for other particulate PAHs. The particulate PAHs are more carcinogen and mutagen as compared to gaseous PAHs. Hence it is deemed necessary to control the emission of PAHs, especially the particular PAHs to reduce the carcinogenic and mutagenic risk to human's health.

ACKNOWLEDGEMENT

The authors are thankful to CPCB (Central Pollution Control Board), New Delhi for providing the relevant information about the Polycyclic Aromatic Hydrocarbons (PAHs) in air and their effects on human health. The authors are also thankful to the Director CSIR- NEERI (National Environmental Engineering Research Institute) and DTU (Delhi Technological University), for providing permission to publish this article.

REFERENCES

[1] L.C.Marr, K. Dzepina, J.L.Jimenez, F.Reisen, H.L.Bethel, J.Arey, J.S.Gaffney,N.A.Marley, & Molina, M. J. "Sources and transformation of particle–bound polycyclic aromatic hydrocarbons" Atmospheric Chemistry and Physics vol.6, pp1733-174, 2006.

[2] Douben, Peter E.T. (2003). PAHs: an ecotoxicological perspective. John Wiley & Sons (Ecological and Environmental Toxicology Series)

[3] S.S. Park, Y.J Kim, C.H.Kang, "Atmospheric polycyclic aromatic hydrocarbons in Seoul, Korea",Atmospheric Environment vol.36, pp 2917–2924, 2002.

[4] World Health organization (WHO), 2000. (Chapter5.9 Polycyclic aromatic hydrocarbons (PAHs) WHO/ Europe www. euro. who. int/__.../ AQG 2ndEd _5_9 PAH.pdf.

[5] G. Na, C. Liu, Z. Wang, L. Ge, X. Ma and Z. Yao,"Distribution and characteristic of PAHs in snow of Fildes Peninsula" Environmental Science, vol.23, pp. 1445-1451,2011.

[6] World Health organization (WHO), 2002. World Health Report 2002: Reducing Risks, Promoting Life. /http://www.who.int/ whr/en/S.

[7] C.E.Bostrom, P. Gerde, A. Hanberg, B.Jerntrom, C.Johansson, T. Kyrklund, A. Rannug, M.Tornqvist, K.Victorin& R. Westerholm. "Cancer risk assessment, indicators, and guidelines for polycyclic aromatic hydrocarbons in the ambient air", Environ Health Perspect 110 (suppl), pp 451-488, 2002.

[8] D.P Singh,R. Gadi, T.K. Mandal, T. Saud, M. Saxena, S.K.Sharma "Emissions estimates of PAH from biomass fuels used in rural sector of Indo-Gangetic Plains of India",Atmospheric Environment,vol. 68, pp120-126, 2013.

[9] Y. Zhang, S. Tau.,"Global atmospheric emission inventory of polycyclic aromatic hydrocarbons (PAHs)", Atmospheric Environment vol. 43, pp 812-819, 2009.

[10] W.F. Rogge, L.M.Hildemann, M.A.Mazurek, G.R. Cass, B.R.T.Simoneit "Sources of fine organic aerosol: 2. Non-catalyst and catalyst-equipped automobiles and heavy-duty diesel trucks" Environmental Science& Technology, vol.27, pp 636–651, 1993.

[11] K.Ravindra, R.Sokhi, G. Van,"Atmospheric polycyclic aromatic hydrocarbons: source attribution, emission factors and regulation-review" Atmospheric Environment,vol. 42, pp 2895-2921, 2008a.

[12] S.E. Manahan, Environ. Chem, sixth ed. Lewis Publisher, New York, 1994.

[13] CPCB, 2003(cpcb.nic.in/.../Publication_363_Document46.pdf.)

[14] B.S.Haynes. In: Bartock, W., Sarofim, A.F. (Eds.), Fossil Fuel Combustion: a Source Book. Wiley, New York, pp. 261–326.

[15] U.E.Wiersum, "The formation of polycyclic aromatics, fullerenes and soot in combustion. The mechanism and the environmental connection. Polycyclic Aromatic Compounds, vol 11. pp 291–300, 1996.

[16] A.L.C.Lima, J.W.Farrington, C.M. Reddy" Combustion-derived polycyclic aromatic hydrocarbons in the environment—a review" Environ Forensics vol.6, pp109–131, 2005.

[17] EUD (European Union Directive), Directive 2004/107/EC of the European parliament and of the council. Official Journal of European Union L23, pp. 3–16. /http://eur-lex. europa.eu/LexUriServ/site/en/oj/2005/l_023/l_02320050126en 00030016.pdf, 2004.

[18] E. Manoli, A. Kouras, C. Samara, C."Profile analysis of ambient and source emitted particle-bound polycyclic aromatic hydrocarbons from three sites in northern Greece", Chemosphere vol.56, pp 867-878, 2004.

[19] M.M.R.Mostert, G.A.Ayoko, S.Kokot, "Application of chemometrics to analysis of soil pollutants." Trends in Analytical Chemistry vol.29,pp430-435,(2010).

[20] H. Guo, S.C. Lee, K.F.Ho, X.M.Wang, S.C. Zou."Particle-associated polycyclic aromatic hydrocarbons in urban air of Hong Kong", Atmospheric Environment, vol. 37, pp 5307–5317, 2003.
[21] M.Tobiszewski, J.Namiesnik" PAH diagnostic ratios for the identification of pollution emission sources-Review", Environmental Pollution, vol.162,pp 110-119, 2012.
[22] D.P Singh,R. Gadi, T.K. Mandal "Characterization of gaseous and particulate polycyclic aromatic hydrocarbons in ambient air of Delhi, India. Polycyclic Aromatic Compounds,vol.32, pp 556–579, 2012, DOI: 0.1080/10406638.2012.683230
[23] M. Akyüz, H. Çabuk, "Gaseparticle partitioning and seasonal variation of polycyclic aromatic hydrocarbons in the atmosphere of Zonguldak, Turkey" Scienceof the Total Environment, vol.408, pp 5550-5558, 2010.
[24] K. Ravindra, E. Wauters, R. V. Grieken, "Variation in particulate PAHs levels and their relation with the transboundary movement of the air masses" Science of the Total Environment, vol. 396, pp100-110, 2008b.
[25] A. H. Neilson," PAHs and Related Compounds", Springer, Berlin 1998.
[26] J. Oda, S, Nomura, A. Yasuhara, T. Shibamoto, "Mobile sources of atmospheric polycyclic aromatic hydrocarbons in a roadway tunnel."Atmospheric Environment, vol. 35, pp 4819–4827, 2001.
[27] C.Pies, B. Hoffmann, J. Petrowsky, Y. Yang, T.A.Ternes, T. Hofmann, "Characterization and source identification of polycyclic aromatic hydrocarbons (PAHs) in river bank soils", Chemosphere vol. 72, pp 1594-1601, 2008.
[28] R.J. Torre-Roche, W.Y.Lee, S.I. Campos-Díaz," Soil-borne polycyclic aromatic hydrocarbons in El Paso, Texas: analysis of a potential problem in the United States/Mexico border region" J. of Hazardous Materials, vol. 163,pp 946-958,2009.
[29] C. Oliveira, N. Martins, J. Tavares, C. Pio, M. Cerqueira, M. Matos, H. Silva, C. Oliveira, F. Camoes." Size distribution of polycyclic aromatic hydro-carbons in a roadway tunnel in Lisbon, Portugal" Chemosphere, 2011. doi:10.1016/ j.chemosphere.2011.01.011.
[30] N.R. Khalili,P.A. Scheff, T.M. Holsen, "PAH source fingerprints for coke ovens, diesel and gasoline engines, highway tunnels, and wood combustion emissions " Atmospheric Environment, vol. 29, pp533–542,1995.
[31] M.B.Yunker,R.W. Macdonald, R. Vingarzan, R.H. Mitchell, D. Goyette, S. Sylvestre. "PAHs in the Fraser River basin: a critical appraisal of PAH ratios as indicators of PAH source and composition" Organic Geochem, vol. 33, pp 489-515, 2002.
[32] A.M. Caricchia, S. Chiavarini, M. Pezza, "Polycyclic aromatic hydrocarbons in the urban atmospheric particulate matter in the city of Naples (Italy)"Atmospheric Environment, vol.33, pp 3731–3738,1999.
[33] P.K. Pandey, K.S.Patel, J. Lenicek, "Polycyclic aromatic hydrocarbons: need for assessment of health risks in India? Study of an urban-industrial location in India" Environmental Monitoring and Assessment.vol. 59, pp 287–319, 1999.
[34] A. Katsoyiannis, E. Terzi, Q.Y.Cai. "On the use of PAH molecular diagnostic ratios in sewage sludge for the understanding of the PAH sources. Is this use appropriate? ", Chemosphere vol. 69, pp 1337-1339, 2007.
[35] G. Grimmer, J. Jacob, K.W.Naujack, "Profile of the polycyclic aromatic compounds from crude oils—inventory by GC GC-MS. PAH in environmental materials, part 3" Fresenius Journal for Analytical Chemistry, vol. 316, pp 29–36, 1983.
[36] W. Esteve, H. Budzinski, E. Villenave, "Relative rate constants for the heterogeneous reactions of NO2 and OH radicals with polycyclic aromatic hydrocarbons adsorbed on carbonaceous particles. Part 2: PAHs adsorbed on diesel particulate exhaust SRM 1650a" Atmospheric Environment, vol.40, pp 201–211, 2006.
[37] M. del Rosario Sienra, N.G. Rosazza, M. Prendez," Polycyclic aromatic hydrocarbons and their molecular diagnostic ratios in urban atmospheric respirable particulate master", Atmospheric. Research. Vol.75, pp 267-281, 2005.
[38] Alves, C., Pio, C., Carvalho, A., Santos, C., "Atmospheric carbonaceous aerosols over grasslands of central Europe and a Boreal forest", Chemosphere vol. 63, pp 153-164, 2006.
[39] World Health organization (WHO), 1998. Selected Non-heterocyclic Polycyclic Aromatic Hydrocarbons (Environmental Health Criteria 202). Geneva.
[40] United States Environmental Protection Agency (USEPA), 1997.Washington, DC, EPA/600/P-95/002F a-c Exposure Factors Handbook (1997 Final Report)| Risk Assessment, https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=12464
[41] C.V.Raiyani, J.P.Jani, N.M. Desai, P.G.Shah, D.J. Parikh, S.K. Kashyap,"Assessment of indoor exposure to polycyclic aromatic hydrocarbons from urban poor using various types of cooking fuels."EnvironmentalContamination. and Toxicology, vol.50, pp 757–763,1993a
[42] World Health organization (WHO), 2010. WHO Guidelines for indoor air quality: selected pollutants www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf.
[43] S.H. Hwang, J.H. Hwang, J.S. Moon & D.H. Lee, "Environmental tobacco smoke and children's health", Korean Journal of Pediatrics, vol. 55(2): pp 35–41,2012. doi: 10.3345/kjp.2012.55.2.35.
[44] Nisbet I, LaGoy P, "Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs)".Regulatory Toxicology and Pharmacology,vol. 16, pp 290–300,1992.
[45] J. Durant, A. Lafleur, W. Busby, A. Lafleur, B. Penman, C. Crespi," Human cell mutagenicity of oxygenated, nitrated and unsubstituted polycyclic aromatic hydrocarbons associated with urban aerosols", Mutation Research/Genetic Toxicology and Environmental Mutagenesis, vol. 371, pp 123-157, 1996a.
[46] J. Durant, A. Lafleur, W. Busby, L. Donhoffner, B. Penman, C. Crespi,"Mutagenicity of $C_{24}H_{14}$ PAH in human cells expressing CYP1A1. Mutation Research/Genetic Toxicology and Environmental Mutagenesis, vol. 446, pp 1-14, 1999b.
[47] Durant J, Busby W, Lafleur A, Penman B, Crespi C. Human cell mutagenicity of oxygenated, nitrated and unsubstituted polycyclic aromatic hydrocarbons associated with urban aerosols. Mutation Research/Genetic Toxicology and Environmental Mutagenesis,vol.371, pp123–157, 1996.
[48] R. L. Miller, K.H. Jung, B.Yan, S.N. Chillrud, F.P. Perera, R. Whyatt, & D. Camann, "Assessment of Benzo(a)pyrene-equivalent Carcinogenicity and Mutagenicity of Residential Indoor versus Outdoor Polycyclic Aromatic Hydrocarbons Exposing Young Children in New York City", International Journal of Environmental Research & Public Health,vol. 7, pp1889–1900, 2010. doi: 10.3390/ijerph7051889
[49] M. Pufulete, J. Battershill,A. Boobis and R. Fielder."Approaches to carcinogenic risk assessment for polycyclic aromatic hydrocarbons: A UK perspective" Regulatory Toxicology and Pharmacology, vol. 40, pp 54–66, 2004.