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Review

A Review on Cancer Probability in Human Beings Due to Environmental Impact of Polycyclic Aromatic Hydrocarbons (PAHs) and Remediation

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Abstract. Environmental consequences of polycyclic aromatic hydrocarbons (PAHs) were studied to evaluate possible human health risks, subsequent cancer probabilities and remediation tools for their eradication. Polycyclic aromatic hydrocarbons (PAHs) are generated globally through incomplete combustion of organic materials and emitted in the environment by various anthropogenic routes including residential heating, coal gasification, liquefying plants, cooking practices, thermal distillation of petroleum and coal, oil spills, ships embankments, incomplete burning of fossil fuels, forest fires, asphalt, engines and vehicles exhaust. Their high lipophilicity and marked tendency for localization in body fats made them easily absorbed through dermis, nasal mucosa and gastrointestinal tract of mammals. The reviewed data show estimated carcinogenic potency equivalent concentrations exceeding the screened value for food stuffs including fish species indicating significant carcinogenic health risks associated with the consumption of fishes. Many remedial measures have been taken to exterminate persistent organic pollutants including PAHs out of which bio-remediation being comparatively safer and economical methodology.

Keywords: carcinogenic, saturated and unsaturated hydrocarbons (PAHs), fat and isolation

Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a large group of organic compounds with two or more fused aromatic (benzene) rings found globally as mixtures in air, water and sediments. They occur naturally in fossil fuels or formed by the incomplete combustion of organic materials or coal gastification sites (Fig. 1) (Behera *et al.*, 2018). PAHs are classified as persistent organic pollutants (POPs) and persistent, bio-accumulative and toxic (PBT) chemicals with various carcinogenic and mutagenic properties. Dietary sources are among the major route of exposures in humans among both smokers, non-smokers, occupational workers and non-occupational persons mostly due to food processing techniques like curing, drying, smoking, roasting, grilling, barbecuing and refining. These food processing steps are known to generate and increase the level of PAHs in the food (WHO, 2000).

All these serious health issues avokes a great matter of concerned around the world and number of regional conventions were held since 1970s to control over them and many legislations have been made. In consonance with US EPA and ATSDR 16 PAHs are listed as reasonably anticipated to be human carcinogen and included in EPA priority pollutant list (Table 1), (EPA, 2015; ATSDR, 2012). Scientific Committee on Food of European Union had also established some legislations regarding exposure of food stuffs by different PAHs sources (Table 2) (EC (SCF), 2011).

Sources and exposures to humans. Humans are exposed by PAHs through a variety of anthropogenic and natural sources by different routes specifically subsequent inhalation, digestion and skin contact (Fig. 4) (Qu *et al*., 2015). The sources includes forest fires, volcanic eruption, oil or natural gas seepage, mining of natural fuels and their destructive distillation, oil spills, incomplete organic fuel combustion, tobacco smoke, industrial by product emissions, contaminated food and different cooking practices (Fig. 2) (Masuda *et al*., 2019; Alomirah *et al*., 2011; Ramesh *et al*., 2011). This has been reported that realistic exposure to toxic chemicals occurs always in mixtures rather than by single chemical.

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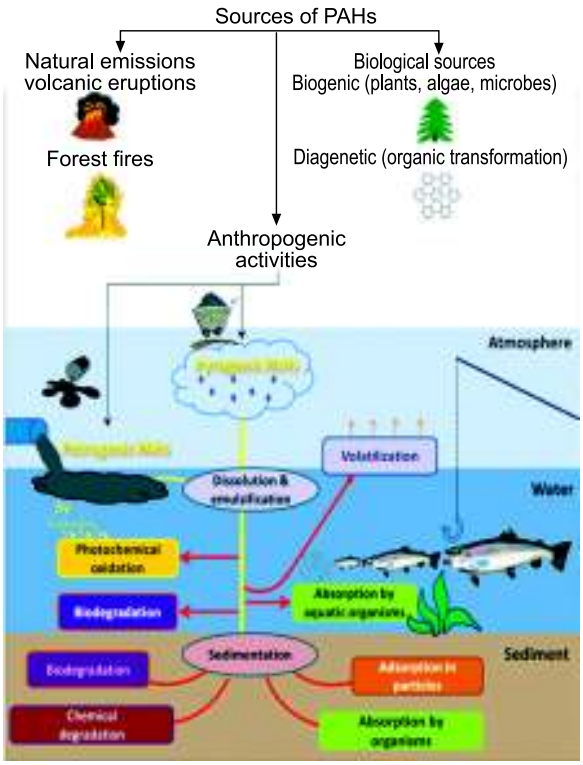

Fig. 1. Environmental sources of polycyclic aromatic hydrocarbons (Behera *et al*., 2018).

However, for study purposes single chemicals are being used and helpful in the research dominion. Therefore, preferably PAHs as mixtures are given more importance to study the interaction between each individual compound and their relevance to human health hazards and exposures. Polycyclic aromatic hydrocarbons (PAHs) are toxic semi volatile organic compounds (SVOCs) that partition between gas and aerosol phases and travel long distances in the atmosphere (Fig. 3) (Friedman *et al*., 2014; Wang *et al*., 2010). The PAHs become trapped in secondary organic aerosol (SOA) during its formation, preventing them from evapouration and degradation *via* oxidation (Zelenyuk *et al*., 2012). PAHs in the atmosphere decreases substantially due to accumulation of SOA species over particles during their atmospheric transport and chemical aging (Jariyasopit *et al*., 2014; Robinson *et al*., 2007; Finley *et al*., 1994). To find the clear impact of PAHs on environment the congested industrial areas has to be constantly monitored for pollution by the environmental protection agency (EPA) of respective countries.

Table 1. The carcinogenic classifications of selected PAHs by specific agencies (ATSDR, 2012)

Agency	(EPA, 2000) PAH Compound(s)	Carcinogenic Classification
U.S. Department of health and human services (HHS)	• Benz(a)anthracene • Benzo(b)fluoranthene • Benzo(a)pyrene • Dibenz(a,h)anthracene • Indeno(1,2,3-c,d)pyrene	Known animal carcinogens
International agency for research on cancer (IARC)	• Benz(a)anthracene • Benzo(a)pyrene	Probably carcinogenic to humans
	• Benzo(a)fluoranthene • Benzo(k)fluoranthene • Indeno(1,2,3-c,d)pyrene	Possibly carcinogenic to humans
	• Anthracene • Benzo(g,h,i)perylene • Benzo(e)pyrene • Chrysene • Fluoranthene • Fluorene • Phenanthrene • Pyrene	Not classifiable as to their carcinogenicity to humans
U.S. Environmental protection agency (EPA)	• Benz(a)anthracene • Benzo(a)pyrene • Benzo(b)fluoranthene • Benzo(k)fluoranthene • Chrysene • Dibenz(a,h)anthracene • Indeno(1,2,3-c-d)pyrene	Probable human carcinogens
	• Acenaphthylene • Anthracene • Benzo(g,h,i)perylene • Fluoranthene • Fluorene • Phenanthrene • Pyrene	Not classifiable as to human carcinogen

For example Gadani in Balochistan province of Pakistan was designated as the world's third largest ship-breaking yard in the 1980s. The ship-breaking operations polluted the environment following several decades due to which subsurface soil, groundwater and sediments are contaminated with PAHs and a variety of pollutants, including dioxins and several heavy metals, reported during the human health and ecological risk assessments. Ship-breaking activities are sources of atmospheric emission of PAHs and high concentrations of PAHs have been found in ambient air in Chittagong in comparison to those found in similar studies performed in other parts of Asia (Nøst *et al*., 2015). Estimated

Fig. 2. Cooking impact of PAHs and its derivatives chlorinated polycyclic aromatic hydrocarbons (ClPAHS) (Masuda *et al*., 2019).

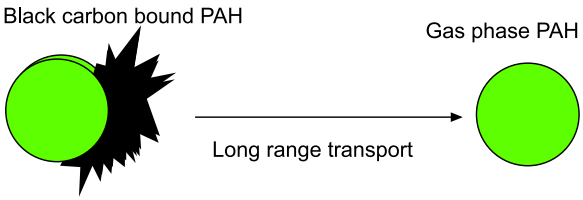

Fig. 3. Influence of secondary organic aerosol (SOA) on long-range atmospheric polycyclic aromatic hydrocarbon transport (Friedman *et al*., 2014).

toxic equivalent quotients indicate elevated human health risks caused by inhalation of PAHs at most sites. Similarly Clifton beach of Karachi has shown the most polluted site after Tasman spirit oil spill (Janjua *et al*., 2013; Meo *et al*., 2009). Thus exposure to PAHs either directly or indirectly causes severe health issues in humans depending on the nature of contact with the oil spill means either through respiration in polluted air, skin contact and indirectly bathing in contaminated water or eating contaminated food. Extremely high levels of PAHs were measured in Clifton beach sediments after a period of first sampling of Tasman spirit oil spill and this site was found to be one of the most highly PAHs contaminated sites in the world relative to other areas and worldwide estuaries of concern (Siddiqi and Munshi, 2015).

The Clifton beach provides an opportunity to study a complex PAH mixtures that may represent world exposure scenario for decade. No doubt, it is a great need to analyze and mitigate risks due to PAHs exposure, which has numerous human health consequences. All

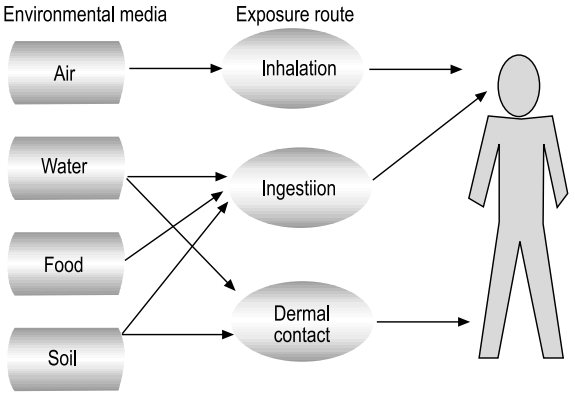

Fig. 4. Multipath ways of human exposure to polycyclic aromatic hydrocarbons (Qu *et al*., 2015).

matrices like sediments, seawater, marine fauna and flora than air must be analyzed for their contribution to PAH exposure, which may have a large impact on dietary exposures to PAHs. Higher concentration of PAHs and the complexity of the mixture have proved that many PAHs are carcinogenic. The sediment-derived PAH mixture components would cancer relevant and confer cancer risk through various activities (e.g., fishing) relevant to sediment exposure. A model complex PAH mixture that may occur in other regions can cause increased cancer risk and detrimental health effects. A common method can be utilized successfully to estimate PAHs exposure (Fillmann *et al*., 2004) and other urinary biomarkers across the Pakistan with a statistical weighted approach (Sobus *et al*., 2015). Efforts have been done to enhance the capabilities to estimate and rank demographic subpopulations by PAHs exposure, targeting those with high exposure. PAHs mixtures have posed significant impact on public health risks by the potential to increase cancer risk in the general population, especially for vulnerable and highly exposed populations being ubiquitous environmental toxicants.

PAHs are supposed to be endocrine disrupting chemicals and their exposure is most threatful for human beings from prenatal days until death (Miller and Jones, 2013). It has been noted that workers exposed to mixtures of PAHs possess increased risk of skin, lung, bladder and gastrointestinal cancers and long-term exposure to low levels of some PAHs may cause cancer such as Benzo (a) pyrene which

Table 2. The annex to regulation (EC) No 1881/2006 is amended as follows; Section 6: "polycyclic aromatic hydrocarbons" (EC (SCF), 2011)

Foodstuffs		Maximum Limits µg/Kg	
6.1	Benzo(a)pyrene, benz(a)anthracene, Benz(b)fluoranthene and chrysene	Benzo(a)pyrene	Sum of Benzo(a)pyrene, Benz(a)anthracene, Benz(b)fluoranthene and chrysene
6.1.1	Oils and fats (excluding coca butter and coconut oil)intended for direct human consumption or use as an ingredient in food	2.0	10.0
6.1.2	Cocoa beans and derived products	5.0 µg/Kg fat as from 1.4.2013	35.0 µg/Kg fat as from 1.4.2013 until 31.3.2015 30.0 µg/Kg
6.1.3	Coconut oil intended for direct human consumption or use as an ingredient in food	2.0	20.0
6.1.4	Smoked meat and smoked meat products	5.0 until 31.8.2014 2.0 as from 1.9.2014	30.0 as from 1.9.2012 until 31.12.2014 12.0 as from 1.9.2014
6.1.5	Muscle meat of smoked fish and smoked fishery products ([25]) ([36]), excluding fishery products listed in points 6.1.6 and 6.1.7. The maximum levels of smoked crustaceans applies to muscle meat from appendages and abdomen ([44]). In case of crabs and crab like crustaceans (Brachyura and Anomura) it applies muscle meat from appendages.	5.0 until 31.8.2014 and 2.0 as from 1.9.2014	30.0 as from 1.9.2012 until 31.8.2014 12.0
6.1.6	Smoked sprats and canned smoked sprats ([25]) ([47]) (sprattus sprattus) bivalve mollusk (fresh, chilled or frozen) ([26]) heat treated meat and heat treated meat products ([46]) sold to the final consumer	5.0	30.0
6.1.7	Bivalve mollusks ([36]) (smoked)	6.0	35.0
6.1.8	Processed cereal based foods and baby foods for infants and young children ([3]) ([29])	1.0	1.0
6.1.9	Infant formula and follow-on formulate, including infant milk and follow-on milk ([8]) ([29])	1.0	1.0
6.1.1.0	Dietary foods for special medical purposes ([9]) ([29]) intended specifically for infants	1.0	1.0

([45]) Lower bound concentrations are calculated on the assumption that all the values of the four substances below the level of quantification are zero

([46]) Meat and meat products that have undergone a heat treatment potentially resulting in formation of PAH, i.e. only grilling and barbecuing

([47]) For the canned product analysis shall be carried out on the whole content of the can. As regards the maximum levels for the whole composite product Art. (2)(1)(c) and 2(2) shall apply." (2) Endnote ([35]) is deleted

is the most common PAH to cause cancer in humans (Rengarajan *et al*., 2015).

Mechanism of cellular metabolism plays an important role in the conversion of chemical carcinogens into reactive species that damage cellular macromolecules and interfere with signaling pathways that cause cancer (Laffon *et al*., 2016). Human colon cells were known to metabolize PAHs and implants the toxic impacts on humans. This review compiles all necessary information available about PAHs, its chronic health hazards and human cancer risks. Health outcomes are addressed in the following categories in this review: physiological effects, genotoxicity, immunotoxicity, carcinogenicity, endocrine toxicity.

Physiological impacts in humans. PAHs can make their way into the human's body through various routes that is inhalation, dermal contact and ingestion. Monte Carlo Simulation techniques have been widely used to provide quantitative estimates of probabilities of exposure and health risks (Table 3) (Qu *et al*., 2015). They are highly lipophilic and easily absorb through mucosal membrane of humans and their toxicity depends exclusively on their biotransformation to toxic metabolites and their interactions at the level of key metabolism enzymes. Induction of metabolism by one PAH may enhance the toxicity of another on the other hand the inhibition of the metabolic enzymes may decrease the toxicity of PAHs metabolites. They possessed adverse effects on human's immune system development, humoral immunity and host resistance (Tarafdar *et al.*, 2020; Abdel-Shafy and Mansour, 2015). The mixed function oxidase enzyme systems are primarily responsible for PAHs metabolism and the first reaction is the formation of epoxides in the human cells. These epoxides then conjugated with glutathione at the detoxification step. The un-conjugated epoxides are converted into phenols and diols that has to be conjugated glucorunic acid or sulfuric to enable excretion (Fig. 5) (Behera *et al*., 2018).

Genotoxicity. A number of PAHs are mutagenic and genotoxic, depending on compound and induce DNA adduct formation *in vitro* and *in vivo* (Fig. 6). Some PAHs probably cause mutations in a number of genes that contribute to cancer development for example the anti-diol-epoxide of B[*a*]P ((±)-anti-BPDE). In human studies, lung tumors from non-smokers exposed to PAHs rich coal combustion emissions had mutations at guanine in K-ras codon 12 and p53 genes (Rengarajan *et al*., 2015).

Table 3. Risk parameters used for monte carlo simulation (Qu *et al*., 2015)

Parameter	Symbol	Units	Infants	Children	Adults	References
Population parameter						
Age	___	Year	0-1	2-18	19-70	
Body weight	BW	Kg	6.79±1.27	37.3±9.1	58.7±12.0	Chen and Liao (2006) Xiao *et al*. (2005)
Inhalation parameter						
Inhalation rate	IR_a	m^3/day	5.05±0.49	9.67±2.39	12.44±1.27	Wang *et al*. (2009)
Ingestion parameter						
Ingestion rate of water	IR_w	mL/day	283.25±91.48	497.35±138.28	1,366±728	USEPA (1997,2008)
Ingestion rate in soil	IR_s	mg/day	0-30	24±4	25(0.1-50)	Stanek *et al.* (2001), USEPA (2008), LaGoy (1987)
Ingestion rate in fish	IR_f	mg/day	4.16±2.37	27.45±5.52	61.25±13.8	
Dermal parameter						
Total skin surface area	A_{bath}	m^2	0.39±0.05	1.09±0.37	1.67±0.10	Wang *et al*. (2008)
Exposed skin surface area	A_s	cm^2	719±1.19	860(430-2,160)	1,530 (760-4,220)	Wang *et al*. (2008) Chen and Liao (2006)
Soil to skin adherence factor	AF	mg/cm^2 /day	0.04	0.65±1.2	0.49±0.54	USEPA (2004), Finley *et al*. (1994)
Time for shower	*t*	min/day	15	18.41±1.32	10.4(3-61	USEPA (1997, 2008)
Dermal absorption factor	ABS	Unitless	0.13	0.13	0.13	USEPA (2004)
Dermal permeability coefficient	K_p	cm/h	0.7	0.7	0.7	USEPA (2004)
Risk model parameter						
Exposure frequency	EF	Days/year	345(180-365)	345 (180-365)	345(180-365)	Smith (1994)
Averaging time	AT	Days	25,550	25,550	25,550	USEPA (997)
Exposure duration	ED	year	1	17	52	USEPA (1997, 2008)

The mean and standard deviation were used for lognormal distributions, minimum and maximum for uniform distributions and mean, minimum for triangular distributions.

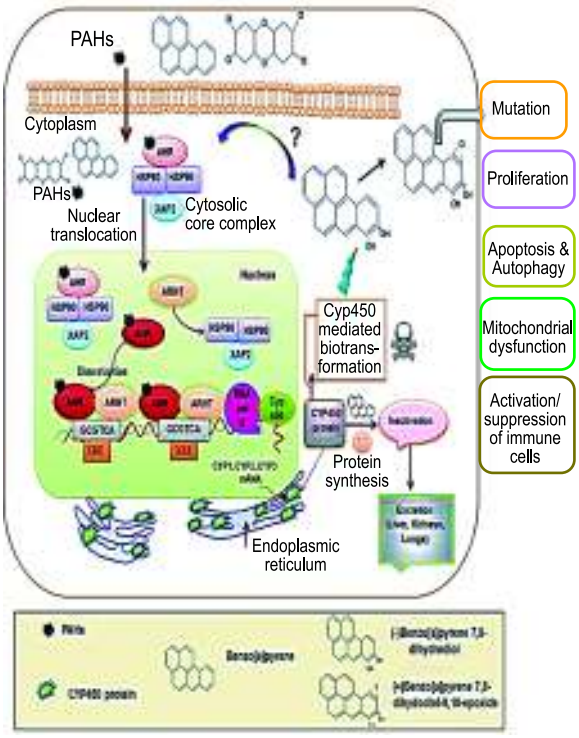

Fig. 5. Toxic multi-pathways of polycyclic aromatic hydrocarbons (Behera *et al*., 2018).

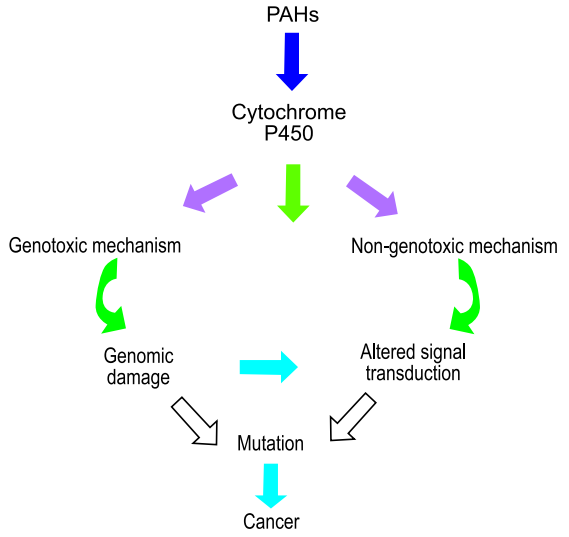

Fig. 6. Genotoxicity/mutagenicity & carcinogenicity of polycyclic aromatic hydrocarbons.

Immunotoxicity. The immune system is the common target for PAH-toxic effects and for this reason PAH-immunogenicity or immunotoxicity has been widely examined in different studies using human cell lines and animal models. Immunotoxicity of PAHs on human beings has been evaluated and it has been examined that human T cells are highly sensitive to suppression of mitogen-effects of PAHs. B (a) P and DMBA are found highly immunotoxic in human system and showed suppression of T lymphocyte mitogenesis (Sakshi and Haritash, 2020).

Carcinogenicity. PAHs with 4 to 7 rings are found to be carcinogenic in experimental animals such as B[*a*]P that induced tumors after exposure to PAHs in mice, rats, rabbits, hamsters and monkeys (EPA, 2015). Like other toxic chemicals, PAHs are also carcinogenic accumulants in both finfish and shellfish, binding to lipid molecules in muscles or tissues. Bioavailability and bio-transformation influenced mechanisms of cancer processes for disposition of PAHs in the body (Copat *et al*., 2013).

The organic fuel is a major source of PAHs contamination in food, generated particularly in unvented stoves cooking practices for example charring meat or deep frying, that can imposed carcinogenic effects by oral route exposures (Fig. 2) (Masuda *et al*., 2019; Balcioglu, 2016). The non-metabolized PAHs are converted into reactive oxygen species such as epoxides and dihydrodiols, forming linkages to cellular proteins and DNA. The resulting biochemical disruptions and cell damage lead to mutations, developmental malformations, tumors and different types of cancers (Fig. 5) (Behera *et al.*, 2018; Wu *et al.*, 2012).

Data sources from occupational studies indicates that mixtures of PAHs are carcinogenic to humans, predominantly causing skin and lung cancers but also bladder and gastrointestinal cancers in some cases. However, it is not clear from these studies whether exposure to PAHs was the main cause, as these workers had been simultaneously exposed to other cancer causing agents (e.g., aromatic amines) Table 4 (Helmfrid *et al*., 2019). The studies on laboratory animals indicates that exposure to high levels of certain PAHs over a long period of time developed lung cancer from inhalation, stomach cancer from ingesting PAHs in food and skin cancer from skin contact. It is clear from ongoing research that BaP is the most common PAH that cause cancer in animals and is notable for being the first chemical carcinogen to have been discovered (Sakshi and Haritash, 2020; Abdel-Shafy and Mansoor, 2015; Rengarajan *et al*., 2015).

Table 4. Basic characteristics of the study population. A restriction criterion of at least five years of residence time in the contaminated area, within 2 km from glass work (controls) and a latency time of at least 10 years between first residence year and time of diagnosis (cases) were applied. In the subsample taking part in the biomonitoring study, study participants who at the time of sampling lived within a geographical distance of 50 km were included. Significant differences ($P < 0.05$) in bold. (Helmfrid *et al*., 2019)

Questionnaire study population N=1846				Bio-monitoring study (a sub-population of the questionnaire study population) N=656			
All	Cases N=361	Controls N=1485	Cases N=127	Controls N=479	Living within 50 km N=50		
Gender	Men, Number (%)	863 (47)	174 (48)	689 (46)	62 (49)	220 (46)	25 (50)
	Women, Number (%)	983 (53)	187 (52)	796 (54)	65 (51)	260 (54)	25 (50)
Age (Year)	Mean (SD)	59 (15)	**69 (11)**	**56 (15)**	**70 (9.6)**	**58 (1)**	54 (18)
	Median	60	70	56	70	59	57
	Range	19–98	19–98	19–97	33–89	29–92	20–92
Residence time in the glasswork area (years)	Mean (SD)	36 (20)	**41 (22)**	**35 (19)**	**47 (18)**	**40 (1)**	26 (25)
	Median	35	42	33	47	40	17
	Range	5–98	5–98	5–91	6–88	5–91	1–80
Residence in glasswork area before age of 5 yrs.	Number (%)	929 (50)	**157 (43)**	**772 (52)**	**56 (44)**	**266 (5)**	23 (46)
Academic education	Number (%)	665 (36)	**97 (27)**	**568 (38)**	35 (28)	153 (32)	18 (36)
Working at glasswork	Number (%)	162 (9.9)	37 (11)	125 (9.5)	12 (11)	48 (12)	5 (10)
Other metal work	Number (%)	204 (12)	32 (9.8)	172 (13)	14 (12)	74 (17)	8 (16)
Smoking status: number (%)	Active smoker	80 (4.4)	14 (3.9)	66 (4.4)	3 (2.4)	20 (4.2)	4 (8)
	Former smoker	789 (43)	**181 (51)**	**608 (41)**	62 (49)	209 (44)	23 (46)
	Never smoker	969 (53)	**163 (46)**	**806 (54)**	61 (48)	251 (52)	26 (52)
Consumers of local vegetarian products	Number (%)	1015 (75)	183 (78)	832 (74)	77 (79)	333 (83)	38 (76)
Consumers of local meat products	Number (%)	541 (47)	78 (44)	463 (48)	31 (40)	177 (50)	19 (38)
Consumers of locally caught fish	Number (%)	158 (14)	**34 (20)**	**124 (13)**	27 (35)	109 (31)	11 (22)
Cancer		**ICD7**					
Digestive system	Number (%)	151–154	41 (10)		15 (12)		
Rectum	Number (%)	154	17 (4.3)		7 (5.5)		
Female genital	Number (%)	171–174	99 (25)		33 (55)		
Breast	Number (%)	170	76 (19)		25 (38)		
Male genital	Number (%)	177–178	79 (20)		36 (58)		
Prostate	Number (%)	178	73 (18)		33 (53)		
Kidney and bladder	Number (%)	180–181	20 (5.1)		4 (3.1)		
Brain	Number (%)	193	24 (6.1)		9 (7.1)		
Lymphatic and hematopoietic tissues	Number (%)	200–208	23 (5.8)		10 (7.9)		
Other sites	Number (%)		93 (23)		20 (16)		

[a]Participants taking part in the biomonitoring study who lived outside the 2 km radius but within 50 km of glassworks at the time of biological sampling.

The enzyme systems involved in the metabolism of carcinogenic PAHs includes cytochrome P450 enzymes, CYP 1A1, 1A2, 1B1 and 3A4. In some case studies, specific mutations found in the Tp53 gene, the most commonly mutated gene in human cancers, associated with exposure to certain carcinogens. For example, the PAHs in cigarette smoke bind preferentially to the Tp53 gene sites called "hotspot" codons, where most smoking associated mutations are also found. Such studies give support to the link between DNA adducts and the cancer risk in humans (Rengarajan *et al*., 2015). The International Agency for Research on Cancer and the US EPA classified a number of PAHs as carcinogenic to animals and some PAH-rich mixtures as carcinogenic to humans Table 1 and 2 (EPA, 2015; ATSDR, 2012).

Endocrine toxicity. PAHs undergoes number of compositional changes and found to be endocrine disrupting chemicals in many epidemiological studies in humans cell lines. The type of function effected and associated doses vary with the alkylation status of PAHs (Lee *et al*., 2017). Estrogen signaling is one of the most studied pathways associated with the endocrine-disrupting activities of PAHs and involves estrogen receptors and aryl hydrocarbon receptors. However, some of the actions of PAHs are contradictory, complex, and un-explainable. Although several possibilities have been suggested, such as direct interactions between PAHs and their receptors and the suppression of their activities through other pathways. The mechanisms underlying the activities of PAHs remain unclear. So, standardized assay protocols for pathway based assessments are considered to be important to overcome these issues (Zhang *et al*., 2016).

Remediation. Many physical and chemical procedures have been used for the remediation of PAHs at oil polluted sites but most of them were found to be challenging, expensive and formed secondary contaminants. Elimination of PAHs from air, water bodies and sediments can take place by means of volatization, photo oxidation, chemical oxidation, bio-accumulation, adsorption, microbial degradation and their adhesion to the soil matrix (Fig. 7) (Abdul-Shafy and Mansour, 2015). The physical characteristics of PAHs such as increasing molecular weight, increased number of rings, insignificant water solubility and high octanol-water partition coefficient decides their availability in the environment and selection of eradication tool (Gaur and Narasimhulu, 2018; Gitipour *et al*., 2018).

Apart from these bio-remediation is found to be the most effective, cost efficient and environmental friendly technique. Many aerobic and anaerobic micro-organisms including bacteria (gram-positive and gram negative), fungi, and algae possess the ability to utilize PAHs as energy source. PAH-bio-degradation could be achieved when microbial species are present in favourable environmental conditions of temperature, pH, nutrients and metabolites (Sakshi and Haritash, 2020). The studies have revealed that the most prominently studied micro-organisms for PAH-degradation are bacteria followed by fungi and algae. The bio-degradation involves bio-transformation of PAHs compounds into simpler metabolic species and also include mineralization of organic compounds into inorganic minerals either aerobically that is H_2O and CO_2 or anaerobically such as CH_4 (Fig. 8) (Sakshi and Haritash, 2020; Babu *et al*., 2019; Kuppusamy *et al*., 2017; Shahsavari *et al*., 2014). Table 5 shows some advantages and dis-

Table 5. Advantages and disadvantages of bio-remediation techniques (Shahsavari *et al*., 2014)

Technology	Key points	Advantages	Disadvantages
Natural attenuation	Using indigenous micro-organisms and natural condition	Cheapest technology	Requires extensive long-term monitoring Not always successful
Bio-augmentation	Addition of hydrocarbon-degrading micro-organisms	Using high biomass of hydrocarbonoclastic micro-organisms	Changes the natural microbial structure Poor adaptation of hydrocarbonoclastic micro-organisms to the contaminated site
Bio-stimulation	Addition of nutrient	More efficient than natural attenuation	Not always successful
Phytoremediation	Using plants and their associated micro-organisms	Supports hydrocarbonoclastic micro-organisms within plant root	Toxicity of contaminants to the plant

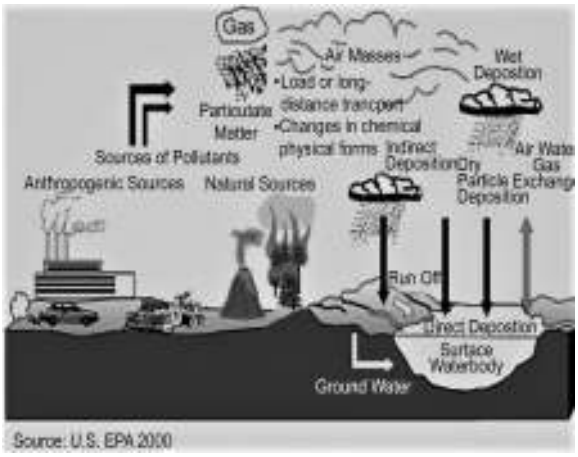

Fig. 7. Elimination of PAHs from the Environment (Abdul-Shafy and Mansour, 2015).

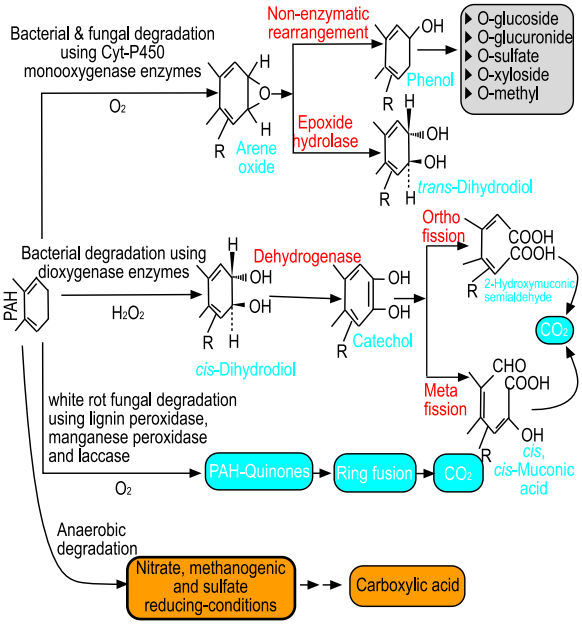

Fig. 8. Bio-degradation pathways of bacteria and fungi (Shahsavari *et al*., 2014).

advantages of bacterial and fungal bio-degradation processes (Shahsavari *et al*., 2014).

Conclusion

Epidemiological exposure. The environmental factors play an important role in cancer susceptibility as an association between ingestion of PAHs and esophageal cancer (Chen and Liao, 2006; Kamangar *et al*., 2005). This can be concluded that the high levels of carcinogenic PAHs in food and indoor air pollution (domestic cheating with coal) have been implicated as the causative factors for esophageal cancer. It is reported that cancer is promoted by the increased intake of PAHs through dietary fat and dietary B(a)P consumption which is a major risk factor for cancer (Yebra-Pimentel *et al.*, 2015; Tabatabaei *et al*., 2010), and the existing data gaps and scope for future studies is also has to be emphasized. This information is also expected to stimulate research on mechanisms of sporadic cancers caused by exposure to environmental carcinogens. Therefore, proper estimations of dietary and non-dietary exposures could help accurately in assessing the risk of exposure to PAHs through diet.

Conflict of Interest. The authors declare no conflict of interest.

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