Exposure Assessment of Polyaromatic Hydrocarbons and Biological Monitoring of Their Metabolites in Different Occupational Group Workers

Thesis submitted in the partial fulfillment of the award of

DOCTOR OF PHILOSOPHY
IN
ENVIRONMENTAL SCIENCE

by
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Dedication

This thesis is dedicated to my Mother, Father, spouse and my son for their endless love, support, encouragement and patience and who are behind my successful achievement

Declaration

I hereby declare that the thesis entitled ***"Exposure Assessment of Polyaromatic Hydrocarbons and Biological Monitoring of Their Metabolites in Different Occupational Group Workers"*** is based on the results of research work carried out by me in The Industrial Hygiene & ToxicologyDivision , Regional Occupational Health Centre (Southern)-National Institute of Occupational Health, Indian Council of Medical Research, Bangalore under the guidance of Prof. J. Narayana, Department of Environmental Science, Kuvempu University, Jnanasahyadri, Shankaraghatta-577451, Shimoga District, Karnataka and Dr. B.Ravichandran,Scientist, Regional Occupational Health Centre (Southern), Indian Council of Medical Research, Poojanahalli Road, Kannamangala Post, Devanahalli TK, Bengalore-562110, Karnataka.

I further declare that this or any part thesis, has not been submitted elsewhere for any degree or diploma in any other University or Institution.

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This is to certify that the thesis entitled **"Exposure Assessment of Polyaromatic Hydrocarbon and biological Monitoring of their metabolites in different occupational group workers"** submitted by Mr. Somnath Sen, for the award of Degree of Doctor of Philosophy in Environmental Science, Kuvempu University, is a record of bonafide research done by him under my co-guidance and it has not been submitted elsewhere for any degree or diploma in any other University or Institution.

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Abbreviations

Polycyclic aromatic hydrocarbons	:	*PAHs*
Naphthalene	:	*NAP*
Acenaphthene	:	*ACE*
Acenaphthylene	:	*ACY*
Anthracene	:	*ANT*
Phenanthrene	:	*PHE*
Fluorene	:	*FLU*
Fluoranthene	:	*FLT*
Benzo(a)anthracene	:	*BaA*
Chrysene	:	*CHR*
Pyrene	:	*PYR*
Benzo(a)pyrene	:	*BaP*
Benzo(b)fluoranthene	:	*BbF*
Benzo(k)fluoranthene	:	*BkF*
Dibenz(a,h)anthracene	:	*DahA*
Benzo(g,h,i)perylene	:	*BghiP*
Indeno(1,2,3-cd) pyrene	:	*IND*
1-hydroxypyrene	:	*1-OHP*
Hydroxyphenanthrene	:	*OHPHE*
American Conference of Governmental Industrial Hygienists	:	*ACGIH*
National Institute for Occupational Safety and Health	:	*NIOSH*
United Nation Environment Protection Agency	:	*USEPA*
World Health Organisation	:	*WHO*

Chapter~ 1

Introduction

Chapter-1

Introduction

1.1 PAHs

Human health risks related to occupational and environmental exposure to hazardous chemicals are current concern and exposure to low doses of chronic exposure resulting in incremental health risks. The first type of measurement used for hazard quantification was ambient monitoring, which was utilised to assess external dose. Another way of assessing hazards was by biological monitoring which was the tool for quantifying internal dose (Franco SS *et. al.,* 2008).

Polycyclic aromatic hydrocarbons (PAHs) are classified as hazardous pollutants. These chemical compounds are formed as combustion by-products as a result of anthropogenic activities. It is widely distributed in the nature and are emitted from pyrolysis or incomplete combustion of organic material at high-temperature during processing of crude oil, coal, or other industrial carbon-containing compounds (Rom, 1983). PAHs are concerned in environmental health for its potent carcinogenicity in man, especially those PAHs having 4-6 aromatic rings (i.e. BaP, DahA, BaA). There was strong epidemiological evidence that exposed groups had increased risks of lung, urinary tract, brain and skin cancers (Hansen, 1988, 1989 and Hammond *et a*l.,1976) and many processes in a variety of workplaces were contaminated with PAHs (Jongeneelen et al., 1988).

The physical properties and biological activity of PAH vary widely. For example, BaP(Bap) has the lowest vapour pressure and NAP has the highest vapour pressure. In the room temperature vapour pressure of NAP was found eleven-fold magnitude higher than that of BaP (Hussein *et al*., 2016; Donald *et al*., 1992). PAHs with lower vapour pressures (e.g., BaP) were tending to particle phase, while PAHs with higher vapour pressures (e.g., NAP) will tend to be associated with the vapour phase. As a result, the relative distribution of PAHs in the two phases available in

the environment Xiang *et al.*, 2007; Yang *et al.*, 2010). Vapour pressure increased markedly with ambient temperature (Murray *et al*., 1974) which additionally affects the distribution co-efficient between gaseous and particulate phases.

Why PAHs are such a concern: PAHs are an alarming group of substances for humans and environmental organisms. Many PAHs are carcinogenic, mutagenic, and/or toxic for reproduction (Crone and Tolstoy, 2010). Some PAHs are at the same time persistent, bioaccumulative, and toxic (PBT) for humans and other organisms. Persistent means that the substances remain in the environment for a long time and are hardly decomposed there. Bioaccumulative chemicals accumulate in organisms – including the human body. Since the degree of exposure to persistent toxic pollutants in the workplace are much higher than the outer environment, the workers generally considered as more risk and there is need of regular monitoring of exposure assessment and internal dose of toxic pollutants like PAHs.

1.2 Occupational Exposure

Exposure of PAHs can occur due to occupational sources (asphalt industry, foundry, coke plants, petrol refineries, aluminum industry) and non-occupational sources (incomplete combustion of biomass, smoking, diesel exhaust, grilled food). Occupational exposure to PAHs in several work environments can lead to body burdens among exposed workers that were considerably higher than those in the general population. In particular, industrial processes that involve the pyrolysis or combustion of coal, use of coal-derived products were major sources of PAHs. PAHs were directly emitted in the work environment at the time of heating and cooling processes at asphalt work process and aromatics and PAHs are emitted for asphalt paving applications (Butler *et al.* 2000, Burstyn *et al*. 2002, Ruhl *et al.* 2006).

PAHs are entering into the body via inhalation, ingestion and dermal contact (Roggi *et al*., 1997) and by all routes (inhalation, ingestion, and skin contact). After PAHs were swallowed, breathed in, or in some cases, passed through the skin, the body

converts PAHs into breakdown products called metabolites that pass out of the body in the urine and feces (Wu *et al*., 1998). It comprises group of similar organic compounds mainly hydrogen and carbon with at least two benzene rings. The properties of the individual PAH depend on the number of hydrocarbon rings. PAHs were generally lipophilic, which means they dissolve poorly in water but well in fats and oils. This tendency increases with a growing number of rings the more fat-soluble are the substance and the better it accumulates in the fatty tissue of organisms in any kind of living organism. PAHs consider as environmentally persistent organic pollutants (Wania *et al.*,1996) and also were either known or suspected to be carcinogenic (IARC, 2010; Schulte, 2007). There was evidence of carcinogenicity in occupations involving exposure to PAH mixtures containing benzo[a]pyrene, such as aluminum production, chimney sweeping, coal gasification, coal-tar distillation, coke production, iron and steel founding, and paving and roofing with coal tar pitch (IARC, 2010; *Baan et al., 2009*; Straif et al., 2005).

1.3 PAHs and Risk

Occupational risk connected with the carcinogenic agent like PAHs in workplace air was very much concern, even if the exposure is lower than permissible limits. Once PAHs entered into the body, they are metabolized in a number of organs (including liver, kidney, and lungs), excreted in bile, urine or breast milk and stored to a limited degree in adipose tissue. The lipophilicity of PAHs enables them to readily penetrate cellular membranes (Yu, 2005). Subsequent metabolism renders them more water-soluble, making them easier for the body to remove. However, PAHs can also be converted to more toxic or carcinogenic metabolites (John *et al.*,1996). Epidemiological studies face difficulties in establishing correlations between exposures and effects on human health. In such situations, the sensitivity of biomarkers allows detection and measurement at exposure end. Biomarkers can be measured after exposure to food, environmental, or occupational sources to elucidate dose-effect relations in risk assessment, clinical diagnosis, and other forms of monitoring. Biomarkers have been considered promising and received the

greatest attention in studying populations exposed to PAHs. 1-OHP was the most widely used metabolite in PAH exposure, since PYR is one of the most abundant hydrocarbons in all PAH mixtures. Also, OHPHE biomarker was the corresponding markers of PHE also got recent attention as metabolites.

1.4 Scenarios of foundry and asphalt industry in India and PAHs exposure

Foundry: The Indian foundry industry is the sixth largest in the world after USA, China, Japan, Russia & Germany. It is second largest after china in terms of units & number of people employed. Over 4,500 recognized foundry units, including small, medium & large scale sectors situated all over the country and approximately 90% are in the smaller scale. (Metalworld, April, 2008). The industry directly employs over 5, 00,000 people and indirectly over 1, 50,000 people. The smaller units were mainly dependent on manual labors. However, the medium & large units are semi/ largely mechanized & some of the large units are world class.

The various types of castings which are produced as ferrous, non-ferrous, aluminum alloy, graded cast iron, ductile iron, steel, etc for application in automobiles, railways and other special applications in our modern life. However, grey iron castings are the major share of approximate 70% of total castings produced. The production of metal casting is a complex process that had long been associated with worker injuries, illness and expose to chemical and physical hazards generated during working hours.

PAHs result from thermal decomposition of carbonaceous ingredients in foundry sand. During casting, PAHs are formed and partly vapourised under the extremely hot and reducing conditions at the mould-metal interface. It is adsorbed onto soot, fume or sand particles and spread throughout the workplace during shake-out and other process. Organic binders, coal powder and other carbonaceous additives are the also predominant sources of PAHs in iron and steel foundries. In some cases, exhaust gases from engines, furnaces and ovens may increase the exposure of workers to these compounds (IARC, 1984).

The pyrolysis of oil, grease, and rubber in the furnace during the melting and the decomposition of the organic ingredients of molding sand in the casting process may produce a complex mixture of organic compounds, including Polyaromatic hydrocarbons. In iron foundries PAHs are present in both the gaseous phase and adsorbed on to dust.

Asphalt: Today, India has a large and extensive road network aggregating to around 4.8 million kilometers, the second largest in the world after USA. Road transportation is the dominant mode of transportation in India in terms of traffic share. It carries almost 86% of the passenger traffic and 63% of the total freight traffic. In 2010, road transport accounted for a share of 5.4 percent in GDP, whereas the overall share of the transport sector was 6.4 percent of GDP. The Indian road construction industry is highly unorganized and fragmented. Only about 0.4% of the 250,000 contractors in India can be classed as medium to large firms (based on the number of people employed per firm). Many of the medium and large construction firms were still family owned and lack professional management and work culture.

Asphalt fume exposure potential, including the quantity and nature of organic compounds, is directly dependent upon the specific application process conditions including temperature. It is known that PAH emissions from asphalt job are highly temperature-dependent. It has also been reported by various authors that only simple aromatics and very low amounts of 2-3 ring PAHs are emitted at temperatures typically employed for asphalt paving applications. In asphalt production plant a single worker controls the entire asphalt plant mixing process. The other persons on site are loaders. These workers tend to be very mobile. These workers come in direct contact with a sustained fume environment and exposed to bitumen fume at the plant site. The paving workers are exposed to hot-mix asphalt while resurfacing roads. On the job-sites, the asphalt was loaded into the front hopper of a paving machine while the screed (attached to the back of the paving machine) is used to adjust the thickness and width of the applied mix. The specific characteristics of the asphalt is varies by job and type of road.

In India, workers engaged in both asphalt and foundry associated jobs were not considered much important to safety aspects on organic pollutants like PAHs and other VOCs compounds in the developing country. These asphalts mixing and road paving workers constitute the largest group considered as an unorganized sector in our country. These unorganized sector of workers, which constitutes a major workforce in the country were exposed to a variety of toxic substances and air pollution in the workplace. Most of these people are working without protective devices and also were not considered as organized sector under Indian Factory Act, 1948.

Therefore, the present study was conducted to evaluate the exposure to PAHs and bio-monitoring its metabolites to find out the risk associated with these two different occupational groups based on the following objectives

1.5 Objectives

- Personal monitoring of respirable suspended particulate matter in occupational groups, namely - (i) Asphalt workers and (ii) Foundry workers.
- Quantification of Total PAH in these exposed occupational group.
- To study the concentration of different biomarkers in urine samples of the above group exposed to PAHs.
- To compare the exposure and biomarkers in these two-different occupational group.

Chapter~2

Literature review

Chapter- 2

Literature review

2.1 PAHs and it's nature

The term PAHs commonly refer to a large class of organic compounds that contained only carbon and hydrogen alongside two or more fused aromatic rings arranged in a straight line, angled, or in clusters. Differences in the configuration of the rings may lead to differences in physicochemical nature. They are relatively insoluble in water, and most can be photo-oxidized and degraded to simpler substances. There are more than different 1000 PAHs, generally present mostly in complex mixtures and rare as a single substance, of which 16 are classified by the U. S. Environmental Protection Agency as priority pollutants for environmental investigation. The sixteen PAHs compounds are NAP, ACE, ACY, ANT, PHE, FLU, FLT, BaA, CHR, PYR, BaP, BbF, BkF, DahA, BghiP and IND.

These priority PAHs are selected due to the availability of data related to potential health effects. PAHs are ubiquitous and persistent as a consequence of natural (forest fires and volcanic eruptions) and human activities (Jongeneelen, 2001). In addition, these PAHs are known to be more persistent in the environment and typically have the highest concentrations in nature (ASTDR 1995). The sixteen PAHs compounds in Table-1 are classified by the USEPA as priority PAHs pollutants and figure-2.1 shown structural compositions of these 16 PAHs compounds.

2.2 Physical and chemical properties of PAHs

The physical and chemical properties are largely determined by the conjugated alpha electron systems, which vary fairly regularly with the number of rings and

molecular weight, giving rise to a more or less wide range of values for each parameter within the whole class. Physical and Chemical Properties relevant to the toxicological and eco-toxicological evaluation of the PAH are summarized in Table-2.2. The general characteristics common to the class are high melting and boiling points; low vapour pressure and very low solubility in water. PAHs are soluble in many organic solvents and are highly lipophilic. Vapour pressure was tended to decrease with increasing molecular weight varying by more than 10 orders of magnitude. This characteristic affects the adsorption of individual PAH onto particulate matter in the atmosphere and their retention in particulate matter during sampling on filters (Thrane & Mikalsen, 1981). Vapour pressure increased markedly with ambient temperature (Murray et al., 1974) which additionally affects the distribution co-efficient between gaseous and particulate phases (Lane, 1989). Solubility in water tends to decrease with increasing molecular weight.

As pure chemicals, these compounds are colorless, white or pale yellow solids. Their physicochemical properties, vapor pressure and solubility vary according to their molecular weight. PAHs possess a highly characteristic UV absorbance spectrum, although some may be fluorescent (Fetzer & Biggs, 1994).

PAHs are present in the atmosphere in the gaseous phase or adsorbed to particulates. In general, PAHs having two or three rings (NAP, ACE, ACY, ANT, PHE and FLU) were present in the air, predominantly in the gaseous/vapour phase. PAHs that had four rings (FLT, PYR and CHR) exist both in the vapour and in the particulate phase, and PAHs having five or more rings (BaP, BbF, BkF,DahA, BgjiP, IND) are found predominantly in the particle phase. Atmospheric residence time and transport distance depend on the size of particles to which PAHs are adsorbed and on climatic conditions. About 90–95% of particulate PAHs are associated with particle diameters < 3.3 µm. Particles with a diameter range of 0.1–3.0 µm, with which airborne PAHs are principally associated, are expected to have atmospheric residence times of a few days and, hence, can undergo long-range transport.

Table-2.1: 16 priority PAHs classified by the U. S. Environmental Protection Agency

PAHs compounds	Ring	Molecular Weight (g/mol)
Naphthalene	2	128.17
Acenaphthene	3	154.21
Acenaphthylene	3	152.2
Anthracene	3	178.23
Phenanthrene	3	178.23
Fluorene	3	166.22
Fluoranthene	4	202.26
Benzo(a)anthracene	4	228.29
Chrysene	4	228.29
Pyrene	4	202.26
Benzo(a)pyrene	5	252.32
Benzo(b)fluoranthene	5	252.32
Benzo(k)fluoranthene	5	252.32
Dibenz(a,h)anthracene	6	278.35
Benzo(g,h,i)perylene	6	276.34
Indeno(1,2,3-cd) pyrene	6	276.34

Source: USEPA, 2004

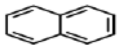
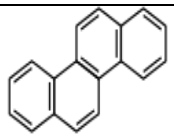
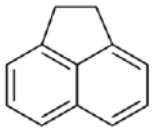
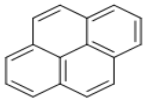
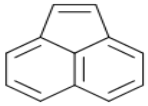
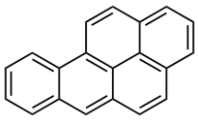
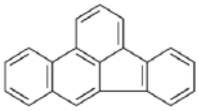
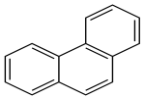
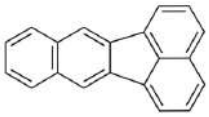
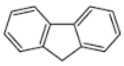
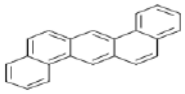
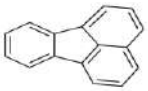
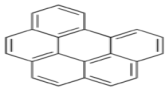
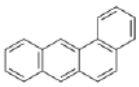
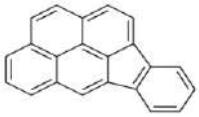
PAHs compounds	Structure	PAHs compounds	Structure
NAP-		CHR-	
ACE-		PYR-	
ACY-		BaP-	
ANT-		BbF-	
PHE-		BkF-	
FLU-		DahA-	
FLT-		BghiP-	
BaA-		IND-	

Figure-2.1: Structure of 16 priority PAHs classified

Source: USEPA, 2004

2.3 Environmental persistence of PAHs

PAHs released to the atmosphere are subject to short- and long-range transport and are removed by wet and dry deposition on to soil, water and vegetation. In surface water, PAHs can volatilize, photolysis, biodegrade or bind to suspended particles or sediments. Based on field observations and laboratory studies with model aerosols, there is indication that abiotic degradation of PAHs on or in particles are hindered in the ambient atmosphere. A possible explanation is that PAHs diffuse partly from the particulates' surface into the particle volume, where degradation by the OH radical was not significant (Behymer & Hites 1988; Finizio *et al.* 1997; Masclet *et al*. 1995; McDow et al. 1996; Offenberg & Baker 2002; Reyes et al. 2000).

2.4 Occupational exposure history perspective

Percival Pott, an English surgeon, reported first that connection between occupational exposure and cancer. In 1775, he described an unusually high incidence of scrotal cancer among London chimney sweeps and suggested that was due to their exposure to soot and ash. Since then, the other coal tar-related cancers have been induced in laboratory animals and found in humans (Kennaway 1995; Kjaerheim 1999). The PAH BaP, which was isolated from coal tar in the 1930s, was determined to be carcinogenic when applied to the skin of test animals. In 1947, the relationship between lung cancer and working conditions of gas industry workers and those working with coal tar was established (Kenneway 1995). An increased incidence of cancers, particularly of the lung, was shown in epidemiologic studies of gas workers (Doll *et al*., 1965). Several epidemiologic studies have shown increased that the cancer mortality rate in workers exposed to PAH mixtures. Exposure to other potentially carcinogenic substances often occurred in these studies [Lloyd 1971; Mazumdar *et al.,* 1975; Redmond *et al*., 1972; and Hammond *et a*l. 1976].

Table-2.2: Physical and chemical properties of Polyaromatic hydrocarbon

Compounds	Melting Point (°C)	Boiling Point (°C)	Vapour Pressure (°C)	n-Icranol :Water Partition Co-efficient	Solubility in water at 25°C (µg/Lit.)	Henry's law Constant at 25°C9 Kpa)	Categories of Half-life in various environment			
							Air	Water	Soil	Sediment
Napthelene	81	217.9	10.4							
Acenaphthylene	92–93	279	8.9×10^{-1}	4.07	–	1.14×10^{-3}	2	4	6	7
Acenaphthene	95	295	2.9×10^{-1}	3.92	3.93×10^{3}	1.48×10^{-2}				
Fluorene	115–116	340	8.0×10^{-2}	4.18	1.98×10^{3}	1.01×10^{-2}	2	4	6	7
Phenanthrene	100.5	342	1.6×10^{-2}	4.6	1.29×10^{3}	3.98×10^{-3}	2	4	6	7
Anthracene	216.4	375	8.0×10^{-4}	4.5	73	7.3×10^{-2}	2	4	6	7
Fluoranthene	108.8	393	1.2×10^{-3}	5.22	260	6.5×10^{-4}	3	5	7	8
Pyrene	150.4	400	6.0×10^{-4}	5.18	135	1.1×10^{-3}	3	5	7	8
Benz[*a*]anthracene	160.7	448	2.8×10^{-5}	5.61	14	–	3	5	7	8
Chrysene	253.8	481	8.4×10^{-5}	5.91	2.0	–	3	5	7	8
Benzo[*b*]fluoranthene	168.3	480	6.7×10^{-5}	6.12	1.2 (20 °C)	5.1×10^{-5}				
Benzo[*j*]fluoranthene	165.4	480	2.0×10^{-6}	6.12	2.5	–				
Benzo[*k*]fluoranthene	215.7	496	1.3×10^{-8}	6.84	0.76	4.4×10^{-5} (20 °C)	3	5	7	8
Benzo[*a*]pyrene	178.1	536	7.3×10^{-7}	6.50	3.8	3.4×10^{-5}	3	5	7	8
Indeno[1,2,3-*c,d*]pyrene	163.6	524	1.3×10^{-8}	6.58	62	2.9×10^{-5}(20 °C)				
Dibenz[*a,h*]anthracene	266.6	594	1.3×10^{-8}	6.50	0.5 (27 °C)	7×10^{-6}	3	5	7	8
Dibenzo[*a,i*]pyrene	282	525	3.2×10^{-10}	7.30	0.17	4.31×10^{-6}				

Class	Half-life (hours)		Class	Half-life (hours)	
	Mean	Range		Mean	Range
1	17	10–30	**5**	1700	1000–3000
2	55	30–100	**6**	5500	3000–10 000
3	170	100–300	**7**	17 000	10 000–30 000
4	550	300-1000	**8**	55 000	>30 000

Source: WHO 1998.

Because of the complex profile of PAHs in the environment and in workplaces, human exposure to pure, individual PAHs has been limited to scientific experiments with volunteers, except in the case of NAP (Rengarajan *et al*, 2015). After dermal application of ANT, FLT and PHE induced specific skin reactions, and BaP induced reversible, regressive verrucae that were classified as neoplastic proliferations. The systemic effects of NAP are known from numerous cases of accidental intake, particularly by children. The lethal oral dose was 5000–15 000 mg for adults and 2000 mg taken over two days for a child. The typical effect after dermal or oral exposure was acute hemolytic anemia, which can also affect fetuses translucently. Similarly, in aluminum plants, asthma-like symptoms, lung function abnormalities and chronic bronchitis have been observed. Coke-oven workers were found to have decreased serum immunoglobin levels and decreased immune function. Occupational exposure to NAP for 5 years was reported to cause cataract (ASTDR, 1990).

Acute or Short-term Health Effects-The effects on human health will depend mainly on the length and route of exposure, the amount or concentration of PAHs one was exposed to, and of course the innate toxicity of the PAHs. A variety of other factors can also affect health impacts including subjective factors such as pre-existing health status and age. The ability of PAHs to induce short-term health effects in humans was not clear. Occupational exposures to high levels of pollutant mixtures containing PAHs have resulted in symptoms such as eye irritation, nausea, vomiting, diarrhoea and confusion. However, it was not known which components of the mixture were responsible for these effects and other compounds commonly found with PAHs may be the cause of these symptoms. Mixtures of PAHs were also known to cause skin irritation and inflammation. NAP was direct skin irritants while ANT and BaP were reported to be skin sensitizers, i.e. caused an allergic skin response in animals and humans (IPCS, 1998).

Chronic or Long-term Health Effects-Health effects from chronic or long-term exposure to PAHs may include decreased immune function, cataracts, kidney and liver damage, e.g. jaundice), breathing problems, asthma-like symptoms, and lung

function abnormalities, and repeated contact with skin may induce redness and skin inflammation. Naphthalene, a specific PAH, can cause the breakdown of red blood cells if inhaled or ingested in large amounts. If exposed to PAHs, the harmful effects that may occur largely depending on the way people were exposed.

The toxic effect of most concern from exposure to PAHs was cancer. Occupational exposure to soot as a cause of scrotal cancer was noted for the first time in 1775. Later, occupational exposure to tars and paraffin was reported to induce skin cancer. The lung was now the main site of PAH-induced cancer, whereas skin tumors have become rarer because of good personal hygiene.

Epidemiological studies have been conducted of workers exposed at coke ovens during coal coking and coal gasification, at asphalt works, foundries and aluminium smelters, and to diesel exhaust. Increased lung tumour rates owing to exposure to PAHs have been found in coke-oven workers, asphalt workers and workers in Södeberg potrooms of aluminium reduction plants. The highest risk was found for-coke oven workers, with a standardized mortality ratio of :1:95 (1.59-2.33) (Costantino *et. al*, 1995). Analysis of the relative risks and the numbers of deaths from lung cancer resulted in the conclusion that 124 deaths occurred among these coke-oven workers over a period of 30 years that can be attributed to exposure to coal-tar pitch volatiles, 2.3% of the cohort.

Workers in industries or trades using or producing coal or coal products were at highest risk for PAH exposure. Those workers include aluminum workers, asphalt workers, carbon black workers, chimney sweeps, coal-gas workers, coke oven workers, fishermen (coal tar on nets), graphite electrode workers, machinists, mechanics (auto and diesel engine), printers, road (pavement) workers, roofers, steel foundry workers, tire and rubber manufacturing workers, and a many job category.

Polycyclic Aromatic Hydrocarbons (PAHs) were a group of chemicals that occur naturally in coal, crude oil and gasoline. Incomplete combustion of organic material results in emission of PAHs (ATSDR, 1996). These molecules consist of two or more

aromatic rings fused in linear, angular or cluster arrangements and by definition were composed of hydrogen and carbon. PAHs containing up to six fused aromatic rings are often known as "small" PAHs while those containing more than six aromatic rings were called "large" PAHs. PAHs may distribute in water, soil and the atmosphere, according to different weather and geographical factors. Although industrial activities such as coke manufacturing or asphalt production were major contributors to PAH emissions, incineration, power generation and several mobile sources also emit a considerable amount of PAHs. Significant sources of PAHs in surface waters include deposition of airborne PAHs, municipal wastewater discharge, urban storm-water runoff, and industrial waste.

Food groups that tend to have the highest levels of PAHs include charcoal broiled or smoked meats, leafy vegetables, grains, and vegetable fats and oils (Yu, 2005). Therefore, workers in these industries and the general population were continually exposed to different concentrations of PAH mixtures. The Agency for Toxic Substances and Disease Registry (ATSDR) has grouped 17 PAHs according to their health effects (ATSDR, 1996). The United States Environmental Protection Agency (EPA) has designated 16 PAH compounds as priority pollutants (EPA, 2009) (Table 2.1). The International Agency for Research on Cancer (IARC) has classified some these compounds as carcinogenic (group 1) or likely carcinogenic (group 2A) to humans, for example benzo[a]pyrene and dibenz[a,h]anthracene, respectively (IARC , 2010). Finally, the National Institute of Standards and Technology has created a classification of PAHs according to their symbols, molecular formulas, class and notation among other properties (NIST, 2010).

IARC reported that workers from industrial settings where airborne PAH levels are high (IARC, 1985). The toxic effect of most concern from exposure to PAHs was cancer. IARC considers several purified PAHs and PAH derivatives to be probable (group 2A) or possible (group 2B) human carcinogens. Some mixtures containing PAHs are known human carcinogens (group 1). The degrees of carcinogens as per IARC classification were shown in Table-2.3.

Table-2.3: IARC list of priority carcinogenicity classification

Compounds	Carcinogenicity Classification Group*	Compounds	Carcinogenicity Classification Group*
NAP	**2B**	**CHR**	**2B**
ACE	**3**	**PYR**	**3**
ACY	**4**	**BaP**	**1**
ANT	**3**	**BbF**	**2B**
PHE	**3**	**BkF**	**2B**
FLU	**3**	**DahA**	**2A**
FLT	**3**	**BghiP**	**3**
BaA	**2B**	**IND**	**2B**

IARC: http://monographs.iarc.fr/ENG/Classification/index.php. Group 1: Carcinogenic to humans; Group 2A: probably carcinogenic to humans; Group 2B: possibly carcinogenic to humans; Group 3: not classifiable as to carcinogenicity to humans; Group 4: probably not carcinogenic to humans.

2.5 Classifications of agents

IARC classifies carcinogens in five categories ranging from carcinogenic to humans (Group 1) to probably not carcinogenic to humans (Group 4). The classification indicates the weight of the evidence as to whether an agent was capable of causing cancer.

Group 1: The agent is carcinogenic to humans. This category is used when there is sufficient evidence of carcinogenicity in humans. In other words, there is convincing

evidence that the agent causes cancer. The evaluation was usually based on epidemiological studies showing development of cancer in exposed humans. Agents can also be classified in Group 1 based on sufficient evidence of carcinogenicity in experimental animals supported by strong evidence in exposed humans that the agent has effects that are important for cancer development.

Group 2: This category includes agents with a range of evidence of carcinogenicity in humans and in experimental animals. At one extreme are agents with positive, but not conclusive evidence in humans. At the other extreme are agents for which evidence in humans was not available, but for which there is sufficient evidence of carcinogenicity in experimental animals. There were two subcategories, indicating different levels of evidence.

Group 2A: The agent is probably carcinogenic to humans. This category is used when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. Limited evidence means that a positive association has been observed between exposure to the agent and cancer, but that other explanations for the observations (technically termed chance, bias, or confounding) could not be ruled out.

Group 2B: The agent is possibly carcinogenic to humans. This category is used when there was limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals. It may also be used when the evidence of carcinogenicity in humans does not permit a conclusion to be drawn (referred to as “inadequate” evidence) but there was sufficient evidence of carcinogenicity in experimental animals.

Group 3**:** The agent is not classifiable as to its carcinogenicity to humans. This category is used most commonly when the evidence of carcinogenicity is inadequate in humans and inadequate or limited in experimental animals. Limited evidence in experimental animals means that the available information suggests a carcinogenic effect but was not conclusive.

Group 4**:** The agent is probably not carcinogenic to humans. This category is used when there is evidence suggesting lack of carcinogenicity in humans and in experimental animals.

The mechanism and location of the deposition of particulate phase PAH in the lung are also affected by particle size. The large particles tend to impact on the upper regions of the lung and small particles diffuse to the surface of the alveoli. Particles in the accumulation mode size range have the lowest faction deposited in the lung.

Data obtained as a result of epidemiological studies under occupational conditions suggest that there was an association between lung cancer and exposure to PAHs. The weight of evidence arising from epidemiological studies based on inhalation and occupational exposure to PAHs suggests an increased risk of harmful health effects, mainly lung cancer. PAHs were also genotoxic carcinogens, inducing chromosomal which has been recognized as cytotoxic and carcinogenic in humans (Boffetta et al., 1997; IARC1987, 1989; Tremblay et al., 1995; Armstrong et al., 1994). PAH emissions from industries were produced by burning fuels such as gas, oil, and coal. PAHs can also be emitted during the processing of raw materials like primary aluminum. Sources of PAHs include emissions from industrial activities, such as primary aluminum and coke production, petrochemical industries, rubber tire and cement manufacturing, bitumen and asphalt industries, wood preservation, commercial heat and power generation, and waste incineration (Lee, 2010; Yang *et al.,* 1998) reported emissions of PAHs from various industrial stacks: a blast furnace, a basic oxygen furnace, a coke oven, an electric arc furnace, a heavy oil plant, a power plant, and a cement plant. Because PAHs were abundant in many petroleum and coal-derived products, workers are exposed to PAHs in industries (Boffetta *et al.,* 1997; Pooeniak, 2005). Exposures to PAHs were greatest in the aluminum, iron, and steel industries (Höflich *et al.,* 2005; Ramírez *et al.,* 2011). Other sources of occupational exposure to PAHs were from inhalation of engine exhaust (Leea *et al*., 2003; Lioy *et al*., 1990). The most toxic effects on humans due to PAHs were cancer.

Exposure of PAHs can occur due to occupational sources (asphalt industry, foundry, coke plants, petrol refineries, aluminum industry) and non-occupational sources (smoking, diesel exhaust, grilled food) and can occur via inhalation, ingestion and dermal contact (Roggi *et a*l., 1997). Historically, the assessment of occupational exposure to PAHs has relied primarily on air monitoring. However, there has been increasing evidence that dermal contact was another primary route of exposure to PAHs (Quinlan *et* al, 1995; Borak *et al.,* 2002). The mounting evidence regarding the exposure of PAHs has largely been due to the increased use of biological monitoring. Asphalt mixture was a combination of bitumen and small amounts of sulphur, oxygen, nitrogen and traces of other minerals.

2.6 PAHs biomarkers for human health risk assessment

Biological monitoring of exposure is "the measurement and assessment of agents or their metabolites either in tissue, secrete, excreta or any combination of these to evaluate exposure and health risk compared to an appropriate reference" (Zeilhuis and Henderson, 1986). In other words, biological monitoring of industrial hazards is the evaluation of the internal exposure "internal dose" of a worker or a group of workers to the hazardous agent/s by a biological method. It is important to define the term "internal dose" before its usage since it may mean the amount of agent absorbed in a specific period of time or the amount already stored in the body (body burden). Biological monitoring is related to "uptake" (intake x fractional absorption) through several pathways simultaneously. It is based upon the knowledge of the impact of man on the agent. An understanding of biological monitoring indices depends on knowledge of the xenobiotic's toxicokinetics including uptake, distribution, absorption, biotransformation, accumulation and elimination.

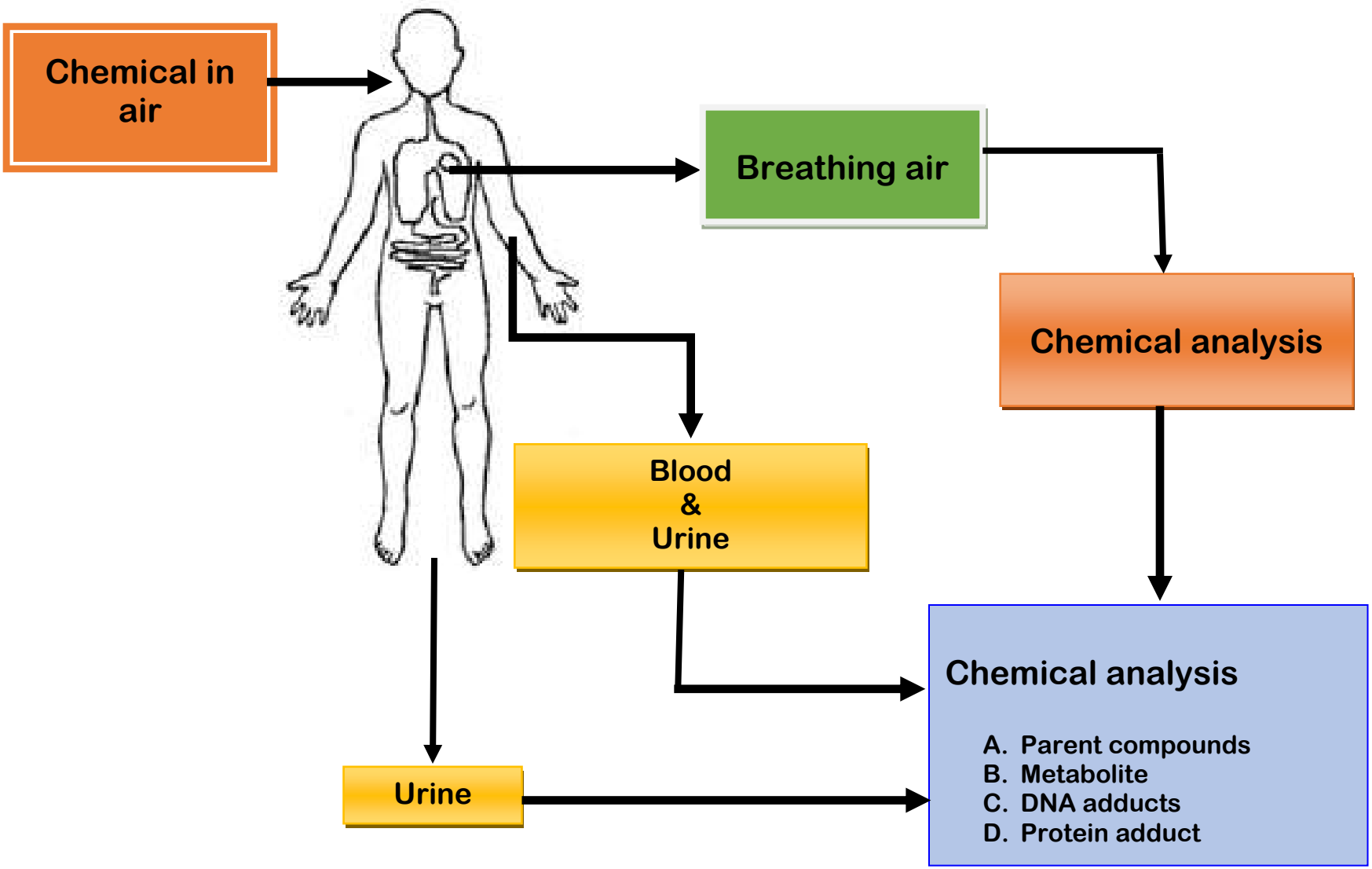


Figure 2.2: Biological Monitoring of chemical exposure

Polycyclic aromatic hydrocarbons (PAH) are widely distributed in the environment, and some were carcinogenic to human beings. The study of biomarkers has helped clarify the nature and magnitude of the human health risks posed by such substances. Biomarkers have been considered promising and have received the greatest attention in studying populations exposed to chemical contaminants. The World Health Organization (IPCF-WHO, 2001) defines biomarker as “any substance, structure, or process that can be measured within an organism or its products and influences or predicts the incidence of harmful effects or disease”.

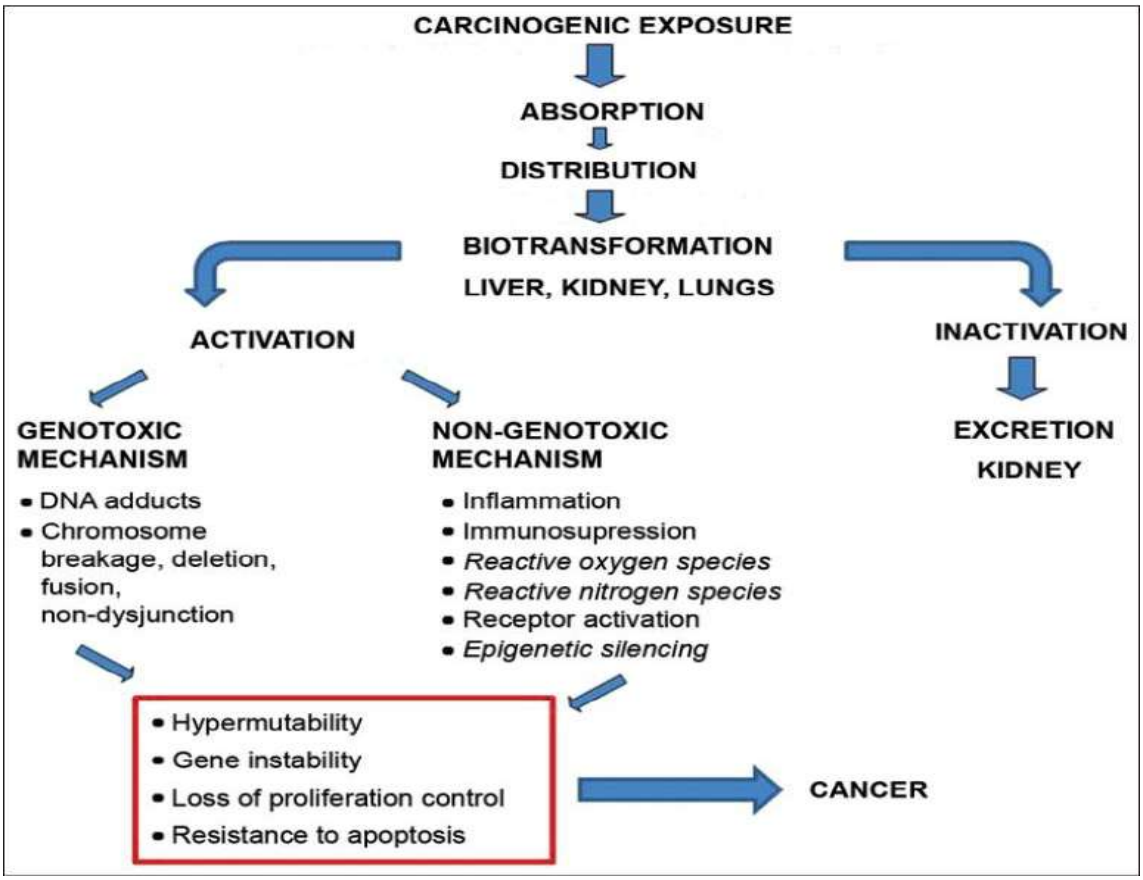


Figure-2.3: Mechanism of carcinogenesis pollutants (Khambeta *et al*., 2014**)**

Biomarkers can be measured after exposure to food, environmental, or occupational sources to elucidate dose-effect relations in risk assessment, clinical diagnosis, and other forms of monitoring. Biomarkers were classified in three categories: (i)

biomarkers of exposure, which involve detection and measurement of an exogenous substance, metabolite, or product of interaction between the xenobiotic and some molecule or target cell; (ii) biomarkers of effect, including measurement of biochemical and physiological alterations that can be related to the occurrence of disease or harm to health; and (iii) biomarkers of susceptibility, indicative of an organism's inherent or acquired ability to respond to the challenge of exposure to xenobiotics.

The integrated application of epidemiological studies, environmental behavior of contaminants, and biomarkers can provide more solid data in relation to the human health risks resulting from environmental exposure to chemical substances like polycyclic aromatic hydrocarbons (PAH). Some PAHs were now recognized as carcinogens or probable carcinogens for human beings and other mammals. Recent studies demonstrate that PAH with more than three aromatic rings account for 70-90% of the total carcinogenic effect related to these sources, thus posing a serious health threat (Jacob *et.al*, 2002).

In the human body, since PAH are lipophilic substances, they were readily dissolved and transported by cell membrane lipoproteins. The absorption rate depends on the specific PAH. In general, they were distributed throughout the body and found in any internal organ or tissue, particularly in lipid-rich tissues and the gastrointestinal tract, through the re-absorption of the product of hepatobiliary excretion. Exposures and effects on human health, the sensitivity of biomarkers allows detection and measurement at reduced concentrations, thus expanding the reach of dose-response studies for assessing sub-lethal effects. Exposure assessment of PAH mixtures can be based on one that was representative of the group, like BaP or a group of PAH-BaA, CHR, BbF, BkF, BaP, DahA, BghiP, and IND (Brandt *et al.*,2003). After the substance enters the body, the unaltered (parental) substance or its metabolites were searched for in the urine, blood, feces, or other bodily fluids or tissues. Since parental PAH generally present a reduced plasma half-life, hydroxy metabolites were the most frequent option for investigation.

Hydroxy PAH metabolites: biomarkers of internal dose: PAH metabolism into more soluble forms was a necessary step for their excretion. The parent compound was generally oxidized by phase 1 enzymes through hydroxylation catalyzed by cytochrome P450 monooxygenase enzymes; hydrolysis and reduction can also occur. These phases I metabolites bind to glutathione, sulfates, or glucuronic acid to form phase II metabolites in order to form more polar and water-soluble substances than the original substances, thereby greatly facilitating their excretion. The metabolites and conjugates were excreted via the urine and feces, but conjugates excreted in bile can be reabsorbed in the intestine. The hydroxyl metabolites can be used as biomarkers based on their ability to indicate the internal dose received (Jacob *et al.*, 2002; Hansen *et al.*, 2008).

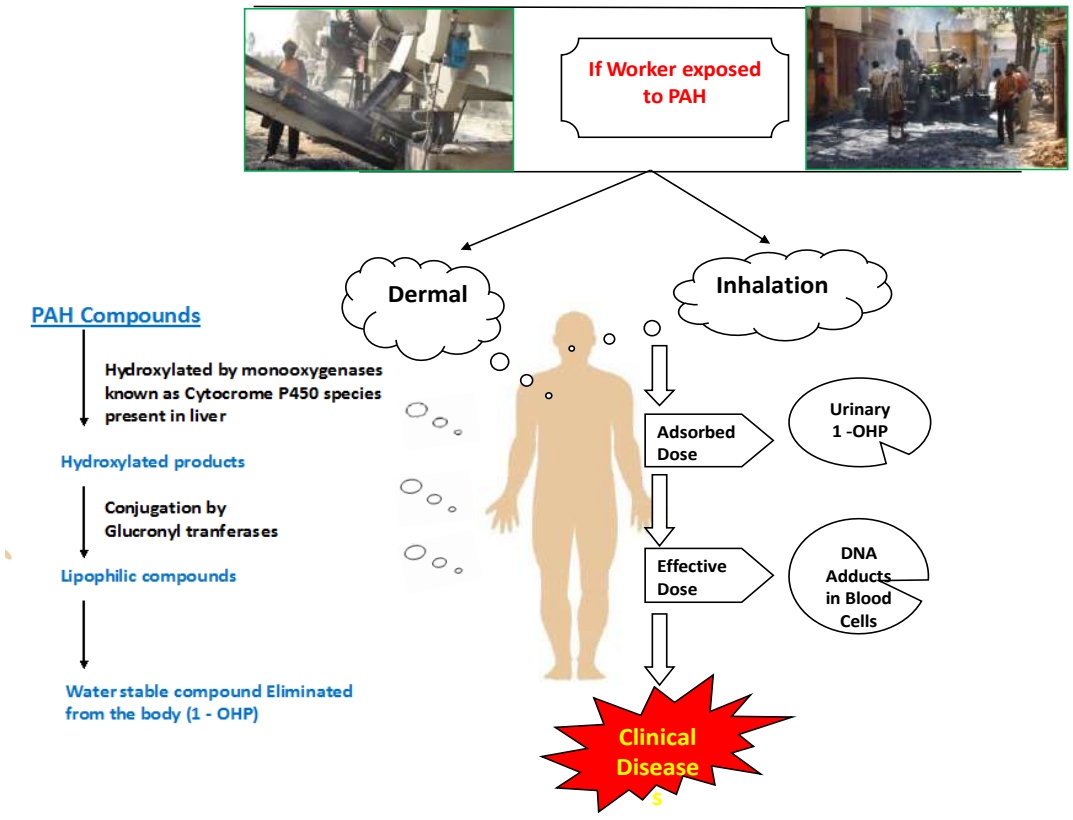


Figure-2.4: Metabolic pathway and mechanism of polyaromatic hydrocarbon in human body

The majority of these reactions result in detoxification, but compounds can be produced that were highly reactive as electrophilic PAH metabolites, which can form covalent interactions with proteins and nucleic acid, resulting in adducts that can compromise normal cell functioning, triggering a series of harmful effects. Metabolites excreted in the urine provide more appropriate estimates of total ingestion as com- pared to exposure assessments based on environmental data. The choice of the urinary metabolite should consider the constituent of the most common PAH. In cases of complex mixtures, more than one biomarker should be considered to ensure adequate evaluation. 1-OHP was the most widely used metabolite in PAH exposure, since PYR was one of the most abundant hydrocarbons in all PAH mixtures and its has principal metabolite 1-OHP formed in mammals. Representing a sensitive biomarker of exposure, 1-OHP was recommended by various authors as the most relevant parameter in estimates of individual exposure to PAH. 1-OHP was excreted in urine; it currently was indicated as the most relevant biomarker of PAH exposure to evaluate total-body exposure to PAH (Jongeneelen *et al.,*1986). The 1-OHP content in urinary excretion was determined not only by the amount of PAH uptake, but also by differences in their distribution, metabolism and excretion. Since there were significant correlations between urinary 1-OHP concentrations and occupational and environmental exposure to all PAH or pyrene concentrations (Jongeneelen *et al.,1998;* Zhao *et al.,1992; Kanoh et al.,1993; Goen et al.,1995; Dor et al.,1999)*. Factors that may cause inter individual differences in urinary 1-OHP levels were lifestyle factors such as smoking, alcohol consumption and dietary intake (Van *et al.,1994)*; personal factors such as age, sex and body mass index (Roggi *et al.,1997)*; and airborne PAH concentrations (Perico *et al.,2001)*. In addition, genetic polymorphisms of enzymes have been suggested to explain inter-individual differences in the rate of metabolism and activation/ deactivation of PAH-derived carcinogens (Alexandrie *et al.,*2000; Chuang *et al.,2007)*.

Comparison of different work environments may, however, be difficult because the proportion of PYR in comparison to BaP and other potentially carcinogenic PAHs may vary. For example, the creosote oil used in a wood impregnation plant

contained about 3.4% PYR and less than 0.0004% BaP. Levels of 2–10% PYR and 0.4–0.6% BaP are found in coal-tar, which was the main PAH contaminant in the coke industry, in the primary aluminium industry and during road paving with tar. Polluted ambient air contains about 6.5% BaP and 1.8–2.7% PYR (WHO 1998).

Several authors have tried to establish admissible levels of 1-OHP in urine for specific exposures. According to Jongeneelen (1992), a urinary concentration of 1-HP in coke-oven workers of 4.4 µg/g creatinine reflects concentrations of coal-tar pitch and BaP in the air of 0.2 mg/m^3 and 2 µg/m^3 respectively. A similar value of 4 µg/g creatinine was proposed by Levin *et al*. (1995). A higher value of 6.1 µg/g creatinine was suggested by Van Rooij *et al.* (1993). Tjoe-Ny *et al.* (1993) assumed that exposure to 0.2 mg/m^3 coal-tar pitch or 5 µg/m^3 BaP would result in a urinary concentration of 1-HP of 8.6 µg/g creatinine. On the basis of the logistical regression between the prevalence of abnormal serum high frequency cells PAHs in air or 1-HP in the post-shift urine of non-smoking workers exposed to PAHs, Buchet *et al.* (1995) concluded that the latter should be kept below 6.4 µg/m^3 and 2.7 µg/g creatinine, respectively.

It was not currently possible to assess risk presented by exposure to PAHs solely on the basis of urinary 1-OHP concentrations. An indirect dose–response relationship between urinary 1-OHP level and the relative risk for lung cancer. However, it was estimated for coke-oven workers: 4.4 µg 1-HP/g creatinine was estimated to be equal to a relative risk for lung cancer of approximately 1.3 (Jongeneelen 1992). Because of the varying composition of PAH mixtures, this risk estimation cannot be used for other workplaces or ambient air, where a correction factor may be necessary.

PAH-DNA: biomarkers of effective dose: PAHs play an undeniable role in the induction of human carcinogenesis. There was evidence on the transformation of healthy cells into cancer cells, using in vitro cell culture experiments, animal studies, and in vivo studies with occupationally or environmentally exposed healthy human volunteers and cancer patients. Chemical induction of carcinogenesis was a complex

process; multiple stages involve mutations in cell growth-regulating genes (protooncogenes) and tumor-suppressor genes. DNA adducts have proven to be promising biomarkers, since they consider individual differences in exposure, absorption, and distribution of chemical agents, metabolism to DNA-reactive forms, detoxification in reactive intermediaries, and cell replacement and repair of DNA damage. DNA adducts were used to assess both exposure and cancer risk in humans.

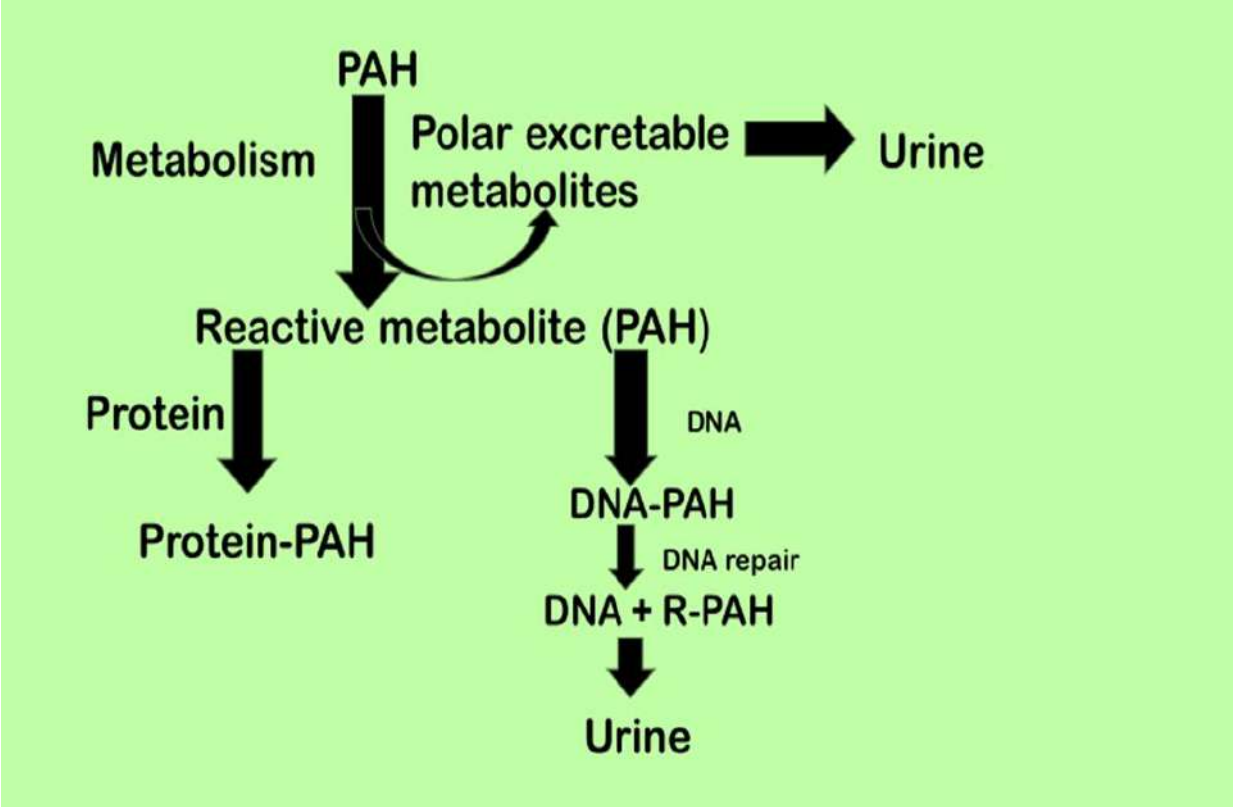


Figure-2.5: Metabolism of PAH leading to protein and DNA adducts (Skipper *et al*., 1990).

Occupational exposure to polycyclic aromatic hydrocarbons (PAHs) has been reported in foundries. A higher risk for DNA damage or oxidative damage lesions was also found in occupationally PAH-exposed groups. A higher concentration of 1-OHP was found in the exposed group (0.322+/-0.289 µg/g creatinine) relative to the control group (0.178+/-0.289 µg/g creatinine) ($p<0.05$). Moreover, higher levels of 1-OHP were found in workers involved in manufacturing processes (0.346 µg/g

creatinine) compared to administrative workers (0.018 µg/g creatinine). A positive correlation was identified between levels of 1-OHP and 8-hydroxydeoxyguanosine (8-OH-dG), DNA strand breakage and malondialdehyde (MDA) in all study subjects. However, when foundry workers were considered based on their specific job categories, a similar trend for 1-OHP and three oxidative damage markers was only found for DNA strand breakage, but not for 8-OH-dG or MDA. Other factors such as furnace equipment, PAH types, and job categories may contribute to different PAH emissions. The study also suggested that co-exposure to metal and PAHs, and smoking status in foundry industries may also cause the oxidative damage in foundry workers (Liu et al., 2010).

Cytogenetic alterations: biomarkers of early effect: In cell cultures of peripheral lymphocytes, cytogenetic alterations like chromosomal aberrations, sister chromatid exchange, and micronucleus induction have been applied as biomarkers of exposure and early effect in exposures to genotoxic carcinogens. Like the majority of biomarkers for genotoxicity, cytogenetic alterations in lymphocytes are estimates obtained from surrogate tissues, presuming that they represent more specific chromosomal alterations, important in the carcinogenesis of target tissues. Evidence has been found in studies showing the high frequency of chromosomal aberrations, but not chromatic exchange or micronucleus induction in peripheral lymphocytes as predictive parameters of increased cancer risk. The relevance of increased frequency of cytogenetic alterations as biomarkers of cancer risk has been corroborated by epidemiological studies suggesting the high frequency of chromosomal aberrations as the best predictive parameter for increased cancer risk.

Genetic polymorphism: biomarkers of susceptibility: Genetic polymorphisms have received increasing attention, since they can modulate the human response to exposures to genotoxic agents, whose role in susceptibility can be studied more easily through the use of biomarkers like cytogenetic alterations. Studies on genetic polymorphism depend on: the biological material examined and the exposure and ethnic composition of the study population. Since cytogenetic markers can represent exposures that occurred months before the tissue sampling, simple measurements

of urinary metabolites or environmental concentrations may not be representative of the most relevant expo- sure period. For different exposure levels, the ex- posed individuals should be grouped in distinct categories and compared to the control group to allow distinguishing exposure-genotype interactions from background biomarker levels. High levels of chromosomal aberrations in peripheral lymphocytes have been observed for the prediction of increased cancer risk for smoking and occupational exposure. Various polymorphisms in enzymes for the metabolism of xenobiotics have indicated an inductive effect on cytogenetic biomarkers. The importance of various genetic polymorphisms in determining the level of cytogenetic alterations depends on the following factors: cytogenetic parameter, chemical agent, and ethnic composition of the study population. Cytogenetic biomarkers can be used to identify sub-groups that are sensitive to carcinogens.

For assessing human exposure to PAH, recent validation studies highlight urinary 1-OHP as a methodology already validated for monitoring exposure and PAH-DNA adducts in lymphocytes as a marker of effective dose. The most promising biomarkers still in the validation process include cytogenetic markers of early effect, evaluation of the frequency of chromosomal aberrations, and micronucleus induction. Future prospects for application of biomarkers to environmental risk assessment of PAH exposure are promising. The expected advances for the coming years are: increased reliability in the exposure assessment and detection of early harm in populations exposed to low doses and to the mixture of chemical substances; increased sensitivity of these studies for the identification of genetic variations linked to chromosomal damage; reduction in the cost of molecular techniques; and increased use of automation. Importantly, biomarkers represent key prospects for expanding scientific research in the environmental health field. However, in the field of science as well, some barriers between areas of knowledge need to be eliminated in order to en- sure the interdisciplinary these studies require, especially in relation to the recognition of the environmental area as an important field in public health.

2.7 Application of risk assessment in occupational PAH exposure

Increasing efforts have focused on risk assessment studies based on models and data for environmental concentrations of the contaminants, a tool already widely adopted in various countries. Risk assessment is understood as consisting of four stages: identification of hazards; evaluation of exposure; dose-response assessment; and risk characterization. Such studies were based on information from experimental toxicity studies and physical and chemical characteristics of the substances and their behavior in the environment. The main advantages of risk assessment are: low cost, lack of need for population interventions; and possibility for use in the assessment of postulated exposure settings, not depending on the existence of real situations of human exposure. However, they are heavily dependent on the existence of toxicological data and models that are not always reliable, especially for low doses, and they do not adequately assess exposure to mixtures of contaminants in fluctuating conditions prevailing in the environment. Recent studies on human exposure to environmental contaminants have incorporated lab- oratory analytical techniques into epidemiological inventories to elucidate the biochemical or molecular basis for the etiology of diseases, thus providing useful information like internal dose and biological effects, using biomarkers.

Analysis of the recent literature shows that the application of biomarkers to environmental risk assessment, especially for human health, has expanded continuously thanks to the advances obtained in characterization and validation studies, although their utilization depends on consistent criteria for defining the study area and population. For assessing human exposure to PAH, recent validation studies highlight urinary 1-OHP as a methodology already validated for monitoring exposure and PAH-DNA adducts in lymphocytes as a marker of effective dose. The most promising biomarkers still in the validation process include cytogenetic markers of early effect, evaluation of the frequency of chromosomal aberrations, and micronucleus induction. Future prospects for application of biomarkers to environmental risk assessment of PAH exposure were promising. The expected

advances for the coming years are: increased reliability in the exposure assessment and detection of early harm in populations exposed to low doses and to the mixture of chemical substances; increased sensitivity of these studies for the identification of genetic variations linked to chromosomal damage; reduction in the cost of molecular techniques; and increased use of automation. Importantly, biomarkers represent key prospects for expanding scientific research in the environmental health field. However, in the field of science as well, some barriers between areas of knowledge need to be eliminated in order to ensure the interdisciplinary these studies require, especially in relation to the recognition of the environmental area as an important field in public health.

Over the last two decades, there has been an increased interest in the assessment of the health risks of populations occupationally exposed to chemical carcinogens. People were exposed to a variety of chemicals as contaminants, both in the general environment and the workplace. PAHs are important to occupational health for several reasons: some are potent carcinogens (Harvey, 1991) and there was strong epidemiological evidence that exposed groups experience excess risk of lung, urinary tract, and skin cancers (Partanen and Boffetta, 1994). Secondly, many processes in a variety of workplaces are contaminated with them (Jongeneelen *et al*., 1988). The acute and chronic toxicity of high and low doses of PAHs in animals have been well researched using in vivo and in vitro techniques (WHO, 1983 and Karcher, 1992). The literature focusing on the effects of long-term exposure to moderate and low levels, such as those found in the roofing and road paving industries was scarce. Hence, estimates of the risk to health of chronic occupational exposure to PAHs have not studied widely. Similarly, a number of animal studies have been conducted on the toxicity of bitumen and bitumen fumes and their effects when administered through different routes. Again, the literature lacks on investigations assessing the risks to health of workers exposed to bitumen fumes. Few occupational health and hygiene studies have been conducted measuring worker's exposure to bitumen/asphalt and their fumes.

PAHs occur in the environment as complex mixtures of many components with widely varying toxic potencies. The profile of compounds are occurring in the air in different occupational environments and in the atmosphere in city agglomerations (e.g. the ratio sum of carcinogenic PAHs and BaP) can differ, as well as the carcinogenic potencies of the individual PAHs (Petry *et al*. 1996). It has therefore been assumed that the development and establishment of potency equivalency factors (PEFs) for PAHs, similar to the concept used in the assessment of mixtures containing polychlorinated dibenzodioxins, dibenzofurans and biphenyls, could help to characterize more precisely the carcinogenic properties of PAH mixtures.

In principle, the cancer risk assessment of PAHs in ambient air can be performed in two ways. One approach was to add the risks from selected individual PAHs as determined from animal experiments. The other approach was to use BaP as an indicator of the mixture of carcinogenic PAHs in air and apply that to the dose–response relationship observed in epidemiological studies. Both of these approaches have considerable weaknesses. WHO has chosen epidemiological data on coke-oven workers for risk assessment in the revised Air Quality Guidelines for Europe (WHO 2000). The same approach has been chosen by the Working Group on Polycyclic Aromatic Hydrocarbons (European Commission 2001).

The approach adopted by The USEPA (1980, 1984) as the basis for risk assessment was to separate the PAHs into two subclasses, consisting of the carcinogenic and non-carcinogenic PAHs, to apply a cancer slope factor derived from assays on BaP to the subclass of carcinogenic PAHs. Nisbet & LaGoy (1992) reviewed relative potency estimates and provided revised ones. The complex version of the application of PEFs was presented by Collins et al. (1998). The development of PEFs and application of the PEF scheme described in this paper was presented below Owing to the absence of chronic inhalation studies on PAH and the variety and uneven quality of data available on the carcinogenicity of PAH, an order of preference for the use of available data in assessing relative potency was developed (Table 2.4).

Table-2.4: Relative potency of individual PAHs compared with B[a]P (TEF values), according to different authors:

Compound	Krewski et al. 1989	Nisbet & LaGoy 1992	Malcolm & Dobson 1994	Kalberlah et al. 1995	USEPA 1993	McClure & Schoeny 1995	Muller et al. 1997	Larsen & Larsen 1998
Naphthalene		0.001	0.001					
Acenaphthene		0.001	0.001	0.001	0			
Acenaphthylene		0.001	0.001	0.01				
Anthanthrene	0.320						0.28	0.3
Anthracene		0.01	0.01	0.01				0.0005
Benz[*a*]anthracene	0.145	0.1	0.1	0.1	0.1	0.1	0.014	0.005
Benzo[*a*]pyrene	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Benzo[*b*]fluoranthene	0.141	0.1	0.1	0.1	0.1	0.1	0.11	0.1
Benzo[*e*]pyrene	0.004		0.01				0	0.002
Benzo[*g,h,i*]perylene	0.022	0.01	0.01	0.01			0.012	0.02
Benzo[*j*]fluoranthene				0.1		0.1	0.045	0.05
Benzo[*k*]fluoranthene	0.061	0.1	0.1	0.1	0.01	0.1	0.037	0.05
Chrysene	0.0044	0.01	0.01	0.01	0.001	0.1	0.026	0.03
Phenanthrene		0.001	0.001	0			0.00064	0.0005
Pyrene	0.81	0.001	0.001	0.001			0	0.001
Indeno[1,2,3-*c,d*]pyrene	0.232	0.1	0.1	0.1	0.1	0.1	0.067	0.1
Dibenzo[*a,e*]pyrene						1.0		0.2
Dibenz[*a,c*]anthracene			0.1					
Dibenz[*a,h*]anthracene	1.11	5.0	1.0	1.0	1.0	1.0	0.89	1.1
Dibenzo[*a,l*]pyrene						100	100	1.0
Dibenzo[*a,e*]fluoranthene							1.0	
Dibenzo[*a,h*]pyrene						1.0	1.2	1.0
Dibenzo[*a,i*]pyrene						0.1		0.1
Fluoranthene		0.001	0.001	0.01				0.05
Fluorene		0.001	0.001	0				

Chapter~3

Methodology

Chapter -3

Methodology

3.1 Study Area

Asphalt Industry: Asphalt processing units located in and around Bangalore were selected for the study. The coordinates are at 12°58′N ,77°34′E to 12.97°N, 77.56°E and it covers an area of 741 km^2. The During the year 2012 a preliminary walk-through survey in all the mixing plants and road where paving work was carried out. The samples were collected during the year 2012-2015. Figure-3.1 shown the location of the asphalt plants.

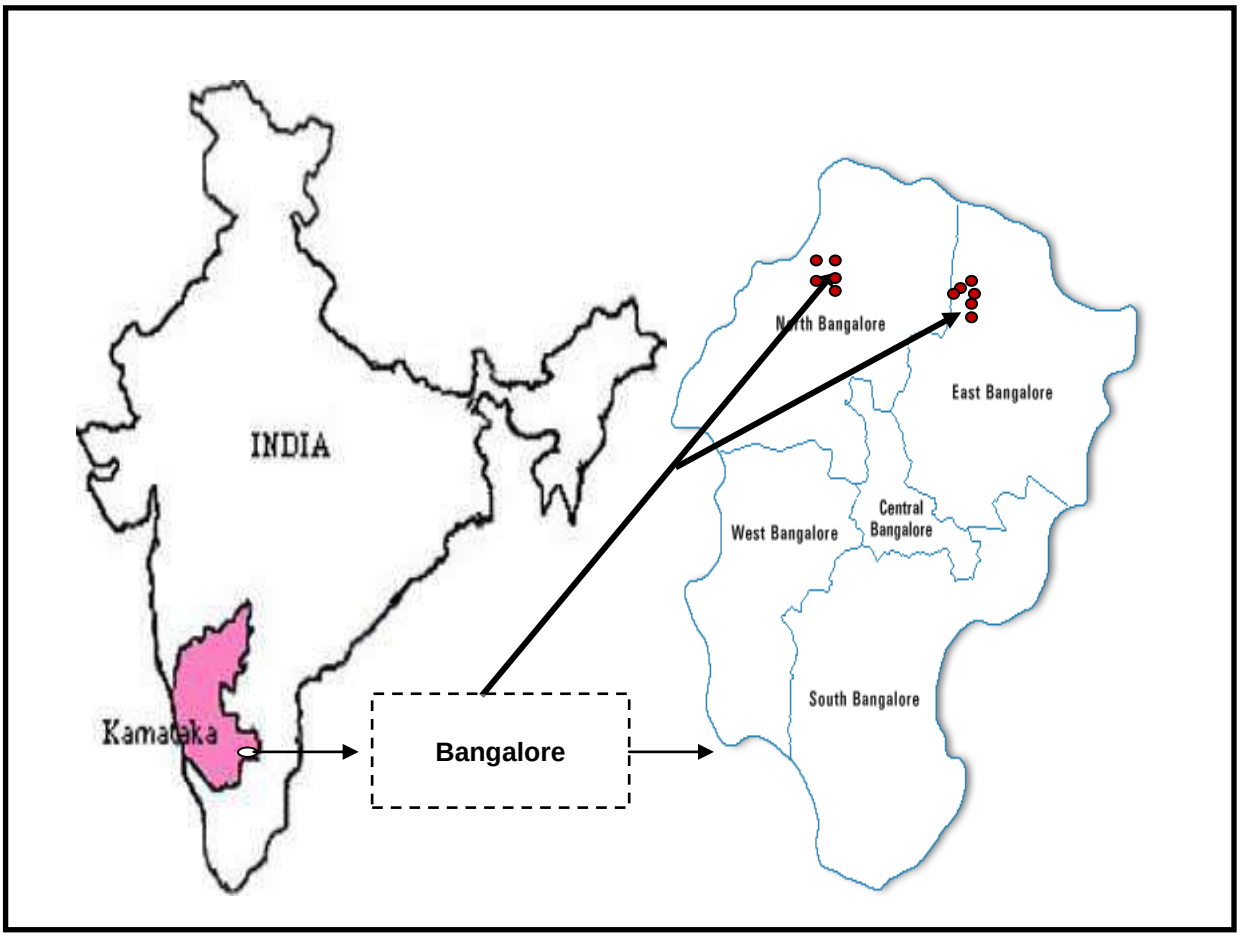


Figure-3.1: Location of asphalt process plants in Bangalore city.

Foundry Industry: The study was conducted in the foundries located in Shimoga town (13° 27′N, 74°37′ E to 14° 39′ N, 75°52′ E; an area of 8477 km^2) in Karnataka (figure-3.2) and Coimbatore city (10°37'N, 76°39'E to 11°31'N, 77°5' covered 7433.72 km^2) in Tamil Nadu (figure-3.3) where large numbers of foundries were situated.

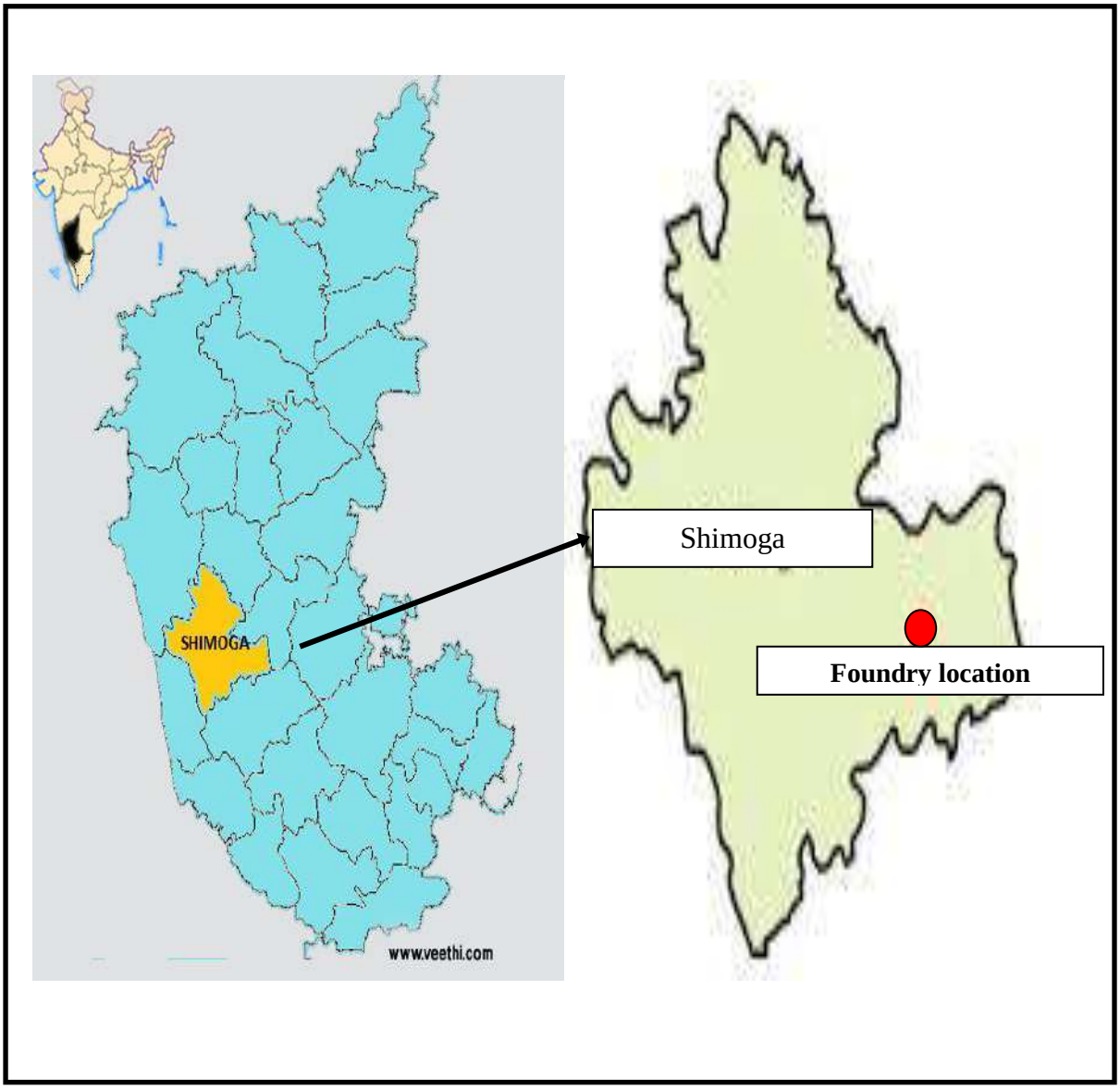


Figure-3.2: Location of the foundry in Shimoga, Karnataka

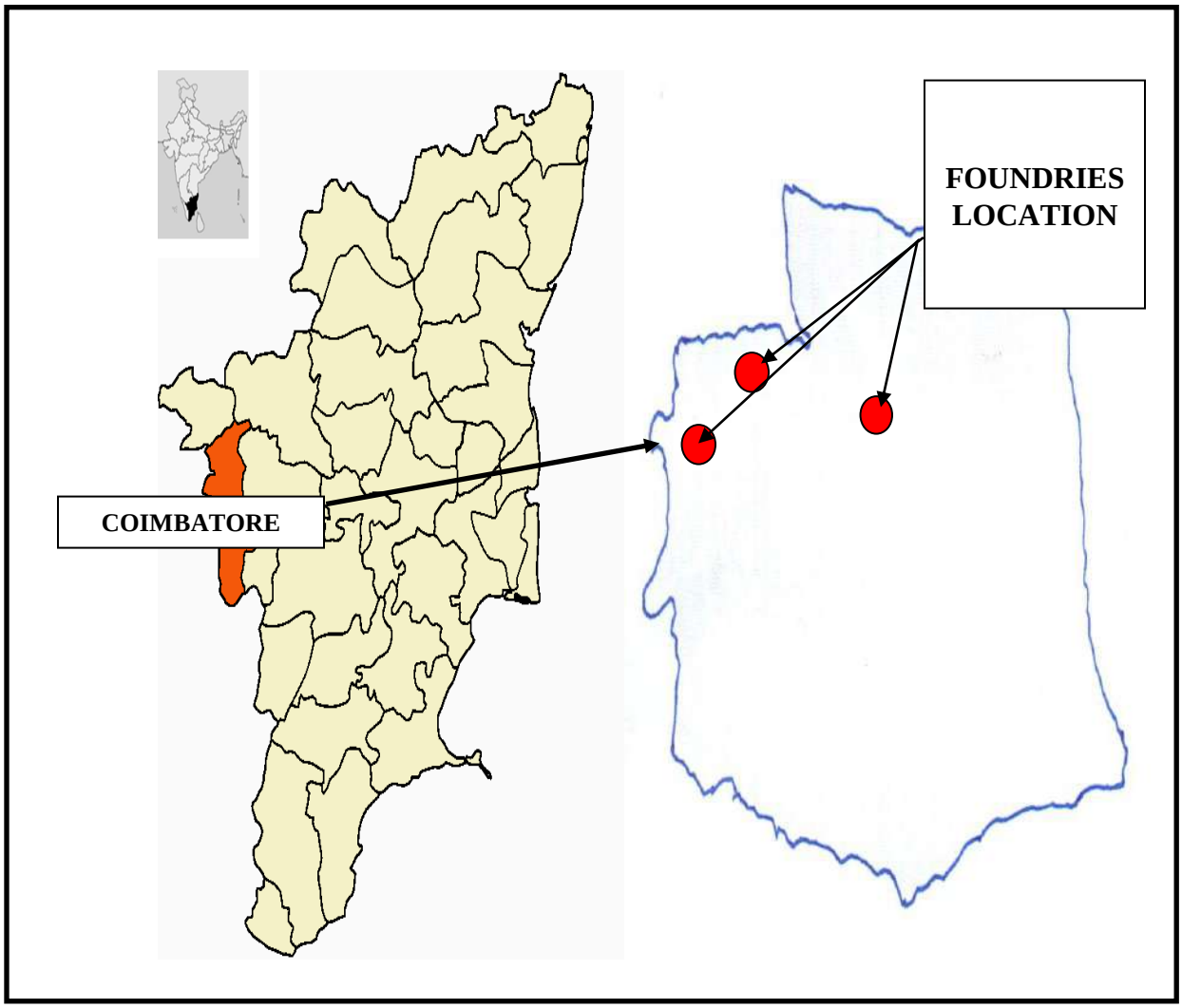


Figure-3.3: Location of the foundry in Coimbatore city, Tamil Nadu.

The environmental monitoring was carried out in various sections of the asphalt and foundry industry. In asphalt industry, the monitoring was carried out in plant where hot bitumen was mixed with stone and in the road paving area. The units considered in the foundry were molding, melting/furnace, shake-out area, blasting, heat treatment and finishing sections (figure-3.4). The biological samples were collected from the workers, those worked in these sections.

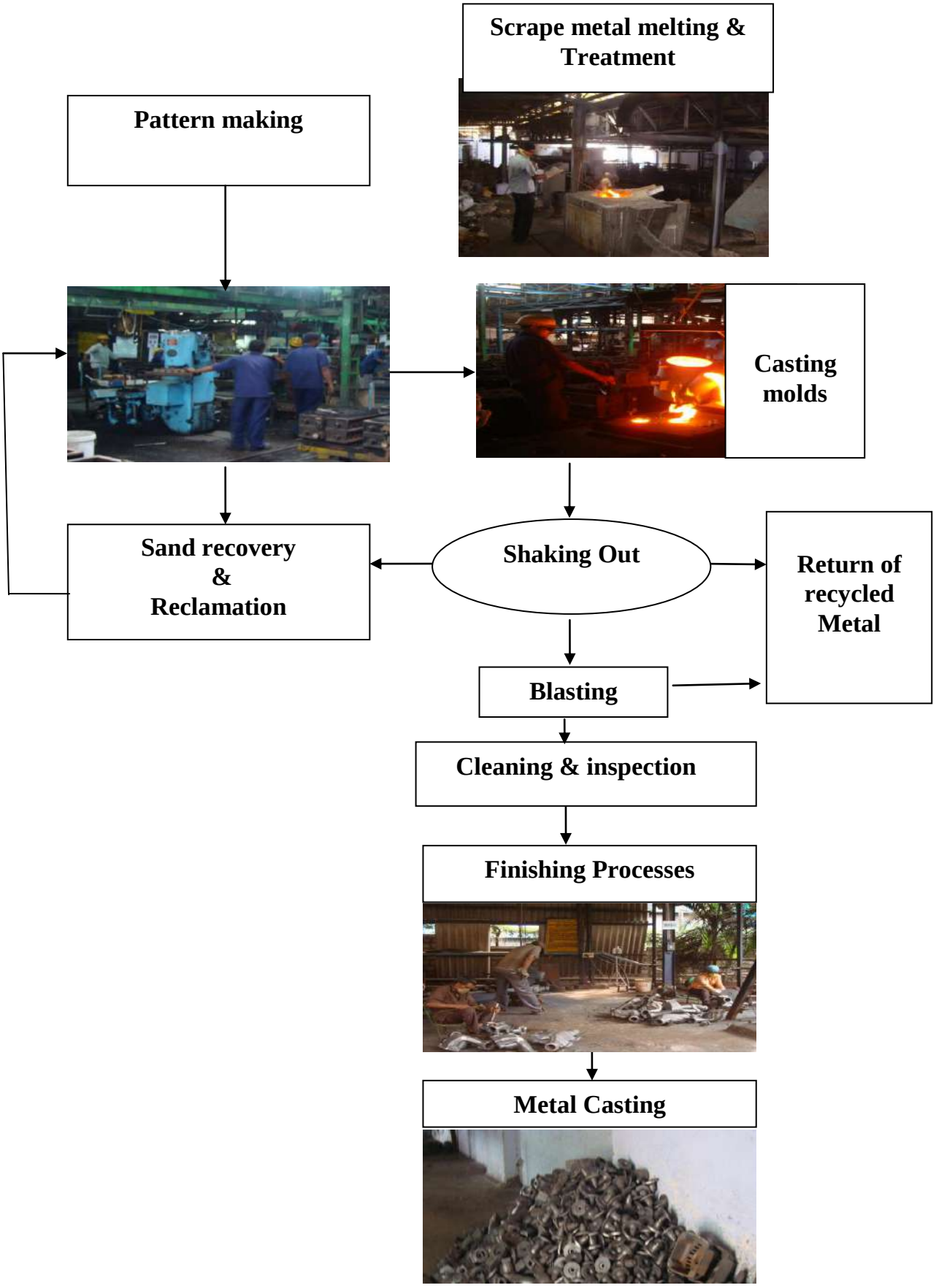


Figure-3.4: Flow Chart of Foundry Process in Study Area

Figure-3.5: Asphalt plant process in the study area

Figure-3.6: Asphalt paving process in the study area

3.2 Study Population

Asphalt Workers: A total of 60 workers belonging to the two job categories such as asphalt mixing plant worker and road paving worker were considered in this study. The hot mixing plant workers undertake the job for transferring hot bitumen into the silo, which comes through the tankers and mixing the stone, gravel into rotating drum/mixer and conveyor belt operators for transferring to asphalting locations. The paving workers apply hot mix asphalt while resurfacing the roads. A consent was obtained from each study participant prior to sampling after explaining the objective of the study to both the occupier of the plant and worker.

Foundry Workers: A total of 93 workers for respirable and 60 for PAHS were considered for the study. Data was collected using a questionnaire on age, work experience, nature of work and lifestyles (including smoking, alcohol, tobacco chewing). At the end of the shift of weekends, urine samples were collected from all the participants. Before the collection of urine, the participants were asked to wash their hands to avoid contamination. All subjects were provided informed consent. The samples were stored at -25°C in the deep freezer till the analysis.

The ethical clearance was obtained from the institutional ethics committee at Regional Occupational Health Centre (Southern), Indian Council of Medical Research, Bangalore (Ref. No-ROHCS/142/ 626A dated 26.09.2012, Appendix-V).

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3.3 Respirable Dust Sample

Sampling of respirable dust at different process in the asphalt and foundries were monitored using SKC personal sampling pumps (Model 224-PCXR8, M/s SKC, Pittsburgh, USA) following NIOSH 0600 method. The pumps were pre-charged and calibrated at the site. The personal sampling pump was equipped with a 37mm aluminum cyclone filter head, loaded with glass filter papers (0.8µm pore size) and was put on the workers during the shift (figure-3.7). The respirable dust was sampled

for 8 hours. At the end of the shift, pumps were removed and filter papers were analysed by gravimetric method.

Figure-3.7: Monitoring of PAHs at workplace using personal sampler

The concentration of respirable dust (mg/m^3) was assessed using the flowing formula

$$C = \frac{(W_2-W_1)\ x10^3}{T\ x\ V}$$

C : Dust concentration in the air in mg/m^3

W_1 : Filter's weight before sampling (mg)

W_2 : Filter's weight after sampling (mg)

T : Time of sampling (min.)

V : Amount of sampling pump's flow (liters/minute (with correction of sampling air capacity over capacity in standard situation)

3.4 Prediction analysis of respirable dust using Bayesian model

In this study an AIHA exposure categorization (Paul *et al*., 2006) scheme and a Bayesian decision analysis (BDA) tool together were used to categorize exposures of workers in the foundry process. When collecting exposure data was to classify the exposure profile, or distribution of exposures into one of five exposure categories: 0, 1, 2, 3, or 4, to trivial (or very low) exposure, highly controlled, well controlled, controlled, and poorly controlled exposures respectively. Using the AIHA exposure categorization scheme, an acceptable exposure group was one where the true group 95th percentile exposure (for a reasonably homogeneous group) was less than the single shift exposure limit. Consequently, an unacceptable exposure group was one where the true 95th percentile exceeds the limit. IHDA-Student 2015 (IH Data Analyst-Student 2015, Exposure Assessment Solutions, Inc. www.OESH.com) was used for data analysis based on Bayesian statistics as a tool for decision making. The BDA tool uses in the AIHA exposure categories given in Table 3.4, and calculates the probability of the 95th percentile of the exposure distribution for each similarly exposed group (SEG) exceeding the exposure limit. The results were presented in the form of three decision charts (prior, likelihood and posterior). We

have assumed a uniform prior for all our calculations indicating that prior to making measurements, there was no evidence to assign higher probabilities to any of the five categories; the likelihood shows the probability of the 95th percentile being located in each of the five categories based solely on the measurements, and the posterior reflects the synthesis of the prior and the likelihood. Since we have assumed a uniform prior, the likelihood and the posterior probabilities were identical.

3.5 Monitoring of polyaromatic aromatic hydrocarbons and analysis

Personal PAHs Sampling: Personal exposure PAHs (both vapour & particulate phase) samples were collected from each worker according to NIOSH Method 5506 during different process of the asphalt and iron foundry. The personal sampling system consisted of a PTFE filter paper (Zefluor, Pall Gelman Sciences, Cat. No. P5PJ037) placed in the cassette holder to collect particulate PAHs, connected in series with an XAD-2 Sorbent tube (ORBO 43, Supelco, Cat. No. 2-0258) to collect the vapour phase PAHs near the breathing zone for 8-hours (figure-3.8). The flow rate was maintained at 2.0 LPM/min and the flow rates were checked before, during and after sample collection using a calibrated Rotameter. After sampling the filter paper and sorbent tubes were wrapped with aluminium foil to prevent sample degradation due to sunlight. Samples were transported in cold chain and stored in -20°C until analysis. The analysis was carried out at Industrial Hygiene & Toxicology Division, Regional Occupational Health Centre (Southern), ICMR, Bangalore Laboratory.

Processing of Samples & PAHs Analysis: The samples collected on filter paper and sorbent tube was extracted with acetonitrile and cyclohexene in an ultrasonic bath for 30 min at room temperature. The extracts were concentrated under rotary evaporator and changed to acetonitrile. All these concentrated samples were filtered through syringe filter (0.45µm Millipore PTFE filters) before analysis in HPLC. All the samples were analysed for a mixture of sixteen PAHs simultaneously. The sixteen PAHs compounds namely were NAP, ACE, ACY, ANT, PHE, FLU, FLT, BaA, CHR, PYR, BaP, BbF,BkF, DahA, BghiP and IND. The excitation and emission wavelength

was 340 nm and 425 nm respectively. Samples (20 µl) were injected using an HPLC system equipped with the fluorescence detector (FLD) with a C18 reversed phase column (250mm x 4.6 nm, 5 µm). A solvent gradient was acetonitrile and deionized water with linear gradient from 60% acetonitrile/ 40% deionized water to 100% acetonitrile at 1.5 ml/min over 50 minutes. The HPLC system was calibrated using an external standard mixture. A standard mixture containing 16 PAHs mixture provided by Sigma Aldrich was used for calibration and quantification. The limit of detection (LOD) of PAHs varied from 0.01-0.20 µg/l, the relative standard deviation (RSD) of PAHs was less than 12% and recovery of the sample from 75-110%.

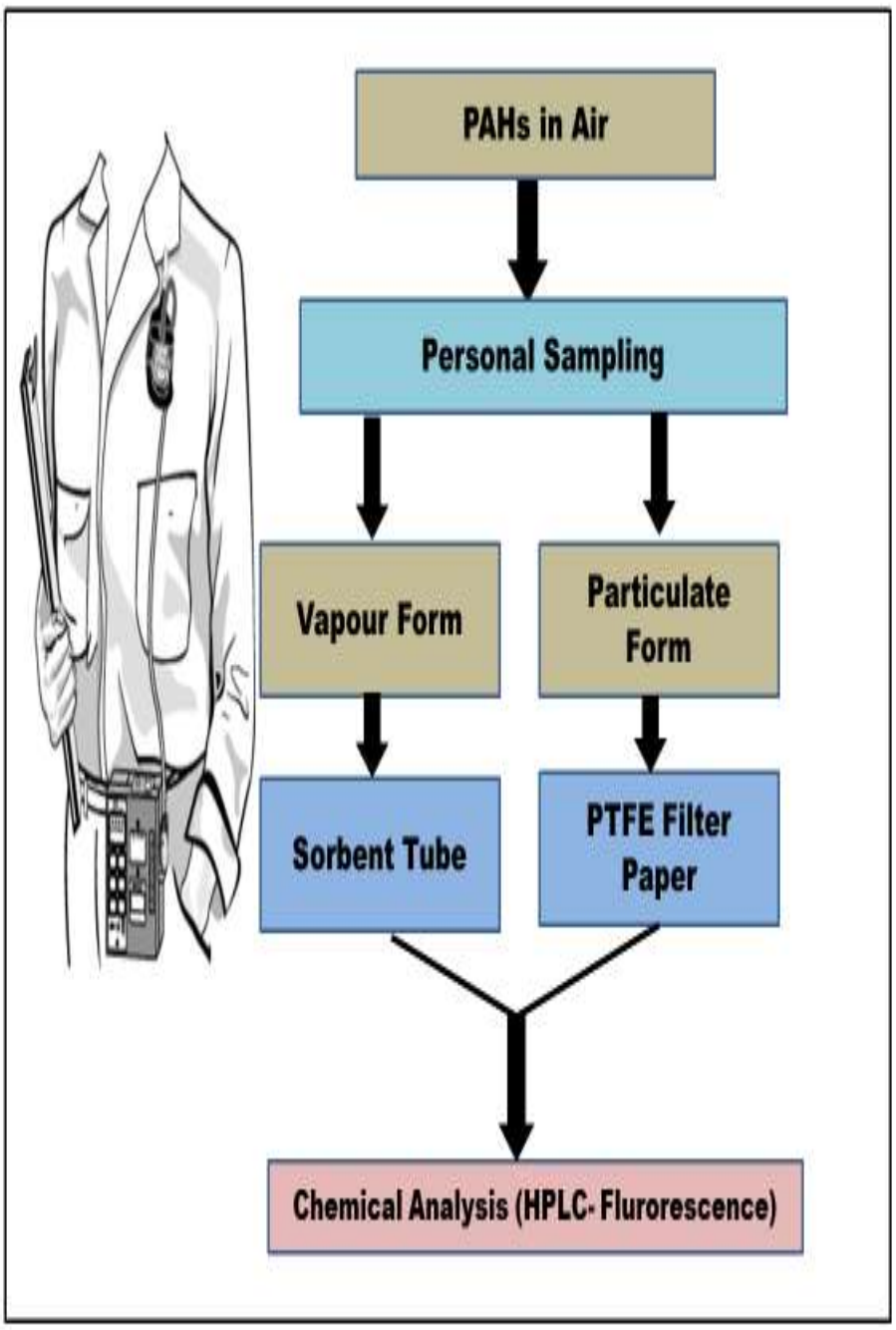

Figure 3.8: Flow diagram of Personal monitoring and analysis of PAHs

Figure-3.9: Personal Sampler with PTFE filter paper and sorbent tube for PAHs Sampling

Figure-3.10: (i) sorbent tube for (vapour phase PAHs) and (ii) PTFE filter paper (particle phase PAHs) samplings

Figure-3.11: Ultrasonic System for PAHs Extraction

Figure -3.12: Rotator evaporator for PAHs re-concentrator

Figure -3.13: Samples for PAHs analysis

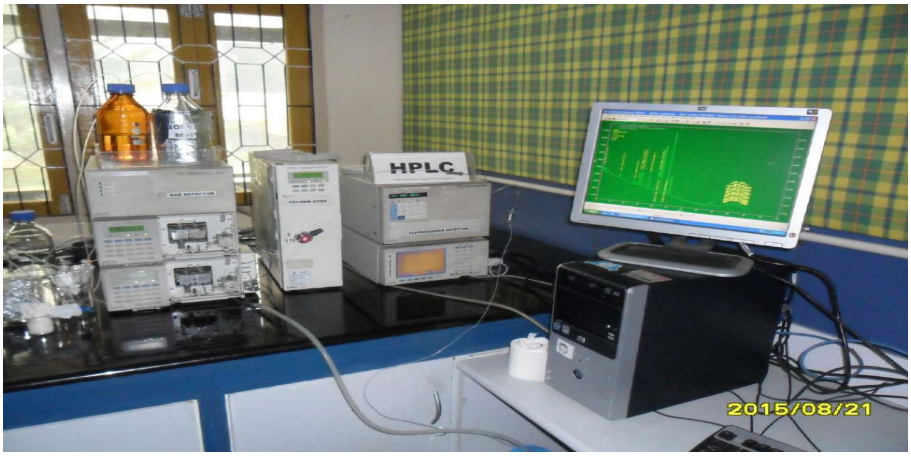

Figure -3.14: High performance liquid chromatography (HPLC-Shimadzu System Model LC-10AVP) for PAHs sample analysis

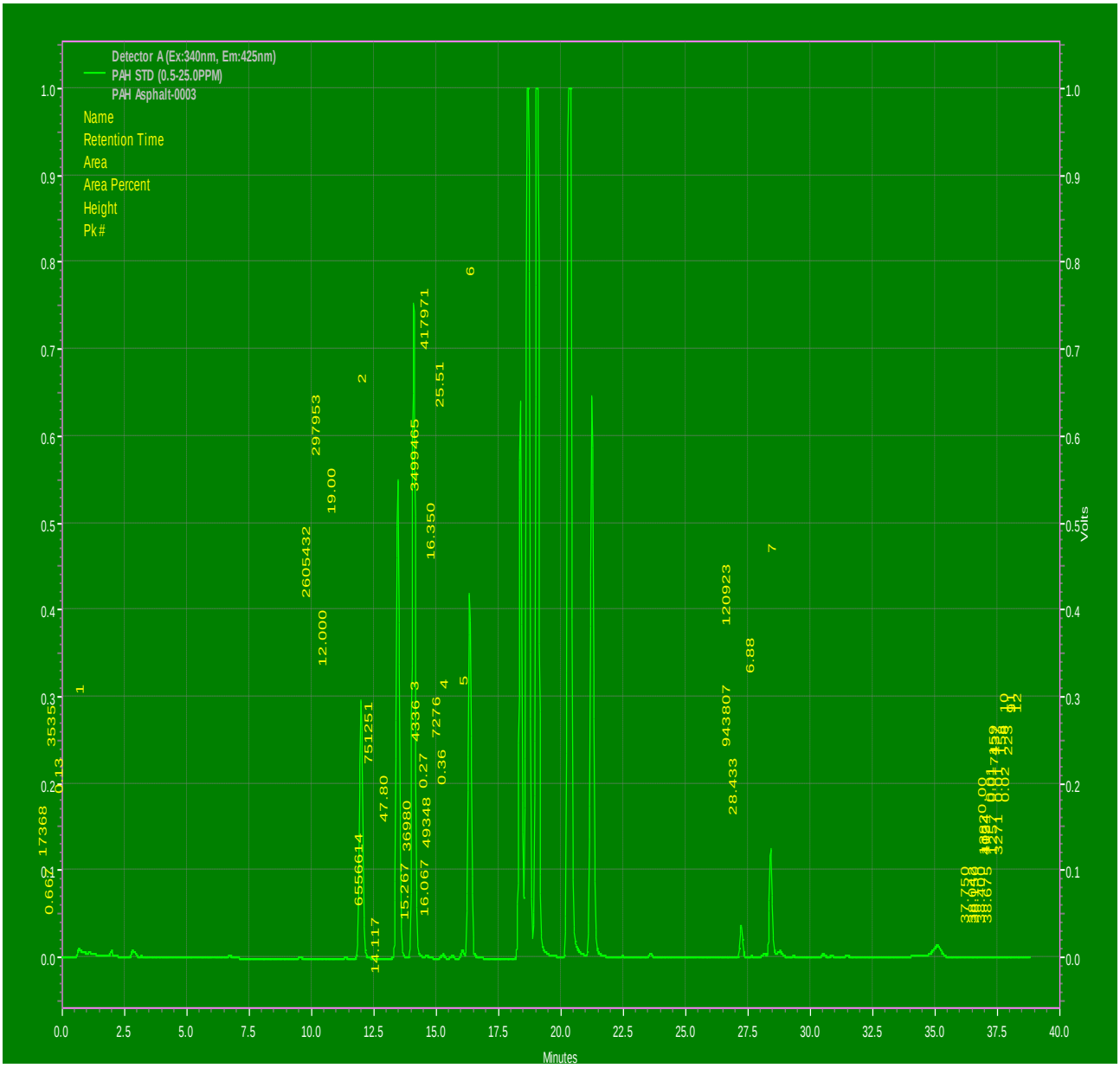

Figure-3.15: HPLC Chromatogram of PAH Standard

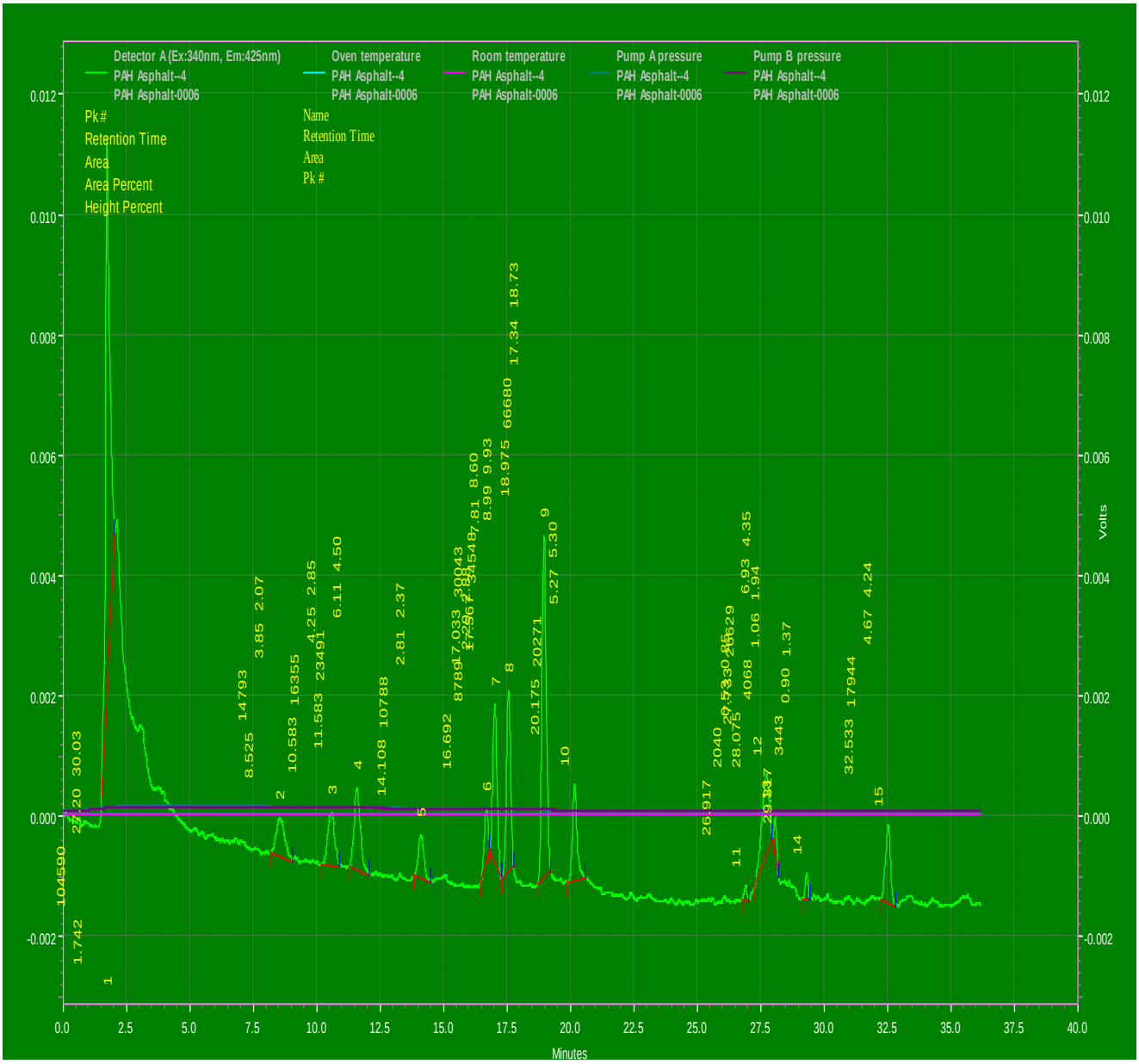

Figure-3.16: HPLC Chromatogram of PAH Sample.

Table-3.1: PAHs retention with concentration and peak area

PAH Compounds	Standard Conc. (ppm)	Retention Time	Peak Area
NAP	25.0	12.00	9450778
ACE	25.0	13.48	15436188
ACY	50.0	14.12	19824697
ANT	5.0	15.26	68275
PHE	2.0	16.06	34405
FLU	1.0	16.36	12686391
FLT	0.25	18.40	571510
BaA	0.50	18.69	1897452
CHR	0.25	19.06	2997931
PYR	0.25	20.33	6136615
BaP	1.0	21.25	20932930
BbF	1.0	23.59	157955
BkF	2.5	27.23	944352
DahA	10.0	28.43	3358289
BghiP	4.0	28.77	122428
IND	2.5	35.10	853662

3.6 Analysis of PAHs Biomarker by HPLC methods

Urine sample Collection: *S*ame numbers of 60 samples in the exposed group belongs to in each category of industry and a total 26 samples comprising from control group were included in the study. The control population were selected in the same factory in non-processing areas which included administration, canteen and store staffs. At the end of the shift during weekends, urine samples were collected from all the participants. Before the collection of urine, the participants were asked to wash their hands to avoid contamination. The subjects agreed for the study were included in the sample collection.

Sample processing and analysis of 1-OHP & OHPHE in Urine: The determination of 1-OHP and OHPHE were carried out following previously developed high performance liquid chromatography (HPLC) method (Jongneelen *et al,*1987; Boos *et al*., 1992; Gtindel *et al.,*1996, Carmella *et al., 2004*). Aliquots (6 ml) of urine were buffered with 12 ml 0.2(N) sodium acetate buffer (pH 5.0) and hydrolyzed with 30µl of arylsulfatase for 16 h keeping inside the incubator at 37°C with the constant shaking condition at 210 rpm. Once conditioning was achieved after overnight incubation, the metabolites were enriched on a special RP-C 18 cartridge (500 mg/3CC cartridge procured from M/s AnalChem Pvt Ltd, Allahabad). The column was conditioned with 5 ml of methanol and 10 ml of water and then the hydrolyzed urine samples were passed through the C18 cartridges without letting the cartridges to get dried. The extracts were dried with nitrogen purging until dryness. The residue was re-dissolved in 2ml of methanol with ultrasonic baths. The aliquot was filtered through 0.45 µm syringe filter prior to HPLC analysis. The 20µl of samples was injected into the HPLC to quantify 1-OHP & total OHPHE metabolites. All the isomers of OHPHE (2, 3, 4 and 9-OHPHE) were measured. The limit of detection (LOD) of 1-OHP and total OHPHE was 0.05 and 0.10 µg/l, respectively and the relative standard deviation (RSD) of 1-OHP was less than 10%, Mobile Phase: Methanol & Water (1:1) with flow rate 1ml/min, Column: C18 reversed phase column (250mm x 4,6 mm, 5 µm), Detector: Fluorescence Detector, Wavelength for OHPHE

metabolites: excitation: 244 nm, emission: 370 nm; Wavelength for 1-OHP: excitation: 241 nm, emission: 386 nm), Temperature of the columns 40°C.

Urinary 1-OHP and OHPHE concentration were expressed as µmol/ mol of creatinine. The urinary 1-OHP levels of each individual were corrected according to urinary creatinine values, which were measured using an automated method based on the kinetic Jaffe's reaction.

Figure-3.17: Urine sample of the study population

Figure- 3.18: -20°C refrigerator for urine sample storage until analysis

Figure -3.19: Semi Auto Analyzer for urinary creatinine estimation

Figure -3.20: Shaking orbital incubator

Figure -3.21: Sample extraction using vacuum manifold

Figure -3.22: Nitrogen Evaporator for re-concentrator urinary PAHs metabolites

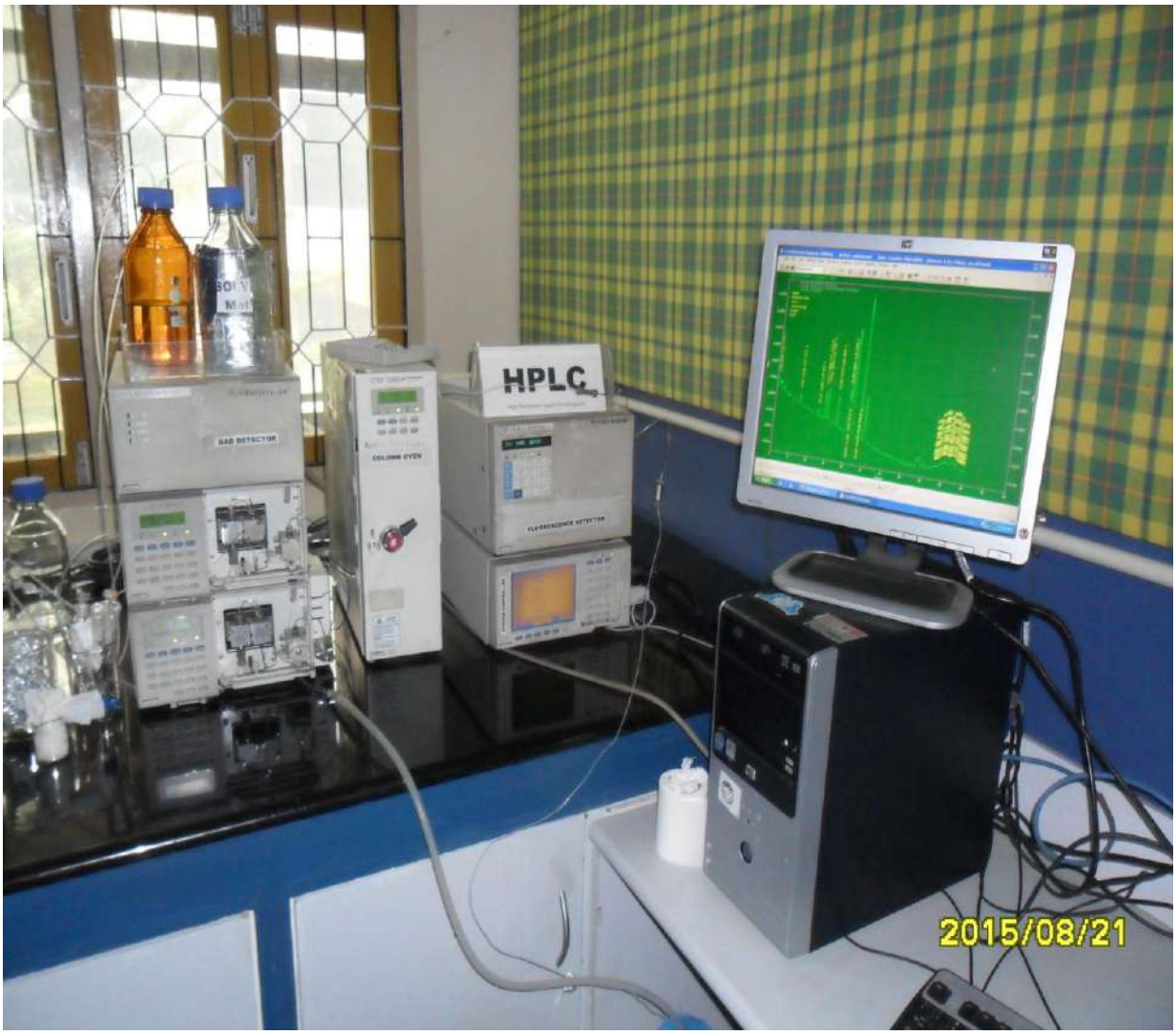

Figure -3.23: High performance liquid chromatography (HPLC-Shimadzu System Model LC-10AVP) for analysis of PAHs metabolites

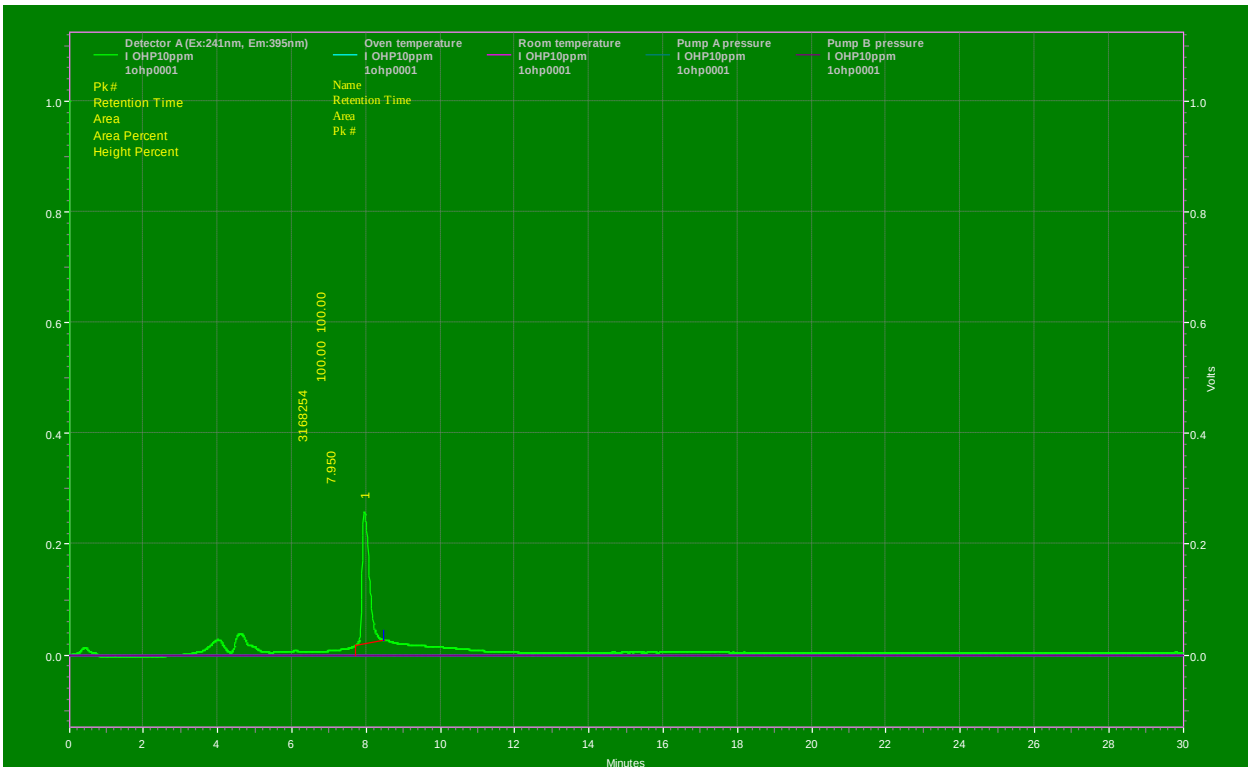

Figure-3.24: HPLC Chromatogram of 1-Hydroxypyrene of Standard.

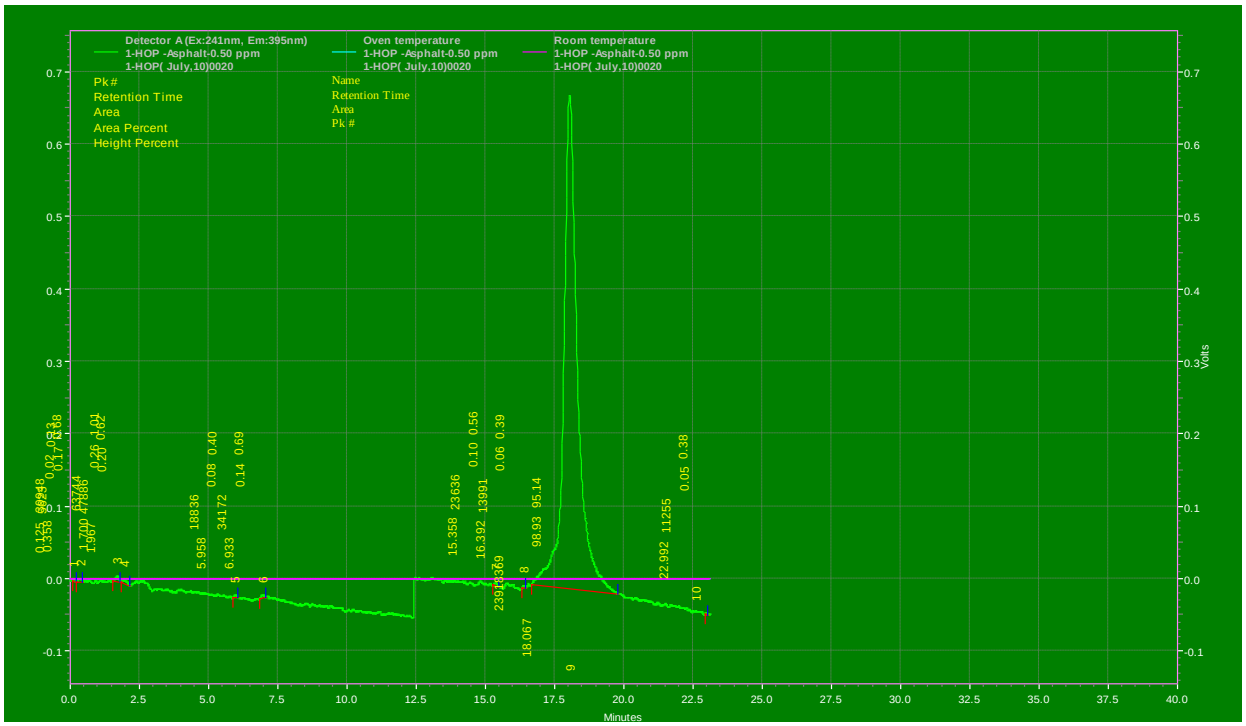

Figure-3.25: HPLC Chromatogram of 1-Hydroxypyrene of Sample.

Figure-3.26: HPLC Chromatogram of 9-Hydroxyphenentherene of Standard

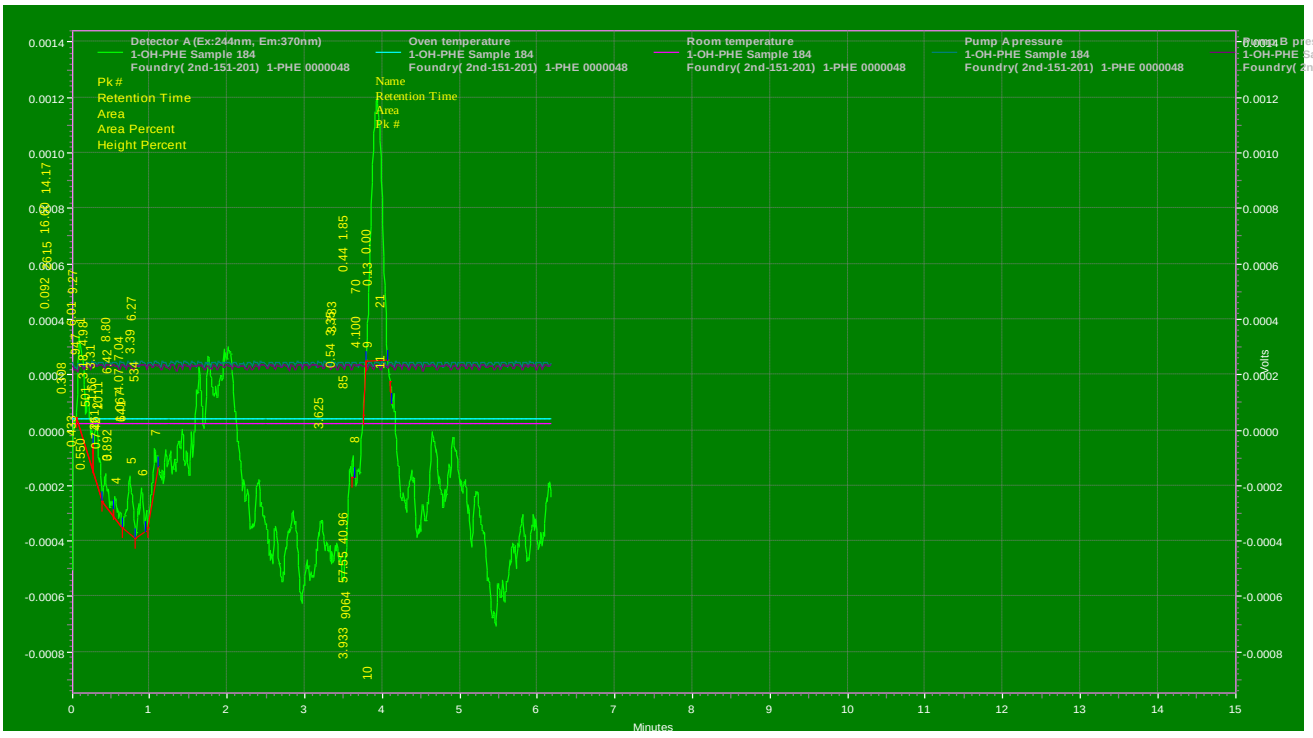

Figure-3.27: HPLC Chromatogram of 9-Hydroxyphenentherene of Sample.

Table-3.2: Calibration Standard of 1-Hydroxypyrene

Conc. (ppb)	Area
0	1215
5	26633
10	46579
20	89576
50	211596
Slope	0.0002
Intercept	-1.013
Correlation	0.9997

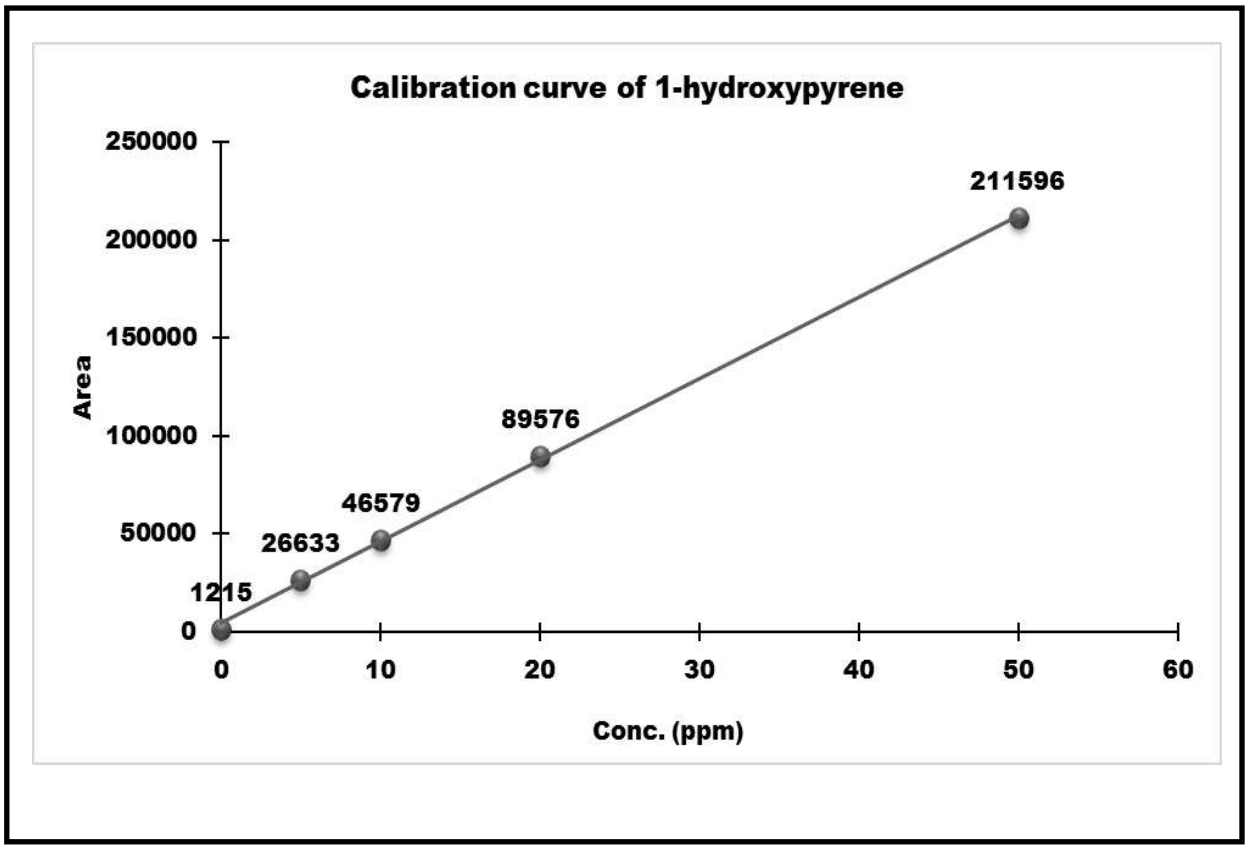

Figure-3.28: Calibration curve of 1-Hydroxypyrene

Table- 3.3: Calibration Standard of Hydroxyphenantherene

Conc(ppb)	Area
0	1546
5	5452
10	12078
25	20454
50	38728
Slope	0.001
Intercept	-3.324
Correlation	0.996

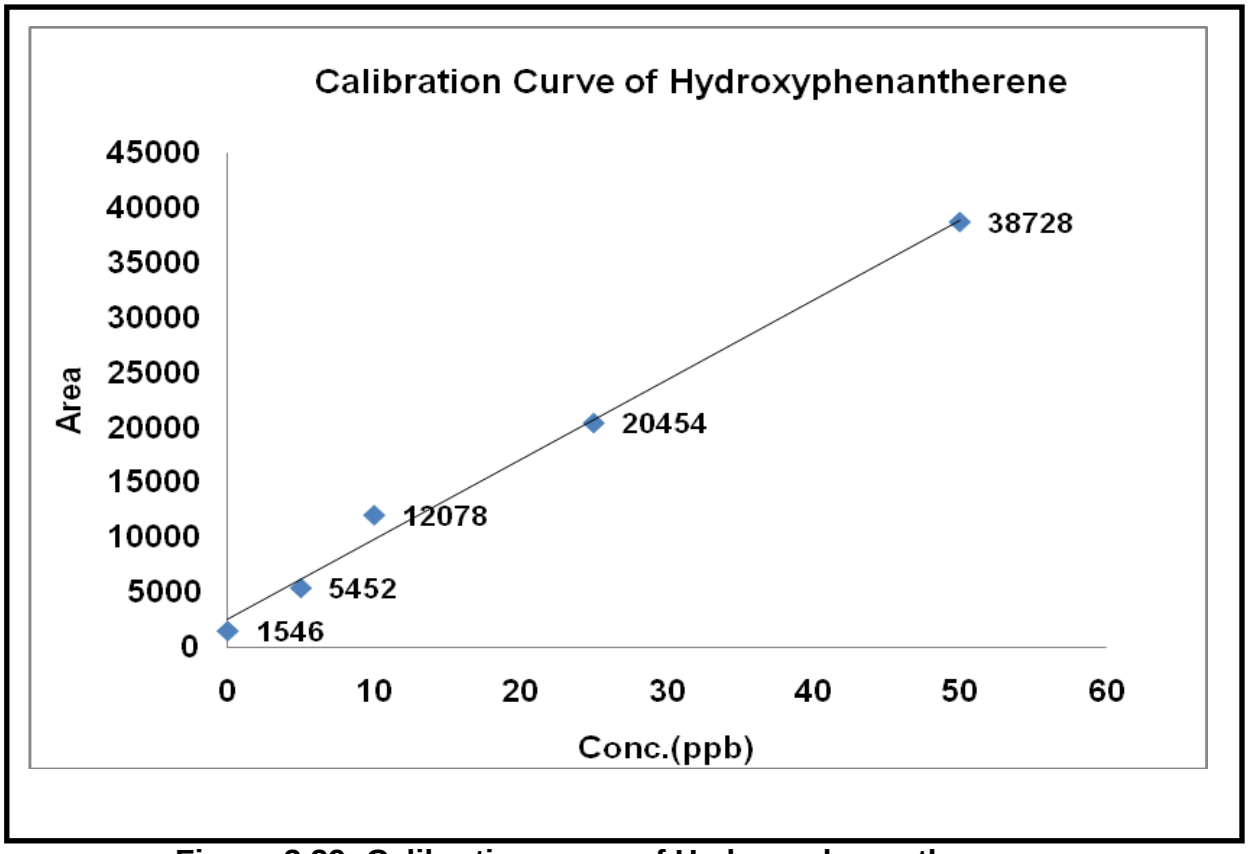

Figure-3.29: Calibration curve of Hydroxyphenantherene

Table -3.4: AIHA exposure categorization scheme

Exposure category[a]	Rule of thumb description[b]	Qualitative description	Recommended statistical interpretation[c]
0	Exposures are trivial to nonexistent—employees have little to no exposure, with little to no inhalation contact.	Exposures, if they occur, infrequently exceed 1% of the OEL	$X0.95 \leq 0.01 \times$ OEL
1	Exposures are highly controlled—employees have minimal exposure, with little to no inhalation contact.	Exposures infrequently exceed 10% of the OEL	$0.01 \times$ OEL $< X0.95 \leq 0.1 \times$ OEL
2	Exposures are well controlled—employees have frequent contact at low concentrations and rare contact at high concentrations.	Exposures infrequently exceed 50% of the OEL and rarely exceed the OEL	$0.1 \times$ OEL $< X0.95 \leq 0.5 \times$ OEL
3	Exposures are controlled—employees have frequent contact at low concentrations and infrequent contact at high concentrations.	Exposures infrequently exceed the OEL	$0.5 \times$ OEL $< X0.95 \leq$ OEL
4	Exposures are poorly controlled—employees often have contact at high or very high concentrations	Exposures frequently exceed the OEL	$X0.95 >$ OEL

[a]An exposure category can be assigned to a SEG whenever the true 95th percentile exposure ($X_{0.95}$) falls within the specified range.
[b]The "Rule-of-thumb" descriptions were based on similar descriptions published by the AIHA.(2)
[C]$X_{0.95}$ = the true group 95th percentile exposure.

Source: Hewett, 2006

3.7 Toxic Equivalency Factors and Risk Assessment

Oxford English Dictionary (Oxford University Press, 1971) defines risk as a "hazard, danger; exposure to mischance or peril". Therefore, to put oneself "at risk" means to participate voluntarily or involuntarily in an activity or event that could lead to injury, damage, or loss. Involuntary risks were negative impacts associated with an occurrence that happens to us without our prior consent or knowledge. Acts of

nature such as being struck by lightning, fires, floods, tornados, etc., and exposures to environmental contaminants were examples of involuntary risks.

To calculate the risk of PAHs compounds exposure in the indoor workplace through inhalation, Risk Assessment Information System toolkit (RAIS,2013, The University of Tennessee) developed by The California Environmental Protection Agency was used which was available as free version on the net. The tool was considered for calculating the risk value, since the workers have performed their duties in indoor environment and inhaled the PAHs from surrounding work atmosphere.

Carcinogenic Air Equation:

Inhalation Ambient Carcinogenic (CDI) Equation (RAIS, 2013):

$$CDI_{iw}\text{-air-}(\mu g/m3) = \frac{Cair(\mu g/m^3) \times (250days/year) \times EDiw(25year) \times ETiw(8hours/day) \times (1day/24hours)}{ATiw\left[\frac{365days}{year}\right] X\ LT(67years)]}$$

Where C_{air}-Concentration of PAH compound; EF_{iw} (exposure frequency - indoor worker) day/year-250; ED_{iw} (exposure duration or total working life) - year-25; ET_{iw} (exposure time - indoor worker) hour-8; AT_{iw} (averaging time - indoor worker)-365; LT (lifetime) year-67 (life expectancy at birth for male in India, WHO,2015).

***The mathematical expression to determine the inhalation ambient air risk is provided below*:**

The Equation Determine the Inhalation Ambient Air Risk (IAAR):

IAAR Value= $CDI_{iw\text{-}air\text{-}ca}$ ($\mu g/m^3$) x Inhalation Unit Risk* $(\mu g/m^3)^{-1}$

**Inhalation Unit Risk (IUR) proposed by California Environmental Protection Agency and establishment of IUR for PAHs listed in* Table-3.5.

Table-3.5: Toxic equivalency Factors (TEFs) for individual PAHs as per Cal/EPA:

Compounds	CAs-Nr	TEFs
NAP	91-20-3	3.4×10^{-05}
ACPy	208-96-8	0
ACE	83-32-9	0
FLU	86-73-7	0
PHE	5801-8	0
ANT	1209-12-7	0
FLA	206-44-0	0
PYR	129-00-0	0
BaA	56-55-3	1.1×10^{-04}
CHR	219-01-9	1.1×10^{-05}
BbF	205-99-2	1.1×10^{-04}
BkF	207-08-9	1.1×10^{-04}
BaP	50-32-8	1.1×10^{-03}
DahA	53-70-3	1.2×10^{-03}
BghiP	191-24-2	0
IND	193-39-5	0.00011

Chapter~4

Workplace monitoring & risk assessment

Chapter-4

Workplace monitoring and risk assessment

4.1 Workplace respirable dust monitoring and risk factor assessment in foundry process

Workplace respirable dust monitoring carried out in the shop floors of the foundry industry throughout the full work shift and emission of dust was collected in the filter paper. The filter paper was connected with respirable dust sampler. The observed respirable dust among the workers in different sections of two study areas (Study area- I (Shimoga) and study area -II (Coimbatore)) is shown in Table-4.1. The dust concentration was predominantly higher in finishing section of both the study area than other sections. By comparing the both the study areas it was found that the dust exposure in molding, melting and blasting sections in the study area II were in the elevated level than in the study area I. Only dust concentration in the finishing section of the study area I was higher than study area II, although the work pattern of study area I & II was similar. The mean concentration of the dust in the finishing section of the study area I has exceeded the permissible limits (3.0 mg/m^3) prescribed by ACGIH. It was also observed that the dust exposure in the heat treatment was lower than other sections, because the heat treatment process was adopted in a closed chamber where dust was not able to spread in the workplace environment. In the finishing section the work practices were manual and the unwanted material attached with the casting products were removed by hand operated equipment. So, the fine dust found in the workplace environment was higher than other section in the both study areas.

Table-4.2 presents the summary statistics for the respirable dust exposure data for each process unit of the foundries. Concentrations (mean±SD) of respirable dust in the molding process were 1.40±0.86 mg/m^3; in the melting process were 1.42±0.63 mg/m^3; in shakeouts 1.63±0.85 mg/m^3; in heat treatment 0.56±0.59 mg/m^3; in felting

2.17±0.61 mg/m^{3}and in finishing 3.30±3.47 mg/m^3 respectively. The levels were found to be relatively higher in the finishing section than the other process units and also the mean level was exceeded the ACGIH standard (TLV 3.0 mg/m^3) of respirable dust (figure-4.1). The highest dust concentration also observed in the finishing section and it was 10.9 mg/m^3. The geometric mean concentration of respirable dust in the finishing process was 2.23 mg/m^3.

Table 4.1: Dust exposure (mg/m^3) in the shop floors among the workers in two study areas of the foundries

Section	Study Area-I		Study Area-II	
	N	Mean ± SE	N	Mean ± SE
Molding	13	1.13±0.12	12	1.71±0.32
Melting	16	1.29±0.14	09	1.65±0.24
Shaking out	10	2.17±0.19	-	-
Blasting	15	1.60±0.22	01	2.09
Heat treatment	04	0.58±0.29	-	-
Finishing	08	3.8±1.56	05	2.52±0.42

- denotes, No Measurement.

From this it is clear that in finishing section the limit has exceeded but in the other process the dust concentration was comparably higher than atmospheric dust. and chronic exposure to dust may have a cumulative effect on workers and under risk.

Figure 4.2A-4.2C shows the results of BDA (the three decision charts) for respirable dust for the molding process considering the exposure limit of 3 mg/m^3 as per ACGIH. A uniform prior probability distribution was used to represent the situation where we have no prior knowledge or expectations regarding this particular process (Figure 4.2A).

Table-4.2: Dust exposure level of workers in foundry process

Section	N	Range	Median	Mean ± SD
		Conc.(mg/m$^{3)}$		
Molding	25	0.5-4.03	1.21	1.40±0.86
Melting	25	0.61-3.11	1.20	1.42±0.63
Shakeouts	16	0.18-3.10	1.60	1.63±0.85
Heat Treatment	4	0.1-1.35	0.43	0.56±0.59
Felting	10	0.81-3.01	2.36	2.17±0.61
Finishing	13	0.73-10.9	2.35	3.30±3.47

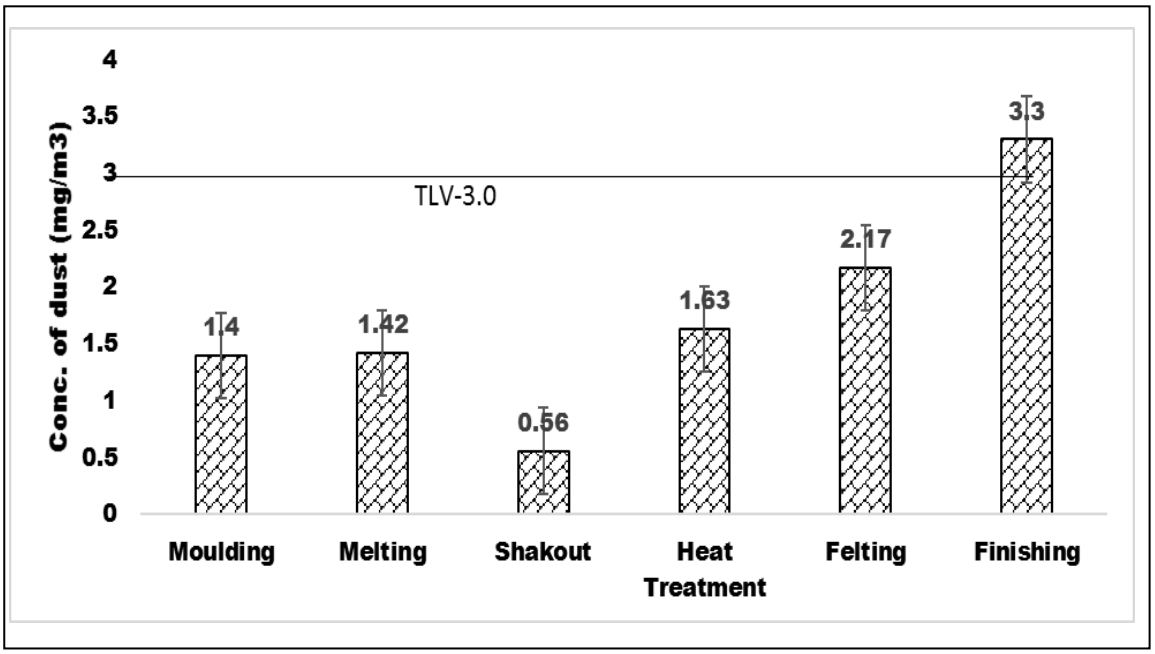

Figure-4.1: Mean respirable dust concentration in different process units in the foundries.

Figure 4.2B shows the probability of likelihood decision in the molding process using monitoring data. Fig.4.2C presents the posterior as final decision probability as the of Figure 4.2A and Figure 4.2B.

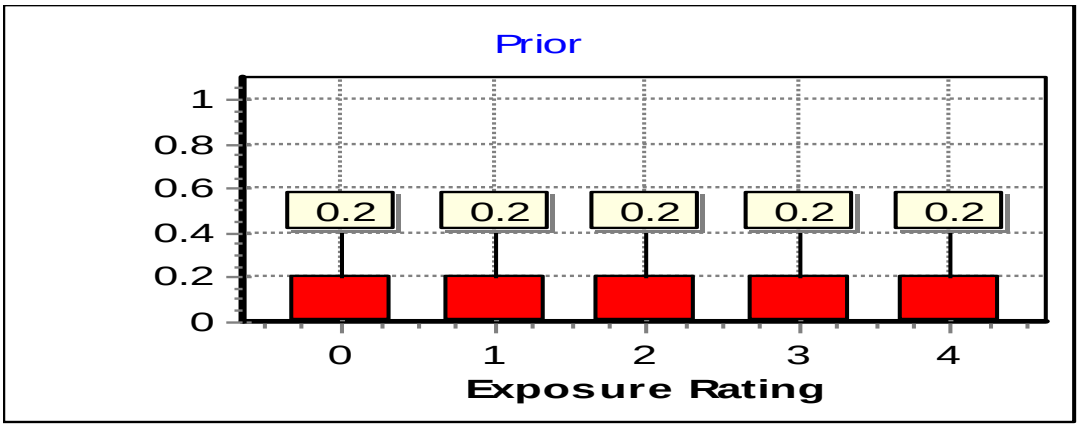

Figure. 4.2A

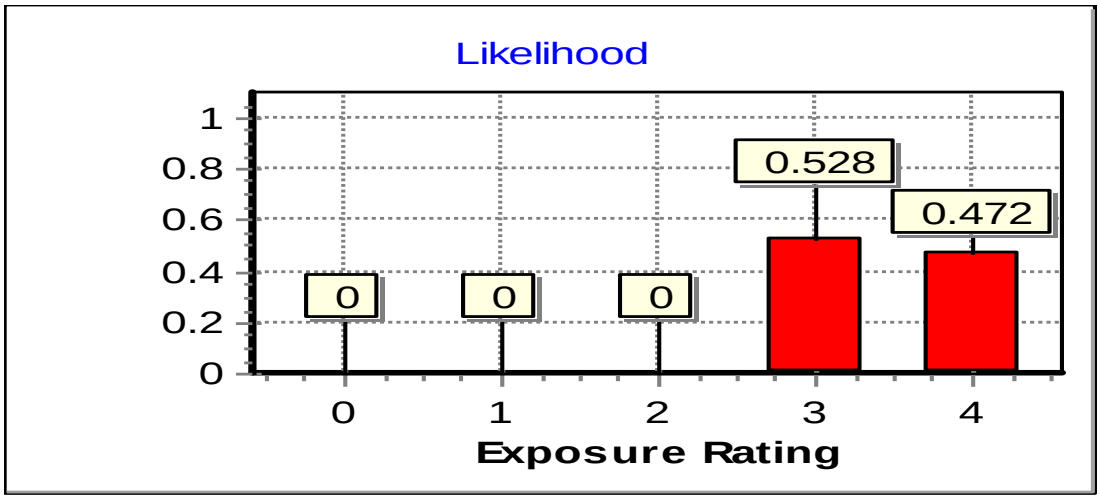

Figure. 4.2B

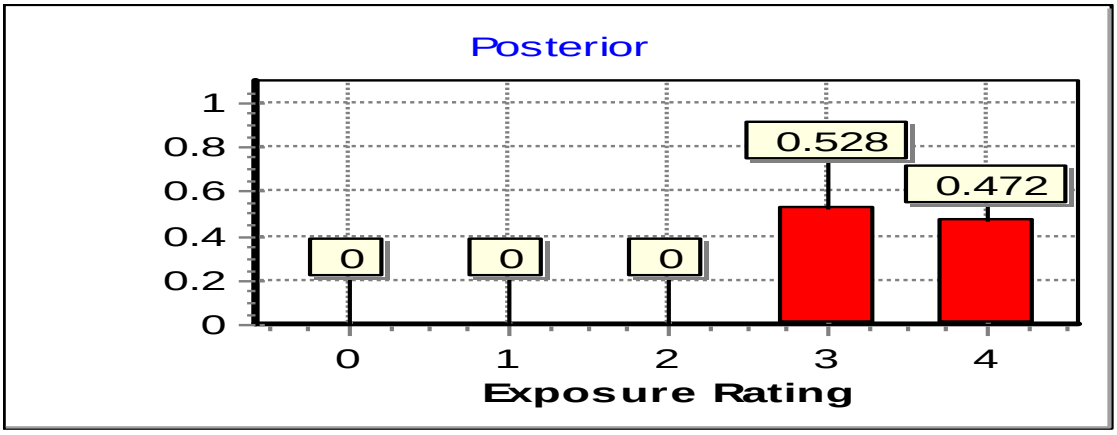

Figure. 4.2C

Figure-4.2(A-C): Bayesian modelling and assessment result of respirable dust concentration at molding unit process in foundry process.

Figures 4.3A- 4.3F were shows the results of the posterior decision probabilities using the Bayesian model based on the results (Table-4.1) of respirable dust identified in different process units of the foundry. Some of the processes were unambiguously Category 4 exposures, e.g., Shakeouts (96.7% probability), Felting (98.1% probability), and Finishing (100% probability), respectively. This was consistent with Table-10 which shows higher median exposures for these three exposure groups. From Figs.4.3(A) and 4.3(B) it was observed that the percentage of highest exposure rating in molding 52.8%, melting 79.4% and heat treatment 40.3% respectively and fall into the exposure category of 3 as per AIHA exposure categories (Table-3.4).

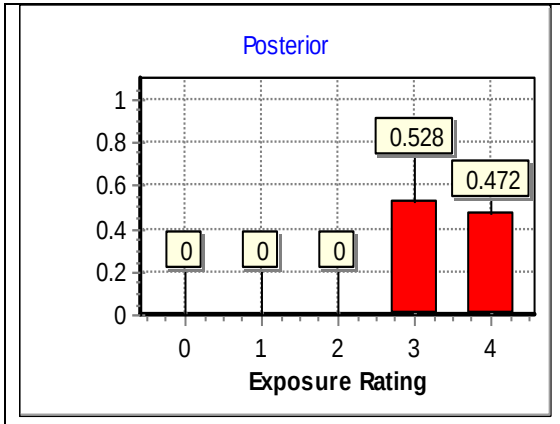

Fig-.4.3A, molding process

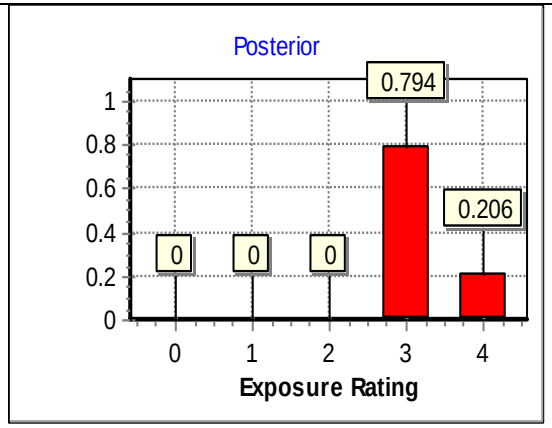

Fig.-4.3B, melting process

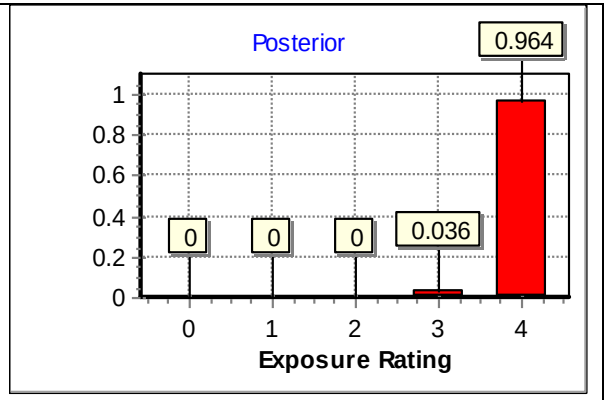

Fig.4.3C, Shakeouts process

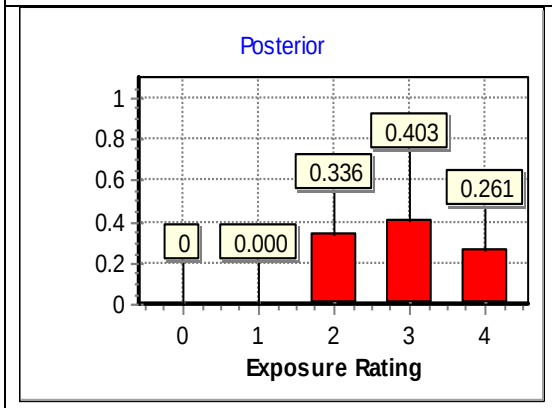

Fig.-4.3D, Heat Treatment process

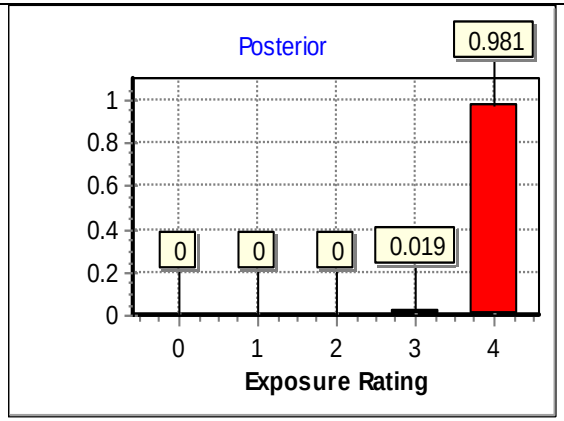

Fig.-4.3E, felting process

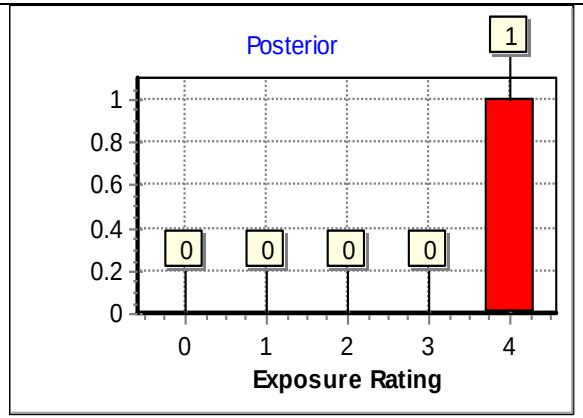

Fig.-4.3F, finishing process

Figure-4.3(A-F): Bayesian modelling and assessment result of respirable dust concentration at different process units in foundry process

Table-4.3 was contained a listing of typical actions and controls as prescribed by AIHA for workplace exposure. By assigning the exposure profile we were able to suggest control measurement in each process to reduce the exposure of respirable dust.

Table- 4.3: Typical actions or controls that result for each final rating

Final Rating	Action or Control
0	No Action
1	General or chemical specific hazard
2	Chemical specific hazard communication
3	Chemical specific hazard communication, Exposure surveillance, Medical surveillance, Work practice evaluation
4	Chemical specific hazard communication, Exposure surveillance, Medical surveillance, Work practice evaluation, Respiratory protection and Engineering controls
4+	+Immediate engineering controls or process shutdown, validate that respiratory protection is appropriate

Source: Hewett, 2006

In the foundry study, we have obtained from the result of prediction about each process unit by Bayesian model that the percentage of the excess rate of respirable dust in the Shakeouts, Felting and Finishing were belonged to the highest grade (grade 4/4+) and molding, melting and heat treatment process were under grade 3. These two outcome final ratings indicating that the workers were frequently inhaling respirable dust. In the molding, melting and heat treatment process unit's workers have frequent contact at low concentrations and infrequent contact at high concentrations. In the Shakeouts, Felting and Finishing unit's workers often have contact at high or very high concentrations. So, it was required to take the fast actions to control and safety measurement. Therefore, it is essential to have an immediate safety adaptation of personal protective equipment of proper respiratory

mask or engineering control like local ventilations or cross ventilation in order to prevent from being exposed to respirable dust to safeguard the workers' health. There should also need of chemical analysis of respirable dust and exposure surveillance like (i) protection of the health of the individual employee, (ii) detection at an early stage any adverse health effects due to exposure of chemical enrich of respirable dust, (iii) assisting in the evaluation of control measures, (iv) detection of hazards and assessment of risk or (v) the disease or health effects associated with exposure.

. 4.2 Workplace respirable dust monitoring and risk factor assessment in asphalts process

Table-4.4 presents the summary statistics for the respirable dust exposure data for each process unit of the asphalts. Concentrations (mean ±SD) of respirable dust in the plant were 0.28±0.25 mg/m^3 and in the paving, were 0.26±0.15 mg/m^3. The levels were found to be relatively higher in the plant than the paving area, but the mean level not exceeding the ACGIH standard (TLV 3.0 mg/m^3) of respirable dust. The highest dust concentration also observed in the plant and it was 1.31 mg/m^3.

Table-4.4: Dust exposure level of workers in asphalts process

Section	N	Range	Median	Mean ± SD
		Conc.(mg/m$^{3)}$		
Plant	38	0.11-1.31	0.18	0.28±0.25
Paving	22	0.05-0.071	0.25	0.26±0.15

Figure-4.4 shows the graphical representation of respirable dust in different process units. From this it is clear that finishing section the limit was exceeded, but in other processes, the dust concentration was comparable higher than atmospheric dust. Chronic exposure to dust may have a cumulative effect on workers and under risk.

Processes which involve the use of bitumen-containing materials at elevated temperatures can release airborne particulates into the workplace. TPM (Total Particulate Matter): this includes aerosol matter from the bitumen and inorganic material such as dust, rock fines, filler, etc. Because TPM methods collect material from non-bitumen sources the resulting values can suggest artificially high exposure values, especially in dusty environments.

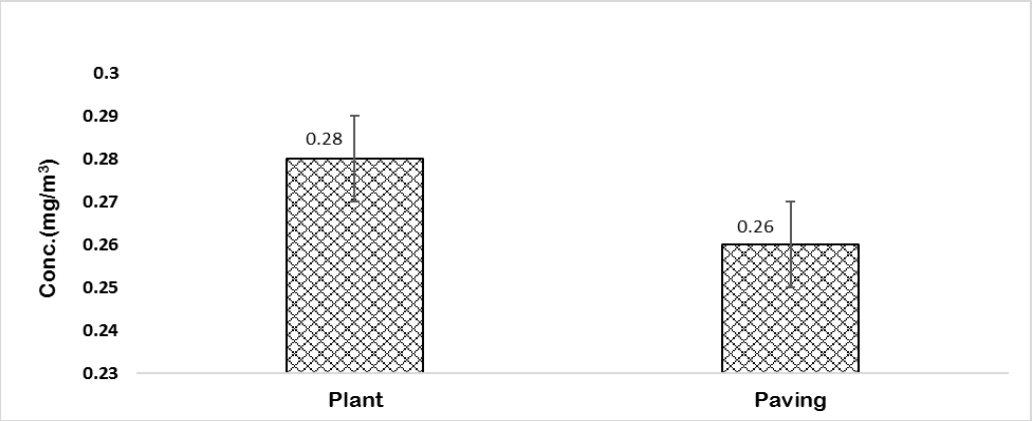

Figure-4.4: Respirable dust concentration in different location of Asphalt Industry.

Personal breathing-zone samples of the mixed aerosol have been taken for analysis of TPM by Brandt *et. al.,* (1985). Airborne concentrations have been measured for 17 jobs in six processes in manufacturing, road application, roofing, and indoor mastic laying. Time-weighted average exposures over 8 h (TWA (8 h)) ranged from 0.2 to 18 mg/ m^3 for TPM. In the present study, it was observed that the range 0.11 to 1.318 mg/ m^3, which was quite less than the above study. But the study carried among the Forty-five workers at 11 paving sites across the United States were evaluated for exposure to paving asphalt (bitumen) fumes was found that the range of individual exposures was 0.03–0.64 mg/m^3 of TPM, with an average exposure of 0.25 mg/m^3 (Anthony et al. 2002) and in the present study the range was more than double in both minimum and maximum range.

The exposure of road pavers to total particulates was studied by Heikkila et al. (2002). 13 paving sites where 11 different asphalt mixtures were laid. The arithmetic mean concentrations of total particulates in the breathing zone of road pavers were

0.6 mg/m^3. The highest bitumen fume concentrations (2.65 mg/m^3) were measured in manual mastic lying, that was, when the paving temperature was higher. In the present study, the mean respirable particulate was 0.28 mg/m^3 with highest concentration was 1.31 mg/m^3.

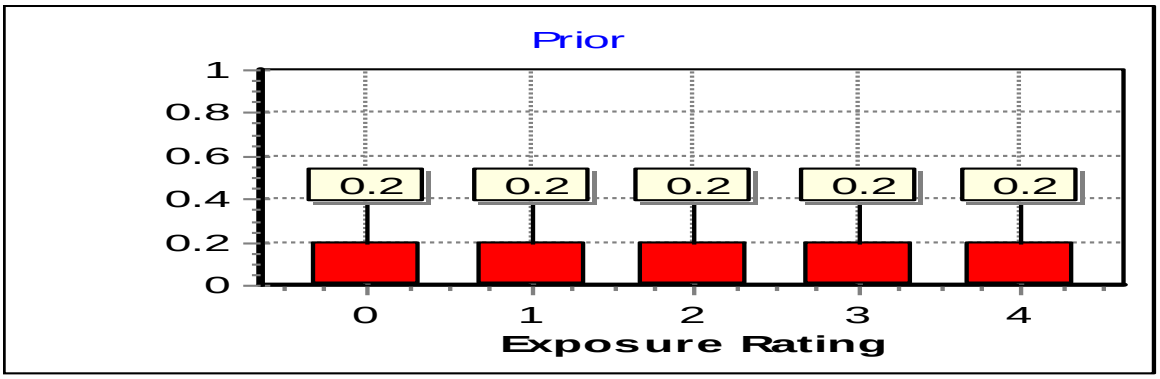

Fig. 4.5A

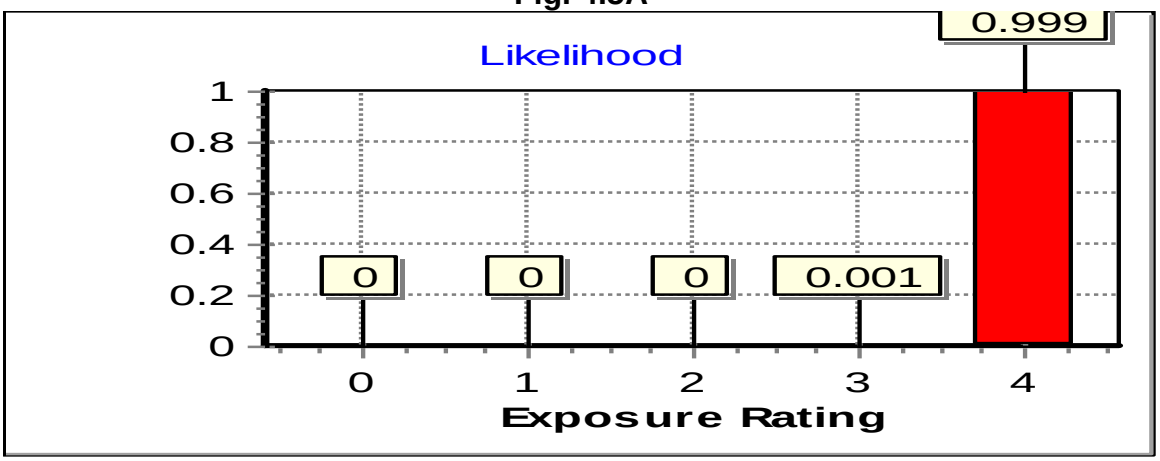

Fig. 4.5B

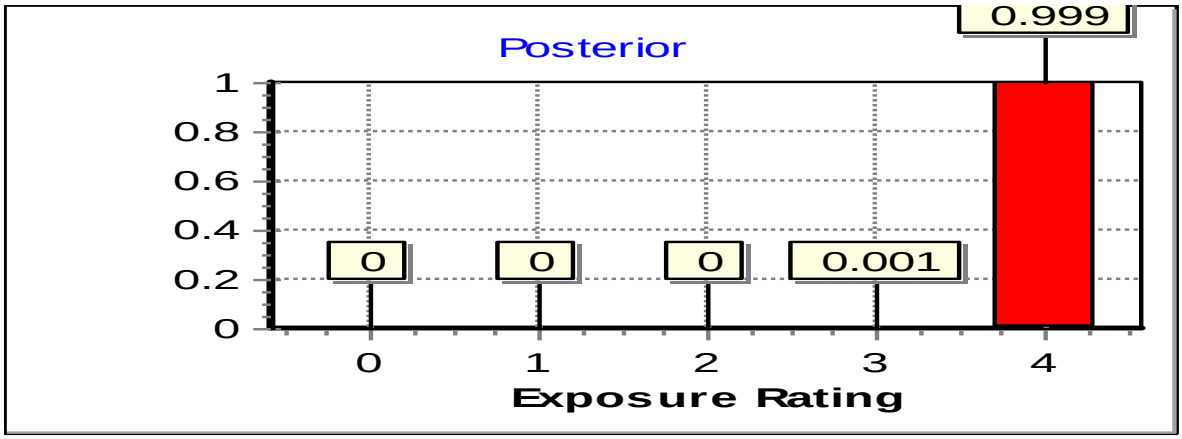

Fig. 4.5C

Figure-4.5(A-C): Bayesian modeling and assessment result of respirable dust concentration at Plant area in asphalt process Industry.

Figure 4.5A-4.5C shows the results of BDA (the three decision charts) for respirable dust in the plant process considering the exposure limit of 0.5 mg/m^3 for fumes as per ACGIH. A uniform prior probability distribution was used to represent the situation where we have no prior knowledge or expectations regarding this particular process (Figure 4.5A). Figure 4.5B shows the probability of likelihood decision in the pant process using monitoring data. Fig.4.5C presents the posterior as final decision probability as the of Figure 4.5A and Figure 4.5B. Figure 4.6A-4.6C shows the results of BDA (the three decision charts) for respirable dust in the paving process considering the exposure limit of 0.5 mg/m^3 for fumes as per ACGIH. A uniform prior probability distribution was used to represent the situation where we have no prior knowledge or expectations regarding this particular process (Figure 4.6A). Figure 4.6B shows the probability of likelihood decision in the pant process using monitoring data. Fig.4.5C presents the posterior as final decision probability as the of Figure 4.6A and Figure 4.6B. Table-4.3 contains a listing of typical actions and controls as prescribed by AIHA for workplace exposure. By assigning the exposure profile we were able to suggest control measurement in each process to reduce the exposure of respirable dust. In the asphalt process, we have obtained the result of prediction about each process unit by Bayesian model. The percentages of excess rate of respirable dust in both the process units (plant and paving) were belongs to the highest grade (grade 4/4+). These outcome final ratings indicating that the workers were regularly inhaling respirable dust. In the plant and paving unit's workers often have contact at high or very high concentrations. So, it was required to take the fast actions on control and safety measurement. Therefore, it was essential to have immediate safety adaptation by personal protective equipment of proper respiratory mask or engineering control like local ventilations or cross ventilation in order to prevent from being exposure to respirable dust to safeguard the workers' health. There should also need of chemical analysis of respirable dust and exposure surveillance like (i) protection of health of the individual employee, (ii) detection at an early stage any adverse health effects due to exposure of chemical enrich of respirable dust, (iii) assisting in the evaluation of control measures, (iv) detection of

hazards and assessment of risk or (v) the disease or health effect associated with exposure.

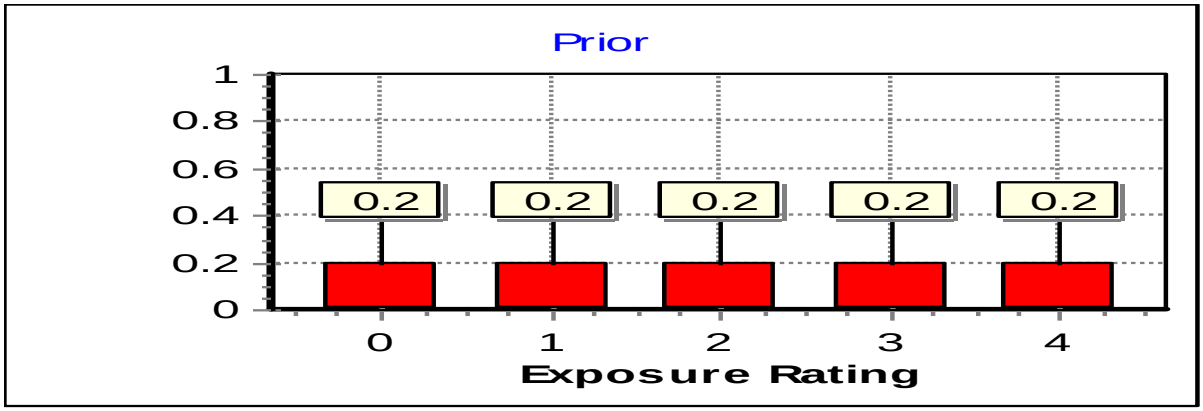

Fig. 4.6A

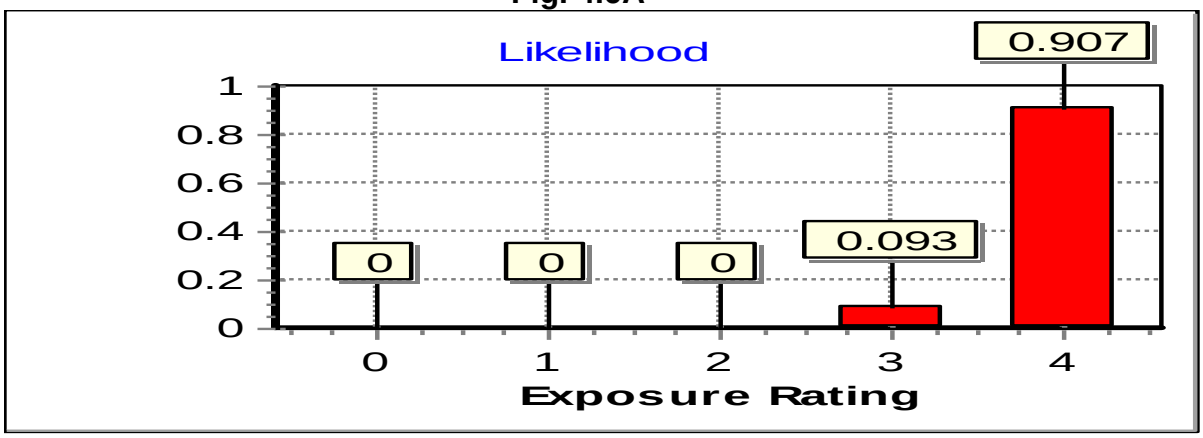

Fig. 4.6B

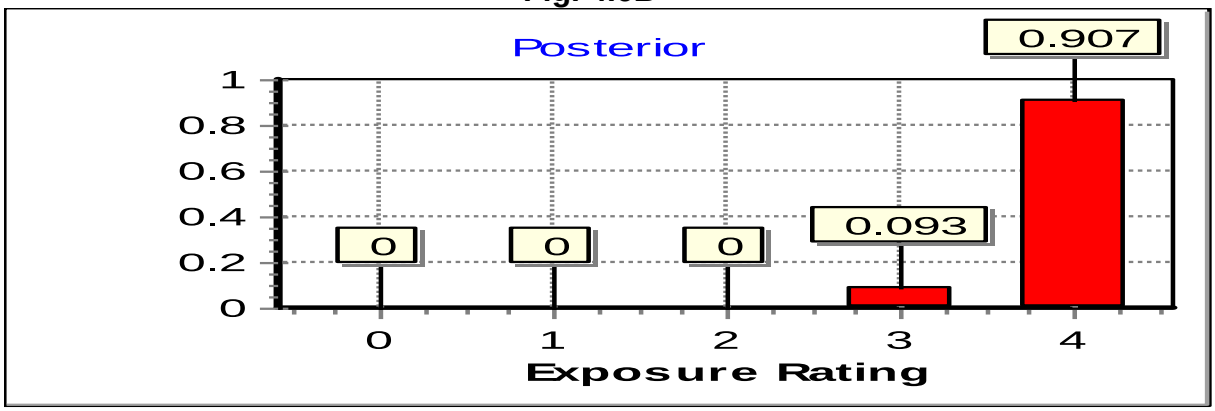

Fig. 4.6V

Figure-4.6(A-C): Bayesian modeling and assessment result of respirable dust concentration at paving unit process in asphalt process Industry.

Chapter~5

PAHs deposition & biomonitoring

Chapter-5

PAHs deposition and biomonitoring

5.1 PAHs depositions in the foundry workers

PAHs are a large group of organic compounds with two or more fused aromatic (benzene) rings. Low-molecular-weight PAHs (two and three rings) occur in the atmosphere, predominantly in the vapour phase, whereas multi-ringed PAHs (five rings or more) are largely bound to particles. Intermediate-molecular-weight PAHs (four rings) are partitioned between the vapour and particulate phases, depending on the atmospheric temperature (Srogi et al, 2007). Particle-bound PAHs are considered to be very hazardous to human health.

The mean concentration of PAHs exposure found among workers of various sections of foundries is presented in Table-5.1. The mean ΣPAHS concentration was 76.36±11.55 µg/m^3 in the foundries with range of 2.78 - 478.43 µg/m^3. All the 16 PAHs compounds detected were classified into two categories: low molecular weight (LM-PAHs containing two to three ring PAHs), and higher molecular weight (HM-PAHs, containing four to six ring PAHs. The PAHs with comparatively LM-PAHs and high vapour pressure, such as NAP, ACPy, ACE, FLU, PHE, ANT was contributing 55.1% (42.02±8.98 µg/ m^3) of ΣPAHs among the foundry workers and the HM-PAHs were 44.9% (37.34±4.03 µg/ m^3) respectively. But the HM-PAHs were carcinogenic or probably carcinogenic to human and defined as toxic compounds.

In my present study, among various PAHs detected, the most abundant PAHs were ACE (14.49±4.09 µg/ m^3), BaP (11.72±1.61 µg/ m^3), NAP (9.45±2.09 µg/ m^3), and ACPy (6.59±1.87µg/ m^3) and BghiP (6.54±1.11 µg/ m^3). Among ΣPAHs monitored for personal exposure, 27% exceeded the value of 100 µg/ m^3 prescribed by The National Institute for Occupational Safety and Health (NIOSH) workplace exposure for 8-hours' Time Weight Average (TWA). BaP is a potent mutagen and carcinogen. It is a public health concern because of its effect in industrial works, as an

environmental and workplace pollutant. BaP is listed as a Group 1 carcinogen by the IARC. In the present study, it was found that the BaP level in the foundry workplace was second highest concentration. Though there were exposure limits for personal exposure to BaP, the atmospheric limits prescribed by CPCB is 1.0 ng/ m^3. So, the foundry workers were exposed to very high level of BaP.

BaP also is classified as having a mutagenic mode of action (MOA) for inducing tumors, and was thought to require metabolic activation to become carcinogenic (USEPA, 2005). BaP is classified by the U.S. EPA as B2: a probable human carcinogen (ATSDR, 1995); based on numerous adult studies in several animal species (primates, rats, mice) that demonstrate BaP can increase the incidence of tumors. BaP was often used as a positive control in tumor formation experiments and in genotoxic (USEPA, 1994). The World Health Organization International Agency for Research on Cancer (IARC) concluded that there was sufficient evidence that BaP is carcinogenic (causes cancer) in experimental animals and that BaP was probably carcinogenic in humans (WHO, 1998).

The personal exposures of various PAH compounds in the different shop floors are shown in Table-5.2. The mean ∑PAHs and total carcinogenic PAHs (BaA, CHR, BbF, BkF, BaP, DahA, BghiP and IND) was 180.21 µg/m^3 and 60.50 µg/m^3 respectively in the molding section and higher than other sections. The personal exposure of BaP which was categorized as a most carcinogenic was 27.64±3.03 µg/m^3 in the blasting section. The exposure loads of ΣPAHs in the finishing section were 84.07±24.12 µg/m^3 followed by blasting (61.10±7.80 µg/m^3), melting (45.19±14.76 µg/m^3) and shaking-out sections (37.06±10.82 µg/m^3). It was found that the exposure limits prescribed by NIOSH has been exceeded in the molding section and workers were exposed to PAHs much higher than boundaries (100 µg/m^3). 80% of personal exposure PAHs exceeded the prescribed limits in the molding section, followed by 33% in finishing, 13% in melting,10% in blasting and 7% in shaking-out process section exceeded the limits.

Table-5.1: Mean of PAHs concentration in the workplace of foundry and their contribution in total PAHs:

PAH Compound	Molecular Weight	Molecular Formula	No. of Rings	Mean±SE ($\mu g/m^3$)	% of Total PAHs
Lower Molecular weight					
NAP	128.18	$C_{10}H_8$	2	9.45±2.09	12.38
ACPy	152.2	$C_{12}H_8$	3	6.59±1.87	8.63
ACE	154.2	$C_{12}H_{10}$	3	14.49±4.09	18.98
FLU	166.23	$C_{13}H_{10}$	3	5.59±2.90	7.32
PHE	178.24	$C_{14}H_{10}$	3	5.45±2.17	7.14
ANT	178.24	$C_{14}H_{10}$	3	0.45±0.11	0.59
Total(2-3rings)	-		2-3	42.02±8.98	55.1
Higher Molecular weight					
FLA	202.26	$C_{16}H_{10}$	4	1.65±0.33	2.16
PYR	202.06	$C_{16}H_{10}$	4	2.45±0.60	3.21
BaA	228.3	$C_{18}H_{12}$	4	2.72±0.96	3.56
CHR	228.3	$C_{18}H_{12}$	4	2.01±0.74	2.63
BbF	252.32	$C_{20}H_{12}$	5	0.45±0.14	0.59
BkF	252.32	$C_{20}H_{12}$	5	1.00±0.59	1.31
BaP	252.32	$C_{20}H_{12}$	5	11.72±1.61	15.35
DahA	278.35	$C_{22}H_{14}$	5	4.23±1.69	5.54
BghiP	276.34	$C_{22}H_{12}$	6	6.54±1.11	8.56
IND	276.34	$C_{22}H_{12}$	6	1.58±0.48	2.07
Total(4-3rings)	-		4-6	37.34±4.03	44.9
∑PAHs	-		2-6	76.36±11.55	100

The PAHs concentration detected in various sections of foundry were compared with the studies conducted elsewhere. PAHs concentration in personal air samples collected among workers employed in various process of foundry in the Danish iron foundry was 9. 6-11.2 µg/m^3 *(Hansen et al. 1994).* However, in the present study the levels were ranged between 2.78 and478.43 µg/m^3. The maximum level (478.43 µg/m^3) recorded in the present study was nine-fold higher than the total PAH concentrations (52 µg/m^3) reported in iron foundries in Ontario, California (Verma *et al.,* 1982). The maximum level of BaP in the present study (45.37 µg/m^3) was forty-fold higher than the level reported in the personal air samples of Canadian foundry workers (Gibson *et al.,* 1977). In the present study, level of total PAHs was 2-4 folds higher than the level recorded in a UK foundry (Unwin *et al.,* 2006), however, lower than the total PAHs (81.01µg/m^3) levels reported in German (Knecht *et al.,* 1986). The study carried out in Taiwan by Chen *et al.* (2011) showed the mean level of PAHs in the painting area (associated with molding activity) and in melting area was 95.51µg/m^3 and 18.42 µg/m^3 respectively which were less than half to present study (molding 82.64 µg/m^3 and melting 23.48 µg/m^3 respectively) levels. Table 5.3 shows the comparative exposure level of PAHs of the iron and steel industry workers in different country.

Table-5.2: Mean concentration of PAHs compounds (µg/m^3) in the personal exposure samples in different sections of the foundry workers

PAHs compounds	Sections				
	Molding(N=10)	Melting(N=15)	Shaking-out(N=13)	Blasting(N=10)	Finishing(N=12)
NAP	27.67±7.77	5.49±2.69	3.19±0.61	1.17±0.34	12.93±5.22
ACPy	21.50±8.91	4.13±2.06	1.32±0.22	0.90±0.16	7.69±3.15
ACE	46.36±18.99	8.78±4.16	2.54±0.63	2.05±0.55	18.39±8.16
FLU	4.12±2.23	12.36±10.96	0.42±0.34	7.78±5.55	2.12±1.02
PHE	8.66±3.73	0.47±0.19	10.44±8.88	6.20±4.30	2.97±2.00
ANT	1.39±0.51	0.32±0.13	0.10±0.03	0.11±0.03	0.49±0.19
FLA	4.25±1.40	0.92±0.29	0.53±0.17	0.29±0.07	2.72±0.64
PYR	5.77±2.51	1.30±0.37	0.85±0.24	0.69±0.10	4.31±1.79
BaA	4.76±1.97	0.44±0.11	0.32±0.10	0.28±0.08	8.49±4.13
CHR	7.23±3.77	0.42±0.11	0.27±0.10	0.50±0.24	2.82±1.47
BbF	1.61±0.70	0.29±0.08	0.11±0.04	0.49±0.25	0.05±0.04
BkF	0.18±0.11	2.65±2.34	0.07±0.05	1.09±0.13	0.57±0.20
BaP	11.09±4.66	3.23±1.22	13.26±2.79	27.64±3.03	7.91±2.77
DahA	20.78±8.58	1.35±0.50	1.46±0.77	0.06±0.04	0.49±0.15
BghiP	13.63±4.97	2.25±0.80	1.38±0.42	10.83±1.73	8.00±1.73
IND	1.23±0.77	0.80±0.30	0.82±0.63	1.03±0.51	4.12±2.08
***CPAHs**	60.50±7.19	11.43±1.14	17.68±4.49	41.92±9.74	32.43±3.64
∑PAHs	180.21±44.81	45.19±14.76	37.06±10.82	61.10±7.80	84.07±24.12

**C-PAHs-Carcinogenic PAHs (sum of BaA, BaA, CHR, BbF ,BkF, BaP ,DahA and IND)*

Table-5.3: PAHs exposures of workers in the iron and steel foundry industry in different country

Country	Mean ∑PAHs µg/m^3	Range µg/m^3	BaP µg/m^3	Reference
Canada	-	-	BDL-1.03	*Gibson et al., 1977*
USA	52	-	-	*Verma et al., 1982*
Germany	81.01	-	-	*Knecht et al., 1986*
Denmark	-	9. 6-11.2	-	*Hansen et al. 1994*
UK	15.8	0.4 – 1912.6	-	*Unwin et al., 2006*
Taiwan	Melting-95.51		-	*Chen et al. (2011)*
India (present Study)	76.36	2.78 -478.43	11.72	*Sen et al*

-Denotes no data

5.2 PAHs depositions in the asphalt workers

Personal exposure to mean concentration of PAHs among workers of two job categories in Asphalt is shown in Table-5.4. The mean ΣPAHS concentrations were 31.26 ±5.51 µg/m^3 with range of 1.64 – 184.71 µg/m^3. All the 16 PAHs compounds detected were classified into two categories: low molecular weight (LM-PAHs containing two to three ringed PAHs), and higher molecular weight (HM-PAHs, containing four to six ringed PAHs. The lower molecular weight of two and three benzene ring PAHs were abundant in both vapour and particulate phase and higher molecular weight PAHs with more than four benzene rings were suspended in the indoor or outdoor were in particulate phase. The percentage of lower molecular weight PAHs were 27.99 % and higher molecular weight PHAs with 72%. In the Asphalt workplace, the higher molecular weight PAHs were nearly threefold in concentration than lower molecular weight PAHs. The high molecular weight PAHs (HMW PAHs) were strongly carcinogenic and mutagenic (Karlsson *et al*., 2008; Laane *et al*., 2006; Ou *et al*., 2004).

Among various PAHs detected, the following PAHs were found in highest concentration BghiP (10.76±2.60 µg/ m^3), BaP (4.66±1.31 µg/ m^3), FLU (3.20±1.23 µg/ m^3), BkF (2.89±0.94 µg/ m^3), and PHE (1.94±0.55µg/ m^3) respectively. Among ΣPAHs monitored for personal exposure, 8% exceeded the value of 100 µg/ m^3 prescribed by The National Institute for Occupational Safety and Health (NIOSH) workplace exposure for 8-hours' Time Weight Average (TWA). It was found that BghiP exposure was highest among the 16 PAHs compounds followed by BaP. The most serious environmental impact of BghiP was its significant accumulation in organisms exposed to it. It was also toxic and a suspected carcinogen. BghiP was very stable and can remain in the environment for a long period of time - it was a Persistent Organic Pollutant (POP). There was little evidence available as to the full effect on human health following exposure to BghiP. In 1987, the International Agency for Research on Cancer designated BghiP as Group 3. Exposure to BghiP at normal background levels was unlikely to have any adverse effect on human health.

In the natural environment BghiP present as part of a mixture of Polycyclic Aromatic Hydrocarbons (PAHs). BghiP has been identified as a "priority hazardous substance" under the Water Framework Directive (European Commission, 2008) and was listed as one of the four PAHs which have a prescribed maximum concentration in drinking water. The most serious environmental impact of BaP was its significant accumulation in organisms exposed to it. BaP was stable and can remain (and travel) in the environment for a long period of time - it was a Persistent Organic Pollutant (POP). Releases of BaP therefore cause concern at a global environmental level as well as on a local scale. Exposure to BaP may damage the reproductive system and cause cancer. Ingestion of BaP may cause gastrointestinal irritation. Dermal contact with BaP may lead to skin irritation. In the natural environment, BaP occurs as part of a mixture of Polycyclic Aromatic Hydrocarbons (PAHs). The full effects of BaPon human health were unknown, however studies have shown that inhalation of PAHs or dermal contact with PAHs for long periods of time can cause cancer. The International Agency for Research on Cancer has designated BaP as a probable carcinogen. However, exposure to BaP at normal background levels was unlikely to have any adverse effect on human health.

The personal exposures of various PAH compounds in the two categories of asphalt mainly Plant and paving were shown in Table-5.5. The mean ∑PAHs and total carcinogenic PAHs (CPAHs) was 64.30 µg/m^3 and 45.18 µg/m^3 in the paving area and 12.13 µg/m^3 and 9.44 µg/m^3 respectively in the plant. So, the paving workers are more exposed to total PAHs and carcinogenic PAHs than Plant workers. BghiP (25.60±5.69 µg/m^3) was high in the paving area followed by BaP (12.16±2.99 µg/m^3). In the plant area BghiP (2.18±0.95 µg/m^3) was high in the paving area followed by BkF (2.18±0.95 µg/m^3). The carcinogenic PAHs load in the paving area was70% of the Total PAHs and in the plant, it was 78%. So, it was clear that the workers in the asphalt industry exposed with more carcinogenic PAHs which was also higher molecular weight PAHs compounds and exists in particulate phase.

Table-5.4: Mean PAHs concentration in the workplace of Asphalt and their contribution in total PAHs:

PAH Compound	Molecular Weight	Molecular Formula	No. of Rings	Mean±SE ($\mu g/m^3$)	% of Total PAHs
Lower Molecular weight					
NAP	128.18	$C_{10}H_8$	2	1.11±0.23	3.54
ACPy	152.2	$C_{12}H_8$	3	0.64±0.19	2.04
ACE	154.2	$C_{12}H_{10}$	3	1.742±0.72	5.50
FLU	166.23	$C_{13}H_{10}$	3	3.20±1.23	10.24
PHE	178.24	$C_{14}H_{10}$	3	1.94±0.55	6.20
ANT	178.24	$C_{14}H_{10}$	3	0.15±0.02	0.47
Total(2-3rings)	-		2-3	8.75±1.73	27.99
Higher Molecular weight					
FLA	202.26	$C_{16}H_{10}$	4	0.46±0.17	1.49
PYR	202.06	$C_{16}H_{10}$	4	0.15±0.02	0.47
BaA	228.3	$C_{18}H_{12}$	4	0.08±0.01	0.26
CHR	228.3	$C_{18}H_{12}$	4	0.06±0.01	0.18
BbF	252.32	$C_{20}H_{12}$	5	1.19±0.26	3.80
BkF	252.32	$C_{20}H_{12}$	5	2.89±0.94	9.24
BaP	252.32	$C_{20}H_{12}$	5	4.66±1.31	14.91
DahA	278.35	$C_{22}H_{14}$	5	1.75±0.37	5.61
BghiP	276.34	$C_{22}H_{12}$	6	10.76±2.60	34.43
IND	276.34	$C_{22}H_{12}$	6	0.51±0.13	1.62
Total(4-3rings)	-		4-6	22.51±4.05	72.00
∑PAHs	-		2-6	31.26±5.51	100

The PAHs concentration detected in asphalt industry researchers were compared with the studies conducted elsewhere. The study conducted in the Greater Boston area (McClean et al., 2004) found that the concentration of total PAHs in the paving was geometric mean 4.1 µg/m^3 with range 0.3-40 µg/m^3 and in the plant 2.6 µg/m^3 with range 0.3-6.4 µg/m^3. In our present the geometric mean total PAHs was 36.27 µg/m^3 with range 1.64-184.71 µg/m^3 in paving area and in the plant area 9.05 µg/m^3 with range 1.80-36.01 µg/m^3. Therefore, it could be concluded that the workers of the present study were exposed higher concentration of PAHs than Greater Boston area asphalt workers.

The results of occupational exposure to PAHs among workers handling bituminous fumes in Polish enterprises carried out indicated that the average concentrations of total PAHs in the breathing zone of workers during road paving were 7.12 µg/m^3 (Pooeniak,2005). Whereas in our study the total PAHs in the breathing zone of workers during road paving was 64.30±11.99 µg/m^3 which was higher than the Polish workers.

In bitumen fumes emitted in investigating processes, volatile PAHs were present primarily in the gaseous phase, ACE, FLU and NAP constituted as main part of all determined PAHs, which was about 72-85%. It was observed in the present study that the volatile PAHs concentration contributed nearly 28 % of the total PAHs.

The exposure of road pavers to polycyclic aromatic hydrocarbons (PAHs) was studied by Heikkila et al., (2002) at 13 paving sites where 11 different asphalt mixtures were laid. The arithmetic mean concentrations of PAHs in the breathing zone of road pavers were 5.03 µg/m3. Airborne concentrations PAHs in the range of 4 to 2508 ng m−3 for a total of eleven selected PAHs found by Brandt et al. (1985) in the breathing zone. Table 5.6 shown the comparative exposure level of PAHs of the asphalt industry workers in different countries.

Table-5.5: Mean concentration of PAHs compounds (µg/m^3) in the personal exposure samples in different section of the asphalt workers

PAHs compounds	Section	
	Plant (N=38)	Paving (N=22)
NAP	0.92±0.19	1.42±0.55
ACPy	0.43±0.16	1.00±0.44
ACE	0.61±0.11	3.64±1.92
FLU	0.64±0.33	7.63±3.14
PHE	0.96±0.02	3.63±1.13
ANT	0.04±.01	0.33±0.12
FLA	0.03±0.01	1.22±0.43
PYR	0.09±0.02	0.25±0.05
BaA	0.07±0.01	0.10±0.02
CHR	0.06±001	0.04±0.01
BbF	1.84±0.38	0.05±0.02
BkF	1.91±0.69	4.58±2.27
BaP	0.32±0.11	12.16±2.99
DahA	1.58±0.50	2.06±0.55
BghiP	2.18±0.95	25.60±5.69
IND	0.46±0.11	0.58±0.32
CPAHs	9.44±2.91	45.18±1.18
∑PAHs	12.13±1.46	64.30±11.99

Table-5.6: PAHs exposures of workers in the Asphalts industry in different country

Country	Mean ∑PAHs µg/m^3	Range µg/m^3	BaP µg/m^3	Reference
Denmark	0.5	-	-	*Byrd and Mikkelsen 1979*
UK	-	0.004-2.51	-	*Brandt et al. (1985)*
USA	Total-220.0 Paving-500.0	Total- ND-440 Paving-240-740		*Zey 1992*
UK	Paving-4.1 Plant-2.6	Paving-0.3-40 Plant-0.3-6.4	-	*McClean et al., 2004*
Poland	Paving-7.12	-	-	*Pooeniak,2005*
Finland	Paving-5.03	-	-	*Heikkila et al., (2002*
India (present Study)	Total-31.26 Paving-64.30 Plant-12.13	1.64-184.71	Total-4.66 Paving-12.16 Plant-0.32	*Sen et al*

-denotes no data

5.3 Biological monitoring of PAHs exposure among the foundry workers

The demographic information of the foundry workers and controls were shown in Table-5.7 Mean age of the foundry workers was tiny compared with the control. The foundry workers were approximately 4years older on average (workers: 37.97±11.49 years and control: 34.5±11.6 years). Also, the percentage of current smoking status, tobacco chewing and alcohol taking were significantly higher among the workers.

Table-5.7: Demographical and lifestyle characteristics of the foundry workers and control subjects:

Variables		Workers N=60 n (%)	Control N=26 n (%)
Age(years)	≤25	12(20.0)	3(11.5)
	26-35	21(35.0)	4(15.4)
	36-45	14(23.3)	12(46.2)
	≥45	13(21.7)	7(26.9)
Mean age ±SD		37.97±11.49	34.5±11.6
Smoking	Yes	19(31.7)	5(19.2)
	No	41(68.3)	21(80.8)
Tobacco Chewing	Yes	11(18.3)	3(11.5)
	No	49(81.7)	23(88.5)
Alcohol	Yes	16(26.7)	7(26.9)
	No	44(73.3)	19(73.1)

Table-5.8 shows the mean urinary 1-OHP levels were significantly (<0.05) higher among the foundry workers (1.35±1.29 µmole/mole creatinine) than the control (0.38±0.73 µmole/mole creatinine). Also, the OHPHE levels of the workers (2.85±1.77 µmole/mole creatinine) was significantly higher than control (1.65±1.38 µmole/mole creatinine), although it was also elevated than the control group.

Table-5.8: Levels of urinary OHPHE and 1-OHP among the foundry workers and control population

Study Group		N	OHPHE µmole/mole creatinine	1-OHP µmole/mole creatinine
Total Workers		60	2.85±1.77*	1.35±1.29*
Section/ processing units	**Molding**	10	3.66±1.52	2.29±1.26
	Melting	15	3.11±2.06	2.16±1.49
	Shaking-out	13	1.54±1.06	0.59±0.80
	Blasting	10	2.99±2.07	0.89±0.76
	Finishing	12	1.77±1.10	0.77±0.83
Control		26	1.65±1.38	0.38±0.73
Values were represented as mean ± SD; *P <0.05(1-OHP: Workers Vs Control)				

The higher level of OHPHE among the workers in molding section (3.66±1.52 µmole/mole creatinine) & melting section (3.11±2.06 µmole/mole creatinine) sections and also 1-OHP level was significantly higher in the molding (2.29±1.26 µmole/mole creatinine) and melting section (2.16±1.49 µmole/mole creatinine) respectively. In personal monitoring of PAHs, it was found that that PYR and PHE PAHs compounds were in higher concentration in molding and melting sections which were inhaled by workers at the time of their duty and got metabolites in the urine.

According to the experience 1-OHP levels were significant with experience of ≥1, <1- ≥5, <5- ≥10 and <10 yrs. (Table-5.9). The OHPHE levels were significant highly only those who were working more than 10yr in the foundry compared with

control and It was indicated that the chronic exposure effect PAHs compounds. Among all the categories of foundry workers the 1-OHP level was higher than the unexposed group.

Table-5.9: Average OHPHE and 1-OHP level in the urine of the study group by experience status and control

Experience (yrs.)	N	OHPHE µmole/mole creatinine	1-OHP µmole/mole creatinine
≥1	9	1.08±1.10	1.91±1.64*
<1- ≥5	18	1.31±1.57	1.12±1.47*
<5- ≥10	16	1.51±1.36	0.73±0.90
<10	18	2.73±2.10*	0.96±0.96*
Control	26	1.65±1.38	0.38±0.73

The concentration of mean PAHs metabolites (OHPHE and with respect to the 1-OHP) with personal habits of smoking, tobacco chewing and alcohol consumption were shown in Table-5.10.

Table- 5.10: The concentration of PAHs metabolites with respect to personal habits among the Study Subjects

Subjects	Habits		N	OHPHE µmole/mole creatinine	1-OHP µmole/mole creatinine
Control (N=26)	**Smoking**	Yes	5	1.64±1.12	0.78±1.20
		No	21	1.65±1.46	0.28±0.57
	Tobacco Chewing	Yes	3	1.06±0.13	0.04±0.02
		No	23	1.72±1.45	0.42±0.76
	Alcohol	Yes	7	1.98±1.95	0.16±0.23
		No	19	1.72±1.45	0.45±0.83
Study Subjects (N=60)	**Smoking**	Yes	19	2.32±1.87	1.52±1.56
		No	41	2.70±1.74	1.28±1.16
	Tobacco Chewing	Yes	11	2.12±1.29	1.89±1.65
		No	49	3.00±1.86	1.42±1.22
	Alcohol	Yes	16	2.14±1.55	1.42±1.52*
		No	44	2.86±1.84	1.41±1.23
Values were represented as mean ± SD,					

The 1-OHP level was observed higher among the workers with smoking habits (1.52±1.56 µmole/mole creatinine) than non-smoker workers (1.28±1.16 µmole/mole creatinine) as well as controls with smoking habits (0.78±1.20 µmole/mole creatinine) and non-smoker control was 0.28 ±.57 µmole/mole creatinine (figure-5.1). So, there is a clear indication that workers were more exposed to PAHs than control and smoking also inducing the increased dose of the inhalation of PAHs and smoking was one of the source of pyrene compound which was metabolized and form 1-OHP. It was also observed that the mean

levels of OHPHE among the non-smoker workers (2.70±1.74 µmole/mole creatinine) were also in elevated range than the non-smoker controls (1.65±1.46 µmole/mole creatinine).

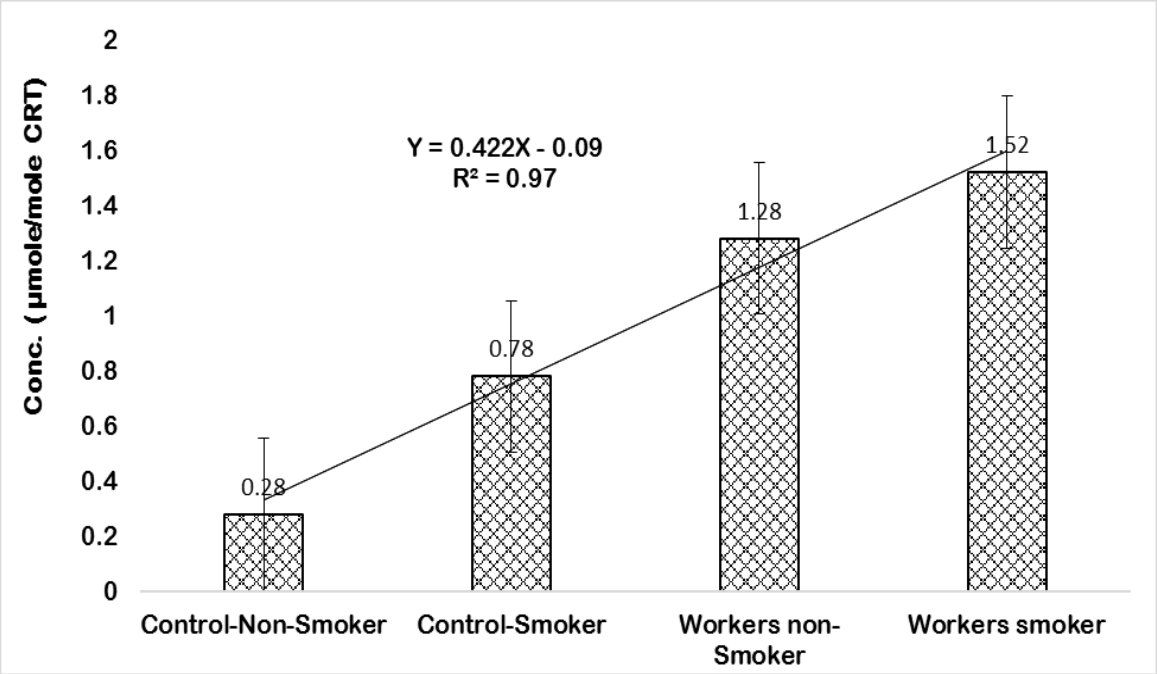

Figure-5.1: 1-OHP level among the foundry workers with smoking habits and control

The mean 1-OHP level of the workers with tobacco chewing habits were higher than the control (1.89±1.65 µmole/mole creatinine vs. 0.04± 0.02µmole/mole creatinine). The mean level of 1-OHP concentration of the workers with non-tobacco chewing habits was 1.42±1.22 µmole/mole creatinine and it was double compared with control (0.42± 0.76µmole/mole creatinine). No significant difference was found between alcoholic and non-alcoholic workers, but the OHPHE and 1-OHP level of the workers with alcoholic consumption were greater than the control (figure-5.2).

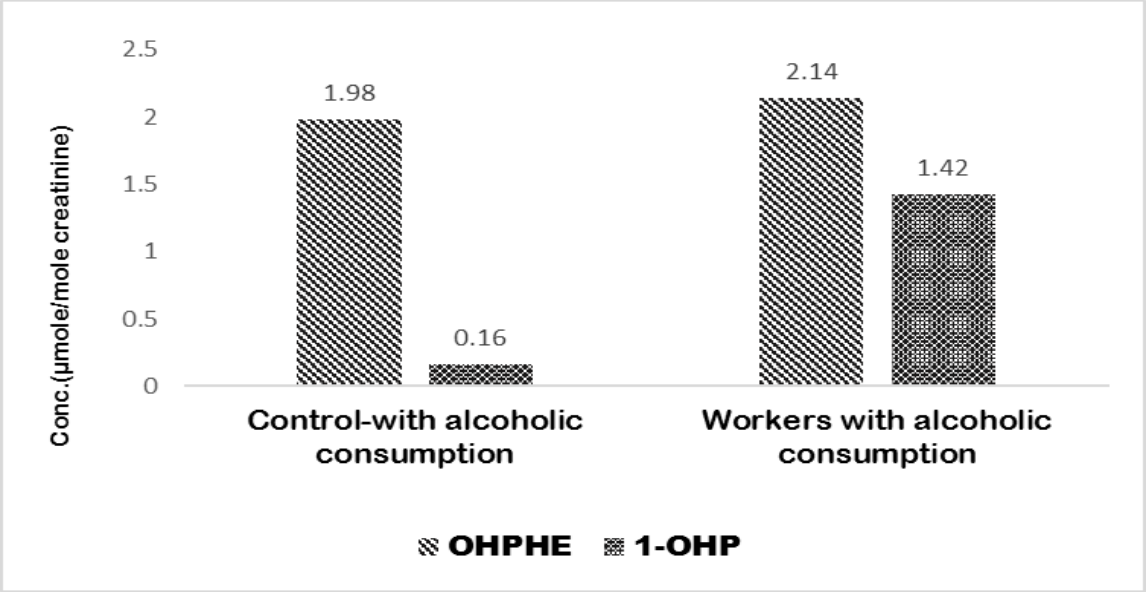

Figure-5.2: OHPHE & 1-OHP level among the workers of the foundry and Control with alcoholic habit.

In this present study, I have measured the urinary levels of OHPHE and 1-OHP of PAHs metabolite among the foundry workers those were exposed to PAHs compound at their workplace. The results show that the urinary level of OHPHE among the foundry workers have higher (2.85±1.77 vs. 1.65±1.38 µmole/mole creatinine) and 1-OHP (1.35±1.29 vs. 0.38±0.73 µmole/mole creatinine, $p<0.05$) than unexposed population. The OHPHE level was higher among the workers in molding and melting sections and both levels were significantly higher than controls This data revealed that workers were at more risk in the molding and melting sections due to high level of personal exposure to PAHs than other process sections. The 1-OHP levels among the foundry workers located in rural Denmark reported 0.42 and 0.11 µmole/mole creatinine among smokers and non-smoker respectively (Ny et al.,1993). The average 1-OHP levels in the finish iron foundry were 2.7, 1.8 and 3.6µmole/mole creatinine among the low, medium and high exposure group of workers respectively (Lauwerys, 1996). A study by Saranya *et al*. (2013) reported 2.15 ±0.87 µmole/mole creatinine of urinary 1 – OHP among foundry workers in Coimbatore, India. The mean 1-OHP levels (0.09 to 5.10 µmole/mol creatinine) reported the present study was compared

with the earlier studies. The mean concentration of 1-OHP was 1.35±1.29 µmole/mole in the present study ranged from among various sections of foundry.

Very few studies have reported the OHPHE metabolites levels due exposure to PAHs in the workplace. A Study conducted among smoker and non- smoker in general populations of Germany reported 1.83 ±0.84 and 1.50 ±1.05 µmole/mole creatinine respectively (Gtindel et al, 1996). Although there was no study available in foundries for OHPHE to compare the levels with the present study, the levels recorded in the present study were higher than the levels reported elsewhere. However, the current study was not observed significant difference between smoker and non-smokers.

Jongeneelen (1985) proposed a biological limit of 2.3µmol/mole 1-OHP for coke oven workers. Ny et al (1993) proposed 4.33 µmole/mole for Soderberg potroom workers. Lauwerys (1996) proposed a tentative urinary 1-OHP limit value was 1.4 µmole/mole. These limits were based on TLV of airborne PAHs concentration and the relationship between airborne PAHs level and urinary 1-OHP concentration. In this study, the mean 1-OHP value of the foundry workers was 1.35±1.29 µmole/mole which was below the recommended biological exposure limits (BEL) prescribed by different authors.

The biological monitoring observed in our study indicates that the foundry workers were exposed to carcinogenic PAHs and increased urinary concentrations of OHPHE and 1-OHP among the foundry workers were the supposition. However, due to lack of authorized biological exposure limits (BEL) value, risk involved was not computed. Moreover, the dermal uptake appears to be a significant pathway of PAHs exposure and dermal absorption also influence the biological level of PAHs metabolites. The epidemiological studies of foundry workers with its urinary metabolites as a dose indicator, will be a more reliable to evaluate the risk basically in the tropical subcontinent of south Asia. Table-5.11 shown the 1-OHP level among the foundry workers of different country which were carried out by different researchers.

Table-5.11: Urinary 1-hydroxypyrene level among the workers exposed to asphalt or asphalt fumes

Occupationally exposed population	Urinary 1-hydroxypyrene (µ mol/mol creatinine)	References
Road Paver **Office worker (control)**	4.2 0.9	*Hatjian et al.1995*
Road Paver **University staff and students (control)**	0.61 0.26	*Burgaz e al.1992*
Road Paver **Nonoccupationally**	0.6 0.26 (Non-Smoker) 0.28 (Smoker)	*Jongeneelen et al. 1988*
Road Pavement (Swedish)	0.96 (pre-shift) 0.60 (post-Shift)	*Järvholm et al.,1999*
Road Paver	1.4	*Mcclean et al, 2004*
Road Paver	1.1 (0.1-9.51)	*Brandt and Watson, 2003*
Road Paver	0.25	*Unwin et al. 2006*
Road Paver **Plant** **Office worker (control)** **Subject**	1.31 1.37 0.38 1.27 (Non-Smoker 1.48 (Smoker)	*Present Study*

5.4 Biological monitoring of PAHs exposure among the asphalt workers

The demographic information of the asphalt workers and controls are shown in Table-5.12. Mean age of the asphalt workers was quite lower compared with the control. The asphalt workers were approximately 7yrs. younger on average (workers: 27.10±7.64 yrs. and control: 34.5±11.6 yrs.). Also, the percentage of current smoking status, tobacco chewing and alcohol taking were significantly lower among the workers.

Table-5.12. Demographical and lifestyle characteristics of the asphalts workers and control

Variables		Workers N=60 n (%)	Control N=26 n (%)
Age(yrs.)	≤25	32(53.3)	3(11.5)
	26-35	22(36.7)	4(15.4)
	36-45	05(08.3)	12(46.2)
	≥45	01(01.7)	7(26.9)
Mean age ±SD		27.10±7.64	34.5±11.6
Smoking	Yes	23 (38.3)	5(19.2)
	No	37(61.7)	21(80.8)
Tobacco Chewing	Yes	23(38.3)	3(11.5)
	No	37(61.7)	23(88.5)
Alcohol	Yes	26(43.3)	7(26.9)
	No	34(56.7)	19(73.1)

Table-5.13 shows the mean urinary 1-OHP levels were significantly (<0.05) higher among the asphalt workers (1.35±0.32 µmole/mole creatinine) than the control (0.38±0.73 µmole/mole creatinine). However, the OHPHE levels of the workers (1.72±0.32 µmole/mole creatinine) were not significantly higher than control (1.65±1.38 µmole/mole creatinine), although it was also elevated than the control group.

Table-5.13: Levels of urinary OHPHE and 1-OHP among the asphalt workers and control population

Study Group		N	OHPHE µmole/mole creatinine	1-OHP µmole/mole creatinine
Total Workers		60	1.72±0.32	1.35±0.32*
Section wise	Plant	38	1.69±0.29	1.37±0.39*
	Paving	22	1.76±0.53	1.31±0.55*
Control		26	1.65±1.38	0.38±0.73
*Values were represented as mean ± SD; *P <0.05(1-OHP: Workers Vs Control)*				

The PAHs metabolites among the different section of the workers also varies. The level of OHPHE among the plant workers (1.69±0.29 µmole/mole creatinine) was less than the paving workers (1.76±0.53 µmole/mole creatinine)., but the 1-OHP level was higher among the plant workers (1.37±0.39 µmole/mole

creatinine) than paving (1.31±0.55 µmole/mole creatinine). Also, both the metabolites (OHPHE & 1-OHP) among the asphalt plant and paver workers were in the elevated range than the control (Fig.5.3). So, it was clearly indicating that the Asphalt workers were exposed to PAHs while performing their duty.

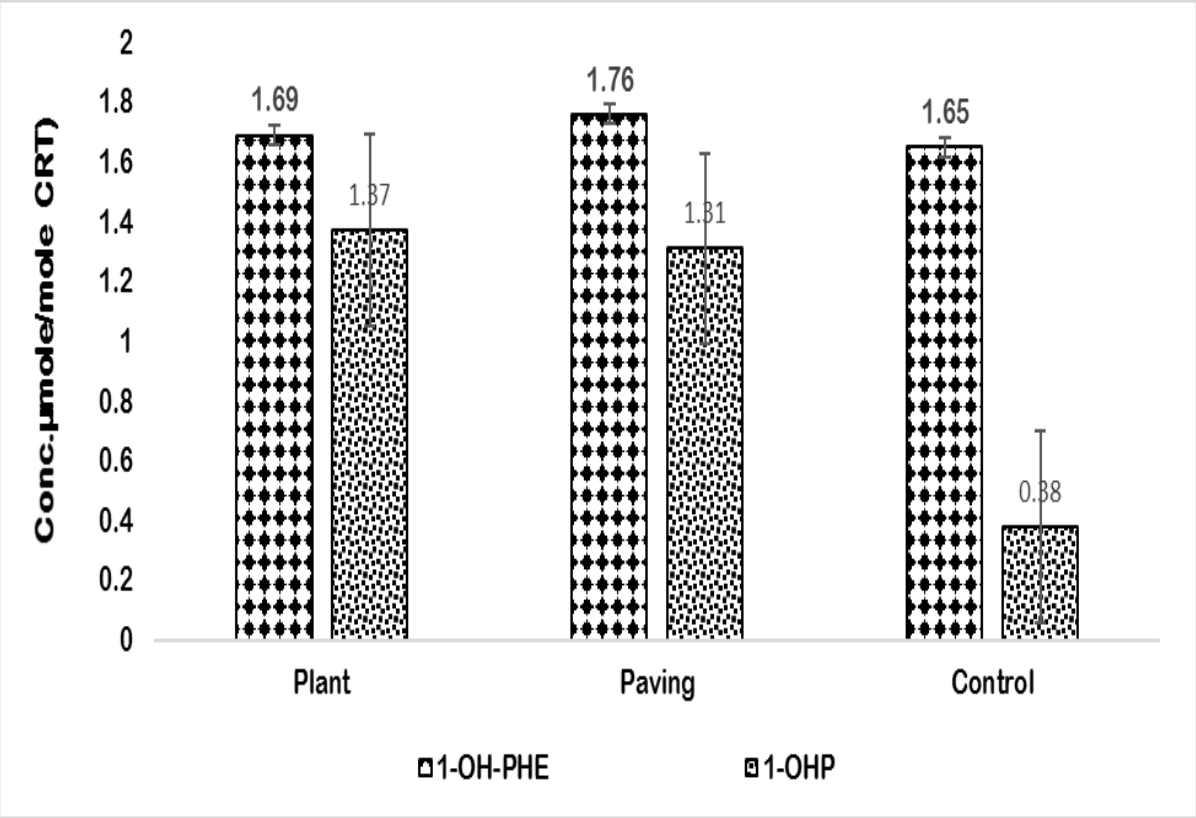

Figure-5.3: OHPHE & 1-OHP level among the workers of the asphalt workers (Plant & Paving) and Control

According to the experience OHPHE (1.99±1.48 µmole/mole creatinine) and 1-OHP (1.80±1.39 µmole/mole creatinine) levels were significantly higher with experience of <5- ≥10 compared to other categories of experience and control (Table-5.14). The OHPHE levels were not increased among the other categories of experience (≥1, <1- ≥5 and <10) compared with control. But the urinary 1-OHP level found in elevated range than the control in all categories of experienced workers.

Table-5.14: Average OHPHE and 1-OHP level in the urine of the study group by experience status and control

Experience (yrs.)	N	OHPHE µmole/mole creatinine	1-OHP µmole/mole creatinine
≥1	28	1.43±0.28	1.27±0.38
<1- ≥5	17	1.55±0.63	1.08±0.28
<5- ≥10	6	1.99±1.48*	1.80±1.39*
<10	9	1.42±0.68	0.75±0.64
Control	26	1.65±1.38	0.38±0.73

Values were represented as mean ± SD, *P values (<0.05) as compared to controls

The concentration of mean PAHs metabolites with respect to the personal habits is shown in Table 5.15. The highest concentration of mean urinary 1-OHP was observed among the asphalt workers with smoking habits (1.48±0.57 µmole/mole creatinine) than non-smoker workers (1.27±0.38 µmole/mole creatinine) as well as Controls with smoking habits (0.78±1.20 µmole/mole creatinine) (figure-5.4). Therefore, it could be noticed in the asphalt work practices that there was a stimulus of PAHs exposure among the workers and smoking habits inducing the level of 1-OHP among the workers.

Table-5.15: The concentration of PAHs metabolites with respect to personal habits among the Study Subjects

Subjects	Habits		N	OHPHE µmole/mole creatinine	1-OHP µmole/mole creatinine
Control (N=26)	Smoking	Yes	5	1.64±1.12	0.78±1.20
		No	21	1.65±1.46	0.28±0.57
	Tobacco Chewing	Yes	3	1.06±0.13	0.04±0.02
		No	23	1.72±1.45	0.42±0.76
	Alcohol	Yes	7	1.98±1.95	0.16±0.23
		No	19	1.72±1.45	0.45±0.83
Study Subjects (N=60)	Smoking	Yes	23	2.17±0.51	1.48±0.57
		No	37	1.43±0.28	1.27±0.38
	Tobacco Chewing	Yes	23	2.51±0.51	2.33±0.68
		No	37	1.22±0.26	0.74±0.25
	Alcohol	Yes	26	1.73±0.33	1.47±0.57
		No	34	1.70±0.39	1.26±0.37
Values were represented as mean ± SD,					

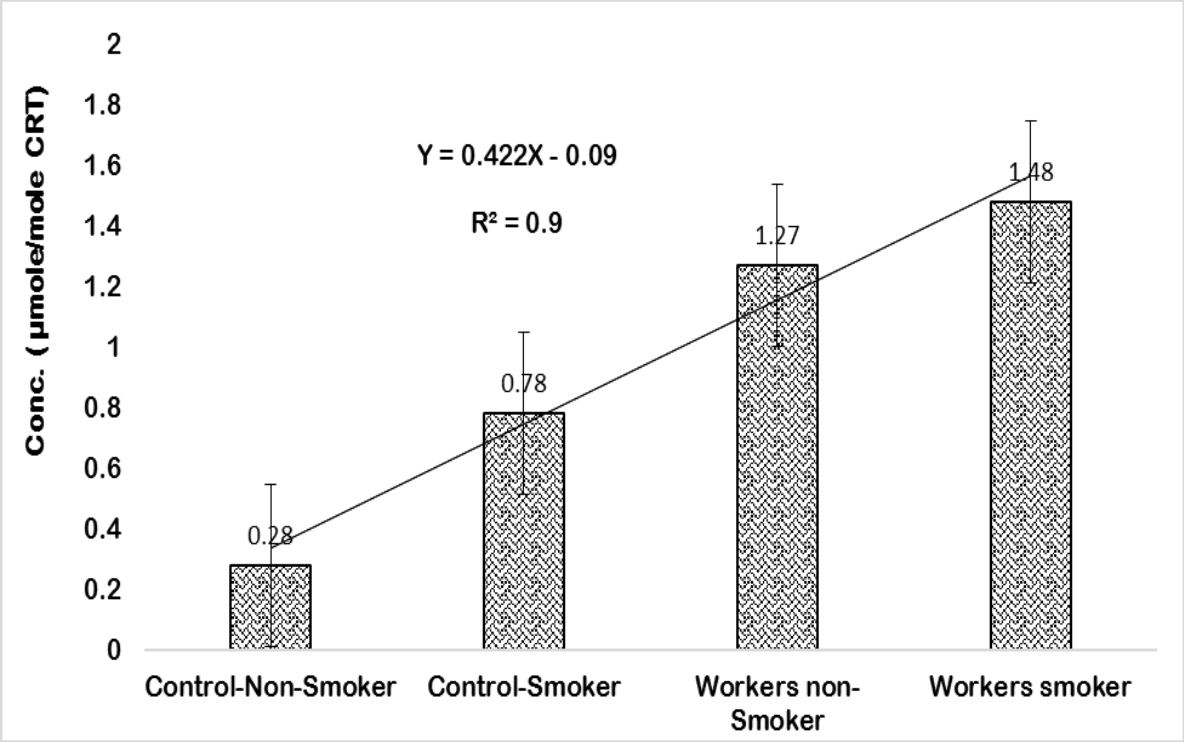

Figure-5.4: 1-OHP level among the asphalt workers with smoking habits and control

It was also observed that the mean levels of OHPHE among the smoker workers (2.17±0.51 µmole/mole creatinine) were also in elevated range than the non-smoker controls (1.43±0.28 µmole/mole creatinine). The mean 1-OHP level of the workers with tobacco chewing habits were higher than the control (2.33±0.68 µmole/mole creatinine vs. 0.04± 0.02µmole/mole creatinine). The mean level of 1-OHP concentration of the workers with non- tobacco chewing habits was 0.75± 0.25 µmole/mole creatinine and it was nearly 2-fold compared with control (0.42± 0.76µmole/mole creatinine). It was found that the mean 1-OHP level was higher among the workers with alcohol habits (1.47±0.57 µmole/mole creatinine) and with-out alcohol habits (1.26±0.37 µmole/mole creatinine) than the controls (with alcohol 0.16±0.23 µmole/mole creatinine; without alcohol 0.45±0.83 µmole/mole creatinine).

The present study with urinary levels of OHPHE and 1-OHP indicated that asphalt workers were exposed to PAHs compound in their workplace. The study carried out among the Swedish road pavement workers (Järvholm et al.,1999) found that the concentration of 1-OHP in urine was higher for the road pavers than for the referents. The geometric means of the urinary 1-OHP was 0.96 (range 0.23-4.0) µmole/mole in post shift compared with 0.60 (range 0.14-2.2) µmole/mole control. The present study found that the geometric means of the urinary 1-OHP was 0.29 (range 0.01-9.51) µmole/mole in post shift.

The study conducted among 20 pavers at Greater Boston area (Mcclean et al, 2004) found that the post shift urinary 1-OHP concentration was 1.4±1.4 µmole/mole and in our study the urinary 1-OHP concentration was 1.31±0.55 µmole/mole among the 22 pavers which was higher than the present study. Another study they found that in bitumen or asphalt application the level of 1-OHP among the workers was 1.1 µmole/mole with range 0.1-2.0 µmole/mole Brandt and Watson, 2003). In the present study, the 1-OHP range was 0.01-9.51 µmole/mole. The range was larger than Brandt study. Study conducted by Unwin et al. (2006) found that the 1-OHP level among the 7 road paver workers in United Kingdom was 0.25 µmole/mole and it was relatively less than then the present research work. Table-5.16 shown the 1-OHP level among the Asphalt workers of different country which were carried out by different researchers.

Jongeneelen proposed a biological limit of 2.3 µmole/mole 1-OHP for cokeoven workers. Ny et al proposed 4.33µmol/mole for Soderberg potroom workers. Lauwerys proposed a tentative urinary 1-OHP limit value as 1.4 µmole/mole. These limits were based on TLV of airborne PAHs concentration and the relationship between airborne PAHs level and urinary 1-OHP concentration. In this study the mean 1-OHP value of the asphalt workers was 1.35±0.32 µmole/mole which was below the recommended biological exposure limits (BEL) prescribed by different authors.

The biological monitoring observed in this present study indicates that the foundry workers were exposed to carcinogenic PAHs and increased urinary concentrations of OHPHE and 1-OHP among the foundry workers were the supposition. However due to lack of authorized biological exposure limits (BEL) value, risk involvement was not computed. More over the dermal uptake appears to be a significant pathway of PAHs exposure and dermal absorption also influence the biological level of PAHs metabolites. The epidemiological studies of foundry workers with its urinary metabolites as a dose indicator, will be a more reliable to evaluate the risk basically in the tropical subcontinent of south Asia.

Table-5.16: Urinary 1-hydroxypyrene in workers exposed to Foundry process by various authors and comparison with present study

Occupationally exposed population	Urinary 1-hydroxypyrene (μ mol/mol creatinine)	References
Foundry workers (Denmark)	0.42 (Non-Smoker 0.11 (Smoker)	Ny et al.,1993
Foundry workers (Finland)	2.7 (Low Exposure) 1.8 (Medium Exposure) 3.6 (high Exposure)	Lauwerys, 1996
Foundry workers (India)	2.15	Saranya et al,2013
Foundry workers **Office worker (control)**	1.35 1.28 (Non-Smoker 1.52 (Smoker) 0.38	*Present Study*

Chapter~6

PAHs induced risk assessment

Chapter-6

PAHs induced risk assessment

6.1 Health risk assessment of foundry workers exposed to PAHs

Health risk assessments were carried out by inhalation of PAHs exposure data in order to quantify lung cancer risk. The toxic equivalent concentration (TEQ) was used to estimate the corresponding lifetime lung cancer risks of workers based on the present findings.

BaP exposure and the lung cancer risk for occupational exposure based on a data bank provided by an epidemiological study conducted by Redmond *et al*., (1976) was repoted. It was suggested the unit risk of 7.0×10^{-5} for a 25 year occupational PAHs exposure was corresponding with the averaged BaP concentration of $1ng/m^3$ (Tsai *et al.*, 2001). The unit risk was proposed to estimate the lung cancer risk caused by the lifetime exposure, therefore, it has been adopted by a recent study for assessing the lung cancer risks of general adult's exposure to the ambient atmospheric PAHs (Tsai et.al 2001; Lin *et al.*, 2008: Zhang *et al.,*2011). However, for PAH exposure to the US Environmental Protection Administration suggested a different risk of 6.4×10^{-7} by using the same data bank based on its total PAH content (USEPA 1984). Since recent studies have indicated BaP can be a better indicator than total PAH content on characterizing the carcinogenic potency of PAHS (Petry *et al.*, 1996; Zhang *et al.*, 2011), the unit risk suggested by Pott was used in this study.

The inhalation ambient air risk associated with occupational exposure to PAHs in the foundry is shown in the Table-6.1 The total unit risk of PAHs in this occupational exposure group was $1.55x10^{-02}$ with Bap (1.05×10^{-02}) & DahA (4.14×10^{-03}) contributing 94.45% of total risk. According to the World Health

Organization (WHO, 2000) Air Quality Guidelines for Europe, the unit risk was 10^{-5}(one extra cancer case in 100,000 exposed individuals in the general population) and USEPA guideline was 10^{-6} (Morrone, 2007; USEPA,1984). In the present study, the estimated lifetime cancer risk value was $1.55x10^{-02}$ (1.5 people may develop cancer risk among 100 foundry workers exposed by PAHs in the workplace). Ramírez *et al.* (2011) estimated an average lifetime lung cancer risk of total PAHs as 1.2×10^{-4} (1.2 additional cases per 10,000 people exposed) in the industrial area of southern Europe. The present study showed a higher risk of PAHs among foundry workers may be due to their continuous exposure adjacent to the source in the closed environment. In the foundry environment, the PAHs emission retention time was much higher due to close environment where as in ambient environment the dilution of air reduces to less retention time and half-life of the PAHs compounds was higher than open atmosphere where direct sunlight with UV light degrade the PAHs compounds faster. The degradation of gas phase PAHs was different from that of particulate phase PAHs. Gas phase PAHs were susceptible to degradation via OH radical; while particulate phase PAHs were susceptible to photo-degradation (Xiang *et al*., 2007). Most of the higher molecular PAHs like BaA, CHR, BbF, BkF, BaP, DahA, BghiP and IND were available in particulate phase and as per carcinogenicity factor these PHAs were more toxicity value than gas/ vapour phase PAHs. So, these PAHs compounds (BaA, CHR, BbF, BkF, BaP, DahA, BghiP and IND) having more a half-life because of non-availability of sunlight in the indoor environment of the foundry and they persist in the foundry environment for a longer time which may affect the workers' health and influence the risk of lung cancer.

Table -6.1: The risk expression estimation value with PAHs compounds in different location of the foundry with total risk value:

PAH Compounds	IUR† (µg/m3)-1	Chronic RfC* (mg/m3)	Inhalation Ambient Air Risk					
			Molding	Melting	Shaking - out	Blasting	Finishing	Total foundry
NAP	**3.4 x10^{-05}**	0.003a	7.67x10^{-04}	1.52x10^{-04}	8.85x10^{-05}	3.24x10^{-05}	3.59X10^{-04}	2.62X10^{-04}
BaA	**0.00011**	-	4.27 x10^{-04}	3.95x10^{-05}	2.87x10^{-05}	2.51x10^{-05}	7.62x10^{-04}	2.44x10^{-04}
CHR	**1.1 x10^{-05}**	-	6.49 x10^{-05}	3.77 x10^{-06}	2.42x10^{-06}	4.49x10^{-06}	2.53x10^{-05}	1.80x10^{-05}
BbF	**0.00011**	-	1.44x10^{-04}	2.60x10^{-05}	9.87x10^{-06}	4.40x10^{-05}	4.49x10^{-06}	4.04x10^{-05}
BkF	**0.00011**	-	1.62x10-05	2.38x10-04	6.28x10^{-06}	9.78x10^{-05}	5.11x10^{-05}	8.97x10^{-05}
BaP	**0.0011**	-	9.95x10^{-03}	2.90x10^{-03}	1.19x10^{-02}	2.48x10^{-02}	7.10x10^{-03}	1.05x10^{-02}
DahA	**0.0012**	-	2.03x10^{-02}	1.32x10^{-03}	1.43x10^{-03}	5.87x10^{-05}	4.80x0^{-04}	4.14x10^{-03}
IND	**0.00011**	-	1.10x10^{-04}	7.18x10^{-05}	7.36x10^{-05}	9.24x10^{-05}	3.70x10^{-04}	1.42x10^{-04}
Total Risk			3.18x10^{-02}	4.75x10^{-03}	1.35x10^{-02}	2.52x10^{-02}	9.15x10^{-03}	1.55x10^{-02}

* *Rfc-inhalation reference concentration.*

6.2 Health risk assessment of asphalt workers exposed to PAHs

The inhalation ambient air risk associated with occupational exposure to PAHs in the asphalt workers is shown in the Table-6.2. The total unit risk of PAHs in this occupational exposure group was $6.41x10^{-03}$ with Bap ($4.18\ x10^{-03}$) & DahA ($1.71\ x10^{-03}$) contributing 92% of total risk. According to the World Health Organization (WHO, 2000) Air Quality Guidelines for Europe, the unit risk was 10^{-5} (one extra cancer case in 100,000 exposed individuals in the general population) and USEPA guideline was 10^{-6} (Morrone, 2007; USEPA,1984). In the present study, the estimated lifetime cancer risk value was $6.41x10^{-03}$ (6.4 people may develop cancer risk among 10,00 people exposed in the asphalt workers). Ramírez et al. (2011) estimated an average lifetime lung cancer risk of total PAHs as 1.2×10^{-4} (1.2 additional cases per 10,000 people exposed) in the industrial area of southern Europe. The present study showed a higher risk of PAHs among asphalt workers may be due to emission of particulate phase PAHs which were more carcinogenic than vapour phase PAHs. Also, the BaP which was the most toxic and carcinogenic PAHs compounds was emitted form bitumen in high concentration and contributing 65% of total cancer risk.

Table-6.2: The risk expression estimation value of PAHs compounds in asphalt workers:

PAH Compounds	IUR† (µg/m3)-1	Chronic RfC* (mg/m3)	Inhalation Ambient Air Risk		
			Plant	Paving	Total Asphalt
NAP	0.000034	0.003a	$2.55x10^{-05}$	$3.94x10^{-05}$	$3.08x10^{-05}$
BaA	0.00011	-	$6.80x10^{-06}$	$8.97x10^{-06}$	$7.18x10^{-05}$
CHR	0.000011	-	$5.90x10^{-07}$	$3.59x10^{-07}$	$5.38x10^{-06}$
BbF	0.00011	-	$1.65x10^{-04}$	$4.49x10^{-06}$	$1.07x10^{-04}$
BkF	0.00011	-	$1.71x10^{-04}$	$4.11x10^{-04}$	$2.59x10^{-04}$
BaP	0.0011	-	$2.87x10^{-04}$	$1.09x10^{-02}$	$4.18x10^{-03}$
DahA	0.0012	-	$1.55x10^{-03}$	$2.02x10^{-03}$	$1.71x10^{-03}$
IND	0.00011	-	$4.13x10^{-05}$	$5.20x10^{-05}$	$4.58x10^{-05}$
Total Risk			$2.24x10^{-03}$	$1.34x10^{-02}$	$6.41x10^{-03}$

. * *Rfc-inhalation reference concentration*

Chapter~ 7

Summary

Chapter- 7

Summary

Risk associated with the PAHs in workplace environment was very much concern, even with low dose of exposure for its carcinogenic property. Also, the degree of exposure level of PAHs in the work environments is much higher than the general population. Once PAHs entered into the body, they are persisting in the body in the form of parent compounds in different organ or metabolised.

In India, workers engaged in both asphalt and foundry associated jobs were not considered much important in the aspects on organic pollutants (PAHs) exposure in the workplace. Also, the majority of the industry having lack of data on the PAHs exposure and its effect and risk involving the workplace. Therefore, the present study was conducted to evaluate the exposure to dust, PAHs, bio-monitoring its metabolites and associated risk in among the foundry and asphalt workers.

Dose PAHs was measured using personal air monitoring devices where gaseous and particulate phase of PAHs was trapped on sorbent tube and filters and then analysed for PAHs content. The Internal dose was measured as urinary PAHs metabolites which was recommended as biomarkers of exposure. The risk assessment was carried out with help of AIHA exposure categorization for dust exposure and the risk of PAHs compounds exposure in the indoor workplace through inhalation, Risk Assessment Information System.

Workplace respirable dust monitoring was carried out in the shop floors of the foundry throughout the full work shift and the levels were found to be relatively higher in the finishing section and it has also exceeded the ACGIH standard (TLV 3.0 mg/m^3) of respirable dust. In the foundry study, we have obtained from the result of prediction about each process unit by Bayesian model that the percentage of the excess rate of respirable dust in the Shakeouts, Felting and Finishing sections.

The levels of respirable dust in the asphalt workplace was not exceeding the ACGIH standard (TLV 3.0 mg/m^3) of respirable dust. The highest dust concentration also observed in the plant and it was 1.31 mg/m^3. In the asphalt process, we have obtained

the result of prediction about each process unit by Bayesian model. The percentages of excess rate of respirable dust in both the process units (plant and paving) were belongs to the highest grade and final ratings indicating that the workers were regularly inhaling respirable dust.

The PAHs exposure among the foundry workers also found in the various sections of workplace the mean ΣPAHS concentration was 76.36±11.55 μg/m^3 in the foundries with ranged 2.78 - 478.43 μg/m^3. The most abundant PAHs were ACE (14.49±4.09 μg/ m^3), BaP (11.72±1.61 μg/ m^3), NAP (9.45±2.09 μg/ m^3), and ACPy (6.59±1.87μg/ m^3) and BghiP (6.54±1.11 μg/ m^3). Among ΣPAHs monitored for personal exposure, 27% exceeded the value of 100 μg/ m^3 prescribed by The National Institute for Occupational Safety and Health (NIOSH) workplace exposure for 8-hours' Time Weight Average (TWA).

The mean ∑PAHs and total carcinogenic PAHs (BaA, CHR, BbF, BkF, BaP, DahA, BghiP and IND) was 180.21 μg/m^3 and 60.50 μg/m^3 respectively in the molding section and higher than other sections. The personal exposure of BaP which was categorized as a most carcinogenic was 27.64±3.03 μg/m^3 in the blasting section. The exposure loads of ΣPAHs in the finishing section were 84.07±24.12 μg/m^3 followed by blasting (61.10±7.80 μg/m^3), melting (45.19±14.76 μg/m^3) and shaking-out sections (37.06±10.82 μg/m^3). 80% of personal exposure PAHs exceeded the prescribed limits in the molding section, followed by 33% in finishing, 13% in melting,10% in blasting and 7% in shaking-out process section exceed the limits.

It was also found that the asphalt workers also exposed to PAHs in the workplace and the mean ΣPAHS concentrations was found 31.26 ±5.51 μg/m^3 with ranges of 1.64 – 184.71 μg/m^3. Among various PAHs detected, the following PAHs were found in highest concentration BghiP (10.76±2.60 μg/ m^3), BaP (4.66±1.31 μg/ m^3), FLU (3.20±1.23 μg/ m^3), BkF (2.89±0.94 μg/ m^3), and PHE (1.94±0.55μg/ m^3). Among ΣPAHs monitored for personal exposure, 8% exceeded the value of 100 μg/ m^3 prescribed by The National Institute for Occupational Safety and Health (NIOSH) workplace exposure for 8-hours' Time Weight Average (TWA). It was found that BghiP exposure was highest among the 16 PAHs compounds followed by BaP. The most serious environmental impact of

BghiP was its significant accumulation in organisms exposed to it. It was also toxic and a suspected carcinogen. BghiP was very stable and can remain in the environment for a long period of time.

An indirect exposure assessment urinary 1-OHP and OHPHE are the indicator of internal dose. It was found that urinary 1-OHP levels were significantly (<0.05) higher among the foundry workers (1.35±1.29 µmole/mole creatinine) than the control (0.38±0.73 µmole/mole creatinine). Also, the OHPHE levels of the workers were at significant higher with control, although it was also elevated than the control group. The 1-OHP level was observed higher among the workers with smoking habits than non-smoker workers as well as controls with smoking habits (0.78±1.20 µmole/mole creatinine) and non-smoker control was 0.28 ±.57 µmole/mole creatinine. In this study, the mean 1-OHP value of the foundry workers was 1.35±1.29 µmole/mole which was below the recommended biological exposure limits (BEL) prescribed by different authors.

Also, urinary 1-OHP levels were significantly (<0.05) higher among the asphalt workers (1.35±0.32 µmole/mole creatinine) than the control (0.38±0.73 µmole/mole creatinine). The PAHs metabolites among the different section of the workers also varied. The level of OHPHE among the plant workers (1.69±0.29 µmole/mole creatinine) was less than the paving workers (1.76±0.53 µmole/mole creatinine). The highest concentration of mean urinary 1-OHP was observed among the asphalt workers with smoking habits (1.48±0.57 µmole/mole creatinine) than non-smoker workers (1.27±0.38 µmole/mole creatinine) as well as Controls with smoking habits (0.78±1.20 µmole/mole creatinine). It was also observed that the mean levels of OHPHE among the smoker workers (2.17±0.51 µmole/mole creatinine) were also in elevated range that the non-smoker controls (1.43±0.28 µmole/mole creatinine). In this study the mean 1-OHP value of the asphalt workers was 1.35±0.32 µmole/mole which was below the recommended biological exposure limits (BEL) prescribed by different authors.

Estimates have been made of the burden of cancer attributable to PAHs factors and of the contribution of risk in of occupational cancer because, there has long been concern that airborne carcinogens contribute to the global burden of cancer, especially of the

lung, which receives the most substantial inhaled doses. The total unit risk of PAHs among the foundry workers was estimated as $1.55x10^{-02}$ with Bap ($1.05\ x10^{-02}$), DahA ($4.14\ x10^{-03}$) contributing 94.45% of total risk and the estimated lifetime cancer risk value was $1.55x10^{-02}$ (1.5 people may develop cancer risk among 100 foundry workers exposed by PAHs in the workplace).

The total unit risk of PAHs among the foundry workers was estimated as was $6.41x10^{-03}$ with Bap ($4.18\ x10^{-03}$), DahA ($1.71\ x10^{-03}$) contributing 92% of total risk and The total unit risk of PAHs in this occupational exposure group was $6.41x10^{-03}$ with Bap ($4.18\ x10^{-03}$) & DahA ($1.71\ x10^{-03}$) contributing 92% of total risk. According to the World Health Organization (WHO, 2000) Air Quality Guidelines for Europe, the unit risk was 10^{-5} (one extra cancer case in 100,000 exposed individuals in the general population) and USEPA guideline was 10^{-6} (Morrone, 2007; USEPA,1984). In the present study, the estimated lifetime cancer risk value was $6.41x10^{-03}$ (6.4 people may develop cancer risk among 10,00 people exposed in the asphalt workers).

The present study demonstrated that PAH exposure and its metabolites in Asphalt and Foundry workers may be risk to their health if proper and suitable precautionary methods such as using appropriate Personal Protective devises are not used. Though the concerned Industry management or proprietor provides the facilities to the workers an awareness should be created among the trade unions, middle level workers and individual worker for effective control measures. This will help in reducing the exposures and will create healthy work environment.

Chapter~8

General Discussion & Conclusion

Chapter-VIII

General Discussion and conclusion

Occupational exposure risk and internal dose of PAHs metabolism comparison between two occupational groups

In this present study, the two categories of workers were included to carry out the exposure assessment of Dust and PAHs and internal dose of PAHs metabolites.it was found that the both the groups of workers were exposed to ubiquitous hazardous pollutant in the workplace at the time of performing their duty and they were chronically exposed in their present environment. Comparing the respirable dust between the two-occupational group it was observed that the foundry workers were chronically exposed to dust concentration was much higher than asphalt workers (foundry:1.76±1.59 mg/m^3 with asphalt: 0.27±0.22 mg/m^3) (Table-8.1). It was also observed that the minimum and maximum range of respirable dust exposure among the foundry workers (0.10-10.90 mg/m^3) was higher than asphalt workers (0.05-1.31 mg/m^3).

Table-8.1: The respirable dust exposure level among the foundry and asphalt workers:

Variable (industry)	Mean±SD	Range
	Conc. (mg/m^3)	
Asphalt	0.27±0.22	0.05-1.31
Foundry	1.76±1.59	0.10-10.90

Exposure to mean levels of PAHs with a range of individual compounds in these two-occupational groups is shown in the Table-8.2. The mean level of total PAHs among

the foundry workers was 79.36±11.55 µg/m^3 and 31.26±5.51 µg/m^3. It was found that the foundry workers were exposed to more than two-fold more PAHs in the workplace than the asphalt workers. Except BbF, BkFand BghiP the mean level of other individual PAHs was also found in the higher range in foundry than asphalt. The mean personal exposure level of BkF was 2.89±0.94 µg/m^3 in the asphalt than foundry (0.45± 0.14µg/m^3). Also, the figure-8.1 represents graphical distribution of personal exposure of PAHs among the foundry and asphalt workers

The mean level of BbF and BghiP were 1.19±0.26 µg/m^3 and 10.76±2.60 µg/m^3 in the asphalt compared with BbF 0.45±0.14 µg/m^3 and BghiP 6.54±1.11 µg/m^3. The carcinogenic total PAHs was also higher in foundry workers (30.25±3.56 µg/m^3) than asphalt workers (21.90±3.97 µg/m^3). Among the PAHs measured in foundry, the ACE compound level was more (180.24 µg/m^3) and in the asphalt the highest concentration was found BaP (59.06 µg/m^3). It was found that 27% foundry PAHs sample exceeded the standard prescribed by NIOSH for workplace exposure than asphalt workers which only exceeded 8%.

Table-8.2: The PAHs exposure level among the foundry and asphalt workers.

PAHs compounds	Foundry	Asphalt
	Conc. (µg/m^3)	
NAP	9.45±2.09 (BDL-74.41)	1.11±0.23 (BDL-12.42)
ACPy	6.59±1.87 (BDL-90.69)	0.64±0.19 (BDL-7.96)
ACE	14.49±4.09 (BDL-180.24)	1.72±0.72 (BDL-35.74)
FLU	5.59±2.90 (BDL-165.46)	3.20±1.23 (BDL-57.71)
PHE	5.45±2.17 (BDL-116.74)	1.94±0.55 (BDL-21.93)
ANT	0.45±0.11 (BDL-3.68)	0.15±0.15 (BDL-1.91)
FLA	1.65±0.33 (BDL-11.54)	0.46±0.17 (BDL-6.56)
PYR	2.45±0.60 (BDL-23.01)	0.15±0.02 (BDL-0.76)
BaA	2.72±0.96 (BDL-42.60)	0.08±0.01 (BDL-0.50)
CHR	2.01±0.74 (BDL-38.95)	0.06±0.01 (BDL-0.25)
BbF	0.45±0.14 (BDL-6.22)	1.19±0.26 (BDL-8.96)
BkF	1.00±0.59 (BDL-35.37)	2.89±0.94 (BDL-48.63)
BaP	11.72±1.61 (BDL-45.70)	4.66±1.31 (BDL-59.06)
DahA	4.23±1.69 (BDL-86.86)	1.75±0.37 (BDL-14.30)
BghiP	6.54±1.11 (BDL-50.36)	10.76±2.60 (BDL-79.06)
IND	1.58±0.48 (BDL-24.66)	0.51±0.13 (BDL-6.29)
CPAHs	30.25±3.56 (2.24-114.50)	21.90±3.97 (BDL-136.40)
∑PAHs	79.36±11.55 (2.78-478.43)	31.26±5.51 (1.64-184.71)

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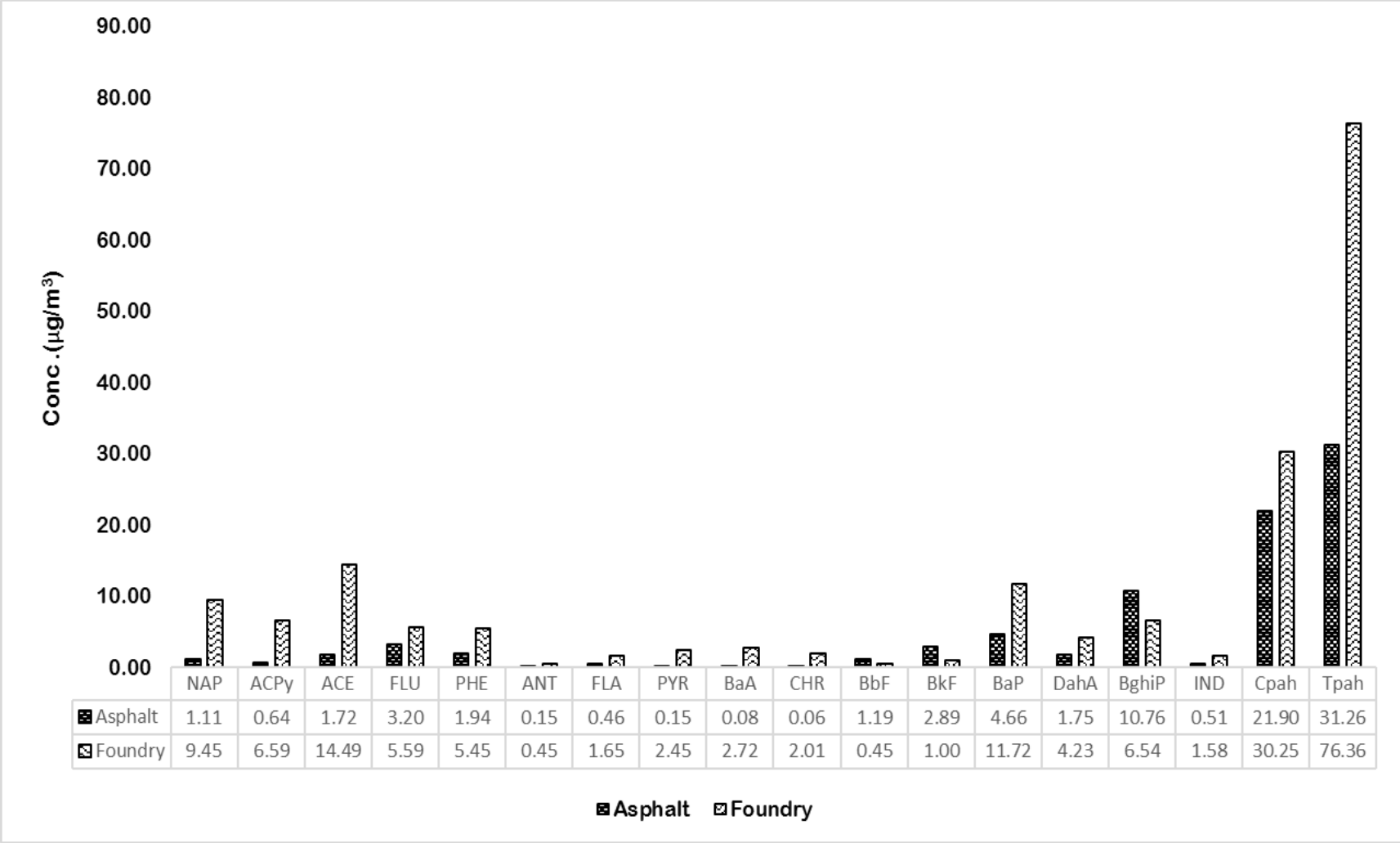

	NAP	ACPy	ACE	FLU	PHE	ANT	FLA	PYR	BaA	CHR	BbF	BkF	BaP	DahA	BghiP	IND	Cpah	Tpah
Asphalt	1.11	0.64	1.72	3.20	1.94	0.15	0.46	0.15	0.08	0.06	1.19	2.89	4.66	1.75	10.76	0.51	21.90	31.26
Foundry	9.45	6.59	14.49	5.59	5.45	0.45	1.65	2.45	2.72	2.01	0.45	1.00	11.72	4.23	6.54	1.58	30.25	76.36

Figure-8.1: Personal exposure of PAHs among the foundry and asphalt workers.

As discussed in Chapter 6.1 Health risk assessment of foundry workers exposed to PAHs, the total unit risk of PAHs in the foundry workers was 1.55x10-02 with Bap (1.05 x10-02) & DahA (4.14 x10-03) contributing 94.45 % of total risk and Chapter 6.2 Health risk assessment of asphalts workers exposed to PAHs, the PAHs risk among the asphalts workers was $6.41x10^{-03}$ with BaP ($4.18\ x10^{-03}$) & DahA ($1.71\ x10^{-03}$) contributing 91.89% of total risk (Table-8.3). As per Air Quality Guidelines for Europe, the unit risk was 10^{-5}(one extra cancer case in 100,000 exposed individuals in the general population) and USEPA guideline was 10^{-6} [WHO,2000; USEPA, 1984]. In the present study, the estimated lifetime cancer risk value was $1.55x10^{-02}$ (1.55 people may develop cancer risk among 100 people exposed in the foundry) and $6.41x10^{-03}$ among the foundry workers (6.41 workers may develop cancer risk among 1000 workers).

Table-8.3: The risk expression estimation value with PAHs compounds in different location of the Asphalt workers with total risk value:

PAHs compounds	Foundry	Asphalt
NAP	$2.62X10^{-04}$	$3.08x10^{-05}$
BaA	$2.44x10^{-04}$	$7.18x10^{-05}$
CHR	$1.80x10^{-05}$	$5.38x10^{-06}$
BbF	$4.04x10^{-05}$	$1.07x10^{-04}$
BkF	$8.97x10^{-05}$	$2.59x10^{-04}$
BaP	1.05x10-^{02}	$4.18x10^{-03}$
DahA	$4.14x10^{-03}$	$1.71x10^{-03}$
IND	$1.42x10^{-04}$	$4.58x10^{-05}$
Total PAHs Risk	$1.55x10^{-02}$	$6.41x10^{-03}$

The urinary level of PAHs metabolites among both the occupational exposure groups were found in the elevated range than the control group (Table-8.4) which has suggested that the workers were chronically exposed to more PAHs than the un-exposed group.

Table-8.4: Levels of urinary PAHs Biomarkers among the study population and control among Foundry and asphalt workers.

Study Group	OHPHE µmole/mole creatinine	1-OHP µmole/mole creatinine
Foundry	2.85±1.77*	1.35±1.29*
Asphalt	1.72±0.32*	1.35±0.32*
Control	1.65±1.38	0.38±0.73

Values were represented as mean ± SD, *P values (<0.05) as compared to controls.

From the figure-8.2 it was observed that the urinary level of OHPHE among the foundry workers (2.85± 1.77 µmole/mole creatinine) were higher than asphalt workers, but there was no difference in the 1-OHP level in both the occupational groups.

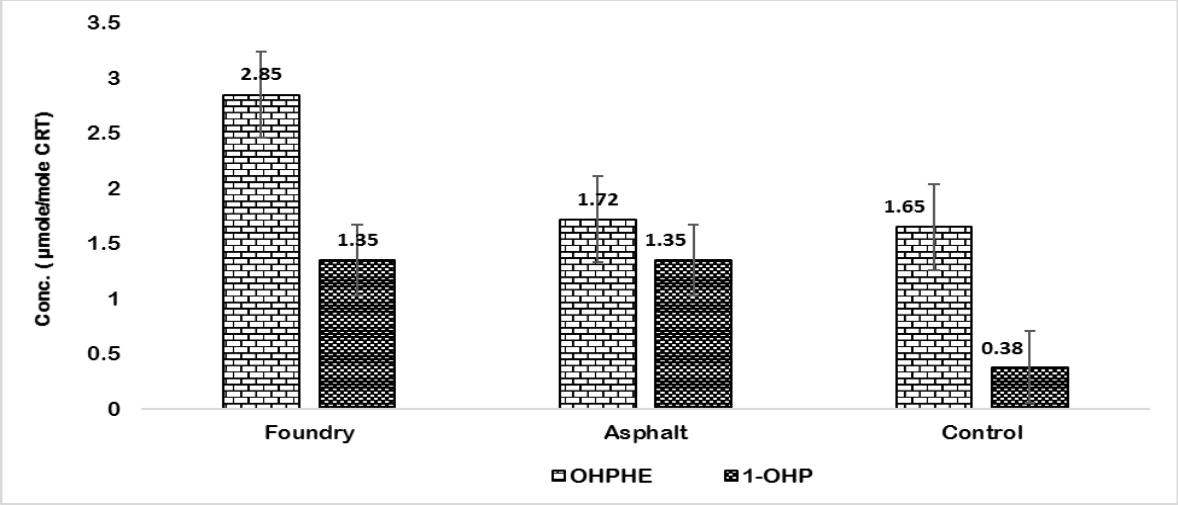

Figure-8.2: Urinary PAHs Biomarkers among the study population and control.

Though in the present study a small population of foundry and asphalt workers were covered to assess the PAHs exposure and internal dose of their metabolites, this study developed an integrated approach for conducting exposure and health-risk assessments associated with PAH exposure. During the present investigation, the two occupational groups (foundry and asphalt) were considered for dust and PAHs exposures and their probability of risk with each job category. It was found that 27% foundry PAHs sample exceeded the standard prescribed by NIOSH for workplace exposure than asphalt workers which only exceeded 8%. It was found that the PHE concentration in the foundry workplace higher (5.45 µg/m^3) than in the asphalt paving industry (1.94 µg/m^3) which influencing the urinary concentration of OHPHE among the foundry workers in elevated range than asphalt workers. The present study showed a higher risk of PAHs among foundry workers than asphalt workers. It may be due to their continuous exposure adjacent to the source and the foundry environment the PAHs emission retention time was much higher due to close environment where as in ambient environment the dilution of air reduces to less retention time. On the other hand, the asphalt workers were working in an open environment where atmospheric factors like temperature and wind velocity helps the chemical agent to distribute in a larger volume of environment and diluting the carcinogenic PAHs. The amount of an individual PAH found in both asphalt and

foundry samples and l as the relative potency of the PAH compounds determine its presumed contribution to the total cancer potency of the sample.

The goal of this type of research study was to prevent the development of occupational diseases among the foundry and asphalt workers which is neglected area in occupational medicine. The monitoring of exposures to dust and PAHs in the workplace may play an important role in detecting excessive exposures before the occurrence of significant biological disturbances and health impairment. Also, workplace airborne concentration of a particular and chemicals contaminant and the subsequent comparison with the appropriate exposure standard(s) was usually the primary technique in the evaluation basic risk associated with working environment. On the other hand, the biological monitoring play an important role to assess the internal dose exposure chemicals and the approach offers important advantages over monitoring the air of the workplace. It also plays greater role to the adverse health effects than environmental measurements. Therefore, it may offer a better estimate of the risk than can be determined from ambient monitoring. Biological monitoring accounts for uptake by all exposure routes. In the absence of biological monitoring data/or wherein the biological monitoring study cannot be conducted, the environmental monitoring plays an important role for evaluating and preventing excessive exposure to toxicants in the workplace.

Exposure assessments were conducted for different reasons and with different objectives. For example, the purpose of an assessment may be to get a rough, order-of-magnitude estimate of the maximal level of a chemical to which a population may be exposed. Alternatively, it may be conducted to get a detailed insight into the contribution of specific products to total exposure to a specific chemical.

There was a lack of information on human exposure to the single PAH compound in factory workers. Risk estimation showed that the contribution of the BaP & DahA compounds had a major contribution to the total risk. The average estimated lifetime lung cancer risk in the present study area was higher than the WHO and the USEPA recommended values. Despite uncertainties associated with the other co-pollutants

quantitative risk assessment calculations, the present study suggested that the inhalation cancer risk due to these PAH exposures were not negligible and should be taken into account for health protection in the future. In the present study, it was recognition that these industrial workers were exposed to dust and PAHs. Therefore, a combined experimental, clinical, and epidemiologic approach is the most effective way for evaluating the potential health risks.

Exposure to hazardous PAHs substances should be routinely evaluated. This may include collecting personal and area air samples. If workers were experiencing any work-related health problems, they should approach a doctor trained to recognize occupational diseases or industry itself having such day to day health checkups.

In the present study, it was found that the both categories of workers were occupationally exposed to dust and PAHs at their workplace and internal dose also indicate that the PAHs metabolites among the workers were higher than the control population. Hence to reduce the exposure through implementation of various control measures, the following procedures can be adopted

I. Reducing exposure

- Wherever possible, enclose the operations and use local exhaust ventilation at the site of chemical release. If local exhaust ventilation or enclosure is not used, respirators should be worn.
- Wearing of protective work clothing should be advised to workers.
- Washing thoroughly immediately after exposure to Asphalt and at the end of the work shift.
- Post hazard and warning information in the work area should be made available.
- Ongoing education and training on - effort, communicating all information on the health and safety hazards of pollutants potentially exposed workers.

II. Workplace controls and practices (Engineering controls)

Unless a less toxic chemical can be substituted for a hazardous substance, engineering controls are the most effective way of reducing exposure. The best protection is to enclose operations and/or provide local exhaust ventilation at the site of chemical release. Isolating operations can also reduce exposure. Using respirators or protective equipment is less effective than the controls mentioned above, but sometimes it is necessary. In evaluating the controls present in the workplace the flowing points to be kept in mind

- how hazardous the substance is
- how much of the substance is released into the workplace and
- whether harmful skin or eye contact could occur. Special controls should be in place for highly toxic chemicals or when significant skin, eye, or breathing exposures are possible.

III. Good hygiene practices in workplace

Good work practices can help to reduce hazardous exposures. The following work practices are recommended:

- Workers whose clothing has been contaminated should be asked to change into clean clothing promptly.

- Eye wash fountains should be provided in the immediate work area for emergency use.

- If there is the possibility of skin exposure, emergency shower facilities should be provided. Immediately wash or shower to remove the chemical.

- At the end of the work shift, washing of the contaminated area of the body that may have contacted PAHs, whether or not known skin contact has occurred. Eating, smoking and drinking where Asphalt is handled, processed, or stored should be avoided. Washing of hands carefully before eating, drinking, applying cosmetics, smoking, or using the toilet should be

taught to the workers.

IV. Personal protective equipment

Workplace controls are better than personal protective equipment. However, for some jobs (such as outside work, confined space entry, jobs done only once in a while, or jobs done while workplace controls are being installed), personal protective equipment may be appropriate. The management or owner of the plant/contractor should provide appropriate personal protective equipment for each hazard and train its employees on how and when to use protective equipment.

Though various reducing method is necessary to protect the workers in asphalt and foundry plants, the recommendations are to be considered as guideline and can applied to adverse situation.

- Avoid skin contact with PAHs. Wear protective gloves and clothing. Safety equipment suppliers/manufacturers can be contacted to provide recommendations on the most protective glove/clothing material for operation.
- All protective clothing (suits, gloves, footwear, headgear) should be worn clean, available each day, and put on before work.

The present study revealed that workers employed in the foundry and asphalt process units were at risk. The urinary levels of OHPHE and 1-OHP of PAHs metabolites concentrations reflected that the workers were exposed to PAHs and other mixture of contaminants at workplace. The workers employed in the melting, shaking out and blasting area are exposed to PAHs emission and these toxic emissions might absorbed in the system. Such cumulative absorption may be retained in different organs and may lead to toxicity.The Bayesian prediction model computed with the respirable dust in various sections indicated that the

workers were frequently inhaling the respirable dust at high concentrations. These respirable dusts might impregnated with PAHs, a known carcinogenic.

All these findings articulate to have immediate safety adaptation by personal protective equipment of using proper respirable mask or engineering control like local ventilations or cross ventilations in order to prevent from exposure to respirable dust to safe guard the health. There should also need of chemical analysis of respirable dust and exposure surveillance methods like protection of health of the individual employee, detection at an early stage for any adverse health effects, detection of hazards and assessment of risk. If such surveillance procedures are adopted in the industries concerned the adverse health effects can be prevented.

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Appendix-I: Dust exposure (mg/m^3) in the shop floors among the workers in study areas-I(foundry)

Sl No.	Working Location	Flow rate (LPM)	Total minutes	Volume of Air (litter)	FW(gm)	IW(gm)	Dust (µg/m^3)
1	Moulding	4.5	420	1890	0.06155	0.05858	1571
2	Moulding	4.5	390	1755	0.06160	0.05893	1521
3	Moulding	4.5	420	1890	0.06062	0.05834	1206
4	Moulding	1.67	422	705	0.06036	0.05915	1717
5	Moulding	1.67	301	503	0.05962	0.05890	1432
6	Moulding	4.5	480	2160	0.06172	0.05904	1241
7	Moulding	1.67	480	802	0.06058	0.0592	1722
8	Moulding	1.67	435	726	0.05904	0.05832	991
9	Moulding	1.67	435	726	0.06034	0.05998	496
10	Moulding	3.0	450	900	0.05866	0.05808	644
11	Moulding	3.0	440	880	0.05924	0.05855	784
12	Moulding	3.0	480	960	0.05882	0.05824	604
13	Moulding	3.0	480	960	0.05984	0.05913	740
14	Melting	4.5	480	2160	0.06010	0.05878	611
15	Melting	4.5	460	2070	0.06092	0.05868	1082
16	Melting	1.67	471	787	0.06006	0.05958	610
17	Melting	1.67	472	788	0.05992	0.05876	1472
18	Melting	4.5	480	2160	0.06168	0.05788	1759
19	Melting	4.5	480	2160	0.06028	0.05810	1009
20	Melting	1.67	480	802	0.05944	0.05756	2345
21	Melting	1.67	405	676	0.05982	0.05887	1405
22	Melting	4.5	480	2160	0.06177	0.05926	1162
23	Melting	4.5	440	1980	0.06049	0.05824	1136
24	Melting	4.5	480	2160	0.06290	0.05912	1750
25	Melting	1.67	480	802	0.05934	0.05852	1023
26	Melting	4.5	480	2160	0.06032	0.05850	843
27	Melting	1.67	463	773	0.06042	0.05844	2561
28	Melting	1.67	479	800	0.05962	0.05892	875
29	Melting	2	480	960	0.06024	0.05929	990
30	Shakout Area	1.67	483	807	0.05973	0.05837	1686
31	Shakout Area	4.5	480	2160	0.06424	0.05918	2343
32	Shakout Area	4.5	480	2160	0.06421	0.05882	2495
33	Shakout Area	1.67	480	802	0.06061	0.05904	1959
34	Shakout Area	1.67	481	803	0.06076	0.05834	3013
35	Shakout Area	4.5	470	2115	0.06412	0.05905	2397
36	Shakout Area	4.5	480	2160	0.06411	0.05835	2667
37	Shakout Area	4.5	480	2160	0.06483	0.05970	2375
38	Shakout Area	4.5	480	2160	0.06326	0.05910	1926
39	Shakout Area	2	480	960	0.05892	0.05814	813
40	Blasting Area	4.5	480	2160	0.06371	0.05958	1912
41	Blasting Area	1.67	480	802	0.05989	0.05868	1509
42	Blasting Area	1.67	480	802	0.06005	0.05928	961
43	Blasting Area	4.5	480	2160	0.06266	0.05920	1602
44	Blasting Area	1.67	480	802	0.06056	0.05856	2495
45	Blasting Area	1.67	480	802	0.05960	0.0591	624
46	Blasting Area	4.5	480	2160	0.06562	0.05892	3102
47	Blasting Area	4.5	480	2160	0.06238	0.0591	1519
48	Blasting Area	4.5	480	2160	0.06534	0.05898	2944
49	Blasting Area	1.67	480	802	0.06010	0.05882	1597
50	Blasting Area	1.67	485	810	0.06040	0.05893	1815
51	Blasting Area	3.0	480	960	0.05890	0.05858	333
52	Blasting Area	3.0	480	960	0.05912	0.05895	177
53	Blasting Area	3.0	480	960	0.05959	0.05850	1135
54	Blasting Area	2	480	960	0.06240	0.06020	2292
55	Heat Treatment	1.67	480	802	0.05988	0.05930	724
56	Heat Treatment	1.67	480	802	0.05898	0.05790	1347
57	Heat Treatment	3.0	480	960	0.05750	0.05740	104
58	Heat Treatment	3.0	480	960	0.05918	0.05906	125
59	Finishing	1.67	481	803	0.05936	0.05862	921
60	Finishing	1.67	483	807	0.05940	0.05852	1091
61	Finishing	4.5	480	2160	0.08228	0.05874	10898
62	Finishing	4.5	390	1755	0.05796	0.05495	1715
63	Finishing	1.67	481	803	0.06706	0.05844	10731
64	Finishing	1.67	466	778	0.05760	0.05508	3238
65	Finishing	4.5	480	2160	0.06066	0.05859	958
66	Finishing	4.5	480	2160	0.06058	0.059	731

Appendix-II: Dust exposure (mg/m^3) in the shop floors among the workers in study areas-II(foundry)

Sl No.	Working Location	Flow rate (LPM)	Total minutes	Volume of Air (litter)	FW(gm)	IW(gm)	Dust (µg/m^3)
1	Moulding	1.67	334	558	0.06034	0.05910	2223
2	Moulding	3.0	340	680	0.05935	0.05884	750
3	Moulding	3.0	340	680	0.05919	0.05839	1176
4	Moulding	3.0	340	680	0.06015	0.05916	1456
5	Moulding	4.5	440	1320	0.06420	0.05888	4030
6	Moulding	4.5	440	1320	0.06012	0.05802	1591
7	Moulding	4.5	440	1320	0.06022	0.05896	955
8	Moulding	1.67	401	670	0.06164	0.05918	3673
9	Moulding	1.67	401	670	0.05908	0.05858	747
10	Moulding	4.5	400	1200	0.05962	0.05860	850
11	Moulding	4.5	400	1200	0.06095	0.05877	1817
12	Moulding	4.5	400	1200	0.05955	0.05812	1192
13	Melting	1.67	318	531	0.05946	0.05884	1167
14	Melting	1.67	312	521	0.06142	0.05980	3109
15	Melting	4.5	220	660	0.06030	0.05926	1576
16	Melting	4.5	210	630	0.06061	0.05905	2476
17	Melting	4.5	210	630	0.06060	0.05946	1810
18	Melting	1.67	455	760	0.06026	0.05928	1290
19	Melting	1.67	452	755	0.05884	0.05810	980
20	Melting	4.5	450	1350	0.06047	0.05885	1200
21	Melting	4.5	450	1350	0.06004	0.05835	1252
22	Blasting	1.67	446	745	0.06192	0.06036	2094
23	Finishing	1.67	358	598	0.06090	0.05948	2375
24	Finishing	1.67	357	596	0.06146	0.05938	3489
25	Finishing	4.5	340	1020	0.06148	0.05908	2353
26	Finishing	4.5	340	1020	0.06088	0.05972	1137
27	Finishing	4.5	340	1020	0.06262	0.05933	3225

Appendix-III: Foundry PAHs Data Sheets

Sl No.	NAP	ACY	ACP	FLU	PHEN	ANTH	FLT	PYR	BaA	CHR	BbF	BkF	BaP	DBahA	BghiP	IND	Total PAHs
1	6.88	0.17	0.22	1.07	0.07	0.04	0.02	0.03	0.00	0.01	0.00	0.05	0.34	0.03	0.15	0.02	9.09
2	74.41	90.69	180.24	22.65	22.93	3.59	8.29	16.40	7.63	7.35	2.89	0.98	1.32	17.56	21.25	0.24	478.43
3	29.45	47.37	123.03	9.14	4.09	3.42	10.83	23.01	10.45	12.58	4.36	0.65	1.92	14.42	50.36	0.44	345.51
4	7.36	9.78	26.62	2.75	2.91	0.56	3.50	8.74	4.04	5.65	1.97	0.00	0.98	16.30	27.02	0.28	118.43
5	65.02	29.00	64.01	0.00	0.00	0.00	3.90	0.00	0.00	0.00	0.00	0.00	0.06	86.86	4.39	0.00	253.23
6	24.11	9.29	20.68	3.01	0.04	0.01	0.28	2.08	4.23	38.95	0.10	0.00	0.37	45.43	0.26	7.91	156.76
7	25.24	6.84	17.66	0.07	0.03	0.21	2.73	3.05	19.31	6.12	0.00	0.06	18.49	2.22	7.35	1.66	111.05
8	2.22	1.99	2.77	0.15	30.14	0.49	0.01	0.96	0.16	0.25	0.54	0.01	15.39	0.43	3.16	0.09	58.76
9	32.12	13.39	27.60	0.72	23.12	3.68	11.54	3.30	0.12	0.01	0.00	0.01	34.47	1.22	15.04	0.12	166.44
10	9.85	6.47	0.75	1.68	3.26	1.94	1.40	0.11	1.61	1.38	6.22	0.02	37.52	23.35	7.30	1.58	104.43
11	1.66	0.44	2.07	0.00	0.12	0.41	1.35	0.01	0.03	0.04	0.00	0.28	0.39	0.00	0.00	0.00	6.80
12	0.31	0.33	1.47	9.71	0.09	0.01	0.25	1.00	0.01	0.12	0.49	1.56	6.14	0.37	10.95	0.35	33.16
13	0.99	1.16	0.00	0.00	0.04	0.02	0.23	0.14	0.00	0.01	0.07	0.23	0.74	0.00	4.65	0.00	8.28
14	0.20	0.18	0.00	1.21	0.11	0.09	0.63	0.73	0.10	0.06	0.03	0.14	2.08	0.43	7.26	0.00	13.26
15	1.80	0.37	1.66	0.00	0.10	0.06	1.52	1.16	0.03	0.05	0.07	0.84	2.36	0.00	0.00	0.00	10.01
16	3.51	0.45	12.15	0.00	0.00	0.07	2.80	1.16	0.56	0.64	1.25	35.37	1.65	0.00	0.53	2.87	63.00
17	2.01	2.17	0.61	0.32	0.27	0.17	0.06	1.00	0.47	0.33	0.26	0.39	2.01	0.27	1.46	0.50	12.32
18	1.16	1.19	3.70	0.00	0.00	0.11	0.00	0.69	0.35	0.66	0.23	0.00	0.15	0.00	1.18	1.41	10.82
19	4.23	4.51	10.03	1.86	0.23	0.29	0.21	1.39	0.67	1.16	0.41	0.11	1.85	3.96	2.32	0.83	34.05
20	41.32	31.44	62.17	5.14	2.27	0.96	1.31	2.67	1.48	1.17	0.60	0.03	0.19	5.22	1.22	0.00	157.20
21	10.60	10.01	23.49	0.85	1.72	0.46	0.78	1.63	0.94	0.77	0.36	0.02	0.09	4.49	0.82	0.08	57.09
22	3.11	3.04	9.00	0.89	0.73	0.21	0.34	0.90	0.58	0.51	0.23	0.03	0.10	1.63	0.52	0.01	21.85
23	9.58	4.37	0.00	165.46	0.00	1.84	3.98	5.90	0.66	0.00	0.00	0.00	2.36	0.00	1.65	3.53	199.33
24	1.48	1.68	4.08	0.00	1.30	0.12	0.35	0.90	0.54	0.60	0.26	0.71	13.02	3.63	1.13	0.38	30.19
25	0.35	0.56	1.24	0.00	0.00	0.03	0.06	0.19	0.13	0.13	0.06	0.03	15.38	0.32	0.04	2.05	20.56
26	1.49	0.28	0.16	0.27	0.14	0.01	0.01	0.03	0.00	0.00	0.00	0.21	0.17	0.00	0.00	0.00	2.78
27	3.10	0.16	0.06	0.00	0.01	0.02	0.00	0.03	0.00	0.00	0.00	0.03	0.26	0.00	0.00	0.00	3.67
28	0.87	0.34	0.15	4.41	0.13	0.01	0.01	0.01	0.00	0.00	0.00	0.02	0.22	0.00	0.00	0.00	6.17
29	0.61	0.71	1.91	0.00	0.00	0.06	0.07	0.35	0.22	0.31	0.13	0.00	19.79	0.12	0.56	0.25	25.07
30	3.47	1.72	2.73	0.00	5.00	0.17	0.09	0.80	0.50	0.58	0.23	0.00	19.92	0.23	1.20	0.00	36.63

Appendix-III: Foundry PAHs Data Sheets

Sl No.	NAP	ACY	ACP	FLU	PHEN	ANTH	FLT	PYR	BaA	CHR	BbF	BkF	BaP	DBahA	BghiP	IND	Total PAHs
31	4.50	1.51	6.80	0.00	4.37	0.34	2.00	3.32	0.39	0.00	0.00	0.01	7.84	6.69	1.27	0.00	39.03
32	1.14	2.00	5.87	0.00	0.00	0.16	0.12	1.34	0.74	1.26	0.45	0.00	28.11	0.15	4.82	0.00	46.18
33	6.75	2.10	0.00	0.00	0.00	0.03	1.12	1.09	0.00	0.47	0.30	0.00	15.33	8.08	2.30	0.00	37.56
34	6.55	2.04	4.90	0.00	0.00	0.01	1.18	1.13	1.20	0.22	0.00	0.00	12.94	0.00	3.92	0.00	34.10
35	1.31	1.75	3.76	0.00	4.97	0.09	0.33	0.74	0.38	0.50	0.19	0.00	19.48	3.04	1.03	0.00	37.55
36	2.25	0.85	2.09	0.76	2.50	0.26	0.47	0.45	0.36	0.08	0.00	0.02	23.40	0.06	0.51	1.27	35.32
37	3.34	1.10	2.07	0.00	116.74	0.13	0.79	1.17	0.14	0.00	0.00	0.00	23.00	0.08	0.66	8.21	157.43
38	6.12	2.55	2.51	0.00	1.81	0.05	0.70	0.54	0.26	0.05	0.07	0.60	1.96	0.50	1.63	0.91	20.25
39	0.93	0.84	1.84	21.97	43.32	0.00	0.55	0.57	0.35	0.06	0.00	0.91	9.83	0.10	4.14	0.00	85.41
40	3.84	1.12	0.13	0.00	0.00	0.19	0.39	0.81	0.10	0.00	0.00	0.45	22.16	0.44	11.24	0.00	40.87
41	1.94	2.00	4.70	53.75	9.31	0.23	0.50	1.08	0.12	0.00	0.00	1.30	30.69	0.00	8.15	0.56	114.33
42	1.35	0.62	0.00	0.00	0.00	0.26	0.32	0.88	0.10	0.00	0.00	0.76	26.53	0.00	10.81	0.00	41.64
43	0.00	0.89	2.72	2.11	9.36	0.10	0.54	1.17	0.71	0.00	1.84	1.69	45.70	0.03	9.97	2.00	78.84
44	0.64	1.29	3.56	0.00	0.00	0.11	0.12	0.77	0.33	2.15	1.74	0.77	20.19	0.01	3.27	3.94	38.89
45	1.02	0.94	4.25	0.00	0.00	0.12	0.01	0.43	0.01	0.32	0.00	1.57	30.98	0.00	21.31	0.00	60.97
46	0.31	0.59	1.69	0.00	0.00	0.01	0.26	0.60	0.67	0.63	0.00	1.06	28.01	0.02	15.63	0.00	49.48
47	0.90	0.70	1.64	0.00	0.00	0.05	0.08	0.50	0.23	1.63	1.34	1.44	36.12	0.03	8.39	3.81	56.85
48	0.72	0.00	0.00	0.00	0.00	0.00	0.08	0.12	0.14	0.16	0.00	0.99	26.19	0.00	15.35	0.00	43.76
49	1.89	1.28	14.07	0.00	0.94	0.11	0.48	0.19	0.01	0.05	0.01	0.04	1.05	0.35	1.32	0.00	21.79
50	4.81	0.75	2.14	0.00	0.65	0.09	2.68	0.48	0.06	0.11	0.49	2.54	6.95	1.04	9.41	0.00	32.18
51	2.67	0.79	0.58	0.00	0.00	0.16	0.81	0.87	0.15	0.07	0.00	0.64	1.26	0.09	6.04	0.00	14.12
52	6.09	3.27	6.04	0.00	24.63	0.48	2.27	4.80	0.65	0.00	0.00	0.60	1.41	0.26	1.48	0.71	52.68
53	1.60	1.32	0.00	0.00	4.63	0.28	1.11	3.02	0.31	0.00	0.00	0.61	1.55	0.61	8.90	0.00	23.93
54	3.13	1.30	5.77	0.00	2.13	0.06	1.28	1.31	1.65	0.27	0.00	0.82	1.75	0.03	2.66	8.80	30.95
55	0.12	0.36	0.83	8.48	0.26	0.04	0.54	0.53	0.59	0.09	0.00	0.88	1.15	0.05	4.75	1.50	20.18
56	46.36	16.57	25.61	0.00	0.93	1.01	4.33	3.82	12.56	5.13	0.00	0.21	1.08	1.33	13.02	4.04	136.01
57	53.88	34.99	98.84	9.55	1.04	2.43	6.91	22.81	11.33	0.27	0.00	0.10	14.31	0.00	19.16	0.00	275.63
58	17.21	21.03	41.12	4.38	0.10	0.49	6.15	6.46	42.60	15.27	0.00	0.11	14.23	1.51	3.04	1.45	175.15
59	5.86	6.16	20.71	3.06	0.05	0.32	4.34	5.27	31.85	10.94	0.00	0.20	29.50	0.07	17.27	24.66	160.27
60	11.50	4.46	5.02	0.02	0.22	0.36	1.69	2.19	0.09	1.62	0.10	0.09	20.70	0.55	9.00	8.33	65.93

Appendix-IV: Asphalt PAHs Data Sheets

SL No.-	Location	NAP	ACY	ACP	FLU	PHEN	ANTH	FLT	PYR	BaA	CHR	BbF	BkF	BaP	DBahA	BghiP	IND	TOTAL PAHs
1	Plant	0.39	0.00	1.20	0.00	0.00	0.02	0.05	0.14	0.07	0.08	0.00	0.00	1.67	0.00	0.00	0.00	3.61
2	Plant	0.00	0.33	0.72	0.00	1.70	0.00	0.03	0.07	0.04	0.03	0.00	0.00	0.00	0.00	0.00	0.00	2.91
3	Plant	0.44	0.33	0.00	4.24	0.00	0.03	0.04	0.05	0.03	0.00	0.01	0.20	0.32	0.00	0.00	0.00	5.68
4	Plant	0.00	0.00	0.36	0.00	0.00	0.01	0.00	0.13	0.00	0.01	0.00	0.00	1.29	0.00	0.00	0.00	1.80
5	Plant	2.13	0.00	0.00	0.00	3.86	0.03	0.06	0.17	0.10	0.11	0.04	0.00	0.98	4.43	0.00	0.00	11.90
6	Plant	0.46	0.76	1.27	9.62	0.00	0.01	0.04	0.08	0.04	0.04	0.04	0.47	0.48	0.00	0.00	0.00	13.31
7	Plant	1.07	0.92	1.76	0.00	4.19	0.00	0.08	0.15	0.07	0.07	0.03	0.26	0.00	0.42	3.19	0.00	12.23
8	Plant	0.10	0.33	0.48	0.00	0.00	0.00	0.01	0.02	0.01	0.01	0.08	1.17	0.21	0.00	0.00	0.00	2.43
9	Paving	0.27	0.33	0.00	0.49	2.51	0.04	0.06	0.06	0.00	0.00	0.00	0.36	8.94	2.01	4.14	0.00	19.20
10	Paving	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.03	0.02	0.00	0.00	0.00	9.70	3.56	0.00	0.00	13.34
11	Paving	0.67	0.58	1.27	0.00	0.00	0.03	0.06	0.13	0.07	0.07	0.03	2.53	0.00	0.65	4.16	0.00	10.25
12	Paving	0.34	0.35	0.80	0.00	0.00	0.01	0.03	0.07	0.04	0.04	0.01	0.93	0.00	0.05	9.29	0.00	11.96
13	Paving	0.19	0.14	0.27	0.00	0.00	0.00	0.01	0.02	0.01	0.02	0.01	0.00	0.00	0.29	0.67	0.00	1.64
14	Paving	0.00	0.00	0.52	0.00	0.00	0.00	0.01	0.04	0.02	0.02	0.01	1.13	0.71	0.00	0.00	0.00	2.45
15	Paving	0.00	0.86	1.94	0.00	0.00	0.04	0.08	0.20	0.11	0.11	0.05	0.35	0.24	0.45	2.94	0.00	7.35
16	Plant	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.02	0.01	0.01	0.01	0.40	0.09	0.29	35.17	0.00	36.01
17	Plant	1.15	0.42	0.38	7.27	0.00	0.00	0.06	0.07	0.00	0.00	0.02	0.00	0.15	0.16	0.78	0.00	10.46
18	Plant	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.10	0.08	0.15	0.06	0.14	2.69	2.97	0.00	0.00	6.21
19	Plant	3.57	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3.57
20	Plant	6.27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.03	0.00	0.00	0.00	0.00	0.00	0.00	6.30
21	Plant	0.00	2.76	0.00	0.00	0.00	0.07	0.00	0.00	0.00	0.04	0.01	0.01	0.01	0.00	0.00	0.00	2.90
22	Plant	0.00	0.00	0.00	1.11	0.06	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.02	0.92	0.00	0.00	2.12
23	plant	2.16	0.22	1.13	BDL	1.78	0.04	0.05	0.53	0.06	0.20	0.44	2.13	0.11	BDL	3.24	0.78	12.86
24	plant	0.66	0.07	0.56	BDL	0.92	0.04	0.01	0.22	0.09	0.05	2.36	2.02	0.04	BDL	0.94	1.03	9.00
25	plant	0.85	0.12	0.69	BDL	1.08	0.05	0.02	0.27	0.09	0.07	2.46	0.19	0.03	BDL	2.96	BDL	8.87
26	plant	1.07	0.05	0.86	0.16	1.45	0.03	BDL	BDL	0.20	0.18	5.19	3.06	0.07	BDL	2.12	1.60	16.06
27	plant	0.56	0.04	0.27	0.04	0.81	0.03	BDL	BDL	0.12	0.03	2.67	1.67	0.03	BDL	1.38	BDL	7.65
28	plant	1.11	0.10	0.71	0.05	1.40	0.06	0.03	0.20	0.03	0.12	5.30	0.17	0.05	0.00	2.07	1.50	12.89
29	plant	0.55	0.11	0.17	0.02	0.92	0.11	0.01	0.02	0.11	0.07	1.41	2.01	0.03	0.00	0.10	0.14	5.75
30	plant	0.49	0.05	0.56	BDL	0.56	0.01	BDL	0.02	0.01	0.07	1.42	1.22	0.03	BDL	BDL	BDL	4.45

Appendix-IV: Asphalt PAHs Data Sheets

SL No.-	Location	NAP	ACY	ACP	FLU	PHEN	ANTH	FLT	PYR	BaA	CHR	BbF	BkF	BaP	DBahA	BghiP	IND	TOTAL PAHs
31	plant	0.32	0.00	0.44	0.03	0.39	0.04	BDL	0.01	0.04	0.02	0.44	0.99	0.02	0.51	0.23	0.08	3.56
32	plant	1.52	0.75	0.36	0.07	1.21	0.05	0.05	0.01	0.07	0.15	8.96	2.62	0.06	5.35	8.08	1.22	30.52
33	plant	0.66	0.10	0.63	0.04	0.83	BDL	BDL	0.02	0.13	0.03	1.62	1.47	0.03	6.73	2.98	0.36	15.61
34	plant	1.32	0.06	0.94	0.00	1.45	0.04	BDL	BDL	0.18	0.05	3.23	2.88	0.09	0.90	2.12	0.50	13.75
35	plant	0.77	BDL	0.61	0.04	1.00	0.09	BDL	0.02	BDL	BDL	1.00	2.76	0.06	BDL	BDL	0.26	6.62
36	plant	1.11	0.07	0.99	BDL	1.79	0.04	BDL	BDL	BDL	0.08	5.71	3.87	0.11	4.81	2.24	2.50	23.33
37	plant	0.80	0.06	BDL	0.46	1.37	0.04	BDL	0.03	0.20	0.14	2.61	3.12	0.07	7.15	1.86	1.64	19.57
38	plant	0.89	0.07	1.06	0.08	0.91	BDL	BDL	0.02	0.15	0.06	4.10	1.54	0.03	6.83	1.59	BDL	17.32
39	plant	1.73	0.09	1.68	BDL	2.32	0.04	BDL	0.07	0.02	0.25	5.39	4.93	0.13	BDL	8.09	0.73	25.45
40	plant	1.00	0.04	1.21	0.08	1.81	0.07	0.01	0.05	0.24	0.05	4.94	3.83	0.08	BDL	1.82	2.15	17.39
41	plant	1.44	4.50	3.31	0.13	0.49	0.15	0.05	0.11	0.11	BDL	1.04	BDL	BDL	BDL	1.23	0.17	12.74
42	plant	BDL	0.26	0.40	0.09	1.37	0.04	0.03	0.35	0.03	0.12	3.00	2.25	0.06	4.17	BDL	0.61	12.79
43	plant	0.22	0.21	0.08	0.28	1.22	0.04	0.05	0.20	0.05	BDL	0.41	0.02	0.04	BDL	0.56	1.11	4.47
44	plant	0.30	3.35	0.27	0.44	1.56	0.06	BDL	0.04	0.24	0.04	6.03	1.21	0.04	14.30	BDL	1.11	28.98
45	Plant	0.00	0.00	0.00	0.00	0.00	0.34	0.29	0.08	0.06	0.08	0.03	25.96	2.99	0.00	0.00	0.00	29.82
46	Paving	0.50	0.13	1.36	21.97	21.93	1.28	0.55	0.59	0.08	0.07	0.02	48.63	6.25	2.29	79.06	0.00	184.71
47	Paving	12.42	0.15	1.05	0.00	0.00	0.02	0.31	0.09	0.21	0.00	0.04	17.01	11.79	3.79	13.26	0.00	60.14
48	Paving	1.90	0.23	25.97	0.00	0.00	0.06	0.16	0.45	0.22	0.03	0.08	0.00	18.91	2.70	26.45	0.00	77.15
49	Paving	0.00	0.25	0.22	1.26	0.00	0.00	1.48	0.04	0.06	0.01	0.03	3.27	9.40	1.21	23.05	0.00	40.30
50	Paving	1.71	7.96	0.00	57.71	0.00	0.06	0.11	0.28	0.20	0.02	0.03	0.00	20.88	1.85	71.43	0.00	162.24
51	Paving	0.36	0.59	3.52	29.43	0.00	0.02	5.63	0.27	0.10	0.02	0.11	6.92	25.10	2.17	46.33	0.00	120.55
52	Paving	1.06	6.27	0.00	0.00	8.78	0.59	0.35	0.71	0.04	0.04	0.07	6.41	11.20	1.78	43.96	0.00	81.26
53	Paving	1.98	0.00	35.74	0.00	0.00	0.07	0.57	0.76	0.03	0.17	0.03	0.00	5.48	0.10	20.01	0.00	64.94
54	Paving	0.00	0.00	2.47	0.00	0.00	0.00	6.56	0.29	0.20	0.00	0.00	0.00	29.68	5.33	0.00	0.00	44.53
55	Paving	1.09	0.46	0.91	29.09	0.00	1.57	0.85	0.00	0.50	0.01	0.41	6.58	3.98	0.00	76.18	2.67	124.30
56	Paving	0.89	0.00	0.00	0.00	0.00	0.50	3.12	0.51	0.00	0.01	0.00	0.00	0.49	0.00	38.81	0.00	44.33
57	Paving	2.59	0.34	0.47	9.22	14.57	0.00	0.07	0.01	0.00	0.00	0.00	0.00	2.37	0.00	0.00	0.00	29.64
58	Paving	0.96	0.27	1.94	0.00	3.02	0.00	5.41	0.22	0.13	0.01	0.10	6.70	24.98	0.00	6.85	1.76	52.35
59	Paving	2.12	1.65	1.02	13.93	18.04	1.91	0.87	0.49	0.10	0.16	0.15	0.00	59.06	9.81	48.47	6.29	164.06
60	Paving	2.27	1.51	0.52	4.75	11.01	1.02	0.59	0.21	0.11	0.10	0.00	0.00	18.32	7.26	48.03	2.11	97.80

ROHCS/142/ 626A

Date: 26.9.12

Communication of Decision of the Institutional Ethics Committee (IEC)

IEC/IRB No. ECR/1086/ROHC/Inst/KA/2013

Protocol title: **IDENTIFICATION OF OCCUPATIONAL HEALTH RISK POLLUTANTS AND THEIR METABOLITES AMONG FOUNDRY WORKERS**
Principal Investigator **Mr.SOMNATH SEN**
Name & Address of Institution: **INDUSTRIAL HYGIENE & TOXICOLOGY DIVISION** **REGIONAL OCCUPATIONAL HEALTH CENTRE(SOUTHERN), ICMR,** **KANNAMANGALA PO, DEVANAHALLI PO, BANGALORE – 562 110, INDIA**
☐ New review ☐ Revised review ☐ Expected review
Date of review (D/M/Y) Date of previous review, if revised application:
Decision of the IEC/IRB: ☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestion/Reasons/Remarks: **Recommended with appreciation**
Recommended for a period of: **2 YEARS**

Please Note:

- Inform IEC/IRB immediately in case of any Adverse events and Serious adverse events.
- Inform IEC/IRB in case of any change of study procedure, site and investigator
- This permission is only for period mentioned above. Annual report to the submitted to IEC/IRB.
- Members of IEC/IRB have right to monitor the trial with prior intimation.

26/9/12

Signature of Member Secretary
IEC/IRB

OFFICER - IN - CHARGE
REGIONAL OCCUPATIONAL HEALTH CENTRE (SOUTHERN)
(ROHCS - NIOH - ICMR)
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