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Workplace Exposure to Polycyclic Aromatic Hydrocarbons (PAHs): A Review and Discussion of Regulatory Imperatives for Nigeria

^{1,3,4,5}Francis O. Okonkwo, ²Chukwunonso E.C.C. Ejike, ³Ursula Berger,
¹Constanze Schmalting, ⁴Rudolf Schierl and ⁴Katja Radon

¹Center for International Health, Ludwig-Maximilians University, Leopoldstraße 7, 80802, Munich, Germany

²Department of Biochemistry, Michael Okpara University of Agriculture, Umudike, Umuahia, Abia State, Nigeria

³Institute of Medical Informatics, Biometry and Epidemiology, Ludwig-Maximilians University Munich, Germany

⁴Institute and Outpatient Clinic for Occupational, Social and Environmental Medicine, Ludwig-Maximilians University, Munich, Germany

⁵Environmental Toxicology and Occupational Health Unit, Department of Biochemistry, Plateau State University, Bokokos, Plateau State, Nigeria

Corresponding Author: Francis O. Okonkwo, Center for International Health, Ludwig-Maximilians University, Leopoldstraße 7, 80802, Munich, Germany

ABSTRACT









Polycyclic Aromatic Hydrocarbons (PAHs) are a class of complex organic chemicals, possessing a fused ring structure with at least 2 benzene rings. They are environmental pollutants that occur naturally in coal and crude oil and in emissions from forest fires and volcanoes. The PAHs also enter the environment via, incomplete combustion (pyrolysis) of organic materials such as coal, garbage, waste tyres, tobacco, etc. Several epidemiologic and laboratory studies have shown that exposure to PAH results in adverse health outcomes ranging from irritation of the exposed surface to cancers. The world is faced with the problem of adequate disposal of used tyres which (among other uses) have unfortunately been used in abattoirs in Nigeria to de-fur kanda, a popular cow skin delicacy, hence exposing the butchers to PAHs. This article reviews the chemistry and biochemistry of PAHs and discusses the need for appropriate regulation of exposure to PAHs especially in Nigerian workplaces.

Key words: Polycyclic aromatic hydrocarbons, occupational exposure, Nigeria

INTRODUCTION

The United States Environmental Protection Agency estimated that 2-3 billion scrap tyres are in landfills and stockpiles across the United States and approximately one scrap tyre per person is generated every year (Reisman, 1997). Globally, more than 240 million vehicle tyres are burnt for various reasons, such as: (1) As a source of cheap fuels in cement kilns, coking plants and allied industries, (2) As a form of waste disposal in municipal refuse dumps and incinerators, (though illegal in most places), (3) As a way of expressing displeasure with government's political and other socio-economic policies during demonstrations and stand-offs; just to mention but a few. In Nigeria,

Table 1: Some representative PAHs, their chemical structure and some physicochemical properties

Compound (IUPAC name)	Mol. wt.	Vapour pressure at 25°C (kPa)	Boiling point °C	Carcinogenicity	Structure
Phenanthrene	178	$2,3 \times 10^{-5}$	339-340		
Anthracene	178	36×10^{-6}	340	+	
Fluoranthene	202	$6,5 \times 10^{-7}$	375-393		
Pyrene	202	$3,1 \times 10^{-6}$	360-404		
Benzo(a)anthracene	228	$1,5 \times 10^{-8}$	435	+	
Chrysene	228	$5,7 \times 10^{-10}$	441-448	+	
Benzo(a)pyrene	252	$7,3 \times 10^{-10}$	493-496	+	
Benzo(g, h, i)perylene	278	$1,3 \times 10^{-11}$	525	+	

OSPAR Commission, 2003

all of the above find application, yet there is, coupled to the above, a nation-wide practice of incinerating tyres in the processing of the skins of slaughtered domestic animals such as goats, sheep and cows.

The skins of animals known locally as pomo or kanda are major culinary delicacies in Nigeria. Other parts like the head, legs and tails, etc., of these animals are notable delicacies and command high prices in the market. For possible economic reasons, butchers find it expedient to use tyres as fuel. In using burning tyres to process edible animal skin, the butchers and the consumers may be unduly exposed to PAHs above safe limits. The International Agency for Research on Cancer reported that PAHs are carcinogenic to humans (IARC, 1983). The use of tyres as fuel for meat processing in Nigeria is therefore worrisome especially as the prevalence of cancers in Nigeria is rising (Ifere *et al.*, 2012). The contamination of the ecosystem with PAHs could also result in ground water pollution especially with the increase in the number of private shallow wells and bore holes in Nigeria. These could pose public health problems and consequently lead to severe economic burdens on both the country's struggling health systems and the populace, majority of whom are extremely poor by World Bank standards.

CLASSIFICATION, CHEMISTRY AND SOURCES OF PAHs

Polycyclic Aromatic Hydrocarbons (PAHs) also referred to as Polycyclic Organic Matter (POM), Polynuclear Aromatics (PNAs) are a class of complex organic chemicals possessing a fused ring structure with at least 2 benzene rings (ATSDR, 1995). Typical examples of PAHs are naphthalene, 2-methylnaphthalene, 1-methylnaphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(1,2,3-cd)pyrene, dibenzo(a,h)anthracene and benzo(g,h,i)perylene (Table 1) (ATSDR, 1995). Poly Aromatic

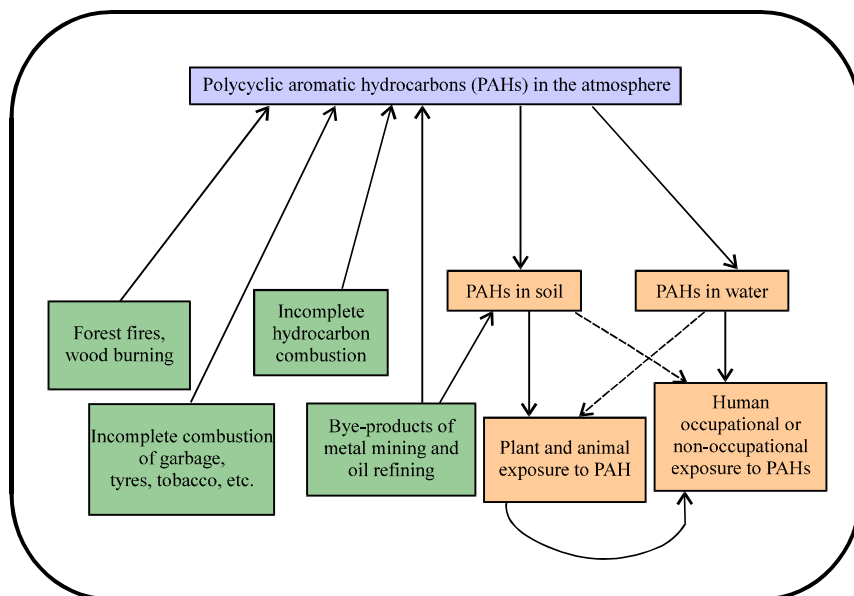


Fig. 1: Sources of polycyclic aromatic hydrocarbons in the environment

Hydrocarbons (PAHs) may also contain additional fused rings that are not the six-sided ring of benzene (Ravindra *et al.*, 2008). They are environmental pollutants found naturally in coal and crude oil and emissions from forest fires and volcanoes. Apart from natural sources, most PAHs entering the environment arise from incomplete combustion (pyrolysis) of organic materials such as coal, garbage, waste tyres and tobacco, etc. (Fig. 1). Others are by-products of industrial processes such as aluminium, iron and steel production in foundries, mining and oil refining (WHO, 2000).

At ambient temperatures, pure PAHs generally exist as colourless, white or pale yellow-green solids. Normally, they have high melting and boiling points, low vapour pressure and very low water solubility. Consequent upon these, they are highly lipophilic and chemically inert. In the atmosphere, they are present in both vapour and particle phases. Most PAHs do not dissolve readily in water although they are commonly found in minute quantities in industrial and municipal effluents. Some PAHs readily evaporate into the air and persist in the atmosphere for months to years often transported through long distances before re-entering the earth through particle settling or during rainfalls (Shen *et al.*, 2011).

Predominantly, two- or three-ringed PAHs (naphthalene, acenaphthene, anthracene, fluorene, phenanthrene) are present in air in the vapour phase; four-ringed PAHs (fluoranthene, pyrene, chrysene) exist both in the vapour and in the particulate phase and PAHs with five or more rings (benzo(a)pyrene, benzo[g, h, i]perylene) are found usually in the particle phase. Upon release into the atmosphere, the fate of gaseous PAH mixtures appear to be determined by their molecular weight, such that PAHs with more than four rings tend to adsorb to particulate matter, while those with less than four rings tend to remain gaseous until removed via., precipitation (Skupinska *et al.*, 2004).

Poly Aromatic Hydrocarbons (PAHs) typically undergo three types of chemical reaction; electrophilic substitution, oxidation and reduction. During their transport and accumulation in the atmosphere, PAHs may undergo chemical and photochemical reactions probably resulting in

compounds that are more toxic than the parent compounds. Most PAHs are likely to react with air and compounds borne in air (e.g., O₃, NO_x and SO₂) to form PAH derivatives such as nitro-PAHs and oxygenated derivatives (Walgraeve *et al.*, 2010; Shen *et al.*, 2011).

The burning of tyres, say in kanda processing releases several compounds and particles [such as Carbon Monoxide (CO), Sulfur Oxides (SO₂), Oxides of Nitrogen (NO_x), Volatile Organic Compounds (VOCs) and other Hazardous Air Pollutants (HAPs), such as Polychlorinated Biphenyls (PCBs), dioxins, furans, hydrogen chloride, benzene and most especially Polycyclic Aromatic Hydrocarbons (PAHs)] into the environment. Metals such as arsenic, cadmium, nickel, zinc, mercury, chromium and vanadium are also released in the process (Carrasco *et al.*, 2002; Ferrao *et al.*, 2008). It is thought that the effects attributable to PAHs may in fact be accentuated by its co-contamination with other chemicals as found in the cocktail of chemicals so-released into the environment (Reisman, 1997).

ROUTES OF EXPOSURE TO PAHs

Exposure is assessed qualitatively or quantitatively. It describes the intensity, frequency and duration of contact and often quantifies the rate at which the chemical or agent crosses the protective layer or is taken-up, the route of the chemical or agent across the protective layer (dermal, oral, or respiratory), the dose (actual quantity of the chemical or agent crossing the layer) and the amount absorbed (USEPA, 1992a; Leung and Paustenbach, 1994). Exposure is a difficult term to scientifically define. There is however some consensus among scientists that exposure entails contact of the human outer protective covering (largely dermal, oral or respiratory) with a chemical or an agent (USEPA, 1989, 1992b; Allaby, 1989).

For most people, PAHs exposure occurs quite regularly through the air, water, soil and food (Fig. 1). Exposure could be in both occupational and non-occupational settings and may involve more than one route of exposure simultaneously. The use of certain cosmetics is yet another hidden means of exposure, especially as there have been reports of high levels of PAHs in say, tattoo ink. Some pharmaceuticals especially dermal preparations, plastics, insect repellents and some household goods contain PAHs. Simultaneous exposure through multiple routes such as concomitant dermal and inhalation exposures usually results in higher doses of the chemical or agent being taken-up (ATDSR, 2009; Le Du-Lacoste *et al.*, 2013).

METABOLISM OF PAHs IN HUMANS

Upon exposure to PAHs just like other xenobiotics, the human body metabolises the PAH into more polar and soluble by-products that are more readily excreted through the kidneys as urine or as exudates from the skin or in the faeces (Fig. 2). In some cases, the metabolism is aimed at enabling the reabsorption of the metabolite through the biliary system. The PAHs metabolism is usually catalysed by Cytochrome P-450 monooxygenase complex. The enzyme complex carries out the oxidation, hydrolysis and reduction reactions that detoxify the xenobiotic (Franco *et al.*, 2008). The oxidation of PAHs by the cytochrome P-450 family of enzymes and the subsequent hydroxylation by epoxide hydrolase yields electrophilic and hydrophilic epoxy diols.

The water soluble products of the reactions usually complexes of glutathione, sulfates or glucuronic acid are excreted from the body as hydroxylated metabolites or conjugates. These conjugation reactions apart from making the metabolites easy to excrete also protects cells from potential damage from the water soluble adducts (Skupinska *et al.*, 2004). The hydroxylated metabolites are usually excreted via., urine and/or faeces while the conjugates are reabsorbed in

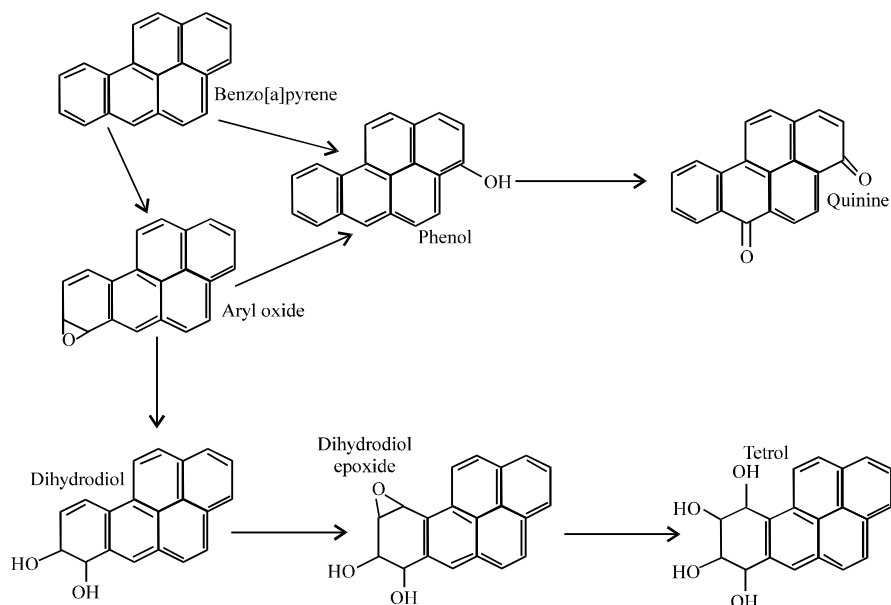


Fig. 2: Scheme of the metabolism of a typical PAH, benzo(a)pyrene

the small intestine if they are biliary conjugates. This metabolic pathway is of great importance as the hydroxyl metabolites in urine are usually measured as biomarkers of PAH exposure (Strickland *et al.*, 1996). In some instances however, products of xenobiotic transformation reactions can form covalent interactions with proteins and nucleic acids resulting in the formation of adducts that can compromise normal cell function, triggering a series of deleterious effects that often lead to cancers (WHO/IPCS, 1998; Jacob and Seidel, 2002; Brandt and Watson, 2003). Benz[a]pyrene, for example, is known to form adducts with guanine and adenine and such adducts result in mutations of the genetic code and could lead to deleterious health consequences if not corrected by the cell's repair systems.

BIOMARKERS OF PAH EXPOSURE

Environmental assessment of PAH mixtures can be based on a representative of the group such as benzo(a)pyrene or a group of PAHs (Brandt and Watson, 2003; Franco *et al.*, 2008). In animals, the unaltered PAH or its metabolites are searched for in the blood, urine, faeces or other body fluids. Pyrene is commonly found in PAH mixtures and its predominant urinary metabolite, 1-hydroxypyrene (1-OHP) is used as an indicator of exposure to PAHs (Granella and Clonfero, 1993; CDCP, 2005; Serdar *et al.*, 2012). The American Conference of Governmental Industrial Hygienists recommends that 1-OHP measurement [first introduced as a biomarker of exposure to PAHs by Jongeneelen *et al.* (1986) be used as an end-of-shift or end-of-week urine sampling method for measuring PAHs' Biological Exposure Index (BEI) (ACGIH, 2005; Heikkila *et al.*, 1995). Its advantage is that pyrene is present in all PAH mixtures at relatively high concentrations. In contrast to other PAH metabolites which are excreted mainly in the faeces, 1-OHP is mainly excreted in the urine.

The determination of thioethers in urine is reported to be of little value, since smoking is a strong confounding factor. Furthermore, no differences in thioether excretion in urine were observed between controls and coke-oven workers or between workers in coke and

graphite-electrode-producing plants (Ferreira *et al.*, 1994a, b). Again, urine samples from workers at an aluminium plant and from coke-oven workers did not show differences in PAH levels in urine between exposed workers and controls (Venier *et al.*, 1985; Haugen *et al.*, 1986).

In Nigeria, neither 1-OHP nor thioethers in urine are available as routine pre or post-shift urine tests as PAH exposure is not regulated by any legislation.

POPULATIONS AT RISK OF PAH EXPOSURE

PAHs are ubiquitous. Hence, entire populations are exposed to PAHs in one way or another to varying degrees. Historically, an English surgeon Percival Pott noted an unusually high incidence of scrotal cancer among London Chimney sweeps in 1775 and suggested that the observation was due to their constant exposure to soot and ash. Since, then PAH-related cancers have been induced in laboratory animals and found in humans especially subjects in contact with coal-tars, soot, coke and other PAHs containing substances (Kennaway, 1955; Kjaerheim, 1999).

Several studies in the scientific literature, especially epidemiologic studies have shown increased cancer mortality in workers exposed to PAH mixtures (Boffetta *et al.*, 1997; Bosetti *et al.*, 2007; Olsson *et al.*, 2010; McClean *et al.*, 2011; Miller *et al.*, 2013; Hogstedt *et al.*, 2013). Workers exposed to PAHs include (but are not limited to) aluminium smelters, asphalt workers, carbon black workers; chimney sweeps, coal-gas workers, coke oven workers, tyre and rubber manufacturing workers, wood workers exposed to creosote etc. (Steenland *et al.*, 1990; Bhatia *et al.*, 1998; Armstrong *et al.*, 2004; Moret *et al.*, 2007; Palm *et al.*, 2011; Serdar *et al.*, 2012; Bruschweiler *et al.*, 2012).

Other groups exposed to PAHs are women who cook over wood fires or coal stoves (Islami *et al.*, 2012) and their fetuses (if they are pregnant) (as PAH and its metabolites have been shown to cross the placenta in various animal studies) (Tang *et al.*, 2012), smokers (Zhong *et al.*, 2011) and tattoo aficionados (Regensburger *et al.*, 2010). Children and pregnant women are the most susceptible sub-groups (Choi *et al.*, 2012). Preparation of food and meat in ways that involve charbroiling, grilling, roasting, barbecuing, etc. and heating of homes with wood or coal fires also increase the risk of exposure to PAHs (Viau *et al.*, 2000; Daniel *et al.*, 2011; Islami *et al.*, 2012).

In Nigeria, all of the above groups are obtainable and also additionally people in some traditional occupations like fish/meat smoking and the relatively new practice of using tyre fire to de-fur edible animal skin and parts.

HEALTH EFFECTS OF EXPOSURE TO PAHs

Poly Aromatic Hydrocarbons (PAHs) exist in nature as mixtures. In nature, a population is exposed to PAH mixtures not single PAHs. Hence, the health effects depend usually on the PAH mixtures the population is exposed to. The structure of a given PAH and the substituted groups in it determine how harmful the PAH will be. However, PAH mixtures are usually not acutely toxic. In fact, most times the acute toxicity observed upon exposure to PAHs may be attributable to other more acutely toxic agents like hydrogen sulphide and sulphur dioxide produced when (for example) tyres are burnt in the open. Such acute effects include but are not limited to headaches, respiratory and dermal irritation and nausea. Naphthalene, the most abundant constituent of coal tar is a skin irritant and its vapours may cause headache, nausea, vomiting and diaphoresis (Rom, 1998).

Some reported health effects associated with occupational exposure to PAHs include, erythema, burns and warts on sun exposed areas of the skin with progression to cancer (the toxic effects of PAH mixtures on the skin are enhanced by exposure to ultraviolet light); irritation and

photosensitivity of the eyes; cough, bronchitis and bronchogenic cancer; leukoplakia, buchal-pharygeal cancer and cancer of the lips; leukaemia and lymphoma, haematuria and kidney and bladder cancers (ATDSR, 2009; Hogstedt *et al.*, 2013; Man *et al.*, 2013).

Carcinogenicity of PAHs is related to the presence of an electron dense region known as the K-region (Pullman and Pullman, 1955; Baird *et al.*, 2005). Since, the populations are exposed to PAH mixtures and many of which have exhibited no carcinogenic potentials, it is postulated that two or more substances within the mixtures may compete with receptors or metabolizing enzymes and such action may result in carcinogenic or chemo-preventive effects (Zeiger, 2003; Baird *et al.*, 2005). Some cancers attributable to PAH mixtures and indicative of route of exposure include lung and bladder cancer (Armstrong *et al.*, 2004; Zhong *et al.*, 2011), esophageal cancer (Islami *et al.*, 2012) and sinonasal cancer (Bruschweiler *et al.*, 2012).

Among women, studies have shown that exposure to PAH mixtures in the first trimester is linked to impairment and restriction of intrauterine growth of the foetus (Choi *et al.*, 2012). This is implicated in the causation of childhood asthma (Tang *et al.*, 2012) and it has adverse effects in the behavioural development of children between the ages of 6 and 7 years (Perera *et al.*, 2012). It is also associated with gastroschitisis, a form of teratogenicity (Lupo *et al.*, 2012).

REGULATION OF EXPOSURE TO PAHs IN NIGERIA AND OTHER DEVELOPING COUNTRIES: HINDRANCES; SUGGESTED SOLUTIONS

No literature was found to substantiate the existence of any PAH standard as a national policy in Nigeria. The existing laws aimed at ensuring a healthy and safe workplace for the Nigerian worker are hardly enforced. There is a dearth of inspectors from the relevant government ministries, departments or agencies. Occupational health and safety practices are scarcely adhered to, except may be in multi-national companies and large corporations. The standards recommended by the ATDSR (2009) in the Table 2 for the regulation of PAHs may however find application in Nigeria.

Table 2: Standards and regulations for Polycyclic Aromatic Hydrocarbons (PAHs)

Agency	Focus	Level	Comments
American Conference of Governmental Industrial Hygienists	Air: Workplace	0.2 mg m ⁻³ for benzene-soluble coal tar pitch fraction	Advisory: TLV* (8 h TWA)†
National Institute for Occupational Safety and Health	Air: Workplace	0.1 mg m ⁻³ for coal tar pitch volatile agents	Advisory: REL‡ (8 h TWA)
Occupational Safety and Health Administration	Air: Workplace	0.2 mg m ⁻³ for benzene-soluble coal tar pitch fraction	Regulation: (benzene soluble fraction of coal tar volatiles) PEL§ (8 h workday)
U.S. Environmental Protection Agency	Water	0.0001 mg L ⁻¹	MCL¶ for benz(a) anthracene
		0.0002 mg L ⁻¹	MCL for benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoran-thene, chrysene
		0.0003 mg L ⁻¹	MCL for dibenz(a, h)anthracene
		0.0004 mg L ⁻¹	MCL for indenol (1, 2, 3-c, d) pyrene

*TLV: Threshold limit value, †TWA (Time-weighted average): Concentration for a normal 8 h workday and a 40 h workweek to which nearly all workers may be repeatedly exposed, ‡REL (Recommended exposure limit): Recommended airborne exposure limit for coal tar pitch volatiles (cyclohexane-extractable fraction) averaged over a 10 h work shift, §PEL (Permissible exposure limit): The legal airborne (PEL) permissible exposure limit for coal tar pitch volatiles (benzene soluble fraction) averaged over an 8 h work shift, ¶MCL: Maximum contaminant level

POSSIBLE INTERVENTION STRATEGIES

Strategies that will mitigate the exposure of Nigerians to PAHs are sorely needed. Such intervention strategies must be simple, cheap and acceptable to all concerned irrespective of educational, socio-cultural, ethnic and religious differences. Below are suggested intervention strategies that will in the long run eliminate totally or ameliorate considerably, the deleterious effects of the exposure to PAHs from burning tyres as used in abattoirs in Nigeria.

A national guideline for the reduction in the level of PAHs in the environment should be developed and an agency set up in the Federal Ministry of Environment and charged with enforcing such guidelines. Such a document should ensure the points listed below are captured, among others:

- Regular health promotion campaigns organized by the relevant authorities aimed at educating the workers and eliciting a change of attitude with respect to work-related exposures to PAHs should be carried out
- Personal Protective Equipment (PPE) in the form of masks, protective heat resistant clothing, sturdy work boots and gloves should be made available to kanda processors/roasters and their habitual use such equipment should be enforced
- Safer means of processing (especially de-furring) animal skin and parts is needed and should be introduced. The use of boiling water is suggested. Though the cost of energy to boil the water may be a problem, organizing the kanda processors into co-operatives and introducing energy saving boilers and recyclers may help in lowering the cost
- Other uses for the animal skin and parts (for example in the leather and tanning industry) should be identified to forestall their being consumed as food. These roasters can now be retrained and absorbed in an alternate industry
- Acquisition of other skills that can give the roasters other opportunities at gainful alternative employment that is healthier and safer will also ensure that fewer people are exposed to PAHs

CONCLUSION

Exposure to polycyclic aromatic hydrocarbons results in a wide array of disease conditions yet human beings are constantly exposed to them through different sources. In Nigeria, the processing of kanda, employs the burning of old tyres and the exposure of the processors to PAHs. Unfortunately, there are (as yet) no regulations aimed at reducing exposure to these harmful environmental pollutants in Nigeria. The development of such regulations and their implementation is central to saving exposed populations from the risks associated with exposure to PAHs.

REFERENCES

- ACGIH, 2005. Polycyclic Aromatic Hydrocarbons (PAHs) Biologic Exposure Indices (BEI). American Conference of Governmental Industrial Hygienists, Cincinnati, OH, USA.
- ATSDR, 1995. Toxicological profile for Polycyclic Aromatic Hydrocarbons (PAHs). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta GA., USA., August 1995.
- ATSDR, 2009. Case studies in environmental medicine: Toxicity of Polycyclic Aromatic Hydrocarbons (PAHs). WB 1519, Agency for Toxic Substances and Disease Registry, Atlanta, GA., USA., July 1, 2009.

- Allaby, M., 1989. Dictionary of the Environment. 3rd Edn., New York University Press, New York, USA., ISBN-13: 9780814705919, Pages: 423.
- Armstrong, B., E. Hutchinson, J. Unwin and T. Fletcher, 2004. Lung cancer risk after exposure to polycyclic aromatic hydrocarbons: A review and Meta-analysis. *Environ. Health. Perspectives*, 112: 970-978.
- Baird, W.M., L.A. Hooven and B. Mahadevan, 2005. Carcinogenic polycyclic aromatic hydrocarbon-DNA adducts and mechanism of action. *Environ. Mol. Mutagen.*, 45: 106-114.
- Bhatia, R., P. Lopipero and A.H. Smith, 1998. Diesel exhaust exposure and lung cancer. *Epidemiology*, 9: 84-91.
- Boffetta, P., N. Jourenkova and P. Gustavsson, 1997. Cancer risk from occupational and environmental exposure to polycyclic aromatic hydrocarbons. *Cancer Causes Control*, 8: 444-472.
- Bosetti, C., P. Boffetta and C. La Vecchia, 2007. Occupational exposures to polycyclic aromatic hydrocarbons and respiratory and urinary tract cancers: A quantitative review to 2005. *Ann. Oncol.*, 18: 431-446.
- Brandt, H.C. and W.P. Watson, 2003. Monitoring human occupational and environmental exposures to polycyclic aromatic compounds. *Ann. Occup. Hyg.*, 47: 349-378.
- Bruschweiler, E.D., B. Danuser, C.K. Huynh, P. Wild and P. Schupfer *et al.*, 2012. Generation of Polycyclic Aromatic Hydrocarbons (PAHs) during woodworking operations. *Front. Oncol.*, Vol. 2. 10.3389/fonc.2012.00148
- CDCP, 2005. Third national report on human exposure to environmental chemicals. NCEH Pub. No. 05-0570, Department of Health and Human Services, Centers for Disease Control and Prevention, Atlanta, GA., USA., July 2005.
- Carrasco, F., N. Bredin and M. Heitz, 2002. Gaseous contaminant emissions as affected by burning scrap tires in cement manufacturing. *J. Environ. Q.*, 31: 1484-1490.
- Choi, H., L. Wang, X. Lin, J.D. Spengler and F.P. Perera, 2012. Fetal window of vulnerability to airborne polycyclic aromatic hydrocarbons on proportional intrauterine growth restriction. *PLoS One*, Vol. 7. 10.1371/journal.pone.0035464
- Daniel, C.R., K.L. Schwartz, J.S. Colt, L.M. Dong and J.J. Ruterbusch *et al.*, 2011. Meat-cooking mutagens and risk of renal cell carcinoma. *Br. J. Cancer*, 105: 1096-1104.
- Ferrao, P., P. Ribeiro and P. Silva, 2008. A management system for end-of-life tyres: A Portuguese case study. *Waste Manage.*, 28: 604-614.
- Ferreira, Jr. M., J.P. Buchet, J.B. Burrion, J. Moro and L. Cupers *et al.*, 1994a. Determinants of urinary thioethers, D-glucaric acid and mutagenicity after exposure to polycyclic aromatic hydrocarbons assessed by air monitoring and measurement of 1-hydroxypyrene in urine: A cross-sectional study in workers of coke and graphite-electrode-producing plants. *Int. Arch. Occup. Environ. Health*, 65: 329-338.
- Ferreira, Jr. M., S. Tas, M. dell'Omo, G. Goormans, J.P. Buchet and R. Lauwerys, 1994b. Determinants of benzo(a)pyrenediol epoxide adducts to haemoglobin in workers exposed to polycyclic aromatic hydrocarbons. *Occup. Environ. Med.*, 51: 451-455.
- Franco, S.S., A.C. Nardocci and W.M.R. Gunther, 2008. PAH biomarkers for human health risk assessment: A review of the state-of-the-art. *Cadernos Saude Publica*, 24: a569-s580.
- Granella, M. and E. Clonfero, 1993. Urinary excretion of 1-pyrenol in automotive repair workers. *Int. Arch. Occup. Environ. Health*, 65: 241-245.

- Haugen, A., G. Becher, C. Benestad, K. Vahakangas, G.E. Trivers, M.J. Newman and C.C. Harris, 1986. Determination of polycyclic aromatic hydrocarbons in the urine, benzo(a)pyrene diol epoxide-DNA adducts in lymphocyte DNA and antibodies to the adducts in sera from coke oven workers exposed to measured amounts of polycyclic aromatic hydrocarbons in the work atmosphere. *Cancer Res.*, 46: 4178-4183.
- Heikkila, P., M. Luotamo, L. Pyy and V. Riihimaki, 1995. Urinary 1-naphthol and 1-pyrenol as indicators of exposure to coal tar products. *Int. Arch. Occup. Environ. Health*, 67: 211-217.
- Hogstedt, C., C. Jansson, M. Hugosson, H. Tinnerberg and P. Gustavsson, 2013. Cancer Incidence in a Cohort of Swedish Chimney Sweeps, 1958-2006. *Am. J. Public Health*, 103: 1708-1714.
- IARC, 1983. Polynuclear Aromatic Hydrocarbons, Part 1: Chemical, Environmental and Experimental Data. Vol. 32, International Agency for Research on Cancer, Lyon, France, ISBN-13: 9789283212324, Pages: 477.
- Ifere, G.O., F. Abebe and G.A. Ananaba, 2012. Emergent trends in the reported incidence of prostate cancer in Nigeria. *Clin. Epidemiol.*, 4: 19-32.
- Islami, F., P. Boffetta, F.J. van Schooten, P. Strickland and D.H. Phillips *et al.*, 2012. Exposure to polycyclic aromatic hydrocarbons among never smokers in Golestan Province, Iran, an area of high incidence of esophageal cancer: A cross-sectional study with repeated measurement of urinary 1-OHPG in two seasons. *Front. Oncol.*, Vol. 2. 10.3389/fonc.2012.00014
- Jacob, J. and A. Seidel, 2002. Biomonitoring of polycyclic aromatic hydrocarbons in human urine. *J. Chromatogr. B*, 778: 31-47.
- Jongeneelen, F.J., R.P. Bos, R.B. Anzion, J.L. Theuws and P.T. Henderson, 1986. Biological monitoring of polycyclic aromatic hydrocarbons: Metabolites in urine. *Scandinavian J. Work Environ. Health*, 12: 137-143.
- Kennaway, E., 1955. The identification of a carcinogenic compound in coal-tar. *Br. Med. J.*, 2: 749-752.
- Kjaerheim, K., 1999. Occupational cancer research in the Nordic countries. *Environ. Health Perspect.*, 107: 233-238.
- Le Du-Lacoste, M., F. Akcha, M.H. Devier, B. Morin, T. Burgeot and H. Budzinski, 2013. Comparative study of different exposure routes on the biotransformation and genotoxicity of PAHs in the flatfish species, *Scophthalmus maximus*. *Environ. Sci. Pollut. Res.*, 20: 690-707.
- Leung, H.W. and D.J. Paustenbach, 1994. Techniques for estimating the percutaneous absorption of chemicals due to occupational and environmental exposure. *Applied Occup. Environ. Hyg.*, 9: 187-197.
- Lupo, P.J., P.H. Langlois, J. Reefhuis, C.C. Lawson and E. Symanski *et al.*, 2012. Maternal occupational exposure to polycyclic aromatic hydrocarbons: Effects on gastroschisis among offspring in the national birth defects prevention study. *Environ. Health Perspect.*, 120: 910-915.
- Man, Y.B., Y. Kang, H.S. Wang, W. Lau and H. Li *et al.*, 2013. Cancer risk assessments of Hong Kong soils contaminated by polycyclic aromatic hydrocarbons. *J. Hazardous Mater.*, 261: 770-776.
- McClean, M.D., K.L. Kelsey, J.D. Sison, C.P. Quesenberry Jr., M.R. Wrensch and J.K. Wiencke, 2011. A case-control study of asphalt and tar exposure and lung cancer in minorities. *Am. J. Ind. Med.*, 54: 811-818.
- Miller, B.G., E. Doust, J.W. Cherrie and J.F. Hurley, 2013. Lung cancer mortality and exposure to polycyclic aromatic hydrocarbons in British coke oven workers. *BMC Public Health*, Vol. 13. 10.1186/1471-2458-13-962

- Moret, S., G. Purcaro and L.S. Conte, 2007. Polycyclic Aromatic Hydrocarbon (PAH) content of soil and olives collected in areas contaminated with creosote released from old railway ties. *Sci. Total Environ.*, 386: 1-8.
- OSPAR Commission, 2003. JAMP guideline on methods and criteria for harmonised sampling and analysis of PAHs in air and precipitation. Ref. No. 2003-8, OSPAR Commission, Monitoring Guidelines.
- Olsson, A.C., J. Fevotte, T. Fletcher, A. Cassidy and A.T. Mannetje *et al.*, 2010. Occupational exposure to polycyclic aromatic hydrocarbons and lung cancer risk: A multicenter study in Europe. *Occup. Environ. Med.*, 67: 98-103.
- Palm, L.M.N., D. Carboo, P.O. Yeboah, W.J. Quasie, M.A. Gorleku and A. Darko, 2011. Characterization of Polycyclic Aromatic Hydrocarbons (PAHs) present in smoked fish from Ghana. *Adv. J. Food Sci. Technol.*, 3: 332-338.
- Perera, F.P., D. Tang, S. Wang, J. Vishnevetsky and B. Zhang *et al.*, 2012. Prenatal Polycyclic Aromatic Hydrocarbon (PAH) exposure and child behavior at age 6-7 years. *Environ. Health Perspect.*, 120: 921-926.
- Pullman, A. and B. Pullman, 1955. Electronic structure and carcinogenic activity of aromatic molecules: New developments. *Adv. Cancer Res.*, 3: 117-169.
- Ravindra, K., R. Sokhi and R. van Grieken, 2008. Atmospheric polycyclic aromatic hydrocarbons: Source attribution, emission factors and regulation. *Atmos. Environ.*, 42: 2895-2921.
- Regensburger, J., K. Lehner, T. Maisch, R. Vasold and F. Santarelli *et al.*, 2010. Tattoo inks contain polycyclic aromatic hydrocarbons that additionally generate deleterious singlet oxygen. *Exp. Dermatol.*, 19: e275-e281.
- Reisman, J.I., 1997. Air emissions from scrap tire combustion. EPA-600/R-97-115, U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC., USA., October 1997.
- Rom, W.N., 1998. Polycyclic Aromatic Hydrocarbons. In: *Environmental and Occupational Medicine*, Row, W. (Ed.). 3rd Edn., Lippincott-Raven, Philadelphia, PA., pp: 1261-1267.
- Serdar, B., D. Lee and Z. Dou, 2012. Biomarkers of exposure to Polycyclic Aromatic Hydrocarbons (PAHs) and DNA damage: A cross-sectional pilot study among roofers in South Florida. *BMJ Open*, Vol. 2. 10.1136/bmjopen-2012-001318
- Shen, G., S. Tao, W. Wang, Y. Yang and J. Ding *et al.*, 2011. Emission of oxygenated polycyclic aromatic hydrocarbons from indoor solid fuel combustion. *Environ. Sci. Technol.*, 45: 3459-3465.
- Skupinska, K., I. Misiewicz and T. Kasprzycka-Guttman, 2004. Polycyclic aromatic hydrocarbons: Physicochemical properties, environmental appearance and impact on living organisms. *Acta Poloniae Pharmaceutica*, 61: 233-240.
- Steenland, N.K., D.T. Silverman and R.W. Hornung, 1990. Case-control study of lung cancer and truck driving in the Teamsters Union. *Am. J. Public Health*, 80: 670-674.
- Strickland, P., D. Kang and P. Sithisarankul, 1996. Polycyclic aromatic hydrocarbon metabolites in urine as biomarkers of exposure and effect. *Environ. Health Perspect.*, 104: 927-932.
- Tang, W.T., L. Levin, G. Talaska, Y.Y. Cheung and J. Herbstman *et al.*, 2012. Maternal exposure to polycyclic aromatic hydrocarbons and 5'-CpG methylation of interferon- α in cord white blood cells. *Environ. Health Perspect.*, 120: 1195-1200.
- USEPA, 1989. Risk assessment guidance for superfund, Volume I: Human health evaluation manual (part A). EPA/540/1-89/002, United States Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC., USA., December 1989.

- USEPA, 1992a. Guidelines for exposure assessment. EPA/600/Z-92/001, U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, DC., USA., May 29, 1992.
- USEPA, 1992b. Supplemental guidance to RAGS: Calculating the concentration term. Publication 9285.7-08I, U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC., USA., May 1992.
- Venier, P., E. Clonfero, D. Cottica, C. Gava, M. Zordan, L. Pozzoli and A.G. Levis, 1985. Mutagenic activity and polycyclic aromatic hydrocarbon levels in urine of workers exposed to coal tar pitch volatiles in an anode plant. *Carcinogenesis*, 6: 749-752.
- Viau, C., G. Hakizimana and M. Bouchard, 2000. Indoor exposure to polycyclic aromatic hydrocarbons and carbon monoxide in traditional houses in Burundi. *Int. Arch. Occup. Environ. Health*, 73: 331-338.
- WHO/IPCS, 1998. Selected non-heterocyclic polycyclic aromatic hydrocarbons. Environmental Health Criteria No. 202, International Programme on Chemical Safety, World Health Organization, Geneva.
- WHO, 2000. Air Quality Guidelines for Europe (WHO Regional Publications, European Series No. 91). 2nd Edn., World Health Organization, Regional Office for Europe, Copenhagen, Denmark, ISBN-13: 9789289013581, Pages: 273.
- Walgraeve, C., K. Demeestere, J. Dewulf, R. Zimmermann and H. van Langenhove, 2010. Oxygenated polycyclic aromatic hydrocarbons in atmospheric particulate matter: Molecular characterization and occurrence. *Atmos. Environ.*, 44: 1831-1846.
- Zeiger, E., 2003. Illusions of safety: Antimutagens can be mutagens and anticarcinogens can be carcinogens. *Mutation Res.*, 543: 191-194.
- Zhong, Y., S.G. Carmella, P. Upadhyaya, J.B. Hochalter and D. Rauch *et al.*, 2011. Immediate consequences of cigarette smoking: Rapid formation of polycyclic aromatic hydrocarbon diol epoxides. *Chem. Res. Toxicol.*, 24: 246-252.