

EXPOSURE PATHWAYS AND THE EFFECTS OF TRACE METALS AND POLYCYCLIC AROMATIC HYDROCARBONS (PAHS) ON HUMANS

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Abstract. The various exposure pathways and health impacts of trace metals (TMs) and polycyclic aromatic hydrocarbons (PAHs) on humans were reviewed. They have been confirmed to be toxic and carcinogenic to humans, especially above safe limits. Various forms of industrial and domestic processes lead to the discharge of trace metals, PAHs, and their compounds in different forms such as gases, vapour, particulates, liquid waste, and solid waste into the air, water-bodies, and soil. TMs and PAHs are deposited in food through the combustion of fossil fuels, grilling of food at high temperatures, pan frying, and microwave methods of cooking. Some heavy metals like chromium, cadmium, and arsenic are said to cause cancer and genetic instability. The cancer-causing potential of PAH is a result of the metabolic transformation undergone by PAH in the human body which forms active metabolites that bind to DNA, a genetic material inside the cell. PAHs have been classified by the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) according to their degree of toxicity and carcinogenicity to humans. Avoiding contact of food with flames and cooking with the heat sources above rather than below food is the recommendation made by the Joint Expert Committee on Food Additives (JECFA) as a measure of PAH reduction.

Key words: Impacts, Metals, PAHs, Pathways, Toxic, Trace.

INTRODUCTION

Human exposure to Trace Metals (TMs) and Polycyclic Aromatic Hydrocarbons (PAHs) in food has aroused tremendously global interest due to their toxic effects on humans [1]. The increasing deposition of PAHs and trace metals in humans results from food improvement production and processing technologies. Trace metals and polycyclic aromatic hydrocarbons (PAHs) are environmental pollutants that can have various exposure pathways in humans. These substances can enter the human body through different routes, leading to potential health risks. Trace metals and polycyclic aromatic hydrocarbons (PAHs) can have various adverse effects on human health, depending on factors such as the level and duration of exposure, the route of exposure, individual susceptibility, and the specific chemical properties of the contaminants. Understanding these exposure pathways is crucial for assessing and managing the risks associated with trace metals and PAHs on human health. These effects

highlight the importance of reducing exposure to trace metals and PAHs through pollution control measures, regulatory actions, occupational safety practices, public health interventions, and consumer awareness. Additionally, monitoring and research efforts are essential for better understanding the health risks associated with these contaminants and developing effective mitigation strategies. The exposure pathways of Trace metals and PAHs may include but are not limited to the following, inhalation, ingestion, dermal contact tobacco smoke, etc. Interestingly, trace metals are metallic elements needed by the body for a specific function but when taken in excessive amounts may result in health problems. Although trace elements, trace metals, toxic metals, and heavy metals are sometimes used interchangeably by some scholars this ought not to be so. Trace metals or trace elements are essential elements needed by plants or animals for proper functioning such as Zn, Fe, Ca, Mg, etc. but toxic metals or heavy metals are metallic elements such as As, Hg Cd, Pb that can induce toxicity at low

level of exposure [1]. TMs and PAHs are produced as a result of the incomplete combustion of fossil fuels or the barbecuing of food with flames of high temperatures. Though grilling of food at high temperatures or smoke from the exhaust is not the only way PAHs are deposited in food other ways include the microwave method of heating, high-temperature cooking, pan frying, etc. Due to the health impact of TMs and PAHs on exposure to humans, international organizations and the European Union have set limits of 0.1 mg/kg for Pb in food [2,3].

TRACE METALS EXPOSURE AND HEALTH HAZARDS

There is global concern about trace metals in the environment due to their persistence, toxicity, and bioaccumulation in the food chain. The release of toxic metals and metalloids into the environment is one of the most dangerous and pernicious forms of pollution [4]. Various forms of industrial and domestic processes lead to the discharge of trace metals and their compounds in different forms; gases, vapour, particulates, liquid waste, solid waste into the air, water-bodies and the soil. Trace metals get into the bodies of man and animals through ingestion (i.e. taking contaminated food and drink) inhalation and thermal contact. Plants absorb trace metals in air through their leaves or from the soil through their roots. Ingestion of trace metals and their compounds can come in several ways. Plants grown in soils contaminated with trace metals absorb them and when eaten by man and animals get into the body system. Aquatic organisms grown in contaminated water bodies and animals contaminated by trace metals transfer the same to man when they are eaten. A major source of trace metal entry into the human system is drinking water. Storage of drinking water in containers lined with trace metals, water from trace metal piping systems, and water from contaminated water bodies all lead to trace metal contamination of body tissues. Ingested trace metals are first

digested (i.e. metabolized) in the alimentary canal before transport to other tissues.

The skin is another port of entry for trace metal pollutants since fat-soluble metals are absorbed by the body's fat lining and may get to inner tissues. Animals with a thick cover of body fat retain metal pollutants in their fat lining and prevent them from getting to sensitive tissues. Prolonged exposure of the skin to trace metal pollutants increases the incidence of entry into the body through the skin. Non-polar and lipid-soluble compounds may readily penetrate the fatty layer of the epidermis (skin is made up of the upper epidermis, middle dermis or true skin, and subcutaneous layer). Mercury, arsenic, chromium, and nickel salts are absorbed by the human body through the skin. Sources include mercury-based bleaching creams which cause skin damage and other problems to man.

Most toxic components of air pollution emanate from automobile exhaust, coal-fired power plants, garbage incinerators, metallurgical and refinery operations, and aerosol cans of cosmetics, pesticides, paints, varnishes, and propellants. Heavy metal pollutants in the air occur as gases and mainly particulates or are adsorbed on particulate matter in the air. Inhalation of air contaminated by heavy metals makes it easier for heavy metals to get directly to the blood without prior processing by digestion in the alimentary canal or detoxication by the liver. Toxicity of trace metals is dependent on dosage [4]. Acute toxicity is a result of large doses of a metal toxicant and symptoms appear rapidly and may result in death. Chronic toxicity is a result of prolonged exposure to small doses and symptoms appear gradually and may also lead to death. The effect of trace metals on man and animals can be additive, antagonistic, or synergistic. For instance, zinc and copper are cadmium antagonists, so adverse effects of high cadmium intake can be reduced by above-normal amounts of Zinc and Copper in the body. Virtually all trace metals, including the essential ones (Iron, copper, Zinc, etc) are toxic if safe limits are exceeded.

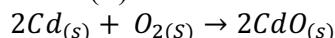
Table 1. W.H.O Permissible limits of trace metals in food

Elements	Permissible limit (mg/kg)
1. Cd	0.2
2. Cu	73.3
3. Fe	425.5
4. Zn	99.4

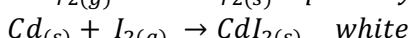
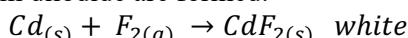
(a) Cadmium is toxic to all man and animal tissues and has no beneficial effect. It stimulates the production of reactive oxygen species causes cell damage and has a particular affinity for sulphhydryl, hydroxyl, and Nitrogen-containing groups [6]. Health effects of cadmium include renal damage and dysfunction, proteinuria, bone lesions, and prostate and lung cancer.

Chemical Reactions of Cadmium

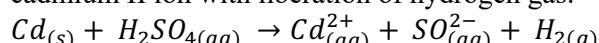
(i) Reaction Cd with O₂: Metallic Cd burns in air to form cadmium (II) oxide



(ii) Reactions of Cd with halogens: When Cd reacts with F₂, Br₂, and I₂, a corresponding cadmium difluoride, cadmium dibromide and cadmium diiodide are formed.



(iii) Reaction of Cd with acid: Metallic cadmium dissolves in dilute sulfuric acid to form an aqueous cadmium II ion with liberation of hydrogen gas.



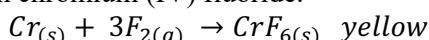
Note: Metallic cadmium does not dissolve in base such as KOH.

(b) Chromium is an essential metal in the human body especially in enhancing insulin activity. However, Cr and its compounds are well-known toxic metals especially Cr⁶⁺ which due to its oxidizing potential easily permeates biological membranes [7]. It can cause renal damage and various forms of cancer.

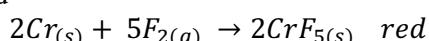
Chemical Reactions of Chromium

(i) Reaction of Cr with O₂: Metallic chromium does not react with oxygen at room temperature. It does not as well react with water at room temperature.

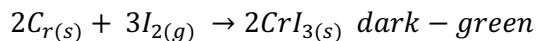
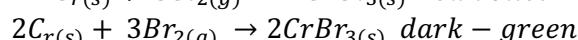
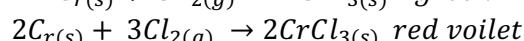
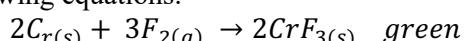
(ii) Reaction of Cr with halogens: At temperature of 400 °C, chromium reacts directly with fluorine to form chromium (IV) fluoride.



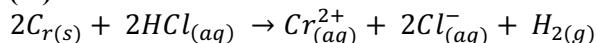
Under mild-conditions, chromium (V) fluoride is formed



Also under mild-conditions, metallic chromium reacts with halogens to form a corresponding trihalides of chromium as indicated in the following equations:



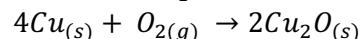
(iii) Reaction of Cr with acid



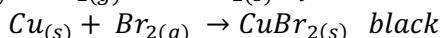
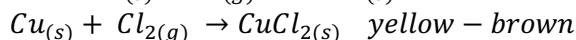
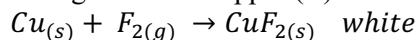
(c) Copper is an essential metal to both fish and man. However, symptoms of acute copper toxicity include hypotension, anemia, and cardiovascular collapse while chronic toxicity leads to sporadic fever, vomiting, diarrhea, and jaundice.

Chemical Reactions of Copper

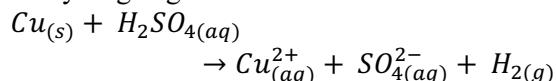
(i) Reaction of Cu with O₂: Copper metals and oxygen react to form Cu₂O as shown



(ii) Reaction of Cu with halogens: When metallic copper react with halogens it forms a corresponding dihalides copper (II).



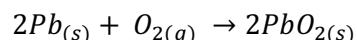
(iii) Reaction of Cu with acid: Metallic copper dissolves in hot concentrated sulfuric acid to form solutions containing aqueous Cu(II) ion together with hydrogen gas.



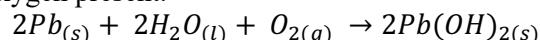
(d) Lead is a well-known toxic metal. After adsorption by the body, it is carried to soft tissues like the brain, lung, spleen, and heart by the blood and finally deposited in the bone where about 90% of total body lead is found. Lead damages the liver, kidneys, brain, central nervous, and reproductive systems [8] causing all kinds of diseases.

Chemical Reactions of Lead

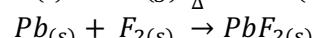
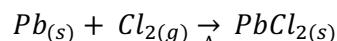
(i) Reaction of Pb with O₂: Lead reacts with oxygen to form lead oxide as shown



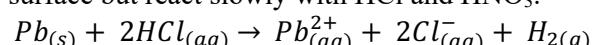
(ii) Reaction of Pb with water: Lead does not react directly with water in the absence of oxygen. The formation of lead (II) hydroxide is as result of oxygen present.

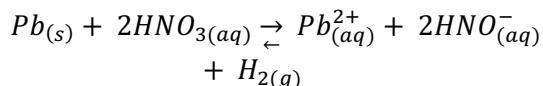


(iii) Reactions of Pb with halogens: Metallic lead when heated reacts with chlorine gas to form lead (II) chloride.



(iv) Reactions of Pb with acids: Lead does not react with sulfuric acid because of passivated PbO surface but react slowly with HCl and HNO₃.

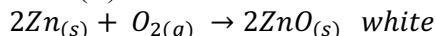




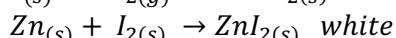
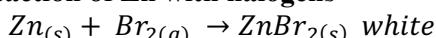
(e) Zinc is essential to man and animals. Acute zinc toxicity in fish can lead to respiratory system damage while chronic toxicity leads to stress and inhibition of normal growth and maturation. Acute Zn toxicity causes diarrhea, and depression of the central nervous system leading to tremors and paralysis, etc. while chronic Zn toxicity causes growth retardation, faulty reproduction, anemia, etc in men.

Chemical Reactions of Zinc

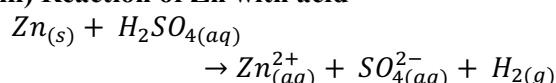
(i) Reaction of Zn with O₂: Zinc metal burn in air to form zinc (II) oxide



(ii) Reaction of Zn with halogens



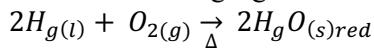
(iii) Reaction of Zn with acid



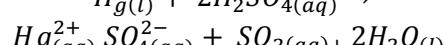
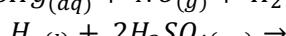
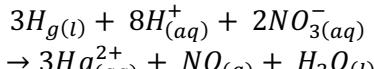
(f) Mercury and most of its compounds are extremely toxic and must be handled with care. The damage caused by elemental Hg is by blocking blood vessels. HgO exists as solid or as liquid metal, its mercurous state (Hg⁺) exists as an inorganic salt and its mercuric state (Hg²⁺) may either form inorganic salts or organo-mercury compounds, the three groups vary in effects. Toxic effects include damage to the brain, kidneys, and lungs. Symptoms typically include sensory impairment (vision, hearing, and speech) disturbed sensation, and lack of coordination. The type and degree of symptom exhibited depend upon the individual toxin, the dose, method, and duration of exposure [9].

Chemical Reactions of Mercury

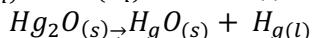
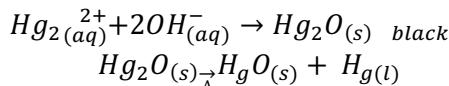
(i) Reaction of Hg with O₂: Hg does not react with O₂ at ordinary temperature rather, oxidizes slowly when heated forming HgO as shown below:



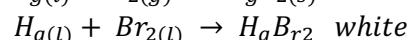
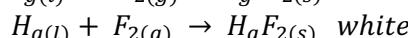
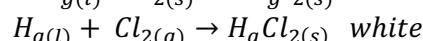
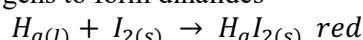
(ii) Reaction of Hg with acid: Hg dissolves in cold nitric acid and hot concentrated H₂SO₄ to form Hg²⁺ salt.



(iii) Reaction of Hg with base: Hg does not react with alkali under normal conditions



(iv) Reactions of Hg with halogens: Hg reacts with halogens to form dihalides



POLYCYCLIC HYDROCARBONS (PAHs)

AROMATIC

Polycyclic aromatic hydrocarbons (PAHs) generally refer to organic compounds containing two or more fused aromatic rings. Examples of PAHs include but are not limited to the following: naphthalene, anthracene, phenanthrene, acenaphthylene, acenaphthene, fluorene, fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene among others.

OCCURRENCE AND POLLUTION OF PAHs

PAHs are among the most widespread organic pollutants. In addition to their presence in fossil fuels, they are also formed by incomplete combustion of carbon-containing fuels such as wood, coal, diesel, fat, and tobacco [10]. Different types of combustion yield different types of PAHs in both relative amounts of individual PAHs and the isomers that are produced. Thus, coal burning produces a different mixture than a motor vehicle or electricity generator combustion or forest fire, making the compounds potentially useful as indicators of the burning history. PAHs are linked to oil spills. Following the massive deep water Horizon oil spill, scientists found PAH levels in soil, and water to be 40 times higher than before the spill [10]. Some authors have concluded that the compounds can enter the food chain through organisms such as fish and PAHs kill fish through cardiac arrest [11]. Very low concentrations of PAHs can slow fish heartbeats and disrupt the development of fish larvae. This may have implications for mammals and other forms of vertebrate life [12]. Studies have shown that high levels of PAHs are found, in meat cooked at high temperatures such as grilling or barbecuing, and in smoked fish. In a study evaluating the genotoxic and carcinogenic risks associated with the consumption of repeatedly heated coconut oil (RCO), it was concluded that dietary consumption of RCO can cause a genotoxic and preneoplastic change in the liver [13,14].

PROPERTIES OF PAHs

The physical and chemical properties of PAHs are dependent on the number of aromatic rings and on the molecular mass. The smallest member of the PAH family is naphthalene, a two-ring compound that is mainly found in the vapour phase of the atmosphere because of high vapour pressure. Three to five-ring PAHs can be found in both the vapour and particulate phases in the air. PAHs consisting of five or more rings tend to be solids adsorbed onto other particulate matter in the atmosphere. The resistance of PAHs to oxidation, reduction, and vaporization increases with increasing molecular weight, whereas the aqueous solubility of these compounds decreases. As a result, PAHs differ in their behaviour, their distribution in the environment, and their effects on biological systems [13].

HEALTH IMPACTS OF PAHs

The toxicity of PAHs is structure-dependent. Isomers (PAHs with the same formula and number of rings) can vary from being non-toxic to extremely toxic. Benzo[a]pyrene, is the first PAH carcinogen to be discovered and is one of the many carcinogens found in cigarette smoke. The USEPA Health Base has classified seven PAHs as probable human carcinogens: benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]floranthene, chrysene, dibenz[a,h]anthracene and indeno [1,2,3-cd] pyrene [15]. High prenatal exposure to PAH is associated with lower IQ and childhood asthma. The Center for Children's Health reported that exposure to PAH pollution during pregnancy is related to adverse birth outcomes including low birth weight, premature delivery, and heart malformations [16]. Studies also show higher levels of developmental delays at age three, lower scores on IQ tests, and increased behavioural problems at ages six and eight. Infants found to have elevated PAH levels in their umbilical cord blood were 46 % more likely to eventually score highly on the anxiety/depression scale than those with low PAH levels in cord blood [15]. Further studies reported that the main contribution to the direct-acting mutagenicity of diesel exhaust particulate were nitro PAHs, including 1,3-, 1,6- and 1,8- dinitropyrenes, 1-nitropyrene, 4-nitropyrene, and 6-nitrochrysene [15, 16, 6, 17, 18].

PAHs undergo metabolic transformation in the human body and may form products that are excreted or active metabolites that may bind to

DNA, the genetic materials inside the cell. The latter pathway is considered to be related to the cancer-causing potential of PAHs. The PAH, benzo[a]pyrene, is classified as "carcinogenic to human" (i.e. Group 1 agent), and three PAHs namely, cyclopenta[cd]pyrene, dibenzo[a,l]pyrene, and dibenz[a,h]anthracene, are classified as "probably carcinogenic" to human (i.e. Group 2 agent) by the International Agency for Research on Cancer (IARC) of the World Health Organization. Since cancer-causing potential is the concern and some PAHs can damage genetic materials inside cells, it is not possible to define a level of exposure that is without risk.

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) states that exposure to PAHs should be as low as practicable [2]. Exposure reduction measures include avoiding contact with foods high flames, and cooking with the heat source above rather than below the food, etc. The Food and Environmental Hygiene Department of Hong Kong conducted a study on PAHs in 2004. Samples of barbecued meats (roasted pork, BBQ pork, roasted ducks, dried beef and dried pork) were collected and analyzed. They found that the higher the cooking temperature, the more PAHs are generated; the closer the distance of the food from the heat source, the higher the PAH levels. Furthermore, the degree of PAH deposition is said to be higher on the skin and fat portion (the outer part) of roasted ducks; and the cooking method of charcoal grilling gives rise to more PAHs in foods when compared with gas grilling and electric oven roasting methods [19].

CONCLUSION

Trace metals and PAHs are toxic and carcinogenic to humans on exposure and it is recommended that high awareness of the exposure pathways and health hazards of these compounds should be created among the general public. Exposure limitations measures are crucial in reducing the effect of these toxicants on humans. Health authorities at all levels in Nigeria should create clear and reliable guidelines and permissible limits for trace metals and PAHs in food.

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