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Electronic Waste Components in Developing Countries: Harmless Substances or Potential Carcinogen

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Author's contribution

Author VOA design the review article, did the literature search and wrote the first and all other drafts of this manuscript. Author VOA read and approved the final manuscript.

Review Article

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ABSTRACT

The continuous increase in the scientific breakthroughs which has existed for some decades, has greatly contributed to the ease of human existence in all ramification. However, these sucesses are not without their short-comings. One of the major challenges faced, is the uncontrolled generation of used electronic appliances or gadgets, commomnly called electronic waste (e-waste), especially in the developing countries. E-waste generated from electrical and electronic equipments is one of the fastest growing global manufacturing activities. In the developed countries, larger percentage of these e-wastes are recycled, thus reducing the potential health risk that could result from exposure to e-wastes. However, in the developing countries, there is little or no machinery in place for recycling and controlling e-wastes, probably because this part of the world are mainly consumers. Most of the e-waste are either used as landfills, disposed and left to decay naturally or burnt. Through these waste disposing practices, the components in the e-wastes either leach into underground water bodies or escape into the atmosphere. Most of these components are potent carcinogens, that pose a great health risk to aquatic lives and humans who are continually exposed to them, no wonder, Africa has been tagged " hub of diseases". There is an urgent need for a more efficient and robust e-waste disposal technology in the developing countries, so that the overwhelming challenges posed by the components of e-waste can be efficiently controlled.

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1. INTRODUCTION

The production of electrical and electronic equipment (EEE) is one of the fastest growing global manufacturing activities. Rapid economic growth, coupled with urbanization and a growing demand for consumer goods, has greatly increased both the consumption and the production of EEE [1]. New electronic gadgets and appliances have infiltrated every aspect of our daily lives, providing our society with more comfort, health and security and with easy information acquisition and exchange [2]. The same hypertechnology that is hailed as a 'crucial vector' for future modern societal development has a not-so-modern downside to it: electronic waste (e-waste) [3]. E-waste broadly covers waste from all electronic and electrical appliances and comprises of items such as computers, mobile phones, digital music recorders/players, refrigerators, washing machines, televisions (TVs) and many other household consumer items [2].

The increasing 'market penetration' in developing countries, 'replacement market' in the developed countries and 'high obsolescence rate' make e-waste one of the fastest waste streams. This kind of waste is posing a serious challenge in disposal and recycling to both developed and developing countries. The dumping of e-waste, particularly computer waste, into underdeveloped and developing countries from developed countries [4] ('green passport' according to Gutierrez) because the latter find its export convenient and economical, has further complicated the problems with waste management. A lot of the reasoning behind the need to control e-waste is for its health and environmental effects. The chemicals and metals usually put into the manufacturing and production of electronics contain properties harmful to physiological system, sometimes their effects are not noticeable right away and often not harmful until the electronics' end of life.

Much of the substances in computers are not biodegradable. The toxic metals contained in them are contaminating the water, air, and soil. While these elements are safely encased in our refrigerators and laptops, e-waste dangers aren't much of an issue, problems occur however when devices break either intentionally or accidentally, the components in them leak and contaminate their immediate environment. When these materials are disposed, their components i.e. heavy metals and other harmful components leach into the underground water and surrounding water bodies, they contaminate these water sources, thus posing a health risk. In the same vein, when some of them are burnt, these heavy metals are released into the atmosphere, thus polluting the air. Properly recycling of electronics waste would not only be a benefit to our environment but for our health. In nature, some of these heavy metals components of electronic, such as lead, mercury, chromium, beryllium etc, are harmless, however, they undergo transformation into the toxic compounds which posed various health risks ranging from mild eye injury to severe cases like DNA damage and cancer.

Due to poor regulations on e-waste recycling and lack of proper control measures in place in developing countries, greater percentage of the population in this part of the world are at the risk posed by these harzadous electronics components. However, developed countries and places that are not home to e-waste landfills are not completely excluded from the effect of e-waste. Pollutants in the air are able to travel to areas far away from the source of generation. As a result of the waste not properly monitored, it can seep into the underground water, thus constituting health hazards to those who are exposed to it. Therefore concerted efforts should be channeled towards combating this challenge posed by this imbroglio. This review is meant to call the attention of the global community, especially that of the developing and

underdeveloped countries, to the imminent danger in which countinuous exposure to these compounds could have on human's health, and also to urgently put a robust e-waste recycling and control machinery in place.

2. WHAT ARE E-WASTES?

Electronic wastes, e-wastes, e-scraps, or Waste Electrical and Electronic Equipments (WEEE) describe loosely discarded, surplus, obsolete, or broken electrical or electronic devices. "Electronic wastes" may be defined as all secondary computers, entertainment device electronics, mobile phones, and other items such as television sets and refrigerators, whether sold, donated, or discarded by their original owners [2]. This definition includes used electronics which are destined for reuse, resale, salvage, recycling, or disposal. Electronic-waste (or *e-waste*) is a collective name for trashed electronic items like obsolete PCs, laptops, fax machines, cell phones, batteries, consumer electronics etc. E-waste is a term used to cover almost all types of electrical and electronic equipment that has or could enter the waste stream [2]. E-waste is any refuse created by discarded electronic devices and components as well as substances involved in their manufacture or use. The disposal of electronics is a growing problem because electronic equipment frequently contains hazardous substances. In a personal computer, for example, there may be lead in the cathode ray tube (CRT) and soldering compound, mercury in switches and housing, and cobalt in steel components, among other equally toxic substances. According to the Environmental Protection Agency (EPA), more than four million tons of e-waste goes to U.S. landfills each year [5].

E-waste has been categorized into three main categories, i.e., Large Household Appliances, IT and Telecom and Consumer Equipment. Refrigerator and washing machine represent large household appliances; PC, monitor and laptop represent IT and Telecom, while TV represents Consumer Equipment [5]. Each of these e-waste items has been classified with respect to 26 common components found in them. These components form the 'building blocks' of each item and therefore they are readily 'identifiable' and 'removable.' These components are metal, motor/ compressor, cooling, plastic, insulation, glass, LCD, rubber, wiring/electrical, concrete, transformer, magnetron, textile, circuit board, fluorescent lamp, incandescent lamp, heating element, thermostat, brominated flamed retardant (BFR)-containing plastic, batteries, CFC/HCFC/HFC/HC, external electric cables, refractory ceramic fibers, radioactive substances and electrolyte capacitors (over L/D 25 mm) [5].

The composition of WEEE/e-waste is very diverse and differs in products across different categories. It contains more than 1000 different substances, which fall under 'hazardous' and 'non-hazardous' categories. Broadly, it consists of ferrous and non-ferrous metals, plastics, glass, wood and plywood, printed circuit boards, concrete and ceramics, rubber and other items. Iron and steel constitutes about 50% of the WEEE followed by plastics (21%), non-ferrous metals (13%) and other constituents. Non-ferrous metals consist of metals like copper, aluminium and precious metals, e.g. silver, gold, platinum, palladium, etc. The presence of elements like lead, mercury, arsenic, cadmium, selenium and hexavalent chromium and flame retardants beyond threshold quantities in WEEE/e-waste classifies them as hazardous waste [6].

2.1 Components of Electronic Waste

Some computer components can be reused in assembling new computer products, while others are reduced to metals that can be reused in applications as varied as construction,

flatware, and jewelry [7]. Substances found in large quantities include epoxy resins, fiberglass, PCBs (Polychlorinated biphenyls), PBDEs (Polybrominated diphenyls ethers), PVC (polyvinyl chlorides), thermosetting plastics, lead, tin, copper, silicon, beryllium, carbon, iron and aluminium. Elements found in small amounts include cadmium, mercury, and thallium [8]. Elements found in trace amounts include americium, antimony, arsenic, barium, bismuth, boron, cobalt, europium, gallium, germanium, gold, indium, lithium, manganese, nickel, niobium, palladium, platinum, rhodium, ruthenium, selenium, silver, tantalum, terbium, thorium, titanium and vanadium[8]. Almost all electronics contain lead and tin (as solder) and copper (as wire and printed circuit board tracks), though the use of lead-free solder is now spreading rapidly. Generally, the components of electronic waste can be arbitrarily divided into two based on their effects: hazardous components and non–hazardous component. The hazardous components include americium, mercury, sulphur, polychlorinated biphenyls (PCB), PBDEs, Cadmium, Lead, beryllium chloride, polyvinyl chloride etc. While the non-hazardous components include tin, copper, aluminium, iron, germanium, silicon, nickel, zinc, gold etc [8].

3. E-WASTE AND ITS EFFECTS ON HUMAN'S HEALTH AND THE ENVIRONMENT

E-waste cannot be considered or treated like any kind of waste, because it contains hazardous and toxic substances such as heavy metals or others such as dioxins and furans (produced when e-waste is incinerated). For instance, lead represents 6% of the total weight of a computer monitor. Ìt has been reported that nearly 36 chemical elements are incorporated in electronic equipment [9]. EEEs are made of a multitude of components, some containing toxic substances that have an adverse impact on human health and the environment if not handled properly. Often, these hazards arise due to the improper recycling and disposal processes, this can have serious health repercussions on those in proximity to places where e-waste is recycled or burnt.

3.1 Effects of the Hazardous E-Waste Components

3.1.1 Lead

Lead is found in many electronic equipment components. For example, in a PC, the largest amount of this metal is found in the CRT (cathode ray tube) of the monitor: 0 to 3% in the panel, 70% in the frit, 24% in the funnel and 30% in the neck [13]. Lead is also present in welding (40%), motherboards, circuits and wiring plastic. Humans are exposed to this metal by particle inhalation and through contaminated foods (via e-waste burning or leaching from landfill) [10,11]. The first effects and symptoms of lead exposure are anorexia, muscle pain, malaise and headache but an extended exposure can cause a decrease in nervous system performance, weakness, brain damage and even death. Lead exerts toxic effects on various systems in the body such as the central (organic affective syndrome) and peripheral nervous systems (motor neuropathy), the hemopoietic system (anemia), the genitourinary system (capable of causing damage to all parts of nephron) and the reproductive systems (male and female) [9]. Likewise, it can affect the reproductive system both in men and women and is considered carcinogen. The chemical structure of this metal is directly affected by its pH but most lead compounds are insoluble in water and remain in that state. Lead doesn't bio-accumulate in fish but it does in other seafood. If broken or incinerated to the environment, particles will be transmitted by air and soil [9].

3.1.2 Mercury

Mercury is found in three specific places in a computer. The largest amount is found in LCD screen fluorescent light, computer or monitor switches, which enable them to shut down while idle, and finally in batteries. Mercury is very volatile and easily liberated by incineration or breaking, which could liberate up to 90% of the mercury contained in the monitor screen, for example. All forms of mercury represent a risk to human health, but mercury in metal form, that is not combined with other components and organic methyl mercury, are the ones that poses the greater risk, especially to the nervous system. Short-term exposures to this compound cause lung damage, nausea, vomiting, diarrhea, high pressure, and, skin and eye irritation. Long or permanent exposure might cause permanent damages to the brain, kidneys and fetus development, besides neurological changes, irritability, tremors, short-sightedness, deafness, memory problems, delirium, hallucinations and suicidal tendencies. Mercury causes damage to the genitourinary system (tubular dysfunction), the central and peripheral nervous systems as well as the fetus [12]. When inorganic mercury spreads out in the water, it is transformed into methylated mercury, which bio-accumulates in living organisms and concentrates through the food chain, particularly by fish [9,13].

3.1.3 Nickel

Nickel is present in the batteries of some electronic equipment (NiCd), which are being gradually replaced with lithium batteries. Likewise, nickel is used in CRT of computer monitors. Nickel causes skin damages and asthma symptoms in about 10 to 20% of the population that has direct contact. The most common harmful health effect of nickel in humans is an allergic skin reaction in those who are sensitive to nickel. Nickel is the most observed cause of immediate and delayed hypersensitivity noticed in occupationally exposed as well in the general population. The metal is not only an allergen but also a potential immunomodulatory and immunotoxic agent in humans [14]. Short-term exposure to nickel could lead to vertigo, nausea, vomiting, insomnia, irritability, which usually last a few hours, followed by an asymptomatic interval of 12 h to 5 days. Other clinical manifestation of acute toxicity include tightness of the chest, nonproductive cough, dyspnoea, cyanosis, tachycardia, palpitations, sweating, visual disturbances, weakness, and lassitude [15]. Long term exposure to nickel has been implicated in asthma, bronchitis, rhinitis, sinusitis, and pneumoconiosis [16]. Studies has revealed that nickel produces some genetic abnormalities like DNA strand break, DNA-protein cross links, nucleotide excision, single gene mutations, sister chromatid exchanges, micronuclei, nucleic acid concentration alteration and cell transformation[17,18,19].

3.1.4 Arsenic

Arsenic is present in small amounts in electronic equipment in forms such as Gallium Arsenide GaAs, which has semi-conductor properties and can be found in electronic equipment diodes. GaAs is carcinogenic and causes skin and lung cancers. The most common means of exposure is direct contact with dust containing this compound especially by workers of semiconductor manufacturers. Gallium Arsenide is an inorganic compound with low water solubility. It is transformed into an organic compound when bio-accumulated in fish and crustaceans [13]. Acute toxicity of arsenic could results in cardiac arrhythmias, anaemia, leucopoenia, sensory loss, nausea, gastrointestinal distress and diarrhoea. Long term exposure have been reported to be responsible for skin lesions, peripheral neuropathy, encephalopathy, hepatomegaly, cirrhosis altered heme metabolism, diabetes, papillary and coritical necrosis [20].

3.1.5 Cadmium

Cadmium is a heavy metal included in many electronic components, such as contact plates, switches, or used to prevent corrosion. Cadmium is particularly found in chip resistors, infrared detectors, and semiconductors. Old monitors contain around 5 to 10 grams of Cadmium and some batteries are made of Nickel Cadmium. It is added as a plastic stabilizer and pigment to wiring, motherboards, PCS, monitors and printed circuit boards.

Cadmium exposure commonly occurs through inhalation and ingestion of food or contaminated water. Inhaling large amounts of Cadmium can cause lung damage and death. Exposure to small amounts over a long period of time can cause high pressure and kidney damage [21]. This metal is a carcinogen [22]. Cadmium enters the environment through water and soil that is absorbed by plants. Low concentrations can cause alterations in the ecology and balance of soil nutrients. This metal can bio-accumulate in mushrooms, oysters, shrimps, mussels and fish. Cadmium is a potentially long-term cumulative poison. Toxic cadmium compounds accumulate in the human body, especially in the kidneys. There is evidence of the role of cadmium and beryllium in carcinogenicity [22,23].

3.1.6 Chromium

Chromium VI, i.e. chromium ions with a charge of +6, is chromium's only toxic form. Its presence is small in electronic equipment where it is used as a plastic hardener and protection layer for some metal components. When electronic components are burned, 99% of Chromium VI stays in residuals and ashes, contaminating soil in a toxic way, which could reach water currents with significant higher risk [23]. The clinical features of acute poisoning are vomiting, diarrhoea, haemorrhage and blood loss into the gastrointestinal tract, causing cardiovascular shock [24]. If the patient survives for more than about 8 days; the major effects resulting from oral ingestion of toxic doses of chromium are liver and kidney necrosis [24]. Other effects of chromium toxicity in humans include irritative dermatitis, ulceration and perforation of the nasal septum, rhinitis, bronchospasm, pneumonia and lung cancer [24].

3.1.7 Polycyclic aromatic hydrocarbons (PAH)

Epidemiological studies in the past on occupational exposure to PAH provide sufficient evidence of the role of PAH in the induction of skin and lung cancers [25]. However, PAH is not actually a component of e-waste, but the critical position it occupies as an important environmental pollutant cannot be overemphasize. In the short term exposure, PAH poses various health risks in humans which depend mainly on length, route and amount being exposed to, and the effect could range from eye irritation, nausea, vomiting, diarrhea, skin irritation, inflammation [25], to complications such as 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 [25].

3.1.8 Polychlorinated biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are a group of organic compounds containing about 209 individual compounds which vary in their chlorine content. PCBs have been used widely in transformer oil, printing inks,hydraulic fliud etc, [26]. PCBs are released into the environment via dumping of old electrical equipment and other e- waste, and also through the dismantling

and recycling of obsolete equipment [26]. Humans are exposed through contaminated food consumption or direct contact at their workplace, (e.g inadequate disassembly of electronic equipment) [11]. Exposure to this compound can cause anemia, damages to the skin, liver, stomach and thyroid. Contamination of pregnant women is very risky and research results show that it can cause toxic effect such as liver damage, nuerotoxicity, immunosuppression, reproductive system damage and carcinogenicity [27,28,29].

3.1.9 Polybrominated diphenyl ethers (PBDEs)

Polybrominated diphenyl ethers (PBDEs) are one of several classes of brominated compound in widespread use as flame retardant additives in plastics and foams, including plastic casings of electronic equipment [30]. There are many different chemicals (congeners) included in this group, differing in the numbers and positioning of bromine atoms in the molecules. Those in common commercial use are “penta” (i.e. a mixture rich in pentabrominated congeners), “octa”, (rich in octabrominated congeners) and “deca” (almost exclusively the decabrominated congener). PBDEs are environmentally persistent chemicals. Some, especially the lower brominated congeners (e.g. “penta-BDE”), are also highly bioaccumulative. Their manufacture and use as additives in plastics and other polymers, in which they are not tightly bound to the polymer matrix, has led to their widespread presence in the environment. PBDEs can be detected in indoor air and dusts in the workplace and in the home [31,32]. They also occur in almost every part of the environment, including sediments [33] and freshwater, and in biota such as marine fish [34], birds eggs [35] and even whales from the deep oceans and the Arctic (36).

PBDEs have also been reported as common contaminants in humans, including reports from Sweden, Spain, Finland and North America [37,38,39,40]. Concentrations of lower brominated PBDEs have shown increasing levels in both blood and breast milk in recent decades, particularly in regions in which “penta” remains in commercial use [41,42,43]. Workers in electronics recycling facilities in Europe have been found to have higher blood levels of PBDEs than other workers, probably as a result of inhalation of contaminated dust [44,45]. Similarly, elevated levels have been reported in the blood of workers (46) and local residents [47] at an e-waste recycling area in China.

For the general population, especially in the developing countries, exposure to PBDEs probably occurs through a combination of food contamination and direct exposure to chemicals from consumer products and/or contaminated dusts, which are normally found in e-waste recyling environments [48]. While their acute toxicity is considered low, chronic exposure to certain PBDEs (especially in the womb) has been associated with abnormal brain development in animals [49], with possible long-term impacts on memory, learning and behaviour [50,51,52]. There are concerns that similar effects may be of relevance in humans [53]. PBDEs also exhibit endocrine (hormone) disrupting properties, interacting with both oestrogen and thyroid hormone systems either as the parent compound or as metabolites [54,54,56,57]. Effects on the immune system have also been reported [58,59]. Furthermore, when plastics containing PBDEs are burned, either in an incinerator or by open burning, the potential exists for formation of brominated dioxins/furans [60] or mixed bromochloro dioxins/furans (61), which appear to be of equivalent or even greater toxicity to chlorinated dioxins [62].

4. MECHANISM OF TOXICITY OF SOME E-WASTE COMPONENTS

4.1 Lead

Lead ranks second among the prioritized hazardous substances issued by the U.S. ATSDR [Agency for Toxic Substances and Disease Registry] in 1999. The noxious effects of this metal have long been well known, especially as regards acute forms of poisoning. However, as for many other contaminants, the threshold level of safety has been drastically lowered recently. Until approximately 30 years ago, chronic lead poisoning was defined by blood lead levels above 80(gr/dl), while today a lead level of 30(gr/dl) in the blood is considered excessive and level at above 10(gr/dl) (0.1ppm) are considered potentially harmful, particularly in children. Once absorbed by the body, mainly through breathing and feeding, lead is not metabolized, but mostly expelled. The remaining portion (about 20%) settles into the tissues and notably: In the blood, where it is carried almost exclusively by the erythrocytes; in mineral tissues (bone and teeth), where it deposits and in soft tissues (kidney, bone marrow, liver and brain).

The presence of lead in the blood stream (inside the red blood cells and mostly linked to haemoglobin) provokes anaemia. Through the blood, lead reaches all other tissues. Because of its capacity to “mimic” calcium, lead is stored in the bones and becomes a stable bone component, particularly in the case of insufficient calcium intake. This lead deposits may be mobilized and return into the blood stream under particular state of physiological stress (pregnancy, breast-feeding, diseases), but also as a consequence of greater calcium intake in the diet. This stable presence of lead in bones make recovery from lead poisoning extremely slow, even when toxic agent has been completely elimated.

4.1.1 Action mechanism

Lead toxicity is largely due to its capacity to mimic calcium and substitute it in many of the funder mental cellular processes that depend on calcium. Lead can cross the cell membrane in several ways which are not well understood. Lead transport through the erythrocyte membrane is mediated by an anion exchanger in one direction and by the Ca-ATPase pump in the other direction. In other tissues, lead permeates the cell membrane through voltage-dependent or other types of calcium channels. Once it has penetrated the cytoplasm, lead continues its destructive mimicking action by occupying the calcium binding sites on numerous calcium-dependent proteins. Lead bind to Calmodulin, a protein which in the synaptic terminal acts as a sensor of free calcium concentration and as mediator of neurotransmitter release. Furthermore, it alters the functioning of the enzyme protein kinase C, a virtually ubiquitous protein which is of crucial importance in numerous physiological functions. Kinase C is normally activated by modulator outside the cell (hormones, neurotransmitters, etc) through an enzyme chain and in a calcium-dependent manner. Other toxic effects include inhibition of heme biosynthesis and binding to sulfhydryl groups (-SH groups) of proteins. Lead binds to the thio-group of the enzymes delta-aminolevulinate synthase and delta-aminolevulinate dehydratase, the first and second enzymes in the biosynthesis of heme (Fig.1), also lead can inhibit the functions of coproporphyrinogen oxidase and ferrochelatase, the last two enzyme in the biosynthesis of heme (Fig. 1).

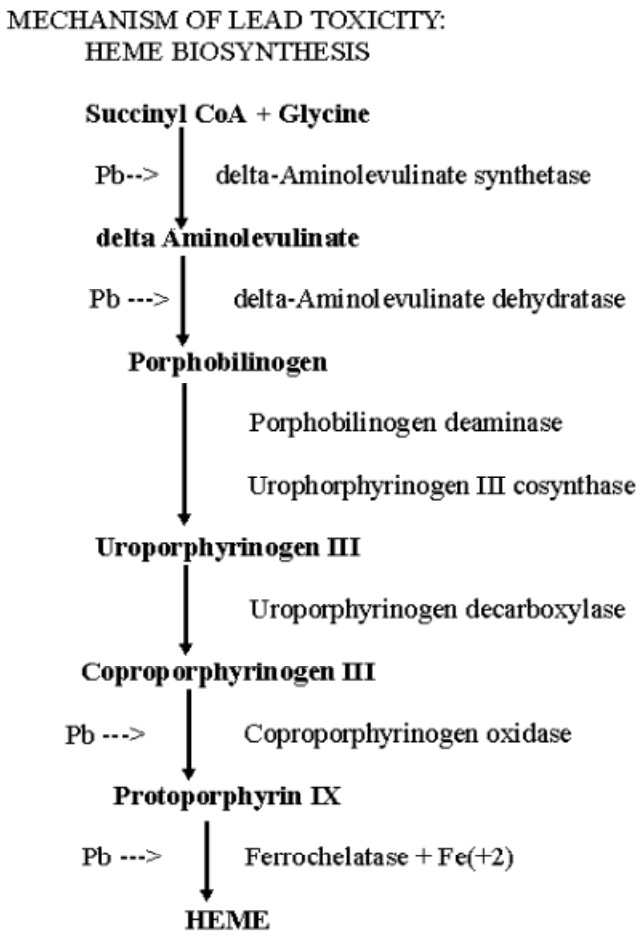

Fig. 1. Mechanism of lead toxicity
Source: Theodore et.al., 2002.

4.2 Mercury

Mercury can exist in three forms, elemental, inorganic and organic, and all are toxic. However, the toxicity of the three forms of mercury is different, mainly as a result of differences in distribution. Some of these toxic properties have been known for centuries. Elemental mercury (Hg) may be absorbed by biological systems as a vapour. Despite being a liquid metal, mercury readily vaporizes at room temperature and in this form constitutes a particular hazard to those who use scientific instruments containing it for example. Elemental mercury vapour is relatively lipid soluble and is readily absorbed from the lungs following inhalation and is oxidized in the red blood cells to Hg^{2+}. Elemental mercury may also be transported in red blood cells to other tissues such as the CNS. Elemental mercury readily passes across the blood-brain barrier into the CNS and also into the foetus. The metallic compound is only poorly absorbed from the gastrointestinal tract, however. Inorganic mercury, existing as monovalent (mercurous) or divalent (mercuric) ions is relatively poorly absorbed from the gastrointestinal tract (7% in humans). After absorption inorganic mercury accumulates in the kidney. Organic mercury is the most readily absorbed (90–95% from the gastrointestinal tract), and after

absorption distributes especially to the brain, particularly the posterior cortex. All the forms of mercury will cross the placenta and gain access to the foetus, although elemental mercury and organic mercury show greater uptake. The concentrations in certain foetal tissues, such as red blood cells, are greater than in maternal tissue. Mercury is eliminated from the body in the urine and faeces with the latter being the major route. Thus, with methyl mercury 90% is excreted into the faeces. Methyl mercury is secreted into the bile as a cysteine conjugate and undergoes extensive enterohepatic recirculation. The half-life of mercury is long but there are two phases, the first being around 2 days, then the terminal phase which is around 20 days. However the half-life will depend on the form of mercury. Thus methyl mercury has a half-life of about 70 days whereas for inorganic mercury this is about 40 days [63]

4.2.1 Mechanism of toxicity of mercury

Mercury is a reactive element and its toxicity is probably due to interaction with proteins. Mercury has a particular affinity for sulphydryl groups in proteins and consequently is an inhibitor of various enzymes such as membrane ATPase, which are sulphydryl dependent. It can also react with amino, phosphoryl and carboxyl groups. Brain pyruvate metabolism is known to be inhibited by mercury, as well as lactate dehydrogenase and fatty acid synthetase. The accumulation of mercury in lysosomes increases the activity of lysosomal acid phosphatase which may be a cause of toxicity as lysosomal damage releases various hydrolytic enzymes into the cell, which can then cause cellular damage. Mercury accumulates in the kidney and is believed to cause uncoupling of oxidative phsophorylation in the mitochondria of the kidney cells. Thus, a number of mitochondrial enzymes are inhibited by Hg^{2+}. These effects on the mitochondria will lead to a reduction of respiratory control in the renal cells and their functions such as solute reabsorption, will be compromised.

4.3 Chromium

Chromium (IV) has long been recognized as a toxin in plant systems and as a carcinogen in human and mammalian systems. The actual mutagenic or toxic species of chromium is one or more of the reactive intermediate produced in the reduction of Cr(IV) to Cr(III). Glutathione is suspected to be a reductant here due to its ability to produce long-lived Cr(V/IV) intermediate during the reduction of chromium(IV). GSH-Cr interaction in plant have been fairly well elucidated [64]. Dichromate reacts with glutathione at the sulfhydryl group forming an unstable glutathione-$Cr0_3$ complex. The Halliwel Asada pathway is the key pathway whereby Cr toxicity or tolerance is mediated.

The high content of dihydro-ascorbate (DHA) in combination with an absence of active scavenging of free radicals and blockage of normal cell cycle progression by DHA is one of the main mechanisms of chromium induced toxicity in plant [64]. Cr(VI) can function as a hill reagent and can inhibit electron transport both in the photosynthetic and mitochondrial apparatus thus accounting for reduced NADPH pool. The critical balance between the available NADPH pool and ROS production by chromium would decide the redox status of a cell in both plants and animals. Chromium-DNA interaction is one of the well explained mechanisms of action of Cr in apoptosis and carcinogenesis. Chromium associate with both DNA bases and the phosphodiester backbone and the binding occur through both covalent binding and electrostatic interactions. The base specific binding of Chromium has revealed a general, but not absolute, preference towards the formation of Cr(III) – guanine DNA adducts and polyriboguanylic acid (poly(G)) in the case of RNA [65].

Cr-DNA crosslinks (Cr-DPCs) have been reported to be extensively developed respectively between DNA and non histone proteins and RNA and cytoplasmic proteins in many animal systems [66]. Cr(VI)-containing compounds are well known carcinogenic compounds. Evidence also have it that chromosomal abnormalities (micronuclei) and genomic instability are possibly involved in the induction of cancer by Cr(VI) [67]. DNA interstand crosslinks (ICLs) are caused by Cr interacting with reaction centers on the complementary strands of DNA. A notion that has received much attention is that intracellular Cr(VI) mediate a fenton-like reaction mediating ROS production which are responsible for nearly all the toxicity and genotoxicity caused by Cr(VI) [68].

4.4 Cadmium

Cadmium is an extremely toxic substance and the major hazard is from inhalation of cadmium metal or cadmium oxide. Although it is present in food, significant oral ingestion is rare and absorption from the gut is poor (5–8%). However, various dietary and other factors may enhance absorption from the gastrointestinal tract. In contrast, up to 40% of an inhaled dose may be absorbed and hence its presence in cigarettes is a significant source of exposure. Cadmium is bound to proteins and red blood cells in blood and transported in this form, but 50–75% of the body burden is located in the liver and kidneys [66]. The half-life of cadmium in the body is between 7 and 30 years and it is excreted through the kidneys, particularly after they become damaged. Cadmium has many toxic effects, primarily causing kidney damage, as a result of chronic exposure, and testicular damage after acute exposure, although the latter does not seem to be a common feature in humans after occupational exposure to the metal. It is also hepatotoxic and affects vascular tissue and bone. After acute inhalation exposure, lung irritation and damage may occur along with other symptoms such as diarrhea and malaise [66]. Chronic inhalation exposure can result in progressive fibrosis of the lower airways leading to emphysema. This results from necrosis of alveolar macrophages and hence release of degradative enzymes which damage the basement membranes of the alveolus. The raised urinary levels of proline and hydroxyproline associated with chronic cadmium toxicity may be due to this damage to the bones [66].

4.5 Polycyclic Aromatic Hydrocarbons (PAH)

Since exposure to PAHs is never to single PAHs, understanding what differences may occur in mixtures of PAHs gives an accurate assessment of the dangers of PAHs. Understanding the dynamics of complex metabolism vis-a-vis single metabolism of PAHs and possible effects on the toxicity expression of PAHs is a necessary advancement to accurately impact and guide remediation strategies. Due to the high lipophilicity of this class of compounds, their bioavailability after ingestion and inhalation is significant. Scientific investigations have shown that detectable levels of PAH occur in almost all internal organs, particularly in organs that are rich in adipose tissue[69].These organs can serve as storage depots from which the hydrocarbons can be gradually released. Once they enter the organism polycyclic aromatic hydrocarbons require a multistep metabolic activation by specific enzymes [69]. The enzyme system primarily responsible for PAH metabolism is the mixed-function oxidase system. The first reaction is an epoxidation. PAH epoxides can then be conjugated with glutathione and this is regarded as a true detoxification reaction. The epoxides that are not conjugated with glutathione are converted into phenols and diols. Although unmetabolized PAHs can have toxic effects, a major concern is the ability of the reactive metabolites, such as epoxides and dihydrodiols, of some PAHs to bind to cellular proteins and DNA. The resulting biochemical disruptions and cell damage lead to mutations, developmental malformations, tumors,

and cancer. Evidence indicates that mixtures of PAHs are carcinogenic to humans [70]. The evidence comes primarily from occupational studies of workers exposed to mixtures containing PAHs and these long-term studies have shown an increased risk of predominantly skin and lung, but as well as bladder and gastrointestinal cancers [70].

4.6 Polychlorinated Biphenyls (PCBs)

PCBs typically cause thymic atrophy, a wasting syn drome, immunotoxic responses, reproductive problems, porphyria and related liver damage. It has been reported that the biological potency of this group of compounds can be linked to their ability to induce some hepatic microsomal enzyme namely benzo(a)pyrene hydroxylase (aryl hydrocarbon hydroxylase, AHH) and cytochromes P-450a, P-450c and P-450d [70].

4.7 Polybrominated Diphenyl Ethers (PBDEs)

Like other persistent organic pollutants congeners, PBDEs congenes are reported to be likely carcinogens, endocrine disruptors, and/or neurodevelopmental toxicants. Among commercial PBDE mixtures, those containing lower congeners are stronger inducers of liver enzymes [i.e., penta-BDE > octa-BDE > deca-BDE][71]. Human data on PBDE carcinogenicity are limited. One study cited an association between adipose tissue levels of PBDE [72] and the risk of non-Hodgkin lymphoma (NHL) among Swedish hospital patients [73]. Other studies cited similar associations for PCB levels and the risk of NHL [74], and for PBB levels and the risk of lymphoma and breast cancer [74,75]. Although PBDEs have not been tested for their ability to bind to the Ah(Aryl hyroxyl) receptor, mechanistic studies indicate that some PBDE congeners exhibit significant Ah receptor-mediated (e.g., dioxin-like) effects, with penta-DBE activity greater than tetra-DBE activity. Ah receptor-mediated activities of PBDEs also have been investigated using the rat hepatoma cell line, 9 congener of PBDEs were able antagonise Ah receptor [76]. Also, it was reported that this is the same mechanism through which PBDEs congeners elicits their genotoxicity, endocrine toxicity,and neurodevelop-mental toxicity effects.

5. CONCLUSION

According to UNEPA (United Nation Environmental Programme Agency, 2010) about 53 million tones of electronic waste was generated worldwide in year 2009 and only 13% was recycled, and most of these recycling were done in the developed countries [77]. Due to poor regulations on e-waste recycling in the developing countries, a lot of methods to retrieve certain metals from electronics and disposing e-waste are polluting the environment. These toxic materials, in the e-waste when disposed indiscriminately or even used in landfills, can leach into the underground water and accumulates in sea foods and plants. This may cause health hazard ranging from tissues or organs damage to chromosomal abnormalities, DNA damage, cancer and eventually death. However, developed countries and places that are not home to e-waste landfills and pollution are not completely excluded from the effects of e-waste. Pollutants can seep into the oceans or travel in the air, thus making e-waste not to be limited by boundaries. Therefore, all steakeholders and the goverments must wake up to the challenges of e-waste by putting up a functioning regulations and well structured recycling programme that will help, if not to stop completely, but to reduce to minimal, the imbroglio of e-waste.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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