**Original Research**

**Toxic metal levels in Nigerian electronic waste workers indicate occupational metal toxicity associated with crude electronic waste management practices**

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**ABSTRACT:** The poor management of electronic wastes (e-waste) in Nigeria exposes workers to toxic chemicals in waste electrical and electronic equipment. In this study, we evaluated the toxic metal levels in Nigerians occupationally exposed to e-waste. Whole blood levels of Lead (Pb), Mercury (Hg), Arsenic (As), Cadmium (Cd), and Chromium (Cr) were determined in Nigerian e-waste workers (n=63) and in age-matched non-exposed participants (n=41), using standard electrothermal atomic absorption spectrometry and inductively coupled plasma-mass spectrometry methods. The results showed statistically significant (p<0.01) elevated body burden of toxic metals in e-waste workers (Pb, 0.95 ± 0.00 µmol/L; Cr, 405.99 ± 6.34 µmol/L; Cd, 108.54 ± 1.60 nmol/L; As, 10.09 ± 0.01 µmol/L; and Hg, 25.02 ± 0.14 nmol/L) compared with non-exposed group ((Pb, 0.03 ± 0.00 µmol/L; Cr, 178.44 ± 5.99 µmol/L; Cd, 56.99 ± 1.42 nmol/L; As, 1.02 ± 0.0008 µmol/L; and Hg, 1.62 ± 0.0672 nmol/L). These data indicate that the elevated body burden of toxic metals in the e-waste exposed population is an indication of occupational metal toxicity associated with crude e-waste management practices in Nigeria. In addition, the potential health implications of exposure to these toxic metals, such as chronic kidney disease, cancer; preceded by genome instability and depressed immune response were highlighted.

**KEYWORDS:** Metal toxicity, e-waste, Nigeria, Occupational exposure.

**INTRODUCTION**

Electronic waste (e-waste, e-scrap, or waste electrical and electronic equipment (WEEE)) describes discarded electrical or electronic devices. These terms are broadly used to describe discarded computers, office electronic equipment, entertainment device electronics, mobile phones, television sets, and refrigerators. This definition includes used electronics that are destined for reuse, resale, salvage, recycling, or disposal (Sthiannopkao and Wong, 2012). The management of E-waste in Nigeria is a serious environmental and health issue that requires serious consideration. Terada (2012) used the serious situation in Nigeria as a case study of e-waste dumping ground in Africa. It was reported that the case of Nigeria was significant because Nigeria is a Party to the Basel Convention and signed the Bamako Convention in February 2008, but is yet to ratify the Bamako Convention. The Bamako Convention
made the trade of hazardous e-waste illegal in sub-Saharan African countries, yet the e-waste trade continues to thrive in Nigeria. Although Nigeria ratified the Basel Convention on May 24, 2004, it still has not ratified the Bamako Convention, and the country remains a dumping ground for e-waste from European and Asian markets (Aginam, 2010; Basel, 2004). It is estimated that 500 containers of second-hand electronics are imported into Nigeria every month from Europe, with each container holding 500 used computers, (Aragbako-Apkore, 2005). About three-quarters of these imported products are reported to be junk that cannot be reused and are dumped in landfills. These e-wastes harbor toxic chemicals that can induce genetic and metabolic alterations in biological systems (Terada, 2012).

The role of toxic chemical carcinogens and oxidative stress, as well as oxidative DNA damage in carcinogenesis are well reported (Anetor, et al., 2013; Terada, 2012; Elst, 2007; Valavanidis, et al., 2009). A growing amount of results provide evidence that toxic and carcinogenic metals are capable of interacting with nuclear proteins and DNA causing oxidative deterioration of biological macromolecules (Valko et al., 2005). Metal-mediated generation of free radicals causes various modifications to DNA bases, enhanced lipid peroxidation, and altered calcium and sulphhydryl homeostasis (Valko et al., 2005). E-waste contains over 1,000 chemicals, most of which have several health implications (Puckett et al., 2000). Among these chemicals are mercury, lead oxide, cadmium, arsenic and polyvinyl chloride, which are known to be carcinogenic, and listed as restricted hazardous substances in WEEE. At the molecular level, heavy metals are capable of activating cells and triggering signaling pathways, induced by targeting a number of cellular regulatory proteins or signaling proteins participating in cell growth, apoptosis, cell cycle regulation, DNA repair mechanism, and cellular differentiation (Valko et al., 2005). The overall effects of this action are the loss of growth regulatory mechanisms in cells, initiating uncontrollable cell proliferation and autonomous cell growth, which is the hallmark of carcinogenesis.

Informal processing of electronic waste in developing countries may cause serious health and pollution problems, though these countries are also most likely to reuse and repair electronics. E-waste disposal is especially problematic when humans and the environment are exposed to hazardous chemicals during the process of dismantling electronic products. Some of the minute fallouts of e-wastes are trapped in dusts.

Humans can become exposed to heavy metals in dust through several routes which include ingestion, inhalation, and dermal absorption (Leung et al., 2006; Leung et al., 2008). In dusty environments, it has been estimated that adults could ingest up to 100 mg dust/day (Leung et al., 2008; Hawley, 1985; Calabrese, 1987). Exposure to high levels of heavy metals can result in acute and chronic toxicity, such as damage to central and peripheral nervous systems, blood composition, lungs, kidneys, liver, and even death (Llobet et al., 2003).

There is a serious association between heavy metal and chemical toxicants, and high cancer risks. China is known to be the largest e-waste dump yard in the world. The emergence of China Cancer villages is well documented (Hong, 2005; Liu and Chen, 2007 and Yingying, 2011), and this may not be not unconnected with the reported heavy chemical pollution, including toxicants in e-wastes. The challenges involved in the operation of environmental laws in China have been reported as a contributory factor to the emergence of China Cancer Villages (Liu and Chen, 2007).

With the unregulated heavy inflow of e-waste into Nigeria, coupled with other widespread health-threatening pollutions, there is increasing possibility that similar health challenges could begin to develop. The present study was aimed to evaluate the blood levels of toxic metals (Hg, As, Cr, Cd and Pb) in Nigerians occupationally exposed to e-waste and in non-exposed participants in Benin City, South-South Nigeria.

PARTICIPANTS AND METHODOLOGY

Study Design and Study Area

This study was designed as a comparative study between occupationally exposed and non-exposed groups. The study was carried out in the Metropolitan City of Benin, Edo State located in South-south Nigeria. Benin City is the capital of Edo State, Nigeria.

Exposed Group

Male Waste Electric and Electronic Equipment (WEEE) Workers (n=63, Mean age of 31 years), working and living in Benin City, formed the exposed group. The states of origin of the exposed subjects comprised of Edo, n=32 (50.8%); Imo, n=15 (23.8%); Delta, n=7 (11.1%); Anambra, n=3 (4.8%); Ekiti, n=2; (3.2%); Enugu, n=2 (3.2%) and Abia, n=2 (3.2%). Only subjects with a minimum of 5 years of occupational exposure to toxic substances in WEEE were enrolled into the study.

Non-exposed Group

Age-matched apparently healthy male participants (n=41), with minimal or no occupational exposure to toxic substances in WEEE, recruited from the Ugbowo Campus Community of the University of Benin formed the non-exposed group in this study.

Inclusion Criteria

(a) The exposed subjects comprised of Electronic Technicians carrying out informal (primitive) e-waste recycling, processing, repair and dismantling repair of
electronic and electrical equipment. Subjects who were occupationally exposed to e-waste for a period of five years and above at the time of sample collection were considered suitable for the study. The five years duration of exposure used in this study is based on E-waste Risk Assessment Report of Adaramodu et al. (2012).

(b) Control subjects were healthy male individuals with minimal or no occupational exposure and with no hobby involving e-waste exposure. The non-exposed participants had no previous demographic and medical history of incidence of cancer.

Exclusion Criteria

E-waste workers who are not exposed to e-waste for a period up to five years at the time of sample collection were considered suitable for the study. Subject with history of any form of cancer, tobacco smoking and alcoholism were excluded from the study. Tobacco smoking and alcohol consumption also served as basis of exclusion for recruiting the apparently healthy control subjects.

Ethical Approval

The protocol for this study was approved by the Health Research Ethics Committee of University of Ibadan/University College Hospital, Ibadan, Nigeria, with a reference number UI/UCH EC: NHREC/05/01/2008a.

Informed Consent

Subjects for this study were adults who were adequately briefed on the research protocol and informed consent was obtained prior to sample collection. The informed consent form used for this study was explicitly explained to the participants in English and in their native language.

Sample Collection

Approximately ten millilitres of venous blood was collected from test subjects (e-waste workers) and control subjects using standard phlebotomy techniques. Blood samples obtained were dispensed into EDTA anticoagulant specimen bottles until time of analysis. Another five millilitres was dispensed into anticoagulant-free bottles to obtain serum. Information about exposure burden was obtained by means of questionnaires provided to the participants of the study.

Laboratory Analysis

The determination of the metal contents of the blood samples was carried out at the International Institute for Tropical Agriculture (IITA), Ibadan, Nigeria. Concentrations of Cadmium, Chromium, Lead, and Arsenic in the samples were estimated by Electrothermal Atomic Absorption Spectrometry, adopting the methods of Olmedo et al. (2010) and Mercury concentration was estimated by Inductively Coupled Plasma Mass Spectrometry adopting the methods of Fong et al. (2007).

Statistical Analysis

Statistical analyses including descriptive statistics shall be carried out using the Statistical Package for Social Scientists (SPSS) version 16.0. All values were expressed as Mean ± Standard Error of the Mean. The Independent Student’s t-test was used to determine significant differences between exposed and unexposed groups and p value < 0.05 was accepted.

RESULTS

Table 1 below shows the pattern of occupational exposure of e-waste exposed workers and compared non-exposed participants in Benin City, Edo State, Nigeria.

Frequency of exposure to e-waste of greater than or equal to 6 days per week, and exposure routes such as hands, eyes, nasal cavity, and oral cavity, and through dermal absorption among the e-waste workers studied. In addition, a majority (88.9%) of the e-waste workers does not use basic protective devices such as apron, hand gloves and facemasks while working.

Table 2 shows the blood levels of the toxic metals in the test and control groups. The results show statistically significant elevated body burden of toxic metals in e-waste workers (Pb, 0.95 ± 0.00 µmol/L; Cr, 405.99 ± 6.34 µmol/L; Cd, 108.54 ± 1.60 nmol/L; As, 10.09 ± 0.01 µmol/L; and Hg, 25.02 ± 0.14 nmol/L) compared with non-exposed group (Pb, 0.03 ± 0.00 µmol/L; Cr, 178.44 ± 5.99 µmol/L; Cd, 56.99 ± 1.42 nmol/L; As, 1.02 ± 0.0008 µmol/L; and Hg, 1.62 ± 0.0672 nmol/L).

DISCUSSION

In this study, we have presented data that indicate a high degree of occupational exposure to toxic e-waste chemicals. Our findings revealed that this exposure is particularly due to the occupational lifestyle of the studied e-waste workers. The majority (88.9%) of the e-waste workers (with exposure burden of ≥ 6 hours per day; ≥6 days per week) work without basic protective devices such as apron, hand gloves and face masks, a practice that facilitates exposure to WEEE-borne toxic and carcinogenic metals and chemicals through almost all body cavities.

The pattern of exposure revealed by this study reflects the significantly higher body burden of toxic metals in the e-waste exposed participants compared with non-exposed population. Our data appear to provide sufficient evidence that there is a systemic build-up of toxic metals in the exposed population, and that this may contribute to synergistic toxicity as a predisposing factor to chronic, acute and sub-acute pathologic conditions associated with heavy metal toxicity.
Micronutrients deficiency has been reported in developing countries. Nutritionally, some metals, such as copper, zinc, selenium and iron, are essential to human health and play critical roles and specific functions, for example, in the effective functioning of enzyme systems catalyzing various biochemical reactions in vivo. Conversely, metals such as beryllium, gold and silver are regarded as xenobiotic, since they serve no useful functions in human health physiology. Other metals such as lead, mercury, cadmium, arsenic, and hexavalent chromium are toxic even at low concentrations (Llobet et al., 2003).

Cadmium

Cadmium has been reported by several authors to be nephrotoxic (Squibb and Fowler, 1984; Goyer et al., 2004). One of the mechanisms involved in cadmium induced renal damage includes the formation of cadmium-metallothionein complex that filters in the proximal tubules and eventually degrades in the kidney, leading to the accumulation of cadmium in the kidney. The accumulated cadmium impairs kidney functions, resulting in renal excretion of calcium and low molecular weight proteins such as albumin and beta2-microglobulin (Roels et al., 1982; Hu, 2002). Adams et al.
(1979) observed that once proteinuria had been established, it persist and progresses even after a change in occupational exposure. Furthermore, cadmium interferes with bone metabolism and calcium transport through the mechanism of molecular mimicry (Hu, 2002). The result of this is bone resorption and softness of bone tissue, making the bones prone to fracture. Cadmium has a cumulative effect and long term storage in the body, with an average half-life of 17 to 30 years (Alissa and Ferns, 2011), a characteristic feature that further potentiates its ability to exert its toxic effects on target organs. More so, cadmium through its antagonistic effects on copper and iron in antioxidant enzymes such as superoxide dismutase, can mediate the generation of free radical species through the Fenton reaction, which can cause oxidative stress and DNA damage (Valko et al., 2005). These considerations suggest that e-waste workers with elevated cadmium levels as observed in this study may face the risk of developing pathologic conditions associated with cadmium toxicity such as cancer (Wafees, 2004), in addition to renal impairment.

**Mercury**

Mercury, which is also WEEE-borne, is known to be toxic through different mechanisms. All forms of mercury have toxic effects in a number of organs, especially in the kidneys (Zalups, 2000), and the central nervous system is particularly susceptible to mercury toxicity (Neustadt and Pieczenik, 2007). The high body burden in e-waste workers observed in this study is a reflection of occupational exposure. Toxicity with mercury was reported to be the cause of Minamata disease, which is a neurological condition first reported in Japan in 1950s (Takizawa, 1979). At lower but more chronic levels of exposure, mercury can cause tremor of the hands, excitability, memory loss, insomnia, and timidity amongst others. The levels found in this report suggests that these events may be replicated in the WEEE workers.

Transport mechanisms of methylmercury result in systemic distribution, which explains its high rate of deposition in both hematopoietic and neural tissues (Zalups, 2000). Inorganic mercury has been found to affect calcium homeostasis (Chavez and Holguin, 1988). Mercury is also known to be involved in metal induced oxidative stress. Copper is a cofactor for super oxide dismutase (SOD), unavailability of copper causes decrease SOD activity. Involvement of mercury (a component of e-waste) in oxidative stress may be due to its ability to displace copper from its binding site. It has been suggested that mercury increases intracellular copper only by increasing influx from extracellular medium, which could particularly increase oxidative stress (Sarafian, 1999; Boveris et al., 1972).

Mercury may also affect the biological role of copper through metal-metal interaction. Metal ions play an important role in biological systems, and without their catalytic presence in trace or ultratrace amounts many essential co-factors for many biochemical reactions would not take place. However, they become toxic to cells when their concentrations surpass certain optimal (natural) levels. Copper is an essential metal. Catalytic copper, because of its mobilization and redox activity, is believed to play a central role in the formation of reactive oxygen species (ROS), such as $O^2-$ and $\cdot OH$ radicals, that bind very fast to DNA, and produce damage by breaking the DNA strands or modifying the bases and/or deoxyribose leading to carcinogenesis. Mercury-mediated oxidative stress and metal-metal interaction (Hg-Cu) may potent deleterious biochemical changes in the Nigerian e-waste exposed population studied.

**Arsenic**

Arsenic as a known carcinogen is well-documented in a number of studies. Exposure to arsenic is linked with a risk of developing tumours of the lung, skin, liver, bladder, and kidney (Waalkes et al., 2004). Chronic arsenic exposure also causes a markedly elevated risk for developing a number of cancers, most notably skin cancer, cancers of the liver (angioscarcoma), lung, bladder, and possibly the kidney and colon (Hu, 2002). Arsenic interferes with mitochondrial ATP production. Once absorbed into the body, it is rapidly redistributed into body tissues. After its distribution, arsenic penetrates into cells and inhibits cellular energy production through mechanisms dependent on the element’s oxidation state. Arsenate has the same oxidation state as inorganic phosphate (p$^{5+}$) and shares many chemical properties with it and can substitute for p$^{5+}$ in glycolysis. Arsenic-poisoned enzymes produce 1-arseno-3-phosphoglycerate rather than the usual 1, 3-bisphosphoglycerate. 1-Arseno-3-phosphoglycerate is unstable and hydrolyzes spontaneously. Glycolysis proceeds so that ATP continues to be produced. However, the arsenic-oxygen bond is significantly weaker than the phosphorus-oxygen bond, and 1-arseno-3-phosphoglycerate hydrolysis yields dramatically less energy. Arsenate can also inhibit the conversion of pyruvate (by poisoning the enzyme complex, pyruvate dehydrogenase) into acetyl-CoA, blocking the Krebs cycle and therefore resulting in further loss of ATP (Lai et al., 2005). Arsenic is known not only to generate reactive oxygen species (ROS) but reactive nitrogen species (RNS) through the damage of lipid membranes and DNA (Valko et al., 2005). The generated reactive oxygen species, causes alterations in the signal cascade and an imbalance in antioxidant levels, in turn triggers cellular apoptosis in cells (Flora et al., 2008). Thus, WEEE-borne arsenic may establish arsenic-induced alteration in energy metabolism, carcinogenesis and apoptotic changes in e-waste exposed populations.

**Lead**

Lead is a major component of e-waste and is often used by WEEE workers in soldering. Lead is a cumulative toxicant that affects multiple body systems, including the neurological,
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