Vol. 26 (4) 107–113 *31 December 2014*

Biokemistri

An International Journal of the Nigerian Society for Experimental Biology

Original Research

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

Godwin O. Igharo[1,2], John I. Anetor[2], Oladele O. Osibanjo[3], Humphrey B. Osadolor[1] and Kingsley C. Dike[1]

[1]Department of Medical Laboratory Sciences, School of Basic Medical Sciences, College of Medical Sciences, University of Benin, Benin City, Nigeria.

[2]Toxicology and Micronutrient Metabolism Unit, Department of Chemical Pathology, College of Medicine, University of Ibadan, Ibadan, Nigeria.

[3]Basel Convention Coordinating Centre for Training & Technology Transfer for the African Region & Department of Chemistry University of Ibadan, Nigeria.

*Corresponding author: Godwin O. Igharo. E-mail: godwingreatman@yahoo.com; +2348038664896

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, (Aragba-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 sulfhydryl homeostasis (Valko *et al.*, 2005). E-waste contains well 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 trigger 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.

Table 1: Pattern of Occupational Exposure of Electronic Waste Exposed Workers and Non-exposed Participants in Benin City, South-South Nigeria

Observation	Exposed Subjects (n=63) Median=31 Years	Non–exposed Subjects (n=41; Median=29 Years)
Duration of Exposure to E-waste Chemicals	≥5.0 years	No occupational exposure
Frequency of Exposure to E-waste Chemicals	≥6 days/week	Nil
Routes of Exposure: Direct	Hands, eyes, nasal cavity, oral cavity, dermal absorption	Nil
Indirect	Environmental (high)	Minimal
% Using Protective Device while Working	Yes { (n = 7) 11.1%} No { (n = 56) 88.9%}	Not Applicable
Specified Protective Devices Used by E-Waste Workers	Apron	Not Applicable

Table 2: Comparison of Blood Levels of Toxic Metals in Nigerian E- Waste Exposed Workers and Non-exposed Participants in Benin City, South-South Nigeria

Toxic Metals	Mean ± SEM		t value	P value	Level of Significance
	Exposed subjects (n = 63)	Non-exposed subjects (n = 41)			
Lead (μmol/L)	0.95 ± 0.00	0.03 ± 0.00	19.26	0.000	Significant
Chromium (nmol/L)	405.99 ± 6.34	178.44 ± 5.99	24.51	0.000	Significant
Cadmium (nmol/L)	108.54 ± 1.6028	56.99 ± 1.4171	2.33	0.000	Significant
Arsenic (μmol/L)	10.09 ± 0.0058	1.02 ± 0.0008	8.47	0.000	Significant
Mercury (nmol/L)	25.02 ± 0.1385	1.62 ± 0.0672	18.67	0.000	Significant

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 Fowlert, 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 beta$_2$-microglobulin (Roels *et al*., 1982; Hu, 2002). Adams *et al*.

(1979) observed that once proteinuria have 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 (Walfees, 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 Minemeta 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 *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 portent 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 (angiosarcoma), 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,

haematological, gastrointestinal, cardiovascular, immune and renal systems, (Wąsowicz *et al*., 2001; Dietert *et al*., 2004). The higher lead level observed in this study may portend risks of lead related pathophysiological disorders in the exposed participants. Lead is known to induce renal tumours, reduce cognitive development, and increase blood pressure and cardiovascular diseases risk for adults (Harmanescu *et al.*, 2011). At the gastrointestinal level, the absorption and distribution of lead is dependent on the nutritional status of an individual. In the presence of micronutrients such as calcium and iron, the absorption of lead will be markedly decreased due to competitive binding to transport mechanism (Hennig *et al.*, 2007; Hernandez-Avila *et al*., 2003). Lead has critical effects on haem and haemoglobin synthesis and changes in the morphology and survival of red blood cells (Flora *et al*., 2008). Activities of aminolevulinic acid dehydratase (ALAD), a cytosolic sulfhydryl enzyme in this pathway is most sensitive to lead insult. Lead also inhibits the activities of ferrochelatase in the last step of heme synthesis. The overall effects are the development of anaemia and decreased haematocrit. These events result in significant elevation of ALAD level and its accumulateion in the blood. This further results in potentiatiating the toxic effects of lead (Ercal *et al*., 2001). Nigerian e-waste workers may be at risk of these toxic effects of lead.

Chromium

Our analyses revealed disturbing levels of hexavalent chromium in the exposed group. Trivalent (Cr(III)) and hexavalent (Cr(VI)) chromium are thought to be the most biologically significant compounds of chromium. Cr(III) is an essential dietary mineral in low doses. Chromium Cr(VI) is toxic even at low concentration. Chromium carcinogenicity was first identified over a century ago and Cr(VI) compounds were among the earliest chemicals to be classified as carcinogens (Stern, 1982). Through the mechanisms of biological reductants such glutathione, ascorbate and lipoic acid; and generation of free radicals involving the Fenton reaction and activation of transcription factors, chromium can cause various degree of DNA damage and exert it's genotoxic effects (Valko *et al.,* 2005). Renal and hepatic damage by chromium have also been reported as well as asthmatic conditions (Bright *et al*., 1997).

Conclusion

The findings from this study lead to the conclusion 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. Our data provides evidence that there is a systemic build-up of toxic metals in the exposed population, and that this may establish synergistic toxicity as a predisposing factor to chronic, acute and sub-acute pathologic conditions associated with heavy metal toxicity.

Conflict of Interest: The authors declare that no conflict of interest in this work.

Acknowledgement: The authors acknowledge TETFUND in support of this work.

REFERENCES

Adams RG (1979) Clinical and Biochemical Observations in Men with Cadmium Nephropathy: A Twenty-year study. *Archives of Industrial Hygiene and Toxicology* 30: 219–321.

Adaramodu AA, Osuntogun BA, Ehi-Eromosele CO (2012) Heavy Metal Concentration of Surface Dust Present in E-waste Components: The Westminister Electronic Market, Lagos Case Study. *ARPN Journal of Science and Technology* 2 (Special Issue) 247–270.

Aginam E (2010) Nigeria: E-waste Concern Re-Echoes at Clean Environment Forum Vanguard (Aug.17, 2010), available at http://allafrica.com/stories/2010008180100.html Accessed 10/02/2013

Alissa EM, Ferns AG (2011) Heavy Metal Poisoning and Cardiovascular Disease. *Journal of Toxicology* 5: 101–121.

Anetor JI, Iyanda AA, Akinseye I, Anetor GO (2013) Strengthening Analytical Capability in Micronutrients: A Prophylactic Approach to DNA Repair Defects, Genome Instability and Carcinogenesis in Developing Countries. Annals de Biologic Clinique 17: 560–561.

Aragba-Akpore S (2005) Red Alert on used Computers, Electronic Devices. The Guardian (Dec. 27, 2005), available at "http://www.ban.org/ban_news/2005/05/227_nigeria.html" http://www.ban.org/ban_news/2005/05/227_nigeria.html. Accessed 18/7/2013.

Basel (2004) Technical guidelines on the environmentally sound recycling/reclamation of metals and metal compounds (R4). Conference of the Parties to the Basel Convention on the Control of Trans-boundary Movements of Hazardous Wastes and Their Disposal, UNEP/CHW.7/8/Add.3.Available at www.basel.int/meetings/cop/cop7/docs/08a3e.pdf. Accessed 18/7/2013

Bittner AC, Echeverria D, Woods JS, Aposhian HV, Naleway C, Martin MD (1998) Behavioral effects of low-level exposure to Hgo among dental professionals: A cross study evaluation of psychomotor effects. *Neurotoxicology and Teratology* 20: 429–439.

Boveris A, Oshino N, Chance B (1972) The cellular production of hydrogen peroxide. *Biochemical Journal* 178: 617-620.

Bright P, Burge PS, O'Hickey SP, Ganon PF, Robertson AS, Boran A (1997) Occupational asthma due to chrome and nickel electroplating *Journal of Occupational Medicine* 52: 28–32.

Calabrese EJ, Kostecki PT, Gilbert CE (1987) How much dirt do children eat? An emerging environmental health question Comment. *Toxicology* 1: 229–241.

Chavez E, Holguin JA (1988) Mitochondrial calcium release as induced by Hg^{2+}. *Journal of Biological Chemistry* 263: 3582–3587.

Dietert RR, Lee JE, Hussain I, Piepenbrink M (2004) Developmental immunotoxicology of lead. *Toxicology and Applied Pharmacology* 198: 86–94.

Elst P, Akankour F, Tjalma W (2007) Management of recurrent cervical Cancer. Review of the literature and case report. *European Journal of Gynaecology and Oncology* 28: 435–441.

Ercal N, Gurer-Orphan H, Aykin-Burns N (2001) Toxic metals and oxidative stress part I: Mechanism involved in metal induced

oxidative damage. *Current Topics in Medicinal Chemistry* 1: 529–539.

Flora SJS, Mittal M, Mehta A, (2008) Heavy metal induced oxidative stress & its possible reversal by chelation therapy. *Indian Journal of Medicine and Research* 128: 501–523.

Fong BMW, Siu TS, Lee JSK, Tam S (2007) Determination of Mercury in Whole Blood and Urine by Inductively Coupled Plasma Mass Spectrometry. *Journal of Analytical Toxicology* 31: 281–287

Goyer R, Golub M, Choudhury H, Hughes M, Kenyon E, Stifelman M (2004) Issue paper on the Human Health effects of Metals. U.S. Environmental Protection Agency Risk Assessment Forum 1200 Pennsylvania Avenue, NW Washington, DC

Harmanescu M, Alda LM, Bordean, DM, Gogoasa I, Gergen I (2011) Heavy metals health risk assessment for population via consumption of vegetables grown in old mining area; a case study: Banat County, Romania. *Chemistry Central Journal* 5: 64

Hawley JK (1985) Assessment of health risk from exposure to contaminated soil. *Risk Analysis* 5: 289–302

Hennig B, Ettinger AS, Jandacek RJ, Koo S, McClain C, Seifried H, Silverstone A, Watkins B, Suk WA (2007) Using Nutrition for Intervention and Prevention against Environmental Chemical Toxicity and Associated Diseases. *Environmental Health Perspective* 115: 493–495.

Hernandez-Avila M, Gonzalez-Cossio T, Hernandez-Avila JE, Romieu I, Peterson KE, Aro A, Palazuelos E, Hu H (2003) Dietary calcium supplements to lower blood lead levels in lactating women: a randomized placebo-controlled trial. *Epidemiology* 14: 206–212.

Hong Q (2005) Why Are There More and More Cancer Villages? *Environmental Economy* 58–59.

Hu H (2002) Human Health and Heavy Metal Exposure. In: Life Support: the Environment and Human Health. 4: 1–10.

Lai MW, Boyer EW, Kleinman ME, Rodig NM, Ewald MB (2005) Acute Arsenic Poisoning in Two Siblings. *Pediatrics* 116: 249–257.

Leung AO, Cai ZW, Wong MH (2006) Environmental Contamination from Electronic Waste Recycling at Guiyu, southeast China. *Journal of Material Cycles Waste Management* 8: 21–33.

Leung AW, Duzgoren-Aydin NS, Cheung KC, Wong MH (2008) Heavy metals concentrations of surface dust from e-waste recycling and its human health implications in Southeast China. *Environmental Science Technology* 42: 2674–2680.

Liu Q, Chen L (2007) On the Lack of Operation System of Environment Law of China in View of a Cancer Village. *Journal of Shiyan Technical Institute* 20: 18–20.

Llobet JM, Falco G, Casas C, Teixido A, Domingo JL (2003) Concentration of arsenic, cadmium, mercury, and lead in common foods and estimated daily intake by children, adolescents, adults, and seniors of Catalonia, Spain. *Journal of Agriculture and Food Chemistry* 51: 838–842.

Neustadt J, Pieczenik S (2007) Heavy-Metal Toxicity: With Emphasis on Mercury. *Integrative Medicine* 6: 26–32.

Olmedo P, Pla A, Hernández AF, López-Guarnido O, Gil LRF (2010) Validation of a method to quantify chromium, cadmium, manganese, nickel and lead in human whole blood, urine, saliva and hair samples by electrothermal atomic absorption spectrometry. *Analytica Chimica Acta* 659: 60–67.

Puckett J, Byster L, Westervelt S, Gutierrez R, Davis S, Hussain A, Dutta M (2000) Exporting Harm: The High-Tech Trashing of Asia. Basel Action Network. E-waste/technotrashfinalcom.pdf. Accessed 17/05/2013.

Roels H, Lauwerys, R, Buhet JP, Bernard A (1981) Environmental exposure to cadmium and renal function of aged women in three areas of Belgium. *Environmental Research* 24: 117–130

Sarafian TA, Verity MA, Vinters HV, Shih CC, Shi L, Ji XD, Dong L, Shau H (1999) Differential expression of peroxiredoxin subtypes in human brain cell types. Journal of Neuroscience Research 56: 206–212.

Squibb KS, Fowlert BA (1984) Intracellular Metbolism and Effects of Circulating Cadmiu-Meallothionein in the Kidney. *Environmental Health Perspectives* 54: 31–35

Stern RM in: S Langård (Ed.), Biological and Environmental Aspects of Chromium, Elsevier, Amsterdam, 1982, 5–47

Sthiannopkao S, Wong MH (2012) Handling e-waste in developed and developing countries: Initiatives, practices, and consequences. *Science Total Environment* http://www.sciencedirect.com/science/article/pii/S0048969712009217. Accessed June 2012.

Takizawa Y (1979) Minamata Disease in Japan. *Environmental Toxicology and Human Health* 1: 1–25

Terada C (2012) Recycling Electronic Wastes in Nigeria: Putting Environmental and Human Rights at Risk. *Northwestern Journal of International Human Rights* 10: 154–172

Valavanidis A, Vlachogiani T, Fiotakis C (2009) 8-hydroxy2-deoxyguanosine:A Critical Biomarker of Oxidative Stress and Carcinogenesis. *Journal of Environmental Science and Health* Part C, 27: 120–139.

Valko M, Morris H, Cronin MTD (2005) Metals, Toxicity and Oxidative Stress. *Current Medicinal Chemistry* 12: 1161–1208.

Waalkes MP, Ward JM, Diwan BA (2004) Induction of tumors of the liver, lung, ovary and adrenal in adult mice after brief maternal gestational exposure to inorganic arsenic: Promotional effects of postnatal phorbol ester exposure on hepatic and pulmonary, but not dermal cancers. *Carcinogenesis* 25: 133–141.

Wąsowicz W, Gromadzińska J, Rydzyński K (2001) Blood Concentration of essential Trace Elements and Heavy Metals in Workers Exposed to Lead and Cadmium. *International Journal of Occupational Medicine and Environmental Health* 14: 223–229.

Yinying Z (2011) The Shadow over Rural China. Chinadialogue,10/02/2011; available at http:// www.shadow over rural chinaliulican on china's cancer villiages.chinadialogue.htm. Accessed July 2, 2013.

Zalups RK (2000) Molecular interactions with mercury in the kidney *Phamacological Review* 52: 113–143.