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ORIGINAL ARTICLE

Exposure assessment and micronuclei induction in populations exposed to electronic waste in South-West Nigeria

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ABSTRACT

BACKGROUND
Informal electronic waste (e-waste) reprocessing in Nigeria is reportedly substantial in Africa, putting the growing exposed population at high risk of metal toxicity. This study aimed to investigate the existence of chromosomal aberration in the growing e-waste exposed populations in Nigeria, using induction of micronuclei (MN) expression in peripheral blood as an indicator.

METHODS
In this cross-sectional study, 632 consenting participants were recruited from South-West Nigeria, consisting of 381 e-waste workers (EWW), 120 environmental e-waste exposed participants (EEP) and 131 age-matched unexposed participants (UP) serving as controls. A validated structured questionnaire was used to assess exposure pattern while frequency of micronucleated polychromatic erythrocytes (MNPCE)/1000PCE in peripheral blood film was determined by modified micronucleus assay.

RESULTS
A duration of exposure of ≥5 years and exposure frequency ≥ 6 hours/day; 6 days/week (9360 hours in any 5-year duration) was observed in both EWW and EEP. Routes of exposure observed in EWW entailed eyes, oral cavity, nasal cavity and skin. EWW that used personal protective equipment (PPE) while working was barely 10.24% while non-PPE users constituted the majority (89.76%) of the studied population. Frequency of MNPCE)/1000PCE in EWW (22.70 ± 0.15) was significantly higher than in EEP (4.17 ± 0.28), which in turn was significantly higher than the lowest frequency (0.99 ± 0.76) observed in UP ($p<0.001$).

CONCLUSION
The observed exposure pattern and the comparatively higher MN induction in the e-waste populations may suggest risk of significant cytogenetic damage and aberrant chromosomal changes associated with occupational e-waste reprocessing in Nigeria.

Keywords: E-waste, micronuclei induction, chromosomal aberration, metal toxicity, exposure pattern

INTRODUCTION

The growth in technological and electronics industries and the consequent increase in production have increased the rate of e-waste generation. Electronic and electrical waste (e-waste), also referred to as waste electrical and electronic equipment (WEEE), is defined as any end-of-life equipment, which is dependent on electrical currents or electromagnetic fields in order to work properly. Included in this definition are small and large household appliances; information technology and telecommunications equipment; lighting equipment; electrical and electronic tools, toys, leisure and sports equipment; medical devices; monitoring and control instruments; and automatic dispensers. Components of electrical and electronic equipment such as batteries, circuit boards, plastic casings, cathode-ray tubes, activated glass and capacitors are also classified as e-waste. [1]

Electronic waste recycling operations have been identified in several locations in China and India. Less investigated locations are in the Philippines, Nigeria (in the city of Lagos), Pakistan (Karachi) and Ghana (Accra).[2] Electronic waste is poorly managed in Nigeria and control of e-waste is inadequate. There has been insufficient enforcement of environmental laws and difficulties in implementing extended producer responsibility (EPR) and producer take-back, together with a general lack of awareness and funds. With no material recovery facility for e-waste and/or appropriate solid waste management infrastructure in place, waste materials often end up in open dumps and unlined landfills.[3] Potentially hazardous chemical elements are the metal components of electrical and electronic equipment, the most common being lead, cadmium, chromium, mercury, copper, manganese, nickel, arsenic, zinc, iron and aluminium.[4]

Heavy metals have many ways by which they cause their pathological effects, these include oxidative stress,[5,6] lipid peroxidation,[5-8] metal-metal interaction.[9] enzyme inhibition,[5,8,10] DNA binding [11] and damage of antioxidant system.[5,12]

The common mode of action for metal-induced carcinogenicity is summarized as follows. Induction of oxidative stress and damage to cellular components, particularly DNA; interference with DNA repair systems, resulting in genomic instability; and interruption of cell growth and proliferation via signalling pathways and dysregulation of oncogenes or tumour suppressor genes such as p53.[13] Induction of oxidative stress is a remarkable phenomenon to explain metal-induced genotoxicity and mutagenicity. Several carcinogenic metals such as arsenic, cobalt, chromium, lead, mercury and nickel induce redox reactions in living systems. These metals induce the production of reactive oxygen species (ROS) (e.g., hydroxyl peroxide and superoxide radicals) and reactive nitrogen species (RNS) (e.g., nitric oxide, peroxynitrite and S-nitrosothiols) in both *in vivo* and *in vitro* systems.[14] These radicals can in turn oxidise biomolecules including DNA and cause breakage, hence mutation and ultimately carcinogenesis.

Genome instability is the occurrence of a high frequency of mutations within the genome of a cell lineage, which mutations can include changes in nucleic acid sequences and rearrangements in the chromosomes or aneuploidy that is central to carcinogenesis.[15]

A systematic review and meta-analysis suggest that occupational and non-occupational exposure to e-waste processing is associated with increased risk of DNA damage measured through MN assay and other types of DNA damage biomarkers.[16] However, more studies from other developing countries in Africa, Latin America, and South Asia are needed to confirm and increase the generalizability of these results.

The present study therefore aimed to investigate the existence and/or extent of chromosomal aberration in the growing e-waste exposed populations in Nigeria, using induction of micronuclei (MN) expression in peripheral blood as an indicator. This thematic area of research has

not been reported among Nigerian e-waste workers.

METHODS

Research design and study area

This was a cross-sectional study carried out from December 2014 to January 2022 in Nigeria in three urban cities (Lagos, Ibadan and Benin) that have been identified and reported as high impact locations for e-waste activities in Nigeria.[17,18]

Study participants

A total of 632 participants were enrolled into the study, viz. 381 e-waste workers; 120 environmental e-waste exposed participants and 131 age-matched unexposed individuals, serving as controls. The inclusion criteria were: (a) e-waste workers, comprising electronics technicians carrying out informal (crude) e-waste recycling, repair, dismantling, purchasing and resale of EEE. Workers who were occupationally exposed to e-waste for a period of five years and above at the time of sample collection were enrolled into the study. The five-year duration of exposure used in this study is based on the e-waste risk assessment report of Adaramodu et al.[19] suggesting that a five-year duration is sufficient for the health risks and effects of e-waste crude reprocessing to become apparent in exposed human populations; (b) environmentally exposed participants, comprising apparently healthy age- and sex-matched traders and non-e-waste workers who had been involved in work and business activities around the e-waste high impact areas for a minimum of five years, and as such were environmentally exposed to e-waste-borne toxicants (including metals), due to unregulated and environmentally unfriendly WEEE disposal practices previously identified;[18,20] (c) control subjects who were healthy male individuals with minimal or no occupational exposure and with no hobby involving e-waste exposure or other toxic substances.

The exclusion criteria were e-waste workers who were not exposed to e-waste for a period up to five years at the time of sample collection and persons with demographic or medical history of any form of cancer, frequent/habitual tobacco smoking and alcohol consumption. Demographic and medical history of incidence of cancer, tobacco smoking and alcohol consumption also served as a basis for the exclusion of apparently healthy controls.

Collection of data

A twenty-item questionnaire was validated and administered to consenting participants to obtain socio-demographic information and occupational exposure pattern.

Sample collection

Approximately 5 mL of venous blood was collected from test subjects (e-waste workers) and control subjects using standard phlebotomy techniques. Blood samples obtained were dispensed into ethylene diamine tetra acetic acid (EDTA) anticoagulant specimen bottles (5 mL). Samples were immediately analysed for the research parameters and where this was not possible, the EDTA blood specimens were stored in the refrigerator (2 to 8 ^{o}C). All samples for the different groups were properly labelled to avoid sample mix-up. All samples were handled using safety gloves and protective lab clothing. Sodium hypochlorite (10%) solution was used to disinfect working areas after analysis. It was ensured that all handling and analysis of samples took place in approved working areas only.

Peripheral blood micronucleus assay

Genotoxicity effect was evaluated using the micronucleus test described by Holland et al. [21] with some modifications. In short, a small drop of peripheral blood was collected over a clean coded slide, a thin smear was made and air-dried in a clean environment for 24hrs and thereafter fixed in absolute methanol for 5 mins. The slides were then stained with May-Gruenwald stain and rinsed in distilled water. The rinsed slides were then stained with 5% Giemsa solution, rinsed properly and dried at room temperature. Slides were mounted with DPX mountant, dried (20-30°C) and cleaned. The frequencies of micronucleated polychromatic erythrocytes (MNPCEs) were estimated by scoring 1000 PCEs per slide.

Statistical analysis

Data collected were analysed using the Statistical Package for Social Sciences (SPSS) version 21.0 (IBM Inc., USA). The analysis of variance (ANOVA) was used to compare means and results were expressed in mean ± standard error of the mean. A p-value of less than 0.05 ($p<0.05$) was considered significant.

Ethical consideration

The protocol for this study was approved by the Health Research Ethics Committee of University of Ibadan/University College Hospital, Ibadan, Nigeria, with reference number UI/UCH EC: NHREC/05/01/2008a**.** Participants for this study were adults who were adequately informed on the benefits of the study and sufficiently briefed on the research protocol, with informed consent being obtained from them prior to sample collection.

RESULTS

Demographic characteristics and anthropometric indices of the study groups of e-waste workers, environmentally exposed participants and the unexposed controls are shown in Table 1 below. From the observations, mean age in the three study groups was not significantly different. Sex frequency indicated that 100% of the participants in each group were males and states of origin implied Nigeria as the nationality of all participants. Alcohol and tobacco use was considerably similar across the three groups of participants. Mean systolic blood pressure did not significantly vary across the three groups ($p>0.05$), diastolic blood pressure in environmentally exposed participants (86.21 ± 1.98 mmHg) was however significantly higher than in the e-waste workers (75.53 ± 1.77 mmHg) and in the unexposed group (70.34 ± 1.67 mmHg), ($p=0.044$). Nutritional indicators show the inclusion of carbohydrate, protein, fat and oil (CPFO) in daily staple food, in addition to observed intake of vitamin-rich supplements among e-waste workers (43.31%); environmentally exposed group (46.67%); and unexposed group (48.85%).

Table 1. Demographic characteristics and baseline health indices of electronic waste exposed and unexposed participants in all study locations

Variables	E-waste workers (n=381)	Environmentally exposed participants (n=120)	Unexposed (controls) (n=131)	p value
Age (years)	37.83 ± 1.37*	34.60 ± 1.10*	35.43 ± 1.77*	0.069
Sex (Frequency)				
Male	381 (100.00)	120 (100.00)	131 (100.00)	
Nationality				
Nigeria	381 (100.00)	120 (100.00)	131 (100.00)	
Tobacco use				
Occasional	81 (21.26)	29 (24.17)	35 (26.72)	0.413
Passive users	300 (78.74)	91 (75.83)	96 (73.28)	
Alcohol use				
Occasional	73 (19.16)	24 (20)	29 (22.14)	0.388
Non-users	308 (80.84)	96 (80)	102 (77.86)	
Blood Pressure (mmHg)				
Systolic	125.23 ± 2.15	116.32 ± 2.02	119.84 ± 1.90	0.082
Diastolic	75.53^{B}± 1.77	86.21^{A} ± 1.98	70.34^{B} ± 1.67	0.054
Nutritional Indicators				
Inclusion of $^{\partial}$ CPFO in Daily Staple Food				
Yes				
No				
Intake of Supplements	381 (100)	120 (100)	131 (100)	
(Vitamin-rich Drugs)	0 (0)	0 (0)	0 (0)	
Yes	165 (43.31)	56 (46.67)	64 (48.85)	0.506
No	216 (56.69)	64 (53.33)	67 (51.15)	
Specified Supplements	Iron-rich Supplements Vitamin-rich Supplements (Vitamin C)	Vitamin-rich Supplements (Multivitamins)	Vitamin-rich Supplements (Multivitamins)	

Data presented as n (%), except for age, systolic and diastolic blood pressure as Mean ± SD
$^{\partial}$CPFO: carbohydrates, proteins, fats and oil

Occupational and environmental WEEE exposures were high for e-waste workers and moderate for the environmentally exposed group. However, environmental WEEE exposure was minimal for the unexposed group, while occupational exposure was not indicated (Table 2). A duration of exposure of ≥5 years and exposure frequency ≥6 hours/day; 6 days/week (9360 hours in any 5-year duration) was observed with both e-waste workers and the environmentally exposed group. Routes of exposure observed in e-waste workers entailed all body organs, viz, eyes, oral cavity, nasal cavity, and skin (dermal absorption). These observations were similar in EEPs, apart from the exposure through the oral cavity which was significantly lower.

In addition, the proportion of e-waste workers that used PPE such as aprons, hand gloves and facemasks while working was barely 10.24% while non-PPE users constituted the majority (89.76%) of the studied population. Among the PPE users, 10.24% used aprons and scarcely used hand gloves, while the rest of the workers used neither face masks nor nose masks in the course of performing daily work tasks.

The frequency of micronucleated polychromatic erythrocytes (MnPCE)/1000PCE in e-waste workers (22.70 ± 0.15) was significantly higher compared with the environmentally exposed participants (4.17 ± 0.28), which in turn was significantly higher than the lowest frequency (0.99 ± 0.76) observed in the unexposed population ($p<0.001$). The maximum MnPCE/1000PCE observed in the participants' groups were: e-waste workers, 34; environmentally exposed, 7; and unexposed, 3; while the minimum MnPCE/1000 PCE were: e-waste workers 15, environmentally exposed 1, and unexposed 0 (Figure 1).

Table 2. Occupational and environmental exposure pattern of study participants

Observation	**E-waste workers (n=381)**	**Environmentally exposed participants (n=120)**	**Unexposed (controls) (n=131)**
Nature of exposure	Occupational / Environmental	Environmental	No occupational exposure
Duration of Exposure to E-waste Chemicals	≥ 5.0 years	≥ 5.0 years	Nil
Frequency of Exposure to E-waste Chemicals	≥ 6 hours/day; 6 days/week (9360 hours in 5 years)	≥ 6 hours/day; 6 days/week (9360 hours in 5 years)	Nil
Medium of Exposure:			
Direct	Hands, eyes, oral cavity, nasal cavity, dermal absorption	Hands, nasal cavity, dermal absorption	Nil
Indirect	Environmental (high)	Environmental (moderate)	Environmental (Minimal)
% Using PPE while Working	Users [(n=39) 10.24 %] Non-users [(n=342) 89.76%]	Not Applicable	Not Applicable
Specified Protective Devices Used by E-Waste Workers	Apron (10.24%) Hand gloves (scarcely) Face/Nose mask (Nil)	Not Applicable	Not Applicable

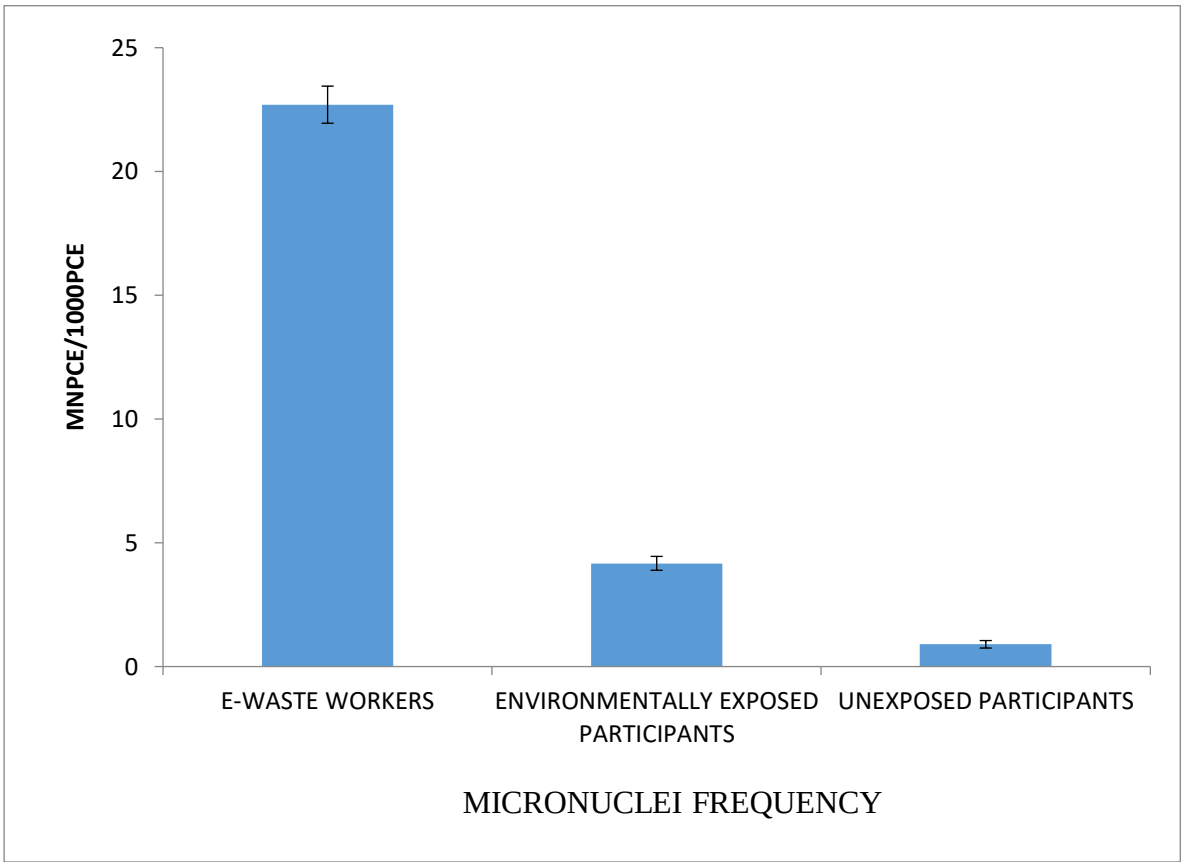

Figure 1. Frequency of MnPCE/1000PCE in e-waste exposed and unexposed participants

DISCUSSION

Waste electrical and electronic equipment (WEEE) have been reported to contain more than one thousand toxic chemicals.[22] Sources of exposure to e-waste have been classified into three sectors, namely informal recycling, formal recycling, and exposure to hazardous e-waste compounds remaining in the environment[22] Exposure through informal and formal recycling can be considered as occupational exposure to hazardous chemicals present in WEEE, and the informal recycling (crude management) adequately describes the mode of exposure of the occupationally exposed participants enlisted for this study. As a fallout of this crude and environmentally unsound methods, the environment in the high e-waste-impact areas is not spared from contamination by the WEEE-borne toxicants,[22,23] a justification that necessitated the inclusion of environmentally exposed participants in this study.

The risk awareness indicators and exposure pattern analysis in this study implied that 248 (64.09%) of e-waste workers, 94 (78.3%) of environmentally exposed participants (EEPs), and 102 (77.86%) of unexposed controls lacked basic awareness of the health hazards associated with WEEE exposure. Among the 133 e-waste workers (34.91%) with basic health risk awareness, 6 (1.57%) specified catarrh/cough; 18 (4.72%) specified electrocution; while 17 participants (4.46%) stated impaired vision; 6 (1.57%) stated hypertension; and the largest proportion, 334 participants (87.68%) indicated uncertain responses as per condition known to them to be associated with WEEE exposure. This lack of basic awareness may be responsible for the workers' poor attitude to the use of personal protective equipment. Also, the non-inclusion of cancer as one of the conditions suspected or known to be associated with WEEE exposure is worrisome and calls for serious occupational health education and a study of this nature. This is in agreement with the high desire for the conduct of a health risk (toxicological) study on e-waste exposure among the participants: 95.28% of e-waste workers; 100% of the environmentally

exposed participants; and 100% of unexposed participants being desirous of the conduct of the study.

Of public importance is this study's observation on the methods of disposal of unwanted WEEE by e-waste workers, viz. the largest proportion (44.36%) is disposed of in the local refuse dump; while another 22.31% of the WEEE is handled by commercial waste managers. More so, 11.02% of the unwanted WEEE is sold to waste scavengers and 22.31% is disposed of through undisclosed methods. Overall, all listed methods of disposal are not technologically driven, and as such are environmentally unsafe and unfriendly. This has been previously copiously reported and also the alarm of the potential public health hazard raised in China [24] and Nigeria.[20, 23]

In addition, the proportion of e-waste workers that uses PPE such as aprons, hand gloves and facemasks while working was barely 10.24% while non-PPE users constituted the majority (89.76%) of the studied population. Among the PPE users, 10.24% used aprons and scarcely used hand gloves and the rest of the workers used neither face masks nor nose masks in the course of performing daily work tasks.

Our observation in the study suggests an occupational practice that enhances exposure to WEEE-borne toxic and potentially carcinogenic metals and chemicals through almost all body cavities, particularly due to the unsafe occupational lifestyle of e-waste workers which shows high level of primitiveness and near-zero safety practices. The outlook portrayed by these data corroborates the observations of Igharo et al.[25] in Benin, a single-location pilot work designed as a preliminary part of this study.

The MN test has been increasingly accepted as a reliable biomarker of genotoxicity in occupationally exposed groups,[15,26] and additionally, the mammalian *in vitro* micronucleus test has been used for the detection of damage induced by toxic test substances to chromosomes or the mitotic apparatus of erythroblasts by analysis of erythrocytes as sampled in the bone marrow and/or peripheral blood cells of animals.[15, 27]

In the present study, the observed significant increase in the frequencies of MNPCEs in both occupational and environmental e-waste exposed populations may suggest elevated induction of chromosomal damage that could be associated with chronic exposure to e-waste-borne genotoxic metals. Our previous study has shown elevated blood levels of key toxic metal levels in Nigerian electronic waste workers.[8]

The formation of micronuclei and binuclei in fish cells caused by their exposure to cadmium, copper and chromium was reported by Garai et al.,[28] thus verifying that heavy metals have cytotoxic and genotoxic effects. Micronuclei (MN) are extra-nuclear bodies that contain damaged chromosome fragments and/or whole chromosomes that were not incorporated into the nucleus after cell division. MN can be induced by defects in the cell repair machinery and accumulation of DNA damage and chromosomal aberrations. A variety of genotoxic agents may induce MN formation leading to cell death, genomic instability or cancer development.[15,16]

Elevated frequencies of micronuclei and other nuclear abnormalities in chrome plating workers occupationally exposed to hexavalent chromium has been reported, and the contribution of tobacco smoking has been reported to be significant. Though smoking status significantly raised MN frequencies in hospital workers occupationally exposed to low levels of ionizing radiation,[29] the possible contribution of tobacco use as a cofounding factor in this study may have been ruled out as the use of tobacco in the three study groups was fairly similar (Table 1). Micronuclei formation in humans is associated with various medical conditions. MN in spermatids may lead to infertility, while a high number of MN in lymphocytes is associated with pregnancy complications and miscarriages.[30] Micronuclei are one of the four main endpoints, together with chromosomal aberrations, aneuploidy, and sister chromatid exchange (SCE) in the identification of cancer initiation. A large number of reports described the correlation between MN and cancer development. A significant increase in MN in lymphocytes was shown in untreated cancer patients. Furthermore, healthy women with mutations in breast cancer genes (BRCA1 and BRCA2) showed a higher increase in MN frequency and a higher radiation sensitivity than did women without a family history of breast cancer. Similar outcomes were also shown in lung cancer patients with a high frequency of spontaneous MN, as well as in patients with pleural malignant mesothelioma, and in those with adenocarcinoma.[31] Cancer-prone patients with Bloom's syndrome and ataxia telangiectasia also possess a high frequency of MN in their lymphocytes.[31] Analysis of European

cohorts indicates that individuals with increased MN are more likely to get cancer 12–15 years after the test was performed.[31]

With regard to the fate of MN in the e-waste population of this study, the role of p53 in apoptosis could be beneficial if the levels are optimal in these individuals. Some micronucleated cells originating from the loss of chromosomes can be eliminated by apoptosis. For instance, nocodazole, a microtubule inhibitor and blocker of cell cycle at M-phase, gives rise to aneuploid, polyploid, and micronucleated cells. It was observed that such MN-carrying cells were apoptotically eliminated through the activation of caspase-8, caspase-9, and effector caspase-3.[31] Interestingly, when MCF-7 cells lacking caspase-3 were treated with nocodazole, MN induction decreased, which allowed the authors to suggest a possible role of caspase-3 in MN formation.[31] There is also data suggesting the reincorporation of MN into the main nucleus and the restoration of normal biological activity in the cell. Alternatively, retention of MN within the cell as an extra-nuclear entity is also possible.[23,30] Further, cells treated with colchicine, vinblastine, bleomycin and arsenic showed a significant induction of MN and p53.[23,31] By analogy with DNA replication in MN, DNA repair may also be compromised by micronuclear envelope trafficking abilities. Multiple studies showed the existence of apoptotic-like DNA degradation in MN that were unable to repair double strand breaks (DSBs).[19,30] Such MN are expulsed from the cell and are lost forever. The effect of MN expulsion on a cell can be dual. If destroyed MN carry extra chromosomes in the cell, then their elimination would be necessary for regaining the normal cellular status, but if the MN chromosome was complementary to the main nucleus, then the cell might lose a certain gene dosage.[19,30]

The apoptosis-induced p53 repair pathway may be susceptible to metal toxicity via different mechanisms such as oxidative stress. Metal toxicity and increased oxidative stress have been reported in Nigerian e-waste workers in our pilot study.[9, 24]

The present study implies that occupational and environmental e-waste exposures portend risk of cytogenetic changes in exposed populations and that there is need for extensive studies in developing nations where the WEEE disposal challenges are huge. The cooperation of larger cohorts of participants and the substantial resource requirement for larger scale study constitute some form of limitations.

CONCLUSION

The observed exposure pattern and the comparatively higher micronuclei (MN) induction in the e-waste populations may suggest risk of significant cytogenetic damage and aberrant chromosomal changes associated with occupational e-waste reprocessing in Nigeria.

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Conflict of Interest

None declared.

Author Contributions

All authors have contributed significantly to the research design, laboratory analysis, statistical analysis and manuscript writing. All authors have read and approved the final manuscript.

Data availability statement

The data used to support the findings of this study is available from the corresponding author upon request

Declaration of Use of AI in Scientific Writing:

Nothing to declare

REFERENCES

1. Balde CP, Kuehr R, Blumenthal K, et al. E-waste statistics: Guidelines on classifications, reporting and indicators. Bonn (Germany): United Nations University, IAS - SCYCLE; 2015.
2. Perkins DN, Drisse MNB, Nxele T, Sly PD. E-waste: a global hazard. Ann Global Health 2014; 80:286-95. http://dx.doi.org/10.1016/j.aogh.2014.10.001.
3. Okorhi JO, Amadi-Echendu JE, Aderemi HO, Uhunmwangho R, Okwubunne AC. Disconnect between policy and practice in developing countries: evidence of managing e-waste from

Nigeria. African J Sci Technol Innov Develop 2019;11:523–31. https://doi.org/10.1080/20421338.2017.1385134.
4. Jinhui L, Huabo D, Pixing S. Heavy metal contamination of surface soil in electronic waste dismantling area: site investigation and source-apportionment analysis. Waste Manag Res 2011;29:727-8. doi: 10.1177/0734242X10397580.
5. Khalid M, Hassani S, Abdollahi M. Metal-induced oxidative stress: an evidence-based update of advantages and disadvantages. Curr Opin Toxicol 2020;20:55-68. https://doi.org/10.1016/j.cotox.2020.05.006.
6. Briffa J, Sinagra E. Blundell R. Heavy metal pollution in the environment and their toxicological effects on humans. Heliyon 2020;6:e04691. DOI:https://doi.org/10.1016/j.heliyon.2020.e04691 .
7. Pizzimenti A, Aragona M, Onesti E, Inghilleri M. Depression, pain, and quality of life in patients with amyotrophic lateral sclerosis: a cross-sectional study. Funct Neurol 2013;28:115–9. doi: 10.11138/FNeur/2013.28.2.115.
8. Igharo GO, Anetor JI, Osibanjo OO, Osadolor HB, Dike K. Toxic metal levels in Nigerian electronic waste workers indicate occupational metal toxicity associated with crude electronic waste management practices. Biokemistri 2014;26:107-13.
9. Mudipalli A. Cadmium carcinogenesis and mechanistic insights. In: Mudipalli A, Zelikoff J. (eds) Essential and non-essential metals. Molecular and integrative toxicology. Cham (Switzerland): Humana Press; 2017. pp 113–142. https://doi.org/10.1007/978-3-319-55448-8_6.
10. Jaishankar M, Tseten T, Anbalagan N, Mathew BB, Beeregowda KN. Toxicity, mechanism and health effects of some heavy metals. Interdiscip Toxicol 2014 ;7:60-72. doi: 10.2478/intox-2014-0009.
11. Balali-Mood M, Naseri K, Tahergorabi Z, Khazdair MR, Sadeghi M. Toxic mechanisms of five heavy metals: mercury, lead, chromium, cadmium, and arsenic. Front Pharmacol 2021;12:643972. doi: 10.3389/fphar.2021.643972.
12. Aaseth J, Crisponi G, Anderson O, editors. Chelation therapy in the treatment of metal intoxication. 1st ed. Elsevier Academic Press;2016.
13. Zhu Y, Costa M. Metals and molecular carcinogenesis. Carcinogenesis 2020 24;41:1161-72. doi: 10.1093/carcin/bgaa076.
14. Surai PF, Kochish II, Fisinin VI, Kidd MT. Antioxidant defence systems and oxidative stress in poultry biology: an update. Antioxidants (Basel) 2019;8:235. doi.org/10.3390/antiox8070235.
15. Rübben A, Araujo A. Cancer heterogeneity: converting a limitation into a source of biologic information. J Transl Med 2017;15:190. https://doi.org/10.1186/s12967-017-1290-9.
16. Issah I, Arko-Mensah J, Agyekum TP, Dwomoh D, Fobil JN. Electronic waste exposure and DNA damage: a systematic review and meta-analysis. Rev Environ Health 2023;38:15–31. https://doi.org/10.1515/reveh-2021-0074.
17. Ibrahim F, Adie D, Giwa A, Abdullahi S, Okuofu C. Material flow analysis of electronic wastes (e-Wastes) in Lagos, Nigeria. J Environ Prot 2013;4: 1011-17. doi: 10.4236/jep.2013.49117.
18. Terada C. Recycling electronic wastes in Nigeria: putting environmental and human rights at risk. Northwestern J Int Human Rights 2012;10:154–72.
19. Adaramodu AA, Osuntogun BA, Ehi-Eromosele CO. Heavy metal concentration of surface dust present in e-waste components: the Westminister electronic market, Lagos case study. Resour Environ 2012;2: 9-13. DOI: 10.5923/j.re.20120202.02.
20. Nnorom IC, Ohakwe J, Osibanjo O. Survey of willingness of residents to participate in electronic waste recycling in Nigeria. a case study of mobile phone recycling. J Clean Prod 2009;17:1629-37. https://doi.org/10.1016/j.jclepro.2009.08.009.
21. Holland N, Bolognesi C, Kirsch-Volders M, et al. The micronucleus assay in human buccal cells as a tool for biomonitoring DNA damage: the HUMN project perspective on current status and knowledge gaps. Mutat Res 2008;659:93–108. doi: 10.1016/j.mrrev.2008.03.007.
22. World Health Organization. Electronic waste (e-waste). Geneva : World Health Organization; 2023.
23. Jiang B, Adebayo A, Jia J, et al. Impacts of heavy metals and soil properties at a Nigerian e-waste site on soil microbial community. J Hazard Mater 2019;362:187-95. doi: 10.1016/j.jhazmat.2018.08.060.
24. Leung AOW, Duzgoren-Aydin NS, Cheung KC, Wong MH. Heavy metals concentrations of surface dust from e-waste recycling and its human health implications in Southeast China. Environ Sci Technol 2008;42:2674–80. doi: 10.1021/es071873x.
25. Igharo OG, Anetor JI, Osibajo OO, Osadolor HB, David OM, Agu KC. Oxidative stress and antioxidant status in Nigerian e-waste workers: a cancer risk predictive study. J Adv Med Med Res 2016;13:1-11. Doi:10.9734/BJMMR/2016/22770
26. Torres-Bugarín O, Zavala-Cerna MG, Nava A, Flores-García A, Ramos-Ibarra ML. Potential uses, limitations, and basic procedures of micronuclei and nuclear abnormalities in buccal cells. Dis Markers 2014;2014:956835. doi: 10.1155/2014/956835.
27. Hayashi, M. The micronucleus test—most widely used *in vivo* genotoxicity test. Genes Environ 2016;38:18. https://doi.org/10.1186/s41021-016-0044-x.
28. Garai P, Banerjee P, Mondal P, Saha NC. Effect of Heavy Metals on Fishes: Toxicity and Bioaccumulation. J Clin Toxicol 2021;11: S18:001.

29. Knudsen LE, Kirsch-Volders M. Micronuclei, reproduction and child health. Mut Res 2021;787:108345. https://doi.org/10.1016/j.mrrev.2020.108345.
30. Stefano B, Randa E, Claudia B, Michael F. Micronuclei frequency in peripheral blood lymphocytes and cancer risk: evidence from human studies. Mutagenesis 2011;26:93-100.
31. Luzhna L, Kathiria P, Kovalchuk O. Micronuclei in genotoxicity assessment: from genetics to epigenetics and beyond. Front Genet 2013;4:131. doi: 10.3389/fgene.2013.00131.