

*Full Length Research Paper*

# Acute toxicity assessment of crude lead-extract from electronic waste materials in Nigeria

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**Lead, with its toxic emission and pollution, is one of the hazardous chemical components of electronic wastes (e-wastes) rapidly generated in developing countries. This study assessed the environmental health effects of crude lead extracted from e-wastes materials, as determined by its acute toxicity (rat, oral). Diluted HNO<sub>3</sub> and hot concentrated HCl tests were used to confirm the presence of lead in the extract. LD<sub>50</sub> (400 mg/kg) showed higher toxicity than the lowest toxic dose of 790 mg/kg reported in literature. There was a perfect positive correlation between the log dose and dead percentage, which was significant at 0.014, with 0.99° of confidence. The R<sup>2</sup> (0.839) and significance F (0.029) showed high reliability. In view of this, we recommend the inclusion of enlightenment and making/enforcing adequate policies for improved management and control of e-waste materials.**

**Key words:** Environmental health effect, toxic chemical component, e-waste.

## INTRODUCTION

Various reasons have been given for rapid rate of generation of electronic wastes (e-wastes) in developing countries. Some hazardous chemical elements and compounds, such as lead, are contained in significant quantity in e-waste materials (Eneh and Agunwamba, 2011). Emission of lead from e-waste materials result in environmental pollution and health hazards, as inhalation of lead emissions result in lead poisoning in adults. Occupation and products are serious routes of exposure to lead poisoning (World Health Products, 2010), which can occur from contact with lead in air, household dust, commercial products and combustion of solid waste. Parents who are exposed to lead in the workplace can bring lead dust home on clothes or skin and inadvertently expose it to their children (Mañay et al., 2008).

With the Industrial Revolution in the 19th century, lead poisoning became common in the work setting. Eisinger

(1982) noted that the 20th century saw an increase in worldwide lead exposure levels due to the increased widespread use of the metal. Inhalation of dust and fumes containing lead compounds cause the disease called morbi metallici. Woolf et al. (2007) observed that people could get lead poison on their fingers from touching a dusty or peeling lead object, and then putting their fingers in their mouths or eating food afterward. Many symptoms of lead poisoning affect many different parts of the body. A single high dose of lead can cause severe emergency symptoms (acute poisoning). However, chronic poisoning commonly occurs from repeated exposure to small amounts of lead which builds up slowly over time. The health problems get worse as the level of lead in the blood gets higher.

Watts (2009) and Brodtkin et al. (2007) stated that no amount of lead is safe. Individuals at highest risk for lead poisoning include those exposed to lead through occupational means. Lead is particularly dangerous because once it gets into a person's system, by being inhaled, swallowed, or in a small number of cases, absorbed through the skin, it is distributed throughout the body. Lead forms a variety of compounds and exists in the environment in various forms. Sanborn et al. (2002) reported that lead is a common environmental

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**Abbreviations:** IN, Irregular nuclei; DNS, denaturation of myelin; NMDA, N-methyl-D-aspartate; PNS, peripheral nervous system; CNS, central nervous system.

pollutant and that it causes environmental contamination that includes industrial use of lead, such as that found in plants.

Spitz et al. (2008), White et al. (2007) and Dimaio et al. (1983) asserted that lead from the atmosphere; soil or dumpsite can end up in groundwater and surface water. The main body compartments that store lead are the blood, soft tissues, and bone. The half-life of lead in these tissues is measured in weeks for blood, months for soft tissues, and years for bone. In adults, 94% of absorbed lead is deposited in the bones and teeth, but children only store 70% in this manner, a fact which may partially account for the more serious health effects of lead on children. Patrick (2006) showed that the estimated half-life of lead in bone is 20 to 30 years, and bone can introduce lead into the bloodstream long after the initial exposure is gone. The half-life of lead in the blood in men is about 40 days, but it may be longer in children and pregnant women, whose bones are undergoing remodeling, and this allows the lead to be continuously re-introduced into the bloodstream.

Fujita et al. (2002) observed that if lead exposure takes place over years, clearance is much slower, partly due to re-release of lead from bone. Many other tissues store lead, but those with the highest concentrations (other than blood, bone, and teeth) are the brain, spleen, kidneys, liver, and lungs. It is removed from the body very slowly, mainly through urine. Smaller amounts of lead are also eliminated through the faeces, and very small amounts through hair, nail, and sweat.

Xu et al. (2009) reported that lead has no known physiologically relevant role in the body, but its harmful effects are myriad. Lead and other heavy metals create reactive radicals, which damage cell structures, including DNA and cell membranes. Lead also interferes with DNA transcription, enzymes that help in the synthesis of vitamin D and those that maintain the integrity of the cell membrane. Anaemia may result when the cell membranes of red blood cells become more fragile as a result of damage to their membranes. Lead interferes with metabolism of bones and teeth and alters the permeability of blood vessels and collagen synthesis. It may also be harmful to the developing immune system, causing production of excessive inflammatory proteins. This mechanism may mean that lead exposure is a risk factor for asthma in children. Lead exposure has also been associated with a decrease in activity of immune cells, such as polymorphonuclear leukocytes. Lead also interferes with the normal metabolism of calcium in cells and causes it to build up within them.

Lanphear et al. (2005) reported that the primary cause of lead's toxicity is its interference with a variety of enzymes due to the fact that it binds to sulfhydryl groups found on many enzymes. Lead is able to mimic other metals that take part in biological processes, which act as co-factors in many enzymatic reactions, displacing them as the enzymes on which they act. Lead is able to

bind to and interact with many of the same enzymes as these metals. But due to its differing chemistry, it does not properly function as a co-factor, thus interfering with the enzyme's ability to catalyze its normal reaction(s). The essential metals with which lead interacts include calcium, iron, and zinc. One of the main causes for the pathology of lead is that it interferes with the activity of an essential enzyme called delta-aminolevulinic acid dehydratase, (ALAD), which is important in the biosynthesis of haeme, the co-factor found in haemoglobin. Lead also inhibits the enzyme ferrochelatase, another enzyme involved in the formation of haeme. Ferrochelatase catalyzes the joining of protoporphyrin and  $Fe^{2+}$  to form haeme. Lead's interference with haeme synthesis results in production of zinc protoporphyrin and the development of anaemia. Another effect of lead's interference with haeme synthesis is the build-up of haeme precursors, such as aminolevulinic acid, which may be directly or indirectly harmful to neurons.

Flora et al. (2008) reported that lead exposure damages cells in the hippocampus, a part of the brain involved in memory. Hippocampi of rats exposed to lead show structural damage, such as irregular nuclei (IN) and denaturation of myelin (DNS). Lead interferes with the release of neurotransmitters, chemicals used by neurons to send signals to other cells. It interferes with the release of glutamate, a neurotransmitter important in many functions, including learning, by blocking N-methyl-D-aspartate (NMDA) receptors. The targeting of NMDA receptors is thought to be one of the main causes for lead's toxicity to neurons. In addition to inhibiting the NMDA receptor, lead exposure decreases the amount of the gene for the receptor in part of the brain. Besides, lead has been found in animal studies to cause programmed cell death in brain cells.

According to Bellinger (2008, 2005, 2004) and Guidotti and Ragain (2007), lead affects every organ of the body, leading to hearing loss, tooth decay, kidney damage, nephropathy, Fanconi syndrome, gout, and other serious health disorders. Xu et al. (2009), Pokras and Kneeland (2008) and Lanphear et al. (2005) also observed that lead exposure is associated with high blood pressure, coronary heart disease, heart rate variability, cardiac automatic dysfunction and death from stroke. Moss et al. (1999), Goyer (1990) and Brudevold et al. (1977) reported that lead affects both the male and female reproductive systems. In men, when blood lead levels exceed 40  $\mu\text{g}/\text{dl}$ , sperm count is reduced and changes occur in volume of sperm, their motility, and their morphology.

A pregnant woman's elevated blood lead level can lead to miscarriage, premature birth, low birth weight, and problems with development during childhood. Lead is able to pass through the placenta and into breast milk, and blood lead levels in mothers and infants are usually similar. A foetus may be poisoned *in utero* if lead from the mother's bones is subsequently mobilized by the changes

in metabolism due to pregnancy. US CDC (2009), Gemmel et al. (2002) and Campbell et al. (2000) observed that lead affects the peripheral nervous system (PNS; especially motor nerves) and the central nervous system (CNS). PNS effects are more prominent in adults and CNS effects are more prominent in children. Lead causes the axons of nerve cells to degenerate and lose their myelin coats.

Ekong et al. (2006), Shadick et al. (2000) and Lin and Huang (1994) reported that the brain is the organ most sensitive to lead exposure. The brains exposed to lead showed decreased volume, especially in the prefrontal cortex. Park et al. (2008), Cleveland et al. (2008) and Navas-Acien (2007) reported that lead poisoning interferes with the normal development of a child's brain and nervous system. Therefore, children are at greater risk of lead neurotoxicity than adults. In a child's developing brain, lead interferes with synapse formation in the cerebral cortex, neurochemical development (including that of neurotransmitters), and organization of ion channels. It causes loss of neurons' myelin sheaths, reduces numbers of neurons, interferes with neurotransmission, and decreases neuronal growth. Jones et al. (2009) and Murata et al. (2009) observed that blood lead levels steadily increase with increasing age.

Thus, the effects, diagnosis, prognosis, epidemiology, therapy, prevention and history, as well as exposure routes and pathophysiology of lead poisoning have been reported. But, there is no report so far on the environmental health effects of crude lead-extract from e-waste materials abounding in homes, offices, workshops and littering the city landscape or public dumpsites in developing countries, where environmental regulations and compliance are weak (Forge, 2007). The purpose of this study was, therefore, to establish the environmental health effects of lead from e-wastes. The specific objectives were to establish the lethal dose 50 (LD<sub>50</sub>) of crude lead-extract from e-waste materials and to recommend some measures for the management and control of pollution from toxic emissions of lead from e-wastes materials.

This study assumed that a dose of crude lead-extract from e-waste materials was not significantly related to the number of deaths of experimental rats to which it had been acutely exposed by oral administration. To correlate the data from acute toxicity tests and to test the hypothesis, the single linear regression (SLR) of the Statistical Package for Social Sciences (SPSS) was done for the log dose of the crude lead extracted from e-waste materials against the dead percentage of the experimental animals. Researchers will benefit from the results of the empirical study, which can serve as a reference point for further studies. Environmental practitioners and policy-makers will also benefit from this study.

## MATERIALS AND METHODS

Experimental rats, analytical reagent grade chemicals and labora-

tory wares were obtained from the laboratory of the Department of Medical Laboratory Sciences, Faculty of Health Sciences and Technology, College of Medicine, Enugu Campus, University of Nigeria, Nsukka. E-waste materials were collected from ICT products repairers and spare-parts vendors in C-To-C Business Plaza, Nkpokiti Street, Enugu, Nigeria.

Lead is applied in electrical and electronic devices as solder, cathode ray tube (CRT) monitor glass, lead-acid batteries, and some formulations of polyvinylchlorides (PVCs) (Slade, 2006; Murali, 2009). Crude lead-extract was obtained from e-waste materials, which were first hand-dismantled into various parts (Royte, 2005). The parts containing old solder were mechanically separated and lead was extracted from circuit boards by use of soldering iron.

The two experiments to qualitatively test for lead in the extract were: shaking in diluted HNO<sub>3</sub> to see if it would readily dissolve, and treating it with hot concentrated HCl to see if it would liberate gas (hydrogen). The modified Miller and Tainter method (Randhawa, 2009) was used for the acute toxicity tests. In the preliminary studies carried out to determine the dose range to be used in the acute toxicity tests, animals in groups (n = 20) of close weight ranges were separately exposed to doses of 50, 100, 200, 400, 800, 1600, 3200, 6400, 12800, and 25600 mg/kg of the crude lead-extract in diluted HNO<sub>3</sub> (1 N) as solvent for 24 h. The control set was exposed to the solvent without the extract. After oral administration, the animals were observed regularly and mortality was recorded at 3, 6, 12 and 24 h. Subsequently, a series of 5 doses (200, 400, 800, 1600 and 3200 mg/kg), between the dose that killed a few or none of the animals and all or most of the animals, were selected for acute toxicity tests.

The percentage of animals that died at each dose level was transformed to probit. The probits for 0 and 100% were corrected, using the formular (Randhawa, 2009):

$$\text{For } 0\%: 100 \times \frac{0.25}{n}$$

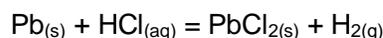
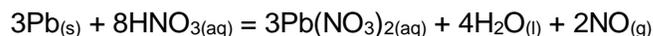
$$\text{For } 100\%: 100 \times \frac{(n - 0.25)}{n}$$

The probit values (y) were plotted against log-doses (x). The log dose corresponding to probit 5 was traced and the anti-log noted as the value of LD<sub>50</sub>, which killed 50% of the animals. The data were correlated by SLR of the dependent variable (y) (dead percentage) of experimental animals against the independent variable (x) (log dose) of lead-extract from e-waste materials. The statistics generated also helped to test the hypothesis.

## RESULTS AND DISCUSSION

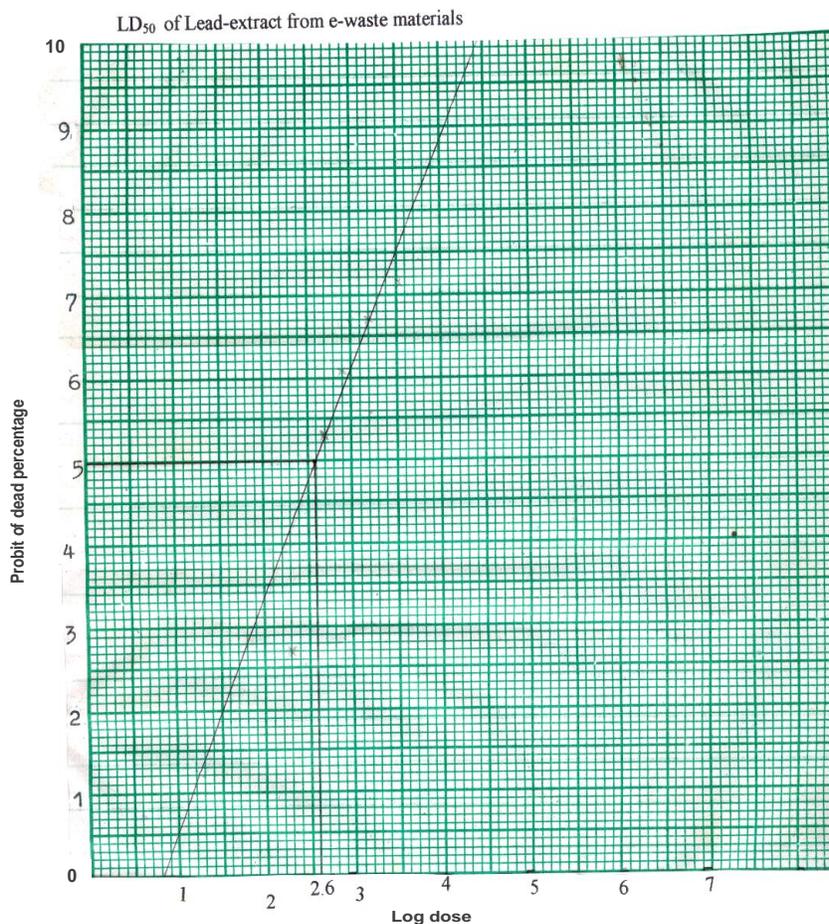
### Test for lead in extract

The crude lead-extract dissolved readily in diluted HNO<sub>3</sub> and reacted with hot concentrated HCl to liberate hydrogen gas. These reactions confirmed that the extract contained lead, as illustrated in the following chemical equations:



**Table 1.** Results of the lethal doses of crude lead-extract from e-wastes (rat, oral).

Group	Dose (mg/kg)	Log dose	n	Death rate	Dead (%)	Corrected (%)	Probit
1	200	2.30	20	0/20	0.00	1.25	2.70
2	400	2.60	20	11/20	55.00	55.00	5.13
3	800	2.90	20	17/20	85.00	85.00	6.04
4	1,600	3.20	20	19/20	95.00	95.00	6.64
5	3,200	3.51	20	20/20	100.00	98.75	7.33

**Figure 1.** Plot of probit of dead percentage against log dose for crude lead-extract (rat, oral).

These findings were in conformity with the report of Ababio (2011) that lead dissolved readily in diluted  $\text{HNO}_3$  and reacted with hot concentrated  $\text{HCl}$  to liberate hydrogen gas.

#### Acute toxicity test (rat, oral) with crude lead extracted from e-wastes

Table 1 contains the data obtained for the acute toxicity tests (rat, oral) with lead extract. Crude lead extract

doses of 200, 400, 800, 1600 and 3200 mg/kg killed 0, 11, 17, 19 and 20 rats, respectively, within 24 h. From the plot of probit values of the dead percentage (y) against log doses (x) (Figure 1), the log dose corresponding to probit 5 was 2.6 and its anti-log was 400. Therefore, the value of  $\text{LD}_{50}$  was 400 mg/kg (rat, oral).

#### Correlation of data and test of hypothesis

Table 2 shows the selected statistics from the SLR

**Table 2.** Selected statistics for acute toxicity analyses for lead-extract.

Statistic	Parameter	Lead
Correlations	Pearson correlation value between the log dose and dead percentage	0.916
	One-tailed significance value	0.014
M/Summary (b)	R <sup>2</sup>	0.839
	Significance F change	0.029

SLR analysis (SPSS).

analysis. At 400 mg/kg, the LD<sub>50</sub> of the crude lead-extract signified a moderate toxicity (rat, oral). This finding was internally validated by a perfect positive correlation (0.916) between the log dose of the crude lead-extract and dead percentage of the experimental animals. The relationship between the two constructs was significant at 0.014, with 0.99° of confidence. The high R<sup>2</sup> value (0.839) and high significance F-value (0.029) showed high reliability. Therefore, the null hypothesis was rejected in favour of acceptance of the alternative hypothesis: "A dose of crude lead-extract from e-waste materials was significantly related to the number of deaths of experimental rats to which it was acutely exposed by oral administration".

#### External validity of the lethality profile of the crude lead-extract from e-waste materials

For external validity, the LD<sub>50</sub> of 400 mg/kg (rat, oral) for lead-extract was compared with the literature values. The lowest lethal dose of lead had been reported as 191 mg/kg for guinea pig and dog. The LD<sub>50</sub> (oral) was 112 mg/kg for rat. The lowest toxic dose (oral) was 790 mg/kg, while the LC<sub>50</sub> (inhalation) was 10 mg/m<sup>3</sup>/24 h for rat. The lowest reported lethal dose (LDLO) of lead was 1,470 µg/kg (14.7 mg/kg) for man (Rahde, 1994 a, b; Sax and Lewis, 1989). In comparison with these literature values, the high lethality profile of lead-extract from e-waste materials is underscored at LD<sub>50</sub> of 400 mg/kg (rat, oral).

Besides, electronic products containing lead components abound in homes, offices, workshops and city waste dump sites, leading to multiple sources of lead emissions and regular inhalation. Thus, the reported LC<sub>50</sub> (inhalation) (10 mg/m<sup>3</sup>/24 h) is easy to attain. Hence, there is a serious cause for intervention against the environmental health effects of lead contained in e-wastes. More so, indoor air pollution and urban air quality, of which emission is a factor, were listed as two of the world's worst pollution problems in 2008 (Blacksmith Institute, 2008). Again, Chukwuma and Asabor (2011) reported that Nigerian scavengers and other operators in e-waste dumpsites were oblivious of the health hazards posed by e-wastes (Figure 2).

Osibanjo and Ogundiran (2010) showed that risk is the sum of hazard and exposure:

$$\text{Risk} = \text{Hazard} + \text{Exposure}$$

Emissions of lead from e-waste materials constitute the environmental health hazard. The exposure is high from the multiple sources of lead emission. Hence, the risk is high. The recent report of the World Health Product (2010) that the primary cause of lead poisoning in adults is inhalation of lead emission lends credence to this worrisome finding.

Woolf et al. (2007) reported that lead is a very strong poison, adding that when lead dust is inhaled some of the poison can stay in the body and cause serious health problems. Lead poisoning commonly builds up slowly over time from repeated exposure to small amounts of lead, with no obvious symptoms. Noji and Kelen (1989) reported that accumulation and toxicity occur if more than 0.5 mg/day of lead is absorbed. The fatal dose is estimated at 500 mg of absorbed lead. Low levels of lead exposure can harm a child's mental development, with over 10 µg/dl being a definite concern. The multiple sources of emission of lead may predispose people to absorption levels greater than 0.5 mg/day and the attendant lead accumulation and toxicity.

Other literature reports on the adverse effects of lead also corroborate the concern for lead emission from e-waste materials. The lowest observed adverse effect level (LOAEL) of lead in human volunteers exposed to particulate lead (inhalation) is 3.2 µg/m<sup>3</sup> (0.032 mg/m<sup>3</sup>). The lowest toxic concentration (inhalation) of lead in humans is 10 µg/m<sup>3</sup> (0.1 mg/m<sup>3</sup>) (ATSDR, 1990). The workplace standard permissible exposure limit (PEL) is 50 µg/m<sup>3</sup> (0.5 mg/m<sup>3</sup>), while the recommended exposure limit (REL) time weighed average (TWA) is 100 µg/m<sup>3</sup> (1 mg/m<sup>3</sup>) (NIOSH, 1978). The threshold limit value-time weighed average (TLV-TWA) for inorganic lead dusts and fumes is 150 µg/m<sup>3</sup> (1.5 mg/m<sup>3</sup>) (ACGIH, 1990). The acceptable daily intake (ADI) and other guideline levels provisional maximum tolerable weekly intake of lead is 3 mg per person or 50 µg/kg (0.5 mg/kg) body weight for adults and 25 µg/kg (0.25 mg/kg) body weight for children (FAO/WHO, 1987). Thus, the inhalation of hazardous lead emissions, enhanced by the multiplicity of emission



**Figure 2.** Operators in e-waste dumpsite oblivious e-waste hazard (Chukwuma and Asabor, 2011).

sources, may predispose the population to these toxicity and mortality levels of lead poisoning.

Pokras and Kneeland (2008) observed that lead poisoning is entirely preventable by, among others, avoiding exposure to lead. Prevention strategies can be divided into individual (measures taken by a family, e.g. removing lead-containing items, such as piping or blinds from the home), preventive medicine (identifying and intervening with high-risk individuals), and public health or nationwide policies (e.g. laws that ban lead in products or reduce allowable levels in water or soil, reducing risk on a population level) (ATSDR, 1990; Glenn, 1986; ILO, 1983).

The  $LD_{50}$  figure of 400 mg/kg (rat, oral) obtained for lead-extract from e-waste materials was higher, and therefore, less lethal than the literature value of 112 mg/kg for rat. This could be explained on the ground that the lead-extract was used in its crude form, so as to reflect the actual components of the gaseous mix of the emissions from e-waste materials containing lead and

other chemical components. Understandably, crude lead-extract is less toxic than pure lead. Besides, electronic and electrical materials contain lead and tin as solder (Nurudeen, 2011). Therefore, the crude lead extracted from e-waste materials could have contained tin as well, thereby further adulterating and lowering the lethal value of the extract.

## CONCLUSION AND RECOMMENDATION

Since the  $LD_{50}$  of 400 mg/kg (rat, oral) obtained for crude lead extracted from e-waste materials sends danger signals, environmental management and control policies and actions against pollution from lead emissions from e-waste materials have become imperative and commends the following recommendations:

- 1) The government and civil society organizations should create awareness on the toxicity of the emissions from e-

waste materials in homes, offices, workshops and littering the city landscape.

2) Citizens of developing countries should be encouraged to dispose of obsolete information and communication technology (ICT) products from homes and offices.

3) Proper disposal approaches should be taught to citizens in developing countries.

4) The government should designate appropriate centers/ bins for collection of e-waste materials for delivery to appropriate dumpsites for proper treatment.

5) Enlightenment campaign on the use of protective equipment, such as nose-mask, and proper hygiene, especially by professionals handling ICT facilities, will be helpful.

6) Recycling, by way of hand-dismantling of e-waste materials and mechanical extraction of old solder by use of soldering iron on the circuit boards, is economically and environmentally desirable.

7) Policies should be made and enforced in developing countries on the use of protective clothes (including shoes), which must not leave the contaminated environment; protective equipment (such as safety nose-masks, glasses, gloves, and ventilation gadgets); face and hand coverings made of impermeable materials; proper hygiene: cleaning spills immediately, wetting floor with a fine spray to avoid stirring up dust, and covering storage receptacles to reduce the circulation of fumes and dusts; and personal hygiene (avoiding eating, smoking, or drinking in contaminated environment); separation of eating facilities from working areas. Other areas to cover in policies formulation and implementation are separation of lead from oxidizers (such as perchlorates, peroxides, permanganates, chlorates and nitrates) and chemically active metals (such as potassium, sodium, magnesium and zinc), since violent reactions occur; pre-employment and pre-placement examinations, periodical examinations, clinical tests, record-keeping and health education, medical surveillance programme, and measurement of air lead levels.

In conclusion, further research using chemical extraction of lead from e-waste materials is recommended as a likely more efficient technique, since the lead-extract might contain tin which also serves as solder in electrical and electronic equipment.

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