LEAD EXPOSURE STUDY
AMONG WORKERS IN LEAD ACID BATTERY REPAIR UNITS
OF
TRANSPORT SERVICE ENTERPRISES
ADDIS ABABA, ETHIOPIA

BY
KEMAL AHMED HUSSEIN (BPharm)

A Thesis Submitted to the School of Graduate Studies
of
Addis Ababa University

In Partial Fulfilment of the Requirements
for
the Master of Science Degree in Toxicology

July 2005
ADDIS ABABA UNIVERSITY

School of Graduate Studies

LEAD EXPOSURE STUDY
AMONG WORKERS IN LEAD ACID BATTERY REPAIR UNITS OF
TRANSPORT SERVICE ENTERPRISES

ADDIS ABABA, ETHIOPIA

BY
Kemal Ahmed Hussein (BPharm)

ADVISORS:
- Ephrem Engidawork (PhD)-AAU
- Gonfa Ayana (MSc)-EHNRI
ACKNOWLEDGEMENTS

First and foremost, I would like to thank Allah (S.W.) for all the strength He has given me to complete this project. Secondly, my advisor, Dr. Ephrem Engidawork and co-advisor, Ato Gonfa Ayana for all the time and effort they have put in assisting me with this project.

Thirdly, Ethiopian Health and Nutrition Research Institute (EHNRI), in particular Dr. Tsehaynesh Melese, for allowing me to use the facilities of the institute. I would also like to thank all the managers and the staff members of the enterprises where the study was conducted for their enthusiasm and willingness in helping me whenever I needed information and specimens for my project.

Prof. Dr. Abdel-Rahman Mohammed, Dr. Amare Mengistu, Dr. Yared Mekonnen and Ato Paulos Nigussie for all the support they have given me for the whole duration of the project. All staff, Department of Pharmacology for their unreserved assistance throughout the project period. All my colleagues for their continuous encouragement and advice throughout the project time. All my family whose patience and endurance made completion of this project a reality. Addis Ababa University (AAU) for the provision of financial support.

Lastly but not least, I thank my wife, Munira in recognition of her invaluable help, patience and understanding. I would not have achieved this without her love and support.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACKNOWLEDGEMENTS</td>
<td>i</td>
</tr>
<tr>
<td>ABBREVIATIONS</td>
<td>v</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
<td>vi</td>
</tr>
<tr>
<td>LIST OF FIGURES</td>
<td>vii</td>
</tr>
<tr>
<td>LIST OF APPENDICES</td>
<td>viii</td>
</tr>
<tr>
<td>ABSTRACT</td>
<td>ix</td>
</tr>
<tr>
<td><strong>1. INTRODUCTION</strong></td>
<td>1</td>
</tr>
<tr>
<td>1.1. Lead and its Exposure</td>
<td>1</td>
</tr>
<tr>
<td>1.2. Toxicology of occupational lead exposure</td>
<td>5</td>
</tr>
<tr>
<td>1.2.1. Effects of lead on the nervous system</td>
<td>9</td>
</tr>
<tr>
<td>1.2.2. Effects of lead on haem biosynthesis and erythropoiesis</td>
<td>12</td>
</tr>
<tr>
<td>1.2.3. Effects of lead on renal function</td>
<td>15</td>
</tr>
<tr>
<td>1.2.4. Effects of lead on digestive and hearing systems</td>
<td>17</td>
</tr>
<tr>
<td>1.2.5. Effects of lead on reproductive system</td>
<td>18</td>
</tr>
<tr>
<td>1.2.6. Effects of lead on blood pressure and cardiovascular system</td>
<td>19</td>
</tr>
<tr>
<td>1.3. Physical effects of lead</td>
<td>20</td>
</tr>
</tbody>
</table>
1.4. Lead exposure preventive and control measures .....................................................…..  20

1.4.1. Personal protective equipment ..........................................................21
1.4.2. General ventilation..............................................................................22
1.4.3. Containment.......................................................................................22
1.4.4. Local exhaust ventilation.................................................................23

2.  OBJECTIVES...........................................................................................................  ..25

2.1. General Objective ..........................................................................................25
2.2. Specific Objectives .......................................................................................25

3.  MATERIAL AND METHODS ................................................................................  .....26

3.1. Study Design and Period ..............................................................................26
3.2. Study Area ....................................................................................................26
3.3. Source and Study Population ......................................................................26
3.4. Sampling Technique and Sample Size ......................................................27
3.5. Data Collection and Measurement ..............................................................27
  3.5.1. Structured Questionnaire........................................................................27
  3.5.2. Laboratory Analysis...............................................................................27
3.6. Quality Control ..............................................................................................29
3.7. Data Analysis ................................................................................................29
3.8. Ethical Consideration ....................................................................................30

4.  RESULTS ...............................................................................................................  .... 31
4.1. Construction of Calibration Curve ................................................................. 32
4.2. The level of urinary δ - ALA ........................................................................ 34
4.3. Serum Creatinine, Creatinine Clearance and Urea levels .......................... 37
4.4. Uric Acid levels ........................................................................................... 38
4.5. Reported Illnesses and Lifestyle Factors ...................................................... 39
4.6. Status of Workplace Safety Measures .......................................................... 42

5. DISCUSSION .................................................................................................. 44

6. CONCLUSION AND RECOMMENDATIONS ................................................ 51
   6.1. CONCLUSION ........................................................................................ 51
   6.2. RECOMMENDATIONS .......................................................................... 51

7. SUGGESTIONS FOR FUTURE WORK ........................................................... 53

8. REFERENCES .................................................................................................. 54
ABBREVIATIONS

ALA: - δ- Aminolevulinic Acid
ALAD: - δ- Aminolevulinic Acid Dehydratase
ALAS: -δ Aminolevulinic Acid Synthetase
ALAU: -Urinary Delta - Aminolevulinic Acid
BUN: -Blood Urea Nitrogen
CC: -Creatinine Clearance
FEP: -Free Erythrocyte Porphyrin
GFR: - Glomerular Filtration Rate
IPCS: -International Programme on Chemical Safety
LEV: -Local Exhaust Ventilation
PbB: -Blood Lead
PBG: -Porphobilinogen
PPE: -Personal Protective Equipment
SC: -Serum Creatinine
SHARP: -Safety and Health Assessment and Research for Prevention
UA: -Uric Acid
WHO: -World Health Organization
ZPP: -Zinc Protoporphyrin.
Table 1: Range of health problems associated with various blood lead levels…………………8

Table 2: Study sites and demographic data of lead exposed workers………………………31

Table 3: Employment duration of lead exposed workers……………………………………31

Table 4: Urinary Aminolevulinic acid mean levels of exposed workers by enterprise………35

Table 5: Categorization of enterprises with the number of exposed subjects based on range of
measured urinary Aminolevulinic acid…………………………………………………………36

Table 6: Urinary Aminolevulinic acid mean levels of exposed and non-exposed subjects by age
group…………………………………………………………………………………………36

Table 7: Urinary Aminolevulinic acid mean levels of exposed workers by sex………………36

Table 8: Urinary Aminolevulinic acid mean levels of exposed workers by employment
duration…………………………………………………………………………………………37

Table 9: Serum creatinine, creatinine clearance and urea levels of exposed and non-exposed
groups…………………………………………………………………………………………38

Table 10: Summary of reported illnesses……………………………………………………40

Table 11: Levels of mean Aminolevulinic acid among alcohol taking and non-alcohol taking
exposed subjects………………………………………………………………………………41

Table 12: Smoking habits, meals at workplace and work related hobbies of exposed subjects…41
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 1</td>
<td>Risk Assessment/ Management Paradigm</td>
<td>21</td>
</tr>
<tr>
<td>Figure 2</td>
<td>Best line fitted standard calibration curve for the determination of δ-ALA</td>
<td>33</td>
</tr>
<tr>
<td>Figure 3</td>
<td>Urinary Aminolevulinic acid mean levels in exposed and non-exposed groups</td>
<td>34</td>
</tr>
<tr>
<td>Figure 4</td>
<td>Distribution of subjects by group of urinary Aminolevulinic acid levels</td>
<td>34</td>
</tr>
<tr>
<td>Figure 5</td>
<td>UA mean levels in exposed and non-exposed groups</td>
<td>38</td>
</tr>
<tr>
<td>Figure 6</td>
<td>Distribution of exposed and non-exposed subjects by group of Uric acid levels</td>
<td>39</td>
</tr>
<tr>
<td>Figure 7</td>
<td>A partial view of storage battery repair unit in one of the enterprises</td>
<td>43</td>
</tr>
<tr>
<td>Figure 8</td>
<td>Disposal of used storage batteries in one of the enterprises</td>
<td>43</td>
</tr>
</tbody>
</table>
## LIST OF APPENDICES

<table>
<thead>
<tr>
<th>APPENDIX</th>
<th>DESCRIPTION</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>APPENDIX A</td>
<td>STRUCTURED INTERVIEW FOR LEADED STORAGE BATTERY REPAIR WORKERS</td>
<td>65</td>
</tr>
<tr>
<td>APPENDIX B</td>
<td>INFORMED CONSENT (ENGLISH AND AMHARIC VERSIONS)</td>
<td>68</td>
</tr>
</tbody>
</table>
ABSTRACT

Lead exposure is common in automobile battery manufacture and repair, radiator repair, secondary smelters and welding units. Urinary Aminolevulinic acid has validity as a surrogate measure of blood lead level among workers occupationally exposed to lead. This study had therefore assessed the magnitude of lead exposure in battery repair workers of three transport service enterprises. To this effect, a cross-sectional study was carried out on lead exposure among storage battery repair workers between November and May 2005 from Anbasa, Comet and Walia transport service enterprises, Addis Ababa, Ethiopia. Subjective information from the workers was obtained by making use of structured questionnaire. Other information was obtained from the walkthrough evaluation of the repair units. Aminolevulinic acid levels in urine were used as an index of the exposure. This was coupled to measurements of other relevant parameters in blood and urine collected from adult subjects working in the repair units as well as age matched control subjects that were not occupationally exposed to lead. Aminolevulinic acid was determined by spectrophotometry, while creatinine clearance, serum creatinine, urea and uric acid levels were determined using AMS Autolab analyser.

Urinary Aminolevulinic acid levels were found to be significantly higher in exposed group (1.6 mg/dl ± 0.2) compared to the non-exposed ones (0.7mg/dl ± 0.1) (p<0.001). Alcohol taking exposed subjects exhibited a significant increase in urinary aminolevulinic acid levels than non-alcohol taking ones (p < 0.05). Moreover, urinary aminolevulinic acid levels of exposed subjects increased with age (p<0.001) as well as duration of employment (p<0.001). Whereas, serum uric acid levels of exposed subjects was significantly higher than non-exposed ones (p<0.05), no statistically significant difference had been found in renal indices and other measured parameters between exposed and non-exposed subjects. From the questionnaire responses and walkthrough observations, it was known that all the repair units do not implement effective preventive and control measures for workplace lead exposure. Taken together, these findings indicated that workers in lead acid battery repair units of the transport service enterprises are not safe from excessively high lead exposure. Thus, strict enforcement of appropriate and cost-effective preventive and control measures is required by all the enterprises.

Key words: Lead exposure, Delta-Aminolevulinic acid, renal indices, uric acid, preventive and control measures.
1. INTRODUCTION

1.1. Lead and its Exposure

Lead (Pb), with atomic number of 82 and atomic weight of 207.19 has a specific gravity of 11.34. Metallic lead is a lustrous blue-grey metal, which rapidly becomes dull in air, and is soft, malleable and ductile. It has a melting point of 327.4°C and a boiling point of 1740°C. It has four naturally occurring isotopes with atomic weights 208, 206, 207, and 204, in decreasing order of abundance. The usual valence state in inorganic lead compounds is +2 (Lead, 2001).

Lead is a metal of antiquity and has been used for many purposes for thousands of years. A variety of forms of lead are used industrially, the most common being lead oxide (PbO) and red lead oxide (lead tetraoxide; Pb₃O₄). Lead oxides are used in the plates of electric batteries and accumulators and in other substances (Stellman and Osinsky, 1997). About 40 to 50% of all lead production is used to make lead-acid batteries (both lead metal and lead oxides are used). A further 20% of production is used as the metal, for example, cable sheathing, solder, ammunition, alloys, weights, ballast, and low-melting alloys. Lead is a major component of many alloys such as bronze and solders (Massaro, 1997). It is used for tank linings, piping, and building construction. Lead-based pigments, such as white lead, have a long tradition of being used in paints, although they have virtually all been substituted by other pigments in the last 20 years for obvious toxicological reasons. Lead compounds are used in glassware and ceramics and as stabilizer in plastics. Red lead is used to make television tubes. About 10% is converted into alkyl-lead compounds and used as antiknock additives in gasoline, although this use is in decline as more and more countries move to limit the concentration of such additives in gasoline (Massaro, 1997). Some of the uses for lead in ancient times (such as lead sheet for lining roofs) remain today.

Solubility in water varies; lead sulphide and lead oxides being poorly soluble and the nitrate, chlorate and chloride salts are reasonably soluble in cold water. Lead also forms salt with such organic acids as lactic and acetic acids and stable organic compounds such as tetraethyl lead and tetramethyl lead (IPCS, 1995).
Lead is a rare metal in the earth's crust and its deposits are scattered throughout the world. The most common ore is galena (lead sulphide-87% lead). Lead is obtained from the ore by smelting to produce lead oxide, which is reduced to lead bullion then refined to remove other metallic impurities (IPCS, 1995). Lead is not bio-degradable and only accumulates where it is deposited. Due to this reason lead levels have increased enormously from the ancient times (Flegal and Smith, 1995). The magnitude of increase has resulted in adverse health effects (Budd et al., 1988).

Lead is a highly toxic metal with no known physiological benefits and is an ubiquitous pollutant in the ecosystem as a result of its natural occurrence and its industrial use. Mankind has used lead for over 6000 years (IPCS, 1995). Lead’s toxicity was recognized and recorded as early as 2000 BC and the widespread use of lead has been a cause of endemic chronic plumbism in several societies throughout the history. There is no such thing as normal or safe levels of lead for its toxicity to humans (George, 1999).

Work is indispensable for the individual, society and for the development of nations. Significant human suffering related to occupation is unacceptable and often results in appreciable financial loss due to the burden on health and social security systems, which negatively impacts production (Goelzer, 1996). There are a number of occupational hazards in all workplaces worldwide due to a lack of adequate prevention and control measures (WHO, 1999). Occupational exposure to lead still occurs in many countries of the world. Especially in many developing countries including Ethiopia occupational lead exposure is entirely unregulated and no monitoring of exposures exists (George, 1999).

Automobile battery manufacture and repair, radiator repair, secondary smelters (including scrap metal refiners) are found in most countries and are common sources of lead exposure. Other occupations where workers have been shown to be at risk from airborne lead include demolition; welding and ship breaking where lead-based paint is present. Significant occupational lead exposures are not limited to traditional heavy industries (George, 1999). Small domestic versions of secondary smelters exist in a large number of countries. These occupations of lead exposure include pottery, ceramic-ware production, artisans producing jewellery and decorative wares, which are often a home based operation or located in close proximity to homes involving women and children (Massaro, 1997). The lead fumes and dust generated in such operations also poses health hazard to
children and adults living near these operations (IPCS, 1995). In developing countries the distinction between home and workplace lead exposure is non-existent (Winder, 1993). In Ethiopia, car battery and radiator repair units, paint factories, leaded gasoline stations, plastic factories, jewellery and pottery productions are common in Addis Ababa including some other major towns of the country. The exposure levels at these units are not known and the extents of health dangers are not reported, too.

Occupational diseases share many common characteristics with infectious diseases (Wu et al., 1995). The prevention of occupational hazards is far more effective and less costly when considered during the early stages. However, hazards can be minimized by replacing the hazardous substance with a non-hazardous or by using these substances without exposing workers. If this does not work or completely prevent the exposure, then the emission of the substance to the air should be prevented or minimized. As the last resort, use of personal protective equipment (PPE) including respirator protective equipment (a device which is worn over at least the mouth and nose to prevent the inhalation of air that is not safe) to the people exposed is necessary (WHO, 1999). In order for controls to be effective, continuous supervision and maintenance is necessary. The workplace control measures should be integrated with other measures such as control of emissions to the atmosphere and waterways and waste disposal so that all these measures work together. Moreover, control of exposure to dust should be a key priority to the top management and workers should continually get feedback from the management. Incentive systems for supervisors and workers should be designed to encourage safe procedures and not concern primarily to the productivity. Generally, more resources are placed into dealing with the consequences of harmful occupational exposure rather than prevention of such consequences (WHO, 1999).

On the other hand, in Ethiopia, non-occupational exposure of the general population to lead is most likely to occur through the ingestion of contaminated food or drinking water and by the inhalation of lead particulates in ambient air (from combustion of leaded petrol, especially in urban areas). Direct inhalation of lead accounts for only a small part of the total human exposure, mainly from lead that is adsorbed to soil and inhaled as dust. Fruits, vegetables and grains may contain levels of lead in excess of background levels as a result of direct deposition of lead on to plant surfaces or plant uptake of lead from soils (Carrington et al., 1993). Foods may also become contaminated during processing (Kocak et al., 1989) and food in lead soldered cans, which are
uncommon, may contain high lead levels. Average blood lead levels of adults with no occupational exposure vary widely depending upon factors such as smoking habits, nutritional status, geographic area, and recreational exposures (for example the use of firearms). In most industrialized countries, blood lead levels in adults without occupational exposure are typically less than 20 - 30 µg/dL (Lead, 2001).

Adults and children may be at risk of lead poisoning from the use of lead utensils, lead glazed ceramics, lead crystal, lead foil on wine bottles, the use of contaminated health foods, traditional remedies, aphrodisiacs or cosmetics and crayons (IPCS, 1995). There have also been cases of lead contamination of heroin and lead toxicity has resulted from the intravenous use of heroin and lead contaminated metamphetamine (Norton et al., 1989). The abuse of petrol by inhalation is relatively common in some groups and sniffing leaded petrol can result in lead toxicity (Burns and Currie, 1995).

Although, most occupational standards are based on airborne lead only, this route of exposure does not fully reflect the total daily exposure of workers. There are many other sources of lead exposure. These include dust, air, drinking water, food and contaminated soil. Airborne lead enters the body when one breathes or swallows lead particles or dust when lead has settled (Lead, 2001). Lead pollution arises mainly from car exhausts but industrial processes, batteries, minerals, and lead arsenate insecticide also contribute to lead in the environment (George, 1999). The use of cooking vessels with lead glaze or made of lead may have been another source in earlier times (Lead, 2001). Point sources, such as primary or secondary lead smelters, may create local pollution problems (Winder, 1993). Since coal, like many minerals, rocks and sediments, usually contains low concentrations of lead, a number of other industrial activities such as iron and steel production, copper smelting and coal combustion must be regarded as additional sources of lead emissions into the atmosphere (Patnaik, 1999). Certain individuals, such as traffic policemen, may have higher blood lead levels than the average member of the urban population because they have greater exposure to car exhausts. The lead in car exhausts is derived from tetraethyl lead, an anti - knock compound added to petrol, which is converted to lead in the engine (Timbrell, 2000).

Cigarette smoke is also a source of inhaled lead (IPCS, 1995). The use of car radiators containing lead solder for the illegal distillation of alcohol (moonshine) and residues of lead arsenate pesticides
in soils used to grow grapes have long been sources of lead contamination of alcoholic beverages. Alcoholic beverages tend to be acidic and there is the possibility that large amounts of lead can be dissolved during preparation, storage and serving (Wai et al., 1979). Sherlock et al. (1986) found higher lead concentrations in draught beers, which are considered most likely due to the draught dispensing equipment that sometimes contains brass or gun metal with low but significant amounts of lead. In general, alcoholic beverages do not appear to be a significant source of lead intake for the average person (IPCS, 1995).

1.2. Toxicology of occupational lead exposure

Lead primarily enters the body through ingestion (eating and drinking) and inhalation (breathing in air). It can also pass through the skin. Lead is absorbed, distributed throughout the body, and excreted (Lead, 2001).

The rate of lead absorption into the body depends on the chemical and physical properties of the form of lead and the physiological characteristics of the exposed person such as age and nutritional status (Lead, 2001). For example, when inhaled, factors such as the lead particle size and shape and the individual's ventilation rate influence how lead will be deposited in and absorbed by the respiratory tract. Large particles, which may be encountered in an occupational setting, tend to be deposited in the upper airways and may be directly absorbed by swallowing and absorption in the stomach. Smaller particles tend to be deposited in the bronchial region of the lung, and particles less than one micron, which is typical for urban air, reach the lower respiratory tract where they can be directly absorbed across the thin walls of the alveolar sacs and enter the blood. Ventilation rate is important because altering the inhalation rate may increase or decrease the amount of the lead ultimately absorbed by the lung (Casarett and Doull's, 2001; Lead, 2001). The absorption rate of lead deposited in the lower respiratory tract is governed by the deposition rate and 30-50% of inhaled lead is completely absorbed. A respiratory deposition/absorption rate of 25-45% has been estimated for children. Absorption via the gastrointestinal tract is highly dependent on the presence of the levels of calcium, iron, fats and proteins. Fasting and diets with low levels of calcium, vitamin D and iron have been shown to increase lead absorption. 20-70% of ingested lead is absorbed (Lead, 2001). Gastrointestinal tract absorption of lead in young children is 42%-48% versus 8%-10% in adults (George, 1999).
When lead reaches the blood, it is distributed primarily among the three compartments: blood; soft tissue such as kidney, bone marrow, liver and brain; and in the mineralising tissues such as bones and teeth. About 95% of the lead body burden in adults is located in the bones, compared with about 70% in children, acting as a reservoir, where it is in continuous exchange with the soft tissue pools. Some 99% of the lead in the bloodstream is bound to erythrocytes. The biological half-time of lead in blood can be as short as 20-40 days, and a steady state is thus achieved in about 6 months, although longer half-time values have been reported in lead workers and may depend on the lead body burden (Casarett and Doull’s, 2001). Lead concentration in bones increases with age and this increase is more noticeable in males in the more dense tibial bones. The half-life for lead in bone is approximately 20 to 30 years (Casarett and Doull’s, 2001). Lead may be released from the bones in decalcification processes related to elderly people, pregnancy, acidosis, thyrotoxicosis or active remodelling processes in the bones of children. It has been suggested that lead may be released from bone tissue after the menopause, and clearly higher blood lead levels have indeed been found in postmenopausal than in pre-menopausal women (IPCS, 1995). Its distribution in the body, its affinity for various binding sites, and differences in cellular composition and structure within tissues and organs modulate toxic effects of lead. As a result there is no single well-defined mechanism that explains the toxicological activity of lead in all tissues (Lead, 2001).

Non-absorbed lead passes through the gastrointestinal tract (GIT) and is excreted in the faeces. In adult subjects, of the absorbed fraction, 50-60% is removed by renal and biliary excretion. Intestinal clearance is about 50% of the renal clearance. Some data indicate that children, particularly infants, retain a higher amount of lead. Lead also is excreted in milk and sweat and is deposited in hair and nails. Placental transfer of lead also is known to occur (Klaassen, 1996).

Lead is a very dynamic element with a wide spectrum of biochemical effects in humans. Studies of the mechanism of lead toxicity at the cellular level implicate cell and sub-cellular membranes as a primary target for lead (Massaro, 1997). Lead-induced alterations of ion transport, particularly calcium ions, are related to a number of health effects associated with lead exposure. Effects on ion-transport lead to inhibition of enzymes by binding to SH-groups of its proteins and/or signalling proteins and interfere with normal cellular processes. The mitochondria appear to be particularly sensitive to lead. Lead causes both structural changes and disturbances in mitochondrial function. Mitochondria exposed to lead expand or swell and there is distortion and loss of the small folds of
the inner membrane called the cristae. The mitochondrial enzymes responsible for cellular respiration are largely located within the cristae. Thus, lead uncouples energy metabolism and inhibits cellular respiration. Lead also alters the mitochondrial distribution of calcium (IPCS, 1995). The overall impact of these effects is to disturb the development and functioning of many organ systems (Lead, 2001).

In humans, depending upon the level and duration of exposure the potential targets for lead include effects on haem biosynthesis, the nervous system, the kidneys and reproductive system, the blood pressure and cardiovascular system, hepatic, hearing, endocrinal and gastrointestinal effects (Casarett and Doull's, 2001). In conditions of low-level and long-term lead exposure, such as are found in the general population, the most critical effects are those on haem biosynthesis, erythropoiesis, the kidney, the nervous system and blood pressure (IPCS, 1995). Health problems associated with various blood lead levels are presented in Table 1 (SHARP programme, 1994).

Hippocrates related gout to food and wine, though the association between gout and lead poisoning was not recognized during this period (450-380 BC). Later during the Roman period, gout was prevalent among the upper classes of Roman society and is believed to be a result of the enormous lead intake (George, 1999). Chronic occupational exposure to lead is related to low urate excretion and a high incidence of gout in lead workers (Lin et al., 2002). Lead induced oxidative stress contributes to the pathogenesis of lead poisoning for disrupting the delicate pro-oxidant-antioxidant balance that exists within mammalian cells (Hsu and Guo, 2002). Moreover, bone lead levels are higher in men who work in blue-collar occupations even if they have not worked in primary lead exposed occupations. This effect is even greater in non-white blue-collar workers and suggests an interaction between occupational exposures and race with respect to cumulative exposure to lead (Elmarsafwy et al., 2002).

Workers in the lead smelting, refining and manufacturing industries experience the highest and most prolonged occupational exposures to lead. The major exposure pathways for workers are inhalation and ingestion of lead-bearing dust and fumes. It is also vitally important to note that occupational exposures can also result in secondary exposure for workers' families if workers bring home lead-contaminated dust on their skin, clothes or shoes. Workers should prevent these secondary exposures by showering and/or changing clothes before returning home. Moreover,
people living near battery recycling centres or other industrial lead sources may be exposed to lead and chemicals that contain lead (IPCS, 1995).

Young children are most susceptible to adverse health effects associated with lead exposure due to their developing central nervous systems and their increased ability to absorb lead. Long-lasting impacts on intelligence, motor control, learning, and neurobehavioral development of children have been documented at levels of lead in the body that are not associated with noticeable symptoms and were once thought to be safe. There is no apparent threshold in the level of lead associated with some of these subtle neurological effects. Severe cases of lead poisoning may result in delirium, convulsions, paralysis, coma, and death (Lead, 2001).

However, most people with lead poisoning do not feel sick or poisoned. In spite of the healthy feeling, high lead levels may still seriously affect health. The longer the high levels exist in the blood, the greater the risk of health problems and the damage may be irreversible (SHARP programme, 1994).

**Table 1**: Range of health problems associated with various blood lead levels.

<table>
<thead>
<tr>
<th>Lead Levels in the Blood (µg/dL)</th>
<th>Severity of Health Problems</th>
<th>Effects of Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-15</td>
<td>Typical level for adults</td>
<td>Average adult blood lead levels in US</td>
</tr>
<tr>
<td>15-20</td>
<td>Symptomless</td>
<td>Foetal effects in pregnant woman</td>
</tr>
<tr>
<td>20-55</td>
<td>Symptomless lead damage. Lead starts to build up at + 20 µg/dL</td>
<td>Decreased blood production; male infertility; nerve damage; decreased hearing; increased blood pressure</td>
</tr>
<tr>
<td>55-80</td>
<td>Serious health damage may happen</td>
<td>Anaemia, kidney failure and reduced neurobehavioral abilities</td>
</tr>
<tr>
<td>80-110</td>
<td>Severe health damage that may occur quickly and be permanent</td>
<td>Brain damage (Encephalopathy)</td>
</tr>
</tbody>
</table>
1.2.1. Effects of lead on the nervous system.

In humans, lead causes damage to a variety of organs, particularly the central nervous system (Massaro, 1997). The mechanisms for lead neurotoxicity are not well understood. One of the mechanisms by which lead may affect brain physiology and biochemistry could be due to either a direct influence upon nerve endings or by influence on neurotransmitter release in some fashion. Low lead concentrations enhance the release of neurotransmitter from presynaptic endings (Cooper et al., 1984). Lead has been shown to stimulate the release of dopamine, acetylcholine, and gamma-aminobutyric acid (GABA) (Minnema et al., 1988). Some of these effects may be due to the ability of lead to alter calcium entry into nerve cell or by an increase in the intracellular calcium levels. Lead may enter cells through calcium channels. Calcium may also competitively inhibit lead uptake in non-excitatory cells such as the adrenal medulla and cannot be discounted as the potential mechanism in nerve cells. Calcium channel blockers likewise may inhibit lead uptake in the same cells (Simons and Pocock, 1987.)

Lead is known to substitute for calcium as a second messenger and can bind to calmodulin. In fact, calmodulin has a greater affinity for lead than for calcium. The calcium calmodulin complex may activate a kinase referred to as calmodulin dependent protein kinase, which is high in nervous tissue and may regulate neurotransmitter release. Synapsin I when phosphorylated is believed to have a role in neurotransmitter release. If lead acts as calcium in the activation of this kinase, this may explain the ability of lead to result in neurotransmitter release (Massaro, 1997). Theoretically, these effects are reversible if lead is removed from the synapse. However, exposure to lead for a long time may result in permanent alteration in cellular responsiveness at pre-synaptic levels.

For over a decade, the hippocampus has been considered to be the principal target of lead in the brain because:

1. The hippocampus contains relatively high concentrations of zinc, and zinc-dependent functions may be sensitive to lead as lead is divalent metal and often competes with divalent ions such as zinc with respect to biochemical processes,
2. The hippocampus contains a dense plexus of cholinergic fibers that are affected by lead exposure, and
3. The hippocampus functions in memory and learning (IPCS, 1995).
Lead appears to impair hippocampal voltage potentials through protein kinase C. Protein kinase C, which is very sensitive to lead, modulates receptor currents affecting long-term potentiation and other forms of synaptic response that may underlie learning and memory (Massaro, 1997). Lead may serve to activate this kinase and consequently inhibit this potential, thereby impairing memory and learning.

Repeated lead exposure of moderate to high levels can cause encephalopathy (a progressive degeneration of certain parts of the brain). Early symptoms of encephalopathy include dullness, irritability, poor attention span, headache, muscular tremor, loss of memory and hallucinations. More severe symptoms occur at very high exposures and include delirium, lack of coordination, convulsions, paralysis, coma and death (Lead, 2001).

The encephalopathy induced by lead toxicity is most likely due to a compromise in blood brain barrier. Brain oedema occurs in the interstitial area and appears due to compromised blood vessel integrity. The brain capillaries and blood vessels have endothelial cells that contain tight junctions and act as a seal or barrier that excludes many plasma proteins and organic molecules and impedes Na\(^+\) and K\(^+\) exchange (Bradbury, 1984). Elevated lead levels disrupt these vessels, and plasma proteins such as albumin enter the interstitial spaces, as do some ions. This increases osmotic pressure, and water accumulates in response. The lack of lymphatic structures within the central nervous system means that the fluid flows into the cerebrospinal fluid. This oedema causes an increase intracranial pressure and restricts blood flow to the brain, resulting in ischemia (Goldstein et al., 1974).

The direct mechanism by which the blood brain barrier and blood vessels that compose the barrier are compromised may be due to the astrocytes appearing to be vulnerable to the toxic effects of lead. The astrocytes cover the vascular walls of the brain vessels, and the permanent neuropsychological deficits seen after exposure to high lead concentrations may be due to impairment of astrocytes function especially in their capacity to regulate the ionic and amino acid concentrations in the extracellular milieu, brain energy metabolism and cell volume. Encephalopathy has been observed in adults at blood lead levels exceeding 1200 \(\mu\)g/L, in children at levels of 800-1000\(\mu\)g/L. The outcome is frequently fatal in children, and those who survive often present with irreversible neurological and neuropsychological sequelae (Perlstein and Attala, 1966).
Consistent neurobehavioral effects, which are to be considered as ‘adverse’ include recurrent seizures, confusion, dizziness, lethargy, headache, disorientation and other CNS symptoms appear in a multiplicity of studies at lead blood levels of ≥40 µg/dL (Whitfield et al., 1972; Haenninen et al., 1979; Fischbein et al., 1980; Awad El Karim et al., 1986). The dose dependency of impairments in performance in neurobehavioral tests, in relation to blood lead levels, may also be drawn from the studies of Mantere et al. (1982) and Stollery et al. (1991). In general, decreases in global performance are reported at blood lead levels > 40µg/dL (Landrigan, 1990). It should be mentioned that the behavioural studies have been almost exclusively conducted in males where (limited numbers of) females are also involved; generally no valid gender specific data are provided.

Lead induces degeneration of the protective Schwann cells in the motor neurons of the peripheral nervous system (nerves of the arms and legs). This cause segmental loss of the myelin covering of the neuron, which leads to decreased nerve conduction velocities and possible neuron degeneration. Lead induces a breakdown in the blood-nerve barrier, allowing lead and fluid to enter the endoneurium and disrupt the myelin membranes. The degeneration of sciatic and tibial nerve roots is also possible. Sensory nerves are less sensitive to lead than motor nerves. Peripheral neuropathy is usually, present only after prolonged high exposure to inorganic lead compounds. This disorder is often referred to as "lead palsy" and symptoms include weakness of the arms and legs (foot drop) and weakness and paralysis of the wrist (wrist drop), fingers and ankles (Lead, 1991).

A study of occupationally exposed workers indicates that motor nerve dysfunction can occur at blood-lead levels below 70 µg/dL, possibly as low as 30µg/dL (Seppalainen et al., 1983), when assessed clinically by the electrophysiologic measurement of nerve conduction velocities. There is some evidence that these effects may be reversible following a 5-month exposure. However, only partial recovery may occur, particularly if lead exposure continues or treatment is not carried out (IPCS, 1995).

Several studies have measured the conduction velocity of electrically stimulated sensory and motor nerves in workers exposed to lead. However, these studies have yielded somewhat mixed results, with many showing a decrease in Nerve Conduction Velocity (NCV) in relation to lead exposure (generally indexed as blood lead concentration) and a few (Araki et al., 1980; Spivey et al., 1980;
showing no effect or occasionally even an increase in NCV associated with lead exposure (reviewed in Davis and Svendsgaard, 1990).

Various reasons may underlie this lack of uniformity in NCV findings. For example, studies may differ in methodological features, in the characterization of lead exposure (e.g. single time-point versus time weighted average blood lead levels) or in the handling of confounding variables such as nerve temperature and age of subjects. Other important factors accounting for some of the apparent inconsistency in this area of research may be the possible antagonistic effect of zinc to lead and differences in nerves selected for measurement in different studies (e.g. slow versus fast fibers)(Murata and Araki, 1991).

Circulating and excreted levels of Aminolevulinic acid (δ-ALA) are likely to be best described as a continuum of effect. Elevated levels of this compound are of importance since neurological features of lead exposure have been ascribed in part to increased circulating levels of δ-ALA (IPCS, 1995).

1.2.2. Effects of lead on haem biosynthesis and erythropoiesis

The normal process of haem biosynthesis and its disturbance by lead are well understood. On the cellular level, the initial and final steps of haem formation are mitochondrial whereas the intermediate steps take place in the cytoplasm (Lead, 2001).

As an overview, porphyrin biosynthesis begins with the condensation of glycine and succinyl CoA, a tri-cyclic acid (TCA) cycle intermediate, to form α-amino-β-keto adipic acid in the presence of the enzyme aminolevulinic acid synthase and vitamin B6. The complex undergoes decarboxylation to form δ-ALA. This reaction occurs in the mitochondria. Subsequently, the compound goes to cytoplasm where two molecules of δ-ALA condense to form porphobilinogen via the dehydratase enzyme. This enzyme is also a zinc-requiring enzyme. The porphyrin haem ring is formed essentially by condensation of four monopyrroles synthesized from the Porphobilinogen (IPCS, 1995).

Lead is known to affect several enzymatic reactions critical in haem synthesis, causing abnormal concentrations of haem precursors in blood and urine. Essentially, lead interferes with the activity of three enzymes:
1. It indirectly stimulates the mitochondrial enzyme Aminolevulinic acid synthetase (ALAS);
2. It directly inhibits the activity of the cytoplasmic enzyme Aminolevulinic acid dehydratase (ALAD); and
3. It interferes with the normal functioning of intramitochondrial ferrochelatase, which is responsible for the insertion of iron (II) into the protoporphyrin ring (Timbrell, 2000; Casarett and Doull's, 2001).

The ALAS and the ALAD enzymes are present in mitochondria and their rates of operation are controlled by feedback (end-product) inhibition by haem (Labbe and Lamon, 1987). The rate-limiting step in the biosynthesis of haem is that catalyzed by the first enzyme in the pathway, ALAS. The amount and activity of this enzyme is regulated so that only trace amounts of the intermediate products in haem synthesis are present. A fall in the haem concentration results in an increase in the activity of ALAS, and an increase in the amount of the enzyme. The level of δ-ALA in blood or urine will increase (Makino et al., 2000; Murata et al., 2003). High levels of activity of ALAS are present in erythroid cells in the bone marrow and also in liver cells (IPCS, 1995). ALAS stimulation has been found in lead workers at blood lead level of about 400 µg/L (Meredith et al., 1978). The ALAD enzyme is involved in synthesis of the porphyrin units. Roels et al. (1976) were unable to determine a threshold for ALAD inhibition in school-age children with blood lead concentrations of 30-300 µg/L, although first effects appeared to be associated with a blood lead of about 100µg/L.

Interference by lead with the formation of haem from protoporphyrin is apparent from increased blood and urine levels of free erythrocyte protoporphyrin (FEP) and coproporphyrin III. The FEP combines with Zinc in the blood to form Zinc protoporphyrin (ZPP), which is the moiety assayed (Hathaway et al., 1991; Gosselin et al., 1984). Whereas ZPP is likely to be confounded by iron status at lead concentrations below 200µg/L (Marcus and Schwartz, 1987), FEP should be a more valid indicator of lead-induced disruption of haem formation at lower blood lead. Based on data from 2004 urban children, a threshold for FEP elevation of 150-180mg/L was found (Piomelli et al., 1982). A reanalysis of the Second National Health and Nutrition Examination Survey (NHANES II) data on 264 children yielded a threshold of 200µg/L (Marcus and Schwartz, 1987).
Effects of lead on erythropoiesis and erythrocyte physiology represent more direct signs of damage to the haemopoietic system than haem precursors in blood or urine. Anaemia is a frequent outcome of chronic lead intoxication. The increases in δ-ALA and FEP are earlier indicators of anaemia, which is generally not observed until the blood concentration reaches 40µg/L (Massaro, 1997). Lead often leads to anaemia by several mechanisms: (1) competing for absorption with the ferrous iron form, (2) inhibiting haem synthesis; and (3) altering the relative composition of cell membranes, including red blood cells, that make them more fragile and likely to haemolyse when passing through tiny capillary spaces.

Bashir et al. (1995) investigated whether there is correlation between lead in blood, exposure and anaemia. These researchers concluded that chronic lead exposure causes normocytic normochromic anaemia and showed dose-response relationship between lead levels and severity of anaemia. In contrary to this, Froom et al. (1999) found that blood samples obtained from 94 workers in lead–acid battery plant in Israel between 1980 and 1993 exceeded 60 µg/dL in 14% of the blood samples. They found no correlation between haemoglobin and blood lead levels. These authors suggested that, a diagnosis of anaemia in a person with blood lead levels up to 80µg/dL should be considered to be due to lead toxicity only after other causes for anaemia are ruled out.

Some other lead exposure studies (Hodgkins et al., 1991; Kononen, 1991; Gittleman et al., 1994; Makino, et al., 1994; Chuang et al., 1999; Suplido and Ong, 2000; Nuwayhid et al., 2001) in lead acid storage battery workers measured different blood lead levels varied with the nature of the industry (battery manufacturing or recycling units). They also found that variations in blood levels are influenced by the employment duration, job category, age, gender, smoking habits and ethnicity.

A threshold effect for occupationally exposed adults was estimated at 500µg/L (IPCS, 1995). Impairment of the erythropoietic system to regenerate from phlebotomy was found in lead–exposed workers with an average blood lead of 445 µg/L relative to controls (Grandjean et al., 1989).
The sensitivity of the haem synthesis pathway to increased lead exposure was in the order: children ≥ women ≥ men. On balance it would appear that lead has discernible effects on the urine level of δ-ALA at a blood lead level of around 1.68µmol/litre (35 µg/dL) (IPCS, 1995).

1.2.3. Effects of lead on renal function

One target organ of lead exposure is the kidney, which given sufficient lead exposure levels over a sufficient length of time may succumb to renal failure. Goyer and Ryne (1973) reported that the proximal tubules of the nephron are lead sensitive. It is manifested clinically by decrease in energy dependent transport functions, including aminoaciduria, hypophosphataemia, glycosuria, and ion transport. The renal proximal tubules are rich in mitochondria and swelling of mitochondria can be observed at a relatively low lead concentrations. The functional changes are thought to be related to a lead effect on mitochondrial respiration and phosphorylation. In exposed workers distortions of the proximal tubules have been noted after a couple of months of exposure.

Furthermore, it is well known that the active form of vitamin D₃ (1, 25-dihydroxycholecalciferol) is produced in the proximal tubules. There is evidence that lead impairs haem containing enzyme systems in the kidney that are involved in vitamin D metabolism. Vitamin D synthesis requires a haem containing hydroxylase enzyme in the kidney for the hydroxylation of 25-hydroxyvitamin D to 1, 25 – dehydroxyvitamin D, which is important in the gastrointestinal absorption of calcium. These effects may occur with lead levels as low as 30µg/dL, below the levels of lead that alters other biomarkers for nephrotoxicity (Rosen et al., 1985).

Typical measures of renal failure, e.g. blood urea nitrogen (BUN) and creatinine are elevated as a consequence of lead-induced renal failure. However, both Serum Creatinine (SC) and BUN are rather insensitive indices of Glomerular Filtration Rate (GFR); a 50 to 70 percent decrease in GFR must occur before increases in Serum Creatinine and BUN develop. These tests are unlikely therefore to detect mild or moderate renal impairment due to lead toxicity and are unlikely to discover a lead nephropathy – because the kidney has a great reserve capacity, these measures of excretory function can be normal or in the normal range despite major unrecognised impairment of kidney function (Massaro, 1997).
Occupational studies on lead-related renal dysfunction found a particular sensitivity to lead for N-acetyl-β-D-glucosaminidase (NAG), a lysosomal enzyme present in renal tubular cells and considered a sensitive but non-specific indicator for early sub-clinical nephrotoxicity. Excretion of blood lead and NAG was found to be associated with blood lead levels below 600 µg/l (Ver-schoor et al., 1987), although negative reports exist as well (Gennart et al., 1992).

Reversible kidney injury has been observed in some workers with repeated low exposure to inorganic lead compounds. Irreversible kidney damage has been observed following long-term, moderate exposures (Gennart et al., 1992). Higher blood lead levels can lead to protein lead complexes in the tubules, which appear as dense accumulations. Proximal tubular dysfunction is usually not demonstrable in the chronic phase of lead nephropathy; but interstitial fibrosis, tubular atrophy and dilatation and arteriosclerotic changes are usually associated with asymptomatic renal azotemia and reduced GFR. Continued accumulation of lead by the kidneys often leads to an increased accumulation of fibrotic connective tissue. These have been found in lead workers with excessive lead exposure for more than two years but these workers did not exhibit renal failure until many years later. It has also been found in workers exposed over ten years (Goyer and Ryne, 1973).

Lilis et al. (1979) conducted two clinical field studies of secondary lead smelter workers. These researchers assessed BUN and Creatinine levels with respect to duration of lead exposure. These studies indicated that a sizeable and significant decrement in kidney function in the secondary lead smelter workers studied was found to be lead-induced. Moreover, the outcome of these studies showed that the decrement of kidney function is age-dependent.

An increased number of deaths due to kidney disease were observed in smelter and lead production workers with moderate lead exposure. There is also some evidence to suggest that chronic low-level environmental lead exposure may affect kidney function (Bernard and Becker, 1988).

Pinto de Almeida et al. (1989) assessed Brazilian lead workers and found that renal dysfunction of workers from Lead exposed group was statistically associated with duration of employment at the smelter and with age. These authors also reported that 32.7% of the exposed group had a greater SC level and these workers also had high mean serum Uric Acid (UA) levels. Moreover, the levels of lead and zinc in blood and δ-ALA did not affect the renal function.
Omae et al. (1990) conducted a cross-sectional study on 165 male lead exposed workers to clarify the quantitative relationship between less severe exposure to lead and its effects on renal function. The mean blood lead concentration was 36.5 µg/dl. The duration of lead exposure was 0.1 to 26.3 years. Renal function indices of these workers from 1972 to 1984 were not different from those of remaining lead-exposed workers whose lead exposure duration was 10 years or less. These authors concluded that long-term and less severe exposure to lead up to 70 µg/dl of blood lead levels might not cause adverse effects on renal glomerular function and proximal tubular function.

Ehrlich et al. (1998) investigated South African battery workers for the association between inorganic lead exposure, blood pressure and renal function. The mean blood lead level was 53.4 µg/dl and the mean exposure duration was 11.6 years (range 0.5 to 44.5 years). The mean historical blood lead level on 246 of 382 workers was 57.3 µg/dl. After adjustment for age and other confounding parameters, it was found that an exposure response relation between lead and renal dysfunction across the range from less than 40 µg/dl up to greater than 70 µg/dl blood lead levels existed. This was found with conventional measures of short and long term lead exposure and of renal function. These authors believe that their finding probably reflected a higher cumulative renal burden of lead exposure among industrial workers in South Africa.

Lim et al. (2001) looked at the renal dysfunction of workers exposed to lead. These authors found that there is a positive correlation between the overall lead exposure and renal dysfunction. The renal parameters were significantly higher among those subjects with atleast one case of blood lead levels above 60 µg/dl.

Wang et al. (2002) investigated the correlation between blood lead levels and renal function indices of BUN, SC and UA among lead battery workers who were exposed to lead. These authors reported that blood lead levels higher than 60 µg/dl had increasing chances of inducing adverse renal effects.

1.2.4. Effects of lead on digestive and hearing systems

Effects on the gastrointestinal tract tend to be observed following high exposure to inorganic lead compounds, although they have sometimes been noted in workers with moderate exposure. Symptoms include loss of appetite, inflammation of the stomach walls (gastritis) and colic, with
severe abdominal pain, cramps, nausea, vomiting, constipation, weight loss and decreased urination. In severe cases of lead exposure, a deposit of lead occurs in the gums near the base of the teeth. This deposit is visible as a blue-grey line (Massaro, 1997).

Wu et al. (2002) conducted a study to investigate the effects of lead and noise exposures on hearing ability. These authors found a significant correlation between a high, long-term lead exposure index (defined by duration of employment and ambient lead concentration) and decreased hearing ability. They also reported that lead via different systems may damage hearing ability and in some cases may cause severe and irreversible damage. However, neither noise exposure alone nor the interaction between noise exposure level and short or long-term lead exposure were correlated significantly with hearing ability. These authors then concluded that measures should be taken against lead exposure for preservation of workers' hearing ability.

1.2.5. Effects of lead on reproductive system

Lead toxicity is known to influence male and female reproductive organs in humans. Increased incidences of spontaneous abortions have been documented in female lead workers and also in the wives of male lead workers (Landrigan, 1991). A few epidemiological studies have been performed on the association between paternal exposure to lead and adverse reproductive outcome. The results suggest an increased risk of spontaneous abortion, prenatal death and low birth weight following paternal occupational lead exposure (Lindbohm et al., 1991; Kristensen et al., 1993). In a Finnish study, a significant increase was observed in the risk of spontaneous abortion among the wives of men whose blood lead was 30 µg/dL or higher during spermatogenesis (Lindbohm et al, 1991).

Reduced fertility has also been reported for men with a long duration of lead exposure (Lin et al., 1996). Coste et al. (1991) conducted a cohort study in a French battery factory from 1977-1982 to explore the relationship between occupational exposure to lead and fertility. Findings of this study were that lead exposure at any level of absorption did not appear significantly associated with a reduction in fertility after controlling confounding factors such as age, French origin, educational level, number of children at start of the period of work, cigarette smoking and exposure to heat.

There is limited evidence of an association between reduced sperm quality (reduced sperm count and motility and increased morphologically abnormal sperm) and blood lead in excess of about 40
Studies of the association between occupational lead exposure with semen quality and infertility among male workers have produced conflicting results. Robins, et al. (1997) studied workers from a South African lead-acid battery factory. The results obtained were that lead in semen ranged from 1 to 87 µg/dl. There was a significant correlation between an increased percentage of sperm with abnormal morphology and higher measures of current blood lead, cumulative blood lead and duration of exposure. However, there were no associations of sperm density or sperm count with any of the lead-exposed measurements. The only valid outcome of that study was the relatively high range of current blood lead levels and high prevalence of abnormalities in semen quality. From a review on male reproductive toxicity of lead (Apostoli et al., 1998), it seems evident that only lead levels above 40 µg/dL in blood are associated with a decrease in sperm count, volume and morphological alterations.

1.2.6. Effects of lead on blood pressure and cardiovascular system

General population studies did not find associations between blood lead levels and cardiovascular morbidity and mortality (Pocock et al., 1988). Possible associations between blood lead and blood pressure have been studied in several large-scale population studies such as the British Regional Heart Study and the US NHANES II (National Health and Nutrition Examination Survey), as well as studies in Belgium, Canada, Denmark and Wales.

Pinto de Almeida et al. (1989) compared a group of 52 workers of a primary lead smelter located in Northeast Brazil to a reference group of 44 workers from a paper mill. These researchers found a strong association between hypertension and renal dysfunction in the lead workers.

A meta-analysis of the available studies by Staessen et al. (1993), covering 19 studies with altogether 28, 210 subjects, revealed similar associations between both sexes. A two-fold increase in blood lead was associated with a 1-mmHg increase in systolic and a 0.7-mmHg increase in diastolic blood pressure. It is concluded that there is a significant weak positive association, possibly without public health implications for hypertension. A causative role for lead is considered unlikely.

Wu et al. (1996) assessed the relationship between occupational lead exposure and elevated blood pressure with consideration of a possible confounding effect of noise exposure. It should be noted
that studies done by these authors showed no relation or correlation between lead exposure and blood pressure. Gerr et al. (2002) in their study that investigated the association between bone lead concentration and blood pressure among young adults concluded that substantial lead exposure during childhood could increase blood pressure during young adulthood.

1.3. Physical effects of lead

The usual components of the car battery are 60% lead, 40% sulphuric acid (10% solution) and polypropylene as the plastic case of the battery. Lead powder poses a higher risk to cause fire than the solid because of its greater contact area with air with resultant higher ignition characteristics. Lead dust cloud has similar risk hazards like gas in causing fire. Accumulations of lead dust should be removed regularly by techniques, which do not generate a dust cloud such as damp sweeping or vacuum cleaning. Also, the electrical equipment in areas where flammable solids and powders are handled or occur must be designed and maintained to the appropriate flameproof standard (Ridley and Channing, 1999). Airborne flammable lead dust in sufficient concentrations can explode posing danger of accidents. The combustible lead dust on the ground may become airborne which may increase and propagate an explosion initiated by flammable gas ignition. This occurs in all oxidisable dusts (WHO, 1999).

1.4. Lead exposure preventive and control measures

Preventive and control measures are meant for reducing the employee’s exposure to lead at workplaces. Workplace design or occupational health includes physical arrangement of employees immediate work area and ambient environmental qualities of the work area (Stokols, 1997). The permissible lead emission values set should ensure that the exposure for a worker does not exceeded the emission threshold limit value (Gerhardsson, 2002). Procedures for health risk assessment of population groups who are at risk of exposure to lead should be directed towards reduction and prevention of the exposure. Risk assessment and management procedures including development of regulatory options are shown in Fig. 1. Workplace assessment entails a process that identifies existing and potential hazards and provides appropriate measures to protect the health and safety of employees (Winder, 1993).
1.4.1. Personal Protective Equipment

Personal Protective Equipment comprises of the disposable facemask, air stream helmet, safety hat, goggles, earmuffs, gloves, safety boots and an overall coat.

In the USA, the Occupational Health and Safety Act /OSHA/ requires the use of PPE to reduce employee's exposures to hazards as the last resort when engineering and administrative controls have failed in reducing the exposures to acceptable levels. However, if PPE is to be used, a PPE programme should be initialised and maintained. The programme should include identification and
evaluation of hazards in the workplace, selecting of an appropriate PPE to be used, maintenance of PPE and its use evaluated. Also, employees should be trained on how the PPE is used.

Stellman and Osinsky (1997) also noted that workers who are exposed to lead should be provided with PPE that should be washed or renewed regularly. According to lead Regulations of 2001, it is stated that:

1. ‘Employers should ensure that the relevant PPE is capable of keeping the exposure at or below the occupational exposure limit for the type of lead used in the industry,
2. The relevant equipment is correctly selected and properly used, training on how to use the PPE is provided,
3. The equipment is kept in good condition and efficient working order,
4. Employer issues no personal protective equipment that has already been used by another person, unless the relevant protection equipment is properly decontaminated and where appropriate sterilised’.

1.4.2. General Ventilation

General ventilation is usually desirable to control the temperature and humidity of the environment or a properly designed system that can act as a back-up control of exposure to airborne substances by diluting the airborne contaminants (WHO, 1999).

A specially trained professional must design the ventilation system in such a way that movement of personnel or the opening of doors and windows do not obstruct the system. Managers should ensure a continuous inspection and maintenance programme for ventilation systems to be effective. They should also involve workers about the use and maintenance of the system (WHO, 1999). In addition, this inspection should be synchronized with work environment monitoring by air sampling to ensure that the plant is continuing to operate effectively (Ridley and Channing, 1999).

1.4.3. Containment

Containment involves placing a barrier between the substance and the people. When the substance is contained and enclosed, it is necessary to have a ventilation system that keeps the enclosure under negative pressure to avoid any leakage or air emission of the substance in and out of the
enclosure. The design of the enclosure has to enable good maintenance and cleaning of the system without causing high exposure. It is acceptable to partially enclose a process by having an opening at the front of an enclosure for maintenance. The worker's breathing zone should not be between the contaminant source and the enclosure (WHO, 1999).

1.4.4. Local Exhaust Ventilation

Local exhaust ventilation (LEV) is the removal of airborne contaminants close to their source of generation or release before they can spread or reach the worker's breathing zone (WHO, 1999). LEV is intended to control mechanically the emission of contaminants such as dust and fumes that are given off during the repair process. Normally, this is done as close to the point of emission as possible using a stream of air to remove the airborne particulate matter and transport it to where it can be safely collected for ultimate disposal. The physical layout and setting of LEV equipment is critical for it to work effectively and comparatively minor alterations can affect its performance. It is therefore important that LEV equipment should be properly designed, manufactured, installed, operated and maintained (Ridley and Channing, 1999). Also it is necessary to ensure that the airflow is sufficient and its direction of flow is appropriate (i.e. away from the workers' breathing zone).

Other types of air emission control systems may be used including wet scrubbers, absorption and adsorption systems, combustion and electrostatic precipitation. All systems generally produce a solid waste from the air emission and release the cleaned air.

Matte et al. (1989) assessed lead exposure in lead acid battery industry in Jamaica. They reported that engineering controls and respiratory protection were observed to be inadequate in the industries studied. Due to these workers had higher mean blood lead levels (> 60µg/dl) and tended to have higher prevalence of most symptoms of lead toxicity. Chuang et al. (1999) also suggested an improvement in engineering controls for reduction of exposure to lead in workers.

Hodgkins et al. (1991) investigated a relationship between air lead and blood lead levels in 132 lead acid battery workers in two plants. These workers were followed for 30 months between 1985 and 1995. Their frequent air lead exposures and lead in blood were determined. These plants converted to more modern technologies around 1978 with associated reductions in mean lead in air exposures
from greater than 100µg/dl to less than 30µg/dl. On the other hand, in Taiwan, the Ministry of Health developed an obligatory surveillance system for blood lead. This assisted in upgrading the occupational disease control to the stage of specific prevention and health promotion (Wu et al. 1995). Further, Jakubowski et al. (1998) conducted a study to evaluate the effectiveness of the directive from the Minister of Health and Social Welfare, Poland in 1996 stating that the blood lead levels determinations in employees occupationally exposed to lead was compulsory. The result of that study indicated that exposure to lead continues to be a serious problem in Polish industry. Lead in blood concentrations exceeded the Polish biological exposure index (BEI) value of 50 µg/dl for men. These results clearly showed the need to improve on compliance of industries to ministerial ordinance.

The improvements of hygienic practice were more effective at lowering blood lead levels than reducing ambient lead level (Lai et al., 1997). These authors concluded that hygienic practice might be the preferential way to reduce lead exposure, especially in developing countries as compared to the engineering controls. Chao, et al. (2002) also reported that in Taiwan there were several reports about elevated blood lead levels in lead battery workers. These authors visited all registered lead acid battery plants in Taiwan and collected their health examination records. The average blood lead concentration was found to be 37.1 µg/dl and 37% of blood lead levels were more than 40 µg/dl, the action level set by the Department of Health, Taiwan. From the results of this study it was suggested that analysis should be performed each year to monitor the effectiveness of occupational hygiene in the workplace of lead battery plants.
2. OBJECTIVES

2.1. General Objective
In developing countries, lead-acid battery repair units are very common and clinical lead poisoning is one of the most important occupational diseases. Moreover, lead exposure at workplaces is likely unregulated (George, 1999). It is therefore important that the exposure levels in workplaces that use lead should be at acceptable ranges. In Ethiopia, biochemical lead exposure studies and Lead Regulations are not available to protect health of workers, that could influence the turnover of the enterprises. The present study was therefore aimed at investigating lead exposure among lead-acid battery repair workers and relates the exposure to health effects.

2.2. Specific Objectives

1. To measure levels of urinary δ-ALA from workers in lead acid battery repair units of Transport Service Enterprises and compare with age-matched non-exposed groups.
2. To assess the effect of duration of exposure on levels of urinary δ-ALA.
3. To investigate the effects of alcohol, age and sex on urinary δ-ALA levels.
4. To compare urinary δ-ALA levels among different enterprises.
5. To measure SC, CC, Urea and UA levels of exposed and non-exposed subjects.
6. To indicate appropriate and cost-effective preventive and control measures to reduce lead exposure at the repair units.
7. To increase general awareness in lead hazards among the public in general and exposed workers in particular.
3. MATERIAL AND METHODS

3.1. Study Design and Period

This cross-sectional study was conducted from November to May 2005 in storage battery repair units for lead exposure assessment. The study was designed after identifying that lead acid-battery is the potential source for lead exposure.

3.2. Study Area

The study sites were operating as repair units of lead acid battery in Addis Ababa, Ethiopia. The study sites were five in number, i.e. Gerji, Kaliti, Lideta, Mekanisa and Shegole.

Working procedure of the storage battery repair at the enterprises include:

1. Water is distilled using distiller and stored in plastic jars.
2. Battery composition is prepared by mixing 2 parts of H$_2$SO$_4$ (10% solution) and 3 parts of distilled water in a polypropylene as the plastic case of the battery. While mixing acid with water, vapour is formed and cooling of the plastic case is done using water. This cooling process lasts for three days.
3. Then the battery cases are filled with the cooled mixture using graduated plastic jug.
4. Specific gravity of the solution is measured using Hydrometer. The specific gravity is required to be between 1.250 and 1.275. If the mixture meets the required specific gravity range, the slow charging process will be done after few minutes and lasts for 3 to 4 hours. Then the battery is tested with voltmeter for its exact voltage, i.e. 6V, 12V, or 24V.
5. The storage battery will be ready for use in buses and heavy trucks after cooling for 2 hours.

3.3. Source and Study Population

The source population was workers in car battery repair units of the enterprises while the study population comprised of workers who had served for over six months in these units.
3.4. Sampling Technique and Sample Size

Purposive sampling method was used and a total of 51 lead acid battery repair workers (45 male & 6 female) whose age range from 23 to 57 were identified and included in the study. Fifty healthy non-exposed age matched subjects (48 male & 2 female) were considered for comparison with the exposed group. Independent variables were age and duration of exposure, while the dependent variables included δ–ALA in urine, renal indices, uric acid levels and occupational illnesses. Demographic characteristics of subjects are shown in Table 2.

3.5. Data Collection and Measurement

3.5.1. Structured Questionnaire

An anonymous questionnaire, which contains both open and close-ended questions, was used to collect information regarding personal data, work practices and health risks (see Appendix A). The same questionnaire was administered and specimens were collected from non-exposed subjects, too. Informal interviews with technical directors and walk through evaluation of the repair units was also conducted.

3.5.2. Laboratory Analysis

i) Sample Collection

Two hours urine samples were collected between 9:00 and 11:00 a.m. from study participants in the repair units using light protected plastic urine containers (aluminium foil wrapped), which contained 2 g barbituric acid as preservative. The measurement of volumes was done using graduated cylinder. In parallel, 3ml blood samples were collected; centrifuged; and serum was separated for analyses. Both serum and urine specimens were refrigerated at 4°C immediately in the dark until analyses. δ-ALA stability is for a maximum of 2 weeks if stored at around pH 3 to 4 in the dark. Identification of specimen with participant name, the date and the time of collection directly on the container and securing it with tape were done (Henry, 1984; Labbe and Lamon, 1987; Tietz, 1987).
ii) Sample Analysis

**Measurement of urinary δ-ALA**

The level of urinary δ-ALA was determined spectrophotometrically according to the method of Tomokumi and Ogata (1972). Briefly, the urine samples were heated with buffered ethyl acetoacetate to produce pyrrole derivatives. This δ-ALA derivative was purified by extraction into ethyl acetate. Ehrlich reagent was then added to produce a reddish colour and absorbance was measured at 553 nm. For the analyses four tubes were prepared as follows:

**Tube A:** Water blank (1ml water +1ml acetate buffer + 0.2ml ethyl acetoacetate + 3ml ethylacetate + 2ml Ehrlich’s reagent).

**Tube B:** Subject specimen blank (1ml urine + 1ml acetate buffer + 3ml ethylacetate + 2ml Ehrlich’s reagent).

**Tube C:** Subject specimen (1ml urine + 1ml acetate buffer + 0.2ml ethyl acetoacetate + 3ml ethylacetate +2ml Ehrlich’s reagent).

**Tube D:** Subject specimen (1ml urine + 1ml acetate buffer + 0.2ml ethyl acetoacetate + 3ml ethylacetate +2ml Ehrlich’s reagent).

Tube A served as a blank for tube B while tube B was blank for tubes C and D. All tubes were heated in a boiling water bath for 10 minutes and allowed to cool in cold water. Then the glass stoppers were removed and centrifuged (1000g x) for 1 minute to separate the phases. 2ml of the upper ethyl acetate phases were removed using volumetric pipette. Then, Ehrlich’s reagent was added, mixed, left for 10 min. and the absorbance at 553nm was taken using water to zero the spectrophotometer. The urinary levels of δ-ALA, as used for index of lead exposure, were then categorized into four groups, i.e. normal range (< 0.6mg/100ml), acceptable (0.6-2.0mg/100ml), high (2.0 – 4.0mg/100ml) and dangerous (> 4.0mg/100ml) (Lane et al., 1968).
**Measurement of creatinine clearance**

Creatinine Clearance was used to assess GFR function after determining the serum and urinary creatinine concentrations and urine volume over 2 hours. Fluitest kit (Biocon® Diagnostic Hecke 8, 34516 Vöhl/Marienhagen, Germany), based on Jaffe Kinetic Colorimetric Method was used for the determination of creatinine. Measurements were done using AMS Autolab analyzer (Roche, Basel, Switzerland) in Clinical Chemistry Laboratory of the Ethiopian Health and Nutrition Research Institute.

**Measurement of urea and uric acid**

Urea Kit (Biocon® Diagnostic Hecke 8, 34516 Vöhl/Marienhagen, Germany), based on Berthelet Method was used for the determination of urea, whilst Uric Acid was analyzed using Uric Acid PAP Kit from Human Biological Diagnostic, Germany. These tests were also run on the same AMS Autolab analyser described above.

**3.6. Quality Control**

- The questionnaire was pre-tested and standardized before data collection began.
- Instruments were calibrated and test kits were quality controlled prior to analysis.
- Standard Operating Procedures (SOPs) were developed and meticulously followed in all analytical steps.

**3.7. Data Analysis**

Data were entered using Excel spread sheet and results were analysed using STAT ver 6. Student t-test was done for urinary δ-ALA levels, sex and alcohol taking habits of both exposed and non-exposed groups. F- ANOVA was also done to relate levels of δ-ALA with duration of exposure, age and enterprises. Values are expressed as means ± SEM. A probability value of less than 5 percent was used as the level of significance. The distribution was regarded as normal distribution and reference values were also considered.
3.8. Ethical Consideration

Before starting the study, each of the exposed and non-exposed subjects were informed briefly about the purpose of the study including its benefits. Each of them had given their consent of participation in the study (see Appendix B) and all information that was obtained about the subjects was kept absolutely confidential. All subjects of the study had a right to quit their enrollment at their will.
4. RESULTS

A total of 51 lead exposed subjects, Anbasa (45.1%), Comet (33.3%) and Walia (21.6%) and 50 non-exposed subjects were investigated for their level of urinary δ-ALA, renal indices and uric acid levels. Eighty-eight percent of the interviewed study participants were all males (Table 2).

Table 2: Study sites and demographic data of lead exposed workers (N=51).

<table>
<thead>
<tr>
<th>Exposed workers</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enterprise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anbasa</td>
<td>23</td>
<td>45.1</td>
</tr>
<tr>
<td>Comet</td>
<td>17</td>
<td>33.3</td>
</tr>
<tr>
<td>Walia</td>
<td>11</td>
<td>21.6</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-35</td>
<td>4</td>
<td>7.9</td>
</tr>
<tr>
<td>36-45</td>
<td>22</td>
<td>43.1</td>
</tr>
<tr>
<td>46+</td>
<td>25</td>
<td>49.0</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>45</td>
<td>88.2</td>
</tr>
<tr>
<td>Female</td>
<td>6</td>
<td>11.8</td>
</tr>
</tbody>
</table>

The workers generally spend a total of 6½ hours on actual work and 1½ hours of break. The duration of employment in the same position ranged from 1 up to 32 years (Table 3). 56 percent of the interviewed workers had been working in the same position since their recruitment, whilst 44 percent of the workers had changed positions that they were placed in when they first joined the enterprises.

Table 3: Employment duration of lead exposed workers.

<table>
<thead>
<tr>
<th>Employment duration (years)</th>
<th>Number of workers</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤10</td>
<td>9</td>
<td>17.7</td>
</tr>
<tr>
<td>11-20</td>
<td>17</td>
<td>33.3</td>
</tr>
<tr>
<td>21-25</td>
<td>9</td>
<td>17.7</td>
</tr>
<tr>
<td>25+</td>
<td>16</td>
<td>31.3</td>
</tr>
</tbody>
</table>

Ten calibrators of known δ-ALA concentrations in µg/ml were analysed and least squares linear regression analysis was performed to establish the best line that relates measured absorbance or optical density to δ-ALA concentration (Fig. 2). The analysis gave the following least squares line equation: \[ Y = 0.06399 \times X + 0.01953 \], where X= concentration of δ-ALA (µg/ml) and Y= absorbance. Urinary δ-ALA level in mg/100ml was calculated using this equation as

\[ X = \frac{Y - 0.01953}{10^{-1}} \], where \(10^{-1}\) changes µg/ml into mg/ml.

0.06399
Figure 2. Best line fitted standard calibration curve for the determination of δ-ALA.

Y = 0.06399X + 0.01953
4.2. Levels of urinary δ-ALA

Biochemical analysis of urinary δ-ALA revealed a two-fold increase (p<0.001) in exposed than non-exposed subjects (Fig. 3). The mean levels of urinary δ-ALA were 1.6mg/dl ± 0.2 in exposed subjects and 0.7mg/dl ± 0.1 in non-exposed ones.

To see within-group distribution of δ-ALA exposed and non-exposed subjects were categorized using classification proposed by Lane et al. (1968).
Accordingly, whilst 84% of subjects from the non-exposed group were within normal range, the percent for exposed ones was as low as 9.8% (Fig. 4). Furthermore, more than half of the exposed subjects had acceptable levels and about a third had high levels. By contrast, among non-exposed subjects about 16% displayed acceptable range and none of them had high levels of urinary δ-ALA. Inter-enterprise analysis of urinary δ-ALA was also done to have an idea whether preventive measures are in place or not. The analysis did not produce any significant difference, despite the levels in Comet appeared to be lower than the other two (Table 4).

**Table 4:** Urinary δ-ALA (mg/dl) levels of exposed workers by enterprise

<table>
<thead>
<tr>
<th>Enterprise</th>
<th>No of workers</th>
<th>Mean δ-ALA (mg/dl) ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anbasa</td>
<td>23</td>
<td>1.83 ± 0.39</td>
</tr>
<tr>
<td>Comet</td>
<td>17</td>
<td>1.26 ± 0.29</td>
</tr>
<tr>
<td>Walia</td>
<td>11</td>
<td>1.87 ± 0.60</td>
</tr>
</tbody>
</table>

Categorization of exposed subjects, based on measured urinary δ-ALA, was also extended by enterprises. It was found that whereas Anbasa contributed to the large proportion (21.6%) of subjects with high urinary δ-ALA levels, Comet with fairly large proportion (5.9%) with normal levels (Table 5). To examine whether urinary δ-ALA levels vary with age, subjects were stratified into different groups and statistical analysis was performed using ANOVA test. The result indicated that urinary δ-ALA levels increased with age in exposed group (F=18.54, p< 0.001) but failed to show any significant difference in non-exposed group (Table 6). Likewise, t-test was employed to assess the impact of sex on urinary δ-ALA levels. Sex difference was not shown to have any effect on the measured values, although levels in male tended to increase than females (Table 7).
Table 5: Categorization of enterprises with the number of exposed subjects based on range of measured urinary δ-ALA.

<table>
<thead>
<tr>
<th>Enterprise</th>
<th>Normal range</th>
<th>Acceptable</th>
<th>High</th>
<th>Dangerous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anbasa</td>
<td>1</td>
<td>11</td>
<td>11</td>
<td>-</td>
</tr>
<tr>
<td>Comet</td>
<td>3</td>
<td>13</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Walia</td>
<td>1</td>
<td>5</td>
<td>5</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>5</td>
<td>29</td>
<td>17</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 6: Urinary δ-ALA (mg/dl) mean levels of exposed and non-exposed subjects by age group.

***p < 0.001

<table>
<thead>
<tr>
<th>Age</th>
<th>Non-Exposed</th>
<th>Exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-35</td>
<td>0.73 ± 0.22</td>
<td>0.61 ± 0.22***</td>
</tr>
<tr>
<td>36-45</td>
<td>0.67 ± 0.12</td>
<td>1.24 ± 0.32***</td>
</tr>
<tr>
<td>46+</td>
<td>0.68 ± 0.14</td>
<td>2.17 ± 0.25***</td>
</tr>
</tbody>
</table>

Table 7: Urinary δ-ALA (mg/dl) mean levels of exposed workers by sex.

<table>
<thead>
<tr>
<th>Sex</th>
<th>n</th>
<th>Mean δ-ALA (mg/dl) ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>45</td>
<td>1.69 ± 0.26</td>
</tr>
<tr>
<td>Female</td>
<td>6</td>
<td>1.34 ± 0.45</td>
</tr>
</tbody>
</table>
The impact of duration of employment on levels of urinary δ-ALA was analysed and δ-ALA was found to be a function of duration of employment (Table 8). Indeed, δ-ALA was noted to significantly increase with duration of employment (F=22.2, p<0.001).

Table 8: Urinary δ-ALA (mg/dl) mean levels of exposed workers by employment duration.

<table>
<thead>
<tr>
<th>Employment duration</th>
<th>Mean δ-ALA (mg/dl) + SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤10</td>
<td>0.54 + 0.16</td>
</tr>
<tr>
<td>11-20</td>
<td>1.45 + 0.31</td>
</tr>
<tr>
<td>21-25</td>
<td>1.90 + 0.37</td>
</tr>
<tr>
<td>25+</td>
<td>2.34 + 0.35</td>
</tr>
</tbody>
</table>

4.3. Serum Creatinine, Creatinine Clearance and Urea levels.

In order to see the long-term effects of lead on kidney, different renal indices were measured in both exposed and non-exposed groups and the results were presented in Table 9. No detectable difference was observed in CC, SC and blood urea levels between exposed and non-exposed groups. However, it is worth noting that CC decreased by about 11% in exposed subjects, although it fell short of reaching statistical significance. In parallel, an attempt was made to look whether there is deviation from reference values or not and interestingly all were found to lie within the normal range. The normal ranges according to the manufacturer of the kit are as follows: SC (male, 0.7-1.3mg/dl and female, 0.6-1.1mg/dl); CC (male, 94-140ml/min and female, 72-110ml/min); and Urea (15-45mg/dl for both sex).
Table 9: Serum creatinine, creatinine clearance and urea levels of exposed and non-exposed groups.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean SC (Mg/dl) ± SEM</th>
<th>Mean CC (Ml/min.)±SEM</th>
<th>Mean Urea (Mg/dl)±SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Exposed</td>
<td>1.15 ± 0.03</td>
<td>115.33± 5.20</td>
<td>21.80± 0.79</td>
</tr>
<tr>
<td>Exposed</td>
<td>1.18 ± 0.02</td>
<td>102.91± 6.27</td>
<td>22.91± 0.79</td>
</tr>
</tbody>
</table>

4.4. Uric Acid levels.

Lead is known to inhibit UA secretion, thereby increasing serum UA levels. Serum UA levels were therefore measured to use it as an indirect measure of lead exposure, along with urinary δ-ALA. Consistent with the aforementioned notion exposed subjects displayed increased UA levels than non-exposed subjects, which were significantly higher by about 8% in exposed subjects than non-exposed ones (p<0.05) (Fig. 5).

Figure 5. UA levels in exposed and non-exposed groups.
Intra-group sub-classification of UA levels using normal ranges supplied along with the kit revealed that about 69% exposed subjects had abnormal serum UA, while this was 36% in non-exposed subjects (Fig. 6). Uric acid normal range was 3.4 -7.0 mg/dl in male and 2.6-6.0mg/dl in female.

![Figure 6. Distribution of exposed and non-exposed subjects by group of Uric acid levels](image)

4.5. Reported Illnesses and Lifestyle Factors.

Reported illnesses and life style factors that were compiled from the structured questionnaire (see Appendix A) included illnesses linked with lead poisoning, alcohol intake, smoking, meals at workplace and work related hobbies which could result in additional exposure to lead. Twenty of the exposed workers interviewed during this study reported that they have suffered from illnesses, which are known to be commonly linked with lead poisoning. A summary of the illnesses reported by 20 exposed workers is presented in Table 10.
Table 10: Summary of reported illnesses.

<table>
<thead>
<tr>
<th>Illness</th>
<th>Number of workers (n)</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>09</td>
<td>17.65</td>
</tr>
<tr>
<td>Visual Problems</td>
<td>13</td>
<td>25.49</td>
</tr>
<tr>
<td>Stomach-ache</td>
<td>08</td>
<td>15.69</td>
</tr>
<tr>
<td>Migraine Headache</td>
<td>01</td>
<td>1.96</td>
</tr>
<tr>
<td>Gout</td>
<td>02</td>
<td>3.92</td>
</tr>
<tr>
<td>Kidney Problems</td>
<td>07</td>
<td>13.73</td>
</tr>
<tr>
<td>Weak Joints</td>
<td>05</td>
<td>9.80</td>
</tr>
<tr>
<td>Hypertension</td>
<td>01</td>
<td>1.96</td>
</tr>
</tbody>
</table>

Six exposed group workers reported that vehicles are the source of dust in the work environment, which in turn causes asthma and eye diseases. Seven workers who reported to suffer from kidney problems had visited the clinic due to the illness. These workers were given sick leave. The workers did not know the cause of the illness. Nine of the workers who had asthma had been absent from work due to this illness. These workers suspect that the cause of the asthma was dust from workplace and solvents such as sulphuric acid exposure from the storage batteries. Eighty-nine percent of the workers reported that they are worried about their health while working in these repair units not because they know about the hazards of lead poisoning but solvents as well as flame exposure. However, six workers were uncertain about their health while working in these units. All workers were not confident about the preventive measures taken by the enterprises towards their health. Regarding health status of non-exposed subjects there was no one with major complaint.

The levels of Aminolevulinic acid were analysed for alcohol taking and non-alcohol taking subjects (Table 11). Thirty-one (60.8%) workers reported that they are taking alcohol and twenty (39.2%) workers are non-alcohol taking. On average, the interviewed alcohol consumers take approximately 6 bottles of draught beers per week.
Table 11: Levels of mean δ-ALA among alcohol taking and non-alcohol taking exposed subjects.

*p < 0.05

<table>
<thead>
<tr>
<th>Exposed group</th>
<th>n</th>
<th>Mean δ-ALA (mg/dl)±SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol taking</td>
<td>31</td>
<td>1.868 ± 0.147*</td>
</tr>
<tr>
<td>Non-alcohol taking</td>
<td>20</td>
<td>1.312 ± 0.165</td>
</tr>
</tbody>
</table>

The level of δ-ALA is significantly higher in alcohol taking workers than non-alcohol taking ones (p < 0.05). Workers were also not aware of the effects of alcohol consumption on blood lead levels. Other life style factors for additional lead exposure in and outside workplace were also obtained from workers responses using the structured questionnaire. These results were compiled and are shown in Table 12.

On average, the interviewed smokers smoke 6-10 cigarettes per day. Workers who were interviewed mentioned that they were not educated about smoking and its effects on ones health. Workers also reported that they do not know about the association between smoking and lead exposure. Surprisingly all the workers were not aware of additional exposure to lead while having meals at the repair units as well as while working outside workplace on bench and industrial electronics. Work related hobbies by 9 of the workers include bench and industrial electronics.

Table 12: Smoking habits, meals at workplace and work related hobbies of exposed subjects.

<table>
<thead>
<tr>
<th>Exposed Workers</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smokers</td>
<td>2</td>
<td>3.92</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>49</td>
<td>96.08</td>
</tr>
<tr>
<td>Have meal at workplace (once)</td>
<td>40</td>
<td>78.43</td>
</tr>
<tr>
<td>Have meal at workplace (twice)</td>
<td>5</td>
<td>9.80</td>
</tr>
<tr>
<td>Never had meal at workplace</td>
<td>6</td>
<td>11.76</td>
</tr>
<tr>
<td>Practice bench and industrial electronics hobbies</td>
<td>9</td>
<td>17.65</td>
</tr>
</tbody>
</table>
4.6. Status of Workplace Safety Measures

Interviews and walkthrough evaluation was done in all the repair units. It was responded by all of the interviewed lead exposed workers that the repair units provide gowns, facemasks and gloves. However, ninety five percent of the exposed workers preferred not to wear personal protective equipment due to feeling discomfort while using them. During the study it was found that all the three enterprises did not implement clear policy regarding the use of personal protective equipment. All exposed subjects of the repair units reported that the enterprises do not provide training regarding lead toxicity.

It was observed during the walkthrough evaluation that all the enterprises workplace are dusty and did not follow lead regulations. Ventilators and extractor fans for controlling lead dust generated at the workplace were lacking at Anbasa and Walia bus service enterprises (Fig. 7). Lack of proper offices and decontamination services were seen in both Anbasa and Walia bus service enterprises. Furthermore, it was reported by 95% the exposed workers that environmental health experts did not regularly supervise all the three enterprises. During walkthrough evaluation non-functional storage batteries were also seen thrown outside the repair units in all the enterprises (Fig. 8).
Figure 7: A partial view of storage battery repair unit in one of the enterprises.

Figure 8: Disposal of used storage batteries in one of the enterprises.
5. DISCUSSION

Low-level long-term occupational lead exposure is known to result in adverse health effects in humans. The present findings indicate that workers at lead acid battery repair units are not safe from excessive lead exposure, as urinary δ-ALA levels were found to be higher in exposed group than non-exposed ones. This cross-sectional biochemical lead exposure study is the first of its kind in Ethiopia, although several studies have been carried out elsewhere (Meredith et al., 1978; Haenninen et al., 1979; Fischbein et al., 1980; Awad El Karim et al., 1986; Pinto de Almeida et al., 1989).

Urinary δ-ALA levels

The application of biomarkers has become a crucial and widely used tool in understanding and assessment of health effects (IPCS, 1994). At present blood lead levels are frequently measured to assess both lead exposure and effect that will facilitate the risk assessment process. However, a large body of evidence indicates that alternative biomarkers for lead that may be easily measured are also of major importance, particularly in the haem biosynthetic pathway (Meredith et al., 1978; IPCS, 1995; Lee, 1999; Sakai, 2000). This study has therefore considered urinary excretion of δ-ALA as a surrogate measure of blood lead in storage battery repair workers; owing to lack of facilities to measure blood lead levels.

δ-ALA is excreted normally in small amounts in urine; but the levels in urine increase with lead exposure. Bauer (1982) reported a five-fold increase in urinary excretion of δ-ALA following lead intoxication. This rise in concentration of δ-ALA during lead exposure is a function first of decreased activity of ALAD, which is uniquely sensitive to lead toxicity, and also other enzymes, including, ferrochelatase. Inhibition of ferrochelatase, the last enzyme in the haem biosynthetic pathway, stimulates the initial and rate-limiting enzyme, ALAS, to produce δ-ALA. However, conversion of δ-ALA to porphobilinogen cannot occur as ALAD is inhibited by lead. This would then result in increased levels of δ-ALA in the blood and plasma, eventually leading to increased δ-ALA urinary excretion (Higashikawa et al., 2000; Mehti et al., 2000; Sakai, 2000; Yokoyama et al., 2000).
ALAD in erythrocytes is inhibited at very low blood lead levels (Hernberg and Nikkanen, 1970). This inhibition shows a dose-dependent relationship with urinary $\delta$-ALA levels, which is thought to be attributed to decreased tubular reabsorption (Selander and Cramer, 1970; Meredith et al., 1978; Lee, 1982).

Increased urinary $\delta$-ALA levels found in exposed subjects in the present study (Fig. 3) is the impact of low-level long-term lead exposure at the repair units and reinforces the notion that $\delta$-ALA can serve as a surrogate marker for lead exposure. In addition, the high urinary $\delta$-ALA levels obtained from about 33.33% of exposed workers (Table 5) is a clear indicator of cumulative lead exposure and appears to be directly related to duration of employment at the repair units (Table 8). The finding that 84% of non-exposed subjects exhibited normal range and none of them had high levels of urinary $\delta$-ALA, rules out other factors and rules in low-level long-term occupational lead exposure to be the factor responsible for the observed high levels of $\delta$-ALA in exposed subjects.

**Employment duration versus $\delta$ALA levels**

The longer the employment period the higher the $\delta$-ALA levels ($p<0.001$). Urinary $\delta$-ALA levels in workers who had served for 25 years was about four times the values measured in those served for ten years and below. It is important to stress here that urinary $\delta$-ALA levels were strongly interrelated with employment duration in lead acid battery repair units (Table 8). One may consider that as the duration of employment increases there will be higher chances of lead exposure. Lee (1982) also found that the urinary $\delta$-ALA of lead workers increased with an increase in the duration of exposure.

**Urinary $\delta$ALA levels among the enterprises**

Comparison of urinary $\delta$-ALA among the enterprises is an indicator for the status of workplace lead exposure preventive and control measures implemented at the repair units. Measured urinary $\delta$-ALA of the three enterprises was compared and no apparent significant difference was observed between the enterprises (Table 4), although workers in Comet tended to have relatively lower levels of urinary $\delta$-ALA. This might have been due to short employment duration of workers, a relatively
better hood system and room facilities of the enterprise. Also this enterprise was with shorter establishment period and located at the peripheral parts of Addis Ababa than two of the enterprises. On the other hand about 21.57% of exposed workers from Anbasa had high urinary δ-ALA, which resulted from its relatively poor preventive and control system compared to the other two.

**Age and sex versus urinary δ-ALA levels**

In order to see the impact of age and sex on the levels of urinary δ-ALA among exposed and non-exposed subjects statistical analysis was done and age was found not to be a necessary or sufficient factor for levels of urinary δ-ALA in non-exposed subjects. In contrast, age had a significant association (p<0.001) in exposed subjects. Plasma lead levels are known to be higher in children and decline with age, as bone density increases and lead starts to redistribute to the skeletal pool. However, in aged people plasma lead again increases due to decalcification of bones and eventual release of lead into the plasma. Given this fact, the association of urinary δ-ALA with age could probably better explained by duration of exposure rather than increase with age *per se*, as the maximum age of an exposed subject is an unlikely age where decalcification of bone starts. Similar findings published in the literature have shown that both age and gender influenced blood lead levels (Hodgkins et al., 1991). In the present study, however, sex was found to have little or no impact on urinary δ-ALA levels among the exposed subjects (Table 7), though females are expected to have higher blood lead levels compared to males. This might have something to do with the few number of females available for comparison i.e. only six of them were found to work in the three storage battery repair units.

**Alcohol intake versus urinary δ-ALA levels**

Lifestyle factors other than the occupational settings can have an effect on the exposure of a toxicant. Such factors usually include smoking and alcohol taking. In this study, the effect of alcohol, particularly draught beer, on urinary δ-ALA levels of exposed subjects was analysed and alcohol-taking subjects displayed increased levels (p<0.05) (Table 11) than their non-alcohol-taking peers. Evidence for this finding comes from the observation of Weyermann and Brenner (1997). These authors reported that alcohol consumption is a culprit for the rise in blood lead levels
observed in exposed alcohol takers. The role of alcohol in blood lead levels is unclear and is still a subject of controversy. Published results indicate that the draught dispensing equipment rather than alcohol \textit{per se} is responsible for the increased lead concentration in alcohol-taking subjects (Sherlock et al., 1986). They argue that the equipment sometimes contains brass or gunmetal that has low but significant amounts of lead. Thus, frequent and prolonged intake of draught beer could result in a slow but steady rise in lead concentration, as observed in this study by measuring urinary δ-ALA.

\textit{Serum creatinine, creatinine clearance and urea levels}

There is a fair evidence to suggest that chronic low level lead exposure may affect kidney function (Bernard and Becker, 1988; Lim et al., 2001). However, level of severity and duration of exposure that leads to renal damage is not clearly defined (Goyer and Ryne, 1973; Omae et al., 1990; Wang et al., 2002). In this study, though urinary δ-ALA increased in exposed subjects and appeared to be related to duration of employment, none of the renal indices were found to be different from the non-exposed subjects. Surprisingly, levels of SC, CC and blood urea levels of both non-exposed and exposed subjects were found to be within the normal range (Table 9). A cross-sectional study conducted in lead-exposed workers showed that lead might not cause adverse effects on renal glomerular and proximal tubular functions when there is long-term and less severe exposure (Omae et al., 1990; Wang et al., 2002). Goyer and Ryne (1973) also reported that workers with excessive lead exposure for over ten years did not exhibit renal failure. Our result is concordant with the aforementioned findings and discordant with that of Pinto de Almeida et al. (1989) who found that exposed workers at the smelter had a greater SC level and renal dysfunction. This difference can be explained by the fact that unlike our work they considered workers of primary lead smelters than repair units.

\textit{Uric acid levels}

A relationship between gout and lead nephropathy has been recognised for centuries and gout occurs more frequently in the presence of chronic lead nephropathy than in any other type of chronic renal disease (Bernard and Becker, 1988). The Autolab analysis results showed that the serum uric acid levels of exposed subjects were significantly higher than non-exposed ones.
The fact that large proportion of exposed subjects revealing high serum uric acid levels than non-exposed ones is an indicator for the possible contribution of lead exposure (Fig. 6). Consistent with our finding, a growing body of evidence indicates that chronic occupational lead exposure is associated with low urate excretion (Pinto de Almeida et al, 1989; Lin et al., 2002).

Attempts were also made to examine additional factors other than lead exposure that might contribute for the rise in the levels of uric acid in both exposed and non-exposed subjects. And it was known that among exposed and non-exposed subjects there was no one who had been taking medication(s) that could contribute for the rise in the levels of uric acid, ruling out this possibility.

**Lead exposure related illnesses**

Occupational lead exposure related illnesses are very common (George, 1999). The present study showed that 39% of exposed workers had some of the common illnesses of lead poisoning. These illnesses include asthma, joint pain, poor sight, stomachaches, gout and renal problems (Table 10). Among the exposed group 89% of them reported that they are very much concerned about their health whilst working in different lead acid battery repair units, which somehow strengthens the possibility of reported symptoms to originate from occupational lead exposure. Therefore, air monitoring, biological monitoring and medical surveillance should be done as stipulated in the lead regulations of 2001. Kononen (1991) conducted a study in lead acid battery workers and found that the greatest absolute and percentage increases above baseline blood lead levels occurred during the first three months of continuous exposure. Employees should not be allowed to have blood lead levels of more than 30µg/dl in the first three months of employment (George, 1999). If so, further steps should be taken, which include consideration of the placement of the worker in more suitable workstation or reassignment of the worker if the levels do not drop down to acceptable levels.

It was interesting to note that 44% of exposed subjects reported that they changed workstations through promotion but not after considering the risks of lead exposure that could result in deleterious health effects. George (1999) also reported that often in many developing nations the long-term health consequences resulting from an unsafe environment are not given proper attention, which would definitely affect productivity of workers.
**Workplace safety measures**

Workplace assessment identified existing and potential hazards, evaluated the nature of the hazard and helped to provide appropriate measures to protect the health and safety of employees. High production of lead dust and fumes observed at workplace are among top risk factors that need special attention by all the studied enterprises. According to Smith (1997) physical surroundings, job demands and technological factors, improper design of the work environment and job activities can cause adverse employee perceptions, psychosocial stress and health problems. Thus, safety and health measures such as general ventilation is usually desirable to control the temperature and humidity of the environment or a properly designed system that can act as a back-up control of exposure to airborne substances by diluting the airborne contaminants (WHO, 1999).

In addition, during this study it was found that all the enterprises have no clear policy regarding the use of PPE and it was also observed that the exposed workers were performing their tasks without using appropriate PPE. With regard to this, Stellman and Osinsky (1997) noted that workers who are exposed to lead should be provided with PPE that should be washed or renewed regularly. Furthermore, all interviewed lead exposed subjects reported that all the studied enterprises do not provide training regarding lead toxicity either preplacement or after employment, if it was practiced would have contributed for the decrease in lead exposure levels at workplaces. According to Lead Regulations of 2001, it is stated that the relevant equipment is correctly selected and properly used as well as training on how to use the PPE is provided. The prevention of occupational hazards is far more effective and less costly when considered during the early stages and use of PPE by the people exposed is necessary (WHO, 1999).

In another finding of this study, the structured questionnaire analysis showed that 88% of exposed subjects had meals at workplaces on regular basis for atleast once per day and were assumed to have additional lead exposure (Table 12). To ensure good performance and well being of workers, ILO (1996) states that all companies should provide drinking facilities, eating areas and rest rooms. Therefore, in all the three enterprises: clothes changing facilities, decontamination services, dining rooms and offices should be considered for renovation and/or construction. According to Lai et al. (1997) improvements of hygienic practices were more effective at lowering blood lead levels than reducing ambient lead level. Therefore, hygienic practices might be the preferential way to reduce
lead exposure at the workplace, especially in a developing country like Ethiopia as compared to the engineering controls.

The present study also reported that environmental health experts did not supervise all the enterprises and non-functional storage batteries were seen thrown outside the repair units (Fig. 8). Lead is highly toxic so that non-functional batteries require proper storage rooms and recycling. In particular, it would be of help if all defunct lead-acid batteries were returned for disassembly and reuse of the lead contained in them; otherwise it will result in lead exposure to other workers of the enterprises. Improving the work environment of the workers is quite important, as the next workers who are assigned to work in the ‘non-fit’ environment would also be exposed to the same hazard that entails an overall decrease in productivity of the enterprises. By and large; occupational exposure to lead remains a big problem in developing countries including Ethiopia. The exposure is likely unregulated in these countries with little monitoring of poisoning being done and only just becoming recognized as a potential problem (George, 1999).
6. CONCLUSION AND RECOMMENDATIONS

6.1. CONCLUSION

Raised levels of urinary δ-ALA and uric acid obtained from the exposed subjects indicated that the possible parallel rise in blood lead levels. These high levels of measured values were attributed from poor preventive and control measures at the repair units. On the other hand, the outcomes of renal indices were found to be in the normal ranges. These values were obtained as expected and which indicates workers at lead acid battery repair units are still safe from serious renal effects of low-level long-term lead exposure. In spite of the healthy feeling lead at any levels may still affect health. It is also known that the prevention of occupational lead exposure is far more effective and less costly when considered during the early stages. Therefore, it is necessary that lead exposures at workplaces are minimized by placement of appropriate and cost-effective integrated preventive and control measures. Regular monitoring of these programmes and exposure levels are also equally important.

6.2. RECOMMENDATIONS

• Medical Examination

Health risks of lead need to be given due attention by the enterprises and periodic medical check-ups should be implemented. Emphasis should be on the following: (1) lead exposure history; (2) personal and workplace hygiene; and (3) gastrointestinal, haematological, renal, reproductive, and neurological problems.

• Biological Monitoring

Blood and/or urine samples should be taken from lead exposed workers on scheduled programs and regular basis for analysis and necessary measures should be taken accordingly. In a developing country such as Ethiopia, which cannot introduce engineering controls quickly to protect storage battery repair workers, biological monitoring would be useful in identifying and lowering excess lead absorption.
• **Housekeeping**

It is recommended that clear assignments of responsibilities regarding the day-to-day cleaning of the work areas and general housekeeping be considered. General housekeeping needs to be done at least after each shift because the workplace was found to be dusty.

• **Personal Protective Equipment**

The enterprises should have a clear policy regarding personal protective equipment (PPE) and ensure that all workers wear the required PPE. There should be regular supervision for the proper use of PPE. In accordance to ILO (1996) clear instructions and proper adaptation trial and training should be given to the workers. The efficacy of PPE program should be checked with ongoing biological monitoring.

• **Personal Hygiene**

It is recommended that workers should take responsibility of ensuring that they wash their hands before each meal to prevent workers from ingesting lead; ensure that work clothes are left in the laundry or in the appropriate storage areas; and take a shower before they leave the workplace so that lead does not spread to the workers' families. Hygienic practices might be the preferential way to reduce lead exposure at the workplace, especially in developing countries like Ethiopia as compared to the engineering controls.

• **Local Ventilation System**

Local Ventilation System - It is recommended that whenever possible the enterprises should install local exhaust ventilation. To meet the high standard of working conditions of industrialized nations, decades of effort are often required. Careful evaluation of the process should be carried out prior to selecting and designing a LEV system. Input should be provided from various disciplines, including engineering, planning, industrial hygiene and labour.
7. SUGGESTIONS FOR FUTURE WORK

- Measured high urinary δ-ALA levels may not actually reflect the true picture of lead exposure. So, a more sensitive indicator based on the tolerable limits, such as lead in blood, is required for and is relevant, to gain a better insight into the magnitude of exposure to lead in the storage battery repair units in Addis Ababa.

- Measurements of air lead levels at workplaces of the repair units need further consideration and study.
8. REFERENCES


Safety and Health Assessment and Research for Prevention (SHARP) programme (1994) occupational lead exposure: An alert for workers, publication No. **17-6-1994**.


Encyclopaedia, 4th ed., Taylor and Francis, p. 34. 22.

Spivey GH, Baloh RW, Brown CP, Browdy BL, Campion DS, Valentine JL, Morgan DE and
Culver BD (1980) Sub clinical effects of chronic increased lead absorption-a prospective

Hypertension caused by low-level lead exposure: myth or fact? Journal of cardiovascular
risk, 1: 87 – 97.


Safety Encyclopaedia, 4th ed., p. 34.19.

Stollery BT, Broadbent DE, Banks HA, and Lec WR (1991) Short-term perspective study of

Suplido ML and Ong CN (2000) Lead exposure among small-scale battery recyclers, Automobile
radiator mechanics and their children in Manila, The Philippines, Environ, Res.,
82 (3): 231–8.

p.833.

Timbrell JA (2000) Principles of Biochemical Toxicology, 3rd ed., Taylor and Francis Ltd., UK,
pp. 338-41.

Tomokumi K. & Ogata M. (1972) Simple method for determination of urinary δ-ALA as an index


Weyermann M and Brenner H (1997) Alcohol consumption and smoking habits as determinants of blood lead levels in a national population sample from Germany. *Arch Environ Health, 52*:233 - 240.


APPENDIX A

STRUCTURED INTERVIEW FOR LEADED STORAGE BATTERY REPAIR WORKERS

The aim of this questionnaire is to gather information regarding: (1) personal information and (2) work practices and health risks.

It would be highly appreciated if you can take a few minutes to answer the following questions as it would assist the researcher in understanding the level of lead exposure in your workplace and make recommendations accordingly.

All answers will be treated confidentially and your anonymity will be maintained throughout the research.

1. PERSONAL INFORMATION

1.1 Name ____________________________ Code No. _________
1.2 Age (yrs) _________________
1.3 Gender _______________________
1.4 Work place/Company ___________________________
1.5 Duration in current job _______________________
1.6 Description of your previous job before joining the current job _____________________
1.7 Duration in previous job _______________________

2. WORK PRACTICES AND HEALTH RISKS

2.1 How many hours do you work per day? _________________________________
2.2 How long are your breaks? _________________________________
2.3 Did you get training for the job you are currently doing? _____________________
2.4 Have you ever suffered from any illness? _________________________________
2.5 How long were you absent from work due to the illness? ______________________
2.6 When were you ill?

☐ Last week / month

☐ 6 months ago

☐ Over a year

2.7 What do you think was the cause of the illness?  

2.8 What do you think could be done to prevent you from getting this illness?  

2.9 Are you worried about your health working here?  

2.10 Do you smoke?  

Yes ☐  No ☐

If yes,

2.11 How many cigarettes do you smoke per day?  

2.12 Do you take alcohol?  

Yes ☐  No ☐

If yes,

2.13 How many units of alcohol do you take per day?  

2.14 How many times a week?  

2.15 Hobby activities  

2.16 Place of residence  

- 66 -
2.17 Do you have meals at the work place?

Yes  No

If yes,

2.18 How many times per day? ______________________________________

2.19 Do you take any medication(s)?

Yes  No

If yes,

2.20 What is (are) the medication(s)?

_________________________________________________________________

.................Thank you for your co-operation..........................
APPENDIX B

INFORMED CONSENT

Name of investigator: Kemal Ahmed Hussein

Name of the institute: Department of Pharmacology,
                      School of Pharmacy,
                      Addis Ababa University,
                      Addis Ababa - ETHIOPIA

Title of the project: Lead exposure study among workers in lead acid battery repair units of Anbasa, Comet and Walia transport service enterprises, Addis Ababa, Ethiopia.

Brief description about the project: Inhalational lead exposure is quite common in storage battery repair units of transport service enterprises. Chronic lead exposure in particular can result in a variety of adverse health effects including interference with haem biosynthesis, damage to the nervous system, the kidneys, reproductive system and so on. The present study will measure levels of delta - Aminolevulinic acid in urine, which is much, related to the concentration of lead in blood. This study will also investigate different renal indices as indicators of lead exposure.

Procedure: (1). Questionnaire will be filled out to collect information regarding workers personal data, work practices and health risks. (2). You will be asked to provide approximately 2 hours (random urine) sample between 9:00 to 11:00 a.m. using light protected plastic containers. (3). Blood specimen (3ml) will be drawn by puncturing antecubital vein by a medical laboratory technician from Ethiopian Health and Nutrition Research Institute.

Benefits & Risks involved: From the results of the present investigation, you will know the level of δ-ALA in your urine. This outcome will help the investigator to propose appropriate preventive and control measures. Further, the present investigation will give result of your renal function status, which helps the physician in deciding the appropriate management measures in case of
illness. Withdrawing of blood from antecubital vein will produce little pain. Rarely, a haematoma may form at the site of puncture though all precautions will be taken to avoid damage to the vein. All the necessary precautions will be taken to prevent transmission of infectious diseases.

**Agreement:** I have fully understood the objectives and uses of this study. I have also read the benefits and risks associated with the project. In addition, I understand that all personal data of mine are strictly confidential. I have not been forced to participate in the investigation. My participation is absolutely voluntary.

Name of the participant __________________   Name of the investigator _________________

Signature ___________________________   Signature ___________________________

Date ______________________   Date ________________ ____________
1. የም.salary

2. የንጆች ትርምሮ የስር

- 70 -
3. ከእተ ይዘት

ลบልታ የራርፋፅ የጭእልካይ ይና የሚበታ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከነስ የጭእልካይ ከስ ከራ ከን
DECLARATION

I, the undersigned, declare that this thesis is my original work, has not been presented for a degree in any other university and that all the sources of material used for the thesis have been duly acknowledged.

Name: **KEMAL AHMED HUSSEIN**  Signature______________________

Place of submission: **ADDIS ABABA**  Date_____________________

SUPERVISORS: -

1. ________________________________ Signature______________________

2. ________________________________ Signature______________________