ASSESSMENT OF LEAD EXPOSURE AMONG CHILDREN IN SELECTED NURSERY SCHOOLS IN LAGOS METROPOLIS, NIGERIA

BY

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ABSTRACT

Lead in the environment comes from paints, petroleum, solder and through various routes such as water, food, air, dust and soil. Children of pre-school age are more exposed to these environmental hazards due to their habits of licking fingers chewing objects or actually eating contaminated dust, soil and food items. This study was undertaken to assess the sources of lead exposure among children in selected Nursery Schools in Lagos Metropolis.

The study was exploratory in nature. For sampling, Lagos Metropolitan schools were divided into three zones as low (Victoria Island), medium (Mile II) and high density (Ojo) areas reflecting various socio-geographical factors and a school was selected in each zone based on simple random method. A random sampling method was employed in selecting the subjects and collecting environmental samples. A total of 150 children comprising of 77 (51.3%) boys and 73 (48.3%) girls. Two hundred and seven (207) different samples were analyzed, 150 hand washings from 150 children. Twenty six (26) toy washings which comprises of various toys such as balls, telephones, empty beverage tins, cars ranging in diameter from 30cm x 15cm to 5cm x 4cm, 9 soil samples, 9 classroom sweepings, 9 roof top water and 4 eye based cosmetic samples.

Lead and other physical-chemical characteristics of the samples were determined viz. pH value, Total Dissolved Solids (TDS), Total Suspended Solids (TSS), Turbidity (FTU) and Conductivity (ms/cm) using standard methods as recommended by American Public Health Association (1998).

The mean lead levels of the boys in low, medium and high-density areas were as follows: 0.232 ± 0. 106mg/hand, 0.044 ± 0.037mg/hand and 0.093 ± 0.076 mg/hand while that of the girls was 0.126 ± 0.089 mg/hand, 0.036 ± 0.027 mg/hand and 0.070 ± 0.070 mg/hand. A positive statistically significant correlation was found between lead in the hand washings and other physical parameters. Lead and turbidity ($r = 0.4$ $p = 0.00$), lead and TSS, ($r = 0.5$, $p = 0.0$), Lead and conductivity ($r = 0.6$, $p = 0$), lead and TSS ($r = 0.6$, $p = 0.0$).

The mean lead values of the toy washings in the low, medium and high density areas were as follows 0.07 ± 0.03 mg/toy, 0.009 ± 0.003 mg /toy and 0.056 ± 0.04mg/toy. A statistically significant linear correlation was found between lead in the toy washings and other physical parameters. ($r = 0.5$). There was no significant association between lead and pH ($r = 0.3$ $p < 0.05$).

The lead levels in the classroom sweeping ranged between 180 – 615 mg/kg which is above the WHO recommended level of indoor lead of 200 mg/kg. The lead levels in the playground samples ranged from 943 – 3602 mg/kg with a [illegible] of 1962 ± 49mg/kg. These observations indicated that the lead levels of the soils were above the Federal Environmental Protection Agency (FEPA) permissible limit of 500mg/kg. The lead levels in the roof top water in the three-study area ranged from 0.05 – 0.25mg/l with a mean of 0.124 ± 0.02mg/l, which is above the WHO, recommended value of 0.01 mg/l of drinking water. The cosmetics samples showed lead levels in the range of 0.19 – 5.76μg/g and a mean of 2.95μg/g.

The in-depth interview revealed that the level of awareness of the various sources of lead in the environment and its associated health hazards was low among the respondents. Based on these results, the lead levels in school environment is due to lead content from dirt (possibly from automobile emissions) on the children's hands, toys soil and cosmetics.

This study shows that children are unduly exposed to lead from their immediate environment. There is need to control lead levels through legislation and health education.

ACKNOWLEDGMENT

I own sincere gratitude and praises to Almighty God for granting me good health and for his Grace during the course of this study. I wish to express my sincere thanks to my supervisors, Prof. M.K.C. Sridhar and Dr. J. F. Olawuyi for their painstaking supervision, encouragement and moral support in making this work come to completion. My sincere thanks also goes to all the lecturers of the Faculty of Preventive and Social Medicine. I am grateful to my parents, Mr and Mrs. Philip Irene, my brothers and sisters among whom are Sister Rose, Brother Julius, Stella, Helen, Patrick, Doris and David. My unreserved thanks goes to my predecessor, Leslie Adogame, Osagie Claudius, Ifeanyi Okekearu, Johnson Etaghene and Linda Aborkor.

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I will not fail to extend my appreciation to all the school proprietors for allowing me to use their schools as study areas.

CERTIFICATION

We certify that this work was carried out by MISS IRENE, AYEHMERE PHILOMENA of the Environmental Health Division, Department of Epidemiology, Medical Statistics and Environmental Health, College of Medicine, University of Ibadan.

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DEDICATION

This work is gratefully and specially dedicated to

GLORY OF GOD THE FATHER, GOD THE SON

and

GOD THE HOLY GHOST.

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ABBREVIATIONS

A.A.S.	:	Atomic Absorption Spectrophotometer.
ALA	:	δ - aminolaevulinic acid
ALAD	:	δ - aminolaevulinic acid dehydratase
ATSDR	:	Agency for Toxic Substance and Disease Registry
BPb	:	Blood lead
CDC	:	Centers for Disease Control
EDTA	:	Ethylene diamine tetra acetic Acid
EPC	:	Environmental Protection Commission
FEPA	:	Federal Environmental Protection Agency
IQS	:	Intelligent Quotients
NIOSH	:	National Institute for Occupational Safety and Health
OECD	:	Organization for Economic Co-operation and Development
RCEP	:	Royal Commission on Environmental Pollution
UNCED	:	United Nations Conference on Environmental Development
UNEP	:	United Nations Environmental Protection

GLOSSARY OF TECHNICAL TERMS USED

Contaminant: An undesirable substance not normally present or an usually high concentration of a naturally occurring substance in water or soil

Contamination: The addition of contaminants. Sometimes considered synonymous with pollution.

Cosmetics: A preparation such as face-cream, body cream, powder etc. intended to make the skin or hair more beautiful.

Environment: Is the sum total of all social, economical biological, physical or chemical factors which constitute the surroundings of man, who is both creator and molder of his environment.

Excretion: The process whereby materials are removed from the body to the external environment.

pH A value taken to represent the acidity or alkalinity of an aqueous solution

Poison: The term used to describe those materials or chemicals that are distinctly harmful to the body.

Pollution: Is the release of harmful substances or energy into the environment by man's activities, which directly or indirectly cause hazard to man and his environment.

Total Dissolved Solids (TDS): The total of all chemicals disolved in a natural water, often related to ionic strength or conductivity.

Toxicity: The capacity of a substance to cause injury to a living organism. It is a measure of a expression of safety and hazard.

Toxicant: A chemical agent or material when present at a concentration capable of producing an adverse response in a biological system, seriously injury structure or function, and causing death.

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CHAPTER ONE

INTRODUCTION

1.1 Background Information

Pollution of the environment is one of the most serious ecological crisis to which we are subjected today. Historically, the development of civilization has caused perturbation of the earth's ecosystem by one or several of a range of human activities resulting in the pollution of air, land and water (Speight, 1996). The quality of the environment and the nature of development are major determinants of health, indeed, the most immediate problems of the world are ill-health and premature deaths caused by biological agents in the human environment in water food, air and soil (WHO, 1992). Little is understood about the exposure to toxic chemicals.

The progress and welfare of present and future generations depend, to a great extent, on positive and timely solutions to socio-economic problems which arise from the relationships between the human populations and nature, it is this which has inspired the United Nations, Educational, Scientific and Cultural Organisation (UNESCO) to look anew at some of the major issues and problems characterizing the contemporary human environment. (UNESCO/UNEP, 1985).

There has been and continues to be, a good deal of public concern about the consequence of environmentally release chemicals especially on health and ecology. Implicit in this regard, the frequently explicit, is the notion that any effects will necessarily be adverse, although it is true to say that human life is not possible in the absence of chemicals, both organic and inorganic (Harrison, 1992).

Health effects from environmental toxicant may be a more serious problem in developing countries because the problem is potentiated by other factors such as lack or failure to enforce regulations, which allow human exposure to genotoxic agents, under nourishment of the lower economic and social classes that comprise the most exposed population from industries and agricultural activities, and parasitic infections in both urban and rural areas. (Speight, 1996).

The sectors of the economy which are likely to contribute to a continuing general increase in environmental pollution are the automobile traffic sectors, general industrial manufacturers, the heavy civil construction sectors and the combustion of fossil fuel. (Onianwa 1985).

The environmental pollution level all over the world continuous to rise as a result of the growth in the volume of industrial production. The most important economic problems concerning the environment include the struggle against hunger, disease and poverty and the improvement of the living conditions of most countries of Africa, Asia, and Latin America (UNESCO/UNEP, 1985).

The principal form of control of environmental pollution is legislation on the protection of air, water, soil, fauna, flora and other components of nature. On the basis of laws regulating relationships of the economy with the environment, a system of ecological standards is established with administrative and economic measures to ensure their maintenance (WHO, 1995).

1.2 Historical Background

The metal is heavy, pliable and very resistant to corrosion and weathering. It is easily melted down and re-used. These properties account for its widespread traditional uses. For instance in building, plumbing, printing, shooting and fishing many of which continue today. Even where substitutes are now preferred or required artifacts from earlier times survive and are still in widespread use. Since the industrial revolution, other properties and uses of lead have been discovered and widely exploited. In its metallic form lead is uniquely suitable for use in heavy electrical insulation, radiation shielding and battery manufacture. Various compounds of lead are used in paints, plastics, ceramics, glass and petrol (RCEP, 1983).

The cumulative effect of centuries of use is that lead is now one of the most widely dispersed of environmental pollutants. Moreover, there is no innocuous form into which it can be converted in the environment. Once in the environment lead and its compounds do not move readily through natural pathways to remote locations such as ocean beds, and they have long environmental residence time compared with other pollutants, particularly in the soil. Thus not only is there widespread human exposure to lead today but future generations too will be exposed to the lead which is already in the environment and is being added to all the time (RCEP, 1983).

Lead is ubiquitous in the human environment as a result of industrialization. It has no known physiological value. Children are particularly susceptible to its toxic effects. Lead poisoning, for the most part, is silent, most poisoned children have no symptoms. The vast majority of cases, therefore go undiagnosed and untreated. Lead poisoning is widespread. It is not solely a problem of innercity or minority children. No socio-economic group, geographic area or racial ethnic population is spared (CDC, 91).

Lead in the environment may enter the body through either inhalation, ingestion, or percutaneous absorption. The later route is considerably less significant than the respiratory and gastrointestinal routes for uptake of inorganic lead (WHO, 1980) lead taken in through the respiratory system is absorbed upto about 10-15% although the efficiency of absorption of lead from drink is critically dependent upon whether the drink is taken before food (high lead uptake) With or after food (low lead uptake) (Sherlock, 1991)

Studies carried out most recently by Adogame, (1997), Osagie, (1998), Okeakaru, (1998) and Aborkor, (1998) revealed that lead levels in indoor and outdoor environment in Lagos and Ibadan city with respect to water, food, dust and soil samples were relatively high above the permissible limits. This therefore, provoked my interest to carry out a similar study among children in Lagos metropolis since no such comprehensive study has been carried out.

1.3 Objectives

1.3.1 Broad Objectives

To assess the level of lead (Pb) exposure among children aged 2-5 in selected Nursery schools.

1.3.2 Specific Objectives

1. To assess the quantity of lead found in the handwashings of nursery school children.

2. To determine if there is any difference between the quantity of lead on the hands of boys and girls.
3. To determine the quantity of lead on toy surface used commonly by children in schools.
4. To identify and assess the high-risk environment for lead exposure e.g play ground, classroom, roof top water and cosmetics.
5. To find out how much knowledge the teachers have about lead poisoning and
6. To recommend measurement to reduce the lead exposure risk among school children.

1.4 Significant of the study

Our environment is in a state of rapid change caused by various human activities that has serious impact on the environment. Lead poisoning is growing day by day due to its health implications.

On this basis, there have been various alarms recently that the level of lead poisoning in Nigeria has reached dangerous proportions, with children as the most vulnerable group. One of such alarms was raised in Nigeria by a group called Alliance to End Childhood Lead Poisoning based in Washington, United State of America. A research of this nature will help to clarify the above assertion.

Again, there is scanty information about lead levels of children blood and environment. This study is aimed at providing baseline data of lead exposure in hands, toys, dust and soil in their immediate environment. Therefore, this research will be useful in categorizing the high-risk environments for lead and designing strategies by FEPA and other governmental and non-governmental bodies to reduce lead levels in the environment.

1.5 Statement of the problem

Daily, we find thousands of vehicles on our roads, pumping tonnes of highly potent neurotoxins into the air, all over the country, especially in the urban, highly commercial centers. The most prominent of the anthropogenic toxins, in addition to that from sources like batteries, paints and water pipes is organic lead as in the form of tetraethyl-lead (TEL) an additive to automobile fuel to improve combustion. This, when established at certain levels in the human system, causes lead poisoning which can result in deformities in unborn children, retarded mental development and cause neuropsychological and behavioural impairment in children. It causes serious nervous system dysfunction, high blood pressure and severe stomach disorder in adults. Damage from regular exposure to lead is irreversible. Children are most susceptible to permanent damage (Agbo, 1997).

Domestic lead main sources are usually lead- based paints and drinking water carried through lead pipes. Lead in based paints are especially harmful to children who chew painted toys, furnishings and eats painting peelings from walls. In 1984, approximately, 17% of children in United States of America were estimated to be at risk of lead poisoning (CDC, 91). Sizeable number of children from families with incomes well above the poverty line have been reported to have elevated blood lead levels. Lead poisoning is associated with learning impairement, reduced IQ, and hyperactivity in children, high blood pressure in adults, underweight and premature newborns (CDC, 91).

1.6 Justification for the study

Lagos metropolis, which is noted by (UNDP, 98) as one of top ten most populous cities in the world by the year 2015, is characterized by by high traffic congestion, indiscriminate waste dumps, poor drainage and high socio- economic activities. These features result in contamination of foods, air, water and soil pollution.

Adedibu, (1990) described Lagos as probably the dirtiest capital of any nation in the world because solid wastes litter the streets, open spaces, markets, car parks and other public places. This therefore, justifies the selection of Lagos metropolis as a study area.

A decade ago, alarmed by mounting evidence that lead poisoning had become a widespread disease, health officials in Australia, Denmark, Germany, Mexico, Scotland and the United States of American began studies to determine how dangerous even low levels of lead are to human beings, especially children.

However, concrete evidence is needed in respect of prevalence of lead in the Nursery schools in Lagos metropolis where children learn and play while their parents are away to work. This data is necessary to stimulate sustained advocacy for the control and prevalence of lead poisoning in Lagos metropolis.

1.7 Scope of the study.

This study is primarily restricted to Nursery school environment in Lagos metropolis. Samples of handwashings, soil from playground, dust from classroom sweeping, roof top water, eye based cosmetic samples were examined from the various sampling sites.

CHAPTER TWO

LITERATURE REVIEW

2.1 Lead

2.1.1 Physical and Chemical Properties of Lead and its Compounds

Lead (atomic number, 82, relative atomic mass, 207.19, specific gravity, 11.34) is a bluish or silvery grey soft metal. The melting point is 327.5°C and the boiling point at atmospheric pressure 1740°C. It has four naturally occurring isotopes (208, 206, 207, and 204 in order of abundance), but the isotopic ratios for various mineral sources may differ. This property has been exploited in non-radioactive-tracer environmental and metabolic studies. The physical and chemical properties of elemental lead and some lead compounds are summarized in table1.

Although lead has four electrons in its valence shell, only two ionize readily. The usual oxidation state of lead in inorganic compounds is therefore +2 rather than +4. The inorganic salts of lead, such as lead sulfide and the oxides of lead, are generally poorly soluble in water. However, the nitrate, chlorate and to a much lesser degree, the chloride are water soluble some of the salts formed with organic acids, e.g., lead oxalate, are also insoluble, but the acetate is relatively soluble as shown in Table 1.

TABLE 1

Physical and chemical data on lead and selected lead compounds

Name	Synonym and formula	Relative atomic/ molecular mass	Melting points (°C)	Boiling points (°C)	Soluble in cold water (g/litre)	Soluble in
Lead	Pb	207.19	327.502	1740	Insoluble	HNO_3; hot concentrated H_2SO_4; hot water, glycerine, alcohol (slightly)
Lead salts						
Acetate	$Pb(C_2H_3O_2)_2$	325.28	280		443	
Carbonate	Cerrusite $PbCO_3$	267.20	315 (decomposes)		0.0011	Acid, alkali; decomposes in hot water
Chlorate	$Pb(ClO_3)_2$	374.09	230 (decomposes)		very soluble	Alcohol
Chloride	Cotunite $PbCl_2$	278.10	501	950	9.9	NH_4 salts, slightly in dilute HCl and in NH_3, hot water (33.1g/litre)
Nitrate	$Pb(NO_3)_2$	331.20	470 (decomposes)		376.5	Alcohol, alkali, NH_3, hot water (1270g/litre)
Ortho-Phosphate	$Pb_3(PO_4)_2$	811.51	1014		0.00014	Alkali, HNO_3
Oxalate	PbC_2O_4	295.21	300 (decomposes)		0.0016	HNO_3
Dioxide	Plattnerite PbO_2	239.19	290 (decomposes)		insoluble	Dilute HCl, acetic acid (slightly)
Monoxide	Litharge PbO	223.19	888		0.017	Dilute HNO_3, acetic acid
Sulfate	Anglesite $PbSO_4$	303.25	1170		0.0125	NH_4 salts, concentrated H_2SO_4 (slightly)
Sulphide	Galena PbS	239.25	1114		0.00086	Acid

Data from Weast (1985)

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Under appropriate conditions of synthesis stable compounds are formed in which lead is directly bound to a carbon atom. Industrially synthesized lead-carbon compounds include tetraethylead and tetramethyllead, which are of importance as fuel additives and hence, are sources of environmental lead (WHO 1993).

2.2 Sources of Human Exposure

The level of lead in the earth's crust is about 20mg/kg (WHO 1993). Lead in the environment may be derived from either natural or anthropogenic sources. Natural sources of atmospheric lead include geological weathering and volcanic emissions and have been estimated at 19,000 tonnes/year, (Nriagu and Pacyna, 1988) compared to an estimate of 126,000 tonnes/year emitted to the air from the mining, smelting and consumption of over 3 million tonnes of lead per year (WHO 1993).

Lead and its compounds enter the environment at any point during mining, smelting, processing, use, recycling or disposal. Major uses are in batteries, cables, pigments, petrol (gasoline) additives, solder and steel products. Lead and lead compounds are also used in solder applied to water distribution pipes and to seams of cans used to store foods, in some traditional remedies, in bottle closures for alcoholic beverages and in ceramic glazes and crystal tablewares. In countries where leaded petrol is still used, the major air emission is from mobile and stationary sources of petrol canbustion (Urban centres). Areas in the vicinity of lead mines and smelters are subject to high levels of air emissions (WHO, 1993).

Airborne lead can be deposited on soil and water, thus reaching human's through the food chain and in drinking-water. Atmosphere leadies also amajor source of lead in drinking water.

2.3 Environmental Transport, Distribution and Transformation

The transport and distribution of lead from fixed, mobile and natural sources are primarily via air transport between environmental compactment such as, vegetation, soil, house dust and water takes place as shown in Fig 1. Most lead emissions are deposited near the source. However, approximately 20% is widely dispersed (Nriagu, 1979. IPCS, 1989), although some particulate matter (<2μm in diameter) is transported over long distances and results in the contamination of remote sites such as artic glaciers (Settle and Patterson, 1980).

Atmospheric conditions and particulate size influence the removal of airborne lead (Nielsen, 1984). Large amount of lead maybe discharged into soil and water, however such material tends to remain localized because of the poor solubility of lead compounds in water. (IPSC, 1989).

Lead deposited in water, weather from air or through run-off from soils, partitions rapidly between sediment and aqueous phase depending upon the pH, salt content, and the presence of organic chelating agents. Above pH 5.4, hard water may contain about 30µglead/litre and soft water above 500µg/litre (Davies and Everhart 1973). Very little lead deposited on soil is transported to surface or ground water except through erosion or geo-chemical weathering; it is normally quite tightly bound (chelated) to organic matter (Lovering, 1976).

Airborne lead can be transferred to biota directly or through uptake from soil. Animals can be exposed to lead directly through grazing and soil ingestion or by inhalation. There is little biomagnification of inorganic lead through the food chain. (IPCS, 1989).

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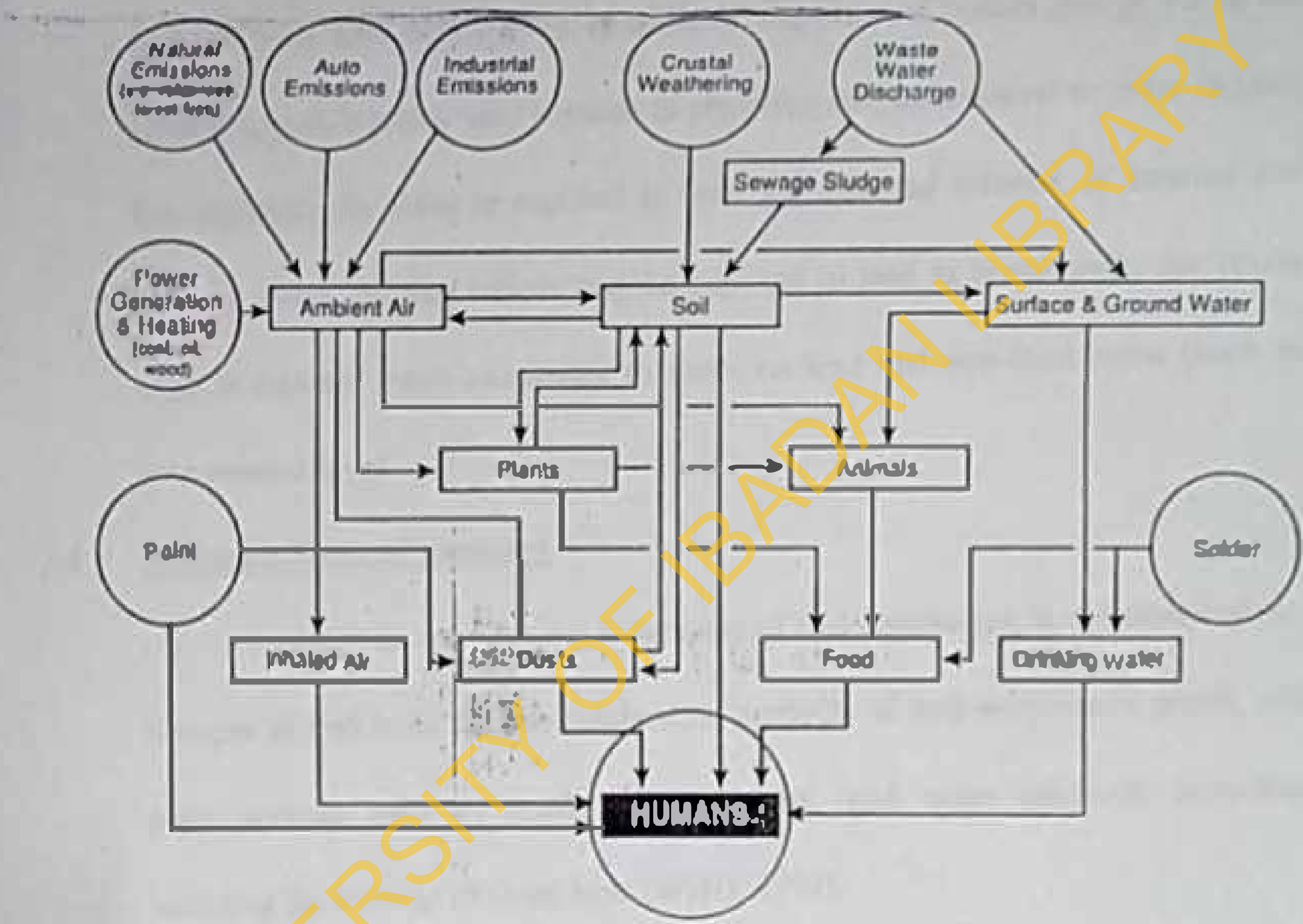

Fig 1 Pathways of human exposure (OECD, 1993)

2.4 Environmental Levels and Childhood Exposure

The general population is exposed to lead simultaneously from many sources and through multiple pathways as shown in Fig. 1 such as air, water food, dust and soils (ATSDR, 1996). In addition, exposure of certain groups within the general population may vary because of physiological, behavioural or other factors. For example, the fetus is exposed to lead via maternal transfer of internal and external doses, nursing infants may be exposed to lead in breast milk, the young child is exposed more intensively to dusts on lead and non-food items (such as lead-painted toys).

2.4.1 Inhalation Route of Exposure

Ambient air can be a major pathway of lead distribution in the environment. Sources of lead in air include combustion products of lead additives in petrol, and point sources such as smelters, incinerators, and some industrial processes including the burning of fossil fuels (WHO, 1993).

Concentrations of lead in air range from 7.6 x 10^{-5} µg/m^3 in remote areas such as Antarctica (Maenhaut et al, 1979) to >10µg/m^3 near lead smelters (Elias, 1985).

Almost all lead in air is bound to fine particles less than 1µm diameter, although some may be solubilized in acid aerosol droplets. The size of these particles varies with the source and with age of the particle from the time of emission. (US EPA, 1986a, WHO, 1987).

2.4.2 Emissions from Motor Vehicles.

In Mexico, where leaded vehicle fuel is still in use, airborne concentration of lead in the urban areas are likely to be in the range of 0.5 – 3μg/m^3 (WHO, 1987). Concentration of between 0.6 and 5. 7μg/m^3 were reported in Europe in 1982 (GEMS, 1985). Where leaded vehicle fuel is no longer used, concentrations are likely to fall to < 0.2μg/m^3 (Elias, 1985).

In South Africa, average atmospheric concentrations of lead were found to be 1.8, 0.86, 0.56 and 0.44μg/m^{-3} in industrial, commercial, park/beach and residential areas in Durban (Nriagu et. al; 1996). In Nigeria, automobile exhaust constitutes 75-80% of the gross air pollution and lead is a major contaminant. The lead levels in Nigerians super grade petrol is in the range of 210-250mg/litre (Ademoroti. 1986).

2.4.3 Stationary Sources

Where emmissions are largely uncontrolled, concentrations of lead in air around stationary sources such as lead smelter range from 10μg/m^3 50m from the smelter to 1.5μg/m^3 to 1cm away (Wang et al; 1992).

2.4.4 Indoor Air.

Davies et al; (1987a) sampled indoor and ambient air lead levels and found that whose there was no interior lead sources such as lead-painted surfaces, air lead concentrations inside dwelling were highly correlated with those outside and averaged approximately 60% of those in the external air.

2.4.5 Air in the working environment.

Airborne lead concentrations in the occupational setting vary considerable according to the type of industry and the level of industrial hygiene practised at each plant. Occupations and operations that may present lead hazards to workers are listed in Table 2.

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TABLE 2

Occupational or operations, which may present lead hazards for workers

Primary and secondary lead smelting	Lead minning
Welding and cutting of lead-painted metal constructions	Plumbing
Welding of galvanized or zinc silicate coated sheets	Cable making
Shipbreaking	Lead casting
Nonferrous foundries	
Storage battery manufacture: pasting, assembling, welding of battery connectors	Type found in printing shops
Production of lead paints	Stereotype setting
Spray painting	Assembling of cars
Mixing (by hand) of lead stabilizers into Polyvinyl chloride	Automobile repair short making
Mixing (by hand) of crystal glass mass	Welding (Occasionally)
Sanding or scraping of lead paint	Lead glass blowing
Burning of lead and enamelling workshops	Pottery/glass making
Repair of automobile radiators	

Adopted from: Hernberg (1973)

2.4.6 Smoking of Tobacco

Lead is present in tobacco, the mean content of lead in filter-tipped cigarettes produced between 1960 and 1980 was 2. 4μ/g. Approximately 5% of this lead may be inhaled. The remainder occurs in the ash and side – stream smoke (Mussalo-Rauhamaa et al, 1986).

2.5 Exposure By Ingestion

2.5.1 Water

Background or natural levels of lead in surface and ground water are generally low. However, water with low pH and only low concentration of dissolved salts (referred to as aggressive) can leach substantial quantities of lead from pipes, solder and fixtures (WHO 1995) Lead – lined reservoirs, cisterns and holding tanks for water (Mushak and Crocetti, 1989) can be a major source of lead contamination of drinking water.

Sampling programmes conducted at the taps in the USA during 1985 – 1988 revealed widespread elevation of lead in drinking-water, often above the WHO guidance value of 50μg/litre (WHO 1984 which has now been revised to 10μg/litre (WHO, 1993).

In two separate studies, Adogame (1997) reported lead levels of 0.01-2.16mg/litre in water samples collected while Okekearu (1998) reported lead levels in tap water in the range of 0.138 – 1.23mg/l for Lagos metropolis.

2.5.2 Food

The proportion of total intake derived from food is dependent on the concentration of lead in air, water and other sources. Detailed data are available from several countries, including Australia (Vahter et al; 1990) and Canada (Dabeka et al; 1987). Children are exposed to additional lead from dust and soil. Data on the lead levels of specific foodstuff or groups of food materials, from which one can estimate a daily dietary lead intake, are available from several countries. (Bolger et al; 1991, Kolbye et al; 1991 and Albert and Badilo, 1991). Data are also available for canned food typically consumed by young children (Carpar and Rigsby, 1989).

The lead levels in infant food in Canada, Mexico and USA are shown in Table 3. In 1987, Dabeka et al; (1987) found the intake of lead by infants fed on evaporated milk stored in lead – soldered cans exceeded the Provisional Tolerable Weekly Intake of 25μg lead/kg body weight, set in 1993 (FAO/WHO, 1993).

These values do not include lead in water used to prepare formulae. It has been reported that infants fed formulae prepared with water containing high levels of lead (> 100μg/litre) have lead intakes exceeding 25μg/kg body weight per week (Galal-Gorchev, 1991b).

TABLE 3

Lead levels (µg/kg food) in cow's milk and infant formula

Product.	Canada. Median (range)[a]	Mexico Average[b]	USA Average[c]
Fluid milk	1.19 (0.01-2.5)	5	
Evaporated milk (canned)	71.9(27-106)	88	10
(cardboard)	—	9	
Infant formula			
Ready to use lead-solder can	30.1 (1.1-12.2)	13	10
Ready to use lead-free can	1.6 (1.5-2)		1
Formular powder (1985)	96.6(3.7-19)		
Powdered milk[d]	—	21	

a. *From: Dabeka and Mckenzie (1987).*
b. *From: Albert and Badilo (1991)*
c. *From: Bolger et al. (1991)*
d. *The concentration of lead in milk consumed by the infant will be highly dependent on the infant will be highly dependent on the concentration of lead in water used to dilute the powdered milk.*

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2.5.1 Dust and Suface Soils.

Dust is a significant source of lead particulary for young children (see Fig 2) as has been demonstrated in several studies correlating children's blood lead concentrations with dust lead levels concentrations with dust lead levels (Rabinowitz et al; 1985, Bronschein et al; 1987, Davies et al 1987a, Laxen et al, 1987, Steenhout, 1987).

The major contributions to lead levels in soil and outdoor dust are from the combustion of fossil fuels (principally leaded petrol), stationary sources such as smelters, peeling and flaking of lead-based paint. Typically, lead levels in road dust in the USA are 800-1300mg/kg in the rural areas to 100-5000mg/kg in urban areas (US-EPA, 1989c).

Flaking lead based paint, paint chips and weathered powdered paint markedly increase intake of lead from surface dust, particularly for urban children with pica (US-EPA, 1986a, Bronschein et al, 1986). Lead – based paint chips have been found to contain 1000-5000- $\mu g/cm^2$ (Billick and Grace, 1978). When lead-based paint is present, interior renovation activities greatly increase household dust lead concentrations (Laxen et al, 1987). Improved control of dust and surface clean-up after lead-based paint removal have been shown to reduce lead exposure of children reoccupying affected houses (Charney et al., 1983).

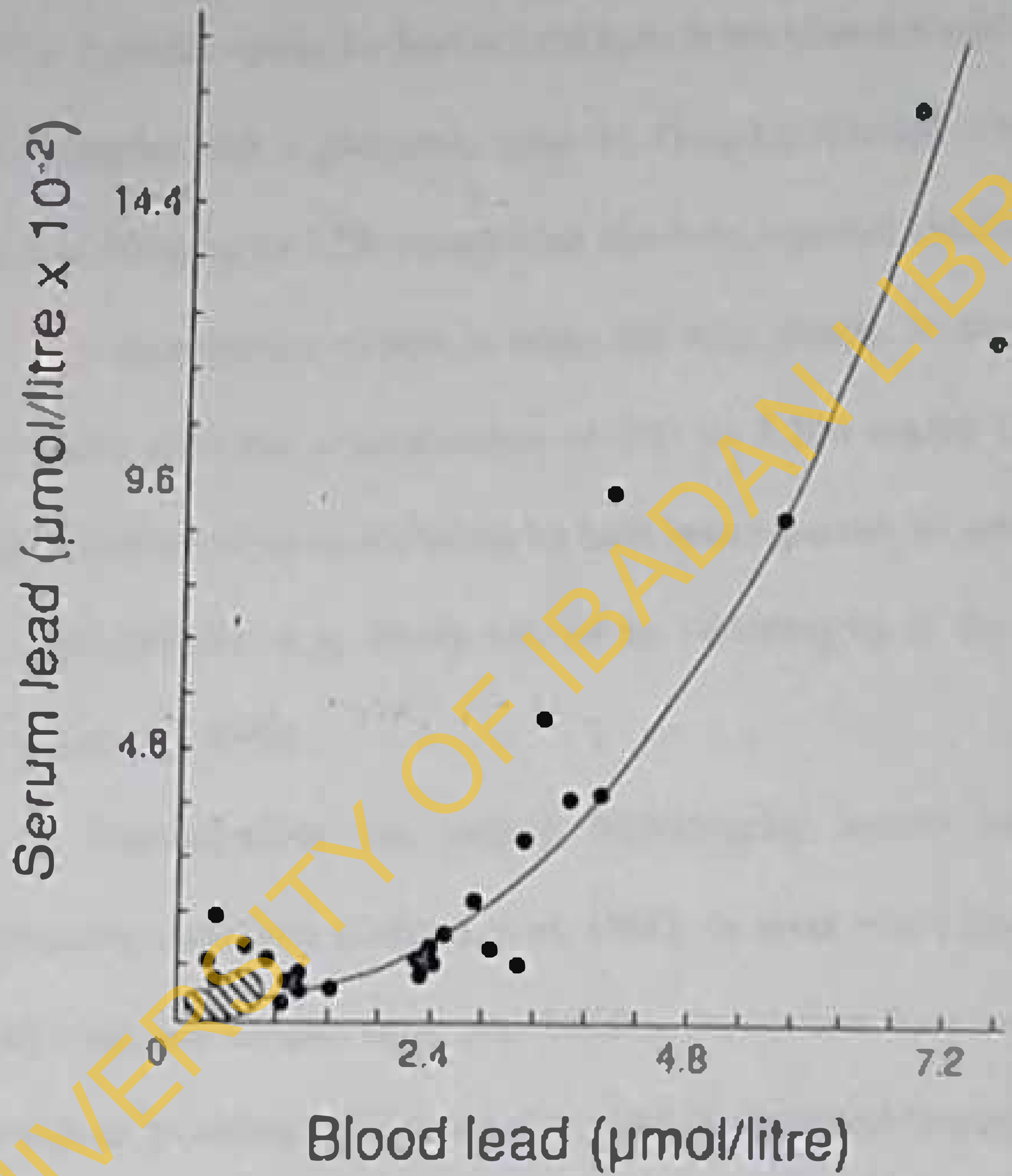

Fig. 3. The curvilinear relationship of serum lead to blood lead levels (the hatched area contains 23 points) (adapted from: Manton & Cook, 1984)

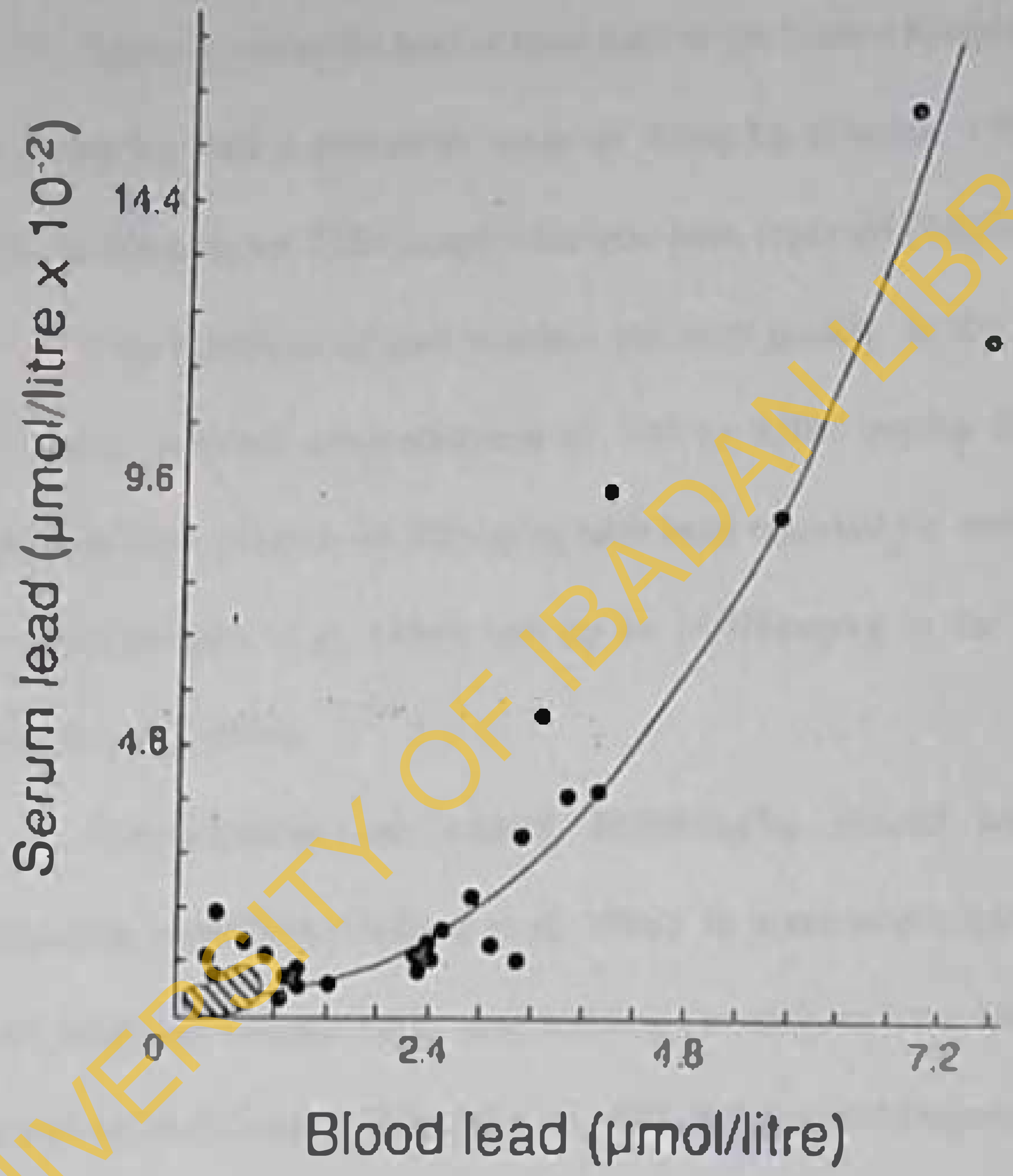

Fig 3. The curvilinear relationship of serum lead to blood lead levels (the hatched area contains 23 points) (adapted from: Manton & Cook, 1984)

2.5.2 Soil

In rural and remote areas, lead in soil is derived mainly from natural geological sources. These natural sources account for 1-30mglead/kg (WHO 1995). Typically values for lead in rural soils in the United Kingdom are 15-106mg/kg with a geometric mean of 42mg/kg (Davies, 1983). A geometric mean of 48mg/kg for 2780 samples has also been reported (McGrath, 1986).

Concentrations of lead in urban soil vary greatly. In the USA, a study of city parks recorded concentrations of 200 to 3,300 mg/kg (US EPA, 1989). Concentrations of up to 10,960mg/kg have been reported for urban garden soils in the USA (Mielke et al; 1984), and up to 14,100mg/kg in the United Kingdom (Culbard et al, 1988).

Concentrations can exceed 20,000mg/kg around lead minning and processing operations (Culbard et al, 1988). In areas where lead-based paint has been used, soil samples taken near building foundations have been reported to be as high as 20,000mg/kg (Schmitt et al; 1988, Knieger and Duguay, 1989).

In general, lead concentrations in soils near reads are high where road traffic density is high. Concentrations decreases exponentially with distance from the road (IPCS, 1989). Continuous application of sewage sludge results in an accumulation of lead in soil. For example, soil receiving heavy applications over a long period was found to contain 425mg/kg, compared with 47mg/kg in an untreated soil (Beckett et al; 1979).

A study carried out in Ibadan by Adogame, (1997) indicated that lead levels in soils were in the range 123-491mg/kg for residential communities, 150-3,862mg/kg for petrol stations, 1,473-11,246mg/kg for battery industry and 71-4,377mg/kg for mechanic villages.

Most recently, a study carried out in Lagos by Osagie (1998), indicated that lead levels in soils were in the range of 454-3,089mg/kg for mechanic villages and 81-983mg/kg for road sides.

2.5.3 Cosmetics and Medicines

Some traditional medicines and customs have been found to result in exposure to high levels of lead, most of which cannot be quantified with any degree of accuracy. Rather than occuring as trace ingredients or trace contaminants, various lead compounds are used as major ingredients in traditional medicines in numerous part of the world (Table 4). Clinically over lead poisoning due to traditional cosmetics and medicines has been identified among infants (Shaltout et al, 1981, Fernando et al, 1986, Sharma et al: 1990. Children and Adults (Pontifax and Grag, 1985, Cueto et al. 1989, Mitchel-Hegg et al, 1990, Gupta et al; 1990). There are case report of lead toxicity secondary to inhalation of lead from traditional remedies (Aslam et al, 1979, Shaltout, 1981, Cueto et al, 1989, Sharma et al, 1990, Michell-Hegg et al, 1990).

Often the use is not limited to adults, these may be used on infants and young children as well as on women. In Kuwait, the leaded "Kohl" also called Al'Kohl' is traditionally applied to raw umbilical stump of the newborn in the erroneous belief of a beneficial astrigent action (Fernando et al, 1981). An additional use of lead metal and lead sulfide is for inhalation of the fumes ("Bokhoor") produced from heating on hot coals in the mistaken belief that this will calm irritable infants and children (Fernando et al, 1981. Shaltout et al, 1981).

Latin American countries also report the use of traditonal medicines with high lead concentrations. For example, the Mexican traditional remedy "azarcon" (lead Chromate and or "greta" (mixed lead oxides), distributed as finely ground powders may contain more than 70% lead. They are used in the treatment of "empacho", a gastrointestinal disorder considered to be due to a blockage in the intestine (Trotter, 1990).

TABLE 4

Sources of lead exposure in traditional medicines and cosmetics

Sources of lead (product)	Comments	References
Surma/Kohl	Used in Indo-Pakistan and other Muslims as eyes preparation; placed on conjuctival surface or as astingent on umbilical cord stump. Antimony originally used but lead cheaper.	Aslam et al (1979); Fernando et al (1981), Shaltout et al, (1981); Sharma et al. (1990)
Hindu folk medicine	Ground seeds and roots as treatment for diabetes (8mglead/g)	Pontifax & Gard (1985)
Bokhoor	Tribal custom to produce lead fumes to ward off evil.	Shaltout et al (1981)
Azarcon	Lead chromate and mixed lead oxides as treatment for gastrointestinal disorders inMexico and southwestern USA	Trotter (1990)
Skin ointments and cosmetics	Cosmetics used by Chinese actors; skin ointment in Europe	Lai (1977)

Source: WHO, 1995

2.6 Kinetics and Metabolism in Humans

2.6.1 Absorption:

The absorption of lead from environmental sources is not dependent solely of entry. It is also dependent on the physical and chemical state in which the metal is presented, and it is influenced by host factors such as age, physiological status, nutritional condition and possibly genetic factors. (WHO 1977).

2.6.2 Absorption after inhalation.

The absorption of lead from air to blood involves two processes: the deposition of air borne particles in the respiratory tract, and the clearance from the respiratory tract from circulation. (WHO 1995). Using the International Radiological Protection Commission (IRPC), document on long dynamics (Task Group on Lung Dynamics, 1966), a model was developed which predicted that 35 inhaled lead is deposited in the respiratory tract (40 to 50% of particles with a mean mass median aerodynamic diameter (MMAD) of 0.5μm, such as are typically generated by automobiles). These are deposited primarily in the alveolar sacs of the lung lead fumes and vapours such as those generated in operations where metals are cut or heated, are of very small size and are respirable. Absorption after deposition will vary according to the solubility of the lead species (e.g. lead carbonate or lead chloride aerosols) and the inherent toxicity to macrophages and cilia (WHO, 1995).

There are no quantitative data on the absorption of leaf in children after inhalation exposure. It is known that young children weighing only one sixth of an adult inhale 40% of the fairly volume of an adult and a proportionately higher daily air volume per unit measure (weight, body area) than do adults (Barltrop, 1972). After controlling for weight and taking into account difference in the anatomy of the respiratory tract between adults and children, James (1978) calculated a rate of deposition of lead particles in children which was 1.6 to 2.7 times that of adults.

2.6.3 Absorption Of Lead From The Gastro Intestinal Tract

In older children and adults without occupational exposure, lead absorbed by the gastro-intestinal tract comes from the intake of lead in foods, beverages and soil/dust. In pre-school children, there is concern over the intake of both food and non-food items (e.g. toys, soil/dust). Young children may take in lead from non-food items, via normal mouthing activities, which in the extreme, is the behavioural trait pica which refers to the ingestion of such materials as soil, ash, paint chips and plaster (US EPA, 1986a). For infants and young children, the extent of absorption of the lead in dust/soil is from the gastrointestinal tract is extremely important, particularly for children living in urban environment.

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Gastrointestinal absorption of lead in humans, as in experimental animals is influenced by dietary factors, nutritional status and the chemical form of the metal. For example, lead ingested during periods of fasting is absorbed to a much greater extent than lead ingested with food (WHO, 1995). Chamberlain et al, (1978) reported 45% absorption of lead chloride in fasting subjects and only 6% in feeding subjects. Using similar procedure, Heard and Chamberlain (1982) reported absorption of 63.3% in fasting subjects.

Ziegler et al. (1978) reported that young children, aged two weeks to two years, absorbed 42% of ingested lead at levels of intake greater than 5µg/kg body weight. Drill et al. (1979) estimated an absorption rate of 17% for lead in paint chips in children aged 2 to 3 years. Using data from Day et al. (1975) and Lepow et al. (1974), it has been estimated that children 2 to 3 years of age ingest about 100mgsoil per day. Using aluminium and titanium as tracers, Clausing et al. (1987) estimated that children aged 2 to 4 years ingest between 50 to 100mg of soil daily. The average amount of soil ingested by young children has recently been estimated to be between 12.5 and 21mg/day (SAHC, 1993).

2.6.4 Nutritional Status and Lead absorption via Gastrointestinal Tract

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It has been known for some time that the absorption and distribution of lead are affected by nutritional status in both experimental animals and humans (Sobel et al. 1940). Vitamin D, calcium and phosphorus have complex and interrelated effects on lead absorption (Fullmer, 1990). Increasing the concentration of 1,25-dihydroxychole-calciferol, the active metabolite of Vitamin D, either exogenously or endogenously, increases gastrointestinal absorption of lead (Fullmer, 1990). However, this effect is dependent upon the duration of lead exposure and the magnitude of body lead stores. This homeostatic mechanism for calcium and its dependence on nutritional status, as well as body burden of lead, is complex (WHO 1995). This may explain the divergent results of the observed interaction in children (Rosen et al. 1980) or the lack of association (Koo et al. 1991) of lead with Vitamin D metabolism.

2.6.5 Dermal absorption

Moore et al. (1980) examined the uptake of lead acetate from two hair-darkening cosmetics through the skin of 8 human volunteers. Only minute quantities of lead (0-0.3% of the applied dose) were detectable in blood, and there was only a slight increase in absorption when the skin was damaged. Lilley et al. (1988) and Florence et al. (1988) have reported the dermal absorption of inorganic lead compound leading to elevated levels of lead in human saliva and sweat.

2.7 The relationship of external Lead expose to blood Lead concentration in children

Blood is the compartment in which lead is most often measured as a marker of exposure. However, it typically represents relatively recent exposures, since the half-life of lead in blood is short (US EPA, 1986a) and has been estimated to be in the order of 36 days from tracer studies (Rabinowitz et al. 1975). Lead in the blood is derived from levels in the current environment and from lead stored in tissues (particularly bone) that re-enters the blood during tissue mobilization (Manton, 1985). Both population and experimental studies have been used to estimate the blood lead.

2.7.1 Ambient air Lead relationships in children

Under ambient conditions (air Lead concentration of 0.1 – 2.0µg/m^3) and PbB levels less than 1.44µmol/litre (<30µg/dl), the relationship has been described as linear (Colombo, 1985). When one calculates the relationship between PbB and the total contribution from air (direct inhalation plus indirect through dust/soil), a value of about 0.14 – 0.24µmol/litre (3 - 5µg/dl) blood per µg/m^3 is obtained (Brunekreef, 1984; US EPA, 1986a).

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2.7.2 Food

The relationship of PbB to dietary intake has been estimated from experimental (Stulk 1974) and as well as population studies (Ryu et al 1983) a slope of 0.0096µmol/litre (0.2µg/dl) per µg lead/day was obtained for infants aged 8 to 196 days.

2.7.3 Drinking Water

In a study by Sherlock et al (1982), a cube root relationship between lead levels in drinking water and blood fitted the data generated.

2.7.4 Soil and dust

It is extremely difficult to choose the most appropriate model to describe the soil/dust to blood lead relationship given the many variables involved in determining the exposure pattern of children and the Kinetics involved between the levels in the environment and the child. A review of the available studies shows the extreme variability in slopes obtained (0.028-0.36µmollead/litre (0.6 – 7.6µg/dl) blood for each 1000µg/gsoil and 0.00096 – 0.35µmollead/litre (0.02–7.2µg/dl) blood for each 1000µg/g dust consumed by children (US EPA, 1986a). Detailed consideration has been given to the process for the assessment of lead contamination in soil and the derivation of soil clean-up criteria by Wixon (1991).

The guideline model uses a PbB target and slope for the soil. Blood lead relationship in the particular community in order to derive a PbB guideline. For house dust, a median value of 0.086μmollead/litre (1.8. μg/dl) blood per 1000μg lead/gdust in children appears to be based on data of reasonable quanlity, as does the 0.105μmol/litre (2.2μg/dl) blood per 1000μglead/gsoil from the same authors.

According to Elwood (1986), studies of the association between lead in house dust and PbB have given inconsistent test results and in general the only studies in which statistically significant association has been found are those where lead levels in dust are quite high. Landrigan et al. (1975), found a highly significant association between PbB and dust lead in an area with a mean level of 4022 μg/lead/gdust but not in two area with means of 922 and 816μglead/gdust. The US EPA (1986a) summarized a series of studies from which the overall relationship was judged to be 0.86μmol/litre (18μg/dlblood per 1000μg lead/g dust. Duggan (1980), reviewed the literature and determined that a slope of 0.24μmol/litre (5μg/dl) per 1000μglead/g dust is reasonable.

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2.8 Distribution

The initial distribution of lead in the body may depend upon the rate of delivery of blood to various organs. However, it would appear that distribution occurs in a similar manner regardless of route of absorption's (Kehoe, 1987).

Lead is distributed to both soft tissues (blood, liver, kidney, etc) and mineralizing systems (bone and teeth). Bone may be affected adversely by lead but also serves as the body's major storage sites. One absorbed, lead is not distributed homogeneously throughout the body but rather into several distinct compartments. Such biokinetic movements have been explained by Rabinowitz et al. (1976) using a three-compartment model.

In adults, approximately 94% of the body burden of lead is in the bones whereas only 73% of the body burden in children is located in this compartment (Barry, 1975, 1981). In view of the extremely long half-life of lead in bone, this compartments can serve as an endogenous source of lead to other compartments long after exposure ceases (O'Flaherty et al., 1982; Kehoe, 1987).

Under steady-state conditions, about 96% of PbB is in the erythrocyte. At PbB concentrations of <1.92μmol/litre (40μg/dl), whole blood and serum lead levels increase linearly in a positive manner. At higher PbB concentrations a curvilinear relationship is apparent and the serum to blood ratio increases dramatically (Manton and Cook, 1984). Such Kinetic relationships may be altered during pregnancy. From invitro data (Ong and Lee, 1980), fetal haemoglobin appears to have a greater affinity for lead than adult haemoglobin.

Bone lead is readily mobilized to blood and the effect is most apparent in people with a history of occupational exposure; bone lead also appears to be a major source of blood lead in older people with previous ambient exposures to lead. Of particular importance is mobilization of lead from bone in pregnant women and nursing mothers (Silbergeld, 1991). The mobilization of lead from bone to the more bio available material blood compartment poses a risk to the fetus and mother.

2.8.1. Transplacental Transfer

Lead is readily transferred from the mother to the developing infant during pregnancy and accumulates in bone during gestation (Barltrop, 1969). The lead concentration in cord blood is 85-90% that of maternal blood. Moore et al. (1982) reported a geometric mean level of 0.67μmol/litre (14μg/dl) in 236 pregnant women. The mean concentration of lead in umbilical cord blood from sample of over 11.000 women was 0.298μmol/litre (6.6±3.2μg/dl) (Bellinger et al.; et al, 1987)

2.9 Elimination and Excretion

In human's lead is eliminated from the body in both urine and feaces. Any dietary (including waterborne) lead not absorbed in the gastrointestinal tract is excreted in faeces. Airborne lead that has been swallowed and not absorbed is also eliminated in this manner (WHO, 1995). Blood lead not returned in the body is excreted in urine or faeces, the latter by billary excretion.

Chamberlain (1983, 1985) examined the relationship between the level of exposure and rate of lead excretion. Renal clearance at PbB levels of between 1.2 and 3.84μmol/litre (25 and 80μgl/dl) was found to increase at a rate approximating the increase in plasma lead.

Chamberlain (1985), estimated endogenous faecal lead loss into the gastrointestinal tract following administration of lead-203 via the inhalations and parentenal routes. These estimates suggested a clearance of approximately 0.5% of administered dose per day PbB concentrations were under 1.2μmol/litre (25μg/dl). A special form of excretion of endogenous lead is through breast milk. Studies of breast milk indicate that lead concentrations correlate with maternal PbB concentrations, (Ong et. al, 1985).

2.10 Biochemical Effects Of Lead In Children

Blood lead (PbB) is distributed between the plasma and the erythrocyte. There is less than 1% in the plasma for PbB level of up to 4.8µmol/litre (100µg/dl) (Manton and Cook, 1984). The curvilinear relationship of serum blood lead is shown in Fig 3. The data shows that the erythrocytes have a capacity to bind lead up to PbB level of about 2.4µmol/litre (50µg/dl). Above this level a fairly rapid increase in the serum levels occurs.

Lead is bound to haemoglobin in blood and has a greater affinity for fetal haemoglobin than adult haemoglobin (Ong and Lee, 1980). Also increased fetal haemoglobin has been found in cases of human and experimental animal poisoning (Albahary, 1972).

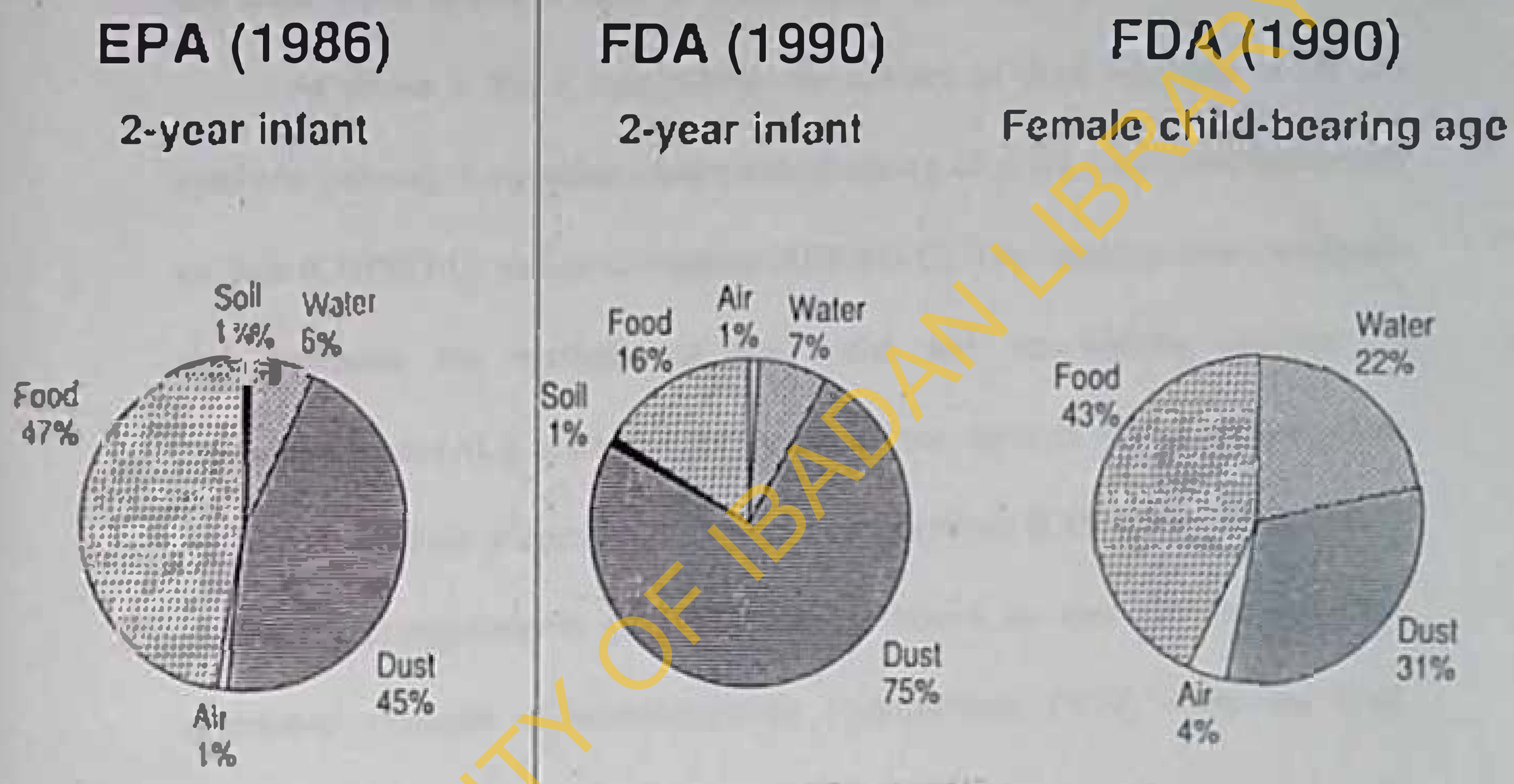


Fig. 2 . Percentage of lead intake from food and other sources in two-year-old infants and women of child bearing age in the USA (Adapted from: Bolger et al., 1991)

2.10.1 Haem Synthesis

Lead is known to affect several enzymatic reactions critical in haem synthesis causing abnormal concentrations of haem precursors in blood and urine and urine. These effects of haem synthesis are shown in Fig 4.

As shown in Fig 4, lead inhibits the activity of three enzymes of the bio synthetic pathway, 5-aminolaevulinate dehydratase (ALA-D), coproporphyrinogen oxidase (COPRO-O) and ferrochelatase (FERRO-C). This depletes haem synthesis and depresses the synthesis of the initial and rate-limiting enzyme 5-aminolaevulinate (ALA systhase). As a consequence, there is increased production and excretion of the precursors ALA and coproporphyrin (COPRO) with increased circulatory protoporphyrin (PROTO) usually bound to zinc. In the red cell, diminished synthesis of monooxygenase (cytochromes P450) comprises drug oxidation and is bound to haemoglobin (WHO, 1995).

2.10.2 Protoporphyrin levels

Lead interferes with the conversion of protoporphyrin to haem by ferro chelatase. This protoporphyrin exists under these circumstances primarily as zinc protoporphyrin with a portion remaining free (Chisolm and Brown, 1979).

A study by Koren et al (1990) of material and umbilical cord lead and free erythrocyte porphyrin (FEP) levels for 95 mother-infant pairs showed a correlation between maternal and cord PbB, with maternal levels exceeding neonatal levels

2.10.3 Coproporphyrin Levels

One of the earliest observed effects of lead poisoning was a rise in coproporphyrin excretion in the urine due to inhibition of coproporphyrinogen oxidase (Campbell et al, 1977).

2.10.4 δ-Amimolaevuline Acid Levels In Urine And Blood

Like protoporphyrin, circulating and excreted levels of ALA are likely to be best described as a continum of effect. Elevated levels of these compounds are of importance. Since neurological features of lead exposure have been ascribed in part to increased circulating levels of ALA (Moore et al, 1987). The rise in concentration during lead exposured is a functiom first of decreared activity of ALA dehydratase (ALAD), which is uniquely sensitive to lead toxicity, and subsequently of increased activity of the initial and rate-limiting enzyme of haem biosythesis, ALA synthase (Meredith et al., 1978).

Other effects of decreased haem synthesis the potential impact of a reduction in the body pool of haem and haem precursor is shown in Fig 5. (WHO. 1995).

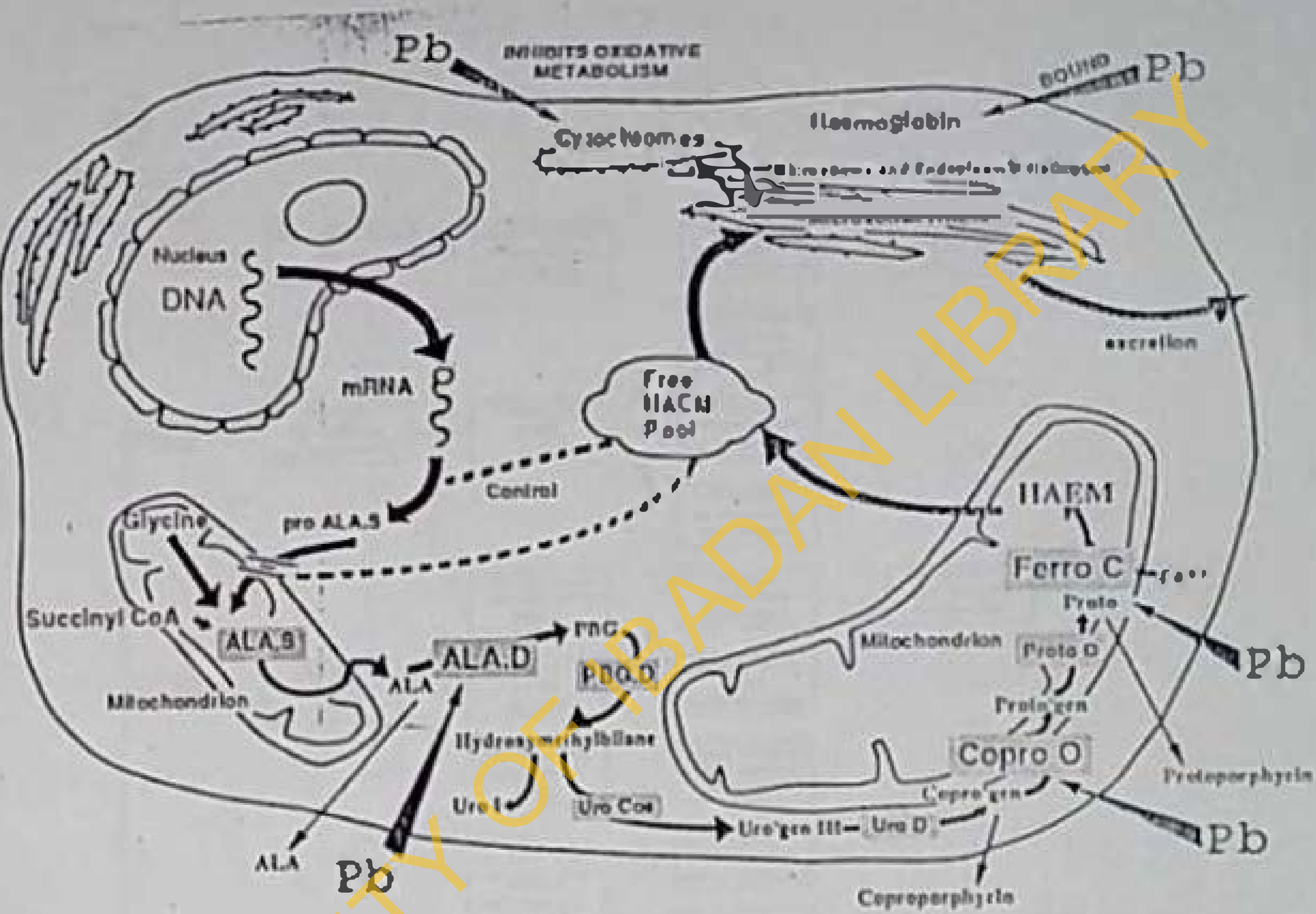


Fig. 4. The effects of lead on haem synthesis (personal communication by M. Moore to the IPCS, 1993)

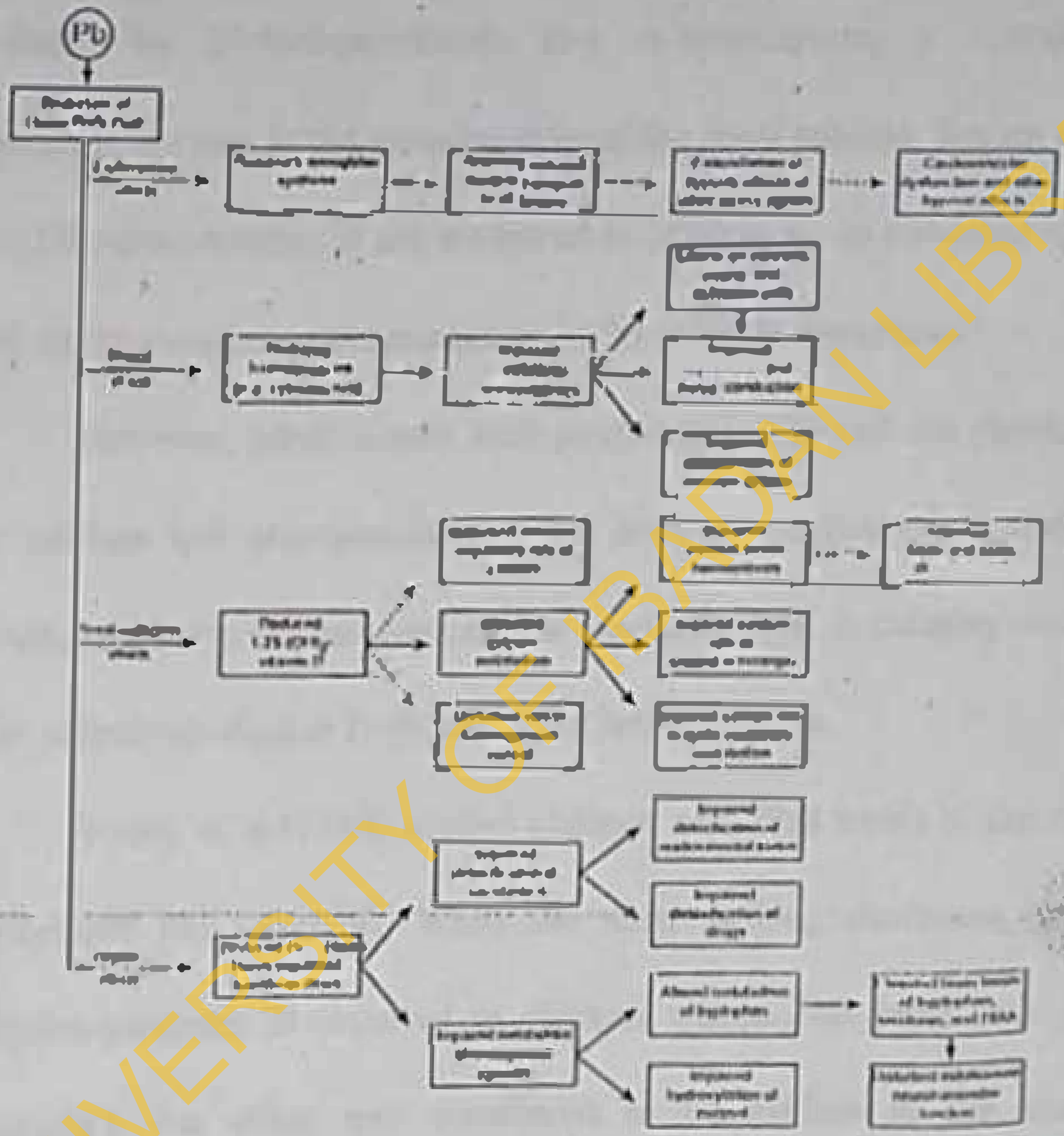


Fig.5 . Potential multi-organ impact of reduction of the haem body pool by lead (US EPA, 1986)

2.10.5 Vitamin D

Formation of the most important vitamin D metabolite, 1,25-dihydroxy vitamin D, is by 1α-hydroxylation of 25- hydroxyvitamin D in the kidney. This is mediated by 25-hydroxyvitamin D-1 α-hydroxytase, a cytochrome P450-dependent enzyme in the mitochondria of the renal tubules. Serum concentrations of 1,25hydroxyvitamin D are measured in children as an indicator of the effect of lead on the enzyme system mediating in the initial hydroxylation.

However, other factors such as dietary intake and the physiological needs for calcium and phosphorus, and the level of calciotropic hormones such as parathyroid hormone, can regulate the production and circulating concentrations of 1,25-dihydroxy-vitamin D (Rosen and Chesney, 1983).

Rosen et al (1980) studied children with PbB levels in the range of 1.58-5.76μmol/l (33-120μg/dl). While the most striking decreases in serum 1,25-dihydroxylvitamin D occurred in children whose PbB level was 2.97μmol/litre (62μg/dl), the effect was considered to be evident in the range of 1.58-2.64μmol/litre) (33-55μg/dl) when compared to age and race. Matched control group with PbB levels in the range of 0.48 – 1.248μmol/litre (10-26μg/dl).

2.10.6 Haematopoetic System

Lead-induced anaemia can be a direct consequence of inhibition of haem biosynthesis, it is not necessarily associated with iron deficiency. It may be associated with alterations of globin synthesis (Albahary, 1972). More importantly, the synthesis of α- and β- globin chains may become asynchronous (White and Harvey, 1972).

The PbB threshold for decreased haemoglobin levels in children is estimated to be approximately 1.92 μmol/litre (40μg/dl), (IPCS, 1977). However, a cross sectional epidemiological study of 579 children aged 1-5 years in 1974, living in close proximity to a primary lead smelter showed that adverse effects on haematocrit may occur at lower PbB levels (Schwartz et al; 1990).

2.10.7 Anaemia

Defined as a haematocrit below 35% was not found at PbB levels less than 0.92μmol/litre (20μg/dl). There was a strong non-linear dose response relationship at higher PbB levels which was influenced by age. Anaemia has been commonly associated with adverse effect of occupational lead exposure. It has an effect that is easily diagnosed clinically and is recognised as a marker of lead toxicity.

Anaemia may result from either a decrease in haemoglobin production or an increase in the rate of destruction of erythrocytes (IPCS, 1995). An analysis, made in 1974 of the association between PbB levels and haematocrit in 579 children (1-5 years of age) living near a primary lead smelter has recently been presented (Schwartz et al; 1990) a haematocrit value of less than 35% was used to indicate an adverse effect. The study concluded that there was no adverse effect of lead at PbB levels below 0.96µmol/litre (20µg/dl). Furthermore, the risk of having a haematocrit value below 35% for a 1-year olds was 2% at PbB levels between 0.96 and 1.87µmol/litre (20 and 30µg/dl). The degree of iron deficiency may account for a substantial proportion of this 2%. In this study, the level at which an effect of lead on the induction of anaemia was demonstrable was about 1.92µmol/litre (40µg/dl).

2.10.8 Pyrimidine – 5' - nucleotidase activity

Inhibition of erythrocyte pyrimidine –5' – nucleotidase leads to accumulation of pyrimidine nucleotides, which has been associated with induction of basophilic strippling (IPCS, 1995).

2.10.9 Erythropoietin Production

Graziano et al. (1991) found depressed serum erythropoeitin levels in females at mid pregnancy and at delivery associated statistically with PbB level. Erythropoietin is a glycoprotein produced in the renal proximal tubules which regulates both steady - state and accelerated erythrocyte production (WHO, 1995).

2.10.10 Neurotic effects in children

The majority of the epidemiological research on the health effects of lead has been focused on children because in comparison with adults, they are more vulnerable to lead in several respects (Davis and Grant, 1992).

2.10.11 Studies of Lowlevel Lead Effects on the Central Nervous System

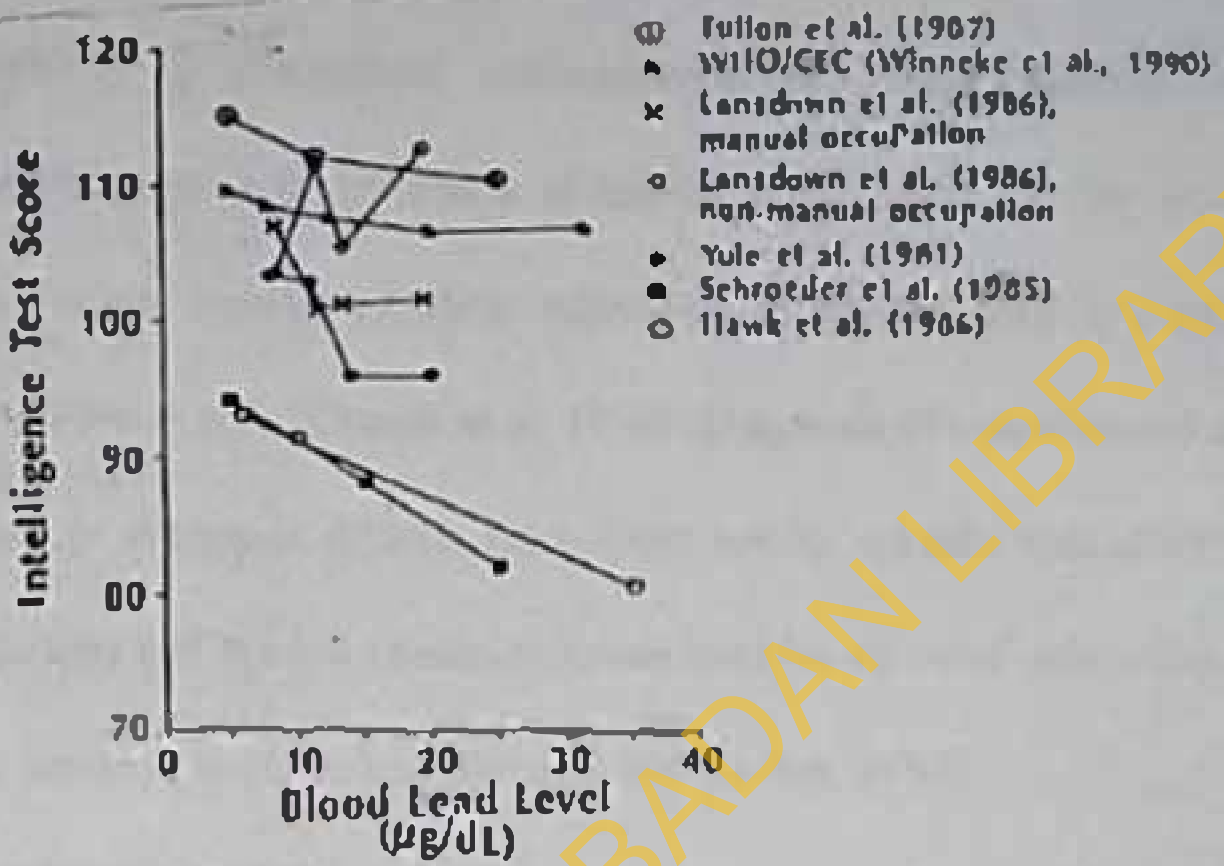
Several well designed and carefully conducted cross-sectional and retrospective cohort studies in many different countries have been conducted (Lansdown et al, 1986, Pulton et al 1987, Fergussion et al; 1988, Silver et al; 1988, Bergomi et al; 1989, Winnecke et al; 1990, Kyngbye et al; 1990, Needleman et al; 1990, Yule et al 1981, Hawk et al 1986, Schroeder et al; 1985) as shown in Fig 6 (the mean intelligence quotient (IQ) scores (in most cases adjusted for potential confounding factors) achieved by children with different blood lead levels from several of these studies some inconsistencies can be found in the results of these studies, but the weight of the evidence clearly supports the hypothesis that decrements in childrens cognition are evident at blood lead levels well below 25µg/dl. No threshold for lead IQ relationship is discernable from there data.

Most investigators report lower IQ scores among the more highly exposed children but these differences have not uniformly reached statistical signficance (that is $p < 0.05$). One way to synthesize the data from different studies is metaanalysis. Evaluation of 24 major cross-sectional studies provides IQ scores are inversely related to lead burden (Needleman and Gastonis, 1990)

Although available evidence is not sufficient to conclude that lead-associated deficits are irreversible. A follow-up study reported that the educational success of a cohort of young adults were significantly inversely associated with the amount of lead in teeth they shed as first and second graders (Needleman et al 1990). In this study, dentine lead levels above 20ppm were associated with a seven-fold risk of not graduating from high school, a six-fold risk of having a reading disability, deficits in vocabularly problems with attention and fine motor co-ordination, greater absenteeism, and lower class ranking. Although dentine lead levels did not correspond in any simple way to blood lead levels, the available pre-school blood lead levels of the more highly exposed children averaged 35μg/dl (Needleman et al, 1979). Increased circumpulpal dentine lead levels (>16ppm) have been linked to higher rates of learning disabilities in a recent Danish study as well (Lyngbye et al, 1990).

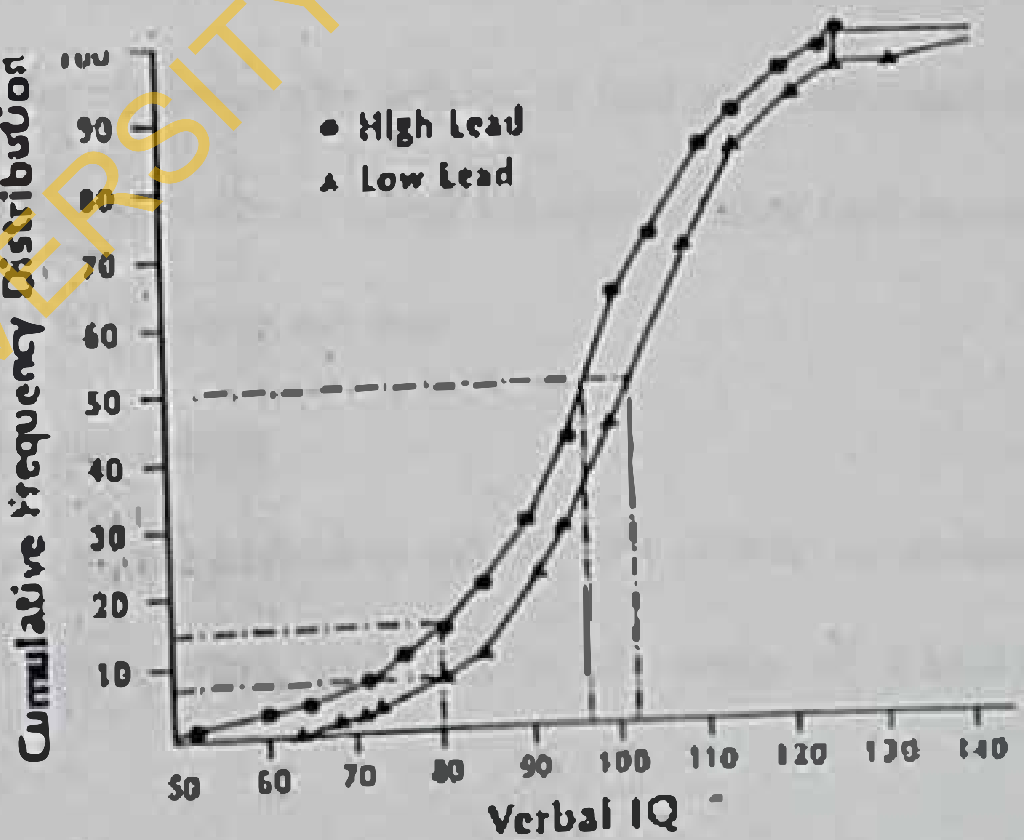
To address methodological limitations of cross- sectional studies of lead and child development, a number of prospective studies begun during the 1980s. Blood lead measurements begun during the prenatal period and continued for several years, along with assessment of development. In several but not all cohorts, prenatal exposures have been associated with slower sensory motor and delayed early congnitive development (Bellinger et al, 1987, Bellinger et al, 1991, Dietrich et al, 1991, Enhart et al, 1986). With low postnatal exposures and favourable socioeconomic conditions some of these early associations may attenuate as children grow older (Bellinger et al, 1991) In addition, several studies have noted that children's cognitive performance in the preschool period may be associated with early postnatal lead exposures (McMicheal et al, 1988, Bellingner et al, 1991). It will be necessary for theses prospective studies to follow the respective cohorts into the school - age years in order for the full implications of these early patterns to become clear.

Questions are frequently raised about the practical significance of the difference frequently observed between the IQ scores of more exposed and less exposed children from the previously described population of children studied by Needleman et al; (1979), a shift in mean IQ score of 4-6 points as a result of lead exposure was associated with a substantial increase in the prevalence of children severe deficits (that is, less than 80) (Fig 7) similarly, in this population the shift was associated with an absence of children who achieved superior function (that is, IQ scores greater than 125).



*Data from prospective studies are not included.

Figure 6 Blood Lead Levels and IQ Scores of Children, from Cross-sectional and Retrospective Cohort Studies



Source: Needleman et al., 1979.

Figure 7. Cumulative Frequency Distribution of Verbal IQ Scores in Children with High and Low Tooth Lead Levels

2.10.12 Renal System

Acute exposure to lead is known to cause proximal renal tubular damage, characterized by generalized aminoaciduria, hypophosphataemia with relative hyperphosphaturia and glycosuria (Chisolm, 1962). Cellular structural changes include nuclear inclusion bodies, mitochondria changes and cytomegaly of the tubular epithelial cells (Cramer et al; 1974). Diagnosis of lead-induced altered renal function or disease is difficult since there are no specific indicators blood level urea nitrogen (BUN) and creatinine levels become elevated only when two thirds of renal function has been lost (Bernard and Becker, 1988).

2.10.13 Cardiovascular System

There is a good evidence that signs of clinical lead poisoning sometimes include evidence of toxic action on the heart (IPCS, 1977). Kopp et al (1988) reviewed the cardiovascular actions of lead and concluded that the degree of cardiovascular involvement during episodes of acute lead intoxication depends on the duration of exposure and dose.

2.10.14 Gastrointestinal System

Colic is seen in children and US EPA (1986a) concluded that the lowest – observed adverse-effect level was in the range of 2.88-4.88μmol/litre (60-100μg/dl).

2.10.15 Liver Effects

The effect of lead on haem synthesis may alter the functional capacity of the hepatic cytochrome P-450 system to the metabolize drugs (IPCS, 1995) Saengo et al. (1984) found decreased urinary excretion of 6-β-hydroxycortisolin 26 children with a mean PbB level of 2. 11µmol/litre (44µg/dl). The decreased formation of the metabolite was attributed to lead inhibition of the cytochrome P-450-dependent mixed function oxidase.

2.10.16 Postnatal Growth and Stature Effects

Several reports have suggested that the physical growth stature of children may be reduced by exposure to lead (Mooty et al; 1975, Johnson ands Tetuna, 1979, Routh et al, 1979), but the influence of other factors (e.g., race and diet) has often made it difficult to isolate lead as a casual agent for such effects in human populations. Multivariate regression analyses of NHANES data for approximately 2700 children in the USA (Schwartz et al., 1986) provided more convincing evidence of a significant association between increasing PbB levels and reduced height, weight and chest circumference after adjusting for age, race, sex and nutritional covariates.

2.10.17 Effects On Chromosomes

Dalpha et al. (1983) examined blood samples taken from 19 children living in a contaminated area near a smelter and having PbB levels in the range of 1.39-3.02 μmol/litre (29-63μg/dl) they found on the SCE frequency.

2.10.18 Effects on The Thyroid Function

Siegel et al. (1989) tested 68 children for thyroid function and for PbB and found no statistically significant relationship between lead and total or free thyroxine.

2.10.19 Effects on Immune System.

A study of 12 pre-school children (Reigart and Grabe 1976) with PbB level > 1.92 μmol/litre (40μg/dl) did not reveal altered immunity in comparison with a control group.

2.11 Clinical Signs and Symptoms of Lead Toxicity in Children

Early signs of lead poisoning in children are highly variable and can be very difficult to discern in the young nonverbal child. Typically, children have no overt signs of toxicity until blood lead levels exceed 20-30 μg/dl. Early signs of toxicity are non-specific and may include sleeping difficulties, decreased appetite and hyperactivity (Sciarillo et al; 1992). Once blood levels exceed 50μg/dl, manifestations become more obvious e.g. marked irritability and anorexia. Seizures may occur at blood lead levels as low as 50-60µg/dl. When lead levels exceed 80-100µg/dl, frank encephalopathy with lethargy, seizures, cerebraloedema, and death occur (Shannon 1998). This can be seen as shown in Fig (8) below.

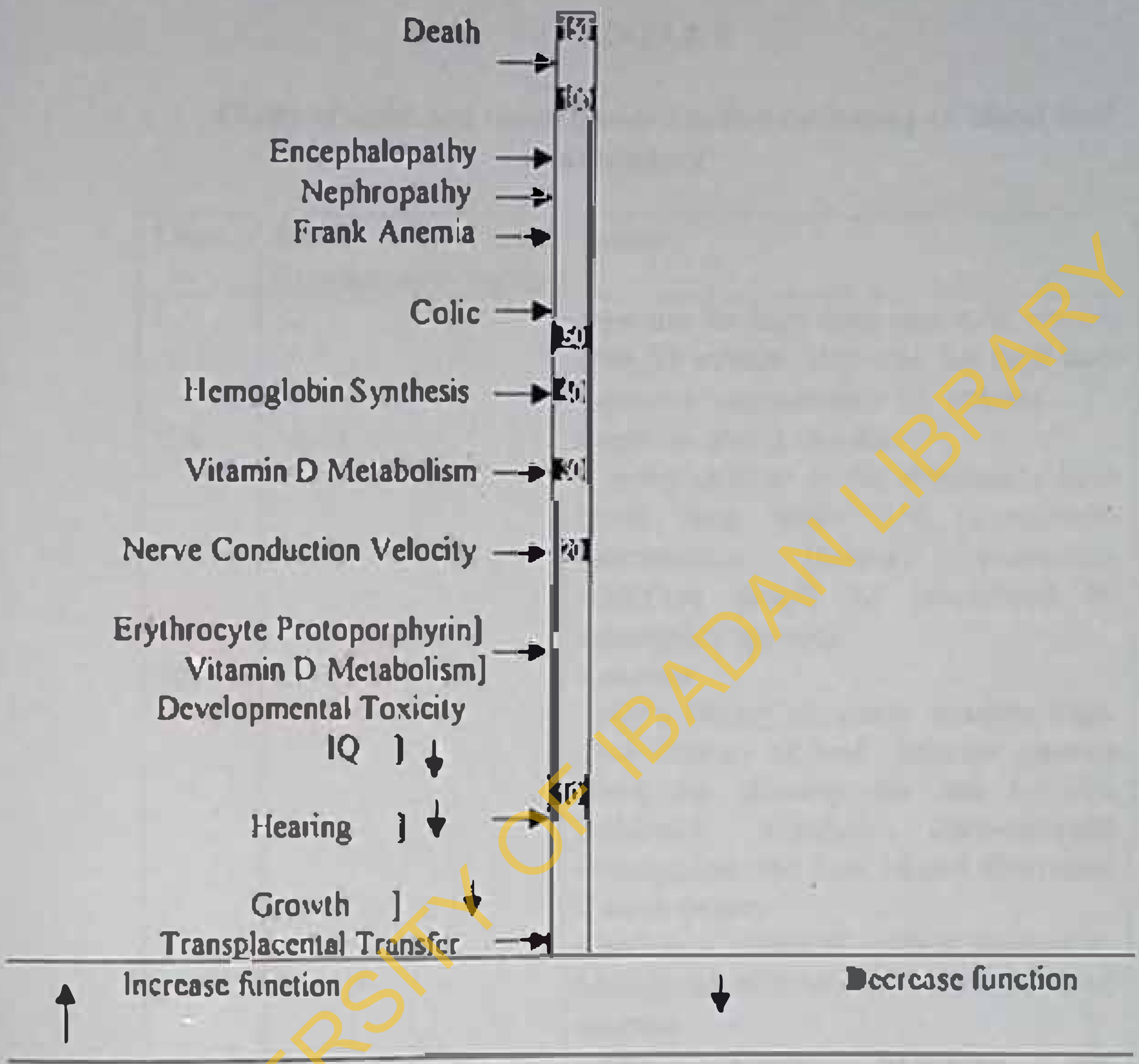
2.12 Prevention and Control of Lead Poisoning in Children

2.12.1 Screening

Screening is important both to ensure that poisoned children are identified and to generate data to target primary prevention activities. Virtualy all children should be screened for lead poisoning, screening children with a high probability of exposure to high-dose sources is the highest priority. Screening should be done using a blood lead test, are (ATSDR, 1993).

2.12.2 Classification on The Basis of Screening Test Results

On the basis of screening test results, children can be classified into categories according to their risk for adverse effects of lead. The urgency and type of follow up are based on these risk classes. These classes are shown in Table 8.



Note: The Levels in this diagram do not necessarily indicate the lowest levels at which lead exerts an effect. These are the levels at which studies have adequately demonstrated an effect.

Sources: ATSDR. 1990.

Fig. 7: Lowest Observed Effect of Levels of Inorganic Lead in Children

TABLE 5

Classs of child and recommended action according to blood lead measurement

Class	Blood Lead Concentration(µg/dl)	Action
I	≤9	Low risk for high dose exposure: rescree after 24 months. High risk for high dose exposure: rescreen after 12 months
IIA	10-14	Rescreen after 3 months. If many children in the community have blood lead levels ≥10, community interventions (primary prevention activities) should be considered by appropriate agencies.
IIB	15-19	Rescreen. Take a history to assess possible high-dose sources of lead. Educate parents about diet, cleaning, etc. test for iron deficiency. Consider environmental investigation and lead hazard abatement if levels persist.
III	20-44*	Conduct a complete medical evaluation. Identify ad eliminate environmental lead sources.
IV	45-69*	Begin medical treatment and environmental assessment and remediation within 48 hours.
V	≥70*	Begin medical treatment and environmental assessment and remediation immediately.

*Based on confirmatory bloodlead level.

Source: ASTDR 1993

2.13 Treatment of Lead Toxicity

2.13.1 Pharmacology of Chelating Agent

Several drugs are used in the treatment of lead poisoning. These drugs, capable of binding or chelating lead, deplete the soft and hard (skeletal) tissues of acute toxicity. All drugs have potential side effects and must be used with caution. The basic pharmacologic characteristics of the various drugs are described below.

2.13 Treatment of Lead Toxicity

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TABLE 6

Chelating Agents Used in Treating Children with Lead Poisoning

Product Name	Generic Name	Chemical Name	Abreviation
Calcium Disodium	Edetate	Calcium disodium	$CaNa_2EDTA$
Versenate	Sodium Calcium	Ethylenediamine tetraacetate	BAL
Bal in Oil	Dimercaprol	2,3-dimecapto-1-propanol	D-penicillamine
Cuprimine	D-penicillamine	3-mercapto-D-valine.	DMSA
Chemet	Succimer	Meso2,3-di-mecaptossuccinic acid.	

Source: ATSDR 1995.

2.14 Prevalence studies of Lead in Children in Africa

The available data on PbB and the actual prevalence of childhood lead poisoning in Africa is limited. The most detailed study of childhood lead poisoning in Africa has been done is South Afric, especially in the Cape Province. Grobler et al. (1985) found the PbB for children in a remote area of South Africa to be 3.4 µg/dl, a value that is higher than the current average for the U.S population. A study conducted by Deveaux et al, (1986) of PbB of socially deprived children, aged between 4 and 6 year of age, from pre –school center in Cape Town BbP to be 16µg/dl and that about 4% of the 300 children studies had PbB of 13µg/dl or higher. Von Schimding and Furgle, (1984) conducted a prevalence study in which involved 120 "coloured", first and second grade children in Cape Peninsula showed a two-fold difference in PbB between the urban (22µg/dl) and rural 11µg/dl) children A follow up study found that 17% of the school children in woodstock (located near the central business district of Cape Town) had PbB>10µg/dl and that about 90% of block children and 68% of white children had PbB > 10µg/dl (Von Schimding et al. 1991). The average PbB for children in schools adjacent to highways was 18-21 µg/dl compared to 13µg/dl in schools removed from heavily traveled roads. Children with high PbB generally showed signs of behavioural and biochemical abnormalities (Von Schimding, 1988). A resurvey of grades one and two pupils in woodstock was done in 1991 following the reduction in lead content of petrol from 0.84 to 0.4g/l (Von Schirnding et al, 1995a), no significant difference was found in average PbB during 1982 (16µg/dl) and 1991 (16µg/dl), and about 62% of the children still showed) PbB >15µg/dl in 1991.

Reported average PbB in various communities of the Capetown. South Africa include 14µg/dl in HoutBay, 15µg/dl in Mitchell's Plain, 16µg/dl in both woodstock and Schotcheskloof (Von Schimding et al; 1995b). Between 93 and 100% of the children in these communities have Bpb > 10µg/dl. In the mining village of Ageneys (North-west Cape), average BPb of children was found to be 16µg/dl and close to the 13µg/dl in the village of Pella located about 40km from Aggeys (Von Schirnding et al, 1995a). The high PbB levels in rural and urban communities are evidence that childhood lead poisoning associated with elvated lead levels in the environment has become pervasive throughout the province and presumably the country as well.

A recent report suggests that 100% of the children 0-2year old and 82% of those 3-5 years who live in the urban areas of Africa have PbB concentration of 10µg/dl or higher (Alliance to end Childhood Lead poisoning, 1994), these estimates are believed to be exaggerated. It is been suggested that 15-30% of the children in some urban areas of Nigeria have PbB levels over 25μg/dl (Nriagu, 1992). High lead levels (28-32μg/g average) were recently reported in the hairs of school children who lived near a major road in Kumasi, Ghana (Golow and Kwansha-Ansah, 1994).

CHAPTER THREE

MATERIALS AND METHODS

3.1 Description of Study Area

Lagos State was created on May 27, 1967 by virtue of State (creation and Transitional Provision) Decree No 14 of 1967, which restricted Nigeria's Federation into 12 States. Prior to this Lagos municipality has been administered by the Federal Government through the Federal Ministry of Lagos Affairs as the regional authority while the Lagos City Council (LCC) governed the city of Lagos. Equally, the metropolitan areas (Colony province) of Ikeja, Agege, Mushin, Ikorodu, Epe and Badagry were administered by the Western Region. The State took-off as an administrative entity on April, 1968 with Lagos Island serving the dual role of being the State and Federal Capital. However, with the creation of the Federal Capital Territory of Abuja in 1976, Lagos ceased to be the capital of the State which was moved to Ikeja.

The state is located on the South-Western part of Nigeria on the narrow coastal plain of Bight of Benin. It lies approximately on the longitude 20° 42'E and 3° 22' East respectively and between lat 60° 22'N and 60° 42'N. It is bounded in the North and East by Ogun State of Nigeria in the West by the Republic of Benin and stretches over 180 Kilometers along the Guinea Coast of the Bight of

Benin on the Atlantic Ocean. Its jurisdiction comprises the city of Lagos and the four administrative divisions (then colony province) of Ikeja, Ikorodu, Epe and Badagry. Politically, Lagos State encompasses an area of 358, 861 hectares or 3,577 sq. km (Lagos State Yearly Diary 1999).

The dominant vegetation of the State is the swamp forest consisting of the fresh water and mangrove swamp forests, both of which are influenced by the double rainfall pattern of the State, which makes the environment a wetland region. Generally, the state has two climatic seasons. Dry (November - March) and Wet (April – October). The drainage system of the State is characterized by a maze of lagoons and waterways, which constitutes about 22 percent or 787sq. Kms (75, 755 hectares) of the State total landmass. The major water bodies are the Lagos and Lekki, Lagoons, Yewa and Ogun rivers, others are Ologe Lagoon, Kuramo Waters, Badagry, Five cowries and Omu Creeks.

Although Lagos State is the smallest State in Nigeria, yet it has the highest population which is over five percent of the national estimate. According to the 1991 national census, the State has a population of 5,725, 116 million out of a national estimated of 88, 922,220. However, based on a UN study and the State Regional masterplan, the State is estimated to have about 8.5 million inhabitants out of this population Lagos metropolitan area is occupied by over 85 percent of the total state population, on an area that is 37 percent of the land area of Lagos State. The rate of population growth is about 300,000 persons per annum with a population density of bout 1,308 persons per sq. kilometer. In the built-up urban areas of metropolitan Lagos, the average density is 20,000 persons per square kilometer. In a recent UN study, the city of Lagos is expected to hit 24 million population mark and thus be among the ten most populous cities in the world by the year 2015. (Lagos State Government, 1999). Lagos State currently has 21 Local Government areas as shown Table 7 and Fig. 8.

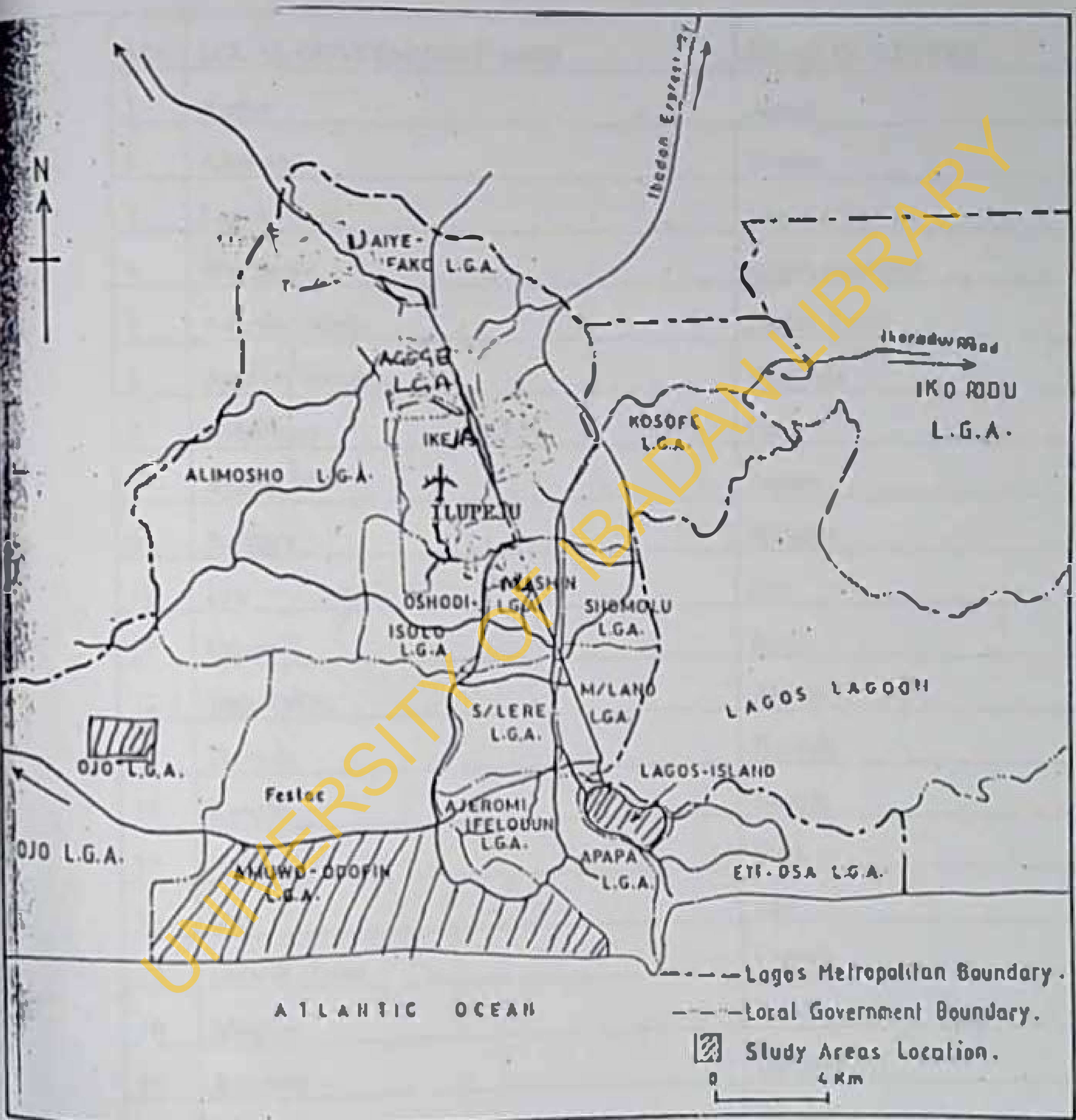

Fig:8 Map of Metropolitan Lagos Showing Local Government Areas and the Study Areas. Sources: Lagos State Survey Dept.

TABLE 7

Local Government Areas of Lagos State and Their Headquarters

S/N	LOCAL GOVERNMENT AREA	HEAD QUARTERS
1	Agege	Agege
2	Alimosho	Ikotun
3	Lagos Island	Lagos Island
4	Mainland	Lagos mainland
5	Amuwo Odofin	Festac Town
6	Ajeromi Ifelodun	Ajegunle
7	Ifako/Ijaye	Ifako
8	Apapa	Apapa
9	Badagry	Badagry
10	Epe	Epe
11	Etiosa	Ikoyi
12	Ibeju Lekki	Akodo
13	Ikorodu	Ikorodu
14	Kosofe	Kosofe
15	Ikeja	Ikeja
16	Ojo	Ojo
17	Oshodi --Isolo	Oshodi
18	Mushin	Mushin
19	Surulere	Surulere
20	Shomolu	Shomolu

Source: Lagos State Secretariat (1999).

3.1.1 Sampling Sites

According to Adedibu (1990), it is possible for ease of analysis and spatial depiction to stratify Lagos metropolis into three different strata based on their respective population density and socio-economic activities among other characteristics into high, medium and low density areas.

Table 8 shows the various communities in their respective category – the method of random sampling was employed in selecting a representative of each high, medium and low-density area.

Ojo, Mile II, and Victoria Island were selected to represent high, medium, and low-density areas.

TABLE 8

The Various Communities in their Respective Category

HIGH DENSITY	MEDIUM DENSITY	LOW DENSITY
Oshodi	Ilupeju	Ikeja GRA
Agege	Yaba	Victoria Island
Ajegunle	Surulere	Ikoyi
Mushin/Idiroko	Apapa	Obalende
Ojo	Mary land	Victoria Gardencity
Bariga	Ikeja	Lekki Peninsula
Dopemu	Festac Town	-
Ojota/ketu	Isolo	-
Badagry	Statelite Town	-
Iyana Ipaja	Yaba	-
Egbeda / Idimu	Okota	-
Idumota	-	-
Oshodi	-	-
Ojodu	-	-

3.1.2 Study Population/Sample Size

The study population comprised children aged 2-5 years who attend Nursery Schools in three randomly selected communities for the study. A list containing the names of schools in these communities was obtained from the Lagos State Ministry of Education, Alausa. There were 43 list of Approved private Nursery/Primary School in Amuwo Odofin Local Education District. Twenty approved Nursery/Primary School in Eti-Osa Local Education District and 56 Approved Nursery/Primary Schools in Ojo Local Education District shown in Appendix I.

The system of simple random technique was used to select a school in each area. Subsequently, Command Nursery/primary School was picked in Eti-Osa local education District.

Marifold Nursery/primary School was picked in Amuwo Odofin local Education District and Nigeria Army Officers Wives Association Nursery School was picked in Ojo local Education district.

All the children within the age bracket of (2-5) years were used as sample size. A 50% proportion was used in the determination of the sample size by

$$A = \frac{Z \& (P)(1-P)}{d^2}$$

Where A = Sample size

Z = Statistic (1.96)

P = Probability of success

D = degrees of freedom

Therefore, with 50% proportion P = 0.5 and at 95% confidence interval d = 0.05

$$A = \frac{(1.96)^2\ (0.5\ (0.5)}{(0.05)^2}$$

$$= 384$$

from this, a sample of 384 children was drawn from the three study areas, but the children in all the schools within the age bracket of 2-5 years were not up to 385, so all the number of children found were used which is 150. Thus a sample of 150 children was drawn from the three selected study area, viz 40 children from Command Nursery/Primary school, Victoria Island and 53 children from Marifold Nursery/Primary School in Mile II and 57 children from Nigerian Army officers Wives Association Nursery School Ojo.

3.2 Materials

Samples of hand washings, toy washings, rooftop water, classroom sweepings, playground soil and lead based eye cosmetics were collected for lead analysis.

3.2.1 Sampling Technique for Handwashings

One hundred and fifty handwashings were obtained, 57 from Nigerian Army Officers Wives Association Nursery School in Ojo, 53 from Marifold Nursery and Primary School and 40 from Command Children Nursery and Primary School. The samples were collected after the children came back from the long break period between (10.30am – 11.30am). The children washed both hands with 1 litre of double distilled water, agitating and rinsing into a plastic bowl which was first rinsed with deionized distilled water to remove traces of heavy metals as shown in Fig. 9.

All the samples were analyzed for lead and some physico-chemical characteristic like pH, total dissolves solids (T.D.S.), total suspended solids (TSS), conductivity Ms/cm and turbidity (FTU).

Figure 9 A Photograph Showing the Method of Hand Washings

3.2.2 Sampling Technique For Toyswashings

A total of twenty-six (26) toys were obtained, five from Command Nursery/Primary School and 10 from Nigerian Officers Wives Association Nursery/Primary School and 11 from Marifold Nursery/Primary School Mile II. The toys were washed with 1 litre of double distilled water into a plastic bowl which was first rinsed with deionized distilled water to remove traces of heavy metals.

All the samples were analyzed for lead and some physico-chemical characteristic like pH, total dissolves solids (TDS), and total suspended solids (TSS), conductivity ms/cm and turbidity (FTU).

3.2.3 Sampling Techniques for Roof Top Samples

A total of nine (9) roof top water samples was obtained from the schools. New and thoroughly cleaned plastic polythene bottles were placed approximated 1.7 to 2m above the ground under the roof at each sampling sites. All the samples were analyzed for lead and physico-chemical characteristics as described earlier.

3.2.4 Sampling Technique for Soils from Playground

Representative grab soil samples were obtained from the three study area. A total of 9 soil samples was collected from the school's playground, three soil samples were collected in each school playground. The use of a hand driven soil

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All the samples were analyzed for lead and some physico-chemical characteristic like pH, total dissolves solids (TDS), and total suspended solids (TSS), conductivity ms/cm and turbidity (FTU).

3.2.3 Sampling Techniques for Roof Top Samples

A total of nine (9) roof top water samples was obtained from the schools. New and thoroughly cleaned plastic polythene bottles were placed approximated 1.7 to 2m above the ground under the roof at each sampling sites. All the samples were analyzed for lead and physico-chemical characteristics as described earlier.

3.2.4 Sampling Technique for Soils from Playground

Representative grab soil samples were obtained from the three study area. A total of 9 soil samples was collected from the school's playground, three soil samples were collected in each school playground. The use of a hand driven soil auger was employed to collect the sample from depth of 0-10cm. The samples were thoroughly mixed and put into clean polythene bags for laboratory analysis. The samples were air-dried, grounded and passed through 2mm mesh sieve before they were pretreated for further analysis.

3.2.5 Sampling Technique for Classroom Sweepings

A total of 9 classroom sweepings were collected from the schools. Brooms and brushes were used appropriately for the collection. Accumulated daily sweepings was collected into polythene bags measuring 5-10 grammes. The samples were air-dried, grounded and passed through 2mm mesh sieve before they were pretreated for further analysis.

3.2.6 Sampling Techniques for Cosmetics Samples

A total of 4 samples were collected from the children through the co-operation of their teachers and parents. These cosmetics samples were pretreated for further analysis.

3.4 Methods

3.4.1 pH Measurement

The pH meter (digital type, made by Sharp England pH Mv/temp meter, Sp –701) was standardized by using phosphate buffer of pH 4 and 9.2. The temperature was regulated at room temperature the calibrated pH meter was then used to read the samples.

3.4.2 Total Dissolved Solids/Conductivity (TDS) and (ms/cm)

This was determined by using direct reading conductivity TDS meter, model 44,600.00 made by HACH England.

3.4.3 Turbidity/Total suspended Solids

This was determined by using digital DR 2000 spectrophotometer (HACH) made by Central Kagaki Corporation Japan

3.5 Further Analysis of Handwashing, Toywashing and Rooftop Water

Evaporation, solvent extraction and adsorption by ion – exchange resin are the common concentration techniques used in trace metal determination by Atomic Absorption Spectroscopy (AAS). Concentration by evaporation (Kakilu, 1985, Abua, 1996) was used here, which involves heating the sample to reduce solvent volume. Samples of 500ml volume were heated on a hot place at a room temperature of 105°C allow for evaporation until about 25ml volume was left. About 5ml concentration nitric acid was added to the sample. This was then diluted to about 50ml to achieve a ten – fold concentration and then subsequently aspirated into the AAS flame.

3.5.1 Pre-treatment and Preparation of Soil Samples

Sieved soil samples of 0.5g was accurately weighted using mettler balance and 20ml of a mixture of concentrated HNO_3 and concentrated HCl (ration 3:1) were added and boiled in a 200ml volumetric flask with a watch glass cover for about 5 –10 minutes until the colour changes as a result of evaporation of nitric acid brown fumes. The samples were cooled and passed through whatman No – 42 filter paper. The filtrate was then diluted to 100ml in a graduated volumetric flask with deionized-distilled water making sure the filter was properly washed. The sample was now aspirated into the standardized flame (AAS). (Dorn et al, 1975, APHA 1998).

3.5.2 Pretreatment of Dust from Classroom Sweepings

The pretreatment method used for dust samples were the same as discribed earlier for the soil samples.

3.5.3 Pre-treatment of Lead Based Cosmetic Sample

Cosmetic samples of 0.5g was accurately weighted via mettler balance and 20ml of a mixture of concentration HNO_3 and concentrated HCL (ratio 3:1) were added and refluxed in a 200ml volumetric flask with a watch glass cover for about 5-10 minutes until the colour changes as a result of evaporation of nitric acid brown fumes. The samples were cooled and passed through a what man No – 42- filter paper. The filtrate was diluted to 100ml in a graduated volumetric flask with demonized distilled water making sure the filter is properly washed. The sample was now aspirated into a standardized flame (AAS Dom et al, APHA 1998).

3.6 Measurement of Lead Using Atomic Absorption Spectrophotometer (AAS)

3.6.1 Principle

This is based on flame absorption rather than flame emission. Metal atoms absorb strongly and at discrete characteristics wavelengths which coincides with the emission spectra of the metal in question. A solution of the sample is converted into an aerosol which is injected into a flame which then convert the sample into atomic and molecular vapour, the atomic vapour absorbs radiation from a hollow cathode lamp at specific wavelengths, this beam then transverse the flame and is focussed on the entrance slits of a monochromator, which is set to read the intensity of the chosen spectra line. Light with this wavelength is absorbed by the metal in the flame and the degree of the absorption is a finction of the concentration of the metal in the sample.

Atomic Absorption spectrophotometer was used for the determination of the concentration of lead from specimen pre-treated and made ready for analysis. The AAS employed was the UNICAM 929 AAS.

Before aspirating the sample of interest the lamp (hollow cathode lamp) was inserted into the turret slot. The machine was put on and the current for the lamp set under use Table 9. The lamp was allowed to warm up for 10 minutes.

The wavelength to the desired line was set and the selector scanned until the required resonance line was obtained (the largest deflection towards zero on the energy meter). The aspiration rate of the nebulizer was checked to ensure that it between 3-7ml/min. The acetylene needle valve was opened nnd immediately the ignition button pressed and released after the flame must have been established as the burner head. The acetylene flow for the element under test was set. their the machine was zeroed and determination process commenced. (Gain setting can be adjusted). The absorbance of the corresponding metal standard Table 9 was read and noted before aspirating the sample, absorbance for the sample was accordingly noted. Fig. 10 shows the calibration curve for lead.

TABLE 9

CONCENTRATIONS AND WAVE LENGTH USED FOR AAS ANALYSIS

Elements	Low Standard (PPM)	Standard High PPM	Wave length of Analysis
Pb	5.0	10.0	216.8
Cu	2.0	4.0	824.8
Ni	2.5	5.0	232.1
Mn	1.0	2.0	279.9
Ag	1.0	2.0	328.9
Fe	2.5	5.0	328.3
Zn	1.0	2.0	213.8

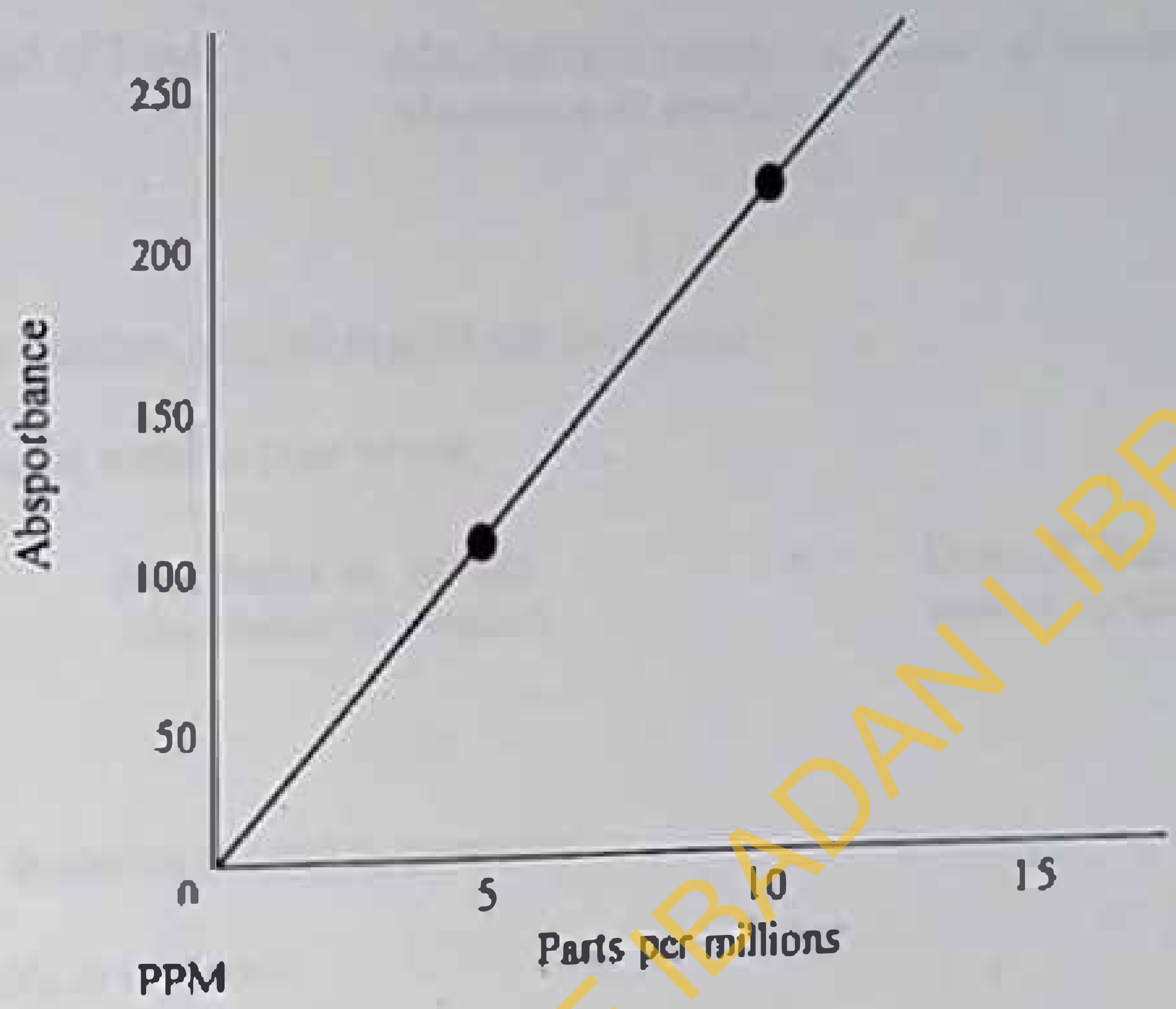

FIG. 10: CALIBRATION CURVE FOR LEAD

3.6.2 Calculation Of Lead In Handwashings, Toy Washings And Rooftop Water

$$\text{mg/l of Lead.} = \frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{Conc. of Standard} \times \text{Factor } 10$$

3.6.3 Calculation of Lead in soil/Dust Sweepings

mg/kg metal in Dust or soil.

$$= \frac{\text{Absorbance of Sample}}{\text{Absorbance of standard}} \times \frac{\text{Conc. of Standard} \times 100}{\text{mass of sample}}$$

3.6.4 Calculation of Lead in Cosmetics

µg/g in cosmestic.

$$= \frac{\text{Absorbance of Sample}}{\text{Absorbance of standard}} \times \frac{\text{Conc. of Standard} \times 1000}{\text{Mass of sample}}$$

$$\text{\% of lead in cosmestic} = \frac{\text{Mass of lead in cosmestic} \times 100\%}{\text{Original weighted sample}}$$

3.7 Indepth Interview Guide

Indepth interview is a qualitative research technique consisting of intensive individual interviews with a small number of respondents to explore what people think and say, do, about a health topic. (Graef, Elder and Booth, 1993).

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An indepth interview guide was developed by the researchers and used for interviewing the teachers about lead and children. As shown in Appendix 2. It consisted of children exposure to lead poisoning and behavioural pattern.

3.7.1 Data Analysis

The in depth interview was collated and edited manually.

3.8 Limitations of the Study

This study was restricted to Lagos Metropolis. Biological and air samples were beyond the scope of this study due to non-availability of facilities. Ideally, the handwashings could have been with very dilute nitric acid. But due to sensitivity of young children hands only distilled water was used.

Figure 11 A Photograph Showing Traditional and Home Made Lead Based Cosmetics (The first 3 are standard brands and the 4th is Traditional with 2 Containers Used to Keep the Material)

Figure 12 A Photograph Showing the Various Types of Toys Used by the Children

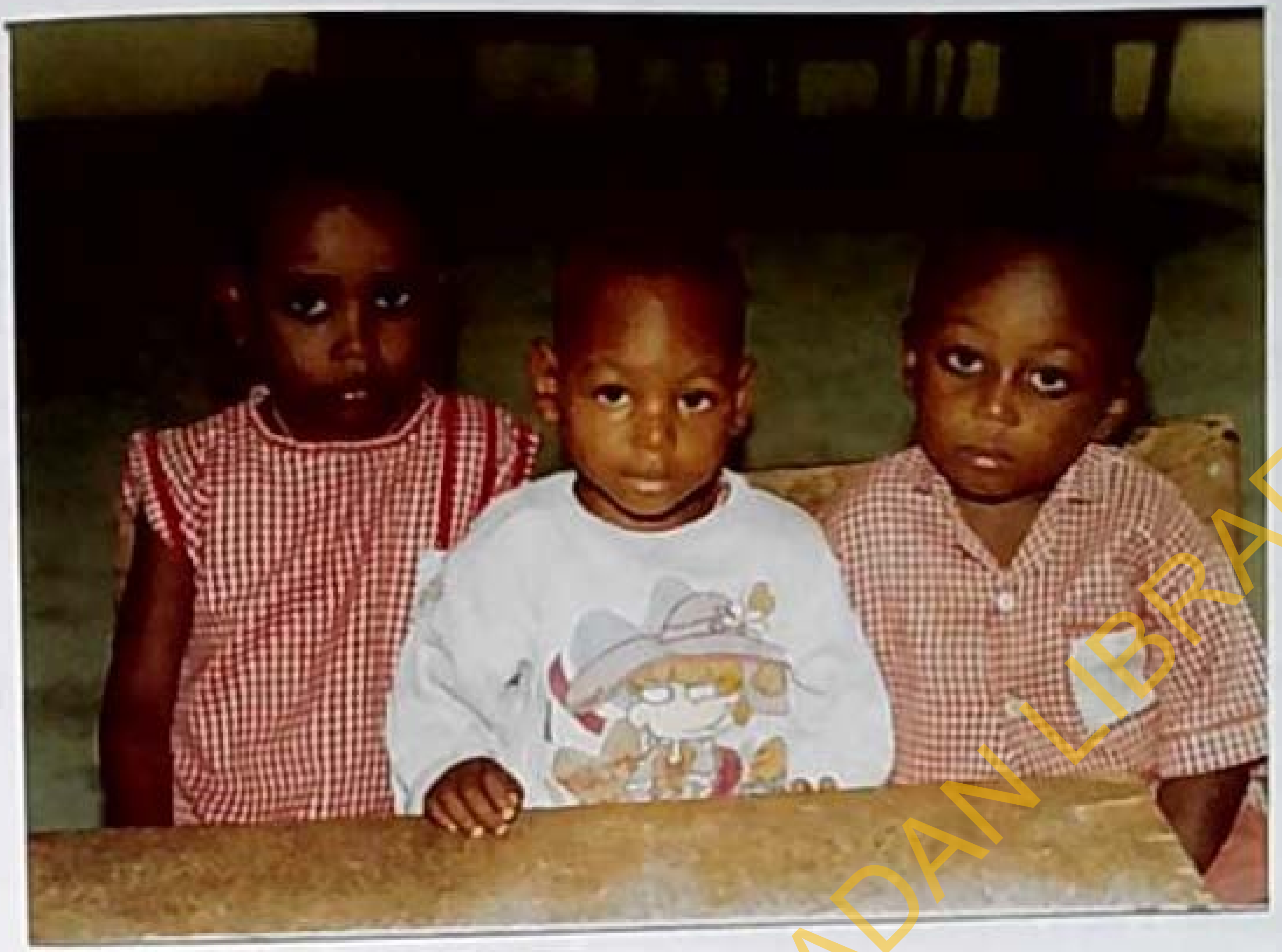
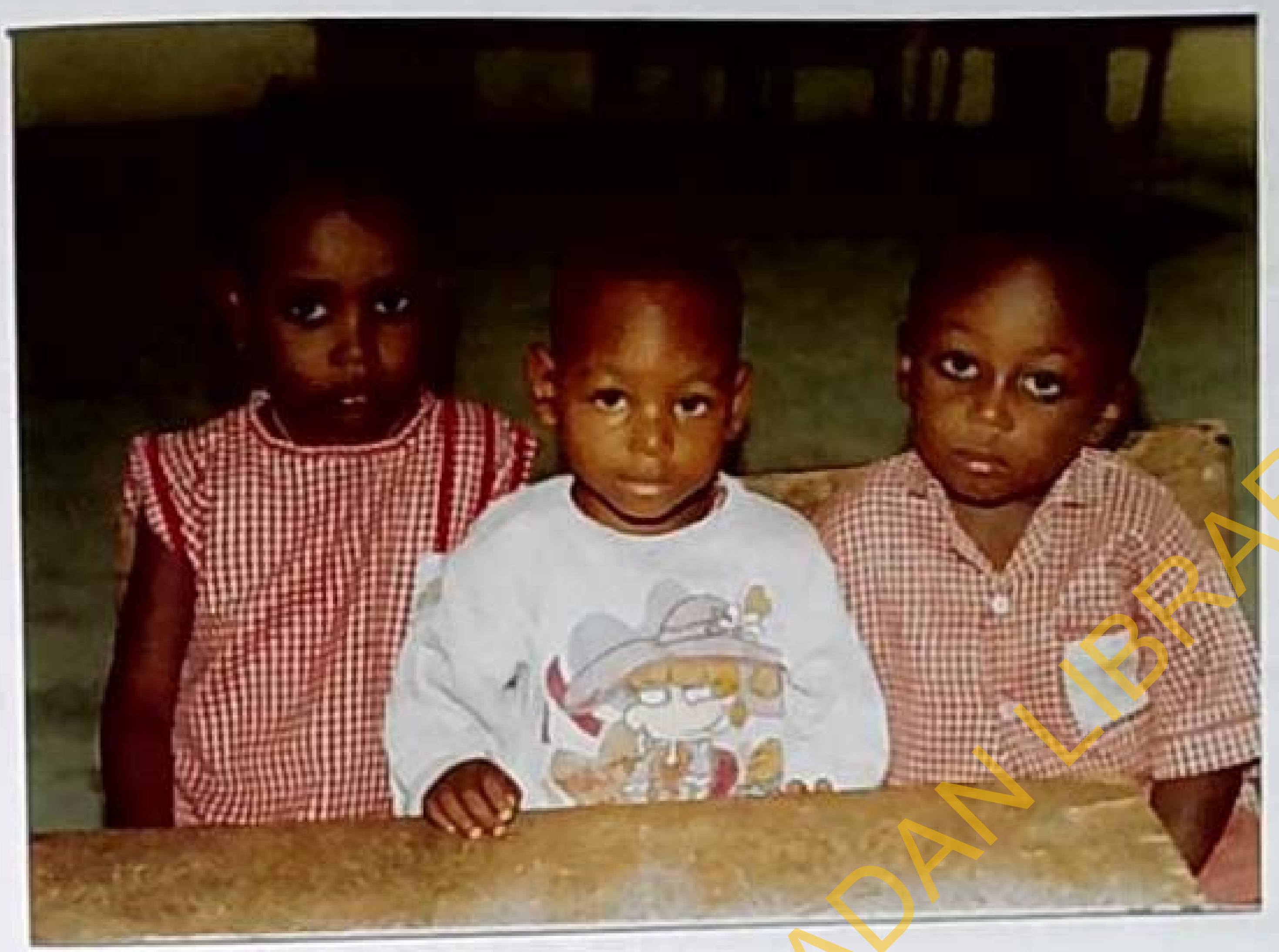


Figure 12 A Photograph Showing Children With Lead Based Eye Cosmetics

Figure 13 A Photograph Showing the Researcher (Extreme Right) with School Teachers and the Children Under Study



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CHAPTER FOUR

RESULTS

The data presented here represents analysis of 207 environmental samples comprising of 150 hand washings, 26 toy washings, 9 soil samples,9 classroom sweepings. 9-rooftop water and 4 lead based cosmetic samples.

4.1 Lead, And Other Physical Chemical Characteristics of Handwasbings

4.1.1 Samples from Low Density Area

Forty handwashings were obtained and analyzed from Command Children and Nursery/Primary School, Victoria Island, 21(52.5%) of them were boys while 19(47.5%) were girls as shown in Table 10. Majority of the children fell into the age range of 3.1 - 4.0 years as shown in Table 13. The boys' hands were more dirty than the hands of the girls hence the high value of TDS, turbidity, as shown in Fig. 15. The mean lead levels of the boys were 0.232 ± 0.106mg/hand while that of the girls was 0.126 ± 0.089mg/hand. A positive statistically linear correlation was observed between lead in handwashing and conductivity value ($r= 0.40$, $p = 0.01$) lead and total dissolved solids ($r= 0.47$, $p= 0.0025$) as shown in Table 14.

The high lead levels in the hands of the boys in this area could be attributed to the fact that they play more in the out door (school playground).

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The high lead levels in the hands of the boys in this area could be attributed to the fact that they play more in the out door (school playground).

The general high lead levels in the hands of the children from the low density area could also be due to the fact that it is the oldest School among the three selected schools, with flaking paints, pervasive dusts, both inside and outside the classrooms and is close to a very busy traffic road in the heart of Lagos. There was a negative correlation between lead and pH value while a marginal correlation was found between lead and turbidity value. (r=0.2, p = 0.4) as shown in Table 14.

4.1.2 Samples from Medium Density Area

A total of 53 handwashings were collected and analyzed from Marifold Nursery and Primary School, Mile II. Twenty nine (54.7%) were boys while 24 (45.3%) were girls as shown in Table 11 majority of the children were in the age group of 2.0 – 3.0 as shown in Table 13. This is due to the fact that it was the only school where kindergarten section was run and hence a large number of children whose parents were working drop their children there.

All the samples analyzed were slightly acidic with a mean pH of 6.545 ± 0.218 for boys and 6.566 ± 0.238 for girls as shown in Table 11. The lead levels in the hand washings of boys were slightly higher than that of the girls as shown in Fig. 14, 0.044 ± 0.037mg/hand as against 0.036 ± 0.027mg/hand. The reason for this, could be said to be that, the school is situated in the residential area, far from high traffic flow, battery chargers shop, refuse dumps and lack of flaking paints.

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The children are also from affluent families hence the school had caregivers (nannies) who insisted the children washed their hands. Other physical parameters were also significantly higher in boys than girls. The highest lead level was recorded among the mean age group 2.0 - 3.1, which is 0.046 ± 0.033mg/hand as shown in Table 13. This could be due to the pica habits in this age group, the children rub their hands on the walls, desk, colour illustrations on books, chew pencils, crayons etc. There was also a significant positive correlation between lead and total suspended solids (r = 0.43 p = 0.0012). Others were not significant as shown in Table 15.

4.1.3 Samples from High Density Area

A total of 57 hand washings was obtained and analyzed from Nigeria Army Officers Wives Nursery/Primary School, Ojo.Twenty seven (47.7%) of the children were boys while 30 (52.6%) were girls, as shown in Table 12. Majority of the children fell into the age range of 3.1 – 4.0 years as shown in Table 13.

All the samples were slightly acidic in both boys and girls as shown in Table 10. The lead levels of the boys and girls were 0.093 ± 0.076mg/hand and 0.070 ± 0.070mg/hand, respectively.

This value could be due to the fact that the school is situated away from a busy traffic road. There was a positive significant correlation between lead and turbidity (r=0.6, p=0.0) lead and TSS (r = 0.59, p =0.0) lead and conductivity (r = 0.51 p= 0.0), and lead and TDS (r = 0.54, p=0.0), as shown in Table 16.

TABLE 10

Physical And Chemical Characteristics of Hand Washings Among Boys And Girls in the Low Density Area

Parameter	Boys	Girls
pH	6.488 ± 0.260	5.50 ± 0.276
Turbidity (FTU)	53.95 ± 33.33	46.10 ± 34.56
TSS (mg/hand)	22.57 ± 19.38	18.63 ± 16.64
TDS (mg/hand)	310.00 ± 164.62	178.42 ± 121.85
Conductivity (ms/cm)	0.617 ± 0.140	0.404 ± 0.404
Pb (mg/hand)	0.232 ± 0.106	0.126 ± 0.080

Name of School: Command Nursery/Primary School
Number of Boys: 21
Number of Girls: 19
Average age of Boys: 3.92 ± 0.61 (52.5%)
Average age of Girls: 3.62 ± 0.54 (47.5%)

TABLE 11

Physical and Chemical Characteristic of Handwashing of Boys and Girls in the Medium Density Area

Parameter	Boys	Girls
pH	6.545 ± 0.218	6.566 ± 0.238
Turbidity (FTU)	10.41 ± 4.59	10.95 ± 4.45
TSS (mg/hand)	5.043 ± 3.212	3.345 ± 2.581
TDS (mg/hand)	50.00 ± 22.83	45.83 ± 16.01
Conductivity (ms/cm)	0.103 ± 0.044	0.089 ± 0.046
Pb (mg/hand)	0.044 ± 0.037	0.036 ± 0.027

Name of School: Marifold Nursery/Primary School
Number of Boys: 29
Number of Girls: 24
Average age of Boys: 3.37 ± 1.78 (54.7%)
Average age of Girls: 3.7 ± 1.04 (45.3%)

TABLE 12

Physical and Chemical Characteristic of Handwashing of Boys and Girls in the High Density Area

Parameter	Boys	Girls
pH	6.399 ± 0.289	6.382 ± 0.286
Turbidity (FTU)	35.26 ± 19.16	32.03 ± 18.72
TSS (mg/hand)	20.25 ± 12.32	18.20 ± 10.27
TDS (mg/hand)	147.40 ± 107.83	133.46 ± 113.23
Conductivity (ms/cm)	0.303 ± 0.228	0.294 ± 0.232
Pb (mg/hand)	0.093 ± 0.076	0.075 ± 0.070

Name of School: Nigerian Army Officers Wives Association Primary/Nursery School.
Number of Boys: 27
Number of Girls: 30
Average age of Boys: 3.66 ± 0.79 (47.4%)
Average age of Girls: 3.76 ± 0.80 (52.6%)

TABLE 13

Physical and Chemical Parameters of Handwashings in Relation to Age

STUDY AREA	AGE	N	pH	Turbidity (FTU)	TSS (mg/hand)	TDS (mg/hand)	Conductivity (ms/cm)	Pb (mg/hand)
LOW DENSITY	2-3	8	6.365 ± 0.254	58.750± 41.712	23.000 ± 22.501	58.750 ± 41.712	0.460 ± 0.032	0.171 ± 0.098
	3-4	25	6.491 ± 0.281	44.000± 27.701	17.200 ± 12.626	44.000 ± 27.701	0.492± 0.317	0.172 ± 0.115
	4-5	7	6.529 ± 0.239	62.710± 43.030	30.571 ± 26.645	62.710± 43.030	0.664 ± 0.494	0.228 ± 0.114
MEAN ± S.D		40	6.461 ± 0.285	55.153 ± 37.481	23.590 ± 20.590	55.153 ± 37.481	0.539 ± 0.106	0.190 ± 0.109
MEDIUM DENSITY	2-3	27	6.478± 0.220	9.815± 3.585	4.577 ± 2.248	65.926 ± 110.009	0.096 ± 0.052	0.046 ± 0.033
	3-4	12	6.664± 1.173	10.833± 3.810	4.917± 4.231	147.500 ± 20.994	0.096 ± 0.039	0.034 ± 0.034
	4-5	14	6.571± 0.252	12.143± 6.225	4.571 ± 2.709	48.571 ± 17.003	0.099 ± 0.038	0.035 ± 0.032
MEAN ± S.D		53	6.571 ± 0.548	10.930 ± 4.540	4.488 ± 3.062	65.357 ± 49.335	0.097 ± 0.043	0.038 ± 0.003
HIGH DENSITY	2-3	22	6.393± 0.314	27.407± 17.508	16.273± 10.872	26.364 ± 107.860	0.284 ± 0.237	0.073 ± 0.066
	3-4	24	6.378± 0.267	39.125± 21.727	21.958 ± 12.651	157.667± 117.363	0.320 ± 0.243	0.106 ± 0.087
	4-5	11	6.412± 0.286	33.727± 11.507	18.909 ± 7.327	129.091 ± 101.436	0.282 ± 0.188	0.064 ± 0.037
MEAN ± S.D		57	6.394 ± 0.289	33.419 ± 16.914	19.046 ± 10.283	104.374 ± 108.886	0.295 ± 0.119	0.081 ± 0.063
Mean of All		150	6.475 ± 0.365	33.167 ± 19.645	15.768 ± 11.311	74.961 ± 65.234	0.310 ± 0.089	0.103 ± 0.068

LOW DENSITY: Command Nursery/Primary School, Victoria Island
MEDIUM DENSITY: Marifold Nursery/Primary School, Mile II
HIGH DENSITY: Nigerian Army Officers Wives Nursery/Primary School, Ojo.

TABLE 14

Simple Correlation Analysis of Children in Low Density Area

	V1	V2	V3	V4	V5
V2	r-0.4042 p.0. 0097	-	-	-	-
V3	r-0.3452 p.0. 0292	0.9040 0. 0.0000	-	-	-
V4	r – 0.1540 p.0 3428	0.7785 0.0000	0.7719 0.0000	-	-
V5	r-0.1742 .0.2824	0.6902 0.0000	0.6431 0.0000	0.9180 0.0000	-
V6	r-0.0921 p 0.5718	0.2004 0.215	0.1836 0.2567	0.4013 0.0103	0.4658 0.0025

Var.: Variable Label
V1: pH
V4: Conductivity
V2: Turbidity
V5: TDS
V3: TSS
V6: Pb

TABLE 15

Simple Correlation Analysis of Children in Medium Density Area

	V1	V2	V3	V4	V5
V2	r- 0.0615 P 0.6619	-	-	-	-
V3	r- 0.2141 p 0.1236	0.6666 0.0000	-	-	-
V4	r- 0.0930 0.5079	0.0728 0.6047	- 0.2929 0.0333	-	-
V5	r. 0.0738 p. 0.5992	0.0021 0.9880	- 0.1594 0.2544	0.3248 0.0176	-
V6	r-0.4171 0.0019	- 0.0701 0.6182	0.4319 0.0012	0.4287 0.0014	0.1997 0.1518

Var.:	Variable Label				
V1:	pH	V2:	Turbidity	V3:	TSS
V4:	Conductivity	V5:	TDS	V6:	Pb

TABLE 16

Simple Correlation Analysis of Children in High Density Area

	V1	V2	V3	V4	V5
V2	r- 0.4640 p 0.0003	-	-	-	-
V3	r-0.4568 p 0.0004	0.9613 0.0000	-	-	-
V4	r-03773 p 0.0038	0.5012 0.0001	0.4378 0.0007	-	-
V5	r-0.3464 p 0.0083	0.5477 0.0000	0.4685 0.0002	0.9395 0.0000	-
V6	r-0.5050 p 0.0001	0.6251 0.0000	0.5920 0.0000	0.5121 0.0000	0.5449 0.0000

Variables in Analysis

Var.:	Variable Label				
V1:	pH	V2:	Turbidity	V3:	TSS
V4:	Conductivity	V5:	TDS	V6:	Pb

TABLE 17

Simple Correlation Analysis in the Three Study Area

	V4	V5	V6	V7	V8
V5	r-0.3684 p 0.0000	-	-	-	-
V6	r-0.3987 0.0000	0.9208 0.0000	-	-	-
V7	r-0.2449 p 0.0025	0.7848 0.0000	0.7118 0.000	-	-
V8	r-0.2158 p 0.0080	0.7079 0.0000	0.6126 0.0000	0.8997 0.0000	-
V9	r-0.2754 p 0.0006	0.5500 0.0000	0.4724 0.0000	0.6086 0.0000	0.6013 0.0000

Variables in Analysis

Var.:	Variable Label				
V4:	pH	V5:	Turbidity	V6:	TSS
V7:	Conductivity	V8:	TDS	V9:	Pb

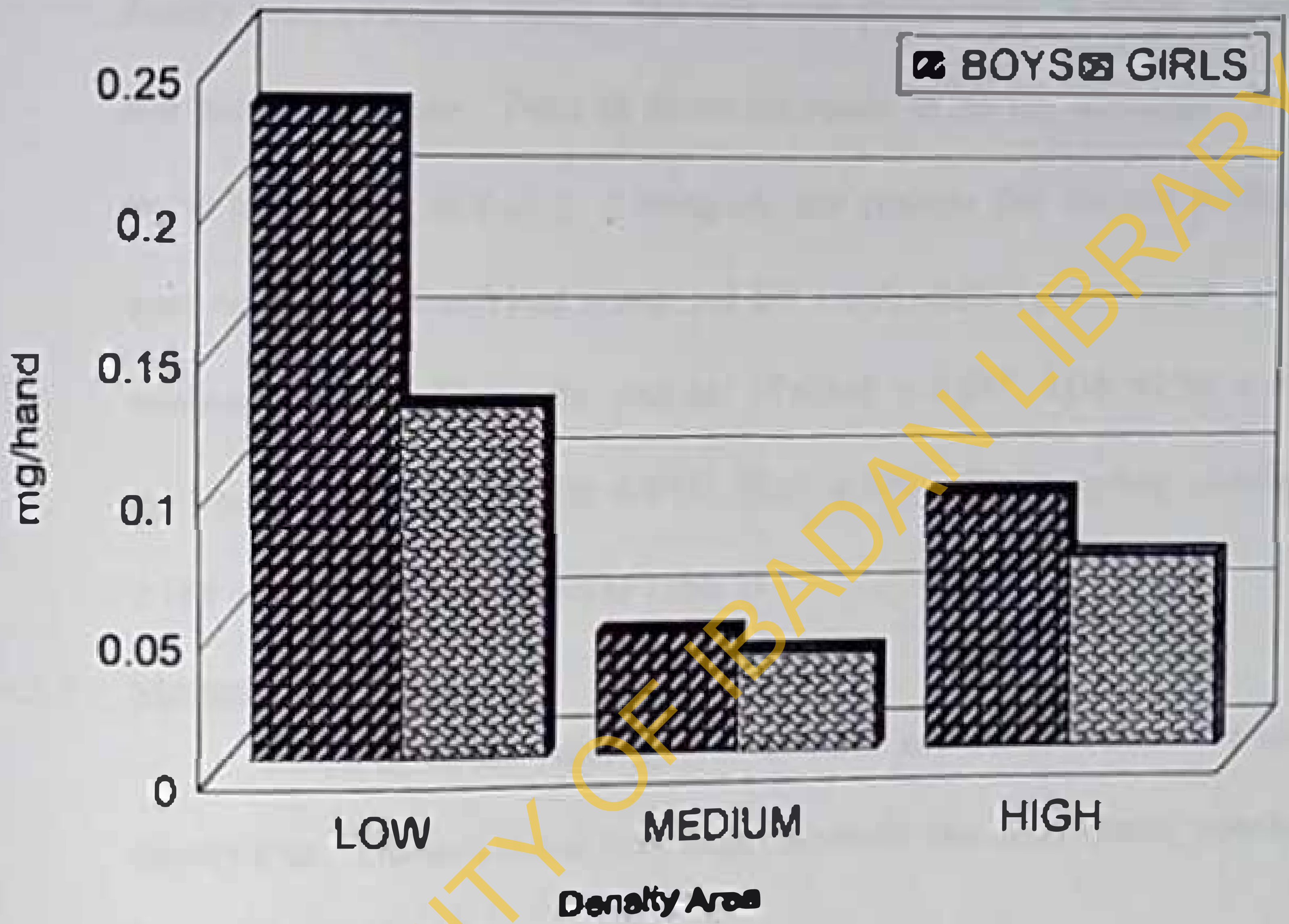

FIG. 14: A Chart Showing Lead Levels Among Boys and Girls in the Study Areas

4.2 Lead Levels and Other Physical Chemical Characteristics of Toywashings

4.2.1 Low Density Area

Five toy washing were collected and analyzed from the School in low density area. (Victoria Island). The toys were plastic birds, footballs, telephones and numeric counters. Table 18 shows the results of the toy washings. The lead levels had a mean of 0.07 ± 0.03mg/toy, this predicts that the toy surface area were contaminated with lead in dust and dirt's in the child's environment. The washings were slightly acidic with pH of 6.662 ± 0.297, TDS 92.00 ± 82.885 mg/toy, Turbidity: 19.40 ± 28.92FTU. TDS: 6.200 ± 9.960 mg/toy; conductivity 0.188 ± 0.169ms/cm as shown in Table 18.

4.2.2 Medium Density Area

A total of 10 toy washings was collected and analyzed from the medium density area. The toys in use were empty cosmetic tins, body cream, powder and beverage container as shown in Fig. 12.

The lead level had a mean value of 0.007 ± 0.003mg/toy. The values were lower than that of the low-density area, this could be attributed to the fact that the School is far away from a major traffic road and the toys were cleaned very morning.

The toy washings were slightly acidic with pH: 6.628 ± 0.273, TDS: 45 ± 16.49mg/toy, turbidity: 9.91 ± 2.11 mg/toy, TSS: 1.50 ± 0.44mg/toy and conductivity 0.09 ± 0.03 ms/cm as shown in Table 19.

4.2.3 High Density Area

In this category 11 toy washings were collected from the School in High density Area (Ojo). The toys comprises of cars building blocks, footballs, bicycles.

Table 20 shows the mean lead level as 0.05 ± 0.43 mg/toy. This value was higher than the value from the medium density area, but lower than the high-density area. This could be due to caretaker's supervision of the children and hygienic condition of the toys.

The toys washing were slightly acidic with pH: 6.365± 25, TDS: 72.00 ± 60.60 38mg/toy; Turbidity: 24.50 ± 19.59mg/toy, TSS: 13.54 ± 11.91mg/toy and conductivity 0.15 ± 0.14 ms/cm as shown in Table 20.

Generally, the toys in all the three study area were slightly acidic with a mean pH: of 6.54 ± 0.34 and of lead value of 0.150 ± 0.63mg/toy. There was a significant correlation between lead and other physical chemical parameters as shown in Table 22.

TABLE 18

Lead Levels and other Physical Chemical Characteristic of Toywashing in Low Density Area

Parameter	Mean ± S.D
pH	6.66 ± 0.29
Turbidity (FTU)	19.40 ± 18.92
TSS (mg/toy)	6.2 ± 0.9
TDS (mg/toy)	92.00 ± 82.88
Conductivity (ms/cm)	0.18 ± 0.16
Pb (mg/toy)	0.07 ± 0.03

TABLE 19

Lead Levels and Other Physical Chemical Parameters of Toywashings from Medium Density Area

Parameter	Mean ± S.D
Ph	6.628 ± 0.273
Turbidity (FTU)	5.90 ± 2.119
TSS (mg/toy)	1.750 ± 0.463
TDS (mg/toy)	45.00 ± 16.49
Conductivity (ms/cm)	0.014 ± 0.033
Pb (mg/toy)	0.009 ± 0.003

TABLE 19

Lead Levels and Other Physical Chemical Parameters of Toywashings from Medium Density Area

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TSS (mg/toy)	1.750 ± 0.463
TDS (mg/toy)	45.00 ± 16.49
Conductivity (ms/cm)	0.014 ± 0.033
Pb (mg/toy)	0.009 ± 0.003

TABLE 20

Lead Levels and Other Physical Chemical Parameters of Toywashing from High Density Area

Parameter	Mean ± S.D
Turbidity (FTU)	24.50 ± 19.59
TSS (mg/toy)	13.54 ± 11.91
TDS (mg/toy)	72.600 ± 60.39
Conductivity (ms/cm)	0.156 ± 0.147
Pb (mg/toy)	0.056 ± 0.04

TABLE 21

Lead and Other Physical and Chemical Analysis

of Toyswashings in The Three Density Areas

Parameter	Mean	SD	p value	t statistics
pH	6.540	0.316	0.0000	107.405
Turbidity (FTU)	15.220	18.477	0.0004	4.281
TSS (mg/toy)	7.220	10.086	0.001	3.721
Conductivity (mg/toy)	0.136	0.121	0.000	5.823
TDS (mg/toy)	64.259	54.007	0.000	6.183
Pb (mg/toy)	0.150	0.0631	-	-

TABLE 22

Simple Correlation Analysis between Lead and Other Physical and Chemical Parameters of Toywashings

Variable	r	pvalue
TDS	0.63	0.077
pH	0.31	0.45
Turbidity	0.71	0.45
TSS	0.53	0.079
Conductivity	0.56	0.011

4.7 Lead Levels in Classroom Sweepings

Nine classroom sweeping were obtained and analyzed for lead. The lead levels ranged from 180-615mg/kg. Three (33%) of the samples were below the WHO recommended background level of 200mg/kg while 6(66%) were above the recommended value as shown in Table 23. The highest value of classroom lead sweepings was obtained from Victoria Island as shown in Fig 16. This could be due to the fact that the school is the oldest among the three selected schools, both the exterior and interior wall paints in the school were flaking (oil paint in origin), the school is situated near a major express way with high traffic density and deposition of lead dust in the atmosphere. Lead content of gasoline in Nigeria is 0.7g/l which create high vehicle emission, while the medium and high density area schools are situated away from a high traffic density and is painted with emulsion paints.

4.8 Lead Levels in School Playground

Nine soil samples were obtained and analyzed for lead. The lead level ranged from 951-3602 mg/kg as show in Table 24. The highest level of playground lead was obtained from the low-density area school of 3602mg/kg as shown in Fig 17. This high value could be due to the high intensity of traffic close to the school, nearest of the playground to the flaking wall paints and deposition of lead aerosols from the atmosphere. This finding is in conformity with Mielke et al (1997). They found out that communities with oldest houses had high median soil lead. All the values obtained were higher than the WHO recommended value of 500mg/kg (out door lead)

TABLE 23

Lead Levels in the Classroom Sweepings in the Three-Study Area

Code	Sampling Site	Source	Nature and Description of Sample	Type Of Building	General Ventilation	Nature Of Paint	Proximity To Heavy Traffic	Age of Building	Pb Levels mg/kg
Low Density 1	Victoria Island	Class room	Dusty Wall Peeling sandy	Bungalow	Fair	Old	Close	30years	600
Low Density 2	Victoria Island	Class room	Dusty, sandy pencil chips crayon	Bungalow	Fair	Old	Close	30 years	627
Low Density 3	Victoria Island	Class room	Dusty, sandy pencil chips crayon	Bungalow	Fair	Old	Close	30 years	600
Mean ± SD									606.75 ± 13.5
Medium Density 1	Mile II	Class room	Dusty, sandy pencils food particles	Bungalow	Good	New	Far	6 years	160
Medium Density 2	Mile II	Class room	Sandy, food particles, chalk paper	Bungalow	Good	New	Far	6 years	190
Medium Density 3	Mile II	Class room	Dusty sandy, paper, pencil chips	Bungalow	Good	New	Far	6 years	184
Mean ± SD									178 ± 15.87
High Density 1	Ojo	Class room	Sandy, Dusty, paper, sticks chalk	Bungalow	Good	Fairly old	Medium	10 years	382
High Density 2	Ojo	Class room	Sandy, dusty food particles	Bungalow	Good	Fairly old	Medium	10 years	330
High Density 3	Ojo	Class room	Sandy dusty food particles	Bungalow	Good	Fairly old	Medium	10 years	390
Mean ± SD									367.33±32.57
Mean ± SD all									384.02±20.65

Class Ventilation: Good (4 or more windows) which can be opened (cross ventilation) Fair (only 2 windows opposite each other).
Poor (less than 2 windows which can be opened) no cross ventilation.

Proximity to traffic: Close (50-100 metres). Medium (100-200 metres) Far (≥ 200 metres)

LOW DENSITY: Low Density School Sweepings sample MEDIUM DENSITY = Medium Density School Sweepings sample

HIGH DENSITY: High density School Sweepings sample

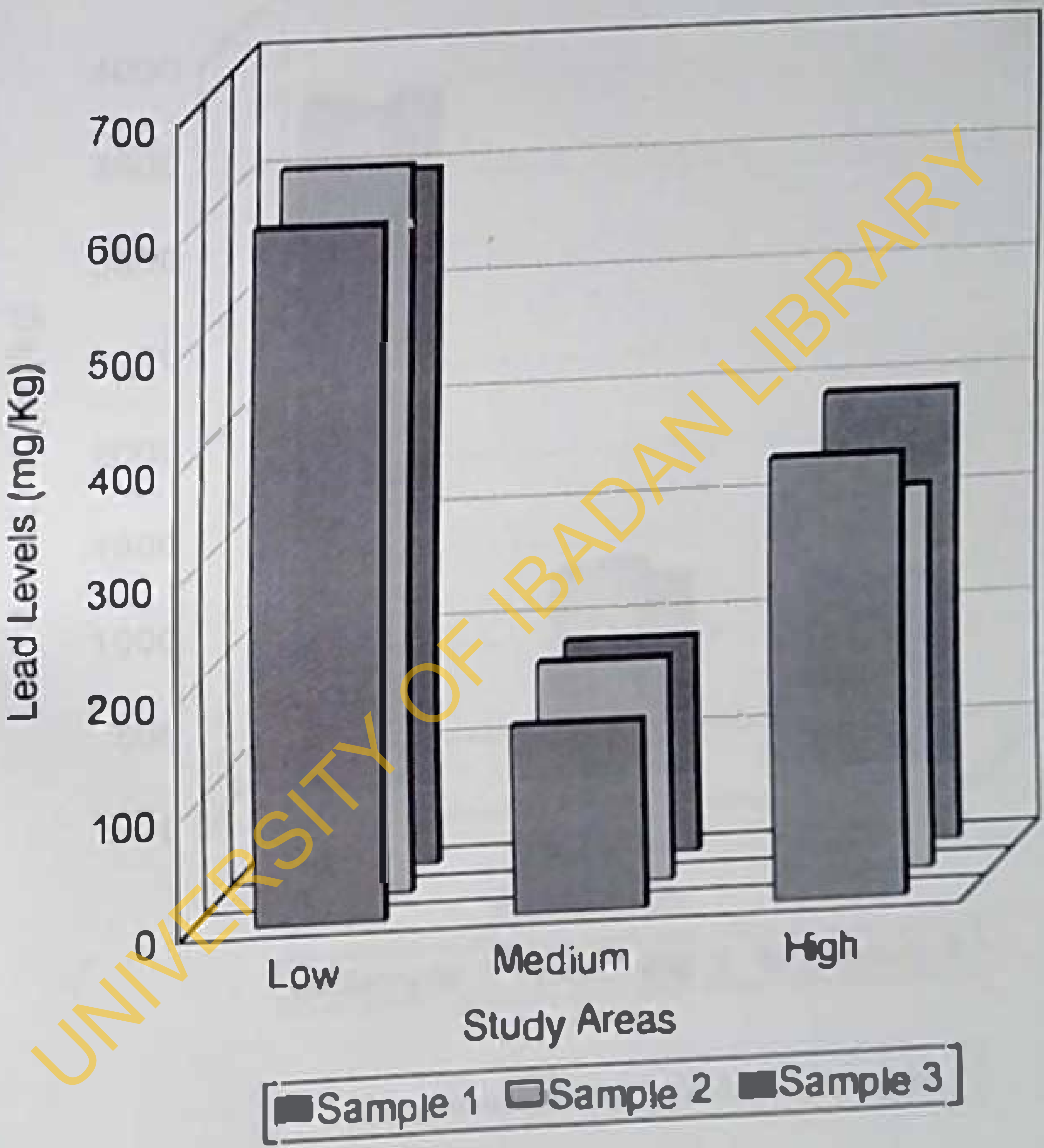
TABLE 24

Lead Levels in the School's Playground in the Three Study Areas

CODE	SAMPLING SITE	SOURCE OF SAMPLE	NATURE DESCRIPTION OF SAMPLE	PROXIMITY TO HEAVY TRAFFIC	LEAD LEVEL mg/kg
Low Density 1	Victoria Island	School playground	Dusty/Sandy	Close	3602
2	Victoria Island	School playground	Dusty/Sandy	Close	3542
3	Victoria Island	School playground	Dusty/Sandy	Close	3600
Mean ± SD					3581.3 ± 34.08
Medium Density 1	Mile II	School playground	Dusty/Loamy	Far	961
2	Mile II	School playground	Dusty/Loamy	Far	945
3	Mile II	School Playground	Dusty/Loamy		950
Mean ± S D					986 ± 66.73
High Density 1	Ojo	School Playground	Sandy	Medium	1063
2	Ojo	School playground	Sandy	Medium	1095
3	Ojo	School playground	Sandy	Medium	1001
Mean ± SD					1319 ± 47
Mean of all					1962 ± 49

FIGURE 16

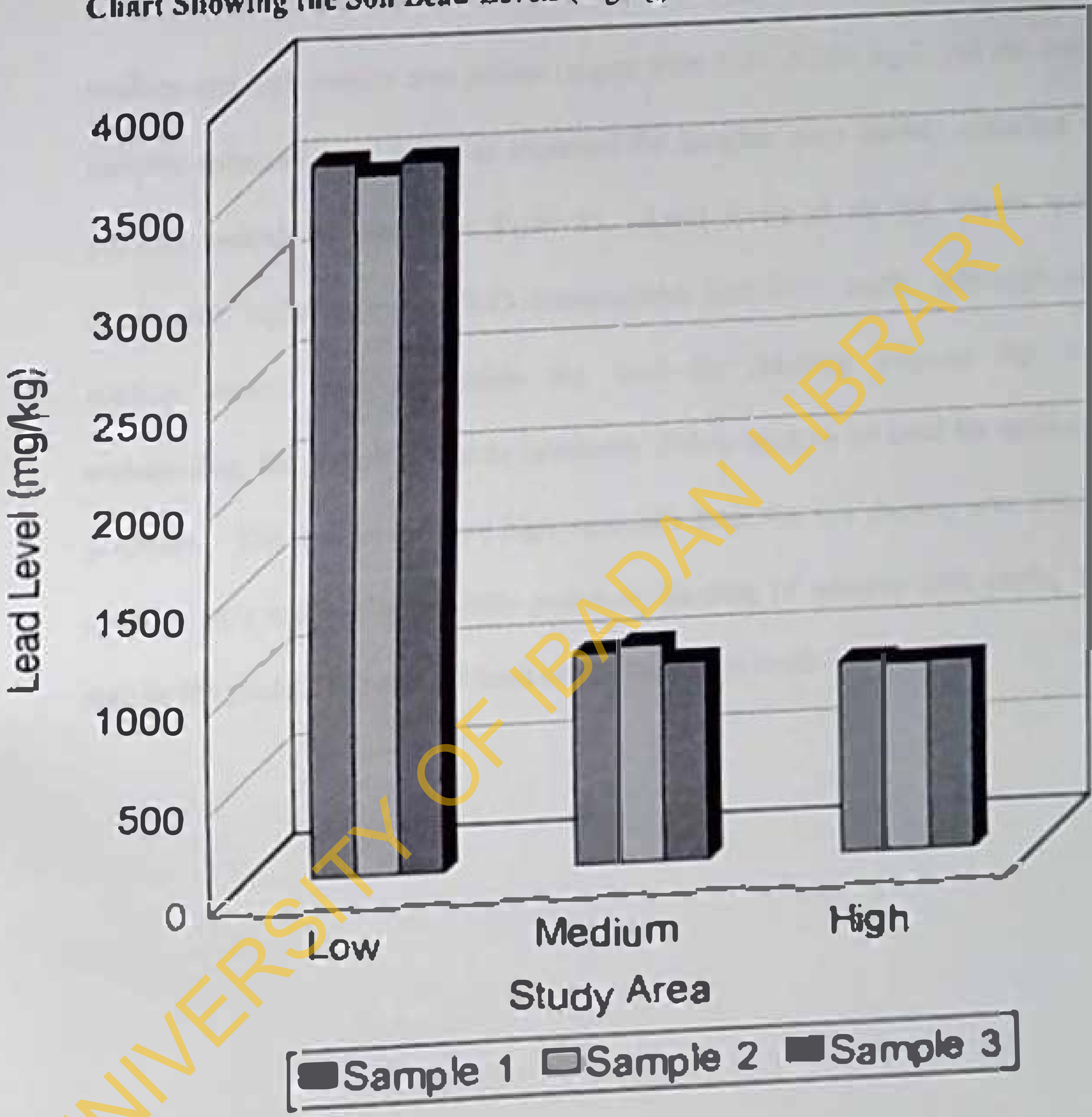
Chart Showing the Sweepings Lead Level (Mg/Kg) in the Three Study Areas



WHO recommended in door lead levels is 200mg/kg

FIGURE 17

Chart Showing the Soil Lead Levels (mg/kg) in the Three Study Areas



WHO recommended outdoor Lead levels is 500mg/kg

4.9 Lead Levels and Other Physical Chemical Parameters of Roof Top Water

Results of the various roof top water samples collected from the low medium and high density area school ranged from 0.05–0.250 mg/l. All the water samples collected were acidic as expected the samples were further subjected to physical analysis as shown in Table 25. Lead levels in all the waters were significantly higher than the WHO recommended limit (0.01 mg/l). Although, the rooftop waters were observably not used for drinking purpose but not withstanding, the waters would be unsuitable if they were to be used for drinking purposes. The lead levels were high especially from the low density area which could be as a result of automobile pollution, leaching of window trim paints, or may be the product of wash off from settled lead from roofs or ledges.

TABLE 25

Lead Levels and Other Physical and Chemical Characteristics of Rooftop Water.

CODE	SAMPLING SITE	pH	TURBIDITY (FTU)	TSS (mg/l)	CONDUCTIVITY (ms/cm)	TDS (mg/l)	Pb (mg/l)
AA1	Victoria Island	5.. .50	136	74	1.16	300	0.250
AA2	Victoria Island	5.82	170	85	1.34	380	0261
AA3	Victoria Island	5.78	200	135	1.06	420	0.280
Mean ± S.D		5.70 ± 0.17	168.67 ± 32.02	98.00 ± 32.51	1.325 ± 0.130	366.67 ± 61.10	0.263 ± 0.015
BB1	Mile 2	5.61	106	90	1.09	240	0.038
BB2	Mile 2	5.10	83	48	0.64	150	0.078
BB3	Mile2	5.01	100	73	0.96	300	0.064
Mean ± S.D		6.01 ± 2.54	100 ± 10.87	71 ± 21.28	0.48 ± 0.28	230 ± 75.49	0.06 ± 0.02
CC1	Ojo	5.38	105	85	0.79	250	0.015
CC2	Ojo	5.93	163	99	0.39	290	0.010
CC 3	Ojo	5.76	89	60	0.26	130	0.090
Mean ± S.D		6.09 ± 0.43	111.3 ± 35.1	81.33 ±19.75	0.48 ±0.27	223.33 ± 83.27	0.05 ± 0.03
Mean of all		5.93 ±2.80	127.89 ± 26.01	83.44 ± 24.51	0.76 ± 0.22	273.33 ± 73.29	0.124 ± 0.02

AA: Low Density Area School
BB: Medium Density Area School
CC: High Density Area School
WHO guideline for drinking water value for lead = 0.01mg/l

4.9 Lead Levels in Cosmetics

Four different cosmetic samples were collected and analyzed for lead. Ninety percent of these cosmetic samples had lead concentration above 50%. The average lead content was 75.82% in all the samples as shown in Table 26.

All the samples collected contained menthol or similar chemical that induce tear formation. Also, when the powder gets into the eye it causes lacrimination which results in the child hand-wiping his/her eye and the lead is ingested when food is subsequently handled or the child suck his/her fingers.

This is against the acceptable legislation that require cosmetic to contain any lead other than trace which could be removed either during or after manufacture.

TABLE 26

Lead Level in Cosmetic Samples

Sample	Description of sample	Material colour	µg/g	% Pb (w/w)
1.	Hashmi Surma	Grey	3.75	87.7
2.	Kajal	Black	0.192	45.6
3.	Hinaka Surma	Grey	1.112	83.3
4.	Tiro	Grey	5.76	86.7

4.10 Personal Interview Guide

Results on behavioral Patterns of Children and Exposure to lead in

4.10.1 Low Density Area

The results of the personal interview guide revealed the following. The School started since 1970, it has been repainted once since the insertion. The children normally rub their hands on the walls, and chew flaking paints. The children normally chew pencils, erasers, papers, crayons, etc. They do not wash their hand before eating, there were teeth marks on the desk, they suck their fingers, and also rub their hands on windowsills, doors, etc. One child was found to be using tiro (lead based cosmetics) and normally cratches his eyes in the morning sections, the teachers complained of perceiving odours from cars exhaust pipes in the School. The school incinerator is very close to the classroom, and they use renovators and charcoal to clean the black boards.

The school had asbestors roofing and a deep well was used as source of drinking water, but the children bring their own water from home, they do not drink the well water. The teachers confessed that they have never heard of lead poisoning before, and they do not know the cause or how it can be controlled.

4.7.2 Medium Density Area

The result of the personal interview revealed that the School started in 1994, and it has been repainted twice. The paints were not flaking. The teachers confessed that the children normally pick paint chips and put it in their mouth. They rub their hands on the walls frequently. The walls of the school was painted with Emulsion paint which had handprints, pencil marks all over. The children do not wash their hands before eating's but were provided with water and soap to do so. The children used a lot of crayons, pencils, and heavy painted illustrations. They chew pencil, chairs, papers, etc. Teeth marks were noticed on their desk and chalks.

Two children were found in the class who used (lead-based cosmetics). The teachers noticed that they do rub their eyes with their hand this could be due to the fact that the preparation contains menthol and other herbs it contains).

The children were provided with wash hand basin to wash their hands after play and before they eat, but they do not like the habit except when been forced. The teachers confessed that is inadequate supervisions (inadequate caretakers to enforce it). The school incinerator is located more than 500m away from the classroom. The school had absebtors roofings and the children normally bring their water from home. The teachers confessed that they have never heard of lead poisoning, causes or prevention.

4.7.3 High Density Area

The result of the personal interview revealed that the school started in 1980 and has always been repainted once, the walls paints were flaking. the teachers said the children normally put any available thing in their mouth both food and non food items. The teachers said two children used lead-based cosmetic and they actually rub their eyes and suck their fingers in the mornings. The children do not like washing their hands before eating even when wash hand basin, soap and towel was provided. The teachers and caregivers said they have never heard of lead poisoning before. The school uses a deep well as source of water. The school incinerator is very far from the school, the school is situated very far from any major road.

CHAPTER FIVE

DISCUSSION

A child an urban centres such as Lagos is exposed to lead from different sources such as paint, gasoline and solder and through different pathways (such as air, food, water, dust and soil)

Pica, the repeated ingestion of non-food substances, has been implicated in many cases of lead poisoning, however a child does not have to eat paint chips to become poisoned. More commonly, children ingest dust and soil contaminated with lead paint which flaked or chalked as its aged. This lead contaminated house dust, ingested via normal repetitive hand – to - mouth activity is newly recognized as a major contributor to the total body burden of lead in children. (ATSDR, 1991).

All automobiles in Nigeria use leaded gasoline, average lead content of regular gasoline being about 0.74g/l (Ogunsola et al, 1994a). Many cars are poorly maintained and characteristically emit blue plumes of bad odour and un-burnt hydrocarbons (Baumbach et al,1995), implying that a higher percentage of lead in gasoline is emitted to the atmosphere (USEPA,1986).

Cottage industries and home lead-works are particularly notorious for exposing children and their families to toxic metals. Some cottage industries in Lagos that employ lead includes type-printing, battery repair, painting, automobile repair and fabrication of plastics. The operations are carried out in crowded residential areas, typically in stores or work shades adjoining the living quarters. There are few controls or restrictions on the lead compounds beings used on the materials being released to the environment, and quite often the homes and surrounding play areas for children are heavily contaminated with lead. The fact that many people live where they work predispose, them and their children to undue exposure to leaden wastes.

Atmospheric lead emissions in Nigeria in recent years have been estimated to be 2800 metric tons per year with most (90%) derived from automobile tail pipe (Obioh et al, 1993). Market places are mostly established around bus stops and vendors, accompanied by their young children, also set their stalls along major roadways with high traffic (and customer volume). After deposition, some of the contaminated dusts get recycled, the trail of dust that accompanies lorries in many parts of the city being, a visible proof of this (Nriagu 1997). Vehicular dust entertainment rate in Nigeria has been estimated to be 6.5g/vehicle km for unpaved roads compared to only 0.1g/vehicle/km for streets in London, England (Akeredolu, 1989).

Lead solder is still found on food cans in Nigeria and contamination of infant foods is likely to be enhanced under hot tropical conditions Lead deposits are relatively scarce in the country and, as far as we know, lead glazing of pottery is not widespread (Nriagu 1997). Cheap, improperly glazed ceramic ware imported from Third World Countries has become common and may be a risk factor especially children from more affluent families. Lead rich medicine and cosmetic represent additional routes of exposure to children. Other potential important sources and pathway include take home exposures from parental occupations and hobbies.

This study has revealed some salient point of interest of these aspects

5.1 Hand Dirt /Dust as a Potential Sources of Lead Exposure

The lead levels in the hands of the children ranged from 0.08-0.39mg/ of hand with a mean of 0.102± 0.07mg/ hand as shown in Table 13. The thought that hand dirt may be a potential source of childhood lead poisoning is not a new one. Studies by (Sayre et al, 1974) shows that a clear relationship was evident between individual hand and blood lead levels. They found out that children with high hand levels (over 30µg/hand) were from homes with high dust levels. (at least 90µg of lead in the household specimens. Since a child's hand contact with the floor, window sills and walls must vary greatly throughout the day, it is expected that a high household lead would produced a correspondingly high lead level on the child's hand.

Mickle et al; (1994) also reported in their study - Surface Dust on Hands for Assessing and Preventing Childhood Lead Exposure- that the average hand lead of children during play in the outdoor environment increased by a factor of 2 2 compared with children playing in the indoor environment. The present data suggest the potential for a childs' ingestion of lead. Since the technique of washing a child's hand in a small plastic bucket with 1litre of double - distilled water, agitating and rinsing was a non-standard method, it may not represent a true lead exposure situation (some metal may still remain embedded in the nails etc). In the absence of any better alternative, my hand washing method was used as a possible approach to the local situation.

The interpretation of the data obtained needs special consideration since the contaminant up to the wrist may not enter the body from food that is eaten mostly with finger. However, it does show that a child's hand has considerable levels of lead, and therefore can potentially contaminate hand or food. The lead values above are significant and raise concerns even though only part of the lead on the hand may be ingested. Any lead ingested adds to the body's uptake.

The fact that one rub off this lead by washings from children's hands indicates that hand contamination readily occurs, and the correlation between lead and other physical parameters like TSS, TDS, turbidity further support this idea. Although adults reside in the same environment, they are less likely to wipe their hands on floors, sills etc, and probably wash their hands more frequently, and certainly do not have the hand-to-mouth behaviour characteristic of preschool children.

This then implies that the lead on children's hand is ingested and absorbed in sufficient quantity to result in an elevated blood lead level. At present, there is no evidence to support this idea.

5.2 Toys as a Potential Source of Childhood Lead Exposure

The total levels of toy washings collected and analyzed ranged from 0.00-0.301mg//toy with a mean of 0.150 ± 0.03mg/toy. This predicts that apart from the lead based paints used to cover toys, the surface areas were also contaminated with dust and dirt's containing lead from the environment. Romieu et al, (1995) recently reported, that in Mexico, the paint used to cover some toys and school equipment had a high lead content. Certainly, this can contribute to the blood lead level in the population studied.

Presently in Nigeria, there is no established standard that regulates the use of leaded paint for children's toys, however, until industry compliance is ensured and until items manufactured are monitored. Studies to assess and monitor blood lead levels will be essential to the public's health.

The researcher observed that some of this children put these toys in their mouth, which can lead to gradual lead poisoning, which can cause serious problems ranging from poor performance in school, hearing loss, even brain damage.

5.3 Classroom Sweepings as a Potential Source of Lead Exposure

Dust is a significant source of exposure to lead particularly for young children (WHO, 1996). Among the nine (9) classrooms dust samples collected, the lead levels ranged from 180-615mg/kg with a mean of 384mg/kg. A mean of 613mg/kg lead level was obtained from the low density area as opposed to 190mg/kg and 390mg/kg from the medium and low density area. The nearness to high traffic and heavy commercial vehicles subjected to rampant Go-slows with considerable stop/start of vehicles may have contributed to the higher results. Only a fence separates the school from the main road in the low density area. Likely source of additional dust lead in the school include lead contained in roof materials, caulking used around windows, paints on the outside surfaces of school building and other structures and street dust. The rate of leaded dust generation/deposition should depend on relative contributions from these sources. (Que Hue et al, (1985), Fergusson and Nicholas (1991)

Reports by Okeakwu (1998) and Aborkor, (1998) shows similar high indoor lead dust levels in Lagos and Ibadan. These results were however higher than the WHO recommended indoor dust levels of 200mg/kg.

Dust control via wet mopping and frequent hand washings has been shown to reduce the blood lead levels in children with high blood lead levels (Charney et al, 1983) but this is not a permanent solution so long as the source of lead in the dust remains.

5.4 Soil as a Potential Source of Lead Exposure

The lead levels in soils in the three study areas ranged from 951-3602mg /kg as shown in table 24. These values were much higher when compared to those reported in earlier studies by (Adogame, 1997, Osagie, 1998, and Aborkor: 1998). This is expected since lead does not disintegrate in the atmosphere. These values are also higher than the WHO recommended value of 500mg/kg.

The mechanism connecting soil lead concentration and blood lead levels is most likely the ubiquitous hand to mouth activities exhibited by children (Sayre et al; 1994). In a study measuring hand lead in day care center children in New Orleans, the amount of hand lead increased significantly after outdoor play compared to after indoor play (Mielke, 1996). The higher the amount of lead dust in outdoor soils, the higher the amount of lead that appears on hands and the greater the potential for children to ingest increased amounts of lead in this route exposure.

Contaminated dusts and soils represent a major source of lead exposure in African children, especially in crowded urban areas. (Nriagu, 1992). Soil Pb intake by infants (1-4 years old) has been estimated to be 0.5 - 7.5µg per kg body weight per day (Nriagu, 1992). When the daily intakes from air inhalation (0.02 - 1.0µg Pb/kg body weight) and foods (0.3 - 1.8µgPb/kg) are also taken into account, it is clear that many children in Lagos metropolis are being exposed to Pb levels that may exceed the WHO recommended Pb intake tolerance of 7µg/kg from all sources (WHO, 1977). Infact, it has been suggested that 10-30% of the children in some urban areas of Africa may be suffering from lead poisoning (Nriagu, 1992).

Children ingest soil and dusts but there is considerable disagreement as to the actual quantity ingested (Stanek and Calabrese, 1995). In general children below the age of 6 years ingest the greatest amount of dust and soil. The amount of lead ingested by a child depends on several factors, education, mouthing behaviour, such as sucking of finger, thumb and toys, hand and face washing behaviour, and nail biting. The amount of lead ingested that is absorbed through the gastro - intestinal track depends on the particle size and form of the lead, the age of the child and his /her nutritional status. (Nriagu, 1998).

Positive association have been reported between soil and dust lead concentrations in urban areas (Rabinowitz and Bellinger, 1988, Thornton et al, 1990,Berry et al, 1994).

A number of studies have reported a high degree of availability of lead in house dust samples; and have found strong significant correlation between lead loading and lead concentration with children's blood lead levels (Davies et al; Thorton, 1990, Berny et al, 1998). Gulson et al; (1994), Murgueytio et al; (1998) concluded that ingestion of lead in dust and soil was the main source and pathway for elevated blood lead in a mining community that they studied. Most of the studies have focused on house dusts.

However, children spend a significant amount of time in schools and lead contamination of dusts in the school environment must be considered a health hazard to the pupils (Duggan and Inskip, 1985, Clark et al; 1991, Lanphear et al; 1998).

For soil lead concentrations up to 5000µg/g an increase in PbB in children from 1.0 to 7.6µg/dl blood per 1000µg/g increase in soil lead have been reported in a number of studies (Steele et al, 1990; Berny et al, 1994). In their recent analysis of pathways of lead exposure in urban children, Lanphear and Roghmann, (1997) showed an increase PbB of children at very low levels of lead in dust, and concluded that current USEPA\HUD guideline levels may be in adequate to protect children. The data from Lanphear et al, (1995, 1998) are for children in urban areas of the United States and may not be directly applicable to children in Lagos metropolis.

Nevertheless, their study and others like it suggest that lead contaminated dusts in schools represent a health hazard to the children and their teachers.

It needs to be emphasized that the socio-ecological conditions in many schools in Lagos metropolis increase the risk of dust lead exposure. The co-location of commercial activities and school (common in Victoria Island), the vibrant outdoor activities during recess (Children can spend hours playing outside), the endemic dusty environment, and infrequent washing of hands increase the likelyhood of hand contamination and hand - to - mouth transfer of lead (Nriagu et al, 1996a). The fact that the school environment in urban areas may be hazardous to children has not received the attention and public concern that it deserves. Few countries currently have guidelines and procedures for dealing with this risk.

Chronic lead poisoning which induces depression, behavioural and neurological disorder in children, hypertension in adults males and negative pregnancy outcomes in females, remains one of the unrecognized public health issues in Nigeria.

5.5 Cosmestics as a Source of Lead Exposure

The results of the cosmetic samples showed lead levels in the range of 0.192 to 5.76µg/g.

This observation is in accordance with Aslam et al, (1979) who analyzed 11 different cosmetic sample and found lead concentration as high as 86%, Fernando et al

(1981) analyzed Thirteen (13) different samples and found lead concentration in the range of 45.6% to 91.8%. The values from my analysis corresponded closely to the lead levels found in Asian Surma materials by them.

Correlation between the use of these materials and elevated blood lead levels has been shown by Ali et al, (1978). In addition modes of gastrointestinal absorption of the materials have been examined Healy, (1980) as well as the implications for mental Health. Aslam et al; (1981) examined blood-lead levels in 62 Asian children in Nothingham and found that the surma users had mean blood-lead concentration of 1.65± 0.68μmol/l while non surma controls had a level of 0.98 ± 0.42 μmol/l. Later studies in Britain by Fernando et al, (1981) confirmed surma to be the principal contribution to the death of a four year old Asian boy in Oldham. They observed the behaviour of children following administration of surma by their mothers and showed that they invariably wipe their eyes with their hands and frequently then suck their fingers. In this, way the lead is deposited into the mouth and whence to the alimentary canal. Of particular concern are the claims made on the containers of these surmas, makes eyes beautiful and protects eyesight or use to prevent eye disease an vision defects.

5.6 Rooftop Water as a Source of Lead Exposure

The lead values of the roof top water ranged from 0.09 - 0.25mg/l as shown in Table 25, this values are in conformity to what Nriagu, (1994) reported Urban sprawl in the developing countries is generally alienated, by high levels of air pollution common air pollutants include oxides of sulfur and nitrogen, toxic metals, organic compounds and particulate matter. The pollutants are removed from the atmosphere by dry deposition and or rainfall. The rainfall and wash out of dry deposited material are on corrugated iron sheets or similar roofing materials.

Besides being impregnated with airborne toxic metals, rain water properties that make it aggressive to metallic objects (Nriagu and Jim 2000). The low pH (as shown in table increase the leaching of metals lead from aerosols and the corrosion potential towards metallic roofing. The low alkalinity, and buffering capacity, as well as high dissolved oxygen, also increases the corrosively of rain water.

In view of a number of prevailing conditions (Such as malnutrition, and immune compromising diseases such as (malaria and cholera) that exacerbate the sensitivity of children to lead toxicity, one would expect the exposure to lead in roof top water to be an important public health problem in Nigeria.

5.7 Teachers Knowledge about Children's Behaviour

The use of in depth interview was necessary as the complement to the analytical work from responses by the teachers it can be deduced that, they do not know anything about lead poisoning and prevention. It also revealed that the teachers are aware of Pica, the repeated ingestion of non-food substances, via normal repetitive hand-to-mouth activity as a source of lead poisoning. There is need to educate teachers about key causes of lead poisoning and potential hazards of lead.

CHAPTER SIX

CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusions

The following conclusions could be made based on results obtained in this study. Lead levels in the samples analyzed were relatively high and this could be assured due to similarities among the communities.

1. The handwashing showed lead values in the range of 0.08 - 0.39mg\hand with a mean of 0.075mg\hand. The low medium and high density had lead level of 0.179 ± 0.097mg\hand, 0.038 ± 0.033mg\ hand and 0.081 ± 0.062mg\hand. The fact that one can wash off this lead with distil water indicates that hand contamination readily occurs
2. The toy washings showed lead values in the ranged of (0.00 -0.301) mg\toy with a mean of 0.150 ± 0.03mg/toy. This predicts that the toy surface area was contaminated with lead in dust and dirt in the child's environment.
3. Classroom sweepings samples indicated varying levels of lead. The mean values of 609mg/kg, 174mg/kg and 330mg/kg and for the low, medium and high-density areas respectively show that air pollution cut across boundaries. Exposure to children through contaminated dust is important

since leaded fuel is still in use and poor combusting vehicles are common in the city of Lagos. This is evident from smoky exhaust pipes that are seen at every place. Inhalation of contaminated dust will increase blood lead levels. Dust is easily found in most parts of the classroom.

4. Soil samples indicated vary levels of lead. The mean value of 3581mg/kg, 935mg/kg and 1019mg/kg respectively. This shows that given that pre-school children play in the school yard, they may ingest lead - contaminated soil via hand - to - mouth activities.

5. Significant concentration of lead was found in the cosmetics; this poses a substantial risk of lead poisoning for the children.

6. Teachers do not have knowledge of lead poisoning, symptoms and ways of prevention of childhood lead poisoning. Information from a health care provider can aid teacher's knowledge.

6.2 Recommendations

- Parents Teachers should be educated about key causes of childhood lead poisoning.
- Children's hands and face should be washed before eating.
- Wash toys and pacifiers before given to children to play with.
- Children should not have access to peeling paint.

- Special attention should be paid to children having access to windows, windowsills and wells.
- Make sure that take-home exposures are not occurring from parental occupations or hobbies.
- Other recommended measures to reduce lead and lead compounds toxicity include.
- Gradual replacement of old metal pipes in house with alternatives like PVC to reduce risk from dissolution of lead from pipes.
- Improved water treatment practices at station continuous inspection of pipes to look for damages which may lead to contamination leading to poor physical and chemical property of water.
- Use of non - lead pigments in paints should be encouraged by SON, and if paints containing lead pigments are used, children should be advised to keep away from the rooms newly painted walls until the paint is fairly dried.
- The installation of vehicular emission control devices e.g Eco flow on older models if vehicles should be encouraged since fairly used old model vehicles are still being imported.
- Encourage public enlightenment campaign of all sectors to warn against possible dangers in the use of old paints can for food and water storage

- Every classroom should have a wash hand basin as shown in fig 17.
- Government should create research funds for development using in relevant institutions to continue research, monitoring and evaluation of environmental toxins.
- Foods should not be stored in open cans, particularly if the cans are imported.
- Pottery or ceramic ware that is inadequately fired or meant for should not be used as food storage.
- Make sure that take-home exposures are not occurring from parental occupations or hobbies parents.
- Children should eat meals regularly, since more lead is absorbed on an empty stomach.
- Parents should make sure that their children diet's contains plenty of iron and calcium.

Figure 17 A Photograph showing Recommended Wash Hand Basin

6.3 Future Outlook

This study would have been more useful if biological samples were taken simultaneously to help in results correlation and interpretations. All these and many other relevant steps if taken in future will guide towards a better and healthier environment fit and lasting for all. Efforts should not be spared by government and agencies like FEPA in trying to relocate some industries to reduce public risk. Researches into safer substitutes to manufacturers should be the goal in future rather than waiting to takecare of pollutants

There is a clear and urgent need for a broader assessment of the magnitude of childhood lead poisoning in Nigeria. At the same time, FEPA authorities should begin to implement policies to reduce the risk of lead poisoning of children in Nigeria. The scientific evidence is large and conclusive, the principal exposure roots are well known, the health effects are well documented and the scientific and medical information gathered from all corners of the world, cry for programs to protect children from lead in their environment. Since automobiles account for must of lead pollution in urban areas in Nigeria it would seem morally indispensable to wait for more evidence for childhood lead poisoning before removing lead from gasoline sold in Nigeria.

The data provided by this study, with other previous researches should act as a guide to future researches in areas of environmental health studies especially with respect to heavy metals like lead, mercury and cadmium etc. The government should release funds to persons and agencies to carry out comprehensive researches with monitoring/evaluation programmes in this respect.

REFERENCES

Aborkor, L. R. (1998): An assessment of Lead in the indoor Environment of Ibadan Metropolis. M. P. H. thesis, University of Ibadan pp. 38

Adedibu, A. A. and Okekunle A. A. (1990): Environmental Sanitation of the Lagos Mainland, Problems and possible solutions. *Environmental Topics* 1 pp 417-440.

Ademoroti, C. M. A. (1986): Levels of heavy metals on barks of trees in Benin-City, Nigeria. *Environ Pollution* (Series B) 11, pp 241-253.

Adogame, A. L. (1997): An assessment of Lead and some heavy metals in the outdoor Environment of Ibadan City. An M. P. H. thesis, University of Ibadan pp 11 – 16

Akeredolu, F. A. (1989): Atmospheric environmental problems in Nigeria – an overview. *Atmos Environ* 23: 783 – 792

Albert, L. A. & Badillo F. (1991): Environmental Lead in Mexico. Rev. *Environ Contamin Toxicol*, 117: 1-49.

Ali, A. R., Smales, O. R. C. and Aslam, M. (1978): Surma and Lead poisoning. *British Medical Journal*, 2, 915.

Alliance to End Childhood Lead Poisoning (1994): The Global Dimension to lead poisoning Washington, DC, 80p.

Apha, (1999): Standard methods for the examination of water and waste water 18th edition. American Public Health Association, Washington, DC.

Aslam M. David SS. & Healy MA (1979): Heavy metals in some Asian medicines and cosmetics. *Publ. Health* (Lond.) 93: 274-284

ATSDR, (1988): (Agency for Toxic Substances and Disease Registry). The nature and extent of Lead poisoning in children in the United States. A report to Congress. Atlanta.

ATSDR, (1993): Toxicological profile for lead. Atlanta, Georgia, US Department of Health and Human Services, Agency for Toxic substances and diseases Registry (TP – 92/12).

Barltrop D. (1969): Mineral metabolism in pediatrics, Oxford, London, Edinburgh, Melborne, Blackwell Scientific Publishers.

Barltrop D. (1972). Children and environmental lead. In: Hepple P ed. Lead in the environment: Proceedings of a conference. London, Institute of Petroleum, pp. 52-60.

Barltrop D. and Meek F. (1979): Effect of particle size on Lead absorption from the gut. *Arch of Environ Health*, 34: 280 - 5.

Barry, P. S. I. (1981): Concentration of lead in the tissues of children. British J. of Ind Med; pp 61-71

Baser, M. E. and D. Marion (1990): A state wide case registry for surveillance of occupational heavy metals absorption. *Am J Public Health* 80: 162-4.

Beckett PH, Davis RD, & Brindley P. (1979): The disposal of sewage sludge onto farmland: the scope of the problem of toxic elements. *Water Pollut Control* 78: 419-436

Bellinger, D., Leviton, D., Waternaux, C., Needleman, H., Rabinowitx, M. (1987): Longitudinal analayses of prenatal and postnatal lead exposure and early cognitive development. *N. Engl J Med* 310: 1037-43.

Belts, P. R., Astely, R. and Rain, D. N. (1973): Lead intoxication in children in children in Birmingham. *British Medical Journal* 1, 402-406.

Bergomi M., Borella, P., Fantuzz, G., Vivoli, G., Sturloni, N., Cavazzutti, G., Tampieri, A., Tartoni, P.L. (1988): Relationship between lead exposure indicators and neuropyschological performance in children. *Dev.Med Child Neurol* 1: 181-90.

Berney P. J., Cote, L. M., Buck, W. B. (1994): Relationship between soil lead, dust lead and blood lead concentrations in pets and their owners. Evaluation of soil lead threshold values. *Environ Res* 67: 84-97.

Bernard BP & Becker CE (1988): Environmental lead exposure and the kidney. *Clin Toxicol*, 26: 1-34.

Bhattacharya, A., Shulka, R., Bornschein, R., Dietrich, K., Kopke, J. (1988): Postural disequilibrium quantification in children with chronic lead exposure: a pilot study. *Neurotoxicology*, 9: 327-40.

Billick IJ & Gray VE 1978): Lead paint poisoning research: review and evaluation 1971-1977. Washinton, DC, US Department of Housing and Urban Development.

Bornschein R. L., (1985): Influence of social factors on lead exposure and child development. *Environ Health Perspect*, 62:343-351

Bornschein R. L., Succop P. A., Kraffl, K. M., C. S. Peace B. Hammond P. B. (1986): Exterior surface dust Lead, interior house dust Lead and childhood Lead exposure in an urban substances in environmental health. Columbia (MO): University of Missouri, pp. 322-32.

Campbell BC, Beattie AD, Moore MR, Goldberg A. & Reid Ag (1977). Renal insufficiency associated with excessive Lead exposure. *Br Med. J. 1*: 482-485

Capar SG & Rigsby EJ (1989): Survey of Lead in canned evaporated milk. *J. Assoc of Anal Chem*, 72: 378-381.

Casells, D.A. and Dodds, E.C (1946): Tetraethyl lead poisoning. *Brit Med J* 2: 681-685.

CDC (1985): Preventing lead poisoning in young children. Atlanta, Georgia, US Department of Health and Human Services, Public Health Service, Centers for Disease Control, pp. 7 – 19 (Publication No. 99-2230)

Centers for Disease Control and Prevention (CDC), (1991): Preventing lead poisoning in young children. A pp. 22-30.

Chamblerlian, A.C. (1985): Prediction of response of blood lead to airbone and dietary lead from volunteer with lead isotopes. Proc. R Soc (London) B 224, pp 149-182.

Charney E, Kessler B, Farfel M, & Jackson D (1983) Childhood lead poisoning of controlled trial for the effect of dust-control measures on blood lead levels. *N Engl. J Med*, 309:1089-1093.

Charney, E. Kessler, B., Farfel, M., Jackson, D. (1983): Prediction of response of blood lead to airborne and dietary lead form volunteer experiment with lead isotopes *Proc. R. Soc. London B* 224, pp 149-182.

Chilsolm J.J. Jr. Mellits E.D., Quaskey, S. A. (1985): The relationship between the level of lead absorption in children and the age, type and condition of housing. *Environ Res* 38: 31-45.

Chisolm JJ & Brown DH (1979): Micromethod for zinc protoporphyrin in erythrocytes: including new data on the absorptivety of zinc protoporphyrine and new observations in neonates and sickle cell disease. *Biochem Med*, 22:214-237.

Chisolm JJ Jr. (1962): Aminoaciduria as a manifestation of renal tubular injury in lead intoxication and a comparison with patterns of aminoaciduria seen in other diseases. *J Pediatr*, 60: 1-17.

Christian, GD (1976): The biochemistry and analysis of lead. *Adv. Clin. Chem.* 18, pp 289-326.

Clark, C. S, Bornshein, R.L., Succop, P., Roda, P. and Peace, B. (1991): Urban lead exposure of children in Cincinnati, Ohio, Chem Specimen Bioavail 13: 163-171.

Clausing P, Brunekreef B, & van Wijnen JH (1987): A method for estimating soil ingestion by children. *Int Arch Occup Environ Health*, 59: 73-82.

Cramer K, Goyer RA, Jagenbury OR, & Wilson MH (1974). Renal ultrastructure, renal function and parameters of lead toxicity in workers with different length of lead exposure *Br J Ind Med*, 31: 113-127.

Culbard EB, Thornton L, Watt JM Wheatley M, Moocroft S, & Thompson M (1988): Metal contamination in British urban dusts and soils. *J Environ Qual*, 17: 226234.

Dabeka RW & McKenzie AD (1987): Lead, cadmium, and fluoride levels in market milk and infant formulas in Canada. *J Assoc Off Anal Chem*, 70(4): 754-757.

Dalpra L, Tibiletti MG Nocera G, Giulotto P, Auriti L, Carnelli V, & Simoni G (1983) SCE analysis in children exposed to lead emission from a smelting plant. *Mutat Ress*, 120: 249-256.

Davies PH & Everhart WH (1973) Effects of chemical variations in aquatic environments: Volume 3 – Lead toxicity to rainbow trout and testing application factor concept. Washington, DC US Environmental Protection Agency (*EPA Report No.* R37301 1C).

Davies DJ, Thornton I, Watt JM, Culbard Eb, Harvey PG, Delves HT, Sherlock JC, Smart GA, Thomas JFA, & Quinn MJ (1987a) Lead intake and blood lead in 2 year olds UK urban children. *Sci Total Environ*, 90: 13-29.

Davies, J.M. and Svendsggard D.J (1987): Lead and child development. *Nature* 32d: 297-300.

Davis JM & Grant LD (1992) The sensitivity of children to lead. In: Guzelian PS, Henry CJ & Olin S ed. Similarities and differences between children and adults: Implications for risk assessment. Washington, *DC. ILSI Press*, PP 15-27.

Davis, R.K., Thorton, A.W. (1989): Inhalation of tetramethylead and tetraethylead. Arch *Environ Health* 6: 473-479.

Dawson G. Relation of lead and social factors to IQ of low-SES children: A partial owned housing a report to congress. Washington (DC) HUD.

Day JP Hart M, & Robinson MS (1975) Lead in urban street dust. *Nature (Lond)*, 253: 343-345.

Deaveaux, P, M.A. Kibet, W.S. Dempster, F. Pocock and K. Forment (1980): Blood lead levels in pre-school children in Capetown. *S. Afri Med J* 69: 421-424.

Delves, H.Y., Shclock, J.L. and Quirin, M.J. (1984): the temporal stability of blood lead concentrations in adults exposed only to the environmental lead. *Human Toxicol* 3 pp 279-88.

Dietrich, K.N., Succop, P.A., Berger, O., Hammond, P., Bornschein, R.L. (1991). Lead exposure and cognitive development of urban preschool children. The Cincinanati lead study cohort at age 4 years (1991). *Neurotoxicology and Teratology*. 13: 203-11.

Dong A, Simisiman GV, & Chesters G (1983) Release of phosphorus and metals from soils and sediments during dispersion. *Soil Sci*, 139: 97-99.

Dorn, C.R., Pierce, J.O., Chase, G.R. and Philips, P.E. (1975): Environmental contamination of lead, cadmium, zinc and cooper in a new lead-producing area. *Environ Research* 9, pp 159-172.

Drill S, Knoz J, Mahar H, & Morse M (1979) the environmental lead problem: An assessment of lead in drinking water from a multi-media perspective. Washington, DC, US Environmental Protection Agency (*EPA*-570/9-79-003, NTIS-PB-296556).

Duggan M.J., Inskip, M.J.: Childhood exposure to lead in surface dust and soil: a community health problem. *Public Health Rev* 13: 1-54.

Elhelu, M.A., Caldwell T.D. and Hirpassa, D. (1995): Lead in inner city soil and its possible effects to children's blood lead. *Arch of Env Health* 2 (50) pp 165-169.

Elias R (1985) lead exposures in the human environment. In: Mahaffey KR ed. Dietary and environmental lead: human health effects. Amsterdam, Oxford. New York, *Elsevier Science* Publishers,pp 79-109.

Elwood L.M. (1984): Greater contribution to blood lead from water than from air. *Nature* 310: pp 138-140.

Elwood PC, Yarnell JWG, Oldham PD, Catford JC, Nutbeam D, Davey-Smith G, & Toothill C (1988b) Blood pressure and blood lead in surveys in Wales. *Am J Epidemiol*, 127: 942-945.

Emhart, C.D., Wolf, A.W., Kenard, M.J., Enhart, P., Filipovich, H.F., Sokol, R.L. (1986): Intrauterine exposure to low levels of lead (1986): The status of the neonate. *Arch Environ Health* 41: 287-91.

FAO/WHO (1993) Evaluation of certain food addivites and contaminants. Forty-first report of the Joint FAO/WHO Exert Committee on food Additives. Geneva, World Health Organization (*Technical Report Series* 837).

Farfel, M.R. and Chisolm J.J Jr (1990): Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. *Am J Public Health*, 80: 1240-5.

Fergussion, D.M., Fergussion, J.E., Horwood, L.J., Kinzett, N.G. (1988). A longitudinal study of dentine lead levels, intelligence, school performance, and behaviour Part II; dentine lead and cognitive ability. *J. Child Psych. Psychiat* 29: 797-809.

Fergussion, J; Nicholas, K. (1991). Trace elements in street and house dust sources and specialization. *Sci Total Environ* 100: 125-150.

Fernando NP, Healy MA, Aslam M, Davis SS, & Husein A (1981) Lead poisoning and traditional practices: the consequences for World Health. A study in Kuwait, *Public Health (Lond)*, 95: 250-260.

Fernando, N.P., Healy, M.A., Aslam, M., Davis, S.S. and Hussain, A. (1981). Lead poisoning and traditional practices: the consequences for world health. A study in Kuwait. *Public Health*. 95: 250-260.

Fullmer CS (1990) Intestinal lead and calcium absorption: Effect of 1,25-dihydroxy-cholcalciferol and lead status. *Proc Sco Exp Biol Med*, 194: 258-264.

Fulton, M., Raab, G., Thompson, G., Laxen D., Hunter R., Hepburn, W.(1987): Influence of blood lead on the ability and the attainment of children in Edinburgh *Lancet* 1: 1221-6.

Galal-Gorchev H (1991a) Global overview of dietary lead exposure. Special issue incorporating the proceeding of the Symposium on the Bioavailability and Dietary Exposure of Lead, Chapel Hill, North Caroline, September 1990. *Chem Speciation Biovailab*, 3(3/4): 5-11.

Gezandheidstraad, (1984): Guidelines for lead, Recommended standards for outdoor air quality. The Hague, Ministry of Health and Environmental Hygiene.

Globler, S.R., Theusen, F.S. and Maresky, L.S. (1996): Evidence of undue lead exposure in Capetown before the advent of lead petrol. *South African Medical Journal* 86(2): pp 169-171.

Golow, A.A. and E.E., Kwaansa-Ansah (1994): Comparison of lead and zinc levels in hair of pupils from four towns in Kumasi Municipal Area of Ghana. *Bull Environ Contam Toxicol*. 53: 325-331.

Goyer, R.A. (1991): Toxic effiects of metals in, casarett and Douls toxicology. The basic seience of poisons. Amdur, M.O. Doull, J. Klassen, CD (eds). 4th edition, Pergemon Press, New York, pp 623-680.

Goyer, R.A. (1993): Lead toxicity. Current concern. *Env. Health Perspect*. 100, pp 177-189.

Goyer, R.A., May, P., Cates, M.M. (1995): Role of chelating agents for prevention, intervention and treatment of toxic metals. *Environ Health Perspect.* 11(103) pp. 1048-1052.

Graef, J. (1990): A case report of lead paint poisoning during renovation of a victorian farmhouse. *Am J Public Health* 1990; 80: 1183-5.

Graziano JH & Blum C (1991) lead exposure from lead crysta. *Lancet* 337: 141-142

Grobler, S.A., Theunsen F.S. and Maresky, C.S. (1996): Evidence of undue lead exposure in Capetown before the advent of lead petrol. *South African Medical Journal.* 86(2) pp 169-171.

Gulson, B.L., Davis, J.J., Mizon, K.J., Korsch, M.J., Law, A.J, Howarth, D. (1994): Lead bioavailability in the environment of children. Blood lead levels in children can be elevated in a mining community. *Arch Environ Health* 49: 326-331.

Gupta AK, Sehgal SK, Mohan M, & Anand NK (1990) Cosmetic plumbism. *Indian Paediatr*, 27: 760-761.

Hammond, P.D. and Aronson, A.L. (1964): Lead poisoning in cattle and horses in the vicinity of a smelter. *Ann N Y Acad Sci III*; 595-611.

Healy, M. and Aslam, M. (1986): Lead containing preparations in the Asian community a retrospective Survey. *Public Health*, 100: 147-151.

Healy, M.A. and Aslam, M. (1986): Eastern Eye *Nursing Times.* Oct. 22, 36/37.

Herberg S (1973) prevention of accupational poisoning from inorganic lead. *Work Environ Health*, 10: 53-61.

HUD Guidelines for the evaluation and control of lead-based paint hazard in housing (1990): Report Number HUD-1539-LBP, US Department of Housing and Urban Development, Washington DC.

IPCS (1989) Environmental Health Criteria 85: Lead – Environmental Effects; Geneva, World Health Organization, pp 106.

James AC (1978) Lung deposition of submicron saerosols calculated as a function of age and breathing rate. In: National Radiological Protection Board Annual Report. Harwell, UK, *National Radiological Protection Board*, pp 71-75.

Johnson NE & Tenuta K (1979) Diets and lead blood levels of children who practice pica. *Environ Res*, 18: 369-376

Kakulu, S.E. (1985): Heavy metal in the Niger Delta. Impact on the petroleum industry on the baseline levels, A Ph.D thesis, University of Ibadan pp 63.

Kehoe RA (1987) Studies of lead administration and elimination in adult volunteers under natural and experimentally induced conditions over extended periods of time. *Food Chem. Toxicol*, 25: 425-493.

Kolbye AC, Mahaffey R, Fiorino A, Correliussen PC, & Jelinek CF (1974) Food exposures to lead *Environ Health Perspect*, 7(Exp issue) 65-75

Koo WWK, Succop PA, Bornschein RL, Krug-Wispe SK, Steichen JJ, Tsang RC Hammond PB & Berger OG (1991) Serum vitamin D metabolites and bone mineralization in young children with chronic to moderate lead exposure, *Pediatrics*, 87: 680-687.

Kopp SJ, Barron JT, & Town JP (1991) Cardiovascular actions of lead and relationship to hypertension: a review, *Environ Health Perspect*, 89: 91-99.

Koren G, Chang N, Gonen R, Klein J, Weiner L, Demshar H, Pizzolato S, Radde I, & Shime J (1990) Lead-exposure among mothers and their newborns in Toronto. Can. *Med. Assoc. J*, 142: 1241-1244.

Lagos State Government Diary (1999): pp 4-6

Lagos State Secretariat Diary (1999) pp 9.

Lai CS (1972) Lead poisoning as an occupational hazards in Chinese opera actors: a case report. *Singap Med J*, 13: 115-117.

Landrigan PJ, Gehlbach SH, Rosenblum BF, Shoults JM, Cancelaria RM, Barthel WF, Liddle JA, Smrek Al, Staehling NW, & Sanders JF (1975) Epidemic lead absorption near an ore smelter; the role of particulate lead. *N Engl J Med*, 292: 123-129.

Landrigan, P.J. (1987). Committee on environmental hazard statement on childhood lead poisoning. *Paediatrics* 79, pp 437-465.

Landsdown, R., Yule, W., Urbanodvicz, M.A. and Hunter, J. (1986). The relationship between blood-level concentrations, intelligence, attainment and behaviour in a school population, the second study. *Int Arch Occup Environ Health* 57: 225-35.

Lanphear, B.P., Burgoon, D.A., Rust, S.W., Eberly, S. and Clike, W. (1998) Environmental exposures to lead in urban children"s blood lead levels. *Environ Res* 76: 120-130.

Lansdown, R. Yule W., Urbanowicz., M.A. and Hunter J. (1986). The relationship between blood-level concentrations, intelligence, attainment and behaviour in a school population. The second study. *Int. Arch Occup Environ Health*. 57: 225-35.

Lapwo ML, Bruckman L, Rubino RA, Markowitz S, Gillete M, & Kapish J (1974) Role of airborne lead in increased body burden of lead in Hartford children. *Environ Health Perstects*, 7 99-102

Laxen DPH, Raab GM, & Fulton M (1987) Children's blood lead and exposure to lead in household dust and water -- a basis for an environemnital standard for lead in dust. *Sci Total Environ*, 66 235-244

Lovering TG ed. (1976) lead in the environment, Washington, DC, US Department of the interior, Geological Survey (Geological Survey Professional Paper No. 957).

Lyngbye, T., Hanson, O.N., Trillings gaard A., Beese, I. and Grandjean, P. (1999) Learning disabilities in children significance of low level lead exposure and confounding effects. *Acta Pediatr. Scand* 79 352-60.

Manton WI & Cook JD (1984) High accuracy (stable isotope dilution) measurements of lead in serum and cerebrospinal fluid. *Br J Ind Med*, 41 313-319.

Manton WI & thal ER (1986) lead poisoning from retained missiles an experimental study. *Ann Surg*, 204 594-599.

Maresky, L.S. and S.R. Globler (1993) Effect of the reduction of petrol lead on the blood lead levels of South Africans. *Sci Total Environ*; 136: 43-48.

McGrath SP (1986) the range of metal concentrations in top soils of England and Wales in relations to soil protection guidelines. In Hemphill DD ed. Trace substances in environmental health, vol 20. Columbia, Missouri, University of Missouri, pp 242-252.

Meanhaut W, Zoller WH, Duce RA, & Hoffman GI (1979) concentration and size distribution of particulate trace elements in the south polar atmosphere. *J Geophys Res*, 84: 2421-2431.

Meredith PA, Moore MR, Campbell BC, Thompson GG, & Goldberg A (1978) delta-Amino levulinic acid metabolism in normal and lead-exposed humans. *Toxicology*, 9 1-9.

MicMichael, A.J., Baghurst, P.A., Wigg, N.R., Vimpani, G.V., Robertson, E.F., Roberts, R.J. (1988) Port Pirie cohort study. Environmental exposure to lead and children abilities at four years. *N Engl J Med* 319 468-75.

Mickle, H.W. (1994) Lead in New Orleans Soils new images of an urban environment. *Environmental Geochemistry and Health* 16 (3/4) 123-128.

Mielke H, Blake B, Burroughs S, & Ryan FJ (1974) Urban lead levels in Minneapolis the case of the Hmong children. *Environ Res*, 34 64-76.

Mitchell-Heggs CAW, Conway M. & Cassar J (1990) Herbal medicine as a cause of combined lead and arsenic poisoning. *Hum Exp Toxicol* 9 195-156

Moore MR, Goldberg A, & Young-Laiwah AAC (1987) lead effects on the haembiosynthetic pathway. Relationship to toxicity. *Ann NY Acad Sci*, 514 191-203.

Mooty J, Ferrand CF, & Harris P (1975) Relationship of diet to lead poisoning in children. *Pediatrics,* 55: 636-639.

Murgueytio, A.M, Evans, R.G., Roberts, D. (1988): Relationship between soil and dust lead in a mining area and blood lead levels. *J Expos Anal Environ Epidemiol* 8: 173-186.

Murthy, G.M. and Rhea U.S. (1971): Cadmium, copper, iron, lead, manganese and zinc in evaporated milk, infant products and human milk. *J Dairy Sci* 54: 1001-1005.

Mushak P., Davis J.M., Grochett, A.F., Grant, L. D. (1989): Prenatal and postnatal effects of low level lead exposure: integrated summary of a report to the US Congress on child hood lead poisoning. *Environ Res* 50: 11-36.

Mushak, P., Davis, J.M., Grochett, A.F. and Grant, L.D. (1989): Prenatal and postnatal effects of low level lead exposure. Integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environ Res* 80: 11-36.

Ndiokwere, C.C. (1984): A study of heavy metals pollution from motor vehicle emissions and its effects on roadside soil, vegetation and crops in Nigeria Environment *POH Serv.* B, 7; pp 33-43.

Ndiokwere, C.C. (1984): A study of heavy metal pollution from motor vehicles emissions and its effects on roadside soil, vegetation and crops in Nigeria. *Environ Pollut Serv* B, 7, pp 35-42.

Needleman, H.L. and Gatsonis, C.A. (1990): Low-level lead exposure and the IQ of children. *JAMA* 263: 673-8.

Needleman, H.L, Gunnoe, C., Leviton, A., Reed, R., Peresic, H., Maher, C., Barret, P.: Deficits in psychologic and classroon performance of children with elevated dentine lead levels. *N Engl J Med.* 300: 368-95.

Needleman, H.L., Schell, A., Bellinger, D., Leviton, A. and Allred, E.N. (1990): The long-term effects of exposure to low doses of lead in childhood: An 11-year follow-up report. *N. Engl. J Med* 322: 83-8.

Neilsen T (1984) Atmospheric occurrence of organolead compounds. In: Grandjean P ed. Biological effects of rofanolead compounds. Boca Raton, Florida, CRC., pp 43-62.

NIOSH, (1978): Criteria for a recommended standard occupational exposure to inorganic lead (revised criteria) NIOSH, DHEW Publ. No. 78-158, United States Govt Printing Office, Washington, DC.

Nriagu J (1979) Global inventory of natural and anthropogentic emissions of trace metails to the atmosphere, *Nature (Lond)*, 279: 409-411.

Nriagu J (1989) A global assessment of natural sources of atmospheric trace etals. *Nature (Lond)*, 338: 47-49.

Nriagu Jerome O. (1999): Global inventory of natural and anthropogenic emissions of trace metals to the atmosphere. *Nature (Lond)* 279; pp 409-411.

Nriagu, J.O. and Myoung-Jin Kim (2000): Trace metals in drinking water. Sources and effects. RA Deininger et al (eds), *Security of Public Water Supplies*, P 115-131.

Nriagu, J.O. (1992): Toxic metal pollution in Africa. *Sc. Total Environ*, 121: 1-37.

Nriagu, J.O., C.C. Jinabhar, R. Naidoo and A. Coutsoudis (1997): Lead poisoning of childre in Africa II. Kwazulu/Natal, South Africa *Sci Total Environ* 197: 1-11.

Nriagu, Jerome O. (1989): A global assessment of natural sources of atmospheric trace metals. *Nature (Lond)* 338; pp 47-49.

Nriagu, O. and Givrin, L. (1998): Lead poisoning of children in Africa IV. Exposure to dust in primary schools in South-Central Durban, South Africa. *Sci Total Environ*, 221: 117-126.

Obioh, I.B., Oluwole, A.F. and F.A. Akeredolu (1988): Atmospheric lead emissions source and strength in Nigeria. : In inventory in heavy metals in the Environment (J. Allan and JO Nriagu, Eds) *CEC* Consultants Ltd, Edinburgh, Scotland, Vol. 2 pp 271-274.

OECD (1993) Risk Reduction Monograph No. 1 lead background and national experience with reducing risk paris, organization for Economic co-operation and Development, 277 pp (report No. OCDE/GD(93)67).

O' Flahery EJ, Hammond PB, & Lerner SI (1982) Dependenc of apparent blood lead half-life on the length of previous lead exposure in humans. *Fundam Appl Toxicol*, 2: 49-54.

Ogunsola, A.J., A.F. Oluwole, O.I. Asubiojo, M.A. Durosinmi A.O (1994) Envrionmental impact of vehicular traffic in Nigeria health aspects. *Sci Total Environ*, 121: 111-116.

Okekaru, I.P. (1998): An assessment of lead levels in the indoor environment of selected areas in Lagos Metropolis. M.P.H. thesis, pp., University of Ibadan pp 15-30.

Ong CN & Lee WR (1980) High affinity of lead for fetal hemoglobin. *Br J Ind Med*, 37: 292-298.

Ong, C.N. (1985): Concentration of lead in maternal blood, cord blood and breast milk. *Arch Dis Child* 60, pp 576-759.

Onianwa, P.C. (1985). Accumulation exchange and retention of trace heaving metals in some mosses from S.W Nigeria. A Ph.D. Thesis, University of Ibadan.

Onianwa, P.C. (1985): Accumulation, exchange and relation of trace heavy metals in some mosses from S.W Nigeria. Ph.D Thesis, University of Ibandan.

Osagie, O.C. (1998). An assessment of lead in the outdoor environment of some areas in Lagos Metropolis, an M.P.H. Thesis, University of Ibadan pp. 11-15

Osibanjo, O. and Ajayi, S.O. (1980). Trace metal levels in tree barks as indicators of atmospheric pollution. *Environment International* 4, pp 239-244.

Osibanjo, O. and Ajayi, S.O. (1989). Trace metal analysis of petroleum products by flame atomic absorpiton spectrophotometry. *Nig J Nat Sci* 4, pp 33-40.

Piomeli, S, Rosen J.F., Chisolm, J.J. Jr. and Graef, J.W. (1994). Management of childhood lead poisoning. *J. Paediatr.* 105 pp 523-532

Pontifax AH & Garg AK (1985) lead poisoning from an asian Indian folk remedy. *Can med Assoc J*, 133: 1227-1228.

Pounds J.G. (1994). Effects of lead on cellular lead metabolism and toxicity, in abstracts from the conference on the role of chelating agents for the prevention, intervention and treatment of exposures to toxic metals. Research Triangle Park, *NC A3* pp 22-23.

Que-Hee S., Peace, B., Clark, S., Boyle, J., Bornschein, R. and Hammond, P. (1985). Evaluation of efficient methods to sample lead sources, such as house dust and hand dust, in the homes of children. *Environ Res.* 38: 77-95

Rabinowitz MB, Leviton A, & Needleman HL (1986). Occurrence of elevated protoporphyrin levels in relation to lead burden in infants. *Environ Res.* 39 257-257.

Rabinowitz, M.B., Bellinger, D.C (1988): Soil lead-blood lead relationship among Boston children. *Bull Environ Contam Toxicol*, 41: 791-797.

Rabinowitz, M.E. (1977). Kinetic analysis of lead metabolism in healthy humans. *J of Clinical Investigations* 58 – 260-270

Romieu,I T.L. Carreon E., Lopez, C., Palazuelous, Y., Rios Manuel and M., Henandex-Avila (1993). Environmental urban lead exposure and blood lead levels in children of Mexico City. *Environ Health Perspect*, 103: 1036-1040.

Rosen JF & Chesney RW (1983) Circulating calcitriol concentration in health and disease *J. Pediatr.* 103: 1-7.

Rosen JF, Chesney RW, Hamstra AJ, Deluca HF, & Mahaffey KR (1980) Reduction in 1,25dihydroxyvitamin D in children with increased lead absorption *N Engl J Med*, 302: 1128-1131.

Rosen, J.F., Markowitz M.E., Bijur P.E,: (1993) Sequential measurements of bone lead content by L-X-ray flourescence in $CaNa_2$ EDTA-treated lead-toxic children. *Environ Health Perspect*

Rosen, J.F., Markowitz, ME., Bijur, P.E. (1993) Sequential measurement of bone lead content by L-Xray flourescence in $CaNa_2$ EDTA-treated lead-toxic children *Environ Health Perspect*

Routh DK, Mushak P, & Boone L (1979) A new syndrome of elevated blood lead and microencephaly, *J. Pediatr Psychol*, 4: 67-76.

Royal Commission on Environmental Pollution, (RCEP) (1983): Lead in the environment, 9th Report.

Saenger P, Markowitz MR & Rosen JF (1984) Depressed excretion of 6β-hydroxycortisol in lead-toxic children, *J. Clin Endocrinal Metab*, 58 363-367.

SAHC (1993) Proceedings of the International Meeting on Non-Occupational Exposure to Lead, Melbourne, Australia, 5-9 October 1992. Adelaide, South Australian Health Commission, pp 56.

Sayre, J.W., Charney E., Vostal J. and Pless I.B. (1974): House and hand dust as a potential source of childhood lead exposure. *Am. J Dis Child* 127: 167-170.

Schmitt MDC, Trippler DL, & Wachtler JN (1988) Soil lead concentrations in residential Minnesota as measured by ICP AES. *Water Air Soil Pollut*. 39: 157-168

Schroedar, H.A. and Balassa J.J. (1961): Abnormal trace metals in man: lead. *J Chron Dis*, 14: 408-425.

Schroedar, S.R., Hawk, B., Otto, D.A., Mushak, P. and Hicks, R.E. (1985): Separating the effects of lead and social factors on IQ. *Environ Res* 38: 144-54.

Schwartz, J, and Otto O (1987). Blood lead, hearing thresholds and neurobehavioural development in children and youth. *Arch Environ Health* 42: 153-60.

Shaltout A, Yaish S, & Fernando N (1981) Lead encephalopathy in infants in Kuwait, *Ann Trop paediatr (London)*, 1 209-215.

Shapiro, H. and Johnson, J.D. (1982) Lead in, McGraw-Hill Encyclopaedia of Chemistry. Parker, S. (Ed) Mc-Graw-Hill Book Company, New York, pp 558-561.

Sharma RR, Chandy MJ, & Lad SD (1990) Transient hydrocepalis and acute lead encephalopathy in neonates and infants, report of two cases. *Br. J. Neurosurg*, 4:141-146.

Shenggao, H. (1997): Lead exposure at an early age substantially increases lead retention in the rat. *J Environ Health Perspect* 105, pp 412-417.

Sherlock JC, Pickford CJ, & White GF (1986) Lead in alcoholic beverages. Food Addit Contam, 3:347-357.

Sherlock, J.C. (191): Exposure of man to heavy metals from foods. Proc Int Conf Heavy metals in the environment Edinburgh 1: pp 6-15.

Shulka R, Bornschein, R.L., Dietrich K.N., Buncher, C.R., Berger, O.G., Hammon, P.B., Succop, P.A. (1989): Fetal and infant lead exposure effects on growth in stature. *Pediatrics* 84: 604-12.

Shwartz J, Landrigan PL, & Baker EL (1990) lead-induced anemia: dose response relationships and evidence for a threshold. *Am J Public Health* 80: 165-168.

Siegel M, Forsyth B, Siegel L, & Cullen MR (1989) The effect of lead on thyroid function in children. *Environ Res*, 49 190-196.

Silbergeld EK (1991) Lead in bone: implications for toxicology during pregnancy and lactation. *Environ Health Perspect*, 91: 68-70.

Silver, P.A., Hugh, P., Williams, S., Faed, J.M., (1988): Blood lead, intelligence, reading attainment and behaviour in eleven year old children in Dunedin, New Zealand. *J. Child Psych. Psychiat.* 29: 43-52.

Smith, J.L. (1976). Metabolism and toxicity of lead. In Trace elements in Human Health and Disease. Prasad A. Oberlas D (eds). Academic Press, New York, pp 443-453.

Snodgrass, G.J., Ziderman, D.A., Gulan, V. and Richards, J. (1973): Cosmetic plumbism. *British Medical Journal* 4, 230.

Sobel AE, Yuska H, Peters DD, & Kramer B (1940). The biochemical behaviour of lead Influence of calcium phosphorus and vitamin D on lead in the blood and bone *J. Biol Chem.* 132: 239-265.

Speight, J. (1996): Environmental Health (1st edition) pp. 4-6, 96-97 and 270-271.

Stanek, E., Calabrese, E.: Daily estimates of soil ingestion in children (1995). *Environ Health Prospect* 103: 227-285.

Steenhout A & Pourtois M (1981) Lead accumulation in teeth as a function of age with different exposure. *Br. J Ind. Med.* 38: 297-303.

Stuit EJ (1974) Biological response of male and female volunteers to inorganic lead *Int Arch Arbeitsmed*, 33: 83-97.

Thorton I., Davies, D.J.A., Watt, J.M. and Quin M.J. (1990): Lead exposure in young children from dust and soil in the United Kingdom *Environ Health Perspect* 89: 55-60.

Trotter RT (1990). The cultural parameters of lead poisoning: A medical anthropologist's view of intervention in environmental lead exposure. *Environ Health Perspect*, 89: 79-84.

UNESCO/UNEP (1985): Living in the environment, a source book for environmental education pp. 3

US EPA (1989a). Air quality criteria for lead. Washington, DC, Environmental Protection Agency (*EPA-600/8-83/02aF-dF*).

US, EPA (1991): Drinking water regulation. Federal Register 56(110): 26460-26546, Washington, DC.

US, EPA (1992): National Air Quality and Emissions Trends Report, 1991. 450-R-92-001, Office of Air Quality Planning and Standard, Environmental Protection Agency, Research Triangle Park, NC.

US, EPA, (1986): Air quality criteria for lead. Vol I-IV. Report No EPA 600/8/8-83/028, Environmental Criteria and Assessment Office, US Environmental Protection Agency, Cincinnati, Ohio.

USEPA (1994): Guidelines on residential lead-based paint, lead-contaminated dust, and lead-contamination soil US Environmental Protection Agency, Office of Prevention Pesticides and Toxic Substances. Washington DC.

Vahter M & Slorach S (1990): GEMS – Global Environmental Monitoring System Exposure monitoring of lead and cadmium: an international pilot study within the WHO/UNEP Human Exposure Assessment Location (HEAL) Programme. Nairobi, United Nations Environment Programme. 82 pp.

Von Schirinding Y.E.R. (1988). Environmental lead exposure among inner-city Cape Town. A case study of associated factors. Ph.D thesis, University of Cape Town, South Africa.

Von Schirinding Y.E.R., D. Bradshaw, R. Fuggle and M. Stokol (1991). Blood lead levels in South African inner-city children. *Environ Health Perspectives*, 94: 125-130.

Von Schirinding, Y.E.R. and R.F. Fuggle (1984). A study of the relationship between low level lead exposure and classroom performance in South African Children. *Int. J. Biol Sci*, 6: 97-106.

Von Schirinding, Y.E.R., M.A. Kibel, R. Fuggle and A. Mathee (1995a). An overview of childhood lead exposure in South Africa. Unpublished Report, Department of Environmental Health, Johannesbourg, South Africa.

Wang J-D, Jand C-S, Hwang Y-H, & Chen Z-S (1992). Lead contamination around a kindergarten near a battery recycling plant. *Bull Environ Contam Toxicol*, 49: 23-30.

Ward, N. I. (1986). Lead contamination of the London Orbital motorway (since its openings in 1996). *J. The Sc of the Total Env.* 93: pp 277-283.

Warley, M.A., Blackledge, P. OGorman, P. (1968). Lead poisoning in eye cosmetics. *British Medical Journal*, 1, 117.

Weast RC ed. (1985). Handbook of chemistry and physics, 66th ed. Boca Raton, Florida. *CRC* Press.

White JM & Harvey DR (1972). Defective synthesis of α and β globin chains in lead poisoning. *Nature (Lond)*, 236: 71-73.

WHO (1980). Recommended health-based limits in occupational exposure to heavy metals. Report of a WHO Study Group. Geneva, World Health Organization (*Technical Report* Series No. 647).

WHO (1985) Guidelines for the study of dietary intake of chemical contaminants. Geneva, World Health Organization (*WHO Offset Publication*, pp 55-56).

WHO (1987) Air quality guidelines for Europe. Copenhagen, World Health Organization, Regional Office for Europe, pp 200-209 (*European Series*, No. 23).

WHO (1992): Commission on Health and Environment. A report on our planet, Our Health. Geneva, Switzerland, pp. 1-14.

WHO (1993) Guidelines for drinking-water quality: 2nd ed. Volume 1: recommendations. Geneva, World Health Organization, 188 pp.

WHO, (1980): Diseases caused by lead and its toxic compounds. In early detection of occupational diseases. World Health Organisation, Geneva pp 85-90.

WHO, (1986): Diseases caused by lead and its toxic compounds. In early detection of occupational diseases. World Health Organisation, Geneva pp. 85-90.

WHO, (1995): Inorganic lead. Environmental health criteria 165, World Health Organisation, Geneva.

Williams, P.I. and Burson, J.L. (1985): Industrial Toxicology. Safety and Health Applications in the Work Place. Van Nostrand Reinhold, New York, pp 197-210.

Winneke, G., Brockhaus, A., Swers, U., Kramer, U. and Neuf, M. (1990): Results from the European Multicenter study on lead neurotoxicity in children implications for risk assessment. *Neurotoxicity and Teratology* 12: 553-9.

Wittners, L.E. (1988): Lead in bones IV. *Arch Environ Health* 43, pp 381-391.

young children. A pp. 22-30.

Yule, W., Lansdown, R., Miller, J. and Urbanowicz, M. (1981): The relationship between blood lead concentrations, intelligence and attainment in a school population. A pilot study. *Dev. Med Child Neurol* 1981; 23: 567-76.

Zieglar, E.E. (1978): Absorption and retention of lead by infants. *Pedatr. Res.* 12: pp 29-34.

Ziehlus, R.L. (1983): Lead alloys and inorganic compounds. In Encyclopaedia of Occupational Health and Safety. Permeggiani L. (ed) Revised 3rd edition Vol. 2. International Labour Organisational (ILO) pp 1200-1204.

APPENDIX 1A

LIST OF APPROVED PRIMARY SCHOOLS IN ETI-OSA LOCAL EDUCATION DISTRICT

S/N	NAME OF SCHOOLS	ADDRESS
1.	Fed. Home Science Ass. Primary School	5, Ruxton Road, Ikoyi
2.	Adrao Int. Primary School	28, Ahmadu Bello Way, V/Island
3.	American Int. Primary School	Victoria Island
4.	Corona Primary School	35, Mekuwen Road, Ikoyi
5.	St. Saviour's Primary School	54, Alexander Avenue, Ikoyi
6.	Command Children Primary School	Bonny Camp. V/Island
7.	Aunty Ayo Primary School	Biaduro Street, Ikoyi
8.	Nigeria Airforce Primary School	V/Island
9.	Crescent Primary School	1004, Fed. Housing Estate V/Island
10.	Faith Villa. Creche Primary School	29, Bourdillon Road, Ikoyi
11.	Greenwood house primary School	8, Omo-Osagie Street SW Ikoyi
12.	Police Children primary School	Police Force H/Q Annex Kam Salem House Moloney, Lagos
13.	Aunty Ayo's Preparatory Primary School	Biaduo Street off Keffi St, SW Ikoyi
14.	Kemson Primary School	17A, Reeve Road, Ikoyi
15.	Virginia Primary School	Plot 854 Bishop Aboyade-Cole Street, Ikoyi
16.	Betty Dolphin Int. Primary School	19B, H.F.P. Way, SW Ikoyi, Lagos Dolphin Estate
17.	Italian Primary School	Admiralty Way Lekki
18.	Star Bright Primary School	Agungi Town
19.	Lagos British Primary School	Kingsway Road, Ikoyi
20.	Martin Int. Primary School	28, Ologun-Agbaje Street, V/Island

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APPENDIX 18

LIST OF APPROVED PRIMARY SCHOOLS IN AMUWO ODOFIN LOCAL EDUCATION DISTRICT

S/N	NAME OF SCHOOLS	ADDRESS
1.	American Model Primary School	Ijegun Satellite Town, 35 Alebiosu Street, Ijegun
2.	Ab-bel International primary School	Amuwo Odofin Housing Estate
3.	Bralay Kiddies Primary School	8, Awomolo Housing Estate, Ijegun
4.	Central Bank Primary School	Satellite Town Ojo
5.	Christian Council of Nigeria Primary School	612, Road, Festac Town
6.	Child Foundation Primary School	Plot 25, Block 3, Amuwo-Odofin, Mile 2
7.	Crest Private Primary School	21, Comfort Olayinka Close, Oluti
8.	Dr. Soyemi Memorial Primary School	7th Avenue, 5 close, Festac Town
9.	Early Life Primary School	3rd Avenue Festac Town
10.	Ebunlayo Children Primary School	1. Ebunlayo Close, Ijegun
11.	Floybis Primary School	21/23, Aku Street, Satellite Town
12.	Happy Children Primary School	2nd Avenue, 205 Road, Close House 14, Festac Town
13.	Happy Family Primary School	9, Casco Street, Kuje Amuwo
14.	Holy Child Primary School	1, Holy Child Way, Satellite
15.	Ideal Day Primary School	1, Ideal Close off Oshinleino Street
16.	King's Cross Primary School	9, Ambrose Aedonwonyi Street, Ijegun
17.	Learning field Primary School	Close 30 Satellite Town
18.	Little Lilies Primary School	2nd Avenue, 23rd Close, Festac Town
19.	Loral Primary School	201, Road, close Festac Town
20.	Leatls Primary School	54, Olusonoye Street, Ijegun Satellite
21.	Mur-yola Primary School	Alakija Junction Navy Town Road Ojo
22.	Nazareth Primary School	Navy Town, Ojo
23.	Prime Montessori Primary School	27, New Road, Satellite Town
24.	Radiance Primary School	O' Close 1st Avenue Festac Town
25.	Royal Kindergarten	28, Ogun Crescent Satellite Town
26.	St. Jude's Primary School	5th Avenue Adjacent 'L' Close Festac Town
27.	Stepping Stone Primary School	Satellite Town Badagry Road, Lagos
28.	Subuola Infant Academy	5th Avenue 'R' Close Festac Town
29.	Stella Maris Primary School	7, Adesina Bello Close, Oluti
30.	Tender Touch Primary School	1st Avenue Festac Town
31	Tender Crown Primary School	20, Comfort Obah Street, Kirikiri
32	The beginners Primary School	Plot 25, Block 3, Amuwo Odofin

APPENDIX 1B

Private

LIST OF APPROVED PRIMARY SCHOOLS IN AMUWO ODOFIN LOCAL EDUCATION DISTRICT

S/N	NAME OF SCHOOL	ADDRESS
33.	Nowa Primary School	Navy Town
34.	Pathfinder Primary School	16, Egbayelo Street, Olodi Apapa, Ojo
35	Rowa Creche Primary School	23rd Opposite J. Close 2nd Avenue F
36	St. Peters Anglican Primary School	49 Wilmer Crescent Olodi Apapa, Ojo
37	Scotfield Primary School	Plot 2248,Amuwo-Odofin private Lay-Out
38	Bolade Children Primary School	Plot 399, Amuwo Odofin Mile 2.
39	Heladin Primary School	2nd Avenue, 23rd T Close, House 10 Festac Town
40	Peridot Primary School	67A, Corporation Road, Crystal Estate
41	Brightest & Best Primary School	5th Avenue V. Close, House 20, Festac Town
42	Christ Hill Children Primary School	Block 24, Plot 3008, Ladisa Crescent
43	Broadway Junior Primary School	12, Dillion Street, Apapa

APPENDIX 1C

LIST OF APPROVED PRIVATE PRIMARY SCHOOLS IN OJO LOCAL EDUCATION DISTRICT

NAME OF SCHOOLS	ADDRESS
Carita Bianchi Pry. School	17, Unity Crescent Igbede, Ojo
Tedoca Pry. School	Ojo
Greenlight Foundation pry.	2, Keniki Ave.Sabo Oniba Ojo
Holy Infant pry. School	8, Ajanaku Street, Ajangbadi
Samuel Primary School	21, Costain Road, Ojo
Esther Foundation Primary School	Yakonde Ilogbo Road, New Site Oke-Iyanu, Ojo
New World	12B, Bale Street, Onikeke Off, Ojo
Global Victory Primary School	15, Segun Oloyede Street, Okokomaiko, Ojo
Sophia Samuel Primary School	14, Ayilara Street, Ojo
Minavester Int. Primary School	Ilogbo Road, Ajangbadi
Palbest Primary School	5, Oladosu Alebiosu Str., off Oroleye St. Afromedia, Ojo
Igbehin-Adun Primary School (For Deaf andDumb/Hearing).	Ojo 9/11, Ajagbe Street, Okokomaiko
Modos Primary School	1, Tedi Road, Ojo Barracks, Ojo
The Sound Foundation Primary School	6/9, Onibuore Ave. off Ilogbo Road, Ojo
Adeniyi Goodwill Primary School	34A, Oretan Street, Ijanikin
Entro Children Primary School	15, Igbockerin Road, Agboroko Iba Town
Paxton Int. Primary School	17/19, College Road, Agric.
Bright Excellent Children Pry. Sch.	2, Oremeji Str., Ilogbo-Elegba
Bedrock Primary School	21/23 Aku Str., Satelite Town
Oakcrest Private School	2, Alafia Str., Ijanikin
Dorbis Primary School	3, Baruwa Str., Amuwo Kuye
King of Kings Private Pry. School	4, Alowonle Str., Okokomaiko
Tina Startright Private Pry. School	222, Ojo Road.

APPENDIX IC

LIST OF APPROVED PRIVATE PRIMARY SCHOOLS IN OJO LOCAL EDUCATION DISTRICT

S/NO	NAME OF SCHOOLS	ADDRESS
1.	St. Michael Pry. School	9, Masebinu Str., Sabalaje
2.	Penny Pry. School	10, Quadri Str., Coker Village
3.	'N' Precious Child Pry. School	House 5, I & C Close, Festac
4.	Elitorial Pry. School	6, Catholic Mission Str., Coker Village
5.	Tola Pry. School	Off Aka Rd. White House B/Stop
6.	Tide Foundation Pry. School	29, Awe Str., Orile Iganmu
7.	St. Mary's Pry. School	11, Rnnotu Str., Kemberi Okokomaiko
8.	Kristobell Jnr. Academy Pry.	Agboroko Village Iba Town
9.	Florentin Priv. Pry. School	5th Ave. M. Close House 23 Festac
10.	Joke-Ayo Int. Pry. School	52/55, Old Ojo Road Suberu, Ojo
11.	Kiddyrille Pry. School	183, Badagry Expressway, Ojo
12.	Daystar Pry. School	8B, Odiche Str. Coker Village
13.	Tonylield Pry. School	6, Yaro Str., Okokomaiko
14.	St. Michael Pry. School	22, Busari Alimi Str. Itire Ilogbo
15.	First Grade Mont. Pry. School	16, Owoseni Str. Mabamu Village Badagry E
16.	Torch of Knowledge Pry. School	1, Babalola Ayodele Str., Igboelerin
17.	Abiodun Bank Anthony Mem. Pry. Foundation Pry. School	Iba New Layout, Itire, Isiwo, Ojo
18	Orire Private Primary School	40, Sadiku Street, Orile-Iganmu
19.	Bright Star Primary School	23, Olaitan Street, Iganmu
20.	Queen Elizabeth Primary School	13, Alhaji Sule Street, Ilemba Hausa
21.	Dorcan Mem. Primary School	62, Cardoso Street, Awori-Ora, Ajegunle
22.	St. Raphael's Primary Schol	7, Dotun Isijola Street, Aka.Okoko
23.	God's Glory Primary School	Tajudeen Nurudeen Street, Ajangbadi Afr. Oj...
24.	Learnfast Child Primary School	9, Maxwell Fekorigba Street, Ilemba Hausa
25.	Bela Primary School	143, Lagos Badagry Expressway, Ojo
26.	Moonlight Primary School	7, Wowo Street, Olodi, Apapa
27.	Mary Fresh Int. Primary School	1, Ajisola Oladapo Street, Ojo
28.	Dayhad Primary School	18, Rashidi Babatunde Street, Okoko
29.	Odofin Christ Primary School	Km 33, Badagry Expressway Rd. Metro B/St
30.	LACOED Staff Primary School	Lagos Street, College of Education Ojo Ilum
31.	Cubic Private Primary School	76, Imam Street, Amukoko
32.	Doseg Primary School	21, Road, C. Close House 1, Festac
33.	Sunray Primary School	5/7, Sunray Close, Ilogbo, Ojo
34.	Sheik Yusuf Arabic & Eng. Pry.	1/3, Makaz Sheik Yusuf Str. Mebanu Layout B/Exp.
35.	Derens Int. Primary School	Ore-Ope St. S/L Quarters Ishasi Odan, Ojo
36.	Olufunwa Child. Pry. School	23, Arufa Str., KM 16 Badagry Expressway, Ojo

APPENDIX II

PERSONAL INTERVIEW GUIDE

Note: (Respond of is the teacher/Caretaker in the classroom with at least one year of teaching experience in the School).

Area: (L.G.A): ....

Name of School: ....

Demographic Information

Gender: Male 1
Female 2

Age______________

When did the School started:______________________________

When last were the classrooms painted______________________

What kind of paint was used______________________________

Do the children in the class play with painting or paints______________

Are Eye pencils/Eye shadow used by the children______________

How many:______________________

Do these children scratch their eyes in the morning______________

Do the children play and chew the following

Pencils and Biros

Suck thumb or lick finger

Play with plastic toys

Rub their Nose Against the painted wall.

Do the children at any time lick or chew the walls

Rub their fingers on the walls

Chew and scratch desk

Do you perceive the smell of exhaust pipe smoke in class

if yes

How often: ______________________________

Do you have waste basket/bucket for your dirty sweeping

Where is the waste basket/bucket located

1. Inside the class
2. Outside the class

Have you ever heard about lead poisoning

Do you know how it can be prevented

RESEARCBERS REPORT (FOR OFFICIAL USE ONLY)

1. How far is the school from the main road:

Very close	(50m)	1
Close	(100 - 200m)	2
Far	(200m)	3
Very far	(500m)	4

2. How is vehicle (motor) traffic around the School

High (more than 100 motors/hr)	1
Moderate (200 - 500 motors/hr)	2
Scanty (50 - 150 motors/hr)	3
Very low (less than 50 motors)	4

3. Presence if industry or commercial activity

1. Near the School	1
2. Mechanic Workshop	2
3. Paint Industry/painter or sprayer	3
4. Petrol Station	4
5. Battery Manufacturing/Dealer/Charger	5
6. More than one of the above	6
7. None of the above	7

Others, Specify_______________________________

4. Any Waste Dump around the School

Yes	1
No	2

5. Nature of Waste Dump

Area covered (large, moderate, small)	1
Smell (high, Moderate, Small	2
Composition (Domestic only)	3
Composition (Domestic and industrial)	4
Composition (Industrial only)	5
(Unknown - Describe)	6

6. Proximity of classroom to waste dump

Very close (50m)	1
Close (100 - 200m)	2
Far (200m)	3
Very far (500)	4

7. How recent does the wall painting of the classroom look?

Newly painted	1
Fairly old paint	2
Old Paint	3
No Paint	4

8. What is the colour of paint?

Specify ....

9. What is the nature of the paint used if newly painted?

Oil paint (still smelly) 1

5. Nature of Waste Dump

Area covered (large, moderate, small)	1
Smell (high, Moderate, Small	2
Composition (Domestic only)	3
Composition (Domestic and industrial)	4
Composition (Industrial only)	5
(Unknown - Describe)	6

6. Proximity of classroom to waste dump

Very close (50m)	1
Close (100 - 200m)	2
Far (200m)	3
Very far (500)	4

7. How recent does the wall painting of the classroom look?

Newly painted	1
Fairly old paint	2
Old Paint	3
No Paint	4

8. What is the colour of paint?

Specify ....

9. What is the nature of the paint used if newly painted?

Oil paint (still smelly) 1

Water paint 2

Not identified 3

Others, specify 4

10. Are there any scratch marks on the wall? ____________

11. Do the children exhibit pica

12. Do the children wash their hands before early.

13. Is adequate water provided for the children to wash their hands

14. Do the children chew and lick papers.

15. How is the ventilation of the class adequate
(Cross ventilation, at least 20 pp. Window)
Fair (less than / window 2
Poor (Windows not easily opened or window seen 3

16. What is the nature of the building
Bungalow 1
One or two storey 2
Two or five storey 3
Above five storey 4

APPENDIX III

LIST OF THE VARIOUS TOYS IN THE THREE STUDY AREAS

Empty Powder Tin (Tony montana)

Empty Powder Tin (Dusting Powder)

Vim container

Bournivita Tin

Cream container

Hair Cream Relaxer

Milo container

Mobil container

Building blocks

Football

Car

Bicycle

Plastic birds

Telephone

Ball

Numeric counter