LEAD CONTENT OF SOME FOOD ITEMS SOLD BY HAWKERS IN ZARIA METROPOLIS

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(Msc/Pharm.Sci/51193/2005-06)

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A THESIS SUBMITTED TO THE FACULTY OF PHARMACEUTICAL SCIENCES ABU ZARIA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF MASTERS DEGREE OF PHARMACEUTICAL CHEMISTRY.

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DECLARATION

I hereby declare that this work is done by me and it is a record of my research, carried out under the supervision of Dr. M.M. Odunola and Dr. M. Garba. This work has not been presented before for the award of any degree. The work of other investigators are dully acknowledged and referred to accordingly.

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ABSTRACT

This study determined lead concentrations in seven types of food sold by hawkers in Zaria and environs. Lead is a heavy metal that serves no useful purpose in the human body.

The food samples were coded A –G respectively and the locations were coded A1 – H1. The samples were analyzed using Atomic Absortion Spectrophotometric Method (AAS), after appropriate treatment.

Lead concentrations of sample A ranged from 0.03 – 0.16mg/L; B ranged from 0.2 – 0.18mg/L; C ranged from 0.01 to 0.22mg/L; D ranged from 0.04 to 0.16 mg/L; Lead concentration of E ranges from 0.02 to 0.04mg/L;F ranged from 0.12 to 0.13mg/L; and G ranged from 0.01 to 0.02mg/L. The concentrations of Lead in the food samples were consistently high for locations D1 and E1. This can be due to the human activities around the areas.

Key Words: Food, Hawkers, Lead, Zaria

CHAPTER ONE

1.0 INTRODUCTION

1.1.1 DEFINITIONS OF A HEAVY METAL

Heavy Metals are chemicals with a specific gravity that is at least five times the specific gravity of water. The specific gravity of water is 1 at 4^{o}C (39^{o}F). Specific gravity is a measure of density of a given amount of a solid substance when it is compared to an equal amount of water. Some well-known toxic metallic elements with a specific gravity that is 5 or more times that of water are arsenic, 5.7; cadmium, 8.65, iron 7.9;lead 11.34 and mercury 13.546(Lide, 1992) .

1.1.2 BENEFICIAL HEAVY METALS

In some quantities, certain heavy metals are nutritionally essential for a healthy life. Some of these are referred to as the trace elements (e.g. iron, copper, manganese, and zinc). These elements, or some of them, are commonly found naturally in foodstuffs, fruits and vegetables, and in commercially available multivitamin products (International Occupational Safety and Health Information Centre 1999). Diagnostic medical applications of heavy metals include direct injection of gallium during radiological procedures, dosing with chromium in parental nutrition mixtures, and the use of lead as a radiation shield around x-ray equipment (Roberts 1999). Heavy metals are also common in industrial applications such as in the manufacture of pesticides, batteries, alloys, electroplated

metal parts, textiles dyes, steel, and so forth. (International Occupational Safety and Health information center 1999). Many of these products are in our home and actually add to our quality of life when properly used.

1.1.3 TOXIC HEAVY METAL

Heavy metals become toxic when they are not metabolized by the body and they are accumulate in the soft tissues. Heavy metals may enter the human body through food, water, air or absorption through the skin when they come in contact with humans in agriculture and in manufacturing, pharmaceuticals, industrial, or residential settings, industrial exposure accounts for a common route of exposure for adults. Ingestion is the most common route of exposure in children (Roberts,1999). Children may develop toxic levels from the normal hand-to-mouth activity of small children who come in contact with contaminated soil or by actually eating objects that are not food (dirt or paints chips) (Dupler, 2001). Less common routes of exposure are during a radiological procedure from inappropriate dosing or monitoring during intravenous (parental) nutrition, from a broken thermometer (Smith et al, 1997), or from a suicide or homicide attempt (Lupton et al, 1985).

As a rule, acute poisoning is more likely to result from inhalation or skin contact of dust, fumes or vapours, or materials in the work place. However, lesser levels of contamination may occur in residential settings, particularly in older homes with lead paint or old pumping (International

Occupational Safety and health Information Centre 1999). The Agency for toxic substances and Diseases registry (ASTDR) in Atlanta Georgia, (a part of the U.S. Department of Health and humans services) was established by congressional mandate to perform specific functions concerning adverse human health effects and diminished quality of life associated with exposure to hazardous substances. The ASTDR is responsible for assessment of waste sited and providing health information concerning hazardous substances, response to emergency release situations, and education and training concerning hazardous substances (ASTDR Mission Statement, 2001).

The Heavy metals most commonly causing problems in humans are lead, Mercury, Cadmium, Arsenic, Nickel, Aluminum. These metals tend to accumulate within the brain, kidneys, and immune system where they can severely disrupt normal function. It is conservatively estimated that up to 25% of the US population suffer to some extent from metal poisoning. Most of the heavy metals in the body are as a result of environmental contamination due to industry. For example, in the United States alone industrial sources contribute more than 600,000tons/year of lead into atmosphere to be inhaled or –after being deposited on food crops, in water, and soil, to be ingested.

Common sources of heavy metals are industrial sources , lead from the solder in tin cans and copper pipes, pesticides species and cooking

utensils, cadmium and lead from cigarette smoke, mercury from dental fillings, contaminated fish, latex paints and cosmetics; and aluminum from antacids and cookware. Some professions with extreme high exposure include battery makers, gasoline station attendants, printers, roofers, solderers, dentists, and jewelers.

Heavy metal poisoning are vague or associated with other problems, early symptoms can include, Headache, fatigue, muscle pains, Indigestion, Tremors, Constipation, Anemia, Pallor, Dizziness, poor coordination.

The person with even mild heavy metal toxicity will experience impaired ability to think or concentrate. As toxicity increase, so do the severity of the sign and symptoms.

Numerous studies have demonstrated a strong relationship between childhood learning disabilities (and other disorders including criminal behaviours) and body stores of heavy metals, particularly lead.

Aluminum interferes with normal neurological function and higher level of this metal have been found among dyslexic children as well as children with other behavioural difficulties.

Antimony accumulates in the hair and is responsible for ailments such as itching skin pustules, bleeding gums, conjunctivitis, laryngitis, headache, weight loss and anemia.

Arsenic toxicity may lead to macrocytosis, anemia, jaundice, brown cutaneous pigmentation. Hyperkeratosis of palms and soles, and white transverse banding of nails.

Barium levels in the hair are related to pathologic processes; how ever one retrospective study indicated that high levels in the hair along with elevated calcium/magnesium ratio correlated with myocardial infarction.

Cadmium exposure has been associated with hypertension and child aggression

Lead is associated with many health problems, causing deficits in central nervous system functioning, and is correlated with both reduced intelligence scores and lowered school achievement

Mercury is considered toxic at any level in the body, because there are known metabolic functions for which mercury is necessary. At high concentrations mercury causes liver and kidney damage and neurological symptoms.

Nickel accumulates with age and smoking, and is known to cause cordermattis, and its observed perturbation of immunoglobulin levels, elevated hair levels may serve as a warning sign for immune dysfunction a long with its potential as helpful maker of cardiovascular risk.

Uranium dust has long been associated with increase incidence of liver cancer. The accumulation of uranium in the hair maybe related to blood group, with types AB and B having apparent affinity for the elements.

Vanadium is recognized as inhibiting Na/K AtPase, and correlations between hair vanadium levels and bipolar disorder indicate that the elevated vanadium found among patients with active symptoms tends to normalize during recovery.

1.2.1 POLLUTION

Pollution is the alteration of surroundings, due to man's activities within and outside the surrounding. The alteration in most cases is unfavourable one which in some cases as result of a man's by product.

Pollution such as gases, smoke particles, lead, asbestos, pesticides and radioactive isotopes are commonly found in the atmosphere. Other pollutants altering environments include, sewage, organic chemicals and toxic metals like zinc, copper, cadmium, lead, mercury etc. most pollutants that concern man occur in nature and to some large extent due to man activities.

Pollutants generated by man, most especially in urban and industrial areas are important due to their adverse effects. There is accumulating evidence that many types of pollutants can become distributed over the whole earth in relatively short periods of time. For example, radioactive fallout from atmospheric nuclear test is detectable throughout the world within days or weeks. Another dramatic example is the world-wide distribution of chlorinated hydrocarbon such as DDT and its metabolites.

Some pollutant remains dangerous indefinitely, for example Beryllium and lead. Others that are eventually broken down into harmless compounds may still persist in the environment for long periods of time.

Another important characteristics of pollutants is that they maybe concentrated biologically so that levels in one part of a ecosystem are much larger than those in other parts. This typically occurs in food chain whereby levels in an organism are higher than those in its food.

Lead is one of the seven metals of antiquity and has accompanied the Eur-Asian and the American civilizations (Waldron, and Staven, 1974). Lead equally represents a potential or real public health hazard. Human kind uses vast quantities of this metal and is widely dispersed throughout the environment. The major sources of lead in the communities which are of significance for the health of communities arise from its industrial and other technological uses The major depressive and non-recoverable use of the lad is in the manufacture of alkyl lead fuel additives. Other important sources of environmental lead pollution relates to the manufacture of batteries, metal sheets. If these substances have values higher than those quoted in water it will cause ill health and death ultimately.

There are other substances that may be injurious to health under certain conditions, if present in high concentrations. Some of them are regarded as essential constituents of drinking water, and too low concentrations of them may also have adverse effects on health.

Fluorides is beneficial when present in concentrations of 0.8 – 1.0mg/l, but will cause mottled enamel of the teeth when present in portable water in levels higher than between 1.0 – 1.5mg/l. Nitrates, which may occur naturally in portable water or may result from sewage from soak –away, access-pool, sewers etc, or from the use of fertilizers on farms, are harmful to infants. Nitrate present in water used for preparing food for infants are reduced to nitrites in the intestinal tract, leading to a disease called infantile methaemoglobinemia. This occur only when the nitrate content is higher than 45mg/l. cumulative effect of nitrate in the body has made a consideration of nitrate level in infant significant at 10mg/l and this is suggested as a level fixed for infants.

A Variety of water-borne disease such as cholera, dysentery, typhoid, paratyphoid, gastro-enteritis, infantile diahoera, leptospirosis, tularaemia and other parasitic infections are known to be transmitted through polluted water.

1.2.2 EFECTS OF POLLUTION

Air pollutants are known to produce eye and respiratory irritations in animals as well as in humans (Heuss and Glasson 1968). Hospital records shows that the infamous smog episode of 1952 in London caused about 4000ll as nerve gas near trains that are carrying it. Water pollutants can also endanger aquatic life and every year millions of fish are reported killed by municipal and industrial wastes (Wilbur, 1979).in 1971, Nigeria

experienced the worst cholera epidemic recorded in its written history. The statistical bulletin published by the Epidemiological unit, Ministry of Health, Western Nigeria now Ogun, Ondo, Osun and Oyo state recorded 14,158 suspected cases; 1,721 of them were confirmed and 609 of them died (Ademoroti (1979); statistical bulletin, Ministry of Health (1973). However, as pipe borne water supply scheme was introduced to rural areas of the country, these figures declined rapidly. Sewage, toxic chemicals and disease organisms can also make water unfit for use by farm animals. There are some types of pollutions known to have adverse effects on animals at levels that do not appear to affect human health. Thermal pollution, the excess heating of water in rivers or lakes can kill fish and pesticides levels in many species of birds have reduced reproduction rates through mechanisms such as interference in calcium metabolism (Woodwell, 1980).

Agriculture and horticulture are also both affected by pollution. Air pollutants having severe effects on vegetation include sulpur oxides from copper and lead smelters, and hydrogen fluoride from fertilizer manufacturing and aluminum reduction. When pollution levels are not high enough to produce noticeable injury, retardation of growth may occur. Complex changes occur in the plant ecosystem with effects on one specie leading to effects on the others since some plants are likely to be more sensitive than others to the pollutants (Air, Water or radiation) (Woodwell,

1980). Sometimes, the undesirable effect of a pollutant is the increase in plant life, as when the introduction of plant nutrients such as phosphorus, nitrogen, and carbon leads to algal blooms in water bodies.

Human beings are probably most concerned about the direct effects of pollutants upon their own health, even though these effects are not necessarily essential to humans, but become toxic at sufficiently high levels or doses. For example, the typical adult requires 10 to 15mg of iron daily and has a body burden of 3g or less yet each year there are a couple of thousand cases of acute iron poisoning (Deichamann and Ferards, 1979). Occasionally, fatalities occur in small children from ingestion of ferrous sulphate tablets; small children need about 5 to 10mg of iron par day but ingestion of 40mg to 1.5g of $FeSP_4$ has been known to produce severe effects. Similarly, copper, which is apparently needed in haemoglobin synthesis, can produce acute poisoning when 5 to 50mg of soluble salts are ingested; the typical intake by adult is probably 1or 2mg daily.

Thus the different toxic doses and actual environmental doses may not be large. Lead, which is not known to be essential to human life, can be toxic at blood concentration above 0.8ppm; yet typical human has a concentration of about 0.25ppm (Paterson (1965).

In determining levels that are toxic to humans, one is generally faced with the following problem. Sufficiently, high levels are toxic or even lethal

to everybody, although there is generally a wide range of sensitivities among the human population. For example, tartaremetic (antimony potassium tartrate) can be lethal at doses as low as 130mg but some persons have survived doses as high as 15g (Gleason, 1979). On the other hand, sufficiently low doses appear to have no toxicological effect. One possible explanation of these facts is that there exists a threshold, a level below which there are no effects. Another possible explanation, however, is that effects are occurring but that experimental techniques are not sophisticated enough to detect them at low dose.

It is well known that the effects of exposure are sometimes not apparent until many years have elapsed. Asbestos is known to produce increased mortality from lung cancer and asbestosis among asbestos workers but these effects take 20 or 30 years to show up (Selikoff, 1979). There is growing evidence that many chronic diseases such as asthma, emphysema and bronchitis are environmentally induced and result from long-term exposures to levels of various substances too low to produce acute effects. Many drugs are toxic at certain does but are of therapeutical value at lower doses, and the two doses are usually not very different. The emetic doses of tartar emetic is 30mg, which is dangerously close to the toxic dose of 130mg/l, although if vomiting occurs, there is no danger (Gleason, 1979). The therapeutic value of drugs is, of course connected with their toxicity.

Pollutants can accelerate the deterioration of materials and construction. Air pollutants, particularly sulphur dioxide gas and the sulphuric acid aerosols into which the gas is converted in the atmosphere, can corrode metals and building materials, increasing the frequency of repair and replacement. In estimation damage is being caused to frescoes in Italy by sulphur dioxide, which converts the pigmented lime plaster to gypsum, thereby increasing its volume and reducing its cohesiveness (Thompson, 1985). Water pollutants, such as suspended particles or dissolved inorganic compounds, can also adversely affect pumps, industrial equipment and bridges.

1.2.3. LEAD AS A MAJOR POLLUTANTS

Lead, the toxic metal known to be harmful to man, has been sneaking unnoticed into the bloodstream of millions of people over many years. It seeps insidiously into our water risking the lives of our unborn children, often going unnoticed until its too late. It enters our homes on the feet of every occupant, picked up from the soil outside. It can cover the work clothes of mechanism, plumbers, lead smelter workers, and other high risk occupations. The same clothes that we wash along with our children's clothes, unknowingly endangering them.

Childhood lead poisoning is the number one environmental health risk for children today. In human population, more than three million

children age six and younger that's one out of six –already have toxic levels of leads in their bodies.

Lead interferes with the development and functioning of almost all body organs and retards the development of the central nervous system and brain. Lead is sometimes called, "brain poison".

Even tiny amounts of lead can cause reduced IQ, reading and learning disabilities, attention deficit disorder and behavioural problems. As a result, lead poisoning is associated with lower educational achievement, higher school drop-out rates, and increased delinquency. It is estimated that lead poisoning has tripled the number of children needing special education.

80% of childhood lead poisoning occurs at home, many homeowners are not aware of hazards associated with lead-based paint and unknowingly poison their own children by not following safe work practices during renovation or by not attending or deteriorating and/or chipping paint.

While it is true that many kids get poisoned by eating paint chips-they taste sweet most children are poisoned by invisible lad dust created when lead paint deteriorates from age, is exposed to the elements, is damaged by water, kids exposed by friction (such as the opening and closing of a door or window), or during home renovation.

In Adults – most adults are poisoned at work, there are laws that seek to prevent this, but many are not yet widely enforced. Any employee,

who maybe exposed to lead in any amount, should have personal air sampling done.

According to centers for disease control, childhood lead poisoning is the number one environmental health risk facing children in industrialized countries today.

Lead is an invisible enemy, often in the form of sample dust that enters our home. Dust that can be both inhaled or ingested especially by young children who tend to put everything in their mouths.

In other words, lead poisoning is still a very real threat present in our environment, damaging the brains and nervous system of many of its victims the majority of which are children.

1.3 FOOD AND LEAD POISONING

Food poisoning is a non-specific term often applied to the syndrome of acute anorexia, nausea, vomiting, or diarrhea, which is attributed to food intake, particularly if it afflicts groups of persons and is not accompanied by fever. The actual cause of such acute gastrointestinal upsets might be emotional stress viral or bacterial infections, food intolerance, or inorganic (e.g. sodium nitrite) or organic (e.g. mushroom, shell fish) poisons, more specially, food poisoning may refer to toxins produced by bacteria growing in food (staiphylococci, clostridia) or to acute food infections with short incubation periods and a mild course (salmonella gastroenteritis, vibrio parahaemolyticus infection, infection with certain stereotypes of

escherichia coli and occasionally shigellosisor cholera). In general, the diagnosis must be suspected when groups of persons with a common food intake develop acute vomiting or diarrhea. Food and stools must be secured for bacteriological and toxicological examination.

Treatment of food poisoning usually consists of replacement of fluids and electrolytes, and very rarely, the management of hypovolemic shock and respiratory embarrassment. If botulism is suspected, polyvalent antitoxin must be administered.

Lead poisoning may occur by ingestion or inhalation of lead dust or fumes. Poisoning is manifested by a metallic taste, anorexia, irritability, apathy abdominal colic, vomiting, diarrhea, constipation, headache, leg cramps, black stools (Lead sulphide), oligura, stupor, convulsions, palsies, and coma. Chronic lead poisoning causes variable involvement of the central nervous system (CNS), the blood –forming organs, and the gastrointestinal tract.

The largest proportion of lead intake is through food. The lead concentrations in food are from many sources. In 1986 the acceptable level of lead in food was 2mg/kg body weight, with discussions of lowering it to mg/kg body weight.

1. Lead in the air: lead from the atmosphere or auto emissions may settle directly on agricultural products, vegetables such as wheat

corn, and spinach accumulate an average of .0015ug/g, that can not be washed of by rain

2. Lead in soil: lead from the air can settle on the soil itself, or the soil can receive lead arsenate, an inorganic pesticides lead will then be taken up plant roots like carrots or potatoes which accumulates an average of .0045ug/g. this may be used as livestock fodder or eaten directly by man. Lead in the air can also settle in the water and be taken up by seafood, such as tuna, which is then eaten by man.
3. Lead from preparation process: food can absorb lead from pans, utensils or contaminated water used in the cooking process. Additionally, low-fired lead glazeled ceramics may be used in the storage or preparation of foods.
4. Lead from dining process: lead can also be ingested from food eaten from dishes such as ceramics ware. It can also be ingested by drinking from leaded glassware, because acidic liquids may leach lead from the glassware into the liquid.
5. Lead in packaging: since the 1920's can have been produced by rolling a tin-plate into a cylinder and soldering the edges together, the scolder and the tin both contain lead.

Nevertheless, an investigation in Oakland found a store selling foods in lead-soldered cans, and some imported foods are still sold in soldered cans. Other packaging can also contain lead, one study found lead in the

coloured pigments used for labels and decorations on soft plastics used in wrapping foods. A lack of education regarding lead in food possesses a problem, especially for pregnant or lactating women.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 REVIEW OF LEAD DETERMINATION

Lead can be determined in the most widely varying flames without noticeable interferences occurring. Usually an air/acetylene flame is used, although a number of authors prefer the air/propane flame. Dagnal and West (1964) found minor interference from aluminum, beryllium, zirconium, phosphate and sulphate in this flame, but these could be largely eliminated by adding EDTA.

The most important resonance lines of lead are listed in the table below.

Although the 217.0mm line is more sensitive than the 283.3nm line, it does not give a better detection limit owing to a poorer signal – to noise ratio, furthermore, more background attention effects occur at the 217.0nm line, so that the 283nm line is used with greater success.

Wave length (nm)	Energy Level (k)	Characteristics Concentration (mg/11%
217.0	0-46069	0.08
283.3	0-35287	0.2
261.4	7819-46061	5
368.4	7819-34960	17
364.0	7819-35287	40

Issaq and Zielinski (1974) found that when dilute aqueous solutions containing lead were stored in glass or polyethylene vessls, substantial

losses occurred within a relatively short time. Acidification with nitric acid prevents these losses.

Since Lead must often be determined in very low concentrations; the detection limit of about 0.01mg/l in the flame is often not adequate. Earlier on, the boat techniques, Kahn et al, (1968) or the Delves system (1970 were applied to increase the sensitivity. The real breakthrough came with the graphite furnace technique and this used principally nowadays for the determination of lead.

Early publications on the determination of lead in various samples reported numerous interferences, both spectral and non-spectral, which in part were dependent on the volatility of lead.

Ottaway (1976) reported considerable interference due to chlorides, simultaneously with the occurrence of signal depression, he observed the spectrum of lead and thus concluded that lead is volatilized in molecular form.

He also observed an interference when lead and magnesium chloride were placed at different locations in the graphite tube, thus, indicating a vapour phase interference. Henn (1977)found substantial interferences through 5% hydrochloric acid; he was able to reduce this effectively by adding molybdenum to the sample and reference solutions. Vickrey et al (1980) found a similar improvement when using zirconium coated graphite tubes.

Behne et al (1975) reported strong signal depression due to perchloric acid; this acid also attacks the graphite tube and thus cannot be used. Nitric acid also interfered in the determination of lead; these authors removed it from the sample solution with formic acid. Julshamn (1977) also found interference due to perchloric acid. Wegscheider et al (1977) reported that all acids depressed the signal for lead except phosphoric acid, which caused signal enhancement. Hodges and Skelding (1981) found that matrix interferences on the determination of lead in urine were lowest when orthophosphoric acid, was added to the sample solution and the graphite tube as conditioned with morhybdenum.

Frech and Cedergren (1975) employed high temperature equilibia equations to examine the interferences and found that chloride interferences were distinctly lower in the presence of hydrogen. These investigations were applied to steel analysis, and would appear that iron plays an important role. In the presence of sodium chloride, hydrogen did not influence the interference adequately. Other authors verified the positive influence of hydrogen during the analysis of steel Maessen et al, (1978) and geological samples Heinrichs (1979).

A solution to the problem was first found when the influence of the graphite surface and matrix modification were thoroughly investigated then atomized off the L'vov platform and found that the majority of the

interference reported in the literature no longer occurred (Manning and Stavin 1978).

Hinderberger et al (1981) verified that lead could be determined free of interference in numerous biological material, including urine by using primary ammonium phosphate as matrix modifier and atomizing off the L'voc platform.

Under these condition, thermal pre treatment temperature up to 950^{o}C can be applied, magnesium nitrate as matrix modifier permits some at higher temperatures. The optimum temperature for atomization off the L'vov platform is 1900^{o}C.

Lead can also be determined by the hydride technique but the sensitivity is not especially good. For atomization in a heated quartz tube, Thompson and Thomerson (1974) obtained a detection limit of 0.1mg/l. Fleming and Ide (1976) were able to increase the sensitivity significantly by adding tartaric acid and potassium dichromate, but they reported that copper and nickel caused severe interferences. Vijan and Sadana (1980) used the hydride technique to determine lead in drinking water after co-precipitation with manganese dioxide.

Many authors have described the combination of chromatographic technique and Atomic Absorption Spectrophotometric techniques to determine organolead compound in petroleum (gasoline) (Segal, 1974; Botre et al,1976; Coker, 1975; Dejeonghe et al, 1980; Robinson, et al,

1977 and in air (Chau, et al 1976), Sirota and Uthe (1977) determined tertraikl lead in tissue after extraction.

Brimheall (1969) and Kirchoff (1969) reported a possible method for the determination of three lead isotopes, 206, 207, and 208 by atomic absorption spectrophotometry, and the construction of isotope hollow cathode lamps.

2.2.1 LEAD EXPOSURE AND HUMAN HEALTH

Lead is one of a limited class of elements that can be described as purely toxic, many other elements including heavy metals such as chromium, manganese, molybdenum, nickel, and selenium, although toxic at high levels, is actually required nutrients at lower levels. This is clearly not the case for lead. In many decades of research, no nutritional value or positive biological effect has been shown to result from lead exposure. Also no case of lead deficiency has ever been noted in the medical literature.

Of all the toxic chemicals known to humanity, lead is probably the most thoroughly studied. The toxicity of lead at high and moderate doses has been known for milliennia, toxicity at lower doses has received perhaps more toxicological and epidemiological attention than any other agent.

Studies on the toxicity of lead can be grouped into three broad categories:

- studies of worker exposure and of populations known to have been exposed to elevated lead levels.
- Epidemiological studies in the general population,
- Animal studies investigating mechanisms of toxicity.

These three types of studies have yielded consistent conclusions about the toxicity of lead.

Occupational studies of lead poisoning are perhaps the most numerous, this reflects not only the widespread use of lead for many different industrial purposes, but also the absence of effective exposure controls for workers in many industrial settings. Sadly, this is not merely a historical problem, cases of severe lead poisoning in workers continue to occur in the international medical literature.

Where single large industrial facility or many small industries, lacking appropriate controls for environmental emissions, are located close to residences, local populations are found who have elevated body burdens of lead. These exposed population studies are probably the second most numerous type of investigation in the toxicological literature, after occupational studies, Studies of populations with known exposures to lead have not resulted in finding a safe exposure level. This realization has led in this century to epidemiological studies addressing lead exposures and effects in the general population and particularly in children. The initial studies predominantly looked at across sections of the population, that is

people with a range of a body burdens at a particular point in time. These have been supplemented in recent years by a number of long-range (Longitudinal) studies that examined lead body burdens and toxicity in a defined population over several years.

With human studies, it is extremely difficult to assess exposure accurately and impossible to assess causes. Animal studies are therefore used to identify the mechanisms by which lead is toxic. Findings from these studies have proven consistent with data from human studies, although many experimental animal appear to be less sensitive than humans to the toxicity of lead. For some effects, this may reflect differences in the sensitivity of the test methods employed.

Lead has been demonstrated to be toxic to a wide variety of organs in both humans and experimental animals. The organ systems tat have been shown to be most sensitive to low level exposures of lead are :

- nervous system: changes in neurotransmitter levels
- biochemistry : impairment of vitamin D metabolism
- reproductive system: irregular uterus and decreased sexual hormone levels.
- Immune system: impaired lymphocyte function and impaired antibody formation.
- Gastrointestinal: colic (severe cramping and nausea, characteristic of high dose poisoning)

- Reproductive: decreased gestation duration and decreased growth rate in offspring.

There is also clear evidence that lead can cause cancer in experimental animals. Thus, the US Environmental Prtection: Agency has classified as a “probable human carcinogen” (Group B2), and the international Agency for research on cancer has deemed it possibly carcinogenic to humans” (Group 2B). The characteristics tumor observed in animals’ has been a bilateral renal carcinoma although other sites and tumor observed. while the cancers observed in animals maybe secondary to other forms of toxicity, there is also evidence for a direct effect of lead on genetic material. Data from studies of worker exposure have been inconsistent with regard to cancer.

Because lead has widely used for millennia, it is widely distributed in the environment and can be detected globally in air, soil, and water. Because there is no level of lead exposure tat appears to be necessary or beneficial, considerable research interest has focused on the question o whether there is any level of exposure that is harmless, and if so, what that level might be. Various types of effect have been noted at different levels of exposure, in a number of cases, variations in study design and the pollution studied have led to slightly different conclusions.

One consequences of the extensive investigation of the toxic effects of lead, using increasingly sensitive measurements, has been the

development of a scientific consensus that there is no demonstrable threshold dose for the manifestations of lead toxicity. In other words, there is no exposure level below which lead appears to be safe. If a safe level exists, current testing techniques are not yet sensitive enough to find it. This distinguishes lead from most chemicals that causes non-cancer toxic effects, where the consensus is that at some non-zero exposure, effects are negligible. The US EPA concluded:

It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood levels so low as to be essentially without a threshold.

On this basis, EPA declined to specify a reference dose (that is, a level of exposure not likely to lead to adverse effects) for lead. Similarly, the US Agency for toxic substances and diseases registry (ATSDR) has not developed minimum risk levels for lead, because no threshold have been demonstrated for the most sensitive effects in humans, "for lead, therefore, any exposure is of potential concern.

The extensive history of lead use, however, also means that lead is ubiquitous. This widely and so long use of it now appears essentially "everywhere". In combination with the lack of evidence for a safe threshold and the limited sensitivity of the techniques employed to study lead, this means that the toxicologist is always faced with the problems not of

determining whether any exposure will lead to an effect, but whether the effect, quantitatively and qualitatively, will be deemed significant in a particular context. Key areas where international scientific consensus has yet developed relate to the exposures at which effects in various organ systems begin to affects human health and become medically significant.

One area of scientific agreement is that children are uniquely susceptible to the effects of lead. Indeed, children appear to be more sensitive to lead than adults in all areas except kidney function. The most critical sphere for children is the potential for damage to their intellectual and behavioural development.

This appears to reflect three key factors:

- young children behave in ways that increase their exposure.
- Children absorb more lead for a given exposure than do adult
- Developing organisms are inherently more susceptible than mature one.

Between the ages of one and three children engage in “oral exploration” of their environment, placing non-food objects in their mouths. They also put their hands in their mouths frequently. This normal behaviour can lead to ingestion of lead contaminated soil and dust. Some children exhibit “pica” behaviour, an abnormal or extreme tendency to eat non-food materials, which can led to even greater ingestion of contaminants.

Children also have a higher inhalation rate, relative to their body mass, than do adults. This leads to greater exposure to airborne contaminants.

It has been estimated that for a given oral dose of lead, children absorb three times more than adults. Of this absorbed dose, children are estimated to contain six times as much as adult do.

Lead is not the only toxic agent to show harmful effects on the nervous system in children at much lower exposure levels than for adults. One possible factor is that the so-called “blood-brain barrier”, which protects the adult brain from toxic agents to some degree, is not fully developed in children. Another is that the nervous system undergoes tremendous development of intercellular connections, and dying back of excess connections. Interference by a toxic agent with these developmental events would be expected to have grave and permanent influences on the ultimate functioning of the nervous system.

Susceptibility to the toxicity of lead also shows an interaction with nutritional levels. This result is that children with inadequate diets are most likely to be adversely affected by lead exposure. In part, this may reflect competition between lead and calcium for absorption, so that diets low in calcium allow greater lead absorption. The interaction of toxicity with nutritional status means that in developed nations lead toxicity may be primarily a problem for the urban poor. In those developing nations where

nutrient deficiencies are a problem, lead contamination of the environment will have more devastating effects than would otherwise be the case.

Exposure to lead for women of childbearing age can have adverse effects on their offspring, should they become pregnant. Lead that is not excreted from the body is eventually sequestered in bone, almost the entire body burden of lead is carried in bone, rather than blood or other tissues. This lead can be mobilized from bone during pregnancy. Lead also transfers readily from the mother to the fetus via the placenta (umbilical cord blood lead levels are typically very close to maternal blood lead levels). Thus, lead exposure in a pregnant woman's history, even in her own childhood, may effect her children. Although the past exposure may not be manifested in adverse effects on a woman herself (because the lead has been sequestered in bone), that exposure may pose a risk to her future offspring.

Given the scientific community's inability to delineate a safe level of exposure to lead, it is perhaps not surprising that regulations and guidelines controlling exposure to lead vary considerably among nations and between national governments and international bodies. Some regulatory or advisory values appear to reflect earlier scientific evaluations, while others are more in accord with more recent evidence indicating toxic effects from lead at doses one considered safe. In general, these

standards have decreased steadily through this century as more of lead's toxic potentials has become known.

Regulations and guidelines typically set forth a "standard" that represents a maximum acceptable concentration of lead in blood, air, water, soil, sediment, foods, etc. concentrations of lead that exceed such a standard lead to efforts to control or rectify the corresponding pollution or to sanctions against a polluting facility. OECD had published information on lead standards (or guidelines) promulgated by individual nations, primarily member states (36). More recently, the International Lead and Zinc study group (ILZSG) has compiled and published similar data. Various other documents contain information on standards for individual nations. In all cases where data were directly available from government publications, those official values were used in this report. (in two cases, data from the respective governments differed significantly from the ILZSG document).

As noted above, most toxicology studies rely on blood lead level as the measure of exposure. This means that blood lead levels measured in population can be compared directly to toxicity studies. (In contrast, when lead is measured in air, soil, food, etc the results must first be extrapolated to corresponding blood levels, before assessment of potential effects.

Official statements on limits on blood lead for the general population (as opposed to workers) ahs been made by several member countries of the OECD.

In Australia, the National Health and Medical research council set a national goal for all Australians to have blood lead than ug/l (except for occupational exposures). Health Canada has established a guideline of 10ug/dl,while Germany has a set guideline of 15ug/dl in adults, 10ug/dl in children and women of childbearing age. Switzerland has stated officially that level as low as 10-15ug/dl may have detrimental effect on a fetus, while prolonged levels above 10ug/dl are detrimental to children. In the United States, the Centers for Disease Control (CDC) have specified a level of 10ug/dl as an action level. For blood lead in children, noting that this level maybe associated with a 2.5 point decrease in intelligence (IQ). In general, these standards have been promulgated fairly recently, and they reflect increasing concern over exposures to lead that were once thought safe. Thus, for example, the Australian goal was set in 1993, while Canada's value was lowered from 25 to 10ug/dl in 1994. the United State CDC revised its value in 1991, it had previously been set as 25ug/dl.

2.2.2 ACTION MECHANISM

Lead's toxicity largely due to its capacity to mimic calcium and substitute it in many of the fundamental cellular processes that depend on calcium. Lead can cross the cell membrane in various ways, which are not

yet entirely understood. Lead transport through the erythrocyte membrane is mediated by the anion exchanger in one direction and by the Ca-AtPase pumping the other. In other tissues, lead permeates the cell membrane through voltage-dependent or other retypes of calcium channels.

One it has penetrated the cytoplasm, lead continues its destructive mimicking action by occupying the calcium binding sites on numerous calcium dependent proteins. Lead binds to calmodulin, a protein that in the synaptic terminal acts as sensor of free calcium concentration and as a mediator of neurotransmitter release. Furthermore, it alters the functioning of the enzyme protein kinase C, a virtually ubiquitous protein which is of crucial importance in numerous physiological functions. Kinase C is normally activated by modulators outside the cell (hormone, neurotransmitters, etc) through an enzyme chain and in calcium dependent manner. Besides many other functions, the activated kinase directly affects the expression of the Immediate Early Response Genes (IERG). Lead has high affinity for the sites, which are typical calcium-binding sites in this protein, pico molar doses can take the place of micro molar calcium doses. In model cell systems it has been demonstrated that lead can stimulate gene expression through a mechanism mediated by protein kinase C and it is postulated that this effect may be correlated with alterations in synaptic functioning.

2.3 THEORY OF ATOMIC ABSORPTION SPECTROPHOTOMETRY

Atomic Absorption Spectrophotometry (AAS) is a classical technique for the determination of metals that are dissolved or suspended in a solution. The solution can be pure water, dilute or concentrated and or organic solvents. The metals can be Alkalis, Alkaline earths, Transmition metals, precious metals, heavy metals, semi-metals and even some refractory metals.

The basic method is quite simple and airily rapid allowing trace metals in the fractional parts –per million range and major components in the percentage range to be determined accurately and precisely.

Atomic Absorption Spectrophotometry (AAS) is the measurement of the absorption of optical radiation by atoms in the gaseous sate and it is based on the ability of an “excited” atom of an element to absorb energy from wavelengths of lights of the same frequency as the element. This creates a decrease in the initial signal energy, and this difference is proportional to the concentration.

Each element has it own series of specific resonance wavelengths, these wavelengths will have specific characteristics for sensitivity, noise and linearity, sensitivity and noise will determine the limit of detection for that element. Linearity will define the range in which the calibration curve will be accurate.

The optical system is set up with a Hollow Cathode Lamp for the element of interest (Analyte, with the appropriate slit and wavelengths selected for this element. A solution of a known concentration of the analyte (standard) is aspirated and the absorbance reading noted. An unknown sample is aspirated, the absorbance reading is ratioed to the standard and a sample concentrated is interpolated from the intensity of the reading.

The unique design of the Buck Model 210-VGP system which is used in this research provides superior sensitivity and stability for analytical applications in many areas of science which include agriculture, food technology, geochemistry, clinical chemistry, petroleum and polymer science, metallurgy, toxicology, biochemistry and ecology.

The Buck Model 210-VGP system can operate in 3 modes: direct measurement, deuterium background correction (D2) and variable Giant Pulse correction (VGP). The two correction modes allow the chemist to analyze samples that have complex or “dirty” matrices which would normally give significant interferences in the analytical determination.

The Deuterium Background Correction mode (D2) employs a quartz lamp filled with deuterium gas that has a “continuum” of radiation from the far ultra violet (<190mmm) to appropriately 350mm, and this constant level of energy can be used to establish a “reference” bam. This reference beam can be used to correct for non-structured, molecular interferences (non

atomic absorptions) and physical (flame) interferences in complex sample matrices. D2 correction is only usable on the UV elements, and may degrade sensitivity significantly if not properly used.

The variable Giant Pulse Correction mode (VGP) uses a time-specific modulation of the Hollow Cathode Lamp to produce an energy "pulse" that contains information on both the sample (Analyte) absorbance and the background (matrix) absorbance. The benefit of VGP correction is that it is usable throughout the spectral range and will correct for additional interferences (structured backgrounds) that D2 will not correct.

2.4 OBJECTIVE AND SCOPE OF THE RESEARCH

Lead serves no useful purpose in human body and its presence in the body can lead to toxic effects, regardless of exposure pathway. Lead toxicity can affect every organ system. On molecular level, proposed mechanisms for toxicity involved fundamental biochemical processes. It includes ability to inhibit or mimic the actions of calcium and to interact with proteins (including those with sulphdryl, amino, phosphate, and carboxyl groups) (ATSDR 1999).

The largest proportion of lead intake is through food. The lead concentration in food is from many sources. In 1986 the acceptable level of lead in food was 2mg/kg body weight.

Food can absorb lead from pans, utensils or contaminated water used in cooking process. It can also be ingested from food eaten from dishes such as ceramics ware.

Lead poisoning is manifested by Anorexia, irritability, abdominal colic, vomiting, diarrhea, constipation, headache, leg crams, black stool, and convulsion etc. chronic lead poisoning causes variable involvement of the central nervous system. Bearing in mind of the above mentioned, it is justifiable to carry out this research so as to ascertain level of Health risk posed by these food sold by Hawkers, most especially the nature of how these food are hawked in locations covered in this research. These areas are densely populated by illiterate and semi-illiterate people who do not know the health implication of eating openly in these areas. Likewise, the hawkers know little or nothing about the danger of this metal, therefore, the objectives of the study are to,

a. determine the lead content (if any) of some foods sold by hawkers
b. discuss the health risk of the people consuming such foods.
c. Compare the lead contents of the same food at the different locations.

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1.1 CHEMICALS AND REAGENTS

All experiments were performed using analytical grade chemicals obtained from British Drug House (BDH). Glass distilled water was used throughout the study. Official method B.P. 1980 was used for the identification of lad (United State Pharmacopoeia, Identification of metals (1980). Stock solution of lead was prepared by dissolving 1.598g of lead nitrate in 50ml of 2M nitric acid and the solution was made to volume in 1 litre volumetric flask with demonized water to make 1000mg/l Pb solution.

Standard working solutions were prepared by suitably diluting the stock solutions. Other reagents used include the following:

- Ammonium pyrrolidine dithiocarbanate (APDC)
- Methyl isobutyl ketone (MIBK) S.G. 1.18
- Hydrochloric acid (HCL)
- Trioxonitrate (v) acid (HNO3) S.G. 1.41 – low in lead for analysis
- Lead Nitrate (pbNO3) (Analytical grade)
- Deionised distilled water
- Perchloric acid
- Hydrofluoric acid

3.1.2 EQUIPMENT AND GLASSWARE

- Analytical balance, Mettler AE240, Mettler Instruments Limited, high Wycombe, HP11, 1QW, Switzerland.
- Oven BS, Model OV – 330, Gallenkamp, England
- PH meter – model HL 7060, Electronic Instrument LTd, USA
- Hot plates, model HL 054, Gallenkamp, England
- Nickel Crucible
- Laboratory Grinder
- Desiccators
- Water bath, model BJE 750A, Gallenkamp England
- Quartz crucible with lid
- Volumetric flasks : 25,50,200,250,1000,2000ml
- Beakers: 50, 250, 500, 1000, 2000ml
- Measuring cylinders 5,10,50,100,500,1000ml
- Pipettes : 1,2,5,10,25ml
- Separating funnel : 250, 500ml
- Mechanical flask shaker, Gallenkamp, England
- Aluminuim foil and filter paper – whatmann No.
- Conical flasks
- Dropper
- Polyethylene sample bottles
- Polyethylene bags

- Spatula
- Fume cupboard
- Retort stand

3.1.3 INSTRUMENTATION

The buck model 210 variant giant pulses correction (VGP) system Atomic Absorption Spectrophotometry (AAS) was used with the following conditions:

Lamp: Lead Hollow Cathode

Source/Lamp current: 10m A

Wavelength: 217.0nm

Band Pass: 0.5nm

Flame type : Air/Acetylene

Stochiometric fuel flow; 0.9-1.21min-1

3.2 LOCATION OF STUDY AREAS

The study areas comprises of Zaria city, Motor Park, Kofan Doka Motor Park, Tudun Wada Motor Park (Agwaro) all in Zaria Local government Area. Other locations are Sabon Gari main park, Kwangila (under Bridge), ABU North-Gate Samaru, Funtua Garage motor park and samaru main market in Sabon Gari Local Government Areas of Kaduna State. These locations are coded as follows:

A1 - Zaria City Motor Park
B1 - Kofan Doka Motor Park
C1 - Tudun Wada Motor Park

D1 - Sabon Gari Main Motor park

E1 - Kwangila (under the bridge)

F1 - ABU North-Gate (Samaru)

G1 - Funtua Garage (Samaru)

H1 - Samaru Main Market

Similarly seven different foods were used:

FOOD CODE

A - Bean Cake

B - Jollof Rice

C - Wara (Soya beans cheese

D - Boiled yam

E - Beans

F - Fried yam

G - Rice and Beans

3.3.1 PRE TREATMENT OF THE SAMPLE

The food samples were dried in an oven at a temperature of 105^{o}C + 0.5^{o}C for one hour and crushed to powdered form. Beakers were thoroughly washed, oven dried and cooled. The weight of the beakers were recorded after which 1g of each of the samples were added into series of weighted beakers and labeled accordingly.

3.3.2 SAMPLE COLLECTION AND PRESERVATION

STRATIFIED SAMPLE COLLECTION

The sample collection method as described by Harper (1976) and Elwood (1982) popularly referred to as stratified sample collection, was adopted and modified for the purpose of this study. The application of standard randomized controlled trial sampling was also extended to this study to achieve an optimal level of success in the sample collection procedure. At the collection site, the followings were recorded: name of sample, time of collection, date of collection and place of collection. Samples were collected in the month of April 2005 and sample analysis started in June 2005 which ended in August 2005.

3.3.3 SAMPLING OF FOOD

Seven different food samples were collected as in 3.2 but not all seven (7) samples were collected at a particular location instead, a particular food sample was collected from all the eight locations. That is moi-moi (labeled A) was collected on the same day from all the location followed by jollof rice (labeled B) in that order.

3.4 PREPARATION OF CALIBRATION CURVE VALIDATION OF METHOD

Five working standard lead solution of concentrations 0.04,0.08,0.12,0.16 and 0.20mg/l were prepared so as to obtain a calibration curve. 100ml of each of this standard solutions was adjusted to pH of 2.5 by adding 1.0ml of 1m Trioxonitrate (v) acid (HN03). Each

standard solution and blank was transferred into an individual 250ml separatory funnel. 1ml ammonium pyrrolidine dithiocaramate was added and the solution was shaken vigorously for 2minutes. The separatory funnel was allowed to stand to let it separate into aqueous and organic layers. The aqueous layer was drained off and discarded. The organic layer was then drained into a 10ml glass stoppered graduated cylinder. The organic extract was aspirated directly into the flame (zeroing the instrument on methyl isobutyl ketone) and the absorbance was recorded.

3.5.1 SAMPLE DIGESTION

The beakers were then left on series of hot plates with low heat to about dryness. The dried beakers were then cooled in dessicator and the weight recorded. The differences in the weight were recorded and the residue removed from the beaker.

Triacid digestion method of analysis was employed using nitric acid,(after checking the limit test of the solvent) perchloric acid and hydrofluoric acid mixture. 0.2 of each pre-treated sample was treated with 5ml of deionised water to dampen the sample,6ml of concentrated nitric acid were then added, followed by 1ml of perchloric acid and heated on a water bath to the appearance of white fumes. 5ml of hydrofluoric acid was added after cooling and the resulting mixture boiled 10min, this was filtered and made up to mark with deionised water in a 100ml volumetric flask. the sample solution were then analyzed as described under preparation for calibration curve. The concentration of lead from each sample was determined from the calibration curve.

3.5.2 ANALYSIS OF LEAD IN FOOD

Analysis of the samples for the lead metal contents was carried out in duplicate on the AAS in accordance with “standard methods

(Apha,1990) and Perkin Elmer (1994). 100ml of this sample solution was adjusted to pH of between 2.2 to 2.8 by adding 1.0ml of 1m Trioxonitrate(v) acid (HN03). Each of these sample solution were transferred into an individual 250ml separatory funnel.

1.0ml of Ammonium pyrrolidine dithiocabamate was added to te solutions followed by the addition of 10ml methyl isobutyl ketone. The solutions were shaken vigorously for 2 minutes and the separatory funnel was allowed to stand to let it separate into aqueous and organic layers. The aqueous layer was drained off and discarded. The organic layer was then drained into a 10ml glass stopered graduated cylinder. The organic extract was aspirated directly into the flame (zeroing the instrument on methyl isobutyl ketone) and the absorbance were recorded. The nebulizer, atomizer and burner were flushed each time with distilled water after each sample solution was aspirated before the next. The instruments stability was also checked at intervals by introducing the highest working standard solution and the blank. The concentration of each sample was determined from the calibration curve subtracting from the blank where necessary.

CHAPTER FOUR

4.0 RESULT

4.1 LEAD CONCENTRATION IN FOOD SAMPLES

The results obtained for levels of lead (mg/L) in different types of food, from eight sampling locations are shown in tables 1- 7. The mean lead Concentrations for each location are also indicated. The summary of mean concentration of lead at different locations and for different food samples was shown in table eight (8).

TABLE 1 : CONCENTRATION OF LEAD IN MOI-MOI

Location	A1	B1	C1	D1	E1	F1	G1	H1
Concentration mg/L	0.080	0.100	0.130	0.040	0.160	0.030	ND	ND

Mean x = 0.0675mg/L

ND = Not Detected

TABLE 2 : CONCENTRATION OF LEAD IN JOLLOF RICE

Location	A1	B1	C1	D1	E1	F1	G1	H1
Concentration mg/L	0.020	ND	ND	0.060	0.180	0.020	ND	0.070

Mean x = 0.0043mg/L

ND = Not Detected

TABLE 3: CONCENTRATION OF LEAD IN WARA (SOYA BEAN CHEESE)

Location	A1	B1	C1	D1	E1	F1	G1	H1
Concentration mg/L	ND	ND	0.010	0.030	ND	ND	0.220	ND

Mean x = 0.0326mg/L

ND = Not Detected

TABLE 4 : CONCENTRATION OF LEAD IN BOILED YAM

Location	A1	B1	C1	D1	E1	F1	G1	H1
Concentration mg/L	0.090	0.155	ND	0.040	0.040	0.134	0.100	0.005

Mean x = 0.0655mg/L

ND = Not Detected

TABLE 5 : CONCENTRATION OF LEAD IN BOILED BEANS

Location	A1	B1	C1	D1	E1	F1	G1	H1
Concentration mg/L	ND	ND	ND	ND	0.020	0.035	ND	ND

Mean x = 0.0069mg/L

ND = Not Detected

TABLE 6 : CONCENTRATION OF LEAD IN FRIED YAM

Location	A1	B1	C1	D1	E1	F1	G1	H1
Concentration mg/L	0.116	0.121	0.124	0.122	0.113	0.118	0.119	0.124

Mean x = 0.1196mg/L

TABLE 7 : CONCENTRATION OF LEAD IN FRIED YAM

Location	A1	B1	C1	D1	E1	F1	G1	H1
Concentration mg/L	0.100	0.118	0.125	0.121	0.130	0.196	0.149	0.157

Mean x = 0.0370mg/L

TABLE 8 : Summary of lead concentration mg/L of all food samples (A – G)at different locations.

	Bean Cake **A**	Jollof Rice **B**	Wara S/Bean Cheese **C**	Boiled Yam **D**	Boiled Beans **E**	Fried Yam **F**	Rice & Beans **G**	Mean ± S.E.M
Zaria City	0.08	0.02	ND	0.09	ND	0.12	0,01	0.06 ± 0.019
Kofan Doka	0.10	ND	ND	0.16	ND	0.12	0.12	0.07 ± 0.026
Tudun Wada	0.13	ND	0.010	ND	ND	0.12	0.13	0.06 ± 0.025
Sabon Gari	0.04	0.06	0.030	0.04	0.02	0.12	0.12	0.06 ± 0.017
Kwangila	0.16	0.18	ND	0.04	0.04	0.11	0.13	0.09 ± 0.027
ABU Northgate	0.03	0.02	ND	0.13	ND	0.12	0.120	0.08± 0.028
Funtua Garage	ND	ND	0.220	0.10	ND	0.12	0.15	0.08 ± 0.033
Samaru M/Mkt	ND	0.07	ND	0.01	0.008	0.12	0.16	0.05 ± 0.025
Mean ± S.E.M	0.07 ± 0.02	0.04 ± 0.02	0.03 ± 0.03	0.07 ± 0.05	0.008 ± 0.005	0.12 ± 0.001	0.14 ± 0.01	

CHAPTER FIVE

5.0 DISCUSSION AND CONCLUSION

5.1 MEAN CONCENTRATION OF LEAD IN BEAN-CAKE (MOI-MOI).

From table 1, it is very easy to see the concentration of lead in bean-cake at various locations, with a mean concentration of lead at 0.07mg/L$\pm$ 0.02 (X$\pm$ SEM). But the concentration of lead at Kwangila under the bridge is the highest (0.160mg/L). This can be attributed to the fact that, hawkers sold the bean-cake (moi-moi) open without cover as found in some locations. The place (under bridge) is characterized as densely populated with a lot of vehicles and it also serves as a motor park for people going to places far and near. The emissions from exhaust of vehicles, the dust due to movements of vehicles, can probably lead to this high concentration in this particular location. Similarly, ABU North-gate recorded the lowest lead concentration of Bean-cake (moi-moi) of 0.030mg/L. here, the hawkers do sell the Bean-cake in a transparent plastic container with cover and the vehicles around this place is very small compared to those other locations. This explains why the concentration of lead in the location is very low since their sources of lead are relatively small.

MEAN LEAD CONCENTRATION OF JOLLOF RICE

The mean concentration of lead at various locations for jollof rice is 0.04mg/L (0.04$\pm$ 0.2). The highest concentration of lead was recorded at

E_1 (0.18mg/L) and lowest at A_1 and F_1 (0.02mg/L). Location E_1 is a very busy place with a lots of human activities like mechanics, sprayers, welders, battery worker etc. this could attribute to the high value.

At location B_1 C_1 and G_1 lead was detected because of the way and manner food is been hawked. At these locations the foods were covered completely with transparent polythene bag and the hawkers were in one place.

MEAN LEAD CONCENTRATION OF FRIED CHEESE "WARA"

The mean $\pm$ SEM lead concentration for this type of food is (0.03 $\pm$ 0.03)mg/L. Lead was detected in these locations with highest value at location G_1 (0.22mg/L). The high value at this location was probably due to the manner in which the food was sold. It was placed in an open tray, uncovered and the hawkers move about from one place to another. Another reason that could amount to this high value was in the preparation processes.

MEAN LEAD CONCENTRATION (mg/L) OF BOILED YAM

The mean $\pm$ SEM (0.07 $\pm$ 0.05) was recorded for this food, lead was not detected at C_1 but recorded in all other locations. B_1 recorded the highest (0.16mg/L) and H_1 the lowest (0.01mg/L). D_1 and E_1 recorded the same value. This might be due to the same conditions in which the foods

were found at these locations. The foods were completely covered with black polythene bag in a bowl.

MEAN LEAD CONCENTRATION (mg/L) OF BOILED BEANS

Lead was detected only in three locations and at very low concentrations compared to other foods. The highest value was (0.04mg/L) at location E_1 and the lowest of all foods and locations was (0.008mg/L) at H_1. The lowest mean $\pm$ SEM of all food and locations was recorded in this food (0.008$\pm$ 0.005). The preparation of this food was simple and the same in all the locations. The food was found in the same manner at all the locations.

MEAN LEAD CONCENTRATION mg/L OF FRIED YAM

The concentration of lead in this food was found to be almost the same in all the locations with mean$\pm$ SEM to be (0.12$\pm$ 0.001)mg/L. Lead was consistently high in all the locations due to the fact that, the food was prepared and sold at roadside openly.

5.7 MEAN LEAD CONCENTRATION mg/L OF JOLLOF RICE & BEANS

A1 location was found to be the lowest (0.001mg/L) and F_1 recorded the highest (0.20mg/L). The other locations had lead concentration ranging from 0.1 – 0.16mg/L. Boiled beans recorded low concentration of lead while in Jallof rice and beans, the concentration was found to be relatively

high. This was probably due to other spices and raw materials used during preparations.

In general, the detection of Pb in all the samples could be due to human activities, which led to the environmental release of lead into the air and thus the exposed food. Other sources of Pb include lead from the solder in tin cans and copper pipes, pesticides species and cooking utensils. Disposal of batteries and other lead containing materials can release lead to the soil contaminating the food materials. Lead in gas will be released into the atmosphere and eventually settle in the soil. Lead can also leach from older plumbing into water used in the preparation of the food. Improperly glazed ceramic containers can also leach lead into stored food.

This study recommends that food hawkers should be stopped from exposing the foods and the safety of food must be protected due to the adverse health effect of lead. The raw materials and preparations of these foods are areas to look at since they can add to the lead concentration in the food items studied. This could be achieved through proper hygiene and cleanness at the preparation venue. Proper enlightenment campaign would bring much needed awareness.

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