CHAPTER ONE

INTRODUCTION

1.1 Background of the study

Dietary patterns are defined as the quantities, proportions, variety or combinations of different foods, drinks and nutrients in diets, and the frequency with which they are habitually consumed. The nutritional quality of a dietary pattern can be determined by assessing the nutrient content of its constituent foods and beverages and comparing these characteristics to age- and sex-specific nutrient requirements and standards for nutrient adequacy (NEL, 2014; ODPHP, 2017). A well designed dietary intake assessment will provide quality data on the dietary patterns of any desired study population.

Dietary intakes assessment is a major component in the assessment of the nutritional status viz-a-viz the nutritional health status of an individual or a population. It entails a comprehensive evaluation of the chemical intakes, of the population in focus, through foods and drinks. This assessment can be done at the national, household or individual level, depending on the objectives of those carrying out the evaluation (Hulshof and Lowik, 1998; Kroes *et al.*, 2002; Shim*et al.*, 2014). This assessment tends to enable the characterization of the chemical intakes through food and drinks to ascertain the adequacy and or toxicity of the dietary component(s) in view.

Minerals are essential inorganic elements needed in small amounts in the diet for the normal function, growth and maintenance of body tissues (Byrd-Bredbenner*et al.*, 2013). At least 16 minerals have been found essential to human health, which have been broadly classified into major (or macro) minerals and trace (or micro) minerals. Several classification schemes exist but the one adapted for the purpose of this study is the scheme that divided the minerals based on the 100 milligram requirement. Macrominerals are regarded as those essential minerals required by the body in greater than 100 milligram quantity per day, whereas microminerals are required in less than

100 milligram per day (Wardlaw and Hampl, 2007). Macrominerals include sodium (Na), potassium (K), calcium (Ca), magnesium (Mg), phosphorus (P), chloride (Cl^-) and sulphur (S). Microminerals are copper (Cu), zinc (Zn), manganese (Mn), iron (Fe), iodide (I^-), fluoride (F^-), selenium (Se), chromium (Cr) and molybdenum (Mo) (Wardlaw and Hampl, 2007; Byrd-Bredbenner *et al*., 2013).

A third other group of minerals is the ultra-trace minerals. These minerals have been found to support health in human and or in some other animals. Ultimately, these minerals may be elevated to the status of essential nutrient. Significant minerals in this category are boron, nickel, silicon, arsenic and vanadium (Wardlaw and Hampl, 2007). The last category is the toxic metals, otherwise called heavy metals.

A heavy metal is a member of a poorly-defined category of elements that exhibit metallic properties, which mainly include the transition metals, some metalloids, lanthanides, and actinides. Many different definitions of heavy metals have been suggested over the years; some are relative to density, some according to atomic number or atomic weight, and some are due to chemical properties or toxicity (Duffus, 2002). Heavy metals, in general, are not biodegradable, have long biological half-lives and have the potential for accumulation in different body organs leading to unwanted side effects (Jarup, 2003; Sathawara*et al.*, 2004). Most of the heavy metals are extremely toxic because of their poor solubility in water. Even low concentrations of heavy metals have damaging effects to man and animals because there is no good mechanism for their elimination from the body. Currently heavy metals are ubiquitous because of their excessive use in industrial applications (Singh*et al.*, 2004; Chen*et al.*, 2005).

According to Paulsen, Luf and Smulders (2007), the major toxic metals of concern to foods are arsenic (As), cadmium(Cd), lead (Pb), mercury (Hg), Cr, Cu and Zn. The first four metals have no nutritional benefit to animals and are toxic at very minute quantity, whereas the last three are mineral nutrients to them but are also toxic, above certain threshold. Thus, the need to monitor these metals in foodstuffs taken by Nigerian consumers cannot be overemphasized. Furthermore, cadmium, lead, copper, manganese, iron and zinc were chosen as representative heavy metals whose levels in the environment represent a reliable index of environmental pollution (Ghaedi*et al.*,

2005; Ghaedi*et al.*, 2008; Karimi*et al.*, 2008). Some major sources of the toxic and essential metals in the environment include metallurgy industries, coal combustion and high traffic density (Sesli*et al.*, 2008).

1.2 Statement of the problem

Malnutrition in all its forms (protein energy malnutrition, micronutrient deficiencies and over nutrition) is a public health concern in developing countries. According to National Demographic Health Survey (NDHS) report of 2013, chronic malnutrition in Nigeria was 37% (Ene-Obong*et al*., 2013; NPC and ICF International, 2014). With the current global and national economic downturn, the situation might be worsened if there are no appropriate interventions. Indeed the hope of reaching the sustainable development goal 2 of 'zero hunger' could ever remain a dream in Nigeria if no proactive steps are taken towards providing current information on our food quality (as regards presence and levels of specific nutrients).

Over the years, several efforts have been made by organisations and individuals to compile food composition data for Nigerian Nutritionists and health professionals (FAO, 1968; Oyenuga, 1968; Platts, 1975; Oguntona and Akinyele, 1995). Most recently, the FAO/UN collaboration released two versions of Food Composition Tables for West Africa (Stadlmayr*et al*., 2010; Stadlmayr*et al*., 2012). However, challenges surrounding the use of these documents include obsolete data, reports on raw food samples, missing food varieties and nutrient values specific to Nigeria, and a lot more. In fact, the several food varieties missing in the Food Composition Tables (FCTs) currently used in Nigeria show that none of them is truly national (Ene-Obong *et al*., 2013). There is a serious lack of analytical data especially on vitamins and minerals even in the latest West Africa FCT released(Stadlmayr *et al*., 2012). These situations have made it difficult for proper dietary assessment of Nigerians, an exercise that could yield appropriate guide to food choices and healthy dietary practices in view of limited resources earmarked for food purchase in various households.

Furthermore, there is very limited information available in literature on the heavy metal contents of food and blood samples of Nigerian residents (Onianwa*et al.*, 2000; Onianwa*et al.*, 2001; Onabanjo and Oguntona, 2003; Babalola and Babajide, 2009; *et al.*, 2009; Ajayi, Charles-Davies and Arinola, 2012; Akan*et al.*, 2014; Lawal, 2014;

Akinyele and Shokunbi, 2015a). Thus, a partial or comprehensive food safety evaluation of consumers in Nigeria using recent data has been a mirage.

1.3 Justification of the study

Knowledge of the nutrient content of foods is fundamental for virtually all nutrition-related projects, programmes and policies. Information on food composition is important for nutrition and health programmes, agricultural and environmental sectors as well as for labelling and trade regulations (Greenfield and Southgate, 2003; Burlingame, 2004). In addition, food composition data can aid the selection of cultivars and varieties that are beneficial for nutritional quality and yield of foods (Toledo and Burlingame, 2006; Burlingame*et al.*, 2009). For all these purposes high quality food composition data are required. Low quality food composition data may misdirect research and lead to inappropriate policies and resource allocations (Harrison, 2004; Pennington, 2008).

There is need for a country specific FCT for Nigeria. Efforts have been made in several developed countries to report the mineral contents of foods in raw and or cooked forms over the years (Saracoglu*et al.*, 2007; FSANZ, 2008; Milloure*t al*., 2011; Chekri*et al*., 2012). Similar concerted efforts exist in some African countries such as South Africa (Wolmarans*et al.*, 2009; Wolmarans*et al.*, 2010; Wolmarans*et al.*, 2013) and Cameroon (Gimou*et al.*, 2014). The increasing prevalence of diet related Non-Communicable Diseases (NCDs) makes the knowledge of the nutrient composition of foods consumed by Nigerian population imperative. However, the support gained to generate this data nationally has been quite minimal (Ene-Obong *et al*., 2013).

Most available reports on mineral contents of foods in Nigeria are quite old, on few food varieties and mostly on raw foods (Onianwa *et al*., 2000; Onianwa *et al*., 2001; Onabanjo and Oguntona, 2003; Williams *et al*., 2009; Akinyele and Shokunbi, 2015a). Furthermore, Stadlmayr *et al*. (2012) clearly emphasized that there is a serious lack of quality analytical data especially on vitamin and mineral contents of West African foods, including Nigeria.

Reports from countries around the world have shown that heavy metals of various biological tissues, especially blood samples, from children and adults have been analysed over the years in a drive towards establishing reference values and enhancing safety of populace relative to heavy metal toxicity. The studies have been carried out on population representatives as sponsored by various scientists, government and non-governmental agencies in those countries, as frequent as possible (Clark*et al.*, 2007; Schulz*et al.*, 2009; Gil*et al*., 2011; Hrubá*et al*., 2012; Christensen, 2013). There is paucity of data on the levels of heavy metals in the blood samples of Nigerian adults. The levels of copper, zinc and magnesium in maternal and cord blood at delivery were reported by Makinde *et al*. (1991). The lead and cadmium levels in blood samples of some industrial workers had been reported (Babalola and Babajide, 2009). Ajayi *et al*. (2012) reported that elevated heavy metal may contribute to recurrent spontaneous abortion in women. Report on Maiduguri residents' blood and urine heavy metal contents is also available in literature (Akan *et al*., 2014). Lawal (2014) reported the levels of lead, cadmium and chromium in the hair, nails and blood samples of electronic repairers in Kaduna. All these reports are of relatively small sample size and somewhat mostly confined in locations. Thus, this study attempted to individually evaluate the levels of macrominerals (potassium, sodium, calcium and magnesium), microminerals (copper, manganese, iron and zinc) and heavy metals (cadmium and lead) in foods from Nigeria, as consumed. It also aimed to report the levels of some selected heavy metals (copper, zinc, iron, chromium, nickel, manganese, cadmium and lead) in the blood samples of some apparently healthy Nigerian adults. This study was designed with an intention to provide current data on Nigerian foods and blood samples in an effort to contribute to the reduction of some of the challenges of nutritional epidemiologists in Nigeria.

The conceptual framework of this study as highlighted in Figure 1.1 is helpful to indicate the overall implication of the outcomes of this study as related to the well-being of consumers in Nigeria.

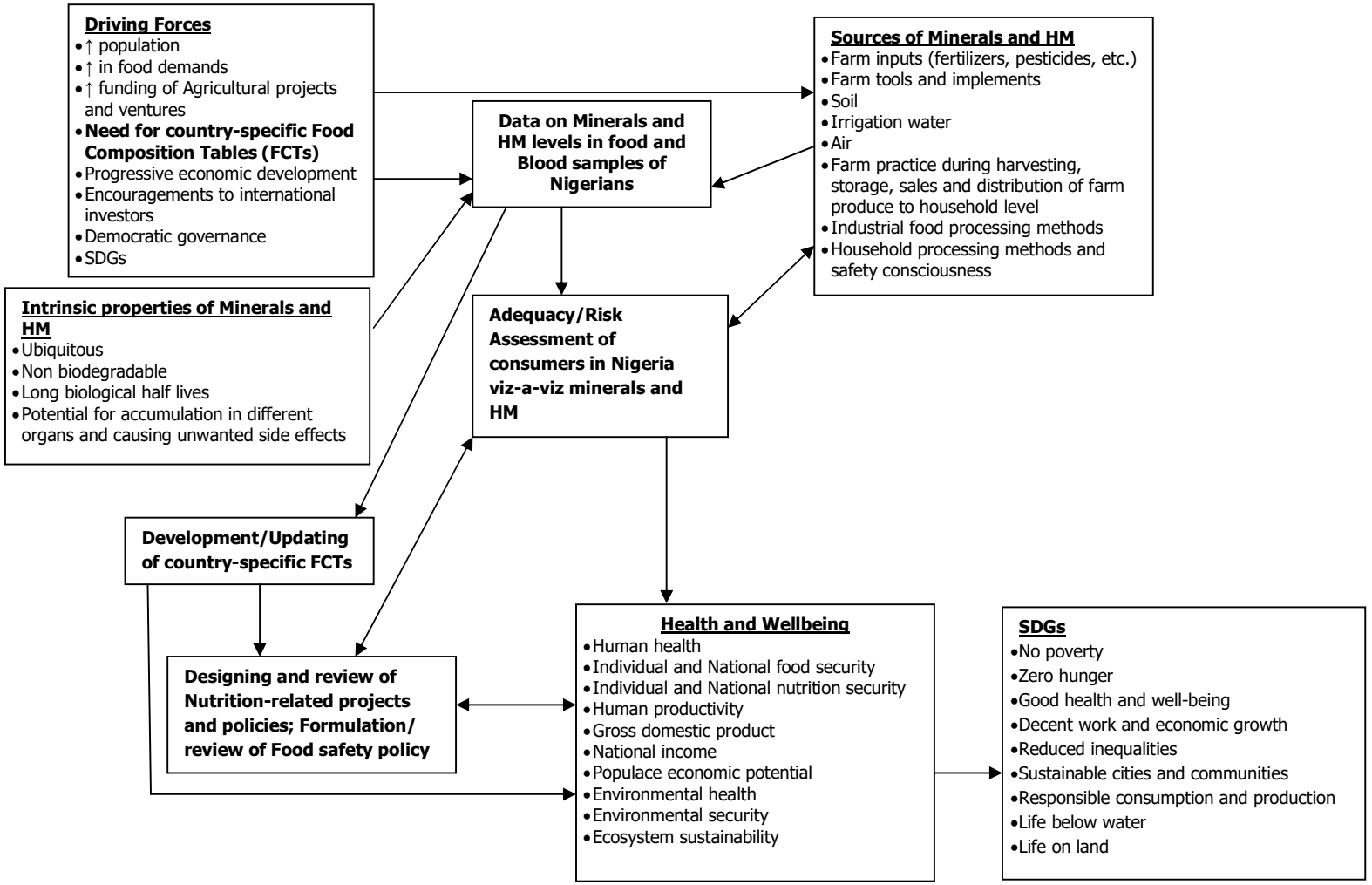

Figure 1.1: Conceptual framework of the study

NB: HM – Heavy Metals; SDGs – Sustainable Development Goals

1.4 Objectives

1.4.1 General objective

The general objective of this study was to determine the dietary patterns, mineral and heavy metal concentrations in the food and blood samples of selected adults residing in Ogun State and Federal Capital Territory (FCT), Abuja.

1.4.2 Specific objectives

The specific objectives were to:

1. assess the dietary patterns of selected adults residing in Ogun state and Abuja,
2. evaluate the macromineral (potassium, sodium, calcium and magnesium), micromineral (copper, manganese, iron and zinc) and heavy metal (cadmium and lead) concentrations in food samples collected 'as consumed' from various major markets and restaurants in Ogun State and Abuja,
3. determine the concentrations of copper, zinc, iron, chromium, nickel, manganese, cadmium and lead in the serum/blood samples of some apparently healthy adult volunteers in Ogun State and Abuja; and
4. compare data obtained from the dietary patterns and blood samples with international standards to ascertain adequacy of intakes and safety of consumers.

CHAPTER TWO

LITERATURE REVIEW

2.1 Dietaryintake assessment

This is one of the five nutritional status assessment schemes done to ascertain the nutritional health status of an individual or a population. It involves the qualitative and/or quantitative evaluation of the chemical intakes of the participants through foods and drinks. This assessment can be done subjectively or objectively; it can also be done at the national, household or individual level, depending on the interest of the person or organization carrying out the evaluation (Hulshof and Lowik, 1998; Kroes *et al*., 2002; Shim *et al*., 2014).

There are five major data useful in assessing dietary intakes, which include data from food supply, data from household food consumption survey, data from dietary surveys among individuals, data collected from complete diets study and data from biomarkers (Hulshof and Lowik, 1998; Kroes *et al*., 2002). The food supply data, household food consumption survey data and data from complete diet study are usually collected by objective observation whereas those from individual dietary survey and biomarkers are collected subjectively (Shim *et al*., 2014). The first two set of data pertains to the national and household level of evaluation, respectively; whereas the last three are carried out at the individual level.

Every set of data relates to a specific level of the food chain and is collected by unique methods. The complete diet method is distinct from other methods. The intake estimation via this method depends mainly on the results from chemical analysis of the duplicate or simulated diet. Biomarker measurements reflect both the types of food consumed and the concentration of the chemical in these foods (Kroes *et al*., 2002).

Over the years, as the economic status of people improves, they tend to eat more outside their homes. Thus, assessment at the individual level is taken to be very vital in most nutritional epidemiological studies. This has led to various advancements of the

individual dietary assessment techniques (Shim *et al*., 2014). This aspect of review will briefly cover description of data collection at the national and household level, while it concentrates more on the collections at the individual level.

2.1.1 Food supply data

Food supply data implies food availability that gives a crude impression of potential average consumption per individual. Food and nutrient losses before consumption, as a result of processing, trimming, waste and spoilage, are unlikely to be adequately accounted for. Food supply data are usually calculated in Food Balance Sheets (FBSs), which are accounts, on a national scale, of annual food production, stock changes, imports and exports, as well as industrial and agricultural use. This produces an estimate of the mean value per individual in the population considered, irrespective of age or gender. Due to their long history, FBSs are usually applied to assess trends of food supply over time (Kroes *et al*., 2002).

Currently food supply data is mainly applied in the exposure assessment regarding contaminants and pesticides residues that are often determined in raw commodities. Many European and non-European countries make use of this strategy as a first step in the risk assessment procedure and this can be useful for comparisons across countries. In addition to the international FBSs, many countries publish national FBSs, which are somewhat better up to date and can slightly differ from the international version (Kroes *et al*., 2002).

2.1.2 Household food consumption survey data

The various foods present at the level of the household can either be estimated using budget surveys or by using consumption surveys. Budget survey gives information on household expenditures on food items and this data is used for economic policy. As regards the household consumption surveys, the quantity of foods and drinks (whether purchased or as gifts) brought into the household are recorded in addition to expenditure data. In most situations, only the expenditures on foods and drinks consumed at home are recorded. In some cases, household surveys may measure some changes in food stocks, along with information on those acquired (Kroes *et al*., 2002).

Generally, household surveys do not indicate how food is handled in the household or on how much of the food is eaten by each member. Household food consumption data can be collected via record keeping by household key informant (usually the mother/wife), by interviews (conducted by trained personnel) or by the two strategies. Data collection regarding non-food items usually cover up to 4 weeks, but in the case of foodstuffs, 2 weeks is more usual (Kroes *et al*., 2002).

In most developed countries, household surveys began since 1940s or 1950s. Only few countries have been carrying it out regularly; some do the surveys every 3–4 years, others only every 5–10 years. There are a number of differences in sampling protocols, grouping of foods, conversion to nutrients, and timing, frequency and data collection techniques (Kroes *et al*., 2002).

2.1.3 Individual dietary survey data

As different from the FBSs and household food consumption surveys, data from individual dietary surveys provide information on mean food and nutrient intakes by specific well-defined groups of individuals. These data gives a better reflection of actual food and drink consumption of individuals. Several methods have been found useful in the collection of individual dietary intakes. Four of them will be discussed in this review. They are food records, 24-hour dietary recall, food frequency and dietary history methods. These methods can be broadly divided into two groups: record and recall methods. Record methods gather information on current intakes over one or more days. Recall methods evaluate previous consumption, which could be over the previous day (24-hour recall) or usual food intake (obtainable through dietary history or food frequency).

2.1.3.1 Food record

Food record is also called dietary record or food diary. This involves the record of foods and beverages (by respondents) and the quantity of each that has been eaten for a specific period, usually between one and seven days. It is ideally expected that the recording is done at the point the food is eaten, so as to avoid dependence on memory. The quantities taken may be weighted, using a scale or house-hold measures (such as, cups or spoons), or their estimates can be obtained using three-dimensional models or

two-dimensional models (pictures) (Kroes *et al*., 2002; Thompson and Subar, 2013). In some cases, the price equivalent of the quantity taken is normally utilized for the estimation of the weight of the food consumed.

When food records of multiple days are taken, they are usually consecutive, and no more than seven days are involved. Research has shown that recording periods above four consecutive days are usually unreliable and misguiding, as reported intakes decrease as a result of respondent fatigue. Since the foods and amounts eaten during consecutive days of reporting may be related, it may be better to collect non-consecutive single-day records in order to increase quality of data obtained on individual's diet (Gersovitz*et al.*, 1978; Thompson and Subar, 2013).

In an effort to increase the quality of data expected from food record survey, each respondent must be trained on the extent of details expected to appropriately describe the foods and drinks taken. The training should cover explanations on the need to indicate amounts consumed, the left-over in plates, the name of the food (as well as brand name, if possible), methods of preparation, recipes for composite diets, and portion sizes. The quality of records can also be enhanced if the investigator reviews the report with the respondent after the first day of taking the food record. At the end of the recording period, it will be very helpful that a trained interviewer review the whole record with the respondent to clarify entries and to probe for foods that have been mistakenly excluded from the records. Food records can also be taken by a trained person other than the respondent in view; such as mother or father reporting for their children or wife reporting for husband and vice versa (Kroes *et al*., 2002; Thompson and Subar, 2013; Shim *et al*., 2014).

The food record method can provide quantitatively accurate information on foods and drinks eaten during the recording period (Gibson, 2005). It helps to minimize the problem of omission and enhances detailed description of foods, since the data are obtained at eating point. To get high quality and reliable food record, respondent must be well motivated as most of the burdens of data collection are transferred to them (Margetts and Nelson, 1997; Thompson and Subar, 2013).

2.1.3.2 24-Hour recall method

In the 24-hour dietary recall, the respondent is asked by trained interviewer to remember and describe all foods and drinks taken in the immediate past 24 hours or in the preceding day. Traditionally, the recall is requested by one-on-one interview, physically or by telephone (Buzzard*et al*., 1996; Casey*et al.*, 1999; Kroes *et al*., 2002). It can also be done with the aid of computer (Slimani*et al*., 2000; Moshfegh*et al*., 2008) or by the use of a paper-and-pencil form; although self-administered electronic version of 24-hour recall has recently become widely available (Vereecken*et al*., 2008; Arab*et al.*, 2010; Arab*et al.*, 2011).

When interviewer-administered, the interviewers should be well-trained as the quality of data obtained is largely dependent on the experience/expertise of such fellow. Best practice requires interviewers to be dietitians or scientists with specialities in foods and nutrition; however, non-nutritionists can be trained on how to use the standardized instrument effectively. It is expected that all interviewers know most of the commonly consumed foods and drinks, their preparation methods, as well as other special foods and drinks prevalent in specific locality. Food models are also well incorporated into this evaluation scheme to enhance accurate quantification as much as possible (Kroes *et al*., 2002; Thompson and Subar, 2013).

Over the years there has been the contention on how best to collect the 24-hour dietary recall. This has made the US Department of Agriculture (USDA) Agricultural Research Service (ARS) to carry out studies on how best to improve the 24-h recall methodology. These studies resulted in the development of the USDA's Multiple-Pass Method in 1999. This is a 5-step dietary interview that includes multiple passes through the 24-h of the previous day, during which respondents are given cues that can help them to recall and describe the foods and drinks they ate within the period in view (Raper*et al.*, 2004; Moshfegh*et al*., 2008). The five steps include: a quick list of foods and drinks; probe of forgotten foods and drinks; time and occasion of eating or drinking; detail cycle of all the foods and drinks listed; and final review.

In 2002, a computer-assisted version of the 5-step method, the Automated Multiple-Pass Method (AMPM), was developed. The AMPM similarly navigates the respondent through the recall, posing standardized questions and supplying response options for

different foods and beverages. It has been used to collect dietary recalls in What We Eat InAmerica (WWEIA), the dietary interview component of the National Health And Nutrition Examination Survey (NHANES) (USDA Agricultural Research Services, 2016). This automated version has been evaluated and validated over the years by several studies (Conway*et al.*, 2003; Conway*et al.*, 2004; Blanton*et al.*, 2006; Moshfegh*et al*., 2008).

2.1.3.3 Food frequency method

In this case, the respondent is usually evaluated using a Food Frequency Questionnaire (FFQ) that is self-administered or interviewer-administered. The FFQ entails a structured list of individual foods or food groups intended to guide respondents to report their usual frequency of consumption of the foods over a given time (such as daily, weekly, monthly or yearly). The FFQ may be brief, pertaining to one or more specific chemicals, with less than 50 food items; or it can be comprehensive, pertaining to large number of nutrients, and having 50 to 150 food items listed (Zulkifli and Yu, 1992; Willett, 1998; Kroes *et al*., 2002). Traditionally, FFQ collects information on frequency of consumption, with little or no detail requested on other dynamics like the methods of cooking, or the ways the foods are being combined in meals (Thompson and Subar, 2013).

Food frequency questionnaire can be qualitative, semi-quantitative or completely quantitative, depending on the objective of the study. Qualitative FFQs gather information mainly on the usual number of times that each of the foods listed is being consumed within a specified period. Semi-quantitative FFQs on the other hand move ahead to estimate the standard portion sizes consumed by the respondents and how frequent, on average, they consume an indicated portion size. A fully quantitative FFQ allows the respondent to freely indicate specific quantity of each of the listed foods typically consumed, within the specified period of time (Kroes *et al*., 2002).

In an effort to quantify amount of food intake, some FFQs incorporate portion size along with each question. The total nutrient or dietary constituent intake estimates are obtained by adding up all the figures obtained after multiplying the reported frequency of each food by the quantity of nutrient or dietary constituent in a specified (or assumed) serving of that food or food group. For best estimation of nutrient intakes for

an assumed or reported portion size of each food listed, each quantitative FFQ should be linked with an appropriate nutrient database. In many situations, the purpose of an FFQ is to obtain a rough estimate of total intakes over a particular period. In some cases, the result of the FFQ is applied to rank individuals by food, nutrient or by food group intakes into categories in a way that group of participants with high and low intake can be separately studied (Kroes *et al*., 2002; Thompson and Subar, 2013).

2.1.3.4 Dietary history method

Originally, as coined by Burke, *dietary history* referred to the gathering of information on the frequency of intakes of various foods as well as the typical make-up of the meals consumed (Burke and Stuart 1938; Burke, 1947). Currently, it is suggested that the term is best reserved for dietary assessment method that are designed to evaluate an individual's total usual food intake in which several details about characteristics of foods as usually consumed are evaluated along with the frequency and quantity of food intake (Kroes *et al*., 2002; Thompson and Subar, 2013).

Broadly considering the dietary history methodology; the respondent is made to supply information about his/her usual eating pattern over an extended time frame (mostly a 'typical' week) and also to recall the specific foods consumed within the preceding 24 hours. Furthermore, the interviewer fills a food frequency checklist as specified by respondent. Last, as a way to cross-check, the respondent is usually required to complete a 3-day dietary record. This broad perspective is still a great reflection of Burke methodology as proposed about seven decades ago. The period of time in view is often the preceding month or several months; or in some cases the preceding year, in an effort to reflect seasonal variation (Burke and Stuart 1938; Burke, 1947; Kroes *et al*., 2002).

Some dietary history instruments are now automated and customized for self-administration, in some case with audio; thereby eliminating the need for an interviewer (Kohlmeier*et al.*, 1997; Mensink*et al.*, 2001). Though some other dietary history instruments have been automated, they are still being administered by an interviewer (Slattery*et al.* 1994; EPIC group of Spain, 1997). In some situations, short-term recalls or records are incorporated in the dietary history instrument for validation or calibration instead of being a part of the tool (Thompson and Subar, 2013).

The main strength of the dietary history method is its assessment of meal patterns and details of food intakes over a relatively longer time frame rather than intakes for a short time frame (as in records or recalls) or only frequency of food intake (as evaluated using FFQ). Details of the method of food preparation can be useful in better evaluating nutrient intake (for example, frying vs. baking), as well as exposure to other factors in foods that relate to the method of processing (for instance, charcoal broiling). Although a meal-based approach, as found in dietary history method, often demands more time from the respondent than does a food-based approach, it may give better cognitive support for the recall process. For instance, the respondent may be better able to indicate total bread intake by reporting bread as consumed at every meal (Thompson and Subar, 2013).

A major weakness of the dietary history method is that respondents are required to make several judgments on both the usual foods eaten as well as the amount of those foods ingested. These subjective tasks may be difficult or impossible for several respondents. Burke emphasized that nutrient intakes estimated from these data should be interpreted as relative rather than absolute. All of these limitations are similarly shared with the food frequency method. Furthermore, the meal-based methodology is not useful for individuals that have no specific eating pattern and could be of limited use for individuals that "graze" (that is, eat throughout the day instead of at defined meal times). The dietary history approach, when carried out by interviewers; requires trained nutrition professionals and is thus of great financial demand. Last, the dietary history as a method is less standardized, and therefore methods applied by researchers differ from place to place and are very challenging to replicate, making comparisons across studies somewhat difficult (Burke and Stuart 1938; Burke, 1947; Thompson and Subar, 2013).

2.1.4 Complete diets

In the estimation of a population's dietary exposure, FAO/WHO recommends the application of total diet studies. In total diet studies, representative samples of most frequently consumed foods are gathered and analysed for the nutritive or non-nutritive component of interest. The reliability of the estimated intakes using total diet study

depends largely on the extent of coverage of the important dietary sources of the chemical(s) of interest (Kroes *et al*., 2002).

The underlisted approaches in total diet studies have been differently established:

(i) Market basket;

(ii) individual food items; and

(iii) duplicate portion.

The market basket approach evaluates the dietary intake of a specific population of interest. In this case, most or all food items that are part of the average diet of the population are purchased and prepared using the standard household procedures; and then pooled together into some of food groups. Each food group is usually analysed for some additives, contaminants and nutrients; depending on the objectives of study. As for the individual food items approach, a food list encompassing most of commonly consumed foods is compiled via national food consumption surveys that covered representative fraction of various age groups and gender. Every food item selected is prepared according to most commonly used household procedures and analysed for specific chemical of interest. In the duplicate portion or duplicate diet approach, the individual daily diet as consumed is collected from members of the study population and analysed for the chemical (s) of interest (Van Dokkum and de Vos, 1990; Macdonald, 1991).

Although not so generalizable, total diet studies provide data that can be used to assess food chemical intake. Total diet study results are usually applied to identify trends in concentrations of pesticide residues, contaminants and nutrients in the food supply to specific population. Total diet studies use only a certain number of foods to represent thousands of foods; thus it is inappropriate to make extrapolations for the amounts of a contaminant in the sampled foods to the amounts consumed by individuals in the population being studied (Kroes *et al*., 2002).

Nevertheless, concentration data on the foods sampled in total diet studies can be used as reference point in intake assessment (Douglas and Tennant, 1997). The duplicate portion method, although being a total diet study, presents some unique features that

are advantageous. Initially, duplicate diet methods give information on individual intakes. However, they are particularly useful for estimating exposure where no national consumption data are available or where an investigation of the exposure of a particular population subgroup is being carried out (WHO, 1999). Along with weighed food intake records, the duplicate portion method is the best possible approach to precisely measure the actual consumption by individuals (WHO, 1985). However, this approach requires a very high commitment from the participants and it is very possible for the participant to modify his/her dietary pattern during the collection period, which could defeat the essence of study.

Total diet studies have been done since the early 1960s in several countries. An example is the US FDA Total Diet Study, conducted on a yearly basis since 1961 (FDA, 2000). At the inception, the main objective was to estimate average intakes or broad exposures of the population to pesticide residues (Harries*et al.*, 1969) and levels of radioactive contamination in foods from atmospheric nuclear testing (Pennington and Gunderson, 1987). The initial purpose in the UK was also targeted at nutrients. At present, regular total diet studies are carried out in various countries such as the USA, the UK Ministry of Agriculture, Fisheries and Food (MAFF, 1996), Australia (ANZFA, 1998), New Zealand (Cressey*et al.*, 2000; Vannoort*et al.*, 2000), and Spain (Urieta*et al.*, 1996; Jalon*et al.*, 1997) and so on, with the purpose of estimating exposure of the general population to a wide range of nutrients, food additives, chemical contaminants and pesticide residues.

In the same vein, duplicate diet studies have been carried out in various countries. Almost 30 research works were summarized by Thomas*et al.* (1997) on duplicate diet studies, most of which have been directed towards dietary intake of essential elements and toxic metals. Very few reports exist on organic chemical contaminants like pesticides and Poly Aromatic Hydrocarbons (PAHs). About 20 years ago, duplicate diet studies were also conducted in the UK and The Netherlands to estimate exposure to nitrates (Vaessen and Schothorst, 1999; Ysart*et al.*, 1999). Also, the UK has two duplicate diet studies to estimate exposure to OTA of the UK population and of vegetarians to natural toxicants (FDA, 2000). Other studies with duplicate diet procedure were the one on food samples from the French community (Noël*et al.*, 2003), where several elements were determined; and the one carried out on population

of Tarragona County, Spain in which dietary metal intakes were also evaluated (Domingo*et al.*, 2012).

2.1.5 Biomarkers

Methodologies depending on biomarker can be employed to evaluate human exposure to food chemicals. They mostly entail two main stages. In the first stage, human volunteer studies (or – for contaminants – total diet studies) are undertaken to confirm whether a quantitative relationship can be found between the dietary intake of the chemical of interest and the amount of the corresponding biomarker quantified in an appropriate biological sample (tissue or body fluid). Some additional details of biological factors will also be needful; including other components of the diet (such as lipids in the case of lipophilic food components), which determine the presence and the level of the biomarker of interest (Kroes *et al*., 2002).

The validation process for a biological marker involves the confirmation of its physiological relevance and reproducibility as well as a vivid understanding of the extent and limitations of the analytical and sampling methods required. After affirming a quantitative relationship, the method is then applied, in the second stage, to individuals in the target population by collecting and analysing appropriate samples. In most situations, the biomarker evaluated is either the food chemical itself or a metabolite. Urine or blood sample is often used for the quantification of biomarkers. However, some other options exist that include especially breast milk, but also hair, adipose tissue, buccal swaps, exhaled air and faeces (Kroes *et al*., 2002).

In some epidemiological studies, biomarkers have been used as indicators to measure the dietary intake of specific nutrients or non-nutritive components of foods (Kim*et al*., 2012; Lim*et al*., 2012; Kho*et al*., 2013). These biomarkers have been established by some researches to be highly correlated with dietary intake levels, free of a social desirability bias, relieving to respondents as regards memory burden, and independent of respondents' ability to describe the type and quantity of foods eaten (Potischman, 2003). Therefore, biomarkers may yield more accurate measures than dietary intake estimates do, in some respects. However, some biomarkers have been known to provide integrated measures that show their absorption and metabolism. They can also be affected by disease or homeostatic regulation; thus, their figures must not be

interpreted as the participant's total dietary intake (Kaaks*et al.*, 2002). Furthermore, the data obtained from biomarkers is very insufficient to provide dietary recommendations that can guide a modification of an individual's dietary pattern. Therefore, direct measurement of individual's dietary intakes may provide better information than biomarkers, in this regard (Wild*et al.*, 2001; Potischman, 2003).

It is noteworthy that biomarkers can give useful information on the order of stages in the aetiology of several chronic diseases. A model has been outlined by the Committee on Biological Markers of the National Research Council (CBM-NRC) (1987) that categorises biomarkers in terms of those showing: internal dose, biologically effective dose, early biological effect, altered structure/function, clinical disease and susceptibility.

An objective consideration of these five dietary intakes assessment methodologies shows that they are quite complementary to each other. The choice of one or more of them during any study will greatly depend on the objectives of study, available funding, population size of study group, period of time available for study as well as level of expertise of the Principal Investigator and other research team members. Comprehensive evaluation of all these details and dynamics will enable the choice of the most appropriate methodologies viz-a-viz research instruments.

2.2 Food composition tables

Food Composition Tables (FCTs) are compilations that give detailed Food Composition Data (FCD) on various nutritionally important food components. They are otherwise called Food Composition Databases (FCDBs) or food composition datasets. They provide values for energy and nutrients which include lipid, protein, carbohydrates, minerals and vitamins as well as some other important food components like fibre (Wikipedia, 2016a). The first FCT released was the one entailing American food materials – Chemical Composition of American Food Materials – as published in the 19th century (Atwater and Woods, 1896). Similarly in the United Kingdom, the first FCT published – The Chemical Composition of Foods – was by McCance and Widdowson (1940). Thereafter these, several others have been published over the years. These publications have been put up in line with the view of McCance and Widdowson (1940) that knowledge of the chemical composition of

foods is the first vital tool for the dietary treatment of disease or in any quantitative study of human nutrition.

Food composition data can also aid the selection of cultivars and varieties that are beneficial for nutritional quality and yield of foods (Toledo and Burlingame, 2006; Burlingame *et al*., 2009). Furthermore, knowledge of the nutrient content of foods is quintessential for virtually all nutrition-related projects, programmes and policies. Information on food composition is vital for various nutrition and health programmes, agricultural and environmental sectors as well as for food labelling and trade regulations of food materials (Greenfield and Southgate, 2003; Burlingame, 2004). For all these roles, high quality food composition data are required. Low quality food composition data may misguide research and lead to ineffective policies and improper resource allocations (Harrison, 2004; Pennington, 2008). The above highlighted realities and benefits have compelled several organizations and scientists to tirelessly support and generate FCTs for various countries or regions across the globe.

There is need for a country specific FCTfor Nigeria. There is also a dare need of the heavy metal levels of the foods consumed in Nigeria. Efforts have been made in several developed countries to report the metal contents of foods in raw and or cooked forms over the years. The following data on mineral and heavy metals of foodstuffs have been useful in the compilation of national FCTs as well as risk assessment of consumers in the respective locations. Jorhem and Sundstroem (1993) reported the levels of lead, cadmium, zinc, copper, nickel, chromium, manganese and cobalt in foods on the Swedish market, 1983-1990. Gumgum*et al*. (1994) detailed some heavy metal pollution in water, sediment and fish from the Tigris River in Turkey. Pennington*et al*. (1995a) reported the Calcium, magnesium, iron, and zinc composition of core foods of the USA food supply of 1982-1991. They also reported the copper, manganese, selenium and iodine contents of the same set of USA food supply (Pennington *et al*., 1995b). Barlas (1999) did a pilot study of heavy metal concentration in various environments and fishes in the Upper Sakarya River Basin, Turkey and reported some contaminations. Other scientist have reported minerals and heavy metals in foodstuff from Spain (Cuadrado*et al.*, 1995; Cuadrado*et al.*, 2000), Slovenia (Milacic and Kralj, 2003), France (Leblanc*et al.*, 2005; Millour*et al*., 2011; Chekri *et al*., 2012), Turkey (Saracoglu*et al*., 2007), and Australia (FSANZ, 2008).

Similar concerted efforts exist in some African countries such as South Africa (Wolmaranset *al*., 2009; Wolmarans *et al*., 2010; Wolmaranset *al*., 2013) and Cameroon (Gimouet *al*., 2014). The increasing prevalence of diet related NCDs makes the knowledge of the nutrient composition of foods consumed by the Nigerian population imperative. However, the support gained from the government to generate this data nationally has been quite minimal (Ene-Obong *et al*., 2013).

2.2.1 Review of the food composition tables used by Nigerian nutritionists and health professionals over the years

The first among the databases accessed by Nigerians was the one published by Food and Agricultural Organization (FAO) of the United States of America as edited by Wu and colleagues in 1968. This project was sponsored jointly by the Nutrition Program, National Center for Chronic Disease Control, US Department of Health, Education, and Welfare (NP/NCCD), and the Food Consumption and Planning Branch, Nutrition Division, FAO. This database contains 1,624 food items including those from plant and animal origins. Fourteen food groups were reported including cereals and grain products; starchy roots, tubers and fruits; grain legumes and legume products; nuts and seeds; vegetables and vegetable products; fruits; sugars and syrups; meats, poultry and insects; eggs; fish and shellfish; milk and milk products; oils and fats; beverages; and miscellaneous. The food components reported are proximate composition, minerals, B-vitamins, vitamin A, ascorbic acid and tryptophan. At the moment, this database is certainly obsolete. However, it was a great lift to food composition effort in Africa and its sub-regions.

Another document published in the same year as that of the FAO was compiled by Oyenuga (1968) entailing the chemistry and nutritive values of Nigerian foods and feeding stuff. This was a 99-page book published by Ibadan University Press. This is also very obsolete and poorly circulated; thus rarely utilized.

Platts (1975) further reported representative values of foods commonly used in tropical countries. This is equally obsolete and has not kept track with changes in tropical foods. This report is very much generalized and not so adaptable to most of the commonly consumed Nigerian foods that are of interest to nutritionists.

In 1995, the Food Basket Foundation International (FBFI) published another document, edited by Oguntona and Akinyele, on the nutrient composition of commonly eaten foods in Nigeria – raw, processed and prepared. This was a very laudable effort as it included several indigenous foods. However, it may lead to overestimation or underestimation of intakes, because of various data supplied in which some dishes were combined in non-conventional ways. Some other data in this document are not at congruent with several other databases, which make them questionable. These challenges and a few others have made this document less adaptable for national use.

Stadlmayr *et al*. (2010) edited an FAO document on the composition of selected foods from West Africa. These data represent the average values of the collected compositional data and is a subset of the archival database that was compiled from March to August 2010 from 7 countries (Benin, Burkina Faso, Ghana, Guinea, Niger, Nigeria and Senegal). The 54-page document has data from more than 1500 food items compiled, out of which 173 foods (of 13 food groups) and 30 components were selected for this table. These data are certainly better refined and adaptable than most of the previously highlighted databases. However, several foods are still missing.

A more recent edition of West African food composition table was also edited by Stadlmayr*et al*. (2012) and published by FAO. This revised version of the 2010 publication extends and updates the number of foods and values of components through data derived from the Mali Food Composition Table, as well as analytical data from scientific articles. The foods represent average values of the collected compositional data from 9 countries (Benin, Burkina Faso, *Gambia*, Ghana, Guinea, *Mali*, Niger, Nigeria and Senegal). This edition includes 472 foods (still of 13 food groups) and 28 components. This document is indeed the best ever obtained for the composition of foods in the West African sub-region. However, the author still emphasized that there is a serious lack of quality analytical data especially on vitamin and mineral contents of West African foods, including Nigeria. This made them to borrow most of these data from other established databases of developed countries. It is also noteworthy that most of the collected data were for raw foods.

2.2.2 Other previous reports on mineral and heavy metal composition of Nigerian foods

Ifon and Bassir (1979) reported the the nutritive value of some Nigerian leafy green vegetables – Part 1: vitamins and mineral contents. The levels of trace elements in hospital diet were reported in Food Chemistry Journal by Akinyele and Osibanjo (1982). Ndiokwere (1984) gave a detailed report on heavy metal pollution from motor vehicle emission and its effect on road soils, vegetation and crops in Nigeria. Estimates of elements in selected tropical fruits and vegetables were given in a published work of Aremu and Udoessien (1990). The reports of Onianwa *et al*. (2000) and Onianwa *et al*. (2001) are quite unique and comprehensive compared with all other reports previously mentioned in this section of review. Onianwa *et al*. (2000) analysed and reported the levels of cadmium and nickel in 78 Nigerian foods, while Onianwa *et al*. (2001) reported the copper and zinc composition of 80 Nigerian foods. The coverage here is quite wide, though the foods were collected from a location (Ibadan) in Nigeria. Onabanjo and Oguntona (2003) reported on the iron, zinc, copper and phytate contents of 20 standardized Nigerian dishes. Williams *et al*. (2009) worked on trace metal levels in fruit juices and carbonated beverages in Nigeria. Last, Akinyele and Shokunbi (2015a) reported concentrations of Mn, Fe, Cu, Zn, Cr, Cd, Pb, Ni in selected Nigerian tubers, legumes and cereals.

A general evaluation of these reports shows that most of them are quite old, reported relatively few food varieties and mostly had data on raw foods. Hence, a careful effort is being made in this study to report the levels of eight minerals and two heavy metals in over one hundred different Nigerian foods as consumed.

2.3 Minerals and heavy metals

Minerals are inorganic substances, present in all body tissues and fluids and their presence is necessary for the maintenance of certain physicochemical processes which are essential to life (Malhotra, 1998). Minerals are inorganic compounds that have been identified in human body as ions (charged atoms) or as part of complex biomolecules. There are 16 minerals discovered to be essential for normal metabolic activities, though some, known to be essential for other animals, might be included in the list as researches advance with years (Wardlaw and Hampl, 2007).

Essential minerals are broadly classified into two categories: the major (or macro) minerals and the trace (or micro) minerals. The macrominerals are the minerals required in amounts greater than 100 milligrams per day, while and the microminerals are thoserequired in amounts less than 100 milligrams per day (Wardlaw and Hampl, 2007; Levetin and McMahon, 2008). The ultra-trace minerals form another group of minerals that is gradually stimulating some interest. These minerals are known to enhance health in humans and some other organisms but are not known to be indispensable to humans. However, some experts believe that with time these minerals might be elevated to essential nutrient status (Byrd-Bredbenner*et al*., 2013). A last category of metals is the heavy metals, which are sometimes regarded as toxic metals. This review focuses on the macrominerals, microminerals and the heavy metals as related to the objectives of the study.

2.4 Selected macrominerals

2.4.1 Potassium

Potassium is a silvery grey metal that was discovered in the early 1800s. Its name was coined from potash, meaning 'extracted in a pot from the ash of burnt trees' (Byrd-Bredbenner *et al*., 2013). Potassium is the major intracellular cation. About 95-98 % of the body's potassium is found within the body cells. It constitute up to about 245 g in a 70-kg individual (Groppe*et al.*, 2005).

2.4.1.1 Sources of potassium

Potassium is found in many foods, especially those not processed. Very rich sources include fruits (like orange, banana, plantain, and watermelon), vegetables (like avocados and leafy green vegetables), and fresh meat. Other good sources of potassium are potatoes, legumes, whole grains and milk. Salt substitutes are often known to contain potassium in place of sodium (Gropper *et al*., 2005; Byrd-Bredbenner *et al*., 2013). In Nigerian diet, fruits, starchy tubers and leafy green vegetables supply most of the dietary potassium. However, traditional processing of the foods usually significantly reduces the potassium content of the final product consumed (Smith and Ojofeitimi, 1995). Diets high in potassium are associated with lower blood pressure (Gropper *et al*., 2005).

2.4.1.2 Needs and metabolic functions of potassium

The adequate intake for potassium for adults is 4700 mg per day (IOM, 2005).Potassium is in fluid-electrolyte balance. It acts along with extracellular ionized sodium to maintain normal osmotic pressures and water balance that ensures cellular fluid integrity. It is envisaged to downplay the effect of high sodium intake and help maintain normal blood pressure. High consumption of potassium from meals tends to suppress the renin-angiotensin system and enhance the excretion of excess sodium and water. This has been a major mechanism by which potassium helps to prevent hypertension from occurring in humans(Williams, 1997; Heaney, 2006a).

Potassium has a great influence on the acid-base balance through its interaction with sodium and hydrogen.It functions with sodium and calcium to regulate neuromuscular excitability and stimulation, transmission of electrochemical impulses, and contraction of muscle fibres. This effect is very significant in the heart muscle. Similar to sodium, it influences the excretion of calcium, though in a reverse direction. Thus, high dietary intake of potassium will result in minimal excretion of calcium(Heaney, 2006a; Byrd-Bredbenner *et al*., 2013).

Potassium is needed for the storage of nitrogen in muscle protein and overall cellular proteins. This is a major reason why potassium is usually added, while carrying out replacement therapy relative to amino acids deficiency (Williams, 1997).

2.4.1.3 Absorption, transport and excretion of potassium

The way by which potassium is being absorbed from the Gastrointestinal Tract (GIT) is not well understood. In recent time, the exact mechanism of potassium transport from the GIT was researched and identified. Up to 90 percent of the dietary potassium is usually absorbed with the small intestine as well as the colon playing major roles (Kliger*et al.*, 1981; Hayslett and Binder, 1982; Agarwal*et al.*, 1994).

Potassium ion can by absorbed across the apical brush border membrane of the colonic mucosal cell by a K^+/H^+-ATPase pump. This pump exchanges intracellular H^+ for luminal K^+ using a similar mechanism as that in which H^+ is secreted along with Cl^- into the stomach as HCl (Gropper *et al*., 2005).

Majority of the potassium is usually excreted from the human body through the kidneys, while a small amount is being excreted in the faeces. Aldosterone is the main regulatory hormone of potassium balance that is achieved through the kidneys. The overall effect of aldosterone on potassium and sodium is reciprocal. Though it stimulates reabsorption of sodium in the kidney tubules, it simultaneously increases the excretion of potassium (Gropper *et al*., 2005).

2.4.1.4 Deficiency and toxicity of potassium

Hypokalemia is regarded as low blood potassium. This is a life-threatening condition. Symptoms include: fatigue, weakness, constipation, and an irregular heartbeat, which impairs the ability of the heart to pump blood. Low dietary intake of potassium can lead to increased blood pressure and risk of stroke (IOM, 2005).

Most times, level of blood potassium depletes drastically due to excessive potassium losses through the urine or GIT. Some drugs used in the treatment of hypertension also tend to lower the amount of blood potassium by increasing the amount excreted in urine. On very few occasions, low dietary intake can lead to low blood potassium. Alcoholics with poor dietary pattern, excessive exercise, diarrhoea, vomiting, diuretics and misuse of laxatives can lead to hypokalemia (Grodner*et al.*, 2004; Byrd-Bredbenner *et al*., 2013). In any of these conditions, deliberate efforts must be made to replace potassium lost and prevent lethal complications.

An elevated blood level of potassium is regarded as *hyperkalemia*. This often occurs in patients with renal failure or those with too-rapid intravenous administration of potassium. Symptoms of this condition include mental confusion, poor respiration, numbness of extremities and erratic heart function (Williams, 1997). This can hardly result from dietary intakes, especially in individuals with normal circulation and renal function. It can result in cardiac arrest, if not promptly reversed (Gropper *et al*., 2005). Hyperkalemia results mainly from intake of supplements, especially when the kidneys are not functioning optimally. No upper level has been set for potassium (Grodner *et al*., 2004; Byrd-Bredbenner *et al*., 2013).

2.4.2 Sodium

Over several years in various parts of the world, salt was scarce and of great worth. Thus, excess intake of sodium is a relatively recent challenge. The word *salary* was coined from the Latin word for salt. Table salt or sodium chloride is about 40 percent sodium (Insel, Turner and Ross, 2010).

About 30 percent of the 105 g sodium within the body (70-kg human) is found on the surface of bone crystals; while the remaining is found in the extracellular fluids largely distributed in plasma, and in nerve and muscle tissues. Sodium makes up approximately 93 percent of the cations in the body, making it the most abundant of all cations (Williams, 1997; Gropper *et al*., 2005)

2.4.2.1 Sources of sodium

Sodium is most frequently taken as sodium chloride (NaCl). Many foods naturally contain sodium. However, it is often added to food while cooking and just before consumption, in some cases. Processed foods add large amount of sodium to our menu, depending on the amount and frequency of consumption. Specific food additives increasing dietary sodium intakes are flavour enhancer (monosodium glutamate), preservative (sodium benzoate), leavening agent (sodium bicarbonate also regarded as baking soda), curing agent (sodium nitrite), wetting agent (sodium phosphate), colour preservative (sodium bisulphite), anticaking agents (sodium aluminium silicate), and so on (Grodner *et al*., 2004; Byrd-Bredbenner *et al*., 2013). An unpublished date recently revealed that many Nigerian are increasingly consuming large percentage of their dietary sodium from stews and vegetable soups. Thus, it is most advisable to consume more of unprocessed foods and consciously reduce the table salt added to foods while cooking.

2.4.2.2 Needs and metabolic functions of sodium

For adults that are below 51 years of age, 1500 mg of sodium is recommended as Adequate Intake (AI). AI decreases to 1300 mg for adults between ages 51 and 70 years. Those older than 70 years are to curtail their intakes further to 1200 mg of sodium per day. Only about 200 mg of sodium is needed to maintain normal physiological roles. However, recommendations allow the consumption of a maximum of 2300 mg sodium per day, in an effort to allow for a varied diet (IOM, 2005; Byrd-

Bredbenner *et al*., 2013). For optimal health of individuals, the amount of table salt should be restricted from menu, as much as possible.

Sodium ion (Na^+) protects the volume of fluid in the extracellular compartment. Its concentration determines the direction of water movement by osmosis from one part of the body to another. Thus, it maintains fluid balance. The acid-base balance of the body is usually regulated by Na^+. The Na^+/K^+-ATPase (also called sodium-potassium pump) mediates in the absorption of glucose (at the small intestine) and thus metabolism of this substrate as well as cellular exchange of Na^+. Similarly, sodium is involved in the absorption of some amino acids in the small intestine. Sodium ions, along with K^+, are vital in the transmission of electrochemical impulses along nerve and muscle membranes; thus maintain normal excitability or irritability of the muscle. Muscle contraction is mainly dependent on these essential roles of Na^+ and K^+(Williams, 1997; Byrd-Bredbenner *et al*., 2013).

2.4.2.3 Absorption, transport and excretion of sodium

About 95 to 100 percent of sodium ingested is normally absorbed. The intestinal tract is the major point of absorption of virtually all dietary sodium intakes. There are three main routes of absorption of sodium, which are: the Na^+/glucose co-transport system – this functions broadly throughout the small intestine; the electro-neutral Na^+ and Cl^- co-transport system – this works both in the small intestine and the proximal portion of the colon; and the electro-genic sodium absorption mechanism – this works primarily in the colon (Gropper *et al*., 2005).

High sodium intake leads to the excretion of the excess through the kidneys. When the level of sodium in the blood is low, aldosterone is released by the adrenal cortex to limit the excretion of sodium via the kidneys. Some sodium (0% to 5%) is usually lost through faeces, tears from eyes and perspiration (Byrd-Bredbenner *et al*., 2013).

2.4.2.4 Deficiency and toxicity of sodium

Due to the presence of significant amount of sodium in various foods and low requirements, deficiency of sodium is rare. Excessive sweat for a prolonged period can lead to sodium deficiency. If such happens, symptoms such as dizziness, shock, coma,

muscle cramps and nausea can be experienced (Smith and Ojofeitimi, 1995; Gropper *et al*., 2005; Byrd-Bredbenner *et al*., 2013).

The upper level for sodium intake by adults is 2300 mg per day. Excess of sodium in blood circulation usually stress the kidneys. For individuals that are sodium sensitive, they develop oedema and hypertension in response to high sodium intake. Occasional very salty meals may produce oedema, but not hypertension. Sodium intakes higher than 2000 mg per day tend to raise calcium excretion via the urine. This creates concern on bone health. However as at date, no direct relationship has been established between osteoporosis and excess sodium intakes (Grodner *et al*., 2004; Borghi*et al.*, 2006; Heaney, 2006b; Insel *et al*., 2010; Byrd-Bredbenner *et al*., 2013). The best way out of sodium toxicity (or high sodium intake) is the intake of additional water to minimize pressure on the kidneys. Ultimately, conscious efforts should be made to reduce sodium intakes to less than 2300 mg per day as this might improve general health of majority by reducing the risk of cardiovascular diseases.

2.4.3 Calcium

Calcium is one of the divalent cations with an atomic mass of 40 g/mol. It is the fifth most abundant element in human body, following oxygen, carbon, hydrogen, and nitrogen. It makes up about 1.9 % of the body by mass (Nordin, 1976). Analyses of human remains show that it constitutes 0.1–0.2 % of early foetal fat-free body mass, which increases to about 2 % of adult fat-free body mass. Concretely, this represents an increase from about 24 g (600 mmol) at birth to 1300 g (32.5 mol) at adult, requiring an average daily positive calcium balance of 180 mg (4.5 mmol) during the first 20 years of life (FAO/WHO, 2004).

Most body calcium (about 99%) is stored in the teeth and bones. In the bones, it exists as hydroxyapatite ($Ca_{10}[PO_4]_6[OH_2]$). Bone remodelling takes place on a daily basis with as much as 700mg calcium being withdrawn and replaced, depending on complex regulation of the body metabolic needs. Apart from the calcium available in bones and teeth, the remaining body calcium exists in the plasma and other body fluids, where they carry out various indispensable metabolic roles. Normal serum calcium is about 10 mg/dl (2.5 mmol/dL) and is stringently regulated between 9 and 11 mg/dL (2.25

and 2.75 mmol/dL) by the actions of calcitriol (1, 25-dihydroxy cholecalciferol) and parathyroid hormone, along with calcitonin (Williams, 1997).

2.4.3.1 Sources of calcium

The dairy products are the major sources of calcium. They include milk (skimmed, low fat and whole), milk products like ice milk, yogurt, cheese, ice cream and puddings. Cottage cheese, cream cheese and butter are though dairy products but not good sources of calcium due to great loss of calcium during processing and or their high fat content. Other good sources of calcium but of non-dairy origins are small fish with soft bones (like sardines, titus and *shawa*), crustaceans (crabs, crayfish, shrimps), mollusks (snails), legumes and tofu fortified with calcium (Smith and Ojofeitimi, 1995; Grodner *et al*., 2004).

Other sources of calcium are the green leafy vegetables (such as bitter leaf (*Vernonia amygdalina*), *soko* (*Celosia argentea*), *tete* (*Amaranthus hybridus*), *ewedu* (*Corchorus olitorius*), water leaf (*Talinum triangulare*), *ugu* (*Telfairia occidentalis*), *ukase* (*Gnetum africanum*), etc.), However, calcium bioavailability from green leafy vegetables is usually limited by the phytate and oxalate contents of these food items. The tannin content of foods is also known to limit the bioavailability of calcium (Smith and Ojofeitimi, 1995; Grodner *et al*., 2004). So, great care should be exercised in relying on food sources high in these anti-nutrients as main calcium supply. Limiting the intake of teas and coffee can also be useful to increase calcium absorption.

Many forms of calcium supplements are also vital to meet calcium needs by humans. They include calcium carbonate, calcium monophosphate, calcium lactate, calcium gluconate, and calcium acetate (Gropper *et al*., 2005). These should however be taken as recommended by a licensed physician. They may not be needful if adequate balanced diets are taken consistently.

2.4.3.2 Needs and metabolic functions of calcium

The Food and Nutrition Board set adequate calcium intake at 1000 mg per day for adult men and women of ages 19 to 50 years. Adults of 51 years and above were to take 1200 mg calcium per day (IOM, 2011). The National Institute of Health (NIH)

panel on osteoporosis further recommended 1500 mg daily calcium intake for postmenopausal women (NIH, 1994).

The metabolic functions of calcium have been divided into three main sections including bone formation, teeth formation and general metabolic functions. As previously mentioned, 99 percent of the body calcium participates in the bone and teeth formation, while the remaining 1 percent carries out the general metabolic functions.About 99 percent of body calcium serves to build and maintain the bones as well as development of cartilage into bone. The osteoblast (bone-building cells) and osteoclast (bone-resorbing cells) are the two groups of cells involved in this strictly regulated bone formation(Williams, 1997; Gropper *et al*., 2005).

Calcium along with some other constituents is mainly utilized by the ameloblasts, which form the teeth. Once these materials are deposited by the ameloblasts, exchange of minerals continues as it occurs in bones. As for the general metabolic functions, five main roles have been identified as summarized subsequently. Calcium is needed for cross-liking of fibrin in blood clotting, thereby stabilizing the fibrin threads. It is very essential in nerve impulse transmission. The release of calcium at the neuromuscular junction causes acetylcholine to eventually stimulate muscle fibre. The release of calcium from the sarcoplasmic reticulum of the myofibril in response to stimulus is the underlying factor controlling muscle contraction. Once the calcium ions are sequestered back in the reticulum, muscle relaxation ensues. Ionized calcium is also notable to control the cell membrane permeability to some substances. Last, four calcium ions are known to bind with each calmodulin molecule for the regulation of certain enzymes in various biochemical pathways, including the one involved in initiating glycogen breakdown (Williams, 1997; Gropper *et al*., 2005; Weaver and Heaney, 2006).

2.4.3.3 Digestion and absorption of calcium

The calcium in foods and supplements are relatively insoluble. Calcium is only absorbed in its ionized form, thus must be released from the salts. The acidic pH of the stomach usually enables the solubilisation of the calcium salts within about an hour (Heaney*et al.*, 1990).

Generally, growing children can absorb up to 75 % of dietary calcium, while adults absorb about 30 %. Absorption of calcium involves two main transport processes occurring in the small intestine. The first transport process functions in the duodenum and proximal jejunum. It is saturable, requires energy, involves a Calcium-Binding Protein (CBP, also called calbindin D_{9k}) and is regulated by calcitriol (1,25-dihydroxy cholecalciferol). The calcitriol-dependent calcium transport system is stimulated once low-calcium diet is taken, especially intakes <400 mg. Increased calcium demand as necessitated by pregnancy, lactation and growth can also activate this absorption mode. Calcium absorption by this active process involves three steps regulated by calcitriol: first, transport across brush border membrane; second, intracellular movement; third, extrusion over basolateral membrane. Low plasma level of ionized calcium lead to an increase in Parathyroid Hormone (PTH) secretion, which further leads to a release of calcitriol and eventually enhances absorption of calcium at the intestinal wall. Calcitriol-induced absorption of calcium is usually made possible by the stimulation of the synthesis of calbindin. Aging, high plasma phosphorus level and oestrogen deficiency often limit the efficiency of this absorption pathway (Sheikh*et al.*, 1987; IOM, 1997; Gropper *et al*., 2005).

The second calcium absorption route occurs throughout the small intestine but especially in the jejunum and ileum. It is non-saturable and passive. This passive paracellular absorption is suspected to occur when the amount of calcium ingested is high. This absorption mode is predominant when the calcium intake is higher (Gropper *et al*., 2005).

The large intestine might also play key role in calcium absorption. Colonic bacteria can release some calcium from pectin, thereby increasing calcium bioavailability. About 4-10 percent of dietary calcium is absorbed in the colon on daily basis. This has made some expert to trace colon cancer to calcium-deficient diet (IOM, 1997).

Factors influencing absorption of calcium

Vitamin D is known to enhance the absorption of calcium. Intake of lactose, other sugars, sugar alcohols and proteins can enhance the absorption of calcium as well (Ziegler and Fomon, 1983; Schaafsma, 1988; Hamalainen, 1994). Fibre as well as phytate may decrease calcium absorption. Phytates bind calcium, limiting its

bioavailability, especially when the phytate : calcium molar ratio is greater than 0.2 (Greger, 1987; Harland and Oberleas, 1987). Oxalate, found in vegetables, fruits, nuts and beverages, may chelate calcium and increase its faecal excretion as it limits its intestinal absorption (Gropper *et al*., 2005).

Divalent cations, along with other minerals, are notable to competitively limit the intestinal absorption of calcium. Magnesium and zinc are known to play significant roles here. The presence of large amount of unabsorbed fat (>7 g faecal fat per day) in the gastrointestinal tract is known to also limit calcium absorption; through the formation of insoluble calcium soaps. Calcium absorption from calcium supplements also vary with the type of salt present in the supplement (Spencer, 1986; Sheikh *et al*., 1987; Gropper *et al*., 2005). Sedentary lifestyle limits calcium absorption and hence leads to low bone density. Medications such as cortisone, tetracycline, thyroxine, anticonvulsants and aluminium-containing antacids are known to be linked with lowered absorption of calcium (Grodner *et al*., 2004).

Age tends to influence the efficiency of absorption. Absorption efficiency is highest in children below 18 years, followed by mature adults, and then the aged ones (Weaver, 1994). Also, post-menopausal women are less efficient in absorbing calcium compared with pre-menopausal women (Hope*et al.*, 1992a). Calcium absorption has been notably enhanced by testosterone. This could be a part explanation as to why males have been noted to have greater absorption of calcium than females of the same age (Hope*et al.*, 1992b; Weaver, 1994).

2.4.3.4 Transport, blood regulation and excretion of calcium

There are three main forms in which blood calcium is being transported. Free (ionized) calcium in blood constitute about 50%; about 10% calcium is complexed with sulphate, phosphate, or citrate; while the remaining approximately 40% is usually bound to albumin and pre-albumin (Gropper *et al*., 2005).

The levels of calcium are strictly controlled intracellularly and extracellularly. Intracellularly, the calcium level is usually kept low and maintained as such by ATP-dependent calcium pumps and by the sequestering of calcium in organelles such as mitochondria, endoplasmic reticulum, vesicles and nucleus. Extracellularly, calcitriol,

PTH and calcitonin are the hormones closely involved in the homeostasis of blood calcium. An overview of the extracellular control is shown in Figure 2.1 (Brown, 1991; Gropper *et al*., 2005).

When blood calcium level is low, parathyroid gland is stimulated to secrete the PTH, which acts on the bone and the kidneys simultaneously. In the kidneys, PTH reduces calcium loss by enhancing its reabsorption. In the bone, PTH enhances calcium release. It also inhibits synthesis of collagen by osteoblasts, thereby stalling osteoblastic-related processes. On the other hand, PTH stimulates the osteoclasts activities indirectly by increasing the loss of inorganic phosphates via the kidneys. As the phosphates are being lost, $Ca_5(PO_4)_3OH$ leaches from the bone and thereby raises the blood level of calcium. Last, PTH also stimulated the synthesis of 1,25-dihydroxycholecalciferol (calcitriol) that further stimulates the calcium uptake at the intestine (Gropper *et al*., 2005; Berdanier and Zempleni, 2009).

Conversely, when blood level of calcium is high, calcitonin is secreted by the parafollicular cells of the thyroid gland and works opposite direction to the PTH. Calcitonin stimulates the osteoblasts and thus lowers serum calcium by simultaneously inhibiting the activities of the osteoclasts and limiting the movement of calcium from the bone. It further inhibits the activation of calcitriol and prevents conservation of calcium by the kidneys. Consequently, PTH-calcitriol and calcitonin complexly regulate blood calcium to allow for very minimal variation in normal individuals (Jaros*et al.*, 1984; Gropper *et al*., 2005)

The excretion of calcium mainly takes place via the urine and faeces. However, under extreme sweating, up to 182 mg calcium can be lost on daily basis through the skin. In most cases, calcium is usually filtered and reabsorbed through the kidneys; so that urinary calcium loss is around 100 to 240 mg (average of 170 mg) on daily basis. Decrease in urinary calcium excretion can be experienced when PTH is secreted, in the presence of potassium, phosphorus, magnesium and boron. On the other hand, it can be increased when protein, boron + magnesium, sodium and caffeine are present. Calcium loss through the faeces is about 45-100 mg daily. When the dietary fibre, phytate and oxalate levels are higher, faecal excretion is increased. Fat malabsorption

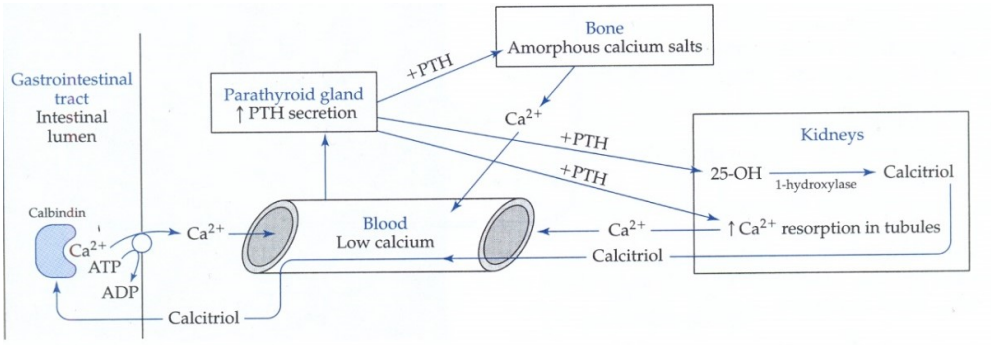

Figure 2.1: An overview of blood calcium regulation by parathyroid hormone (PTH) and calcitriol in response to low blood calcium concentrations (Gropper *et al*., 2005)

disorders and excess magnesium intake can also lead to increased calcium excretion via the faeces (Charles*et al.*, 1991; Calvo*et al.*, 1991; Lemann*et al.*, 1993).

2.4.3.5 Deficiency and toxicity of calcium

Calcium deficiency can result from inadequate calcium intake, poor calcium absorption and/or excessive losses of calcium. The bones and muscles are often affected by poor intakes of calcium. Once the bone density is lower than normal range in children, rickets develop. Hypocalcemia (low blood free ionized calcium level) can lead to tetany, a condition showing intermittent muscle contractions that fail to relax, especially in muscles of the arms and legs (extremeties). Other common signs of tetany are muscle pain, muscle spasms and tingling or numbness in the hands and feet (paresthesia). Deficiency of calcium in adults leads to the development of osteoporosis, that is, a loss of bone mass (bone minerals and protein matrix). This could be either type I, which is usually found in post-menopausal women between 50 and 65 years, and basically affect the vertebrae and distal radius; or type II, which presents in men and women above 70 years, affecting the hips, pelvis, vertebrae, humerus and tibia (Harward, 1993; Sowers, 1993; Nieves*et al.*, 1998).

A tolerable upper limit of 2500 mg per day has been recommended for adults of ages 19 to 50 and 2000 mg per day for those that are 50 years and above. Intakes of calcium greater than this threshold will lead to hypercalcemia and deposition of calcium in soft tissues alongside with alkalosis of the system. Calcification of the soft tissues is usually the case in patients that have renal failure, especially when their plasma calcium and phosphorus are high. Hypercalcification sometimes leads to constipation, headache, decreased absorption of other minerals and irritability; and patients with idiopathic hypercalciuria (daily urinary calcium >4 mg/kg body weight) can further develop kidney stones containing calcium, if they take calcium in excess (Brown and Wolfson, 1993; IOM, 1997; Patel and Goldfarb, 2010; Byrd-Bredbenner *et al*., 2013).

2.4.4 Magnesium

Magnesium was first found in Magnesia (within Greece). It is a silvery white substance that is largely found in the soil and ocean water (Byrd-Bredbenner *et al*., 2013). It is the 4th most abundant cation in human body, but the 2nd most abundant,

after potassium, within the cells. It makes up about 0.05% of human body weight, of which 55-60% is found in bones and 20-25% in muscle and other soft tissues. Only 1% of body magnesium is found in the extracellular fluids. Plasma magnesium ranges from 1.6 to 2.2 mg/dL (Gropper *et al*., 2005; Insel *et al*., 2010).

2.4.4.1 Sources of magnesium

Magnesium is widely found in nature, especially in foods that are not processed. Magnesium-rich foods are nuts, legumes, and whole grain cereals (especially barley and oats). Green leafy vegetables are also very rich in magnesium, due to their chlorophyll contents. Other good sources include chocolate, parsley, peas, carrots and brown rice. Animal products such as milk, meat, eggs and some seafood are known to supply moderate amount of magnesium. Hard tap water is known to contain magnesium (as well as calcium) (Williams, 1997; Gropper *et al*., 2005; Insel *et al*., 2010; Byrd-Bredbenner *et al*., 2013).

Magnesium forms like magnesium sulphate ($MgSO_4$), magnesium chloride ($MgCl_2$), magnesium oxide (MgO), magnesium gluconate, magnesium lactate, magnesium citrate, and magnesium acetate, are found in multivitamins and mineral supplements. Intake of magnesium supplements at different time from when other mineral supplements (especially iron) are taken is quite vital to enhance magnesium bioavailability (Gropper *et al*., 2005). Magnesium contents of some food are usually adversely affected by food preparation and processing. Removal of germ and outer layers of whole wheat can deplete up to 80 percent of the magnesium content of this whole grain (NRC, 1989).

2.4.4.2 Needs and metabolic functions of magnesium

The Recommended Dietary Allowances (RDAs) for magnesium for men and women 19 to 30 years are 400 mg/day and 310 mg/day respectively with an addition of 10 to 20 mg/day magnesium for adults above 30 years. Women of ages 19 to 30 years should take 350 mg magnesium when pregnant, whereas those from 31 to 50 years should take 360 mg. As for those lactating, women 19 to 30 years should take 310 mg and those from 31 to 50 years should take 320 mg magnesium (IOM, 1997).

Magnesium ion along with calcium ion is involved in the regulation of PTH level. Raised level of magnesium ion reverses the effect of calcium ion on PTH. It is very vital in protein synthesis as it enables aggregation of ribosome and the binding of messenger RNA to ribosome subunits. A change in intracellular level of magnesium ion yields neuromuscular response to stimuli. Lower level of cellular magnesium leads to inhibition of cardiac and smooth muscle contraction. Activation of several enzymes in the oxidative breakdown of glucose as well as some of those involved in oxidative phosphorylation requires magnesium ion. DNA synthesis and catabolism as well as RNA transcription cannot be achieved without magnesium. Thus, magnesium is required in the molecular processes leading to cellular growth and reproduction. The acyl CoA synthetase of the fatty acid β-oxidation pathway requires magnesium. It is also involved in the regulation of potassium ion channels and some other ion channels (Williams, 1997; Gropper *et al*., 2005).

2.4.4.3 Absorption, transport and excretion of magnesium

The absorption of magnesium occurs throughout the small intestine, but especially at the distal jejunum and ileum. It is usually absorbed by passive diffusion when dietary intake is very high or by a carrier-mediated active transporter, when intake is low. Increased absorption takes place when the dietary intake is low and or when magnesium status is below marginal value; and decreased absorption takes place when dietary intake is high (Fine*et al.*, 1991; Hardwick*et al.*, 1991; Kayne and Lee, 1993; Williams, 1997). A schematic representation of magnesium absorption and transport is shown in Figure 2.2.

Several factors are known to influence the absorption of magnesium in the intestine. Large amount of unabsorbed fatty acids tend to bind and limit the absorption of magnesium. Phytate and some non-fermentable fibres are known to slightly lower the amount of magnesium absorbed. Calcium and phosphorus can inhibit magnesium absorption by the formation of complexes in the intestinal tract (Brink and Beynen, 1992; Siener and Hesse, 1995; Coudray*et al.*, 2003; Gropper *et al*., 2005). On the other hand, some studies have reported protein as enhancing the absorption of magnesium. More so, pharmacological doses of vitamin D and carbohydrates like

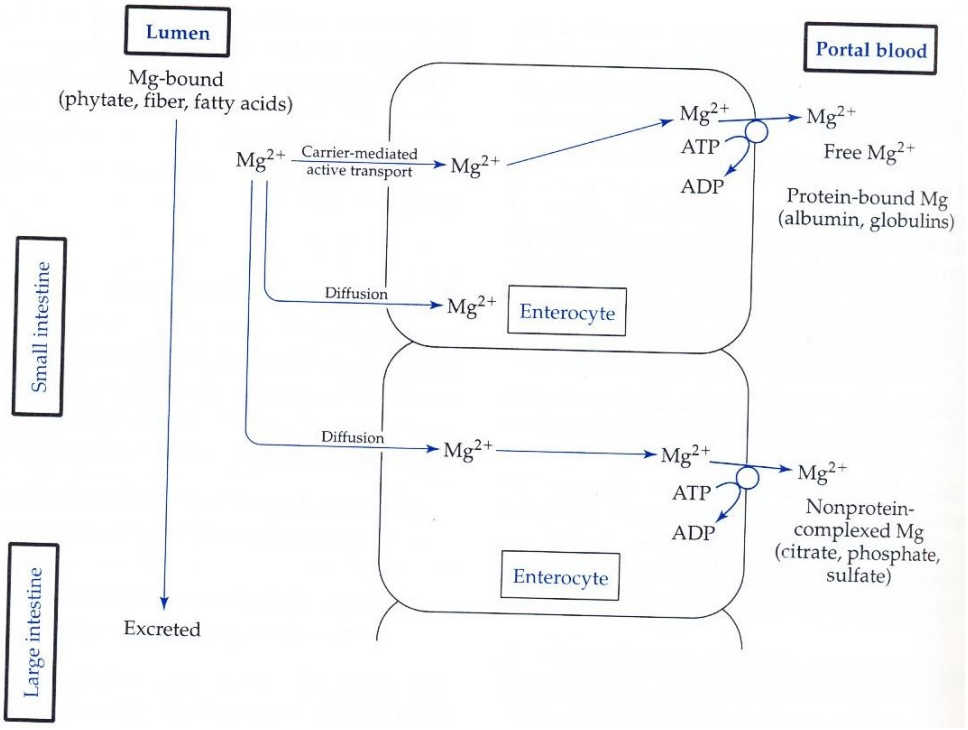

Figure 2.2: Magnesium absorption and transport (Gropper *et al.*, 2005)

lactose and fructose have been reported to stimulate the absorption of magnesium (Ziegler and Fomon, 1983; Holbrook*et al.*, 1989; Hardwick *et al*., 1991; Brink and Beynen, 1992; Rude, 1993; Milne and Nielsen, 2000).

The forms in which magnesium is transported are quite diverse. Within the plasma, about 13 percent is complexed with citrate, phosphate, sulphate or other ions; about 33 percent is bound to protein (30 percent usually bound to albumin and 3 percent bound to globulin) and the remaining 50 to 55 percent is free. The gastrointestinal absorption, renal excretion and cation flux across the membrane are the major factors coordinated to maintain the plasma magnesium between 1.6 and 2.2 mg/dL. Parathyroid hormone is known to control these factors in a way to raise plasma magnesium level eventually (NRC, 1989; Gropper *et al*., 2005).

Studies have shown that when appropriate amount of magnesium is ingested only about 5 percent is usually excreted via the kidneys. About 65 percent is reabsorbed at the loop of Henle and 20 to 30 percent is reabsorbed in the proximal tubule (Rude and Singer, 1981; Elin, 1987; Wester, 1987). Depending on the extent of sweat released, up to 15 mg of magnesium can be excreted through the sweat per day (Wester, 1987). Intake of excess magnesium will lead to decreased absorption of the mineral and excretion of large amount in the faeces (NRC, 1989). Alcohol, caffeine consumption, diuretic medications and protein tend to increase urinary excretion of magnesium; whereas PTH yields an opposite effect (McCollister*et al.*, 1963; Mahalko*et al.*, 1983).

2.4.4.4 Deficiency andtoxicity of magnesium

Deficiency of magnesium is often linked to the presence of other diseases. Poor magnesium status can be related to hypertension, renal disease, postsurgical complications, toxaemia in pregnancy or diabetes mellitus (Wester, 1987; Rude, 1993; Ma*et al*., 1995; Frakes and Richardson, 1997). Conditions like excessive vomiting, and/or diarrhoea, diuretic use, burns, protein malnutrition, parathyroid disease or excessive alcohol use tend to raise the possibility of developing magnesium deficiency (Williams, 1997, Gropper *et al*., 2005). Plasma magnesium level of < 1.5 mg/dL is regarded as hypomagnesemia and it occurs shortly after magnesium deficiency, leading to a number of other biochemical changes (Fatemi*et al.*, 1991; Kalepouris and Agus, 1998).

Poor magnesium deficiency resulting from inadequate dietary magnesium has not reported but deficiency has been induced in humans under research protocols. This led to symptoms such as hallucination, vomiting, personality changes, nausea, spasms and tremors, muscle weakness and anorexia. Other related complications could lead to death, if left uncontrolled (Rude and Singer, 1981). Animals deficient in magnesium often become very irritable and with severe deficiency eventually suffer convulsions and usually die (Wardlaw and Hampl, 2007).

The Food and Nutrition Board of Institute of Medicine (1997) recommended upper intake level of 350 mg magnesium from supplement and other non-food sources for individuals that are 9 years and above. An excessive intake of magnesium is unlikely to yield toxicity, except in those with kidney disorders (Wester, 1987). Older people are at higher risk of toxicity due to possible impaired renal function (Wardlaw and Hampl, 2007). Intake of magnesium salt ($MgSO_4$) up to 3-5 g may have cathartic effect yielding nausea, double vision, diarrhoea, dehydration, slurred speech and plasma level of about 9-12 mg/dL. Acute magnesium toxicity from excessive intravenous infusion can even result in depression and paralysis (IOM, 1997).

2.5 Selected Microminerals

2.5.1 Copper

2.5.1.1 Chemistry and sources of copper

For several years, copper alloyed with tin has been utilized by humans to build various useful items. However, it has just been in recent time that it was recognized as an essential nutrient for man and other animals. In the 1920s copper was discovered to be needed, in addition to iron, for haemoglobin biosynthesis. This was realised when anaemic animals treated with iron showed no improvement until copper was provided alongside. Early work of Cartwright and fellow researchers (Hart*et al.*, 1928) indicated the relationship between the two metals relative to heme biosynthesis. Additional functions of copper have been reported from that time (Frieden, 1983).

Copper (Cu) is one of the transition metals in the fourth period of the periodic table. It has an atomic mass of 29 and a molecular mass of 63.4 and usually exists in two oxidation states, cuprous (Cu^{+}) and cupric (Cu^{2+}). There are two naturally occurring

isotopes, which are Cu^{63} and Cu^{65}; and two radioisotopes which are ^{64}Cu and ^{67}Cu. The ^{64}Cu has a half-life of 12.7 hours, whereas ^{67}Cu has a half-life of 62 hours (Berdanier and Zempleni, 2009).

Copper is available in nearly all foods in varying amounts. Legumes and nuts are rich in copper, whereas dairy products are poor sources. Shellfish, whole grains, beef liver, shrimp and raisins are excellent sources as well. With low intakes, absorption is markedly higher (56% of intake) than when intake is high (12% of intake). The form of copper (cuprous or cupric) ingested determines the percentage of intake that is absorbed eventually (Johnson and Kays, 1990). Ascorbic acid, zinc, tin, and iron tend to affect the absorption of copper negatively.

2.5.1.2 Metabolic functions of copper

Copper, zinc and iron are all involved in the regulation of gene expression. Copper, relative to metallothionine, has been described extensively by Chen*et al.* (1985).A specific metallothionine locus (CUP1) encodes metallothionine, a 6570 molecular weight protein that binds heavy metals. The CUP1 promoter responds to copper indirectly. Rather, this role is played by an Upstream-Activating Sequence (UAS) present as a tandem sequence designated UASp and UASd located between -105 and -108 bp from the transcription start site. Both copper and zinc are required for the synthesis of metallothionine (Berdanier and Zempleni, 2009).

Ten genes have been shown to require copper response element for their expression (Wang*et al.*,1996). Seven of these had substantial similarity with: ferritin mRNA, fetuin mRNA, mitochondrial 12S and 16S rRNA, and with mitochondrial tRNA for phenylalanine, valine and leucine. These similarities suggest importance of copper in mitochondrial gene expression, which in turn relates to the observation of lower oxidative phosphorylation in rats that are deficient of copper (Hoshi*et al.*, 1993; Johnson*et al.*, 1995; Matz*et al.*, 1995).Copper is a vital part of a mitochondrial respiratory enzyme, cytochrome C, and it serves as a cofactor for several enzymes listed below. Of the remaining RNAs identified as having a copper–response element, no gene product has yet been found. The gene products might be enzymes requiring copper as a cofactor or could be copper transport proteins. This would follow similar pattern as the gene expression regarding zinc and iron. Enzymes requiring copper as a

cofactor include ascorbate oxidase, lysyl oxidase, α-amidating enzyme, monoamine oxidase, dopamine β hydroxylase, amine oxidase, cytochrome C oxidase, cytoplasmic superoxide dismutase, ferroxidase II, metallothionine, phenylalanine-4-monooxygenase and tyrosine oxidase (Berdanier and Zempleni, 2009).

2.5.1.3 Absorption, transport and excretion of copper

Copper absorption takes place mainly in the small intestine and partly in the stomach (Weiss and Linder, 1985; Fields*et al.*, 1986; Turnlund*et al.*, 1989). Copper status largely determines the level of absorption; where the need is great, uptake is high and vice versa. The amount absorbed is also affected by the food combination eaten and on the presence of other divalent minerals that may compete for uptake. Generally, efficiency of absorption is quite low with an average uptake of 12%. Copper absorption is negatively affected by phytate (Sandstead, 1982) and by zinc (Oestreicher and Cousins, 1985; Weiss and Linder, 1985). The unabsorbed copper is usually excreted in the faeces. Copper transported to the intestine from the liver via the bile is also excreted in the faeces. About 2 mg copper per day is excreted via the bilary route. Small amount of copper is also excreted in the urine and via the skin and hair. The amount lost through the bilary excretion, urine, skin and hair ranges from 12 to 43 percent of the dietary intakes. The copper excreted in urine by human is quite small, about 10 to 50 μg per day (Linder and Roboz, 1986).

Once copper is absorbed by the enterocyte, it is passed to the blood where it binds to either albumin or transcuperin (Harris, 1991). The half-life of albumin-bound copper is about 10minutes. The copper is then transported to the liver where it is incorporated into *ceruloplasmin*, which is an alpha-globulin transport protein. Eachceruloplasmin molecule can carry six atoms of copper. Blood levels of copper are about 1 mg/L whereas ceruloplasmin is about 150-600 mg/L. This is because ceruloplasmin is not only useful in transporting copper to all parts of the body; it also has enzyme activities as a ferroxidase, an amide oxidase, and as a superoxide dismutase. As a ferroxidase it helps in releasing iron from its liver storage sites to transferrin in the plasma. It is needed in the conversion of ferrous iron to ferric iron and in the linkage of the ferric iron to napotransferrin to form transferrin which, in turn, transports the iron to the reticulocyte for synthesis of haemoglobin. Its role in iron metabolism relates to the

cuprous-cupric interconversion. Averagely, the copper carried by the ceruloplasmin is 50 percent cuprous and 50 percent cupric (Berdanier and Zempleni, 2009).

Although ceruloplasmin seems to be the most active of the copper transporters, other transporters still participate in the transportation of copper to the cells for storage and or utilization. Albumin can serve this role as well as a 270-kDa protein, transcuperin, and amino acids, such as histidine (Weiss and Linder, 1985).The liver is the main user and storage site for copper. The copper levels in the liver remain relatively constant from time to time. Bilary excretion and ceruloplasmin release are the major mechanisms used to regulate copper levels in the liver. Ceruloplasmin contains most of the body copper: 70-90 percent of approximately 1 μg/mL copper in the plasma (Berdanier and Zempleni, 2009).

Trancuperin is known to compete with albumin at the intestine for copper, yet it acts in the portal circulation as a donor of copper to albumin. The existence and roles of transcuperin in the maintenance of copper status has not been fully elucidated. Although the transport of copper in the blood has received considerable attention, its transport into the cell has not been well studied. Copper moves through the plasma membrane via fixed membrane transporter proteins. These membrane proteins may either reversibly bind the copper or form channels through which the copper passes. The kinetics of copper transport has been studied. The Km values are uniformly in the low micromolar range, whereas the Vmax is varies greatly, depending on cell type, incubation conditions and media used (Berdanier and Zempleni, 2009).

2.5.1.4 Deficiency of copper

Copper deficiency is rare in humans, especially those regularly taking varied food items. A major characteristic of copper deficiency is anaemia and poor wound healing akin to what is seen in the deficiency of vitamin C. This anaemia does not resolve with iron supplementation. The indispensable roles of copper in the synthesis of connective tissue, especially collagen makes individuals deficient of this mineral to show symptoms like weakness, osteoporosis, lassitude, joint ache, small petechial haemorrhaging, and arterial aneurysms. Heart rupture is a frequently reported characteristic in copper-deficient rats (Wildman*et al.*, 1995).

Central nervous system degeneration can be equated to a decline in respiratory-chain activity; however, in the order of enzymes needing copper to act, this enzyme is about the last to be affected in a copper-deficient animal. Reduced immune response has also been reported. Copper-deficient animals have been shown to have lowered T lymphocyte and neutrophil activities (Hopkins and Failla, 1995).Copper and chromium depletion has been reported to negatively affect lymphocyte proliferation. Both minerals are needed simultaneously to elicit this response to a mitogen challenge (Rhee*et al.*, 2004).

Other signs of the deficient state include increased levels of plasma cholesterol, neutropenia, achromatism, twisted, kinky hair, and hemacytic, hypochromic anaemia (Klevay*et al*., 1984).Copper-deficiency cam cause elevated cholesterol levels in the blood, elevated 3-Hydroxy-3-Methyl-Glutaryl-Coenzyme AReductase (HMGCR) activity, and increased hepatic glutathione levels in rats (Kim*et al.*, 1992).If the rise in glutathione level is inhibited, then the hypercholesterolemia associated with copper deficiency is truncated. Heme biosynthesis is lowered in copper deficient swine (Lee*et al.*, 1968).

2.5.1.5 Toxicity of copper

Under normal circumstances, ingestion of excess copper is rare. Toxicity has been reported in children taking accidental overdose, those taking copper-contaminated food or water and in Wilson disease (a genetic disorder emanating from excess storage of copper). Although copper toxicity can develop if the exposure is high enough and long enough, the body can protect itself from occasional excess intake by lowering its level of absorption. The symptoms of toxicity include nausea, vomiting, diarrhoea and abdominal pain (Berdanier and Zempleni, 2009; Byrd-Bredbenner *et al*., 2013). Turnlundhas reported that young men consuming between 0.75 and 7.53 mg copper per day were able to attain positive copper balance regardless of intake. Likely, the figure given for optimal intake is a little high because of the paucity of data on copper status under controlled conditions (Turnlund *et al*., 1989). The upper level for copper is 10 mg per day and intakes higher than this tend to increase the risk of liver damage (IOM, 2001).

There are two genetic disorders that have enhanced the understanding of the function and metabolism of copper. In the first one, Menke's syndrome, copper absorption is faulty. Intestinal cells absorb the copper but cannot release it into the circulation for use.Parenteral copper can correct most of the conditions that look like copper deficiency, but care must be taken in its administration. Overdependence on this route can easily yield toxicity. Unfortunately, parenterally administered copper does not reach the brain and cannot prevent the cerebral degeneration and premature death characteristics of patients with Menke's disease (Prohaska, 1986; Kelly and Palmiter, 1996).

Another genetic disorder relative to copper toxicity is Wilson's disease.This condition is also associated with premature death and is due to a poor incorporation of copper into cerulloplasmin and lowered bilary excretion of copper. This results in an accumulation of copper in the liver and brain. Early signs of Wilson's disease include liver dysfunction, neurological disease, and deposit of copper in the cornea manifested as a ring that looks like a halo around the pupil. This lesion is called the Kayser-Fleischer ring. Renal stones, renal aciduria, neurological deficit, and osteoporosis also characterize Wilson's disease. Periodic bleeding, which removes some of the excess copper, can be helpful in managing Wilson's disease as can treatment with copper-chelating agents such as D-penicillamine, and increasing the intake of zinc, which limits copper absorption (Yang*et al.*, 1983; Reed*et al.*, 1995; DiDonato*et al.*, 1997; Yang*et al.*, 1997).

2.5.2 Manganese

2.5.2.1 The essentiality of manganese

Manganese (Mn), an essential trace mineral, found in all tissues is required for normal amino acid, lipid, protein, and carbohydrate metabolism. Manganese-dependent enzyme families include oxidoreductases, transferases, hydrolases, lyases, isomerases, and ligases. Manganese metalloenzymes include pyruvate carboxylase, arginase, isocitrate dehydrogenase, glucokinase, glutamine synthetase, phosphoenolpyruvate decarboxylase, acetyl-CoA carboxylase and manganese Superoxide Dismutase (Mn-SOD). The optimal functioning of several organ systems is dependent on manganese. It is needed for normal immune function, regulation of blood glucose level and cellular

energy metabolism, reproduction, digestion, bone growth, and it aids in defense mechanisms against free radicals. Manganese, along with vitamin K supports blood clotting and hemostasis (Aschner and Aschner, 2005; Berdanier and Zempleni, 2009).

Due to insufficient data, no formal Recommended Dietary Allowance (RDA) for manganese has been established, but the US National Research Council has established an Estimated Safe and Adequate Dietary Intake (ESADDI) of 2–5 mg per day for adults (Greger, 1998). Furthermore, the Institute of Medicine (2001) has established an Adequate Intake (AI) for manganese. Adequate intake is defined as a nutrient consumption value that is experimentally derived or is an approximation of an observed mean nutrient intake for a group of apparently healthy individuals. An AI is established when there is not sufficient scientific evidence to calculate an Estimated Average Requirement (EAR). The EAR is the daily intake value that is estimated to meet the nutritional requirement, as defined by a specific indicator of adequacy, in one-half of the apparently healthy individuals in a life stage or gender group. The AI replaces the ESADDI. The manganese AI for adult men and women is 2.3 and 1.8 mg per day, respectively. Developmental life stage can also influence dietary manganese requirement. Adequate intakes for newborn (< 6 months of age) infants are approximately 3 µg per day while intakes increase to 600 µg per day by 7–12 months of age. Children between 1–3 and 4–8 years of age have manganese AI of approximately 1.2 and 1.5 mg per day, respectively. Pregnant and lactating women (14 to 50 years) have AI of 2.0 and 2.6 mg per day respectively (IOM, 2001).

2.5.2.2 The absorption, distribution, and elimination of oral manganese

Manganese is poorly absorbed via the gastrointestinal tract. The use of radioactive manganese (^{54}Mn) has helped to establish that about 1 to 5 percent of the ingested manganese eventually reaches blood circulation (Davis*et al.*, 1993). In adults the net absorption from gut (mean ± SD) of radiolabeled ^{54}Mn from a meal containing 1 mg manganese is 1.35 ± 0.51 and 3.55 ± 2.11 percent for adult men and women, respectively (Finley*et al.*, 1994). The mean (±SD) retention 10 days after ingestion of 0.3 mg manganese was reported as 5.0 ± 3.1 percent in young adult women (Davidsson*et al.*, 1988). Gender differences also exist for manganese absorption, with men absorbing significantly less manganese compared to women. It has been

postulated that reduced gut manganese absorption in men reflects the higher iron status and serum ferritin levels in men (Finley *et al*., 1994; Finley, 1999; IOM, 2001).

Once absorbed, manganese is mostly bound to gamma-globulin and albumin, and a small percentage of Mn^{3+} is bound to transferrin that also carries iron (Aisen*et al.*, 1969). Manganese absorption at the gut is affected by a number of factors. For instance, the level of manganese in the diet is known to influence the absorbable amount from the gut as well as its excretion through the bile. Adjustments to high dietary manganese intake include lowered gut absorption, increased liver metabolism, and higher biliary and pancreatic excretion of this mineral (Malecki*et al.*, 1996; Finley and Davis, 1999; Dorman*et al.*, 2001; Dorman*et al.*, 2002). Manganese absorption from the diet is also affected by the presence of ascorbic acid, phytate, other trace minerals and other dietary constituents (Davidsson*et al.*, 1991).

Competition between manganese and iron at the gut has been reported to be most likely mediated through the Divalent Metal Transporter 1 (DMT-1) (Gunshin*et al.*, 1997). In addition, manganese absorption from the gut is also influenced by individual's age. Absorption of manganese is high at the neonatal period (Keen*et al.*, 1986). Compared with adults, human infants also have higher retention of ingested manganese during the early neonatal period (Zlotkin*et al.*, 1995; Dorner*et al*., 1989), with formula-fed term infants retaining approximately 20% of oral intake. The developing rodent brain takes up about 8% of the total orally ingested manganese during the early neonatal period (Keen *et al*., 1986).

The mammalian tissue manganese levels has a ''normal'' range of 0.3–2.9 μg manganese per gram wet tissue weight (Keen and Zidenberg-Cherr, 1994; Rehnberg, 1982). Tissues like brain that have high energy demand and retina with high pigment content usually contain the highest manganese levels. Uniquely, liver, kidney, pancreas and bone, also have high manganese levels.

Manganese is usually excreted through the bile (Davis *et al*., 1993; Malecki *et al*., 1996). Irrespective of the level of manganese ingested, adult humans maintain relatively stable tissue manganese levels, achieved by tightly controlled regulation of absorption and excretion rates. Within the liver, manganese is conjugated with bile and excreted into the intestine. Reabsorption of small manganese portion takes place in the

intestine, establishing de facto an enterohepatic circulation (Schroeder*et al.*, 1996). Biliary excretion is not well developed in neonatal animals, thus exposure during this period may result in higher risk of toxicity due to increased delivery of manganese to the brain and other tissues. In mice, rats, and kittens, there is an almost complete absence of biliary manganese excretion within the neonatal period (Cotzias*et al.*, 1976). Only a small fraction of the absorbed manganese dose is usually excreted via the pancreas (Davis *et al*., 1993). Urinary excretion of manganese is generally low.

2.5.2.3 Deficiency and toxicity of manganese

Inadequate dietary intake and manganese deficiency, though rarely found, lead to reduced fertility and birth defects, impaired growth, poor bone formation and skeletal defects, abnormal glucose tolerance, and altered lipid and carbohydrate metabolism (Freeland-Graves and Llanes, 1994; Keen*et al.*, 1999). Men experimentally placed on manganese-depleted diets developed an erythematous rash on their torsos (Friedman*et al*., 1987), and women consuming <1 mg manganese per day in their diet developed altered mood and increased pain during the premenstrual phase of their estrous cycle (Penland and Johnson, 1993). Incidentally, naturally induced deficiency of manganese has not been documented in humans.

While naturally induced deficiency states have not been found in humans, manganese-induced neurotoxicity from excess respiratory or dietary exposures has been well described. There are numerous propositions regarding potential mechanisms of manganese-induced neurotoxicity(Fitsanakis*et al.*, 2004). Oxidative stress is one of various factors implicated in manganese-induced neurotoxicity (Aschner, 1997).

A tolerable Upper Level (UL) intake of 11 mg per day of total manganese intake from water, food and supplements has been set for adults (19 years and above) based on no-observed-adverse-effect level for Western diet. Beyond this stipulated amount of 11 mg per day, the risk of toxicity is high. Toxicity is not expected to ensue in infants (0 to 12 months), provided they take their foods strictly from breast milk and formula/complementary foods. The UL set for other age groups has been extrapolated from the data for adult, considering the body weight of members of these other groups. It ranges from 2 mg per day for children 1 to 3 years to 9 mg per day for adolescents 14 to 18 years (IOM, 2001).

Manganese toxicity can be found in patients with liver failure, due to its route of excretion. Manganese toxicity secondary to liver failure results in accumulation of manganese in liver and brain, yielding neurologic abnormalities (Hauser*et al.*, 1994; Reynolds*et al.*, 1994). Manganese toxicity having symptoms similar to those of Parkinson's disease was reported in about 25 percent of miners and metal mill workers in Chile, Russia, former Yugoslavia and North Africa. This toxicity was known to culminate mainly from inhalation mineral dust in the mills and mines (Wennberg*et al.*, 1991; Berdanier and Zempleni, 2009).

2.5.3 Iron

2.5.3.1 Chemistry and essentiality of iron

Iron (Fe), 26th element in the periodic table, is the fourth most prevalent mineral in the Earth's crust. Several centuries ago, the Neolithic man learned to mine iron as well as fabricate tools from iron. Tonics have been processed from iron by the Romans, but the clinical recognition of iron as an essential nutrient was not achieved until the 17th century (Christian, 1903). Sydenham was the first to suggest that chlorosis (a sickness in adolescent female characterized by pale skin colour) was due to iron deficiency anaemia. He showed that the condition can be effectively treated with iron salts (Berdanier and Zempleni, 2009).

In 1713, Remmery and Jeffrey demonstrated the presence of iron in the blood preparation, and in 1852 Funke demonstrated that the red cell was rich in this mineral. Thus, it was realised that iron and red cell number were related, and that red cell function of carrying oxygen greatly depended on its haemoglobin viz-a-viz iron content (Christian, 1903).

Various forms of iron exist in the environment. It commonly exists in a trivalent form (Fe^{3+}) as ferric oxide or hydroxide or its polymers. These salts are not well absorbed until they are solubilized or ionized by intestinal contents. Both ferric (Fe^{3+}) and ferrous (Fe^{2+}) salts are available in the diet, but only ferrous slats are absorbable from the Gastrointestinal Tract (GIT). Ferric compounds must be reduced to their ferrous forms for them to be absorbed. The low pH of the stomach gastric juice helps to achieve this feat during digestion (Berdanier and Zempleni, 2009).

The food sources of iron tend to determine its bioavailability. Soybean, for instance, contains some inhibitors of iron uptake. Diets such as those in Asia contain numerous soybean products, and iron absorption is grossly affected by these soybean anti-nutrients. Tannins, phytates, certain fibres (not cellulose), carbonates, phosphates, and low protein diets also negatively affect the apparent absorption of iron. On the other hand, citric acid, ascorbic acid, fructose, high-protein foods, methionine, lysine, histidine, cysteine, and natural chelates (such as heme), all support the apparent absorption of iron. Zinc and manganese minimize iron uptake by about 30 to 50 percent and 10 to 40 percent respectively. Excess iron reduce zinc uptake by 13-22%. Stearic acid, a major fatty acid in meat, also enhances iron uptake (Morris, 1983; Fairweather-tait, 1987; Johnson, 1990; Looker*et al.*, 1995). In animal tissues and foods of animal origin, iron is present in various metalloproteins that include haemoglobin, myoglobin, transferrin, ferritin, cytochromes, and so on (Donovan*et al.*, 2006).

2.5.3.2 Absorption, excretion and metabolism of iron

The apparent absorption of iron (the amount absorbed from food) can vary from less than 1% to more than 50%. The percentage that is absorbed depends on the nature of the diet, on the type of iron compound in the diet, and on regulatory mechanism in the intestinal mucosa that reflects body's physiological need for iron (Berdanier and Zempleni, 2009).

Two types of iron are present in food, namely the heme iron that is mainly found in animal products, and non-heme iron that is inorganic iron bound to various plant proteins. Most of the iron in the diet (usually above 85 percent) is present in the non-heme form. The absorption of the non-heme iron is strongly influenced by its solubility in the duodenum. Absorption of non-heme iron depends on the meal composition and is dependent on enhancers of absorption like animal protein and by reducing agents like vitamin C. On the other hand, heme iron is absorbed more efficiently as it is independent of enhancers. Although heme iron accounts for a smaller percentage of iron in the diet, it often provides (quantitatively) more absorbable iron to the body than dietary non-heme iron (Berdanier and Zempleni, 2009).

Iron uptake by the gut, iron in use and reuse, and iron loss are all integrated in a closed system (Donovan *et al*., 2006). This is as shown in Figure 2.3 below. The intake through the gut is very inefficient, and there is virtually no mechanism apart from blood loss that rids the body of its iron excess. The aggregate body iron content averages 4.0g in men and 2.6g in women (Table 2.1). There are two groups of iron containing compounds that are considered essential to life; they include essential iron compounds as well as storage and transport iron compounds (Crichton and Charloteaux-Wauters, 1987).

Essential iron compounds in the body are haemoglobin, myoglobin, and the cytochromes; making about 78 percent of the body iron. The second groups of molecules are those involved in iron transport (transferrin) and storage (ferritin, hemosiderin). In addition, there are some enzymes whose active sites have iron-sulphur centres (Crichton and Charloteaux-Wauters, 1987).

Haemoglobin is a tetramer (that is, with four polypeptide chains) having a molecular weight of 64,500 that contains two α-subunits and two β-subunits. In haemoglobin, iron is coordinated with a tetraporphyrin moiety which, in turn, is bound to a polypeptide chain. These give the protein allosteric properties in the uptake and release of oxygen. Each polypeptide subunit has a ferrous iron atom, which amounts to 0.34% of the protein by weight (Berdanier and Zempleni, 2009).

Transferrin is the iron-transport protein that carries ferric iron around the sites of its absorption, storage, and utilization. It is a β-glycoprotein, molecular weight 76,000 that binds two atoms of ferric iron per mole. Iron is transferred from the intestinal mucosa to transferrin and is carried through the blood to peripheral tissues that have transferrin receptor sites. Transferrin is mainly synthesized in the liver, brain, and testes, as well as other tissues. The regulation of transferrin gene varies from cell type to cell type, and each cell type has its own array of transcription factors and promoters that control the quantity of transferrin produced. The amount of transferrin synthesized is inversely related to the iron supplied from foods and drinks. While there is low

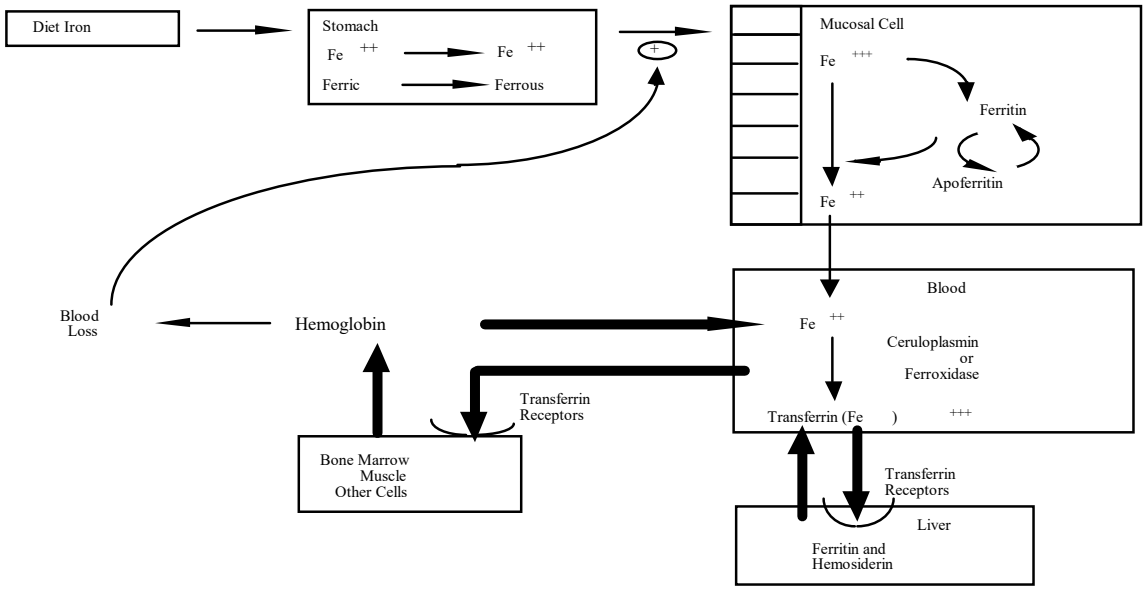

Figure 2.3: An overview of iron uptake and uses. This is an apparently closed system with the recycling and conservation of iron absorbed from the gut (Berdanier and Zempleni, 2009)

Table 2.1: The Body Content of Iron

Type of iron	Male 70kg	Female 60kg
Essential iron	3.100g	2.100g
Haemoglobin	2.700g	1.800g
Myoglobin, cytochromes and other enzyme	0.400g	0.300g
Storage and transport iron	0.900g	0.500g
Ferritin	0.897g	0.407g
Transferrin	0.003g	0.003g
Total Iron	4.000g	2.600g

(Berdanier and Zempleni, 2009)

intake, more transferrin is usually produced so as to optimize iron availability and vice versa (Zakim, 1992; Kuvibidila*et al.*, 1996).

Once iron enters the cell, it is chelated to a storage protein, ferritin. Ferrochelatase is the enzyme that facilitates this chelation. This reaction points to the ultimate destination for most of the iron that enters the cell. Chelation of iron to its storage protein occurs at the outer mitochondrial membrane. Ferritin has a molecular weight of 45,000 and it is composed of 24 subunits that form an outer shell within which there is a storage capacity for polynuclear hydrous ferric oxide phosphate. Its synthesis is highly regulated at the level of translation by ferric atom (Rogers and Munro, 1987; Munro*et al.*, 1993; Proudhon*et al.*, 1996) and post transcriptionally, by a cytoplasmic protein called the iron regulatory protein (Cairo*et al.*, 1996). When iron is present, ferritin mRNA is available for translation. In the absence of iron, the mRNA folds up in a way that the translation start site is hidden. Over 30% of the ferritin weight may be iron. Ferritin is available in the gut, liver, reticulo-endothelial cells, and the bone marrow.

Hemosiderin is a denatured form of ferritin that has about 33 percent of the iron stores. The posttranscriptional regulation of ferritin synthesis is regarded as a protective mechanism in the presence of excess iron. In this situation the iron regulatory protein binds to the iron-responsive element in the ferritin mRNA thereby reducing ferritin synthesis. When the iron-responsive protein detaches from the iron-responsive element, ferritin synthesis resumes. Aconitase, an enzyme that catalyses the dehydrogenation of citrate to isocitrate has been found to serve as an iron-regulatory protein (Beinert and Kennedy, 1993). Ferritin also serves as detoxicant of zinc and as a zinc ion donor (Price and Joshi, 1982). This is useful in the management of zinc overload.

The hormone hepciden has been indicated as key to the regulation of iron balance. When elevated it serves to raise the amount of iron in macrophages and lower gastrointestinal iron uptake (Nicolas*et al*., 2002; Sullivan, 2007). The synthesis of hepciden is induced by infection, inflammation, and elevated iron intake. Lower levels of hepciden are seen in patients with iron deficiency, anaemia, hypoxia, and hereditary

hemochromatosis. Hepciden mRNA falls quickly after blood loss (Nicolas *et al*., 2002).

2.5.3.3 Iron-containing materials in the body

The cytochromes are enzymes involved in electron transport system which is found mainly in the mitochondria. Cytochrome P-450, a specialized cytochrome, is used to oxidize organic compounds. This cytochrome is found in the endoplasmic reticulum. Whereas the cytochrome P-450 enzymes are involved in the detoxification of drugs and chemicals, they also convert carcinogens to active forms. The cytochrome P-450I is involved in this carcinogen activation whereas P-450IIE has the tendency to form oxygen radicals that are cytotoxic as well as carcinogenic (Parke*et al.*, 1991). Other cytochromes produce oxygen radicals by futile cycling. In some respects the ability to generate peroxide radicals has a protective effect in that peroxides can kill invading pathogens. Peroxides formation is in fact the first line of defence against such intruders. Other enzymes, in which iron is not bound to heme, include iron sulfur proteins, metalloflavoproteins and some glycolytic enzymes (Berdanier and Zempleni, 2009).

Since the life span of a red blood cell is about 120 days in humans, the iron flow through the plasma space totals about 25-30 mg per day in adults (that is, 0.5 mg per kg body weight). The amount of iron corresponds to the destruction of about 1 percent of the circulating haemoglobin mass per day. Iron is conserved in the body in males and postmenopausal female much better; only 10 percent is being lost per year in normal male, or about 1 mg per day. This loss of 1 mg per day has to be made up by dietary intake of iron, which is only about 10 percent efficient in absorption, thus, requiring about 10 mg of dietary intake of iron per day. In menstruating females on the other hand, the loss is increased to 2 mg per day, which means that the intake and absorption of iron must be increased or the females will develop iron deficiency. In contrast to the turnover of haemoglobin in the red cell, tissue iron-containing compounds which include the cytochrome enzymes and a variety of other non-heme enzymes are heterogeneous with respect to life span (Berdanier and Zempleni, 2009).

Iron entry into the body is normally regulated in the mucosal cells of the small intestine. Its iron gate is very sensitive to the iron stores, so if the iron stores are low,

which is true for most women and children, the intestinal mucosa takes up iron and increases the amount absorbed from diet. On the other hand, if the body iron store is optimal as found in healthy men and postmenopausal women, the percentage of iron absorbed is low. This mechanism minimizes risk of toxicity from iron overload. In infancy lactoferrin, an iron-binding protein in human milk enhances the absorption of iron through the lactoferrin receptors on the surface of the intestinal mucosa of infants. This explains why iron absorption from human milk is higher. Milk is not usually considered a good source of iron but for the breast fed infant, this lactoferrin-iron mechanism enables adequate supply and thus prevents deficiency from developing. As the infant matures, however, this mechanism becomes insufficient to supply the required iron intake (Berdanier and Zempleni, 2009).

On the average, only about 10% of dietary iron is absorbed. In order to be absorbed, the iron must be in the ferrous state. Upon entry into the enterocyte, it is incorporated (in part) into ferritin in the ferrous state. As it enters the enterocyte, it is transported in the ferrous state, probably bound to cytoplasmic proteins. When the iron is pumped out of the enterocyte it must be oxidized to the ferric state in order to bind to transferrin. This is accomplished by ceruloplasmin, which contains eight copper ions in the divalent state. Ceruloplasmin copper is reduced by the iron, resulting in the formation of cuprous ions in ceruloplasmin and ferric iron in transferrin. Transferrin is recognized in the periphery by cells that have transferrin receptors. The transferrin receptors vary, depending on the tissue and condition. Tissues such as erythroid precursors, placenta, and liver, which have a large number of transferrin receptor, have a proportionately high intake of iron. When these cells are in an iron-rich environment, the number of receptors decreases and, conversely, when they are in an iron-poor environment, the number of receptors increases. The up- and down regulation of transferrin receptors is accomplished at the level of transcription (Zakim, 1992; Munro *et al*., 1993).

The presence of oxygen tends to oxidize a small percentage of iron each day, and the formation of ferric iron in the haemoglobin molecules converts it to met-haemoglobin, which has no capacity to absorb and release oxygen. In order to minimize this effect of the oxidation of ferrous iron in haemoglobin by cellular oxygen concentrations, met-haemoglobin reductase, which is also NADH-dependent enzyme, reduces the ferric

iron in met-haemoglobin back to the ferrous state that makes haemoglobin functional (Berdanier and Zempleni, 2009).

2.5.3.4 Iron deficiency

Iron deficiency is probably the most common nutritional deficiency known within the world population. This is so due to the fact that iron is poorly absorbed and because many diets, especially those consumed among the third-world populations, are not so rich in bioavailable iron. Diets containing leafy green vegetables, whole grain cereals and legumes contain only non-heme iron, which is poorly absorbed. Furthermore children and women are at a constant risk for iron deficiency. Assessments of iron deficiency include the determination of levels of tissue ferritin, red cell number, receptor activity, heme iron, and haemoglobin levels (Berdanier and Zempleni, 2009).

The appearance of clinical iron deficiency anaemia usually manifest in three stages:

- The first involves depletion of iron store as measured by decreased ferritin, which reflects the ferritin (iron store) supply in the body. This stage is without loss of essential iron compounds and without any evidence of anaemia.

- The second stage is characterized by chemical changes that reflect the deficiency of iron required for normal production of haemoglobin and other iron compounds. This is shown by a decrease in transferrin saturation levels and an increase in erythrocyte protoporphyrin-so-called iron-deficiency without anaemia.

- In the final stage the appearance of iron deficiency anaemia manifests with depressed haemoglobin production and change in the mean corpuscular volume of the red blood cell to produce microcytic hypochromic anaemia. This is expressed clinically as pallor and weakness. When iron deficiency becomes severe, the nails take up a spoon shape. Several changes in intermediary metabolism have been reported in iron-deficient rat models. These include an increase insulin sensitivity in peripheral tissue, an increase in hepatic glucose production (gluconeogenesis), a decrease in the conversion rate of thyroxin to triiodothyronine, impaired oxidation of fatty acids, and ketogenesis, an increased need for carnitine, evidence of oxidative damage to

membranes of red blood cells, abnormal monoamine metabolism in the brain (increased dopamine synthesis and down regulation of dopamine receptors), increased serum cholesterol and triglycerides and slightly less pentose shunt activity. In addition to these metabolic changes, iron-deficient rats had a poor immune response to pathogen challenge. There was a decrease in antibody production and decrease in the natural killer population (Hill*et al*., 2007). These metabolic anomalies have not been documented in iron-deficient humans; nevertheless it is envisaged that there will be lots of similarities.

Treatment of iron deficiency anaemia

The treatment of iron-deficiency anaemia is a pharmacologic activity that entails administration of large doses of iron, usually amounting to 60 mg of essential iron or 300 mg of ferrous sulphate, once or twice daily. This treatment is normally maintained for 2 to 3 months to normalize haemoglobin levels and iron stores. These parameters are usually monitored till satisfactory results are realised (Berdanier and Zempleni, 2009).

2.5.3.5 Toxicology of iron

Excess iron intake can lead to toxicity. This can occur acutely in children who take in iron pills or iron vitamin supplement, not realizing the toxicity potential. Severe iron poisoning is characterized by damage to the intestine with vomiting, bloody diarrhoea, acidosis, and sometimes liver failure. Effective treatment includes induced emesis. Food and electrolyte treatment has substantially decreased the mortality rate from about 50% in 1950 to less than a few percent in recent years (Berdanier and Zempleni, 2009).

Chronic overload of iron can result either from chronic excess intake or from a genetic disorder, hemochromatosis. Hemochromatosis is intermittent in nature. The genetic disorder is due to defect of gene on chromosome 6. Two descendent of the British king George III have been diagnosed with the disease, and it has been suggested that the intermittent psychiatric state of King George III during the American Revolution might have been due to hemochromatosis. Hemochromatosis is characterized by higher iron absorption with damage to the brain, pancreas, liver, and heart. These

damages have been associated with diabetes, liver failure and heart failure. There is also an associated psychiatric abnormality that is likewise intermittent in character. People having hemochromatosis may also develop hepatocellular carcinoma and cancer of colon.

Two lines of evidence have been put forth that support this suggestion. The first line concerns the production of free radicals (Thompson and Godin, 1995). Excess iron can stimulate the generation of free radicals and damages from these radicals could be so great that natural repair will be inadequate. The second line concerns the fact that cancer cells, like normal cells, require iron as an essential ingredient of metabolism. Having an abundance of iron in the system could raise the rate of survival and proliferation of the cancer cells. Several population studies have provided support for these lines of thought. In these studies a dose-response relationship was reported. That is, there was a positive correlation of iron intake, ferritin levels, and colon cancer development (Berdanier and Zempleni, 2009).

Carcinogenesis can also be instigated by other minerals in relation to iron. Nickel sulphide, for example, is a potent carcinogen having the kidneys as target. In the presence of high-to-moderate iron levels, the activity of the nickel compound increases. In a situation of excess copper resulting from a genetic disorder involving the copper transporter, hepatic cancer develops, which is potentiated by high iron levels. It would appear in these last examples that iron serves as cancer promoter rather than as an initiator as previously described for colon cancer (Berdanier and Zempleni, 2009).

Although excess iron intake can be harmful, it should be noted that optimal iron intake can protect against lead toxicity, since lead competes with iron for intake by the enterocyte. If the transport is fully saturated by its preferred mineral, iron then the lead will be poorly absorbed and thus excreted in the faeces. Those with optimal iron intake are at less risk for lead toxicity than are those whose iron intake is marginal or deficient (Berdanier and Zempleni, 2009).

2.5.4 Zinc

Zinc (Zn) is the last transition element of the fourth period of the periodic table. It has an atomic number of 30 and atomic mass of 65.4. It has up to 15 isotopes, of which ^{65}Zn, is the most useful. The radioisotope has a half-life of 244 days. Zinc is a good reducing agent and it forms stable complexes with other ions as well as form a wide range of salts with members of the halogen family, carbonates, phosphates, sulphates, oxaloacetate, and phytate (Berdanier and Zempleni, 2009).

2.5.4.1 Needs and metabolic functions of zinc

The RDA for zinc is 11 mg/day for adult males and 8 mg/day for adult females (IOM, 2001).Zinc has two major sets of functions. The first is its role as a cofactor for more than 70 enzymes. The enzymes requiring zinc for catalyses are partially highlighted below. In this role, zinc binds to the histidine and cysteine residues of the enzyme thereby stabilizing and exposing the active sites of the enzymes such that catalyses of the reactions in question can occur optimally (Berdanier and Zempleni, 2009).

Some enzymes needing zinc as a cofactor include lactate dehydrogenase, alcohol dehydrogenase, angiotensin converting enzyme, cytoplasmic superoxide dismutase (also requires copper), alkaline phosphatase, carbonic anhydrase, carboxypeptidase A, B, and D, DNA and RNA polymerases, pyruvate dehydrogenase, proteases and peptidases, thymidine kinase, aspartate transcarbamoylase, thymulin, d-amino levulinate dehydrase, fructose 1,6 bisphosphatase, transcarboxylases, reverse transcriptase, leukotriene hydrolase, glyoxalase, phosphodiesterase, elastase, 5' nucleotidase, adenosine deaminase, and transcription factor Sp1 (Berdanier and Zempleni, 2009).

Even though these enzymes require zinc as a cofactor, they appear to function at near-normal levels in zinc-deficient animals. This could be partly because these enzymes are intracellular enzymes and tenaciously retain the zinc needful to continue to function. There seems to be a hierarchy of zinc need by the living organism. Tissue stores and dispensable zinc uses are raided well before the intracellular zinc needed by these enzymes is negatively affected. Furthermore, deficient states are characterized

by higher zinc absorption efficiency, which gives added protection to the system from self-destruction (Berdanier and Zempleni, 2009).

The Second vital role is the binding of zinc to certain DNA-binding proteins found in the nucleus. In this role, zinc binds also to histidine and cysteine residues of the linear portions of these binding proteins, giving them a sausage-like shape. These proteins, with zinc attached, are called zinc finger proteins or simply *zinc fingers* (Conte*et al.*, 1996).Some nutrients (such as vitamin A, vitamin D and hormones like steroids, insulin-like growth factor I, growth hormone, and so on) have their effects on the expression of specific genes simply because they can bind to specific zinc fingers, which in turn binds to very specific DNA regions (Berdanier and Zempleni, 2009).

In view of this second vital role of zinc, if there is mutation in the genes encoding these DNA-binding proteins in a way that they lack the two required residues (each of histidine and cysteine in the linear part of their structure), then the functional attributes of these vitamins and hormones at the genetic level will be truncated. Instances of such mutations have been reported, as well as instances where these zinc fingers have been deliberately modified as a therapeutic approach to disease control. Zinc containing transcription factor Zif268 has been modified with the result of a loss in sequence specific recognition of DNA by viruses, thus disrupting the viral invasion and destruction of their target cells. Although this modification was an *in vitro* set-up, which was not replicated *in vivo*, this approach might have therapeutic application in future years as a means to circumvent the consequences of viral disease such as found in AIDS (Wu*et al.*, 1995).

The synthesis of Metallothionine (MT 1 and MT-2) is regulated by zinc through its action on the expression of the genes for these proteins. The level of MT-1 is a very sensitive indicator of zinc deficiency (Oberleas, 1993).In addition to its function in metallothionine transcription, as a cofactor of several enzymes and in the zinc fingers; zinc is also required for the stabilization of membranes and it provides structural strength to bone as part of the bone mineral apatite (Berdanier and Zempleni, 2009).

2.5.4.2 Absorption, transport and excretion of zinc

Similar to iron, zinc absorption is relatively poor (Halsted*et al.*, 1974).Out of the approximately 4-14 mg per day consumed; only 10-40 percent is absorbed. Absorption is decreased by the presence of binding agents or chelating agents that make the mineral unabsorbable or poorly absorbed. Agents like fibre, phosphate, and phytate (inositol hexaphosphate) can render it unavailable. Zinc bound by these agents is excreted through faeces. People who are geophagic (Pica) and/or who consume large amounts of phytate-containing foods (mainly cereal products) are at higher risk of being zinc deficient. Oberleas (1993) has proposed that diets having a phytate-to-zinc ratio higher than 10 will induce zinc deficiency, if largely consumed on a long term basis, regardless of the total zinc content in these diets. Clay, a mixed mineral soil fragment, can also render zinc unavailable.

Zinc binds to ligands that contain sulphur, nitrogen, or oxygen. Zinc will form complexes with phosphate groups (PO_4^{3-}), chloride (Cl^-), and carbonate groups (HCO_3^-) as well as with cysteine and histidine. Buffers such as N-(2-hydroxyethyl-)-1-piperazine ethane sulfonic acid (HEPES) have very minimal effect on how zinc binds to these ligands. Unlike iron, zinc exits in only one valence state: Zn^{2+}. The 70kg human absorbs 1-2 mg zinc per day (Figure 2.4) using both a nonsaturated and a saturable process. The former is passive diffusion whereas the latter involves zinc binding metallothionine protein. After entry into the enterocyte the zinc binds to Cysteine-Rich Intestinal Protein (CRIP), which in turn transfers it to either metallothionine or (through the serosal side of the enterocyte) to albumin, which carriers it to its point of utilization (Figure 2.5). Vitamin D enhances zinc uptake, probably due to its positive effect on the synthesis of metallothionine. From the enterocyte, it is sent to the serum where 77 percent is loosely bound to albumin, approximately 20 percent is tightly bound to α-2 macroglobulin and 2-8 percent is ultrafilterable (Masuoka and Saltman, 1994). The liver seems to be a main site of Zn^{2+} uptake after it has been absorbed. There is both rapid uptake ($t_{1/2}$ = 20 seconds) and a slower linear uptake. The ultrafiltrate is excreted either in the urine (0.5-0.8mg per day) or in the faeces through biliary excretion (Failla and Cousins, 1978).

2.5.4.3 Storage of zinc

Zinc is found in all types and tissues. However, it is notably stored in the β-cells of the islets of Langerhans in the pancreas. There, zinc is incorporated into the hormone insulin. Each insulin molecule contains two to four atoms of zinc as part of its crystalline structure. Zinc may play a role in insulin release but the details of this role have not yet been completely elucidated. Pharmaceutical preparations of insulin needed by diabetics for hormone replacement therapy contain zinc. It should be noted that not all species integrate zinc in the insulin structure (Berdanier and Zempleni, 2009).

2.5.4.4 Zinc interactions

Zinc can sometimes be displaced on the zinc fingers by some other divalent metals. Iron for instance has been used to displace zinc on the DNA-binding protein that binds oestrogen. This protein binds to the oestrogen response element of the DNA in the promoter regions encoding oestrogen-responsive gene products. When this happens in the presence of H_2O_2 and ascorbic acid, damage to the proximate DNA and the oestrogen response element, occurs. It has been suggested that in this circumstance of an iron-substituted zinc finger, free radicals are more readily generated with the consequence of genomic DNA damage (Conte *et al*., 1996). This mechanism has been proposed as how excess iron (iron toxicity) could multiply the cellular changes that occur in carcinogenesis.

In excess, cadmium can also substitute for zinc in the zinc fingers. In this substitution, the resultant zinc fingers are non-functional. Due to the importance of these fingers in cell survival and renewal, cadmium substitution is often lethal. Cadmium toxicity is an acute illness taking quite short time for the symptom of cell death to become so evident. The metallothionine proteins, in addition to binding zinc and copper, also bind other heavy metals such as mercury and cadmium. Ferritin, the iron storage protein, can also bind zinc. When zinc is available in excess, it can replace iron on this protein. Other interaction includes the copper-zinc interaction. Copper in excess can interferewith the uptake and binding of zinc by metallothionine in the enterocyte. Metallothionine has a greater affinity for copper than for zinc, and thus zinc is

leftbehind, whereas copper is transported to the serosal side of the enterocyte for export to

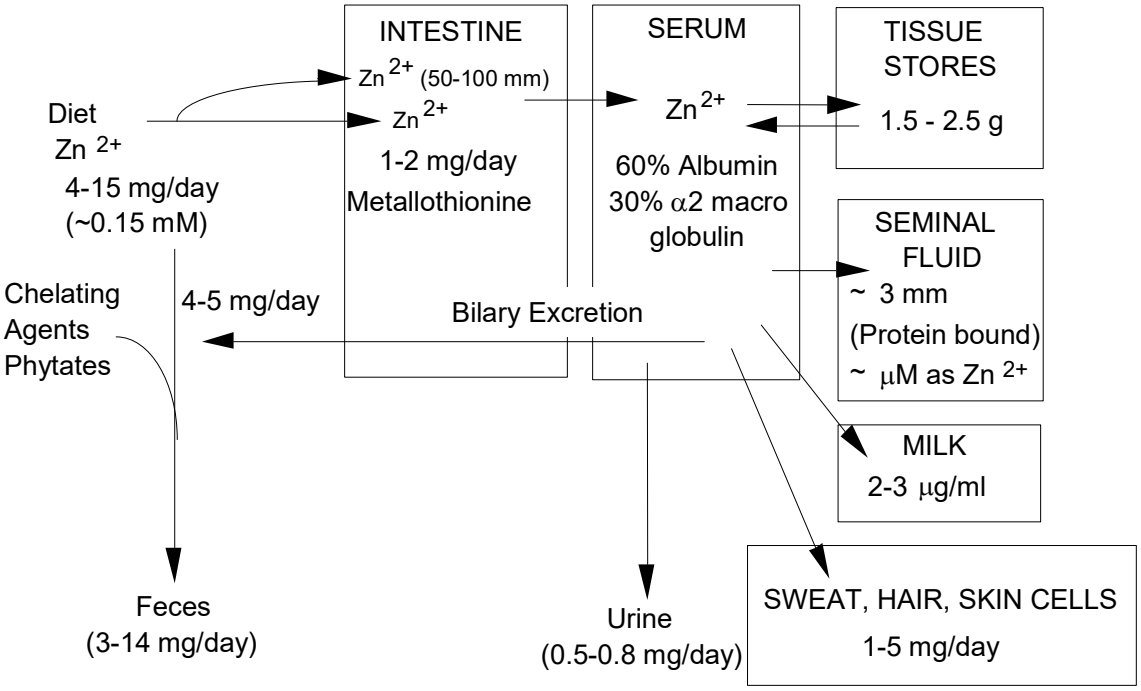

Figure 2.4: Zinc balance in a normal adult humans (Berdanier and Zempleni, 2009)

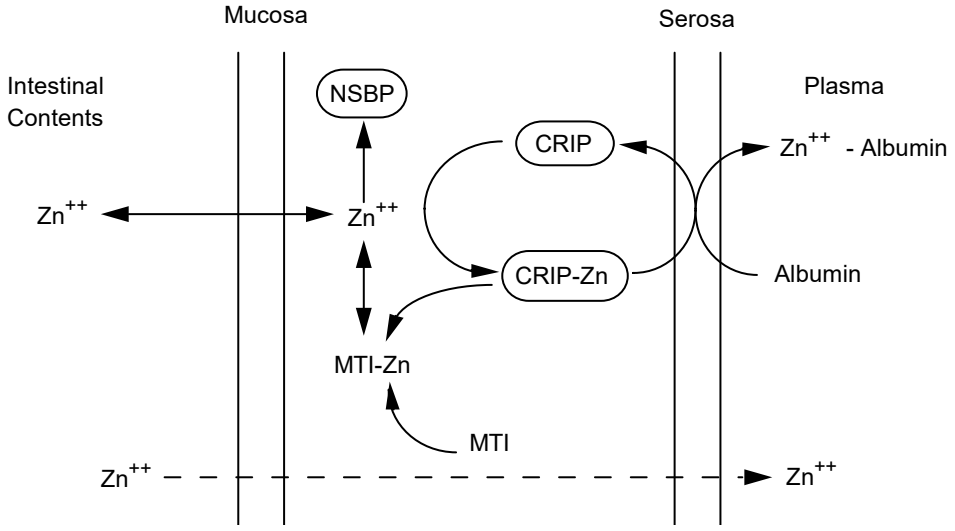

Figure 2.5: Intestinal absorption of zinc. Passive diffusion is slow at the lower part of the diagram whereas mediated transport involving Metalothioine (MT1), the Cysteine-Rich Protein (CRIP) and the

Nonspecific Binding Proteins (NSBP) are shown in the upper part of the diagram (Berdanier and Zempleni, 2009)

the plasma whereupon the copper rather than the zinc is picked up by albumin and transported to the rest of the body. Fortunately, excess copper in the normal diet is uncommon. Zinc is normally present in significantly high amounts, and the interaction is of very slight impact in the overall scheme of zinc metabolism (Berdanier and Zempleni, 2009).

2.5.4.5 Status and deficiency of zinc

Zinc status can be difficult to assess sensitively. Plasma and neutrophil zinc level can give a static measure of status; however, these blood levels can only evaluate the amount of zinc transport, not the functional state of the individual. Measurement of alkaline phosphate activity is quite useful because it is a zinc requiring enzyme and is sensitive to zinc deprivation. The level of metallothionine I in the blood is also a very sensitive indicator of zinc intake adequacy (Reeves, 1995).

Until the early 1960s, zinc as an essential mineral for human had not been established. Prasad in 1961 (Praad, 1984)andHalsted in 1958and 1963 (Halsted *et al*. 1974) described conditions in humans later found to be due to insufficient zinc intake. Among the symptoms were growth failure, hypogonadism, enlarged liver and spleen, anaemia, rough skin and mental lethargy. These characteristics are notable due to lack of zinc as a cofactor in many enzymatic reactions, and because of the absence of zinc in serving as an essential component of the DNA-binding zinc fingers. Detailed studies of population having these symptoms among its members revealed the custom of clay eating as well as diet that were very low in animal protein and high in cereal products. Geophagia (clay eating) can affect bioavailability of not only zinc but also iron and other minerals needed for optimal growth and development.

In Iran, Prasad and Halsted found that the provision of iron and protein supplements reversed the anaemia and enlarged spleen and liver conditions. Pubic hair and the gonad also began to develop. It was confounding to explain all the clinical features (and their reversal) only on the basis of iron deficiency and/or protein deficiency due to the fact that other researchers had not reported the features of hypogonadism as part

of the iron or protein deficient state. However, animal studies demonstrated this feature as being a characteristic of zinc deficiency. Later, Prasad in Egypt gave report on growth retardation and testicular atropy in young men. Geophagia was not a custom in this study population and there were no signs of enlarged spleens and livers (McNall*et al.*, 1995).

The dietary patterns were somewhat close to those found in Iran, in that the diets were high in cereal and low in animal protein. Zinc level in hair, plasma, and red cells were less than normal. The zinc deficiency signs of growth failure and sexual immaturity are due to an individual's inability to support cell division and hence tissue growth which can also be a consequence of decreased expression of insulin-like growth factor and growth hormone (McNall*et al.*, 1995).

The skin symptoms of deficiency are the most obvious because skin cells turn over very rapidly (approximately 7 Days) these symptoms include a moist eczematoid dermatitis found in the nasolabial folds and around other body folded portions. There is a failure in zinc-deficient individuals to replace these regularly desquamated cells. In infants and young children, inadequate zinc intake can result abnormal CNS development as well as impaired skeletal development (Keen*et al.*, 1993). In the later instance, zinc deficiency results in an impaired calcium uptake probably due to a decreased synthesis of intestinal calbindin.

Poor immune response and decreased taste sensitivity are also experienced in the zinc deficient state. These features also relate to the role of zinc in cell turnover. Immunity requires antibody synthesis involving zinc fingers whereas the taste sensation involves short-lived epithelial cells on the surface of the tongue and oral cavity. Other zinc deficient states have been reported in severely traumatized individual and in patients with end-stage renal disease with or without dialysis; more zinc is lost through the kidneys than a normal individual (Mahajan*et al.*, 1982). In renal failure, excess zinc is lost through the dialysate when patients are maintained on regular dialysis treatment. Children that are anephric and are placed on dialysis must be monitored with respect to their zinc status. Failure in this regard will result in growth their failure and lack of sexual maturation. Zinc supplementation can still reverse these symptoms (Berdanier and Zempleni, 2009).

2.6 Selected heavy metals

2.6.1 Cadmium

Cadmium (Cd) is an element of the group II-B, with an atomic mass of 112.41. Pure cadmium is a soft, silver-white metal, that is not usually found in its pure form, but as mineral compounds with some other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulphur (cadmium sulphate, cadmium sulphide), (ATSDR, 1998). Cadmium is very similar to zinc; they mainly differ in their boiling points, 907 ^{o}C and 767 ^{o}C, respectively. Both form divalent cations with a complete shell of electrons, which accounts for the lack of ligand field stabilization of their complexes (Valle, 1995).

2.6.1.1 Historical background and sources of cadmium

Cadmium was discovered as an element in 1817 and its utilization in industries has been quite minor until about 60 years ago. The first reported human health effect related to cadmium exposure was the damage to the lungs of cadmium-exposed workers, published in 1938. About a decade later, in 1948, Friberg reported some unusual conditions – proteinuria and emphysema – in workers of the cadmium battery industry. Shortly after, pathological bone fractures and server pain (named Itai-Itai disease) occurred after World War II, in Toyama Japan, which was confirmed as a consequence of cadmium exposure (Nordberg, 2004). Nowadays, cadmium is still an important industrial metal. It is extracted during the production of other metals, such as zinc, lead and copper and it is used in industrial and household products, mainly in batteries, pigments, metal coatings, plastics and some metal alloys (ATSDR, 1998).

Despite the fact that cadmium has been a widespread industrial and environmental pollutant, it is usually present in food at very low quantities, and dietary intakes and gastrointestinal absorption are minimal. The main source of toxic exposure is by the inhalation route of cadmium particles or fumes during industrial operations (OEHHA, 2006). Naturally occurring cadmium levels are extremely low. Reports have shown

that cadmium concentrations in non-contaminated soil vary from 0.01 to 5 mg per kg of soil (Kabata-Pendias, 2004); nevertheless, fertilizers produced from phosphate ores constitute a major source of spread of cadmium pollution (Chen*et al.*, 2007). In addition, the inappropriate disposal of cadmium containing wastes has increased its emission in densely populated areas globally. Although cadmium is used in a number of industrial applications, the main source of cadmium intake is through smoking and food (Jarup, 2003).

2.6.1.2 Cadmium uptake by plants

The electrochemical potential gradient of the plasma membrane in plant root drives cadmium and other cations into the root cells (Huang*et al.*, 1992; Wang*et al.*, 1994; Blaylock and Huang, 2000). However, external factors such as iron level can reduce cadmium uptake. For instance, in *Hordeum vulgare* (barley) iron levels of 0–10µM reduced cadmium uptake (Sharma*et al.*, 2004a; Sharma*et al.*, 2004b). In *Thlaspi caerulescens* Ganges ecotype, iron deficiency up-regulates the expression of genes encoding for Fe(II) uptake which promotes the uptake of cadmium (Lombi*et al.*, 2002). On the other hand, it seems that in *Lactuca sativa*, manganese enriched medium enhances the uptake of cadmium, 64% of which is accumulated in the cell walls and potentially transferred to the consumers (Ramos*et al.*, 2002).

As regards cadmium transport inside plants, Diatloff*et al.* (2006) reported for the first time that a Low affinity Cation Transporter (LCT1) responsible for calcium transport in wheat is also responsible for cadmium transport in the yeast *Pichia pastoris*. It is most likely that this transporter is also involved in cadmium transport in many plants. *Salsola kali*, a potential cadmium hyper-accumulator handles cadmium through the production of Low Molecular Weight Thiols (LMWT), mainly in roots and stems (de la Rosa*et al.*, 2004). However, these same researchers have proposed that LMWT are at most contributing to half of the total cadmium binding in leaves (de la Rosa*et al.*, 2005).

In rice, the concentration of cadmium in grains is governed somewhat by its uptake and transport from roots to shoots, and to a greater extent, by the transport of Cd from shoots to grain. In a China study by Liu*et al.* (2007), it was discovered that about 0.73% of the total Cd taken up by six rice cultivars was transferred to the grain. This

represents an average of 1.02 mg per kg, which is 100 times higher than the concentration allowed by the European Union for cadmium concentration in rice grain (Olsson*et al.*, 2005).

2.6.1.3 Cadmium compounds in animals

In mammals cadmium is transported as a cadmium–protein complex, especially cadmium–metallothionein, and then stored in kidneys, liver, and intestinal mucosa (Cooke and Johnson, 1996) with a retention time of as long as 10 years (Pokorny and Ribaric-Lasnik, 2000). Cooke and Johnson (1996) have reported that in an ecosystem around a refinery site, the common shrew (*Sorex araneus*), an insectivorous mammal, stores up to 273±15 mg cadmium per kg dry weight in kidneys, while the field vole (*Microtus agrestris*), a herbivore mammal, stored up to 88.8±23.3 mg cadmium per kg dry weight in kidneys as well. As pointed out by Kan and Meijer (2007), the level of cadmium in the feed, the exposure period to the feed and the chemical form of cadmium in the feed can largely be used to predict the levels of cadmium in kidneys and liver. Although there are lots of detailed reports on cadmium toxicity and its transfer within the food chain, more information is still needful to unravel the metabolic pathways followed by cadmium compounds obtained by higher consumers of the food chain, especially humans.

2.6.1.4 Cadmium in the food chain

Studies have shown that environmental heavy metals like cadmium are moved within the food chain in a way that they affect both producers and consumers (Veltman*et al.*, 2008). To assess the roles of environmental factors on metal bioavailability, researchers have developed indices like *bioaccumulation factors*, defined as the ratio of the level of a chemical in an organism to the level in the soil. However, the bioaccumulation factors are affected by several environmental conditions in-built within the environment and the organisms (Veltman *et al*., 2008). In an experiment involving cadmium-fed plants at 0–100 µg per g, the primary consumer (the snail *Helixaspersa*) and the secondary consumer (larvae of the beetle *Chrysocarabus splendens*) displayed cadmium bioaccumulation factors of 1.87–3.9 µg per gin *H. aspersa* and less than 1 µg per g in *C. splendens*, the secondary predator (Scheifler*et al.*, 2002). The study also revealed that the cadmium exposure to the snails produced

31% beetle larvae death, which further showed the potential toxicity of cadmium movement in the food chain.

Scheifler*et al.* (2006) have also reported that snails absorbed the most cadmium from soil, not from plants. Another study have shown that cadmium is transferred from plants to snails in a concentration dependent manner (Gimbert*et al.*, 2008), which corroborate the potential hazard of cadmium hyper-accumulator plants. Food is a significant exposure route for several heavy metals, particularly in people consuming regionally contaminated foods. According to Chary*et al.* (2008), people that restrict their diet to locally grown food produce, such as subsistence farmers, are particularly at risk from soil contamination (if such exist in their area), because the cadmium in their diets is not diluted by food from other non-contaminated areas, as it is in the majority of the economically developed communities.

Chien *et al*. (2002) calculate the exposure risk to toxic metals through food by using a quotient called the Target Hazard Quotient (THQ). The THQ includes the exposure frequency and the concentration of the contaminant, among other parameters. They considered that when THQ was lower than 1, the level of daily exposure to the human population was safe. Using the THQ index Zheng*et al.* (2007) concluded that inhabitants living 500–1000 m from the Huludao Zinc Plant in China have acquired THQ values from vegetables higher than 1, which means they were at risk of cadmium toxicity.

Furthermore, Yang*et al.* (2006) reported that in the upper Wu Jiang River basin, province of Guangdong, China, rice plants irrigated with untreated mining wastewater had 0.24 μg cadmium per gram unpolished rice, and the dietary intake of cadmium was calculated to be 2.2 and 1.5 μg cadmium per kg body weight per day for a 60-kg adult and 40-kg child, respectively. These values exceed the provisional tolerable daily intake set by FAO/WHO, which is 1 μg cadmium per kg body weight. Although, rice is consumed as the staple food in Guangdong, and hence, rice contributes a major part to the total daily food intake, there are other sources of cadmium intake, such as dairy products and vegetables, which should also be considered in the risk assessment.

According to Millis*et al.* (2004), the consumption of vegetables is the main source of cadmium for humans. These scientists have indicated that the heterogeneity of soil

results in variation in element concentration in plants, which might lead to an inaccurate health risk assessment. Another factor that must be considered is the genetic makeup of crops. Antonious and Kochlar (2009) tested the heavy metal uptake capacity of 23 capsicum accessions from the USDA germplasm collection at the Kentucky State University Research Farm, Franklin County, KY. These researchers discovered that the accession PI-246331 accumulated significantly higher cadmium than the others.

In various parts of the world (e.g. Mexico, Pakistan, and China), crop land is irrigated with wastewater, and studies on metal transfer from crops to humans are just starting to be performed in these countries. Among crop plants, rice has a special place due to its unique capacity to absorb cadmium. Chaney*et al.*(2004) have reported that rice has the ability to accumulate soil cadmium in grains, excluding iron, zinc and calcium (even though the soil contains 100 times more zinc than cadmium). This poses a real threat to farmers consuming polished rice which is deficient in iron, zinc, and calcium (Chaney *et al*., 2004).

According to Khan*et al.* (2008), the daily intake of cadmium from food crops normally cultivated in wastewater irrigated land seems to be too low, based on the health risk indexes, to pose a threat for the human population. However, in a study that reported data collected for 16 years, Kobayashi*et al*. (2002) found that in the Jinzu River basin, the higher total cadmium intake by humans appears to be related to an adverse effect of this element on life prognosis. These researchers found that the mortality rate in people ingesting above 2.0 mg cadmium from rice cultivated in a cadmium-polluted area was higher compared to people ingesting less than 2.0 mg of cadmium.

Thus, a holistic approach to studying risk of cadmium toxicity to humans will incorporate at least two of the following schemes: dietary intake from the contaminated foods compared with provisional tolerable daily intake set by FAO/WHO; target hazard quotient calculated relative to the contaminated sources; and epidemiological survey on the impact of the contamination on specific population.

2.6.1.5 Toxicity of cadmium

The most dangerous characteristic of cadmium is that it accumulates throughout a lifetime due to its very long biological half-life of about 20 to 30 years in humans (Hideaki*et al*., 2008). The Provisional Tolerable Monthly Intakes (PTMI) set by FAO/WHO for cadmium is 25 µg per kg body weight (JECFA, 2011). Considering a 60 kg adult with 30-days monthly evaluation, a maximum of 50 µg cadmium daily intakes is allowable. This regulation has helped to guide on how much of this contaminant is allowable from food and water intakes. Excess intakes of cadmium tend to result in various debilitating health conditions, some of which are listed subsequently.

This metal is a serious occupational and environmental contaminant that may indicate a serious health hazard to humans and animals (WHO, 1992; Jarup*et al.*, 1998; Staessen*et al*., 1999). Cadmium represents serious environmental hazards because of its possible absorption through the GIT, penetration through placenta during pregnancy, and damages to plasma membranes and DNA (Kabata-Pendias, 2004). Furthermore, according to Peijnenburg*et al.* (2000), cadmium is the metal of greatest concern because it is the only known metal that might pose human or animal health risks at plant tissue levels that are not generally phytotoxic, when other metals are considered. In humans, cadmium may cause kidney damage, and according to WHO (1992), safe cadmium levels are less than 10 nmol per mmol creatinine (about 200 mg cadmium per kg kidney cortex) as measured in the kidneys. Cadmium not only attacks kidney and bones, it also affects the female reproduction system, which implies a serious threat for humans and other mammals. Cadmium also severely affects the female endocrine system.

Considering the cellular level, cadmium stimulates both the damaging and repair processes in which the cellular oxidation-reduction status has a vital role (Cuypers *et al*., 2010). As reviewed by Zarros*et al.* (2008), there are evidences that cadmium provoke indirect oxidative damage on the DNA, leading to: induction of cellular proliferation; inhibition of the apoptotic mechanisms; and inhibition of the DNA repair mechanisms (Zarros *et al*. 2008). Oxidative stress is thus believed to be the major molecular basis behind cadmium-induced cytotoxicity.

Rani*et al.* (2014) reviewed the myriad effects of cadmium on various organs of humans. Cadmium usually affects the proliferation and differentiation of cells, cell cycle progression, DNA synthesis, apoptosis and other cellular processes (Aimola*et al*., 2012). Another main impact of cadmium may be on the blockage of DNA repair (Giaginis*et al.* 2006; Hartwig, 2010), thereby, instilling cellular genotoxicity. Jarup (2003) and Hellstrom*et al*. (2001) reported studies carried out on adults (20-80 years) in Belgium and Kalmar exposed to different levels of cadmium. The people were notable to experience various levels of nephrotoxicity. Some other studies confirmed kidneys dysfunction similarly (Iwata*et al.*, 1991, Iwata*et al.*, 1992). A set of studies has reported that long-term exposure of cadmium to humans led to increased skeletal fragility and decreased mineral density (Bhattacharyya, 2009).

In another study, relationship between low cadmium dose and bone density in environmentally and occupationally exposed individuals in the three communities of Fliseryd, Oskarshamn and Linköping were studied (a total of 1064 participants 16–80 years). The results revealed that even at low cadmium level exposure, cadmium may lead to osteoporosis (Alfvén*et al*., 2000). Another target organ of cadmium toxicity is the lung. Cadmium enters the lung via house dust, smoking and/or occupational exposure (Hogervorst*et al*., 2007). The major sources of cadmium intoxication related to acute respiratory distress syndromes and other lung diseases are inhalation of tobacco cigarette smoke and occupational exposure to cadmium-containing fumes (Elinder*et al*., 1976; Barbee and Prince 1999; Godt*et al*., 2006; Sarkar*et al*., 2013). Several grades of cadmium-induced hepatotoxicities have also been reported from different studies (Rikans and Yamano 2000; Martelli*et al*., 2006; Van Kerkhove*et al*.,2010).

2.6.2 Lead

Lead (Pb) is a post-transition metal that is somewhat inert, when available in solid form. It has atomic number 82 and atomic mass 207. It is a soft and malleableheavy metal. Freshly cut solid lead has a bluish-white colour that quickly tarnishes to a dull greyish colour when exposed to air; the liquid metal has shiny chrome-silver lustre. The density of lead is 11.34 g per cm^3. Lead has the second highest atomic number of all practically stable elements. It shows high tendency for covalent bonding. It is

mostly found in the oxidation state of +2. Its weakened metallic character is shown by its general amphoteric nature: lead and its oxides react with both acids and bases (Polyanskiy, 1986; Wikipedia, 2016b). Lead occurs in Earth's crust majorly as the mineral galena (lead (II) sulfide) and, to a lesser extent, as anglesite (lead (II) sulfate) and cerussite (lead carbonate) (JECFA, 2011). Lead is viewed as having low solubility and availability for plant uptake because it precipitates as phosphates and sulphates, chemicals commonly found in the rhizosphere of plants (Blaylock and Huang, 2000). Lead is a wide spread toxic metal that has no known biological functions.

2.6.2.1 Sources of lead

The main environmental source of lead is metal smelting (Caussy*et al*., 2003), but agriculture, industry, and urban activities are also important sources of lead pollution (Marchiol*et al*., 2004). The use of lead-glazes in ceramics is ubiquitous and has been implicated as a frequent source of food contamination. In Mexico, the frequency of use of traditional, low-temperature, lead-glazed pottery has been associated directly with increased blood lead levels of children (Rojas-López, 1994). The use of older cracked pottery, storage of acidic foods, and cooking in the pottery have been reported to increase the amount of leached lead from the glaze. Children of potters engaged in producing leaded ceramic-ware, a cottage industry in many countries, have been seen to have higher risk of exposure to lead (WHO, 2010).

Over 80 percent of the lead daily intake is contributed by the ingestion of food, dirt and dust. The level of lead in plant-based foods depends on soil concentrations and is greatest around lead or zinc mines, lead smelters, and battery recycling plants. Cereals and cereal-based products, milk or formula can expose adults and or infants to high level of lead. The use of lead-soldered food and beverage cans may significantly raise the lead level, especially when acidic foods or drinks are stored in them. Since some alcoholic beverages are somewhat acidic, the use of any lead-containing materials for their production or distribution will increase the risk of lead toxicity. Smoking of tobacco can also raise lead level in the body (WHO, 2010). So, great care must be taken in minimizing the use of lead-containing materials for production, storage and distribution of foods and drinks.

High levels of lead in dust have been seen among populations engaged in electronic waste recovery and recycling, and elevated blood levels of lead have been reported in children grossly engaged in this processes (Leung*et al*., 2008; Zheng*et al*., 2008). As found in some traditions and culture, pregnant women eat soil, ceramic fragments or other non-food substances. Some of these items contain high levels of lead. Such intake can expose these women to high amount of blood lead. Since lead can cross freely from the maternal to the foetal circulation during pregnancy, serious prenatal brain damage can come up (Shannon, 2003; Erdem*et al*., 2004). Thus, to protect the next generation, children should be prevented from electronic recovery and recycling processes. More so, pregnant women should be discouraged from pica tradition, especially those related to the intakes of lead-contaminated non-food items.

2.6.2.2 Absorption, distribution, metabolism and excretion of lead

Lead is usually absorbed from the gastrointestinal tract as influenced by physiological factors such as age, fasting, pregnancy, calcium and iron status, and the physicochemical characteristics of the ingested material. Absorption is higher in children than in adults and is higher when food is absent in the tract. Absorbed lead is usually transferred to soft tissues, including liver and kidney, and to bone tissue, where it accumulates over the years. Under certain conditions such as pregnancy and osteoporosis, bone resorption can result in increased blood lead concentrations. Lead readily crosses the placenta and is transferred into breast milk. In humans, the half-life of lead is approximately 30 days in blood and 10–30 years in bone. Urine and faeces are the major routes of excretion. Its toxicity has been partly attributed to inhibition of enzymes and interference with calcium, magnesium and zinc homeostasis (JECFA, 2011).

2.6.2.3 Lead toxicity and management

The PTWI retained by FAO/WHO for lead is 25 µg per kg body weight (JECFA, 1999). On this note, a threshold of approximately 214 µg lead should not be exceeded by a 60 kg adult on daily intake basis. JECFA reassessed lead in June, 2010 in view of new data available and thereby withdrew the PTWI guideline value on the grounds that it was inadequate to protect against health problem and IQ loss (JECFA, 2010).

Since no new figure has been set, the value retained in 1999 is still useful currently as a benchmark for toxicity risk assessment.

Lead is linked with myriads of toxicity in adult and children across a wide range of exposures, down to the least blood lead concentrations evaluated. These toxic effects extend from acute, clinically obvious, symptomatic poisoning at high levels of exposure down to subclinical effects at lower amount of intake. Lead poisoning can negatively affect almost all organ system in human body. The mostly targeted organs are the central and peripheral nervous systems, the renal, immune, cardiovascular, gastrointestinal, endocrine, and haematological systems (WHO, 2010). Reports from various researches suggest that no level of blood lead is safe to humans, especially children (Chandran and Cataldo, 2010).

Studies have shown that the source of lead to an infant's blood seems to be a mixture of about two thirds dietary and one third skeletal lead of the mother (Gulson*et al*., 2003). From the time of conception onward, lead that has been stored in the mother's skeleton in years past is released into the circulation under the metabolic stress of pregnancy. Throughout pregnancy, lead readily crosses from the maternal to the infant circulation, and the blood lead concentration of the infant equilibrates with that of the mother with time (Markowitz, 2000; Dart*et al*., 2004). Once in the infant, lead can penetrate the immature blood–brain barrier to gain entrance to the developing brain and cause some damages, even when the levels of exposure is very low (Lidsky and Schneider, 2003).

One mechanism underlying the neurotoxic effect of lead is its unique ability to replace other monovalent and divalent cations, especially sodium (Na^{+}), calcium (Ca^{2+}), magnesium (Mg^{2+}), iron (Fe^{2+}) and zinc (Zn^{2+}), at the molecular level in living organisms (Godwin, 2001; Lidsky and Schneider, 2003). In these molecular machineries, the binding is often with greater affinity than even Ca^{2+} and Zn^{2+}. These interactions allow lead to affect different biologically significant processes, such as metal transport, apoptosis, intercellular and intracellular signalling, energy metabolism, ionic conduction, cell adhesion, diverse enzymatic processes, protein maturation, genetic regulation and release and uptake of neurotransmitters (choline, dopamine and GABA). The developing central nervous system is particularly

susceptible in all of these processes (Bressleret *al*., 1999; Markowitz, 2000; Garzaet *al*., 2006). Various report have specified that these neuro-behavioural changes associated with early exposure to lead seems to be persistent and irreversible (Burnset *al*., 1999; Dietrichet *al*., 2001; Cecilet *al*., 2008; Wrightet *al*., 2008).

Another well-established mechanism lead toxicity is the orchestration of oxidative stress through its direct and or indirect effects on the antioxidant molecules and enzymes. Lead covalently interacts with the Sulphurhydryl (-SH) groups of Reduced Glutathione (GSH) and antioxidant enzymes – glutathione reductase, glutathione peroxidase and glutathione-S-transferase – thereby inactivating them (Hunaitiet *al*., 1995; Kasperczyket *al*., 2004; Ahamed and Siddiqui 2007). Some additional important antioxidant enzymes possibly inhibited by lead include superoxide dismutase and catalase (Floraet *al*., 2007).

At the central nervous system, lead is known to cause asymptomatic impairment of neuro-behavioural function in children at doses smaller than what can produce clinical encephalopathy. Early cross-sectional studies of the association between lead and Intelligence Quotient (IQ) had shown that clinically asymptomatic children with high blood lead had a four- to five-point reduction in mean verbal IQ scores compared with those from the same communities with lower blood lead (Landriganet *al*., 1975; Needlemanet *al*., 1979). At the peripheral nervous system, the motor axons are the prime target of lead toxicity. Lead-induced pathological changes in these fibres include segmental demyelination and axonal degeneration (WHO, 2010). Lanphearet *al*. (2005) pooled analysis of data from seven cohorts and confirmed that an increase in blood lead level from less than 1 µg/dL to 10 µg/dL was associated with a six IQ point decrement, which is considerably greater than the decrement associated with an increase in blood lead level from 10 µg/dL to 20 µg/dL. The reports of this pooled analysis have been confirmed by several other studies (Despréset *al*., 2005; Fraseret *al*., 2006; Huet *al*., 2006; Chiodoet *al*., 2007; Surkanet *al*., 2007).

In kidneys, lead aggregates in the mitochondria of kidneys causing both structural and functional damages. These damaging effects include swelling of the mitochondrial as well as prevention of respiratory chain function and oxidative phosphorylation for ATP generation. This leads to impairment of energy-dependent processes, including

tubular transport (Carocci*et al*., 2016). Lead can result in acute and chronic nephropathies. In acute lead nephrotoxicity, lead is absorbed by the proximal tubular cells and it binds to specific lead-binding proteins causing abnormal excretion of glucose, phosphates, uric acids, bicarbonates, and certain amino acids, a condition referred to as Fanconi's syndrome. On another other hand, chronic lead nephropathy is much more severe and causes irreversible morphological and functional changes in proximal tubules with a characteristic pathology of proximal tubule nuclear inclusion bodies that progress to tubulo-interstitial disease (accompanied by hypertension) and fibrosis. Lead accumulation in the proximal tubule leads to hyperuricaemia and gout, probably by inhibiting uric acid secretion, and also to diminished renal clearance, tubular reabsorption and glomerular filtration rate (Gonick, 2008; Rastogi 2008; Carocci *et al*., 2016).

Patients with diabetes and hypertension are at higher risk of clinical renal dysfunction at lower exposures to lead. A systematic review concluded that a slight positive relationship between exposure to lead and blood pressure has been identified in several studies in various settings, and that some of these studies have identified a dose-response relationship. This review drew a conclusion that the association between lead and hypertension is somewhat causal (WHO, 2010).

At low levels, such as below 10 µg/dL, the immune system (Bunn*et al*., 2001; Karmaus*et al*., 2005) and reproductive system (Wu*et al*., 2003; Iavicoli*et al*., 2006) are also grossly affected. Lead interferes with semen quality, lowering it (Telisman*et al*., 1990), reduces the volume of ejaculation, semen density, total sperm number and motility, and increases the percentage of pathological spermatozoa. Other effects of high blood lead are reduced libido, abnormal spermatogenesis, chromosomal damage, infertility and changes in serum testosterone (Goyer 1993; Wu*et al*., 2012). It has been discovered that women with severe lead intoxication are highly susceptible to prolonged and abnormal menstruations, infertility, miscarriage, stillbirth, premature membrane rupture, pregnancy hypertension and premature delivery (Flora*et al*., 2011).

Anaemia develops as a classic clinical sign of lead toxicity in erythrocytes. The level of lead in the blood greatly determines the severity and prevalence of lead-induced anaemia. Infants and iron deficient children are at higher risk of lead-induced clinical

anaemia. The anaemia induced by lead is caused mainly by disruption of heme biosynthesis; however, a higher rate of erythrocyte destruction may also have affected (Schwartz*et al*., 1990). Three enzymes are usually inhibited by lead in the heme biosynthetic pathway as shown below in Figure 2.6.

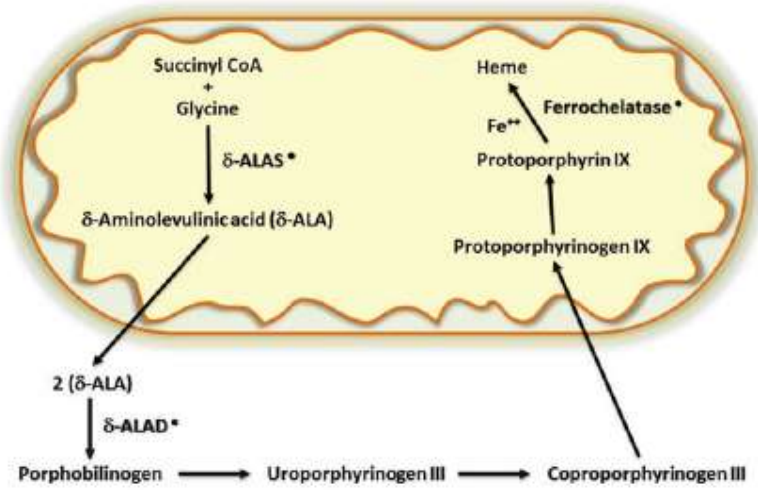

Figure 2.6:Inhibition of lead in the heme biosynthetic pathway (Carocci *et al*., 2016)

The initial and final steps of heme biosynthesis take place in the mitochondria, while some of the intermediate steps are carried out in the cytoplasm. Lead thus inhibits the first and the last mitochondrial enzymes – δ-Aminolevulinic Acid Synthase (δ-ALAS) and ferrochelatase, respectively – involved in the biosynthetic pathway as well as δ-Aminolevulinic Acid Dehydratase (δ-ALAD), which catalyses one of the cytosolic steps. It down-regulates these three enzymes in a dose-dependent manner (Piomelli, 2002). Heme synthesis only decreases when the activity of δ-ALAD is inhibited by 80–90 percent, and this occurs at a blood lead concentration of around 55 μg/dL (Ahamed*et al*., 2005).

It is now well established that pharmacologic therapy with chelating agents does not reverse neurocognitive defects in children who have lead neurotoxicity. Case identification is not an ultimate solution to reducing the ill effects of lead in a community, rather primary prevention encompassing environmental management, family education, and nutritional supplementation are critical. Lead poisoning can be effectively managed with a longitudinal and multidisciplinary approach (Yeoh*et al*., 2008; Chandran and Cataldo, 2010). Efforts should be channelled much more towards prevention rather than curing of lead toxicity.

2.6.2.4 Lead uptake by plants

As lead is not an essential element, plants do not have specific channels for its uptake. Instead, this element is attached to carboxylic groups of mucilage uronic acids on root surfaces (Morel*et al*., 1986; Sharma and Dubey, 2005). However, it is still unclear on how lead goes into the root tissue. Although some plants species (*Allium cepa*, *Hordeum vulgare* and *Zea mays*) tolerate lead through complexation and inactivation, other species (*Brassica napus* and *Phaseolus vulgaris*) experience toxicity because lead disrupts some metabolic pathways once it enters (Wierzbicka, 1999). In a few plant species, excess lead inhibits seed germination, plant growth and chlorophyll synthesis, among other deleterious effects (Xiong, 1998; Begonia*et al*., 2004).

Several studies have shown that most of the absorbed lead stays in roots, which makes the root the first plant-barrier for upward translocation of lead (Blaylock and Huang, 2000). Once inside the roots, most lead ions are bound to ion exchangeable sites in the cell walls and extracellular precipitation as carbonates and phosphates (Sahi*et al*.,

2002; Sharma and Dubey, 2005). The unbound lead is transported via calcium channels located near the endodermis (Huang and Cunningham, 1996; Antosiewicz, 2005). As reported by Cobbett (2000), lead, like other toxic metals, is complexed by the cysteine-rich low molecular weight polypeptides generally known as phytochelatins. However, lead is transported to stems and leaves in structures similar to lead sulphide, lead acetate, and lead nitrate in *Sesbania drummondii* (Sharma *et al*., 2004a,b). In addition, Lopez*et al*. (2009) have reported the formation of different lead complexes in stems and leaves of alfalfa.

2.6.2.5 Lead compounds in animals

It appears that the complexation of lead determines its level of toxicity. Lead (II) acetate (also known as sugar of lead) was used by the Roman Empire as a wine sweetener, and some believed this as a major cause of dementia that is known in several of the Roman Emperors. Leaded gasoline contains organic lead; so, since it was banned in the U.S. from 1976, exposure to organic lead is generally limited to work place. Organic lead can be more toxic when compared with inorganic lead because it is more readily absorbed by the body. Thus, potential exposures to organic lead should not be taken with levity. Lead in blood serum is bound to proteins or complexed with low molecular weight sulfhydryl compounds (such as, cysteine, homocysteine and glutathione,). Some other potential lead ligands in serum are low molecular weight molecules like citrate, oxalate, ergothioneine, and histidine (ATSDR, 2007). In addition, Hwang*et al*. (2009) reported that in humans there is a relationship between lead exposure and hearing losses.

2.6.2.6 Lead in the food chain

Limited studies have been published on the transport of lead in the food chain. Laskowski and Hopkin (1996) reported that the garden snail (*H. aspersa*) can store 43% of its lead from food, mainly in the soft tissue. The can be of great concern for the food chain. In addition, Milton*et al*. (2002) have reported that in Wales mine tailings, there is a strong relationship between lead in grass and the grasshopper *Chorthippus brunneus*. However, further studies are needed to determine the contributions of the plant lead-compounds to the food chain.

From the report of Ma (1996) the main route of exposure of humans and mammals to lead is via the food chain. This report indicated that in wildlife, a liver lead level above 10 µg per gram dry weight or a kidney lead level above 25 µg per gram dry weight can be taken as criteria for acute lead poisoning. The author also specified that more researches will be needful to establish the range of lead in blood or tissues that will give a no-adverse-effect diagnosis. In a study carried out in Spain, Capdevila*et al*. (2003) reported an average lead content of 0.073 µg per gram in edible vegetables. They estimated that the contribution of edible vegetables (mainly onions and parsley) to the total daily intake of lead was 14.6 µg.

Some other reports from animal experiments exist. Anders*et al*. (1982) treated adult white Carneaux pigeons with inorganic lead (6.25 mg lead per kg dry weight per day, gastric intubation) for up to 64 weeks and found that these birds had a marked and rapid hypochromic normocytic anaemia with an elevated erythrocyte porphyrin. In another study, red-tailed hawks treated with lead acetate at 0.82 mg lead per kgdry weight per day for 3 or 11 weeks developed an alteration in the heme biosynthetic pathway after the week 1. More so, the erythrocyte porphobilinogen synthase (aminolevulinic acid dehydratase) activity was significantly reduced and did not return to normality until 5 weeks after the experiment was terminated (Redig*et al*., 1991). Ku*et al*. (1978) found that rats fed with 300 mg lead per litre as lead acetate and phospholipid-bound lead accumulated with similar concentration of lead in tissues and bones.

In humans, Quintanilla-Vega*et al*. (1995) reported the presence of cytosolic protein that is responsible for the binding of lead with an apparent *K*d of 10^{-9} M in the brain. Other scientists have reported that in humans, two binding polypeptides are responsible for the binding of lead in the kidneys (Smith*et al*., 1998). One of them is thymosin and the other is acyl-CoA binding protein. These scientists indicated that these lead binding proteins have a *K*d ≈14nM and account for over 35 percent of the lead in kidney cortex.

2.7 Study locations

Ògùnstate is one of the states in South-West geopolitical zone of Nigeria. It was created on February 3, 1976 from the former Western State, by the military Head of State, Major General Olusegun Obasanjo. It has a land space of 16, 980.55 Km^2 (10, 544.92 square miles). It is bordered at the south by Lagos State, by Oyo and Osun states at the north, by Ondo state at the east and by the Republic of Benin at the west. It is between latitudes 6.20 and 7.90 north of the equator and longitude 2.50 and 4.40 east of Greenwich Meridian (Figure2.7) and has Abeokuta as the largest and capital city. It is fondly regarded as the 'Gateway to Nigeria'. As at 2006 census, the total number of residents was recorded as 3,751,140 (Wikipedia, 2017). Being so close to Lagos State and with diverse culture, Ogun State has most (at least 95 percent) of the food products consumed within the South-Western Nigeria.

Ogun state is well known to have several large industrial estates and as a major manufacturing hub in Nigeria. Major factories in Ogun include: Coleman Cables in Sagamu and Arepo, Dangote Cement factory in Ibese, Lafarge Cement factory in Ewekoro, Nestle in Agbara and Sagamu, and Procter and Gamble in Agbara, amongst several others (Wikipedia, 2017).

The State has twenty (20) Local Government Areas, which include: Abeokuta North, Abeokuta South, Obafemi Owode and Odeda in Abeokuta Health Zone (HZ); Ijebu East, Ijebu North, Ijebu North East, Ijebu Ode and Ogun Waterside in Ijebu Ode HZ; Ewekoro, Imeko Afon, Yewa North (formerly Egbado North) and Yewa South (formerly Egbado South) in Ilaro HZ; Ado-Odo/Ota, Ifo and Ipokia in Ota HZ; and Ikenne, Odogbolu, Remo North and Sagamu in Sagamu HZ (Wikipedia, 2017). Thus, Abeokuta South, Ijebu Ode, Yewa South (Ilaro), Ipokia and Ikenne (Ilisan) were randomly selected from each of the HZs. The selected sub-locations for study areas are shown in Figure 2.7.

The Federal Capital Territory (FCT), often referred to as FCT-Abuja, is a federal territory in North Central geopolitical zone (loosely known as Middle Belt) of Nigeria. Abuja, which is the capital city of Nigeria, is located within FCT. This territory was formed in 1976 from regions of Nasarawa, Niger and Kogi states. Geographically, it is located at the centre of Nigeria. The FCT is usually administered by the Federal

Capital Territory Administration that is overseen by a minister emanating from a presidential appointment; as different from the States, which are administered by elected Governors (Wikipedia, 2016c).

This territory is just at the north of the confluence of Niger and Benue Rivers. It is bordered by the Niger state at the West and North, by Kaduna state at the northeast, by Nasarawa state at the east and south, and by Kogi at the southwest (Figure 2.7). It is between latitudes 8.25 and 9.20 north of the equator and longitude 6.45 and 7.39 east of Greenwich Meridian. The FCT has a landmass of about 7,315 km^2 (4,542.62 square miles). As at 2006 census, the population of FCT residents was reported as 1,406,239. However, this population has been projected to reach 2,238,800 by the National Population Commission of Nigeria in 2011. A nice feature of Abuja as a benefit of its central location is that it shares the savannah grass with the northern Nigeria. This produces an overall effect of rich soil for Agriculture and enjoys an equable climate that is neither too hot nor too cold all year round (City Population, 2015; Wikipedia, 2016c; FCDA, 2017).

The FCT is currently divided into six local Area Councils (ACs), which are: Abaji, Abuja Municipal, Bwari, Gwagwalada, Kuje, and Kwali. Each AC operates with similar structure to what is obtainable in the LGAs of the States. Five (Abaji, Abuja Municipal (Karu), Bwari (Dutsen-Alhaji), Kuje, and Kwali) of the six ACs were randomly selected for study as sub-locations in Abuja. These selected sub-locations for study are shown in Figure 2.7.

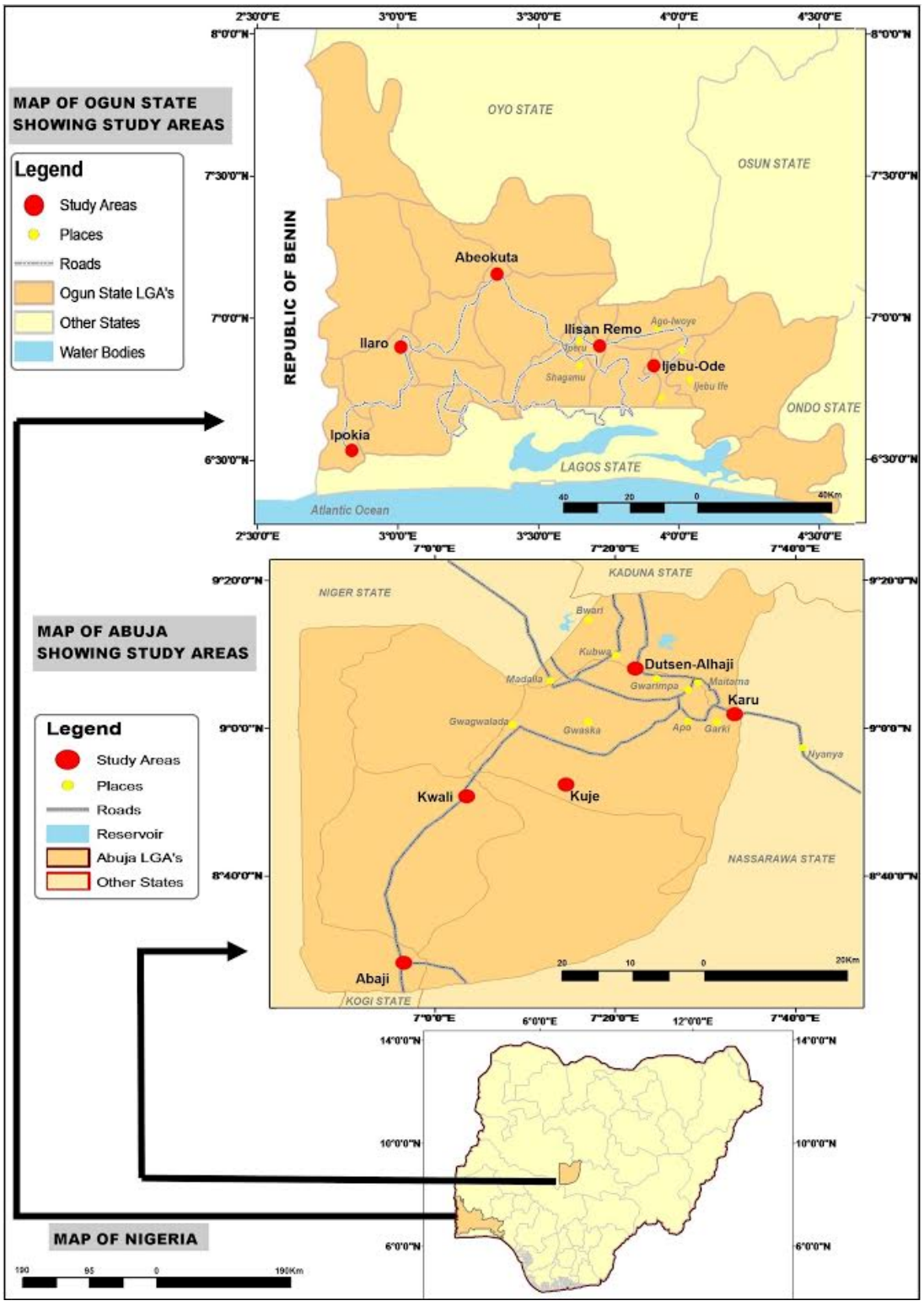

Figure 2.7: Study Sites in Ogun State and Federal Capital Territory (FCT), Abuja

CHAPTER THREE

METHODOLOGY

3.1 Study design and locations

The study was cross-sectional and comparative in design involving both survey and laboratory analyses. A five-stage sampling technique was used. Ogun State and Federal Capital Territory (FCT), Abuja, were purposively selected to represent two distinct geographical locations within the country. Ogun State was then stratified into five Health Zones (HZs) with several Local Government Areas (LGAs) and FCT was stratified into six Area Councils (ACs). One LGA was randomly selected from each of the five HZs in Ogun State, making five LGAs altogether, while five ACs were randomly selected from the six ACs in FCT. A town or city was further randomly selected from each LGA and AC respectively; making ten towns/cities altogether. Each selected town/city (sub-location) was then stratified with the selection of at least three representative strata for study. In each stratum, respondents were invited by the contact person to meet at a designated venue for questionnaire administration and blood sample donation.

Dietary pattern survey was carried out in each of the ten selected towns and cities in Ogun State and Federal Capital Territory, Abuja. Blood samples of volunteers were collected during this survey for evaluation of heavy metal concentration. A food list generated from the survey was used to purchase the market baskets for food analyses. Ipokia, Ilaro, Abeokuta-South, Ilisan and Ijebu-Ode were the selected sub-locations in Ogun State, while Kwali, Kuje, Abaji, Dutsen Alhaji and Karu were the selected sub-locations in Abuja (Figure 2.7).

3.2 Dietary pattern survey

This involved the administration of a questionnaire (Appendix 1) covering the demographic, socio-economic characteristics as well as dietary diversity and food

frequency pattern of the adult participants (18 years and above) in each of the ten locations.

The sample size was calculated using the conventional sample size formula:

$$\text{Sample size} = \frac{Z_{(1-\alpha/2)}{}^2 \, p\,(1-p)}{d^2}$$

Where $Z_{(1-\alpha/2)}$ = standard normal variate from probability table (1% type 1 error was chosen for this study which yields a score of 2.58)

p = prevalence of those with good dietary pattern (50% maximum score was taken)

d = absolute error or precision (5% was selected)

This gives a minimum of 666, approximated to 700 participants. A target of at least 150 participants in each of the 5 sub-locations under Ogun State and Abuja respectively was set. A projection of 20 percent of volunteer participants was also set for blood samples collection in each location.

However, 2,027 adult participants were eventually studied, which included 1044 and 983 of them from five sub-locations each in Ogun State and FCT, Abuja, respectively. On the other hand, 336 adult participants volunteered to give their blood samples for heavy metal analysis. This included 205 and 131 of them from five sub-locations each in Ogun State and FCT, Abuja, respectively.

3.3 Classification of the Socio-Economic Status (SES) and dietary patterns

The study population was stratified into three Socio-Economic Status (SES) based on the household head income, housing material, primary source of energy for cooking, primary method of sewage disposal, and the possession or non-possession of television, radio, video CD player, fan, fridge and cable television subscription like MultiChoice's Digital Satellite Television (DSTV). The three strata are the low, medium and high SES based on the 11-point scale developed from the factors listed above. Based on the distribution of the participants after a principal component analysis, those having 1-9 points were classified as having low SES, those with 10 points were classified as having medium SES and those with 11 points were classified as having high SES.

Dietary Diversity Questionnaire (DDQ), Food Frequency Questionnaire (FFQ) and two non-consecutive multiple-pass 24-hr dietary recalls were used to describe dietary patterns of participants. The DDQ of the Food and Agriculture Organization of the United Nations (FAO) was adapted, administered and analysed according to the guidelines (FAO, 2011). It included a 16-point food group-scale that queries the groups of foods consumed by the individual, a day before the interview. The 16 food group-scale was then collapsed into 9 food groups at the point of analysis, in line with the guidelines. The participants that consumed less than six food groups were classified as having low Dietary Diversity Score (DDS), while those that consumed six or more food groups were classified as having high DDS. All the 2027 adult participants were involved in the completion of the DDQ.

The FFQ included eleven food frequency options, varying from once daily to rarely or less than once monthly consumption pattern. For convenience of reporting, the eleven options were collapsed into five main options, which are: daily or greater than four times weekly, 3-4 times weekly, 1-2 times weekly, 1-3 times monthly and rarely or less than once monthly consumption patterns. All the 2027 adult participants were involved in the completion of the FFQ.

For the 24-hr dietary recall, the USDA's Multiple-pass method was adopted in which trained interviewers were made to carry out the 5-step dietary interview to each volunteer participant. The interview was conducted at two non-consecutive days for each volunteer, including a week day and a weekend day. About one-third of the participants were interviewed with 386 being from Ogun State and 349 from Abuja.

3.4 Food selection and sample collection

For inclusion in the food list, individual foods that were consumed by at least 5% of the study population on a daily basis (27 of them, mostly staples) as determined from the result of the food frequency questionnaire (Table 4.6) were of greatest priority. Then, several other foods were added to allow for appropriate regional food diversity representation and seasonality variation. A total of six hundred and five (605) individual food samples representing 142 food items were purchased for analyses between August and October, 2014. The food items were purchased as consumed from two to six major food vendors (for each food item) selected by systematic random

sampling from the major market in each of the sub-locations. The mean number of individual food sample purchased was 4.2 (min: 2, max: 23, Table 4.1).The variation in number of samples was dependent on availability (in the market) and the size of the specific food item. Eleven food groups (tubers, starches and their products; cereals and cereal products; legumes and legume products; fruits; leafy and fruity vegetables; condiments, sauces and soups; dairy products; beef, poultry and eggs; fish; oil seeds; and sugar and cocoa product) were included within the food purchased and analysed. In the same order of the food groups indicated, specific food items analysed were cassava products, yam and yam products, plantain, various kinds of rice, maize and their products, wheat products, various kinds of beans and their products, various kinds of fruits, various kinds of leafy and fruity vegetables, various kinds of stews, soups and condiments, milk and milk products, beef, poultry, egg, different kinds of fish, various kinds of nuts, sugar and cocoa product. Specific details of these food items are highlighted in Table 4.1.A subsample of each was obtained for analysis. Only wholesome samples were used for the analysis. These consist of edible portions 'as consumed', whereas bruised or rotten parts were removed.

3.5 Food preparation

All the food items were obtained from hygienic food stalls or vendors. They were collected in the form 'as consumed' by the residents of each sub-location. Food samples were transferred to the laboratory immediately after collection to minimise deterioration of highly perishable ones. In some cases, skin, bone and head of some fish were removed after collection; some fruits had to be washed and peeled, shells of oil seeds like coconuts and walnuts had to be removed before further processing was done. All preparations were done using tap water as available in the laboratory and in the communities where they were collected. Thereafter, they were all oven-dried at 100 ± 5 ^{o}C using hot air oven (Gallenkamp, England) and pulverized with Eurolex blender (model GM 1153, Eurolex India). Individual food samples were separately stored in polyethylene bags at –20 ^{o}C until when needed for analysis.

3.6 Processing and analytical instruments for metal analyses

The dry ashing was done using the Carbolite muffle furnace (model S302AU, Bamford, Sheffield, England). The Varian atomic absorption spectrometer (AA240FS series, California, USA) was used for the determination of macro- and micro-minerals in the food samples. Both determinations were carried out using air/acetylene flame. Determination of cadmium and lead in food samples was done using Varian 210 Graphite Furnace Atomic Absorption Spectrometer (GFAAS), which requires argon gas for graphite tube conditioning. A Buck Scientific flame atomic absorption spectrometer (210 VGP model, East Norwalk, Connecticut, USA) was used for determination of the metal levels in blood samples. Operating parameters for the metals determined were set as recommended by the manufacturer. Analysis was carried out at the most sensitive analytical spectral lines of the metals. Details of the instrumental conditions are available in Tables 3.1, 3.2 and 3.4.

Table 3.1: Instrumental conditions for Flame Atomic Absorption Spectrometer (FAAS) for the determination of macrominerals and microminerals in food samples

Element	Acetylene (L min^{-1})	Air (L min^{-1})	Wavelength (nm)	Slit width (nm)	Lamp current (mA)	Lamp mode
Potassium	2	13.5	766.5	1.0	5	NON-BGC[a]
Sodium	2	13.5	589.0	0.5	5	NON-BGC[a]
Calcium	2	13.5	422.7	0.5	10	NON-BGC[a]
Magnesium	2	13.5	285.2	0.5	4	BGC-D2[b]
Copper	2	13.5	324.8	0.5	4	BGC-D2[b]
Manganese	2	13.5	279.5	0.2	5	BGC-D2[b]
Iron	2	13.5	248.3	0.2	5	BGC-D2[b]
Zinc	2	13.5	213.9	1.0	5	BGC-D2[b]

[a]NON-BC, no background correction
[b]BGC-D2, deuterium background correction

Table 3.2: Instrumental analytical conditions and graphite furnace programmes for determination of cadmium and lead in food samples by GFAAS

Working conditions	Cadmium	Lead
Wavelength (nm)	228.8	283.3
Slit width (nm)	0.5	0.5
Lamp current (mA)	4	10
Ar flow (L/min)	3.0	3.0
Sample volume (µL)	20	20
Modifier (µL)	5	5
Heating program temperature (^{o}C) (ramp time (s), hold time (s) (flow interrupted at atomization stage)		
Drying 1	110 (5, 35)	110 (5, 35)
Drying 2	130 (3, 15)	130 (3, 15)
Ashing	250 (3, 10)	400 (3, 10)
Atomization	1800 (0.8, 3)	2100 (0.9, 3)
Cleaning	2100 (0.9, 2)	2300 (0.9, 2)

3.7 Sampling of food and processing for metal analyses

This was done at the Departments of Agronomy and Chemistry, University of Venda, Thohoyandou, Limpopo province, South Africa. The dry ashing was done by the method of Crosby (1977) as modified by Akinyele and Shokunbi (2015b) by weighing 1 g of each dried sample into a porcelain crucible and dry-ashed in a muffle furnace by stepwise increase of temperature up to 500 ^{o}C in 1 h and then leaving to ash at this temperature for the next 12 h. The residue was dissolved in 10 mL of 1 M nitric acid (67%, v/v), filtered into a 50 mL volumetric flask using Whatman filter paper (No 41) and made up to mark with nitric acid (1 M). The blank digest was similarly processed, one per batch of 15 food samples. The Limits of Detection (LOD) and Quantification (LOQ) were calculated in accordance with the NF EN 13804 (AFNOR, 2002). They were specified as 3 and 6 times the standard deviation of the mean of 21 independent blank tests after due correction by weight of sample (1g) and the dilution (1:50). The LOD obtained ranged from 0.00006 to 0.251 mg/100g edible portion while LOQ ranged from 0.00012 to 0.502 mg/100g edible portion (Table 3.3). The mean values obtained for all the metals determined in the food samples were derived on dry weight basis. These values were then converted to equivalent mean values on wet weight basis as currently reported, using the derived equation below:

B = A *(1 – m/100) where A = mineral concentration on dry weight basis; B = mineral concentration on wet weight basis; and m = moisture content of the food sample.

3.8 Blood sample collection and preparation

Blood samples were collected from each of the 10 study locations. A total of 336 volunteers, who were apparently healthy adults (18 years and above) with no personal sign or history of sickle cell anaemia, diabetes, hypertension and related non-communicable diseases, participated in blood specimen donation. Respondents that recently completed the use of or were currently on antimalarial, anti-convulsant, antiretroviral, chemotherapeutic and anti-tuberculosis drugs were excluded. Smokers were also excluded from blood sample donation.

Table 3.3: Limits of Detection (LOD) and Limits of Quantification (LOQ) for macrominerals, microminerals, and heavy metals in food samples

Element	LOD (mg/100g EP[a])	LOQ (mg/100g EP[a])
MACROMINERALS		
Potassium (K)	0.207	0.414
Sodium (Na)	0.251	0.502
Calcium (Ca)	0.178	0.356
Magnesium (Mg)	0.160	0.320
MICROMINERALS		
Copper (Cu)	0.0154	0.308
Manganese (Mn)	0.0164	0.328
Iron (Fe)	0.0140	0.0280
Zinc (Zn)	0.0146	0.0292
HEAVY METALS		
Cadmium (Cd)	0.00006	0.00012
Lead (Pb)	0.00044	0.00088

[a]Edible portion (EP) based on fresh weight

All blood samples were collected by licensed Nurse or certified medical laboratory scientist via cubital venepuncture, into containers pre-labelled with the volunteer'sidentification number and date. For each participant who provided written consent, 5 mL blood sample was collected for the measurement of the concentrations of copper, zinc, iron, chromium, nickel, manganese, cadmium and lead. From each 5 mL blood sample collected, about 3.5 mL was dispensed into plain tube for the analyses of serum copper, zinc, iron, chromium and nickel, while about 1.5 mL was dispensed into heparinized tube for the analysis of whole blood manganese, cadmium and lead.

The samples collected in plain tubes were allowed to coagulate and the serum obtained after centrifuging with a bench centrifuge at 3000 g for 10 minutes. Following collection, the samples were kept in a cold chain system supported with ice packs (at about 0–4 ^{o}C) and were processed within 48 h for long-term storage at −20 ^{o}C until analysis. Samples were typically analysed within three months of collection.

3.9 Sampling of blood and processing for metal analyses

In an effort to obtain the most accurate heavy metal levels in the blood samples, a recovery experiment was set up in which blood samples of known metal concentrations were digested with various acids/mixtures of acids. The results obtained are as presented in Tables 4.3a and 4.3b. Eventually, digestion with concentrated HCl and HNO_3 of 3:1 was noted to yield the best set of results and thus was adopted. Briefly, 5ml of the acid mix was measured into 25ml beakers and 200µl of serum or whole blood sample was added respectively into separate beakers. The preparation was digested on a hot plate at 100^{o}C for about one hour or till a clear digest is obtained. The digest was then allowed to cool down, filtered using Whatman filter paper (No. 41), into a 10ml volumetric flask and made up to mark with distilled-deionized water. This way organic matter was destroyed in the sample and a high level of concentration of the sample solution was achieved. The blank digests and spiked samples were similarly handled. The sample solutions were analysed for heavy metal contents using a Flame Atomic Absorption Spectrometer (FAAS) (Buck Scientific 210 VGP model). The levels of copper, zinc, iron, chromium and nickel were determined in the serum samples while the levels of manganese, cadmium and lead were

determined in whole blood samples. The instrumental analytical conditions of the investigated metals are as highlighted in Table 3.4.

Table 3.4: Instrumental analytical conditions of investigated elements

Metal	Wavelength (nm)	Slit (nm)	Sensitivity Check (µg/ml)	Linear Range (µg/ml)	Flame Type Colour
Copper	324.8	0.7	2.00	5.00	A-A, lean/blue
Zinc	213.9	0.7	0.50	2.50	A-A, lean/blue
Iron	248.3	0.7	2.50	5.00	A-A, lean/blue
Chromium	357.9	0.7	2.00	5.00	A-A, rich/yellow
Nickel	341.5	0.2	7.00	8.00	A-A, lean/blue
Manganese	279.5	0.7	1.25	2.50	A-A, lean/blue
Cadmium	228.9	0.7	0.75	2.00	A-A, lean/blue
Lead	283.3	0.7	10.00	20.0	A-A, lean/blue

3.10 Quality assurance

Some criteria were implemented after due validation for the determination of the 12 metals in the blood and food samples. The results of these metal analyses were validated using five quality control checks:

1. Samples were generally carefully handled to avoid contamination. All glass wares were properly cleaned with 2M nitric acid and then rinsed thoroughly with distilled-deionized water or the ultra-pure water ($18M\Omega cm^{-1}$) (Merck Milli-Q, Germany). The concentrated acids used for the digestion were analytical grades from Merck, Germany. Ultra-pure water was used throughout the processing and analysis steps.

2. For every batch of 15 individual food samples ashed, one blank sample was usually processed. One blank sample was processed for every 12 digest, in the case of the blood samples. This was to monitor any cross contamination or protocol memory effects. All sample values were presented after due subtraction of the mean of the blank values.

3. In the analyses of all elements, the calibration curves had no outliers ($r^2 > 0.995$; four points). Whenever an outlier was observed, a re-calibration was done.

4. In a way to ascertain the trueness of the results and possible instrumental interferences, recovery studies were done. In the case of food sample 15 different samples were spiked before being dry-ashed and read on the AA240FS and GFAAS. Spike concentrations varied from 1 µg/L for cadmium and lead to 10000 µg/L for potassium which corresponds with 0.05mg/kg for cadmium and lead to 500 mg/kg for potassium. All recovery results were within 94-108% (Table 4.2).For the blood samples, 8 different samples were used relative to the adopted acid mixture for whole blood and serum analysis. The recovery results were within 69-99% (Tables 4.3a and 4.3b).

5. All samples were analysed in duplicate to eliminate errors that could come up from each batch and to monitor whether the results are repeatable – reproducible. The extent of variation between duplicate results was calculated as Relative

Standard Deviation (RSD). Results were accepted only when RSD was < 15%. Otherwise, samples were reanalysed.

3.11 Ethical considerations

Ethical approval (with reference number: UI/EC/13/0053) for this study was obtained from UI-UCH ethical review Board. A scanned copy of the approval document is as presented in Appendix 2. Every protocol was run in accordance with guidelines regulating studies on human subjects from National Institute of Health.

3.12 Statistical Analyses

Some data were analysed using descriptive statistics, while others were analysed by independent student *t*-test of Statistical Package for Social Sciences (SPSS) version 21.0 and differences were considered statistically significant at $p< 0.05$.

CHAPTER FOUR

RESULTS

4.1 Frequency distribution of participants in study sites

Figure 4.1 shows the frequency distribution of participants of this study from the ten sub-locations. Two thousand and twenty-seven (2027) apparently healthy adults participated in the study. Ilisan had the highest (220) number of participants while Karu has the least (192). About half (51.4%) of the participants were from Ogun State.

4.2 Characteristics of Food Samples Analysed

Table 4.1 shows some characteristics such as groups, scientific names, common names and number of individual food samples used in the analyses of the food samples studied. Virtually all Nigerian staple foods are included on the list. Cereals and cereal products group (28) has the highest number of individual food items, followed by Condiments, sauces and soups (27) and then fruits (19).

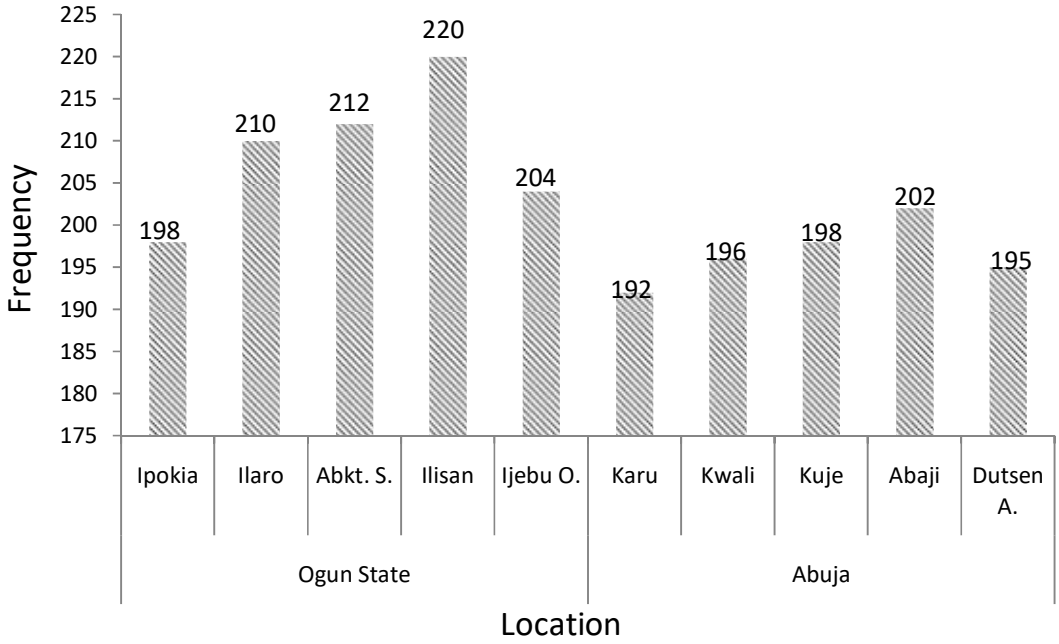

Figure 4.1: Frequency distribution of participants by location

Table 4.1: Characteristics of the food samples collected for metal analyses

Food group	Scientific name	Common name and preparation mode	Number of samples (n)
Tubers, starches and their products	*Manihot esculenta*	Fufu (cooked)	11
	Ipomoea batatas	Sweet potato (fried)	4
	Dioscorea rotundata	Amala (yam powder, cooked)	10
	Dioscorea rotundata	Pounded yam	6
	Dioscorea rotundata	Yam (white, fried)	8
	Dioscorea rotundata	Yam (white, boiled)	2
	Dioscorea rotundata	Poundo yam (cooked)	2
	Dioscorea rotundata	Yam pottage (white yam)	2
	Dioscorea alata	Cake (water yam, fried)	2
	Manihot esculenta	Eba (white gari, cooked)	9
	Artocarpus altilis	Bread fruit (boiled)	2
	Musa paradisiaca	Plantain (mature ripe, fried)	6
	Musa paradisiaca	Plantain chip (mature unripe, fried)	4
Cereals and cereal products	*Oryza sativa*	Rice (Long Grain, boiled)	14
	Oryza sativa	Rice (Short Grain, boiled)	3
	Oryza glaberrima	Local Rice (boiled)	4
	Oryza sativa	Jollof Rice	5
	Oryza sativa	Fried Rice	3
	Oryza sativa	Masa (rice cake, fried)	3
	Oryza sativa and *Vigna unguiculata*	Garogaro (rice, beans, boiled; along with fresh sauce)	2
	Zea mays	Maize (yellow, roasted)	6
	Zea mays	Maize (yellow, boiled)	2
	Zea mays	Maize (white, roasted)	2
	Zea mays	Tunwo (white maize, cooked)	4
	Oryza sativa	Tunwo (rice, cooked)	3
	Triticum aestivum	Bread (white)	12
	Triticum aestivum	Whole wheat bread	3
	Triticum aestivum	Malt bread	2
	Zea mays	Agidi (white maize, cooked)	10
	Zea mays	Kokoro (white maize, fried)	2
	Triticum aestivum	Semovita (cooked)	8
	Zea mays and *Arachis hypogaea*	Donkwa	2
	Pennisetum glaucum	Fura	2
	Avena sativa	Oat (cooked)	2
	Zea mays	Custard (cooked)	2

Table 4.1: Characteristics of the food samples collected for metal analyses (continued)

Food group	Scientific name	Common name and preparation mode	Number of samples (n)
Cereals and cereal products	*Triticum aestivum, Arachis hypogaea* and *Cocos nucifera*	Granola	2
	Zea mays	Cornflakes	2
	Triticum aestivum	Spaghetti (boiled)	2
	Triticum aestivum	Jollof spaghetti	2
	Triticum aestivum	Noodles (plain, boiled)	6
	Triticum aestivum	Noodles (boiled, with steamed or fried egg)	3
Legumes and legume products	*Vigna unguiculata*	Bean pottage (White bean)	4
	Vigna unguiculata	Bean (Drum, plain, boiled)	8
	Vigna unguiculata	Bean (White, plain, boiled)	2
	Vigna unguiculata	Bean (Olo 2, plain, boiled)	2
	Vigna unguiculata	Bean (Pewu, plain, boiled)	2
	Vigna unguiculata	Akara (White bean, fried)	3
	Vigna unguiculata	Akara (Pewu bean, fried)	2
	Vigna unguiculata	Akara (Drum bean, fried)	2
	Vigna unguiculata	Moi-moi (White bean, cooked)	2
	Vigna unguiculata	Moi-moi (Olo 2 bean, cooked)	2
	Vigna unguiculata	Okpa bean (boiled)	2
	Vigna unguiculata	Moi-moi (Okpa bean, cooked)	4
	Glycine max	Tofu pie	2
	Glycine max	Tofu (fried)	4
	Arachis hypogaea and Oryza sativa	Kunu (gyada)	2
Fruits	*Musa sapientum,* Musa acuminata	Banana (ripe)	14
	Daucus carota subsp. Sativus	Carrot (raw)	3
	Cucumis sativus	Cucumber	9
	Citrus sinensis	Orange	14
	Carica papaya	Pawpaw	9
	Ananas comosus	Pineapple	7
	Citrullus lanatus	Water melon (fruit without seed)	15
	Citrullus lanatus	Water melon (seed)	2
	Cucumis melo	Golden melon	2
	Psidium guajava	Guava	4
	Phoenix dactylifera	Date	3
	Cyperus esculentus	Tiger nut (yellow, raw)	2
	Cyperus esculentus	Tiger nut (brown, raw)	2
	Malus domestica	Apple (wine)	6

Table 4.1: Characteristics of the food samples collected for metal analyses (continued)

Food group	Scientific name	Common name and preparation mode	Number of samples (n)
Fruits	*Malus domestica*	Apple (green)	12
	Malus domestica	Apple (wine/green)	2
	Pyrus communis	Pear apple (European Pear)	4
	Citrus tangerine	Tangerine	4
	Prunus 'Black Amber'	Plum	2
Leafy and fruity vegetables	*Solanum melongena*	Garden egg (light yellow)	7
	Solanum melongena	Garden egg (green)	7
	Solanum melongena	Garden egg (reddish)	2
	Solanum melongena	Garden egg (lemon)	2
	Capsicum anuum Group	Green bell pepper (raw)	3
	Pisum sativum	Green peas (raw)	6
	Phaseolus vulgaris	Green bean (raw)	2
	Brassica oleracea var. capitata	Cabbage (raw)	7
	Brassica oleracea var. capitata	Cole slaw	3
Condiments, sauces and soups	Sodium chloride	Salt	4
	Parkia biglobosa	Locust bean (raw)	6
	-	Stew (for rice, with vegetable oil)	23
	-	Stew (for tunwo)	2
	-	Stew (with palm oil)	4
	-	Stew (for masa)	2
	-	Stew (for swallow)	2
	-	Stew (for local rice)	2
	Gallus gallus domesticus	Egg stew	2
	Arachis hypogaea	Groundnut soup	2
	Vigna unguiculata	Gbegiri	2
	Irvingia gabonensis	Ogbono soup	2
	Telfairia occidentalis	Fried ugwu	2
	Abelmoschus esculentus	Okra (plain)	4
	Abelmoschus esculentus	Okra soup	4
	Amaranthus hybridus	Tete soup	2
	Corchorous olitorius	Ewedu (plain)	7
	Celosia argentea	Soko soup	2

Table 4.1: Characteristics of the food samples collected for metal analyses (continued)

Food group	Scientific name	Common name and preparation mode	Number of samples (n)
Condiments, sauces and soups	*Telfairia occidentalis*, *Cucumeropsis mannii*	Ugwu soup (with egusi)	7
	Amaranthus hybridus, Cucumeropsis mannii	Tete soup (with egusi)	3
	Amaranthus hybridus	Efo riro (tete)	2
	Celosia argentea, *Cucumeropsis mannii*	Soko soup (with egusi)	2
	Talinum triangulare	Waterleaf soup	4
	Talinum triangulare, *Telfairia occidentalis*	Water leaf (with ugwu) soup	2
	Celosia argentea	Efo riro (soko)	3
	Gnetum africanum, *Cucumeropsis mannii*	Ukase soup (with egusi)	2
Dairy products	-	Nunu (locally fermented milk)	2
	-	Powdered low-fat milk (raw)	2
	-	Powdered full-cream milk (raw)	3
	-	Cheese (local)	2
Beef, poultry and eggs	*Bos taurus indicus*	Beef (boiled, in stew)	11
	Bos taurus indicus	Beef (fried, in stew)	9
	Bos taurus indicus	Ponmo (raw)	6
	Gallus gallus domesticus	Chicken thigh (fried)	5
	Meleagris gallopavo	Turkey wing (fried)	5
	Gallus gallus domesticus	Egg (boiled)	4
	Gallus gallus domesticus	Egg (boiled, in stew)	3
Fish	*Clupea harengus*	Shawa (fried)	6
	Clupea harengus	Shawa (tiny, fried)	2
	Clupea harengus	Shawa (smoked)	4
	Trachurus trachurus	Kote (fried)	5
	Scomber scombrus	Titus (fried)	4
	Scomber scombrus	Titus (smoked)	2
		Ebolo (smoked)	2
	Clarias gariepinus	Cat fish (roasted)	5
	Farfantepenaeus notialis	Cray fish (roasted)	4
		Panla (roasted)	4
		Panla (fried)	3

Table 4.1: Characteristics of the food samples collected for metal analyses (continued)

Food group	Scientific name	Common name and preparation mode	Number of samples (n)
Fish (continued)	Stockfish	Stockfish	3
	Pseudotolithus elongatus	Apo (roasted)	3
		Palamu (roasted)	3
Oil seeds	*Arachis hypogaea*	Ground nut (roasted)	6
	Arachis hypogaea	Ground nut (boiled)	6
	Anacardium occidentale	Cashew nut (roasted)	2
	Juglans regia	Walnut (boiled)	2
	Cocos nucifera	Coconut (fresh)	3
Sugar and cocoa product	*Saccharumspp.*	Refined sugar (raw)	4
	Theobroma cacao	Cocoa product (raw)	2
		TOTAL	605

4.3 Recoveries of macrominerals and microminerals from food items

The mean recoveries of potassium, sodium, calcium, magnesium, copper, manganese, iron, zinc, cadmium and lead from various food samples analysed after sample processing with the method of Crosby (1977) as modified by Akinyele and Shokunbi (2015b) are as presented in Table 4.2. The recoveries ranged from 94-108% and thus can be rated to be highly quantitative. Spiking at low and high concentrations of all the metals yielded excellent recoveries.

Table 4.2: Recovery (%) of macrominerals, microminerals and heavy metals from various food items

Metal	Sample weight (g)	Spiked concentration[a] (mg/100g)	Mean Recovery concentration (mg/100g)	Mean Percentage recovery (%)
K	1.00	10.0	10.4	104.1 ± 3.6
	1.00	50.0	47.0	94.4 ± 1.9
Na	1.00	1.00	1.08	107.6 ± 4.4
	1.00	10.0	10.0	100.2 ± 2.1
Ca	1.00	5.00	5.15	102.8 ± 1.5
	1.00	25.0	24.0	96.4 ± 2.3
Mg	1.00	10.0	9.50	95.3 ± 1.8
	1.00	40.0	42.4	106.0 ± 3.3
Cu	1.00	0.100	0.108	108.2 ± 4.9
	1.00	0.500	0.515	103.4 ± 2.1
Mn	1.00	0.100	0.106	105.9 ± 3.8
	1.00	0.500	0.485	97.3 ± 2.6
Fe	1.00	0.200	0.200	100.4 ± 2.3
	1.00	1.00	1.04	104.1 ± 3.2
Zn	1.00	0.200	0.212	105.6 ± 4.1
	1.00	1.00	1.03	103.3 ± 1.9
Cd	1.00	0.005	0.0052	103.0 ± 2.2
	1.00	0.020	0.0208	104.2 ± 2.7
Pb	1.00	0.005	0.0052	103.4 ± 1.6
	1.00	0.020	0.0214	106.8 ± 4.5

[a] corresponding to 0.1, 0.4, 2.0, 4.0, 10.0, 20.0, 100, 200, 500, 800 and 1000 µg/100 mL respectively, for a sample weight of 1.0g and a final volume of 50 mL

4.4 Recovery experiments on whole blood and serum samples

The average recoveries (%) of the microminerals and heavy metals from whole blood and serum using individual acid or acid mixtures are as shown in Tables 4.3a and 4.3b. For the whole blood results (Table 4.3a), nitric acid only, nitric acid:hydrochloric acid (3:1), nitric acid:hydrochloric acid (1:3) and hydrochloric acid only yielded recoveries of 47-90%, 50-98%, 71-99% and 48-100%, respectively. Similarly, serum samples digested with nitric acid only, nitric acid:hydrochloric acid (3:1), nitric acid:hydrochloric acid (1:3) and hydrochloric acid only produced recoveries of 66-93%, 62-92%, 72-95% and 59-94%, respectively (Table 4.3b). Digestion with nitric acid:hydrochloric acid (1:3) produced the best set of recoveries considering all the metals on a general note, hence this acid mixture was selected for digestion of the rest of samples for the whole blood and serum analyses.

Table 4.3a: Recovery (%) of microminerals and heavy metals from whole blood samples

Metal	Spiked sample digested with HNO_3 only	Spiked sample with HNO_3 :HCl of 3:1	Spiked sample with HNO_3 :HCl of 1:3	Spiked sample digested with HCl only
Cu	85	70	71	48
Zn	90	98	99	100
Fe	57	76	95	69
Cr	47	50	84	66
Ni	57	70	71	74
Mn	71	71	80	72
Cd	73	81	93	72
Pb	56	69	90	73

Table 4.3b: Recovery (%) of microminerals and heavy metals from serum samples

Metal	Spiked sample digested with HNO_3 only	Spiked sample with HNO_3 :HCl of 3:1	Spiked sample with HNO_3 :HCl of 1:3	Spiked sample digested with HCl only
Cu	79	70	77	59
Zn	93	92	95	94
Fe	81	80	87	86
Cr	73	66	74	73
Ni	75	72	72	73
Mn	68	66	90	78
Cd	74	77	79	73
Pb	66	62	69	72

4.5 Distribution of study participants by gender and religious practice

The gender distribution of the study population is presented in Figure 4.2. The number of women (1320) far exceeded that of men (707) at both Ogun State and Abuja. Figure 4.3 shows the distribution of participants by religious practice. At both locations, majority were Christians, followed by Muslims. There were very few traditional worshippers among the respondents.

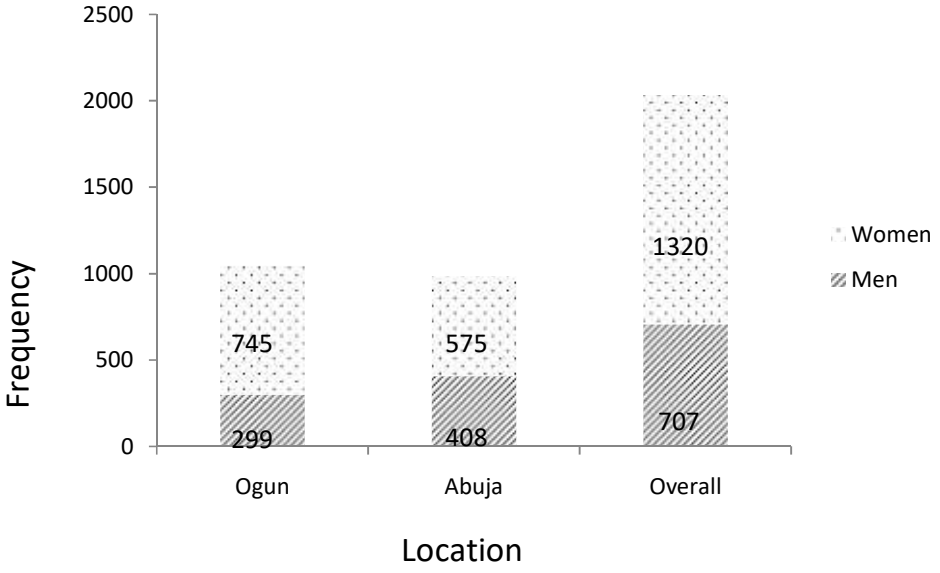

Figure 4.2: Gender distribution of participants by location

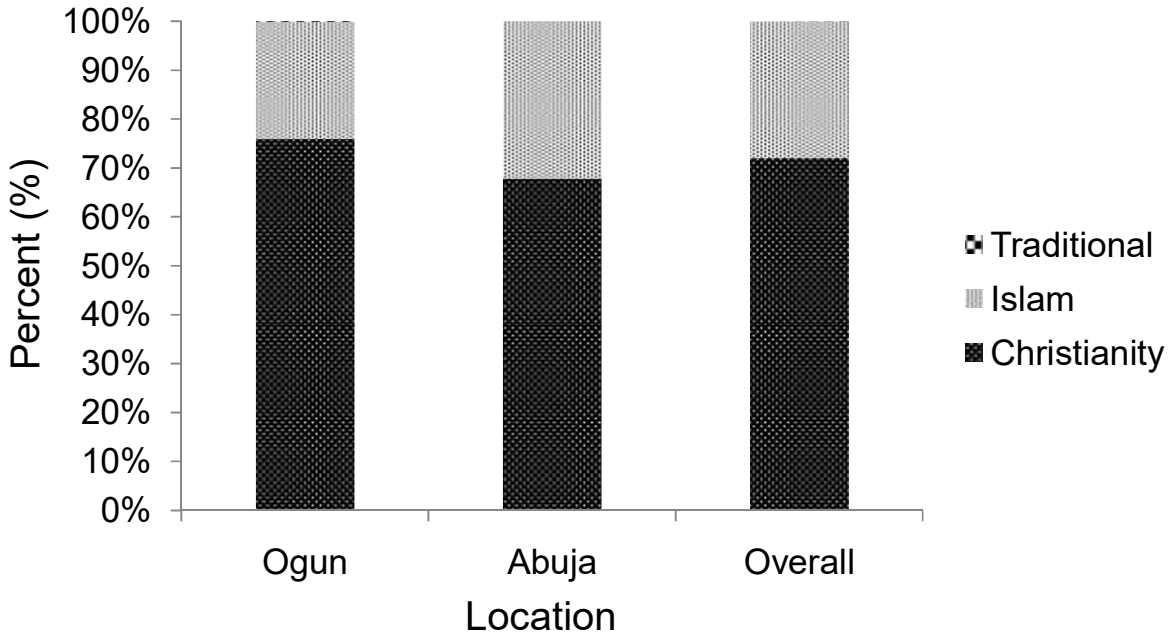

Figure 4.3: Percent distribution of participants by religious practice

4.6 Distribution of study participants by highest level of education

Figure 4.4 shows the distribution of the participants according to their highest level of education attained. Majority of them attended school up to the tertiary level, especially participants from Ogun State. Most participants that attended school up to primary or secondary school level were from Abuja. Eighty-two of them had no education: 38 (3.63%) from Ogun and 44 (4.48%) from Abuja. Less than 20 (0.69%) of them had informal education.

4.7 Occupation of household heads and spouses

Figure 4.5 shows the occupation of the household heads and spouses of those that participated in the study. Civil servants had the highest proportion while the artisans had the least at both locations. Among participants from Ogun State, the household heads were civil servants > private company workers > artisans > traders > farmers. The household heads in Abuja were civil servants > farmers > private company workers > traders > artisans. Most spouses in Ogun State were civil servants, followed by traders, then private company workers. In Abuja, most spouses of the participants were farmers, followed by civil servants and then traders.

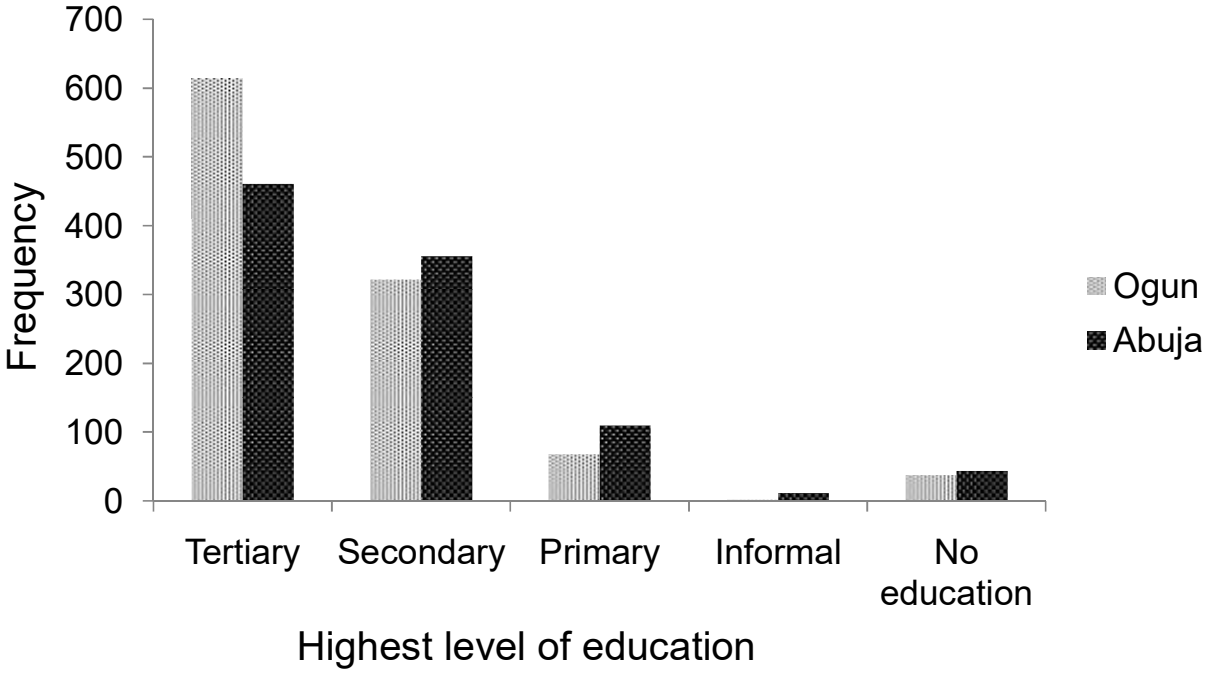

Figure 4.4: Frequency distribution of participants by highest level of education attained

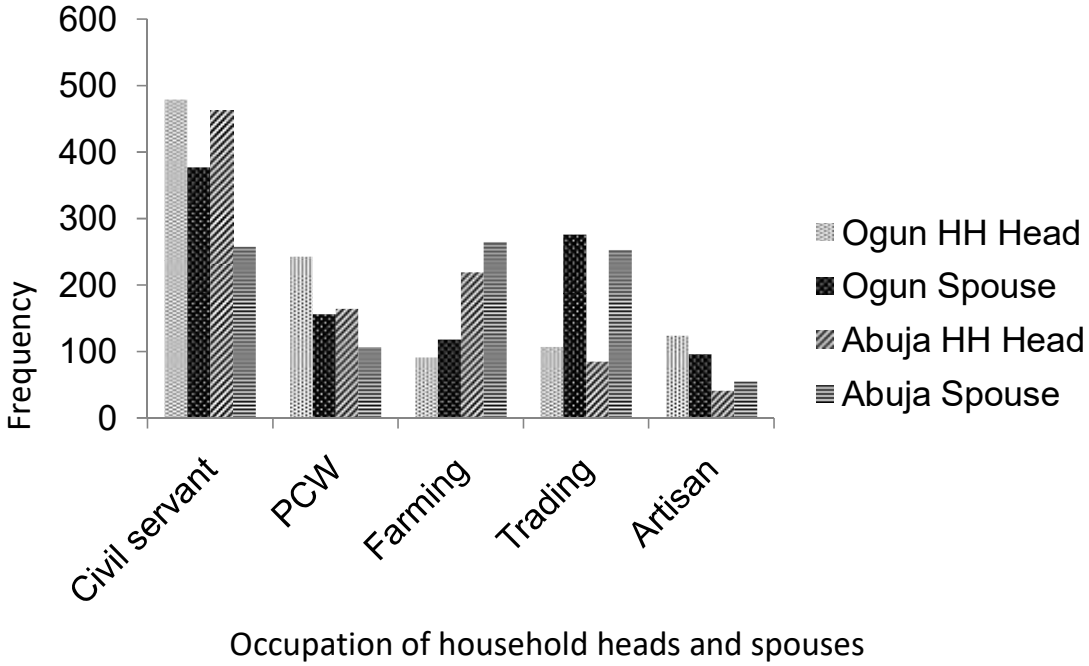

Figure 4.5: Frequency distribution of participants by occupation of household heads and spouses

PCW – private company workers

4.8 Socio-economic status of participants

The result of the Socio-Economic Status (SES) stratification is as presented in Figure 4.6. Many (47.5%) of the participants in Ogun State fell within the high socio-economic class whereas many (42.7%) of those from Abuja fell within the low socio-economic class. The trend of proportion of participants in the three classes was somewhat inversely between Ogun State and Abuja participants. In Ogun State the proportion of participants moves from majority being of high SES, followed by medium SES, then the low SES. Among Abuja participants, a reverse is the case; with majority being with low SES as previously highlighted.

A further disaggregation of the SES by location is shown in Figure 4.7. This shows that the major contributors to the high SES in Ogun state were participants from Ilaro, Abeokuta South and Ilisan, in decreasing order. As for participants in Abuja, the highest contributor to the low SES were participants from Kuje, followed by those in Kwali, and then those in Dutsen Alhaji. Coincidentally, participants from Dutsen Alhaji were the major contributors to the high SES of Abuja participants. In Ogun State, participants from Ipokia and those from Ijebu-Ode were the major contributors to the low SES of the location.

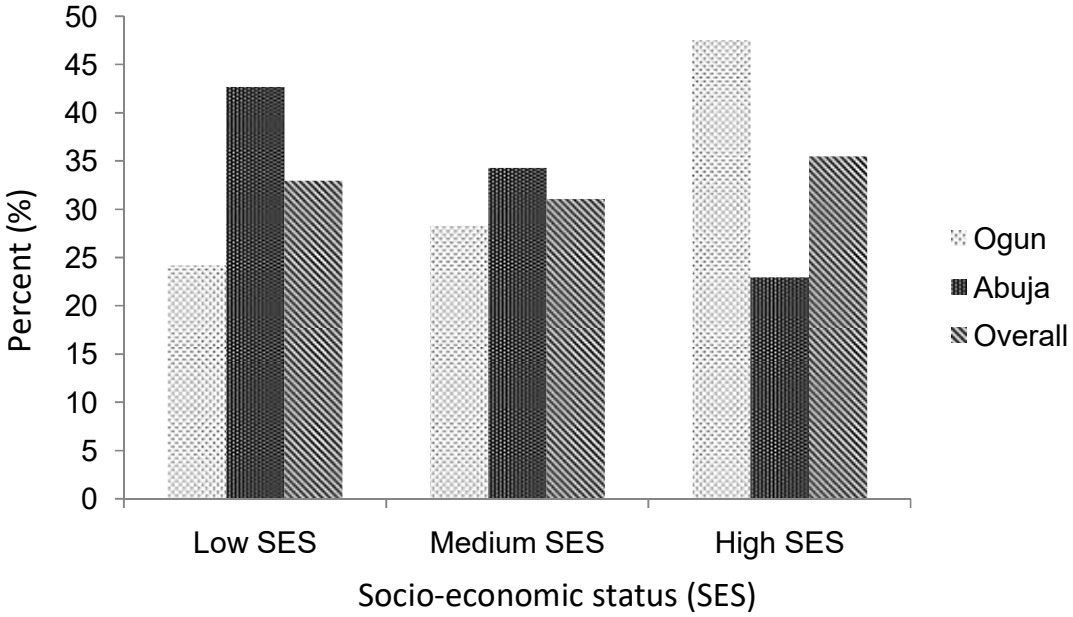

Figure 4.6: Percentage of participants with different socio-economic status by location

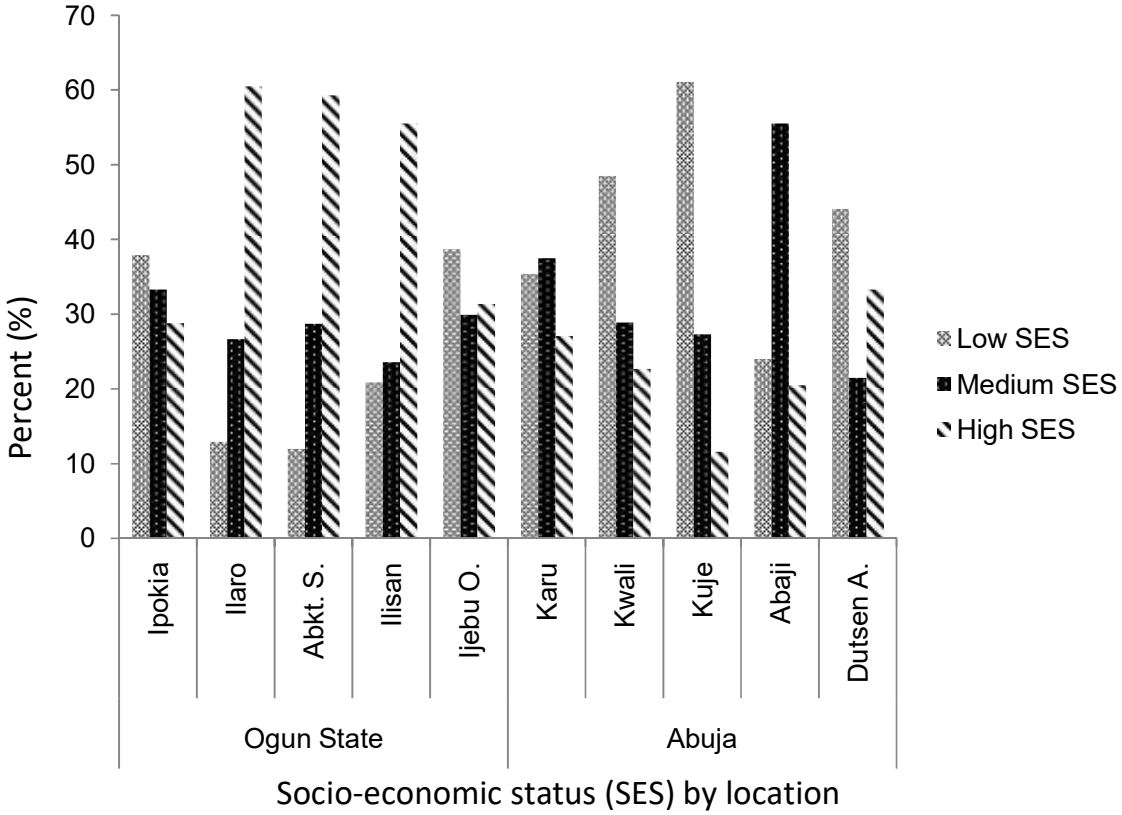

Figure 4.7: Percent distribution of participants with different socio-economic status (SES) by location further disaggregated

4.9 Age distribution and Body Mass Index (BMI) of participants

Further characteristics of the participants are shown in Table 4.4. A total of 2027 adults participated in the survey, with the majority coming from Ogun State. It can also be noticed that participants within 26-35 years were in the majority in Ogun State (41.1%) as well as Abuja (46.4%). Young adults in age bracket 18-25 years were the second leading group among the participants in both locations. Only 10 people older than 60 years participated in the whole survey. The same table also display the Body Mass Index (BMI) of the study participants. It is noteworthy to mention that the pregnant women within the study groups were excluded before the BMI classification was done. This dropped the total number to 1953. Most of the participants in Ogun State (49.2%) and in Abuja (47.8%) were within the normal range of BMI classification. Less than 3% of the adults in both locations were undernourished. Up to 14.8% from Ogun State and 12.5% from Abuja fell within various classes of obesity.

The BMI classification of the participants was further disaggregated based on gender and location (Table 4.5). Majority (71.9%) of the obese participants were women, with many of them being from Ogun State. The trend of those with overweight status was just the same as that of obese participants in the two locations. However, 32.8% of the total women were overweight, while 39.5% of the whole men had overweight status. Women in Ipokia, followed by those in Kwali were the ones with high frequency of undernutrition status. On the other hand, women in Ijebu-Ode, followed closely by those in Karu were those with participants mainly in the obese group. Slightly more than half (51.8%) of men in Ogun State was either overweight or obese compared with those in Abuja (48.8%).

Table 4.4: Age and Body Mass Index (BMI) of participants

Characteristics	Ogun	Abuja
Age (years)	**(n = 1044)**	**(n = 983)**
18-25	318 (30.5)	253 (25.7)
26-35	429 (41.1)	456 (46.4)
36-45	193 (18.5)	190 (19.3)
46-55	86 (8.2)	66 (6.7)
55-60	15 (1.4)	11 (1.1)
61-70	3 (0.3)	7 (0.7)
BMI (Kg/M^2)	**(n = 987)**	**(n = 966)**
Undernutrition (<18.5)	29 (2.9)	20 (2.1)
Normal (18.5-24.9)	486 (49.2)	462 (47.8)
Overweight (25.0-29.9)	326 (33.0)	363 (37.6)
Obese (>30.0)	146 (14.8)	121 (12.5)

Table 4.5: Disaggregated distribution of the Body Mass Index (BMI) of participants by gender and location

Location	Men BMI Status				Women BMI Status			
	Underwt	Normal	Overwt	Obese	Underwt	Normal	Overwt	Obese
OGUN	**4**	**140**	**112**	**43**	**25**	**346**	**214**	**103**
Ipokia	-	15	12	15	17	95	30	8
Ilaro	-	17	22	10	3	54	59	20
Abkt.-S.	-	37	12	-	1	82	49	26
Ilisan	4	40	44	6	2	56	38	16
Ijebu-O.	-	31	22	12	2	59	52	33
ABUJA	**7**	**200**	**166**	**32**	**13**	**262**	**197**	**89**
Karu	-	16	28	8	-	56	52	32
Kwali	4	38	22	2	8	81	31	6
Kuje	3	57	50	-	-	39	32	15
Abaji	-	77	27	16	-	23	40	18
Dutsen A.	-	12	39	6	5	63	42	18

Ogun: n = 299 (men), 688 (women); Abuja: n = 405 (male), 561(female).
Underwt – underweight, overwt – overweight, Abkt.-S. – Abeokuta-South, Ijebu-O. – Ijebu-Ode.

4.10 Dietary diversities of participants in Ogun State and Abuja

The dietary patterns of the study population are reported in Figures 4.8, 4.9 and 4.10 as well as Tables 4.6 and 4.7. The 9 aggregated food groups are as shown in Figure 4.8. From the Figure, the food groups mainly consumed by the participants were starchy staples; vitamin A rich fruits and vegetables; other fruits and vegetables; and meat and fish, in a decreasing order. Organ meat was the least consumed, followed by eggs, then legumes, nuts and seeds.

Figure 4.9 shows the percentage of the population in Ogun State and Abuja having a low Dietary Diversity Score (DDS) of less than 6 food groups being consumed on the day prior to the day of interview. More people had lower diversification of their diet in Ogun state than in Abuja as shown by the Figure. A further disaggregation of those with low DDS based on location is presented in Figure 4.10. Ipokia and Kuje were the two sub-locations with the least dietary diversification in Ogun State and Abuja, respectively. On the other hand, majority of the participants in Abeokuta-South (78.3%) and Dutsen Alhaji (80%) had high DDS.

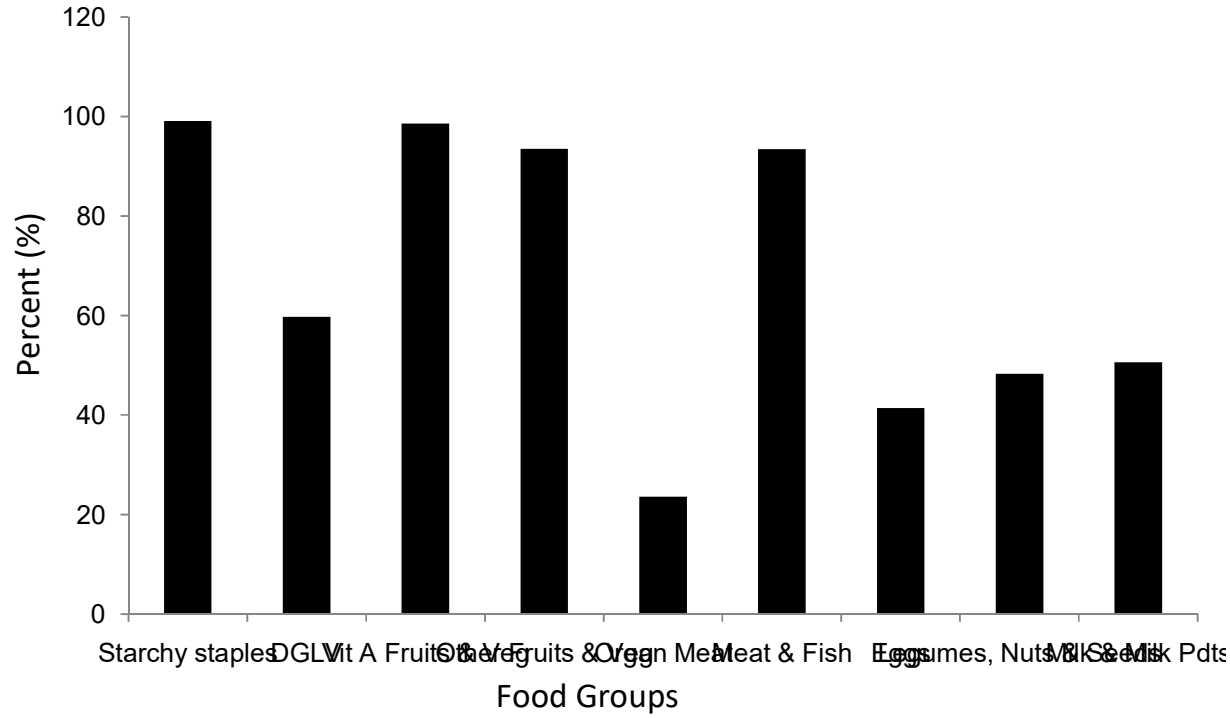

Figure 4.8: Percentage consumption of various aggregated food groups in the dietary diversity questionnaire

DGLV – dark green leafy vegetables

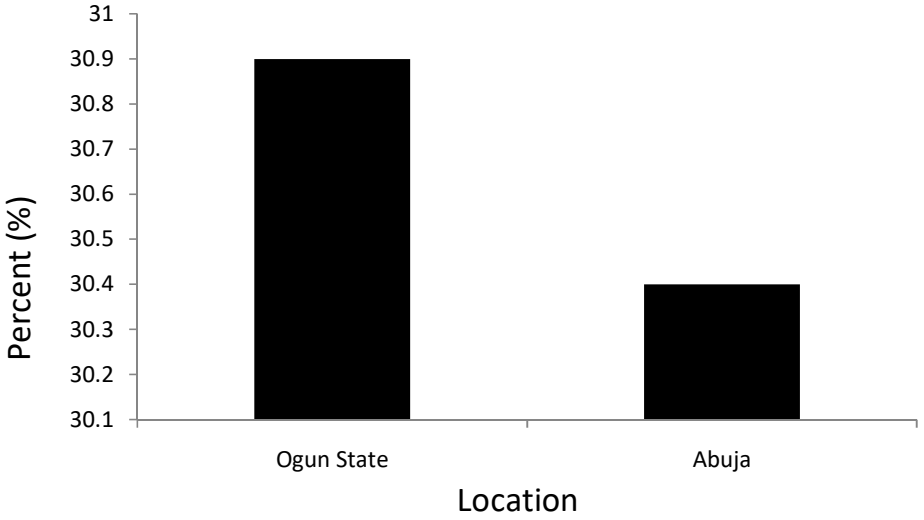

Figure 4.9: Percentage of the participants in Ogun State and Abuja having a low Dietary Diversity Score (DDS) of less than 6 food groups

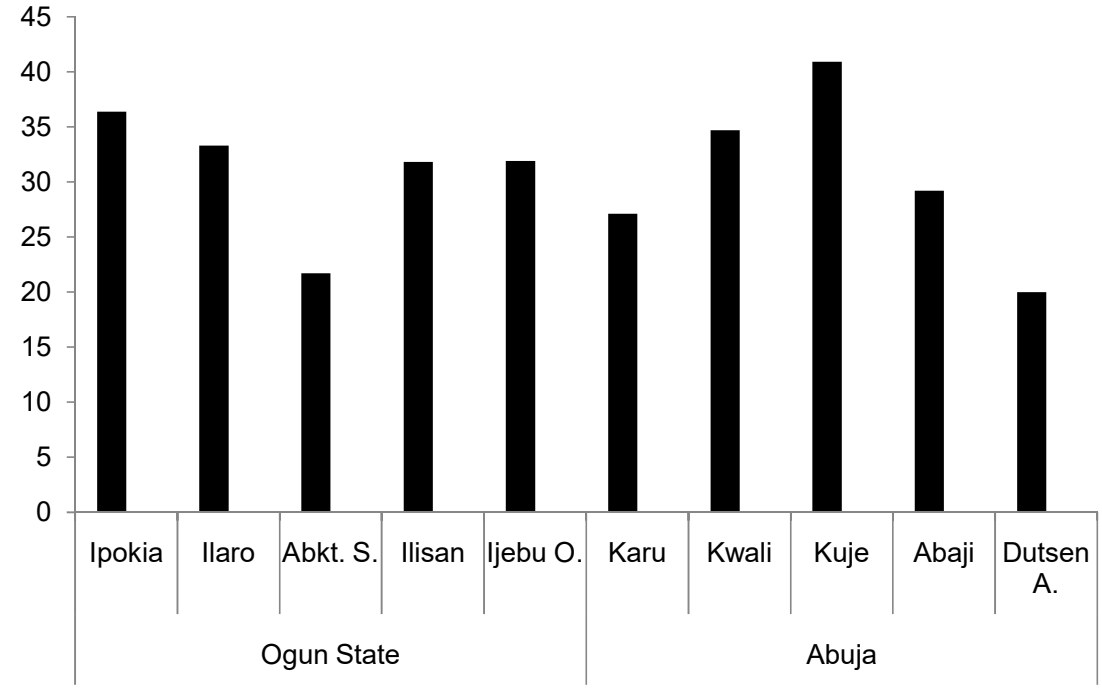

Figure 4.10: Percentage of the disaggregated participants in Ogun State and Abuja having low Dietary Diversity Score (DDS) of less than 6 food groups

4.11 Frequency of consumption of various foods by all participants

The frequency of consumption of various foods by Ogun State and Abuja participants (2027) is summarized in Table 4.6. Among the known staples in Nigeria, rice had the highest level of consumption with 49% of the study population consuming it daily or more than four times weekly basis, followed by maize and its products (with pap/akamu having 27.2%), then cassava and its products (with garri having 17.6%) and wheat and its products (with white bread having 15.1%, noodles having 8.9% and biscuit having 21.1%). As for yam and its products, barely 5% consumed them daily; rather most of the study group consumed them 1-2 times weekly with frequency ranging from 26-50.7%.

The major protein source to the respondents was fish (55.6%), followed by beef (37.8%), milk (30.1%), groundnut (20.6%) and then cowpea (12.5%) looking at their daily or more than four times weekly food consumption pattern. About 21% of the participants consumed each of tea and beverages daily or more than four times weekly. However, other drinks and snacks were consumed at less than 5% rate and several (22.6-63.1%) rarely or consumed them less than once monthly.

Orange (29.6%) was the most frequently consumed fruit on daily or more than four times weekly basis, by the respondents; followed by water melon (6.7%) and banana (5.7%). As regards the vegetable, ‘ewedu’ was the most frequently consumed (17.4%) on daily or more than four times weekly basis, followed by ‘ugu’ and ‘soko’ jointly having 27.5% and then ‘tete/aleiho’ having 12.3% level of consumption.

From the whole list of foods on the FFQ, tunwo dawa was specified as the most rarely consumed food (77.5%) by the study participants; followed closely by date (71.4%) and tofu (71.3%).

Table 4.6: Overall percentage (%) food consumption pattern for Ogun State and Abujaparticipants

Food Group	Food description	1-3 X daily, > 4 X weekly	3-4 X weekly	1-2 X weekly	1-3 X monthly	Rarely or < once monthly
Roots, Tubers and their products	Garri	17.6	14.8	35.0	13.1	19.5
	Fufu	2.7	6.2	23.4	14.2	53.6
	Yam, boiled	4.8	17.3	50.7	14.6	12.6
	Yam, fried	2.7	13.0	35.3	16.4	32.6
	Amala	5.1	16.7	29.1	16.5	32.6
	Yam, pounded	1.7	8.9	26.0	22.9	40.6
	Yam, pottage	2.5	7.6	33.1	21.3	35.5
	Sweet potato, fried	2.5	10.7	23.5	17.4	45.9
Cereals, Grains and their products	Ogi/Pap	27.2	14.6	29.3	10.9	18.0
	Eko/Agidi	3.9	12.5	22.3	12.8	48.5
	Tunwo Masara	4.6	10.3	17.5	6.3	61.4
	Maize	5.9	14.2	37.7	14.6	27.7
	Corn flakes, Golden Morn	3.7	11.1	22.8	14.1	48.3
	Tunwo dawa	3.3	6.4	9.8	3.1	77.5
	Rice	49.0	35.5	11.6	1.6	2.3
	Tunwo Shinkafa	0.7	5.2	17.8	6.8	69.6
	White bread	15.1	38.1	30.3	5.7	10.8
	Biscuit	21.1	25.3	28.0	7.3	18.5
	Semovita	4.9	16.7	46.8	9.2	22.4
	Noodles	8.9	20.0	35.1	27.0	9.0
Legume and legume products	Beans	12.5	19.9	55.8	6.7	5.1
	Moimoi	1.7	16.0	51.9	20.2	10.2
	Akara	6.8	14.0	41.9	13.0	24.3
	Tofu	2.8	7.1	11.9	7.0	71.3
Meat, fish, Poultry	Beef	37.8	21.3	28.2	6.6	6.1
	Poultry	5.1	17.4	41.6	25.8	10.2
	Fish	55.6	28.8	12.0	1.7	2.0
	Egg	9.5	27.1	49.2	8.5	5.6

Table 4.6: Overall percentage (%) food consumption pattern for Ogun State and Abuja participants (continued)

Food Group	Food description	1-3 X daily, > 4 X weekly	3-4 X weekly	1-2 X weekly	1-3 X monthly	Rarely or < once monthly
Milk	Milk, powdered	30.1	16.8	36.0	5.8	11.3
	Milk, evaporated	8.5	16.9	26.3	16.4	31.8
Tea, beverages and other drinks	Tea	20.7	12.0	26.8	8.1	32.3
	Beverages	21.3	25.5	30.1	8.9	14.2
	Viju and Nutri milk	2.2	5.3	26.8	18.0	47.7
	Yoghurt	3.6	6.5	28.5	22.2	39.2
	LaCasera	0.9	6.1	22.5	23.9	46.6
	Pepsi, Mirinda, 7up, Coke, Fanta	4.1	13.1	29.2	20.8	32.9
	Fayruz, Mountain dew	1.5	6.1	20.2	21.9	50.4
	Malts	1.8	13.2	38.1	24.4	22.6
	Zobo	1.4	13.4	12.5	9.6	63.1
Fruits	Orange	29.6	23.8	34.6	5.4	6.6
	Mango	2.4	9.0	19.1	19.5	50.0
	Pawpaw	3.3	9.8	33.9	21.3	31.7
	Guava	1.2	3.8	16.4	18.6	59.9
	Pineapple	3.4	13.6	40.0	24.1	18.9
	Water melon	6.7	27.2	32.7	16.4	17.1
	Carrot	2.2	9.4	35.0	25.7	27.7
	Banana	5.7	25.2	41.7	14.2	13.2
	Plantain	4.1	33.5	38.5	13.6	10.3
	Apple	4.8	6.5	38.9	37.3	12.5
	Date	5.7	7.4	10.4	5.1	71.4
Oil seed nuts	Groundnut, boiled/roasted	20.6	12.3	38.8	10.5	17.8
	Cashew nut	1.2	3.4	15.3	17.4	62.8
	Coconut	1.7	4.4	26.9	24.5	42.5
Vegetables	Ugu and Soko leaves	27.5	19.7	39.7	6.6	6.6
	Okra	7.0	19.5	26.5	13.1	33.9
	Waterleaf	5.8	16.9	38.8	9.9	28.6
	Tete leaf	12.3	19.5	37.6	11.2	19.3
	Ewedu	17.4	22.5	25.7	8.4	26.0
	Ogbonna	1.8	8.9	23.3	15.4	50.6

Table 4.6: Overall percentage (%) food consumption pattern for Ogun State and Abuja participants (continued)

Food Group	Food description	1-3 X daily, > 4 X weekly	3-4 X weekly	1-2 X weekly	1-3 X monthly	Rarely or < once monthly
Snacks	Cookies	3.1	4.9	16.7	15.9	59.4
	Doughnut	1.8	9.3	21.8	16.9	50.2
	Chips	2.6	9.1	32.5	19.0	36.8
	Pufpuf/ Buns	3.2	15.9	33.3	15.2	32.5
	Meat pie	0.5	9.3	31.7	26.2	32.3
	Cake	1.0	6.3	24.3	31.8	36.7
	Fish roll	2.3	7.3	24.9	21.7	43.9

4.12 Disaggregated frequency of consumption of various foods by participants in Ogun State and Abuja

The FFQ results were further disaggregated on Table 4.7 for some additional clarifications, especially as to which foods are more frequently consumed in each main location of study. Among the staples, this table revealed that rice, maize products (like pap/akamu), garri and wheat products (like biscuits and noodles) are much more consumed by study population in Ogun State; while other maize products (like eko/agidi, tunwo masara, boiled/roasted maize, and corn flakes/golden morn), wheat products (like white bread and semovita) and yam and its products were better consumed by participants in Abuja on daily or more than four times weekly basis. Among the protein sources, participants in Abuja consumed more fish, beef, poultry, akara, moimoi, evaporated milk and groundnut than those in Ogun State on daily or more than four times weekly basis. Those in Ogun State consumed more of cowpea, tofu, egg and powdered milk than their Abuja counterpart.

Participants in Abuja consumed more tea and beverages on daily or more than four times weekly basis than those in Ogun State. Most of the drinks and snacks were rarely consumed at a higher percentage by participants in Abuja compared with those in Ogun State. They also consumed more fruits (orange, water melon, carrot, banana, date, gwava, pawpaw and mango) and vegetables (ugu and soko, okra, tete/aleiho, and ogbonna) on daily or more than four times weekly basis compared with participants in Ogun State. Overall, tunwo masara, tunwo dawa, tunwo shinkafa and date were somewhat regionally consumed in Abuja.

Table 4.7: Disaggregated percentage (%) food consumption pattern by Ogun State and Abuja participants

Food Group	Food description	Loca-tion	1-3 X daily, > 4 X weekly	3-4 X weekly	1-2 X weekly	1-3 X monthly	Rarely or < once monthly
Roots, Tubers and their products	Garri	Ogun	23.4	16.3	34.4	10.6	15.3
		Abuja	11.5	13.1	35.7	15.8	23.9
	Fufu	Ogun	1.4	9.6	23.4	16.1	49.5
		Abuja	4.0	2.5	23.4	12.1	58.0
	Yam, boiled	Ogun	2.6	22.8	50.2	14.6	9.9
		Abuja	7.2	11.5	51.2	14.5	15.6
	Yam, fried	Ogun	0.6	11.6	32.8	16.0	39.1
		Abuja	4.9	14.5	38.0	16.9	25.6
	Amala	Ogun	4.4	22.1	31.7	13.3	28.4
		Abuja	5.9	11.0	26.3	19.8	36.9
	Yam, pounded	Ogun	0.3	6.8	17.5	24.9	50.5
		Abuja	3.2	11.1	35.0	20.8	30.0
	Yam, pottage	Ogun	1.0	6.8	27.4	23.4	41.5
		Abuja	4.2	8.5	39.1	19.0	29.2
	Sweet potato, fried	Ogun	1.1	10.9	15.3	18.5	54.1
		Abuja	3.9	10.5	32.2	16.2	37.2
Cereals, Grains and their products	Ogi/Pap	Ogun	21.4	14.8	29.8	13.4	20.6
		Abuja	15.2	8.2	28.8	14.3	33.5
	Eko/Agidi	Ogun	3.8	13.6	24.2	14.8	43.5
		Abuja	4.0	11.3	20.3	10.6	53.8
	Tunwo Masara	Ogun	-	5.5	11.4	6.7	76.4
		Abuja	9.5	15.4	23.9	5.8	45.5
	Maize	Ogun	1.8	10.6	33.2	18.2	36.1
		Abuja	10.2	17.9	42.4	10.7	18.8
	Corn flakes, Golden Morn	Ogun	3.4	15.7	26.1	13.7	41.1
		Abuja	4.0	6.3	19.2	14.4	56.1
	Tunwo dawa	Ogun	0.5	0.5	1.5	1.6	95.9
		Abuja	6.3	12.7	18.5	4.6	57.9
	Rice	Ogun	51.7	38.3	6.7	1.6	1.6
		Abuja	46.1	32.6	16.9	1.5	3.0

Table 4.7: Disaggregated percentage (%) food consumption pattern by Ogun State and Abuja participants(continued)

Food Group	Food description	Loca-tion	1-3 X daily, > 4 X weekly	3-4 X weekly	1-2 X weekly	1-3 X monthly	Rarely or < once monthly
	Tunwo Shinkafa	Ogun	0.3	0.8	0.5	1.6	96.8
		Abuja	1.1	10.0	36.1	12.2	40.6
	White bread	Ogun	13.9	40.5	31.5	6.8	7.3
		Abuja	16.4	35.6	29.0	4.5	14.5
	Biscuit	Ogun	25.8	27.4	21.3	7.1	18.5
		Abuja	16.1	23.0	35.1	7.4	18.4
	Semovita	Ogun	1.8	18.2	45.6	9.4	25.0
		Abuja	8.2	15.1	48.0	9.1	19.6
	Noodles	Ogun	9.9	21.6	38.6	20.8	9.1
		Abuja	7.8	18.2	31.4	33.6	9.0
Legume and legume products	Beans	Ogun	15.5	24.7	46.8	7.3	5.7
		Abuja	9.3	14.9	65.3	6.0	4.6
	Moimoi	Ogun	0.9	16.5	49.6	20.1	12.9
		Abuja	2.5	15.6	54.3	20.3	7.2
	Akara	Ogun	2.6	11.3	39.8	14.9	31.4
		Abuja	11.3	16.8	44.3	10.9	16.8
	Tofu	Ogun	3.4	3.1	7.5	5.4	80.7
		Abuja	2.1	11.3	16.7	8.6	61.2
Meat, fish, Poultry	Beef	Ogun	33.6	24.3	24.9	8.9	8.3
		Abuja	42.2	18.1	31.8	4.1	3.8
	Poultry	Ogun	4.0	20.7	47.7	19.0	8.6
		Abuja	6.3	13.8	35.1	33.0	11.8
	Fish	Ogun	54.1	29.7	10.8	2.0	3.4
		Abuja	57.1	27.8	13.1	1.3	0.6
	Egg	Ogun	11.4	36.0	43.7	5.6	3.4
		Abuja	7.5	17.7	55.1	11.7	7.9
Milk	Milk, powdered	Ogun	30.4	18.3	35.0	4.7	11.7
		Abuja	29.9	15.2	37.0	7.0	10.9
	Milk, evaporated	Ogun	8.0	15.4	32.6	14.3	29.7
		Abuja	9.1	18.4	19.7	18.7	34.1
Tea, beverages and other drinks	Tea	Ogun	16.0	12.1	26.4	8.7	36.8
		Abuja	25.7	12.0	27.2	7.5	27.6

Table 4.7: Disaggregated percentage (%) food consumption pattern by Ogun State and Abuja participants (continued)

Food Group	Food description	Loca-tion	1-3 X daily, > 4 X weekly	3-4 X weekly	1-2 X weekly	1-3 X monthly	Rarely or < once monthly
Tea, beverages and other drinks (contd.)	Beverages	Ogun	20.2	26.7	31.9	8.4	12.7
		Abuja	22.5	24.1	28.2	9.5	15.8
	Viju and Nutri milk	Ogun	3.0	6.7	28.4	19.0	43.0
		Abuja	1.4	3.8	25.2	16.9	52.7
	Yoghurt	Ogun	4.6	8.6	29.1	21.0	36.7
		Abuja	2.5	4.2	27.8	23.6	41.9
	LaCasera	Ogun	0.5	9.5	23.5	21.9	44.6
		Abuja	1.3	2.4	21.5	26.0	48.7
	Pepsi, Mirinda, 7up, Coke, Fanta	Ogun	4.6	15.7	28.4	20.4	30.8
		Abuja	3.6	10.3	29.9	21.3	35.0
	Fayruz, Mountain dew	Ogun	0.9	8.8	21.1	19.4	49.8
		Abuja	2.1	3.3	19.2	24.4	51.0
	Malts	Ogun	1.3	16.5	38.2	19.9	24.0
		Abuja	2.2	9.7	37.9	29.1	21.0
	Zobo	Ogun	1.0	7.7	8.0	7.8	75.7
		Abuja	1.8	19.5	17.4	11.5	49.7
Fruits	Orange	Ogun	28.0	25.4	32.8	7.4	6.5
		Abuja	31.3	22.1	36.6	3.4	6.6
	Mango	Ogun	1.1	7.9	14.1	19.9	57.1
		Abuja	3.9	10.2	24.4	19.0	42.5
	Pawpaw	Ogun	2.3	12.8	32.8	20.2	31.9
		Abuja	4.4	6.5	35.2	22.5	31.4
	Guava	Ogun	0.5	2.9	12.8	14.7	69.2
		Abuja	1.9	4.9	20.1	22.9	50.2
	Pineapple	Ogun	2.7	13.9	37.3	22.2	23.9
		Abuja	4.1	13.3	42.9	26.0	13.6
	Water melon	Ogun	3.2	26.7	27.8	20.7	21.6
		Abuja	10.5	27.7	37.8	11.8	12.2
	Carrot	Ogun	0.8	8.9	31.0	27.9	31.4
		Abuja	3.7	10.0	39.3	23.3	23.8
	Banana	Ogun	4.8	27.2	43.6	10.5	13.9
		Abuja	6.7	23.1	39.7	18.1	12.4

Table 4.7: Disaggregated percentage (%) food consumption pattern by Ogun State and Abuja participants (continued)

Food Group	Food description	Loca-tion	1-3 X daily, > 4 X weekly	3-4 X weekly	1-2 X weekly	1-3 X monthly	Rarely or < once monthly
Fruits (contd.)	Plantain	Ogun	4.5	39.6	38.5	9.8	7.7
		Abuja	3.7	27.0	38.5	17.8	13.1
	Apple	Ogun	6.2	8.0	42.5	32.0	11.3
		Abuja	1.5	3.1	30.8	49.2	15.4
	Date	Ogun	-	1.9	1.7	2.0	94.3
		Abuja	11.7	13.3	19.5	8.3	47.1
Oil seed nuts	Groundnut, boiled/roasted	Ogun	11.9	12.6	39.4	12.5	23.7
		Abuja	29.9	12.0	38.1	8.4	11.5
	Cashew nut	Ogun	0.9	3.0	8.7	20.0	67.3
		Abuja	1.5	3.8	22.3	14.4	58.0
	Coconut	Ogun	-	5.3	21.7	24.4	48.6
		Abuja	3.6	3.6	32.3	24.5	36.0
Vegetables	Ugu and Soko leaves	Ogun	25.7	20.8	37.5	8.7	7.3
		Abuja	29.4	18.5	41.9	4.3	5.9
	Okra	Ogun	1.7	13.2	20.4	15.9	48.8
		Abuja	12.5	26.2	33.0	10.2	18.1
	Tete leaf	Ogun	9.0	18.2	38.2	10.5	24.1
		Abuja	12.3	18.5	38.6	12.2	18.3
	Ewedu	Ogun	22.1	26.4	28.8	6.1	16.5
		Abuja	12.3	18.3	22.4	10.8	36.1
	Ogbono	Ogun	0.6	9.5	14.8	16.1	59.1
		Abuja	3.1	8.3	32.5	14.6	41.5
Snacks	Cookies	Ogun	2.9	5.7	20.2	14.0	57.3
		Abuja	3.3	4.2	12.9	18.0	61.6
	Doughnut	Ogun	1.7	10.0	23.2	15.0	50.1
		Abuja	1.8	8.6	20.3	18.8	50.4
	Chips	Ogun	2.9	9.3	33.1	18.7	36.0
		Abuja	2.3	9.0	31.7	19.4	37.5
	Pufpuf/ Buns	Ogun	2.8	15.8	32.5	15.0	33.9
		Abuja	3.7	16.0	34.1	15.4	30.9
	Meat pie	Ogun	0.4	13.3	35.6	25.9	24.8
		Abuja	0.6	5.1	27.5	26.6	40.3
	Cake	Ogun	0.6	6.3	26.7	35.3	31.0
		Abuja	1.4	6.2	21.8	28.0	42.6
	Fish roll	Ogun	2.6	8.2	22.6	21.3	45.3
		Abuja	1.9	6.3	27.3	22.2	42.3

4.13 Mean daily mineral and heavy metal intakes of study participants

The mean daily metal intakes ofall the study participants based on the foods analysed are as shown in Table 4.8. This result shows that participants' daily mineral intakes (mg/person/day) were 1968.6±783.9, 2747.4±1065.2, 334.3±169.0, 389.3±131.5, 2.8±1.0, 3.7±1.7, 23.4±8.2 and 8.7±3.4 for potassium, sodium, calcium, magnesium, copper, manganese, iron and zinc while cadmium and lead were 5.3±4.2 and 64.6±46.8µg/person/day, respectively. The recommended daily intakes as well as the tolerable Upper intake Levels (ULs)/ Provisional Tolerable Daily Intakes (PTDIs) are also highlighted on the table. Relative to the recommended daily intakes, the mean values of potassium and calcium are far lower than the recommendations, while those of magnesium and zinc are within range. The mean daily intakes of manganese and iron are slightly higher than the recommendations, whereas those of sodium and copper are about double and triple of the recommendations respectively. The daily intakes of all the metals are lower than the ULs/PTDIs, except for that of sodium that exceeded the stipulated value of 2300 mg/person/day.

Table 4.8:Mean daily mineral and heavy metal intakes of participants

Metal	Mean ± SD (Min, Max)	Recommended Intake	UL/ PTDI
MINERAL	(mg/person/day)		
Potassium	1968.6±783.9 (366.3, 8369.0)	4700.0[a]	ND
Sodium	2747.4±1065.2 (34.4, 9309.9)	1500.0[a]	2300.0[a]
Calcium	334.3±169.0 (84.6, 1161.8)	1000.0-1300.0[b]	2500.0[b]
Magnesium	389.3±131.5 (125.8, 1081.2)	310.0-420.0[b]	ND
Copper	2.8±1.0 (0.86, 7.16)	0.9[c]	10.0[c]
Manganese	3.7±1.7 (0.7, 10.7)	1.8-2.3[c]	11.0[c]
Iron	23.4±8.2 (5.2, 60.0)	8.0-18.0[c]	45.0[c]
Zinc	8.7±3.4 (1.49, 31.1)	8.0-11.0[c]	40.0[c]
HEAVY METAL	(μg/person/day)		
Cadmium	5.3±4.2 (0.0, 23.1)	-	60.0[d]
Lead	64.6±46.8 (0.0, 247.6)	-	214.0[e]

[a] – IOM (2005); [b]–IOM (1997); [c] –IOM (2001); [d] – JECFA (2003), for 60kg adult human; [e] – JECFA (1999), for 60kg adult human; UL – Tolerable Upper Intake Level; PTDI – Provisional Tolerable Daily Intake; ND – no data.

4.14 Disaggregated mean daily mineral and heavy metal intakes of participants in Ogun State and Abuja

The daily metal intakes of the studied population are reported in Table 4.9, disaggregated into the two main locations – Ogun and Abuja. This disaggregation shows clearly that the patterns of mineral intakes at the two main locations are somewhat different, though the patterns of heavy metal intakes are similar. The mean daily intakes of sodium, copper, manganese and iron by participants in Ogun State are significantly higher ($p<0.05$) than those of participants in Abuja. A reverse is the case for the mean calcium intakes at the two main locations. A comparison of this disaggregated result with the recommended daily intakes and ULs/PTDIs shows similar patterns to what was previously highlighted in the data that was not disaggregated in Table 4.8.

Table 4.9: Mean daily mineral and heavy metal intakes of participants by locations

Metal	Ogun (n = 386)	Abuja (n = 349)	Recommended Intake	UL/ PTDI
MINERAL		(mg/person/day)		
Potassium	2014.1±780.2 (366.3, 5043.0)	1977.5 ±709.3 (779.3, 8369.0)	4700.0[a]	ND
Sodium	2976.5±1102.4 (34.4, 9309.9)*	2498.8±937.1 (652.5, 7111.7)	1500.0[a]	2300.0[a]
Calcium	306.1±156.7 (84.6, 1113.3)	365.5±176.2 (85.1, 1161.8)*	1000.0-1300.0[b]	2500.0[b]
Magnesium	400.5±134.9 (125.8, 897.6)	386.5±122.5 (132.7, 1081.2)	310.0-420.0[b]	ND
Copper	2.9±1.0 (0.9, 6.6)*	2.6±0.8 (0.9, 7.2)	0.9[c]	10.0[c]
Manganese	4.0±1.8 (0.8, 10.7)*	3.4±1.5 (0.7, 0.7)	1.8-2.3[c]	11.0[c]
Iron	25.4±8.5 (5.2, 59.9)*	21.3±7.2 (5.7, 60.0)	8.0-18.0[c]	45.0[c]
Zinc	8.9±3.4 (1.5, 24.3)	8.5±3.3 (3.0, 31.1)	8.0-11.0[c]	40.0[c]
HEAVY METAL	(µg/person/day)			
Cadmium	5.5±4.5 (0.0, 21.2)	5.6±4.2 (0.0, 23.1)	-	60.0[d]
Lead	64.0±48.2 (0.0, 226.0)	69.9±46.0 (0.0, 247.6)	-	214.0[e]

Values represent mean±SD (min, max); * - Mean value is significantly higher (at $p<0.05$) than that obtained from the other location; ND – no data.
[a] – IOM (2005); [b] –IOM (1997); [c] –IOM (2001); [d] – JECFA (2003), for 60kg adult human; [e] – JECFA (1999), for 60kg adult human; UL – Tolerable Upper Intake Level; PTDI – Provisional Tolerable Daily Intake

4.15 Disaggregated mean daily mineral and heavy metal intakes of participants in Ogun State and Abuja compared with recommendations

Considering the recommended daily intakes and ULs/PTDIs, some dynamics can be further obtained from Table 4.10 as related to the mean levels of minerals and heavy metals habitually consumed by the participants. The table shows that all participants from both main locations consumed below the recommended amount of potassium, whereas all consumed copper within the recommended level. Majority of study group, especially from Ogun State consumed above the safe level of sodium. Most of the participants consumed less than the required level of calcium through the foods analysed. Conversely, most of them had manganese and iron intakes falling within the recommended range at both locations. Virtually all the participants consumed far less than the toxic level of heavy metals.

Table 4.10: Mean daily mineral and heavy metal intakes of participants by locations compared with recommendations

Metal	Ogun (n = 386)	Abuja (n = 349)	Recommended Intake	UL/ PTDI
MINERAL	(mg/person/day)			
Potassium	2014.1±780.2 (100.0, 0.0, 0.0)	1977.5 ±709.3 (100.0, 0.0, 0.0)	4700.0[a]	ND
Sodium	2976.5±1102.4 (4.1, 24.1, 71.8)	2498.8±937.1 (7.2, 37.0, 55.9)	1500.0[a]	2300.0[a]
Calcium	306.1±156.7 (99.5, 0.5, 0.0)	365.5±176.2 (98.9, 1.1, 0.0)	1000.0-1300.0[b]	2500.0[b]
Magnesium	400.5±134.9 (27.2, 33.4, 39.4)	386.5±122.5 (29.3, 35.6, 35.1)	310.0-420.0[b]	ND
Copper	2.9±1.0 (0.0, 100.0, 0.0)	2.6±0.8 (0.0, 100.0, 0.0)	0.9[c]	10.0[c]
Manganese	4.0±1.8 (7.5, 92.5, 0.0)	3.4±1.5 (14.3, 85.7, 0.0)	1.8-2.3[c]	11.0[c]
Iron	25.4±8.5 (0.3, 97.4, 2.3)	21.3±7.2 (0.6, 99.1, 0.3)	8.0-18.0[c]	45.0[c]
Zinc	8.9±3.4 (42.0, 58.0, 0.0)	8.5±3.3 (54.2, 45.8, 0.0)	8.0-11.0[c]	40.0[c]
HEAVY METAL	(µg/person/day)			
Cadmium	5.5±4.5 (100.0, 0.0)[f]	5.6±4.2 (100.0, 0.0)[f]	-	60.0[d]
Lead	64.0±48.2 (99.5, 0.5)[f]	69.9±46.0 (99.7, 0.3)[f]	-	214.0[e]

Values represent mean±SD (% consuming below recommendation, % consuming within recommendation and safe level, % consuming above safe level); [*] - Mean value is significantly higher (at $p<0.05$) than that obtained from the other location; ND – no data.

[a] –IOM (2005); [b] –IOM (1997); [c] –IOM (2001); [d] – JECFA (2003), for 60kg adult human; [e] – JECFA (1999), for 60kg adult human; [f] – Values represent mean±SD (% consuming within safe level, % consuming above safe level);

UL – Tolerable Upper Intake Level; PTDI – Provisional Tolerable Daily Intake

4.16 Disaggregated mean daily mineral and heavy metal intakes of participants by location and gender

Table 4.11 presents the mean daily intakes of the minerals and heavy metals disaggregated by location and gender. The male and female participants in Ogun State had similar mineral and heavy metal intakes, with only calcium being significantly higher ($p<0.05$) in the male category. In the case of participants in Abuja, the mean cadmium and lead intakes of the males were the only metals significantly higher ($p<0.05$) than those of the female group.

Table 4.11: Mean daily mineral and heavy metal intakes of participants by locationsand gender

Metal	Ogun (n = 386)		Abuja (n = 349)	
	Male (n=97)	Female (n=289)	Male (n=141)	Female (n=208)
MINERAL	(mg/person/day)			
Potassium	2118.1±802.4	1979.2 ±772.2	1963.4±565.4	1987.0 ±794.9
Sodium	3011.6±1339.5	2964.8±1014.9	2454.7±828.6	2528.7±1007.0
Calcium	337.2±164.1*	295.7±153.2	384.2±179.5	352.8±173.7
Magnesium	413.2±131.8	396.2±136.1	390.1±114.9	383.9±127.9
Copper	3.0±1.0	2.9±1.0	2.7±0.7	2.5±0.9
Manganese	4.3±2.0	3.9±1.8	3.4±1.5	3.3±1.5
Iron	25.9±9.1	25.3±8.3	21.5±6.2	21.1±7.8
Zinc	9.3±3.4	8.8±3.5	8.6±2.7	8.3±3.6
HEAVY METAL	(µg/person/day)			
Cadmium	5.1±4.8	5.6±4.4	6.6±4.5*	4.8±3.8
Lead	61.3±51.5	64.8±47.2	83.5±47.5*	60.7±42.8

Values represent mean±SD; * - Mean value is significantly higher (at $p<0.05$) than the value obtained from the other gender in the same location

4.17 Moisture content and mean concentrations of macrominerals in some Nigerian foods analysed 'as consumed'

Table 4.12 shows the moisture content and upper bound mean concentrations of macrominerals in some Nigerian foods 'as consumed'. The mean levels of potassium (K) ranged from 2.92 mg/100g in custard to 1520 mg/100g in stock fish. The levels in the foods decreased from 1520 mg/100g in stock fish to 1510 mg/100g in 'ebolo' fish and then to 1440 mg/100g in powdered full-cream milk. The fish group contained most samples with very high levels of K. Fried plantain chips (969 mg/100g), cornflakes (821 mg/100g), akara of drum bean (mg/100g), date (607 mg/100g), green peas (390 mg/100g), water leaf soup (413 mg/100g), fried beef in stew (309 mg/100g), boiled walnut (867 mg/100g) and cocoa product (825 mg/100g) had the highest levels of K in the tubers, starches and their products; cereals and cereal product; legumes and legume products; fruits; leafy and fruity vegetables; condiments, sauces and soups; beef, poultry and eggs; oil seeds; and sugar and cocoa product groups, respectively.

The mean sodium (Na) level was highest in salt (30,700 mg/100g) and not detected in cornflakes. All the fruits, leafy and fruity vegetables (except cole slaw), and most cereals and tubers normally prepared without addition of salt had relatively low mean levels of Na. Most items within the 'legumes and legume products', 'condiment, sauces and soups' and fish groups had high mean levels of Na.

The mean levels of calcium (Ca) ranged from 0.65 mg/100g in plum to 770 mg/100g in powdered full-cream milk. Other rich sources of Ca include powdered low-fat milk (740 mg/100g), stockfish (330 mg/100g), local cheese (293 mg/100g), cray fish (171 mg/100g), 'donkwa' (111 mg/100g), fried kote (80.1 mg/100g), and locust bean (69.8 mg/100g).

Magnesium (Mg) mean concentration was 1.16 mg/100g in refined sugar and highest in cashew nut (426 mg/100g). Other rich sources of Mg are: roasted cray fish (416 mg/100g), roasted groundnut (272 mg/100g), boiled walnut (248 mg/100g), stock fish (229 mg/100g), and roasted 'palamu' fish (208 mg/100g).

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Tubers, starches and their products	Fufu (cooked)	11	67.8	53.4 (25.4-92.8)	5.02 (1.87-8.59)	20.6 (10.8-40.3)	19.5 (4.63-35.7)
	Sweet potato (fried)	4	53.0	404 (381-426)	162 (137-214)	30.9 (22.1-44.1)	28.9 (17.0-51.3)
	Amala (yam powder, cooked)	10	76.3	191 (121-254)	15.1 (7.15-28.5)	14.9 (7.45-32.7)	26.3 (10.3-41.4)
	Pounded yam	6	70.3	186 (142-269)	9.02 (2.84-15.8)	10.4 (4.11-18.0)	27.5 (10.4-45.1)
	Yam (white, fried)	8	54.5	313 (249-389)	214 (125-382)	20.6 (12.8-31.7)	38.7 (12.7-79.1)
	Yam (white, boiled)	2	64.2	161 (161-162)	336 (335-336)	27.0 (18.7-35.2)	72.2 (71.1-73.3)
	Poundo yam (cooked)	2	73.9	15.6 (14.8-16.4)	12.0 (11.7-12.4)	4.20 (4.05-4.35)	5.50 (4.00-7.00)
	Yam pottage (white yam)	2	68.8	247 (226-271)	236 (215-252)	5.90 (5.63-6.41)	14.8 (14.1-16.0)
	Cake (water yam, fried)	2	63.3	554 (542-566)	353 (345-360)	5.03 (4.98-5.08)	21.6 (20.8-22.4)
	Eba (white gari, cooked)	9	72.6	96.3 (35.7-181)	11.8 (4.92-18.9)	22.5 (12.9-33.5)	27.6 (10.9-43.8)
	Bread fruit (boiled)	2	79.5	221 (219-223)	12.2 (12.0-12.4)	18.1 (13.44-24.7)	44.3 (42.2-46.4)
	Plantain (mature ripe, fried)	6	43.3	448 (350-678)	12.8 (3.85-24.1)	4.51 (3.86-5.79)	58.1 (38.3-71.6)
	Plantain chip (mature unripe, fried)	4	1.95	969 (931-1050)	194 (105-280)	5.91 (5.18-6.62)	91.6 (84.0-98.3)

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'(continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Cereals and cereal products	Rice (Long grain, boiled)	14	71.0	34.3 (18.7-51.4)	205 (100-355)	8.83 (3.59-15.3)	13.5 (2.40-36.3)
	Rice (Short grain, boiled)	3	71.7	11.3 (10.9-11.7)	333 (328-337)	11.5 (10.7-12.2)	19.9 (19.7-20.1)
	Local Rice (boiled)	4	71.9	49.6 (16.7-93.4)	158 (117-236)	10.5 (8.89-13.5)	36.2 (11.8-48.1)
	Jollof Rice	5	65.8	98.3 (74.0-130)	362 (326-409)	15.3 (11.2-18.9)	24.6 (16.3-31.0)
	Fried Rice	3	62.5	93.2 (81.1-106)	334 (257-469)	5.45 (5.18-6.03)	7.37 (6.96-8.22)
	Masa (rice cake, fried)	3	56.4	27.2 (16.0-48.9)	163 (146-197)	29.8 (16.0-37.2)	26.5 (12.4-51.8)
	Garogaro (rice, beans, boiled; along with fresh sauce)	2	67.1	76.2 (62.1-105)	180 (128-206)	62.2 (50.3-81.3)	48.8 (43.9-52.7)
	Maize (yellow, roasted)	6	42.8	211 (89.1-325)	8.73 (2.46-27.5)	30.7 (16.0-55.3)	71.9 (25.6-112)
	Maize (yellow, boiled)	2	72.7	195 (189-201)	7.54 (6.99-8.09)	19.1 (18.5-19.7)	47.2 (44.8-49.6)
	Maize (white, roasted)	2	54.6	126 (122-131)	35.7 (33.7-37.8)	39.6 (34.4-44.8)	93.6 (88.4-99.0)
	Tunwo (white maize, cooked)	4	77.9	60.3 (21.4-138)	28.9 (4.66-74.3)	22.7 (15.2-36.9)	32.6 (22.6-52.2)
	Tunwo (rice, cooked)	3	77.0	19.4 (12.5-30.5)	27.4 (12.5-49.3)	5.29 (2.11-9.88)	9.58 (5.02-17.2)
	Bread (white)	12	30.7	140 (75.6-216)	256 (120-335)	30.3 (16.5-63.8)	51.0 (14.7-99.5)
	Whole wheat bread	3	26.8	174 (153-196)	148 (128-167)	37.9 (37.2-38.7)	94.9 (82.0-108)
	Malt bread	2	28.3	220 (208-233)	116 (116-117)	37.5 (36.9-38.0)	24.4 (23.2-25.6)
	Agidi (white maize, cooked)	10	86.4	25.6 (6.07-67.1)	5.28 (0.58-16.7)	7.98 (3.70-24.2)	11.5 (3.67-32.3)

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'(continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Cereals and cereal products (contd.)	Kokoro (white maize, fried)	2	5.23	250 (233-260)	538 (516-580)	3.15 (2.80-3.50)	94.8 (88.3-98.5)
	Semovita (cooked)	8	76.2	47.1 (27.9-112)	5.06 (2.07-10.2)	14.6 (9.19-21.0)	14.3 (7.34-21.9)
	Donkwa	2	18.0	325 (255-395)	219 (167-274)	111 (79.6-145)	181 (163-210)
	Fura	2	57.5	119 (109-136)	8.09 (7.25-10.1)	13.1 (8.43-18.2)	49.6 (45.0-54.5)
	Oat (cooked)	2	89.0	36.8 (35.7-38.0)	3.56 (3.26-3.86)	4.79 (4.22-5.36)	4.04 (3.47-4.60)
	Custard (cooked)	2	91.0	2.92 (2.90-2.93)	92.8 (91.3-94.3)	26.4 (26.0-26.8)	18.0 (15.9-20.2)
	Granola	2	1.63	91.0 (81.9-99.5)	232 (182-304)	30.4 (27.5-35.0)	19.4 (14.0-29.7)
	Cornflakes	2	1.17	821 (811-831)	2.55 (1.80-3.30)	8.50 (7.00-10.0)	70.0 (65.0-75.0)
	Spaghetti (boiled)	2	63.5	156 (151-161)	53.6 (52.4-54.8)	1.70 (1.65-1.75)	14.7 (14.3-15.2)
	Jollof spaghetti	2	67.0	130 (126-135)	317 (312-322)	2.01 (1.99-2.03)	18.2 (17.7-18.8)
	Noodles (plain, boiled)	6	61.0	69.9 (59.5-89.2)	589 (487-734)	1.15 (0.612-2.09)	9.76 (8.06-11.3)
	Noodles (boiled, with steamed or fried egg)	3	59.4	73.0 (53.5-100)	594 (380-861)	3.43 (2.47-4.16)	10.3 (7.81-12.4)
Legumes and legume products	Bean pottage (White bean)	4	70.0	351 (274-480)	441 (377-576)	16.8 (12.7-21.4)	54.0 (49.2-59.5)
	Bean (Drum, plain, boiled)	8	64.2	331 (83.9-495)	282 (66.2-443)	18.5 (9.80-29.2)	59.9 (16.9-93.2)
	Bean (White, plain, boiled)	2	60.9	433 (310-557)	297 (287-306)	20.2 (16.2-24.1)	63.7 (50.3-77.0)

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'(continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Legumes and legume products (contd.)	Bean (Olo 2, plain, boiled)	2	68.6	298 (269-335)	248 (233-262)	28.5 (25.2-32.7)	87.3 (76.5-102)
	Bean (Pewu, plain, boiled)	2	67.8	74.9 (74.8-75.1)	147 (137-158)	16.4 (16.3-16.5)	30.7 (26.2-35.2)
	Akara (White bean, fried)	3	53.3	349 (339-364)	362 (337-410)	28.3 (16.5-38.6)	76.9 (71.3-86.5)
	Akara (Pewu bean, fried)	2	54.3	502 (443-599)	404 (262-488)	19.3 (18.9-19.9)	61.7 (55.3-64.6)
	Akara (Drum bean, fried)	2	50.5	541 (387-696)	620 (619-622)	18.5 (18.0-19.2)	60.9 (54.4-67.4)
	Moi-moi (White bean, cooked)	2	68.5	277 (207-314)	368 (335-386)	27.0 (25.1-29.1)	61.6 (59.2-66.2)
	Moi-moi (Olo 2 bean, cooked)	2	73.2	269 (261-276)	484 (344-623)	16.5 (16.3-16.7)	32.9 (29.0-36.7)
	Okpa bean (boiled)	2	59.6	266 (252-280)	516 (514-518)	16.8 (14.4-19.1)	71.7 (69.5-74.1)
	Moi-moi (Okpa bean, cooked)	4	64.1	286 (244-336)	456 (427-486)	22.3 (19.6-26.9)	85.7 (72.1-98.7)
	Tofu pie	2	33.8	226 (215-236)	432 (428-436)	30.6 (30.0-31.2)	58.0 (56.3-59.6)
	Tofu (fried)	4	51.5	141 (109-184)	499 (435-579)	50.3 (47.6-53.6)	47.5 (40.5-55.5)
	Kunu (gyada)	2	89.6	29.6 (28.8-30.3)	1.70 (1.62-1.78)	4.51 (3.87-4.96)	8.22 (7.37-9.06)

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'(continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Fruits	Banana (ripe)	14	75.1	263 (204-338)	7.14 (3.24-15.9)	3.38 (1.44-7.42)	41.7 (31.5-53.7)
	Carrot (raw)	3	88.1	312 (234-363)	11.2 (6.19-16.1)	9.27 (6.77-10.8)	18.6 (16.8-21.5)
	Cucumber	9	96.6	108 (86.3-150)	1.81 (0.78-4.06)	11.2 (11.7-12.6)	11.3 (7.39-17.1)
	Orange	14	89.3	126 (83.3-199)	1.46 (nd-3.13)	18.6 (14.0-25.1)	13.7 (10.3-19.8)
	Pawpaw	9	89.5	109 (78.9-141)	3.19 (1.51-6.43)	13.5 (11.5-17.3)	11.8 (5.74-20.6)
	Pineapple	7	84.8	84.9 (52.1-109)	2.58 (1.07-5.53)	10.6 (6.54-15.6)	23.5 (16.4-29.0)
	Water melon (fruit without seed)	15	93.4	87.6 (61.0-121)	2.78 (1.13-5.89)	4.59 (3.60-6.49)	8.92 (5.20-15.1)
	Water melon (seed)	2	53.2	266 (263-268)	13.9 (13.8-13.9)	6.26 (6.18-6.35)	143 (142-145)
	Golden melon	2	93.5	144 (138-149)	1.53 (1.41-1.65)	7.41 (7.32-7.50)	14.7 (14.2-15.2)
	Guava	4	84.0	260 (177-380)	4.52 (3.16-7.34)	15.4 (8.68-19.8)	15.9 (11.3-23.4)
	Date	3	10.0	607 (565-644)	16.1 (10.5-26.7)	38.2 (26.3-47.4)	56.2 (48.1-74.9)
	Tiger nut (yellow, raw)	2	47.5	302 (296-308)	10.6 (10.1-11.1)	27.6 (27.0-28.2)	54.8 (54.5-55.2)
	Tiger nut (brown, raw)	2	14.5	473 (462-483)	8.54 (7.99-9.08)	26.2 (23.2-29.1)	74.5 (70.9-78.1)
	Apple (wine)	6	83.8	85.2 (70.6-105)	3.29 (2.14-5.25)	4.63 (3.48-6.41)	9.11 (4.38-24.0)
	Apple (green)	12	84.7	76.4 (50.0-116)	3.32 (1.06-6.67)	2.71 (1.65-3.76)	6.59 (3.47-13.1)
	Apple (wine/green)	2	83.0	85.2 (84.9-85.4)	3.20 (3.04-3.35)	6.5 (6.00-7.00)	7.30 (5.78-8.82)
	Pear apple (European pear)	4	84.2	81.9 (69.0-105)	3.34 (2.41-4.86)	10.9 (7.00-12.1)	14.7 (10.8-21.9)
	Tangerine	4	89.8	144 (134-160)	2.85 (1.67-3.82)	15.1 (12.2-16.9)	13.0 (9.61-15.1)
	Plum	2	81.9	169 (167-172)	5.23 (4.91-5.55)	0.650 (0.610-0.715)	7.44 (7.07-7.81)

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'(continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Leafy and fruity vegetables	Garden egg (light yellow)	7	92.9	151 (132-162)	2.24 (1.08-3.47)	6.29 (5.07-8.07)	16.0 (13.8-20.4)
	Garden egg (green)	7	91.8	159 (128-187)	3.16 (1.90-5.69)	6.33 (4.42-7.15)	20.6 (16.9-23.2)
	Garden egg (reddish)	2	91.7	141 (118-157)	7.75 (7.34-8.35)	5.73 (4.34-6.34)	24.9 (24.1-25.6)
	Garden egg (lemon)	2	92.5	131 (105-158)	2.41 (2.15-2.67)	4.92 (3.81-5.63)	14.3 (11.9-16.8)
	Green bell pepper (raw)	3	94.4	180 (163-189)	1.74 (1.45-2.01)	6.55 (4.67-9.15)	13.1 (11.4-14.9)
	Green peas (raw)	6	74.7	390 (266-554)	9.11 (3.43-15.6)	21.9 (17.1-29.2)	55.0 (33.6-65.8)
	Green bean (raw)	2	92.1	231 (181-282)	3.93 (3.23-4.62)	31.1 (29.3-32.9)	29.8 (25.7-33.8)
	Cabbage (raw)	7	93.1	204 (152-233)	4.00 (2.59-5.66)	15.6 (13.6-21.3)	14.7 (11.4-21.4)
	Cole slaw	3	81.1	203 (192-210)	142 (120-170)	14.1 (13.6-14.6)	18.0 (16.5-21.1)
Condiments, sauces and soups	Salt	4	2.3	207 (180-243)	30700 (28600-33300)	7.61 (5.56-8.78)	56.5 (29.4-87.6)
	Locust bean (raw)	6	65.8	35.6 (15.7-51.8)	503 (103-957)	69.8 (56.3-84.4)	67.3 (49.8-101)
	Stew (for rice, with vegetable oil)	23	71.5	273 (96.4-489)	786 (267-1580)	9.94 (5.90-24.7)	27.9 (11.5-50.3)
	Stew (for tunwo)	2	81.9	58.1 (57.3-58.9)	410 (408-411)	14.1 (12.2-16.0)	44.0 (42.8-45.3)
	Stew (with palm oil)	4	79.0	245 (199-296)	577 (401-706)	11.4 (6.65-20.2)	22.0 (13.6-30.8)
	Stew (for masa)	2	86.6	191 (190-191)	513 (479-546)	13.5 (12.4-14.7)	29.8 (29.0-30.6)
	Stew (for swallow)	2	76.1	217 (213-222)	484 (434-534)	5.83 (5.39-6.26)	16.7 (12.9-20.6)
	Stew (for local rice)	2	82.9	156 (138-167)	547 (480-585)	4.22 (4.11-4.28)	14.6 (13.9-15.3)
	Egg stew	2	65.4	301 (290-313)	986 (980-991)	29.7 (29.5-29.9)	22.8 (22.7-22.9)

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'(continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Condiments, sauces and soups (contd.)	Groundnut soup	2	84.1	102 (96.0-107)	644 (634-654)	15.1 (14.3-15.9)	28.9 (28.8-29.0)
	Gbegiri	2	71.3	209 (205-215)	421 (391-454)	19.7 (19.3-20.1)	38.0 (37.0-38.9)
	Ogbono soup	2	70.3	109 (108-109)	679 (678-680)	46.7 (45.6-47.8)	42.5 (41.7-43.2)
	Fried ugwu	2	72.2	226 (211-241)	400 (381-419)	49.6 (48.3-50.8)	93.6 (89.0-98.1)
	Okra (plain)	4	91.5	127 (118-137)	407 (374-436)	26.3 (21.9-39.2)	25.7 (18.2-33.6)
	Okra soup	4	93.2	96.2 (89.0-118)	394 (331-484)	38.6 (28.7-57.4)	19.4 (18.3-21.0)
	Tete soup	2	56.6	187 (177-197)	1170 (1140-1200)	39.8 (39.6-39.9)	113 (112-114)
	Ewedu (plain)	7	93.5	97.5 (62.0-146)	316 (237-525)	30.8 (16.0-49.2)	23.8 (17.1-34.2)
	Soko soup	2	60.1	202 (199-206)	690 (661-719)	35.7 (35.6-35.8)	71.6 (70.2-73.0)
	Ugwu soup (with egusi)	7	68.6	190 (153-290)	457 (344-637)	57.4 (41.2-94.4)	91.3 (64.8-133)
	Tete soup (with egusi)	3	74.2	133 (105-171)	507 (384-639)	58.6 (55.9-83.1)	86.0 (70.7-105)
	Efo riro (tete)	2	79.9	251 (226-277)	725 (608-843)	31.3 (25.3-37.3)	72.1 (64.7-79.5)
	Soko soup (with egusi)	2	70.2	237 (170-272)	697 (554-785)	44.6 (23.3-55.9)	155 (148-163)
	Water leaf soup	4	71.6	201 (183-217)	552 (526-580)	38.2 (30.1-48.8)	72.7 (59.8-87.1)
	Water leaf (with ugwu) soup	2	72.0	254 (249-258)	423 (416-429)	58.7 (52.4-65.1)	74.1 (72.5-75.7)
	Efo riro (soko)	3	73.9	199 (161-230)	721 (575-927)	39.8 (37.8-41.6)	76.2 (71.1-82.0)
	Ukase soup (with egusi)	2	83.0	93.2 (92.2-94.2)	341 (326-356)	23.1 (22.2-24.1)	42.9 (41.9-44.0)

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'(continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Dairy products	Nunu (locally fermented milk)	2	92.3	106 (95.5-115)	22.5 (21.3-24.7)	51.6 (54.3-72.8)	16.7 (12.1-22.0)
	Powdered low-fat milk (raw)	2	3.6	1430 (1400-1450)	249 (244-254)	740 (720-760)	91.8 (91.5-92.1)
	Powdered full-cream milk (raw)	3	3.9	1440 (1320-1550)	337 (294-400)	770 (730-815)	95.0 (89.7-97.2)
	Cheese (local)	2	63.4	118 (109-126)	448 (435-462)	293 (285-301)	52.4 (49.2-55.5)
Beef, poultry and eggs	Beef (boiled, in stew)	11	65.5	201 (121-265)	494 (312-850)	18.8 (13.9-31.2)	35.0 (24.8-55.1)
	Beef (fried, in stew)	9	46.7	309 (231-418)	563 (352-840)	21.2 (14.8-27.6)	35.5 (29.2-46.9)
	Ponmo (raw)	6	79.0	6.95 (2.57-10.8)	15.7 (11.5-19.9)	5.85 (3.11-7.14)	6.51 (3.81-9.46)
	Chicken thigh (fried)	5	44.5	251 (178-291)	363 (153-573)	34.7 (28.0-43.9)	40.3 (29.1-56.8)
	Turkey wing (fried)	5	44.3	266 (241-305)	480 (268-869)	39.0 (31.6-45.8)	49.7 (39.6-64.7)
	Egg (boiled)	4	74.6	148 (96.8-177)	152 (135-170)	47.1 (38.8-58.3)	16.8 (14.3-19.7)
	Egg (boiled, in stew)	3	72.2	180 (138-228)	377 (216-470)	48.2 (44.8-50.1)	18.9 (16.9-20.7)
Fish	Shawa (fried)	6	40.9	684 (567-869)	320 (267-377)	59.9 (55.1-68.5)	69.4 (48.9-95.8)
	Shawa (tiny, fried)	2	24.3	876 (867-886)	244 (240-248)	37.4 (36.7-38.1)	77.8 (77.0-78.6)
	Shawa (smoked)	4	53.9	518 (427-560)	232 (159-263)	52.9 (32.0-68.4)	55.9 (47.4-66.9)
	Kote (fried)	5	52.6	382 (152-678)	438 (277-629)	80.1 (71.2-93.9)	53.2 (25.7-83.0)
	Titus (fried)	4	48.3	369 (333-453)	274 (133-430)	34.5 (32.1-38.8)	47.4 (38.1-57.7)

Table 4.12: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed'(continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Potassium	Sodium	Calcium	Magnesium
Fish (contd.)	Titus (smoked)	2	45.3	455 (424-475)	276 (243-319)	27.1 (26.6-28.0)	54.0 (52.4-57.0)
	Ebolo (smoked)	2	22.3	1510 (1490-1540)	474 (463-485)	71.0 (69.2-72.7)	116 (111-122)
	Cat fish (roasted)	5	13.9	845 (434-1140)	303 (226-436)	65.3 (48.5-85.7)	133 (109-168)
	Cray fish (roasted)	4	11.4	859 (704-1090)	1080 (762-1410)	171 (135-213)	416 (358-492)
	Panla (roasted)	4	61.3	464 (164-838)	455 (339-587)	37.9 (30.3-46.9)	124 (117-136)
	Panla (fried)	3	40.1	623 (400-845)	659 (624-700)	34.4 (26.6-44.7)	108 (77-135)
	Stockfish	3	5.5	1520 (1510-1540)	585 (581-589)	330 (297-364)	229 (223-234)
	Apo (roasted)	3	10.4	1240 (1170-1300)	501 (479-526)	24.8 (22.4-29.8)	149 (136-173)
	Palamu (roasted)	3	6.5	1330 (1200-1430)	714 (645-766)	37.1 (33.2-41.7)	208 (183-248)
Oil seeds	Ground nut (roasted)	6	3.11	717 (523-841)	377 (222-587)	45.8 (40.6-48.5)	272 (196-375)
	Ground nut (boiled)	6	40.5	327 (258-394)	294 (110-447)	42.8 (38.6-49.5)	147 (114-209)
	Cashew nut (roasted)	2	4.78	554 (547-567)	45.5 (44.0-47.9)	47.8 (39.6-53.6)	426 (407-451)
	Walnut (boiled)	2	36.4	867 (849-885)	12.4 (12.1-12.6)	76.1 (69.7-80.1)	248 (242-254)
	Coconut (fresh)	3	41.9	317 (170-398)	24.2 (16.5-36.4)	16.3 (10.2-24.0)	97.6 (75.5-132)
Sugar and cocoa product	Refined sugar (raw)	4	1.00	33.3 (30.0-39.1)	1.97 (1.56-2.62)	4.20 (2.80-5.80)	1.16 (0.900-1.60)
	Cocoa product (raw)	2	1.55	825 (784-867)	120 (113-127)	66.5 (65.8-67.1)	195 (191-200)

Values are presented as mean (min.-max.); nd – not detected (value is below Limit of Detection (LOD)). LOD (mg/100 g sample) = 0.207, 0.251, 0.178 and 0.162 for K, Na, Ca and Mg, respectively.

4.18 Moisture content and mean concentrations of microminerals in some Nigerian foods analysed 'as consumed'

Table 4.13 highlights the mean levels of copper (Cu), manganese (Mn), iron (Fe) and zinc (Zn) in 143 Nigerian foods 'as consumed' at individual food level. The mean Cu levels ranged from non-detectable in 'ogbono', fried 'ugu', waterleaf and 'tete/aleiho' soups to 3.77 mg/100g in roasted cashew nut. Other rich sources of Cu are cray fish (2.15 mg/100g), boiled walnut (1.47 mg/100g) and roasted cat fish (1.29 mg/100g). The legumes and legume products, beef, poultry and eggs, fish, as well as nuts are generally rich sources of Cu.

The mean Mn level was highest in smoked 'ebolo' fish (3.74 mg/100g) and non-detectable in pottage yam, custard, egg stew, 'gbegiri' and smoked 'shawa'. Rich sources of Mn include oil seeds, leafy vegetable soups, legumes and legume products, cereals (such as granola, bread, 'donkwa', roasted maize) and plantain chip.

The mean concentrations of Fe ranged from 0.187 mg/100g in cucumber to 14.7 mg/100g in cashew nut. Other significant sources of Fe are roasted cray fish (11.6 mg/100g), cocoa product (10.8 mg/100g), local cheese (9.06 mg/100g), smoked 'ebolo' fish (8.15 mg/100g), roasted ground nut (7.96 mg/100g) and 'donkwa' (7.92 mg/100g). The legume and legume products, green leafy vegetable soups, beef, poultry and eggs, fish, oil seeds and cocoa products are generally rich in Fe.

The mean levels of Zn ranged from 0.069 mg/100g in orange to 6.42 mg/100g in fried beef in stew. Cashew nut (5.68 mg/100g), roasted crayfish (5.57 mg/100g), smoked 'ebolo' fish (5.24 mg/100g), roasted 'palamu' fish (4.68 mg/100g) and boiled beef in stew (4.44 mg/100g) are other rich sources of Zn. Generally, milk, cheese, beef, poultry eggs, fish and oil seed are excellent sources of Zn, among the individual foods analysed.

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed'

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Tubers, starches and their products	Fufu (cooked)	11	67.8	0.125 (0.035-0.260)	0.162 (0.042-0.440)	0.760 (0.228-1.80)	0.217 (0.069-0.427)
	Sweet potato (fried)	4	53.0	0.257 (0.187-0.372)	0.460 (0.189-0.938)	1.38 (0.625-2.72)	0.303 (0.116-0.694)
	Amala (yam powder, cooked)	10	76.3	0.220 (0.118-0.433)	0.248 (0.097-0.473)	2.02 (1.09-3.29)	0.391 (0.238-0.714)
	Pounded yam	6	70.3	0.201 (0.145-0.283)	0.214 (0.086-0.482)	1.55 (0.520-3.51)	0.325 (0.197-0.474)
	Yam (white, fried)	8	54.5	0.297 (0.145-0.419)	0.295 (0.085-0.681)	2.35 (0.828-5.12)	0.571 (0.283-1.12)
	Yam (white, boiled)	2	64.2	0.626 (0.611-0.640)	0.639 (0.578-0.701)	4.22 (3.91-4.52)	0.865 (0.794-0.937)
	Poundo yam (cooked)	2	73.9	0.086 (0.079-0.092)	0.073 (0.070-0.077)	0.292 (0.280-0.304)	0.369 (0.363-0.376)
	Yam pottage (white yam)	2	68.8	0.032 (nd-0.050)	nd	1.29 (0.915-1.97)	0.502 (0.329-0.622)
	Cake (water yam, fried)	2	63.3	0.365 (0.318-0.412)	0.021 (nd-0.031)	2.15 (2.04-2.26)	0.420 (0.398-0.443)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Tubers, starches and their products	Eba (white gari, cooked)	9	72.6	0.133 (0.065-0.313)	0.340 (0.102-0.605)	2.00 (0.625-3.39)	0.234 (0.096-0.583)
	Bread fruit (boiled)	2	79.5	0.181 (0.173-0.188)	0.343 (0.313-0.372)	1.93 (1.84-2.01)	0.228 (0.215-0.241)
	Plantain (mature ripe, fried)	6	43.3	0.265 (0.120-0.455)	0.193 (0.138-0.241)	1.75 (0.702-2.43)	0.485 (0.209-0.897)
	Plantain chip (mature unripe, fried)	4	1.95	0.408 (0.333-0.474)	1.80 (1.24-2.70)	2.87 (2.19-3.45)	0.646 (0.492-0.890)
Cereals and cereal products	Rice (Long grain, boiled)	14	71.0	0.216 (0.111-0.386)	0.154 (0.031-0.502)	1.59 (1.06-3.58)	0.332 (0.164-0.969)
	Rice (Short grain, boiled)	3	71.7	0.184 (0.170-0.198)	0.418 (0.414-0.422)	2.70 (2.61-2.79)	0.319 (0.258-0.379)
	Local Rice (boiled)	4	71.9	0.249 (0.180-0.312)	0.339 (0.128-0.689)	1.78 (1.21-2.57)	0.384 (0.291-0.474)
	Jollof Rice	5	65.8	0.132 (0.082-0.183)	0.112 (nd-0.235)	2.12 (1.95-2.43)	0.313 (0.230-0.440)
	Fried Rice	3	62.5	0.127 (nd-0.321)	0.064 (nd-0.150)	2.11 (1.77-2.69)	0.424 (0.310-0.596)
	Masa (rice cake, fried)	3	56.4	0.203 (0.173-0.220)	0.357 (0.243-0.566)	2.12 (1.58-3.12)	0.917 (0.347-1.90)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Cereals and cereal products	Garogaro (rice, beans, boiled; along with fresh sauce)	2	67.1	0.222 (0.193-0.255)	0.522 (0.446-0.641)	2.84 (2.24-3.51)	0.911 (0.741-1.03)
	Maize (yellow, roasted)	6	42.8	0.277 (0.120-0.837)	0.407 (0.154-0.823)	1.40 (0.862-3.31)	1.32 (0.776-2.06)
	Maize (yellow, boiled)	2	72.7	0.223 (0.218-0.228)	0.348 (0.328-0.368)	0.394 (0.352-0.435)	0.673 (0.662-0.683)
	Maize (white, roasted)	2	54.6	0.273 (0.255-0.290)	0.768 (0.733-0.803)	3.99 (3.75-4.24)	1.06 (1.03-1.09)
	Tunwo (white maize, cooked)	4	77.9	0.077 (nd-0.129)	0.121 (0.028-0.278)	0.914 (0.410-1.57)	0.287 (0.131-0.560)
	Tunwo (rice, cooked)	3	77.0	0.162 (0.124-0.216)	0.153 (0.127-0.186)	0.277 (0.197-0.373)	0.363 (0.210-0.655)
	Bread (white)	12	30.7	0.409 (0.244-0.641)	0.861 (0.410-1.65)	2.94 (1.08-6.74)	1.06 (0.640-1.75)
	Whole wheat bread	3	26.8	0.476 (0.382-0.569)	0.732 (0.508-0.957)	2.23 (1.84-2.62)	1.34 (1.08-1.60)
	Malt bread	2	28.3	0.287 (0.260-0.315)	0.608 (0.607-0.610)	3.41 (3.39-3.43)	1.36 (1.34-1.37)
	Agidi (white maize, cooked)	10	86.4	0.130 (0.078-0.293)	0.069 (0.032-0.190)	0.634 (0.144-1.70)	0.208 (0.110-0.431)
	Kokoro (white maize, fried)	2	5.23	0.027 (nd-0.030)	0.232 (0.099-0.337)	5.80 (2.98-7.54)	2.22 (2.04-2.36)
	Semovita (cooked)	8	76.2	0.141 (0.067-0.411)	0.359 (0.175-0.635)	0.831 (0.353-1.86)	0.308 (0.121-0.552)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Cereals and cereal products	Donkwa	2	18.0	0.537 (0.469-0.608)	1.52 (1.28-1.63)	7.92 (6.84-9.24)	4.20 (3.72-4.53)
	Fura	2	57.5	0.308 (0.242-0.385)	0.571 (0.413-0.712)	4.75 (1.75-7.65)	1.24 (0.990-1.62)
	Oat (cooked)	2	89.0	0.188 (0.142-0.233)	0.356 (0.353-0.358)	0.428 (0.406-0.450)	0.246 (0.244-0.248)
	Custard (cooked)	2	91.0	0.061 (0.046-0.076)	nd	0.250 (0.244-0.256)	0.024 (nd-0.028)
	Granola	2	1.63	0.527 (0.397-0.649)	1.79 (1.64-2.00)	3.29 (2.83-4.08)	1.78 (1.62-2.06)
	Cornflakes	2	1.17	0.446 (0.421-0.471)	0.246 (0.236-0.256)	0.760 (0.735-0.785)	0.566 (0.551-0.581)
	Spaghetti (boiled)	2	63.5	0.136 (0.126-0.146)	0.278 (0.266-0.289)	0.587 (0.582-0.592)	0.456 (0.431-0.481)
	Jollof spaghetti	2	67.0	0.149 (0.144-0.154)	0.248 (0.233-0.263)	2.16 (2.06-2.27)	0.538 (0.514-0.561)
	Noodles (plain, boiled)	6	61.0	0.141 (0.029-0.490)	0.206 (0.180-0.234)	0.932 (0.646-1.46)	0.369 (0.137-0.632)
	Noodles (boiled, with steamed or fried egg)	3	59.4	0.479 (0.240-0.765)	0.209 (0.178-0.218)	1.12 (0.765-1.37)	1.01 (0.361-2.16)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Legumes and legume products	Bean pottage (White bean)	4	70.0	0.283 (0.178-0.408)	0.366 (0.103-0.481)	1.67 (1.12-2.25)	0.869 (0.371-1.06)
	Bean (Drum, plain, boiled)	8	64.2	0.352 (0.188-0.496)	0.734 (0.388-1.38)	1.91 (1.27-3.25)	1.18 (0.942-1.66)
	Bean (White, plain, boiled)	2	60.9	0.400 (0.319-0.480)	0.645 (0.423-0.866)	1.25 (1.12-1.37)	1.03 (0.898-1.16)
	Bean (Olo 2, plain, boiled)	2	68.6	0.409 (0.365-0.434)	1.04 (0.976- 1.09)	3.64 (3.34-4.07)	1.13 (1.04-1.26)
	Bean (Pewu, plain, boiled)	2	67.8	0.499 (0.331-0.666)	0.568 (0.540-0.595)	1.61 (1.56-1.65)	1.05 (1.03-1.07)
	Akara (White bean, fried)	3	53.3	0.401 (0.314-0.477)	0.745 (0.594-1.01)	3.75 (2.31-5.81)	1.64 (1.12-2.04)
	Akara (Pewu bean, fried)	2	54.3	0.362 (0.271-0.478)	0.573 (0.536-0.597)	2.63 (1.74-3.57)	1.30 (1.07-1.47)
	Akara (Drum bean, fried)	2	50.5	0.393 (0.377-0.408)	0.668 (0.500-0.835)	2.47 (1.62-3.33)	1.31 (1.03-1.60)
	Moi-moi (White bean, cooked)	2	68.5	0.332 (0.270-0.431)	0.760 (0.735-0.797)	2.93 (2.47-3.67)	1.09 (0.887-1.40)
	Moi-moi (Olo 2 bean, cooked)	2	73.2	0.050 (0.043-0.056)	0.395 (0.234-0.556)	5.57 (4.62-6.52)	0.806 (0.776-0.835)
	Okpa bean (boiled)	2	59.6	0.351 (0.349-0.352)	0.665 (0.625-0.704)	4.42 (4.01-4.83)	3.01 (2.83-3.18)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Legumes and legume products	Moi-moi (Okpa bean, cooked)	4	64.1	0.328 (0.266-0.388)	0.725 (0.605-0.871)	2.96 (2.30-3.86)	1.45 (0.771-2.46)
	Tofu pie	2	33.8	0.249 (0.224-0.274)	0.769 (0.741-0.796)	2.05 (1.89-2.22)	1.14 (1.10-1.18)
	Tofu (fried)	4	51.5	0.759 (0.663-0.944)	1.66 (1.31-2.13)	7.31 (5.34-9.26)	2.51 (2.06-3.07)
	Kunu (gyada)	2	89.6	0.024 (0.017-0.030)	0.128 (0.124-0.132)	0.259 (0.220-0.299)	0.203 (0.203-0.204)
Fruits	Banana (ripe)	14	75.1	0.185 (0.118-0.341)	0.442 (0.146-1.07)	0.692 (0.290-1.25)	0.286 (0.188-0.521)
	Carrot (raw)	3	88.1	0.135 (0.110-0.154)	0.279 (0.257-0.308)	0.372 (0.338-0.404)	0.415 (0.256-0.596)
	Cucumber	9	96.6	0.041 (0.028-0.079)	0.055 (0.031-0.117)	0.187 (0.106-0.273)	0.102 (0.079-0.133)
	Orange	14	89.3	0.069 (0.031-0.161)	0.059 (0.029-0.116)	0.252 (0.042-0.591)	0.069 (0.025-0.254)
	Pawpaw	9	89.5	0.078 (0.031-0.198)	0.046 (0.029-0.059)	0.389 (0.104-0.723)	0.105 (0.064-0.192)
	Pineapple	7	84.8	0.116 (0.048-0.267)	0.517(0.172-1.03)	0.597 (0.221-1.13)	0.097 (0.037-0.194)
	Water melon (fruit without seed)	15	93.4	0.050 (0.028-0.134)	0.040 (nd-0.060)	0.273 (0.130 0.634)	0.078 (0.050-0.113)
	Water melon (seed)	2	53.2	1.19 (1.16-1.21)	0.822 (0.782-0.862)	2.36 (2.29-2.43)	1.48 (1.42-1.54)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Fruits	Golden melon	2	93.5	0.027 (0.019-0.030)	0.055 (0.053-0.057)	0.308 (0.263-0.352)	0.144 (0.126-0.162)
	Guava	4	84.0	0.183 (0.136-0.241)	0.158 (0.107-0.239)	0.412 (0.273-0.591)	0.143 (0.076-0.271)
	Date	3	10.0	0.331 (0.263-0.435)	0.344 (0.293-0.433)	1.10 (0.970-1.30)	0.361 (0.145-0.574)
	Tiger nut (yellow, raw)	2	47.5	0.260 (0.220-0.297)	1.04 (0.989-1.08)	1.79 (1.63-1.95)	0.944 (0.890-0.998)
	Tiger nut (brown, raw)	2	14.5	0.292 (0.258-0.326)	1.15 (1.13-1.16)	2.61 (2.54-2.68)	1.20 (1.18-1.22)
	Apple (wine)	6	83.8	0.108 (nd-0.214)	0.129 (0.030-0.315)	0.772 (0.223-1.45)	0.125 (0.040-0.347)
	Apple (green)	12	84.7	0.081 (nd-0.179)	0.068 (0.031-0.147)	0.362 (0.090-0.616)	0.138 (0.024-0.299)
	Apple (wine/green)	2	83.0	0.072 (0.067-0.076)	0.068 (0.059-0.077)	0.246 (0.192-0.299)	0.032 (0.022-0.035)
	Pear apple (European pear)	4	84.2	0.064 (0.047-0.105)	0.076 (0.055-0.095)	0.401 (0.274-0.549)	0.215 (0.143-0.358)
	Tangerine	4	89.8	0.045 (0.021-0.067)	0.058 (0.026-0.087)	0.433 (0.323-0.553)	0.213 (0.117-0.269)
	Plum	2	81.9	0.025 (0.017-0.030)	0.048 (0.042-0.053)	0.661 (0.648-0.673)	0.150 (0.130-0.169)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Leafy and fruity vegetables	Garden egg (light yellow)	7	92.9	0.092 (0.064-0.131)	0.117 (0.048-0.209)	0.248 (0.133-0.321)	0.117 (0.078-0.180)
	Garden egg (green)	7	91.8	0.181 (0.122-0.239)	0.165 (0.091-0.228)	0.443 (0.278-0.690)	0.194 (0.154-0.254)
	Garden egg (reddish)	2	91.7	0.175 (0.165-0.185)	0.129 (0.121-0.135)	0.299 (0.290-0.315)	0.187 (0.174-0.199)
	Garden egg (lemon)	2	92.5	0.076 (0.065-0.087)	0.094 (0.079-0.109)	0.288 (0.238-0.338)	0.158 (0.140-0.176)
	Green bell pepper (raw)	3	94.4	0.123 (0.063-0.202)	0.144 (0.115-0.212)	0.319 (0.256-0.402)	0.113 (0.093-0.135)
	Green peas (raw)	6	74.7	0.445 (0.133-0.658)	0.690 (0.354-1.08)	2.54 (1.68-3.41)	1.63 (1.07-2.04)
	Green bean (raw)	2	92.1	0.096 (0.085-0.107)	0.499 (0.399-0.599)	0.936 (0.846-1.03)	0.416 (0.362-0.469)
	Cabbage (raw)	7	93.1	0.036 (0.026-0.061)	0.210 (0.117-0.344)	0.382 (0.185-0.700)	0.193 (0.118-0.318)
	Cole slaw	3	81.1	0.051 (nd-0.123)	0.089 (nd-0.235)	0.305 (0.135-0.570)	0.173 (0.100-0.280)
Condiments, sauces and soups	Salt	4	2.3	0.228 (0.107-0.416)	0.030 (nd-0.111)	3.13 (2.11-3.71)	0.170 (0.022-0.352)
	Locust bean (raw)	6	65.8	0.665 (0.312-0.882)	2.55 (1.87-3.77)	2.37 (0.534-4.97)	1.76 (1.23-2.17)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Condiments, sauces and soups	Stew (for rice, with vegetable oil)	23	71.5	0.138 (0.057-0.237)	0.161 (0.025-0.726)	3.10 (1.07-8.64)	0.286 (0.073-0.700)
	Stew (for tunwo)	2	81.9	0.142 (0.132-0.151)	0.321 (0.277-0.365)	1.80 (1.63-1.97)	0.412 (0.403-0.421)
	Stew (with palm oil)	4	79.0	0.173 (0.064-0.395)	0.125 (0.068-0.213)	1.84 (1.21-2.35)	0.381 (0.286-0.455)
	Stew (for masa)	2	86.6	0.125 (0.118-0.132)	0.237 (0.213-0.261)	1.94 (1.87-2.00)	0.356 (0.258-0.454)
	Stew (for swallow)	2	76.1	0.159 (0.109-0.208)	0.065 (0.054-0.075)	3.75 (2.79-4.70)	0.276 (0.219-0.332)
	Stew (for local rice)	2	82.9	0.110 (0.085-0.149)	0.093 (0.060-0.139)	1.65 (1.39-2.13)	0.244 (0.205-0.280)
	Egg stew	2	65.4	0.106 (0.097-0.114)	nd	1.96 (1.86-2.05)	0.857 (0.833-0.880)
	Groundnut soup	2	84.1	0.125 (0.108-0.141)	0.198 (0.179-0.216)	2.30 (2.16-2.43)	0.295 (0.259-0.330)
	Gbegiri	2	71.3	0.152 (0.137-0.168)	nd	0.915 (0.834-0.985)	0.687 (0.610-0.753)
	Ogbono soup	2	70.3	nd	0.291 (0.286-0.295)	3.82 (3.69-3.94)	0.186 (0.182-0.190)
	Fried ugwu	2	72.2	Nd	3.45 (3.40-3.51)	2.99 (2.94-3.04)	0.643 (0.635-0.651)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Condiments, sauces and soups	Okra (plain)	4	91.5	0.228 (0.174-0.294)	0.168 (0.135-0.199)	1.04 (0.903-1.25)	0.316 (0.177-0.431)
	Okra soup	4	93.2	0.063 (0.043-0.080)	0.217 (0.174-0.258)	0.661 (0.464-0.999)	0.232 (0.116-0.335)
	Tete soup	2	56.6	0.034 (0.021-0.039)	1.10 (1.06-1.14)	5.44 (5.31-5.57)	0.103 (0.095-0.111)
	Ewedu (plain)	7	93.5	0.125 (0.068-0.203)	0.440 (0.290-0.880)	1.78 (0.646-4.37)	0.300 (0.179-0.439)
	Soko soup	2	60.1	0.056 (0.055-0.057)	1.70 (1.68-1.73)	5.36 (5.23-5.48)	0.525 (0.497-0.552)
	Ugwu soup (with egusi)	7	68.6	0.367 (0.134-0.704)	0.576 (0.193-1.01)	3.70 (2.03-7.20)	1.17 (0.684-1.43)
	Tete soup (with egusi)	3	74.2	0.290 (0.233-0.396)	0.812 (0.599-1.03)	4.80 (2.09-6.66)	0.901 (0.553-1.49)
	Efo riro (tete)	2	79.9	0.298 (0.264-0.331)	0.674 (0.528-0.819)	2.37 (2.03-2.71)	0.673 (0.604-0.741)
	Soko soup (with egusi)	2	70.2	0.439 (0.257-0.530)	3.13 (2.32-3.77)	4.15 (2.90-4.94)	1.03 (0.832-1.14)
	Water leaf soup	4	71.6	0.153 (0.115-0.195)	1.86 (1.10-2.66)	3.14 (2.36-4.00)	0.508 (0.389-0.606)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Condiments, sauces and soups	Water leaf (with ugwu) soup	2	72.0	0.227 (0.204-0.249)	1.27 (1.23-1.30)	4.28 (4.16-4.41)	1.14 (1.06-1.22)
	Efo riro (soko)	3	73.9	0.186 (0.073-0.365)	1.99 (1.82-2.20)	4.03 (2.74-5.70)	0.803 (0.650-0.890)
	Ukase soup (with egusi)	2	83.0	0.054 (0.048-0.059)	0.898 (0.868-0.928)	1.52 (1.49-1.55)	0.492 (0.462-0.522)
Dairy products	Nunu (locally fermented milk)	2	92.3	0.131 (0.096-0.159)	0.910 (0.666-1.17)	0.361 (0.334-0.397)	0.368 (0.231-0.503)
	Powdered low-fat milk (raw)	2	3.6	0.258 (0.236-0.279)	0.023 (0.019-0.029)	2.39 (2.34-2.44)	2.81 (2.80-2.82)
	Powdered full-cream milk (raw)	3	3.9	0.437 (0.377-0.485)	0.027 (0.018-0.031)	4.86 (2.13-9.40)	3.53 (2.43-5.18)
	Cheese (local)	2	63.4	0.701 (0.686-0.716)	1.33 (1.31-1.35)	9.06 (8.85-9.26)	2.86 (2.64-3.07)
Beef, poultry and eggs	Beef (boiled, in stew)	11	65.5	0.384 (0.249-0.523)	0.221 (0.112-0.441)	3.15 (2.16-4.98)	4.44 (2.15-7.09)
	Beef (fried, in stew)	9	46.7	0.644 (0.448-1.06)	0.114 (0.066-0.154)	4.67 (1.76-7.12)	6.42 (2.89-13.2)
	Ponmo (raw)	6	79.0	0.206 (0.103-0.371)	0.046 (nd-0.099)	0.901 (0.314-1.35)	0.204 (0.139-0.241)
	Chicken thigh (fried)	5	44.5	0.702 (0.422-1.13)	0.646 (0.412-0.950)	4.74 (2.18-8.71)	4.09 (3.29-5.81)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Beef, poultry and eggs	Turkey wing (fried)	5	44.3	0.735 (0.589-0.915)	0.485 (0.325-0.598)	6.19 (5.40-7.59)	4.26 (3.49-5.24)
	Egg (boiled)	4	74.6	0.344 (0.202-0.495)	0.051 (0.00-0.086)	1.76 (1.56-1.93)	1.07 (0.718-1.37)
	Egg (boiled, in stew)	3	72.2	0.214 (0.201-0.224)	0.030 (nd-0.035)	2.18 (1.63-3.04)	1.10 (0.826-1.27)
Fish	Shawa (fried)	6	40.9	0.524 (0.280-0.852)	0.029 (nd-0.062)	2.09 (1.22-3.18)	2.15 (1.12-3.78)
	Shawa (tiny, fried)	2	24.3	0.685 (0.678-0.692)	0.552 (0.529-0.575)	3.17 (3.14-3.19)	4.53 (4.46-4.60)
	Shawa (smoked)	4	53.9	0.356 (0.187-0.483)	nd	2.62 (1.77-4.71)	1.13 (0.899-1.24)
	Kote (fried)	5	52.6	0.536 (0.197-0.868)	0.057 (0.029-0.156)	2.73 (1.75-4.13)	1.50 (0.825-2.66)
	Titus (fried)	4	48.3	0.504 (0.242-0.663)	0.028 (nd-0.032)	2.17 (1.03-3.98)	1.49 (1.29-1.78)
	Titus (smoked)	2	45.3	0.709 (0.625-0.852)	0.030 (0.021-0.033)	1.67 (1.54-1.85)	0.952 (0.859-1.00)
	Ebolo (smoked)	2	22.3	1.20 (1.08-1.32)	3.74 (3.63-3.85)	8.15 (7.99-8.32)	5.24 (5.06-5.42)
	Cat fish (roasted)	5	13.9	1.29 (0.73-2.06)	0.85 (0.479-1.66)	6.21 (3.53-7.93)	3.33 (1.85-7.84)
	Cray fish (roasted)	4	11.4	2.15 (1.86-2.49)	0.652 (0.434-0.940)	11.6 (8.77-16.5)	5.57 (4.89-6.84)
	Panla (roasted)	4	61.3	0.705 (0.495-1.06)	0.122 (0.076-0.194)	1.17 (0.616-1.91)	2.06 (1.51-2.69)
	Panla (fried)	3	40.1	0.729 (0.539-0.943)	0.108 (0.098-0.117)	1.44 (1.20-1.73)	1.88 (1.69-2.16)
	Stockfish	3	5.5	1.07 (1.01-1.11)	0.995 (0.968-1.02)	4.17 (4.07-4.28)	4.22 (3.93-4.50)
	Apo (roasted)	3	10.4	1.09 (0.829-1.25)	0.108 (0.094-0.135)	4.17 (3.81-4.53)	2.35 (2.00-2.87)
	Palamu (roasted)	3	6.5	0.973 (0.899-1.08)	0.461 (0.362-0.56)	4.43 (3.97-4.91)	4.68 (4.13-5.61)

Table 4.13: Moisture content (g/100g) and concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' (continued)

Food group	Individual food sample	Number of samples (n)	Moisture	Copper	Manganese	Iron	Zinc
Oil seeds	Ground nut (roasted)	6	3.11	1.13 (0.788-1.88)	1.89 (1.03-3.09)	7.96 (5.18-9.67)	3.33 (2.00-4.92)
	Ground nut (boiled)	6	40.5	0.69 (0.406-0.854)	1.20 (0.858-1.93)	3.11 (1.65-5.08)	2.06 (1.55-2.47)
	Cashew nut (roasted)	2	4.78	3.77 (3.63-4.00)	3.26 (3.06-3.42)	14.7 (14.2-14.9)	5.68 (5.28-5.08)
	Walnut (boiled)	2	36.4	1.47 (1.40-1.55)	1.09 (1.09-1.09)	5.54 (5.34-5.74)	3.10 (3.02-3.18)
	Coconut (fresh)	3	41.9	0.79 (0.453-1.08)	1.32 (0.862-2.00)	4.47 (3.95-5.48)	1.62 (1.38-2.60)
Sugar and cocoa product	Refined sugar (raw)	4	1.00	0.210 (0.102-0.345)	nd	2.27 (1.85-2.64)	0.139 (0.045-0.279)
	Cocoa product (raw)	2	1.55	0.726 (0.717-0.738)	0.357 (0.348-0.365)	10.8 (10.1-11.5)	1.78 (1.74-1.81)

Values are presented as mean (min.-max.); nd – not detected (value is below Limit of Detection (LOD)). LOD (mg/100 g sample) = 0.016, 0.017, 0.014 and 0.015 for Cu, Mn, Fe and Zn, respectively.

4.19 Comparison ofmean concentrations of macrominerals in some Nigerian foods analysed 'as consumed' in Ogun State and Abuja

The mean levels of macrominerals in selected foods from Ogun State and Abuja were compared for any significant difference at $p<0.05$ (Table 4.14). In all, 46 different foods were compared between the two locations. Nine food groups were represented in this comparison. They are tubers, starches and their products; cereals and cereal products; legumes and legume products; fruits; leafy and fruity vegetables; condiments, sauces and soups; beef, poultry and eggs; fish; and oil seeds. The mean K levels were found to be significantly different ($p <0.05$) in 17 foods that cut across 7 of the 9 food groups represented. Legumes and legume products as well as leafy and fruity vegetables were the groups with no significant differences ($p> 0.05$) between the samples from the two main locations. Twenty-four different food samples had their mean Na levels being significantly different ($p< 0.05$), when similar samples collected from Ogun State and Abuja were compared. These 24 samples included at least one sample from all the 9 food groups represented in Table 4.14. In the case of Ca, only 10 out of the 46 food samples compared between the two locations had their mean values being significantly different ($p< 0.05$) from each other. These samples included tubers, starches and their products; cereals and cereal products; and fruits. The mean concentrations of Mg in 25 individual food samples separately collected from Ogun State and Abuja were significantly different ($p< 0.05$) from each other. These foods included at least one food from each of all the 9 food groups represented. Generally, among these significantly different food samples, the mean K and Na levels were higher in more samples from Ogun State than Abuja; while the mean levels of Ca and Mg were higher in more samples from Abuja than Ogun State.

Table 4.14: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed' in Ogun State and Abuja

Food group	Individual food sample	Location	Moisture	Potassium	Sodium	Calcium	Magnesium
Tubers, starches and their products	Fufu (cooked)	Ogun	69.4	59.3 (32.8-78.6)	5.35 (2.01-8.59)	23.3 (14.8-37.2)	26.5 (9.78-35.7)*
		Abuja	66.5	48.7 (25.4-92.8)	9.77 (1.87-8.26)	18.5 (10.8-40.3)	14.0 (4.63-21.8)
	Sweet potato (fried)	Ogun	55.2	425 (424-426)*	138 (137-138)	22.3 (22.1-22.5)	17.4 (17.0-17.7)
		Abuja	50.9	383 (381-384)	186 (157-214)	39.4 (34.8-44.1)*	40.5 (29.7-51.3)*
	Amala (yam powder, cooked)	Ogun	75.3	178 (121-254)	14.9 (7.15-21.0)	18.4 (8.65-32.7)*	31.0 (10.3-41.4)*
		Abuja	77.8	210 (198-222)	15.4 (8.83-28.5)	9.66 (7.45-12.1)	19.2 (13.9-24.2)
	Pounded yam	Ogun	73.2	156 (142-171)	10.3 (5.60-15.8)	12.8 (9.29-18.0)	39.5 (35.4-45.1)*
		Abuja	68.3	206 (181-269)*	8.14 (2.84-12.2)	8.76 (4.11-16.7)	19.4 (10.4-29.7)
	Yam (white, fried)	Ogun	53.9	311 (249-389)	209 (125-382)	18.5 (12.8-31.7)	37.6 (12.7-79.1)
		Abuja	55.1	316 (306-325)	223 (222-223)	23.7 (23.4-23.9)*	40.4 (40.1-40.7)
	Eba (white gari)	Ogun	72.9	90.8 (35.7-181)	13.0 (4.92-18.9)*	24.6 (12.9-33.5)*	29.2 (10.9-43.8)
		Abuja	71.9	116 (113-119)	7.33 (7.31-7.35)	15.3 (14.8-15.8)	21.8 (20.9-22.8)
	Plantain (mature ripe, fried)	Ogun	41.5	470 (350-678)	16.5 (11.0-24.1)*	4.20 (2.58-8.37)	52.9 (38.3-59.5)
		Abuja	46.9	406 (406-407)	5.55 (3.85-7.25)	5.13 (4.98-5.31)	68.5 (65.4-71.6)*

Table 4.14: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Potassium	Sodium	Calcium	Magnesium
Cereals and cereal products	Rice (Long grain, boiled)	Ogun	69.5	37.5 (24.6-46.6)	216 (100-355)	11.1 (8.32-13.2)*	23.2 (12.8-36.3)*
		Abuja	72.0	32.2 (18.7-51.4)	197 (105-327)	7.32 (3.59-15.3)	6.99 (2.40-13.9)
	Jollof Rice	Ogun	65.2	112 (88.3-130)*	356 (326-380)	14.9 (11.2-18.9)	21.8 (16.3-26.7)
		Abuja	66.7	75.7 (74.0-77.1)	372 (352-409)	16.1 (12.7-18.7)	29.3 (26.0-31.0)*
	Maize (yellow, roasted)	Ogun	44.3	112 (89.1-152)	13.6 (2.58-27.5)	31.6 (23.4-55.3)	52.3 (25.6-112)
		Abuja	41.9	277 (248-325)*	5.50 (2.46-8.28)	30.1 (16.0-39.7)	84.9 (64.9-112)
	Tunwo (white maize, cooked)	Ogun	79.7	27.5 (26.5-28.5)	69.4 (64.4-74.3)*	26.4 (25.9-26.9)*	51.6 (51.1-52.2)*
		Abuja	77.0	76.7 (21.4-138)	8.67 (4.66-13.6)	15.8 (15.2-16.7)	23.1 (22.6-23.4)
	Bread (white)	Ogun	31.5	153 (75.6-216)	226 (120-323)	27.9 (16.5-63.8)	52.9 (14.7-99.5)
		Abuja	29.3	116 (103-126)	310 (292-335)*	34.6 (12.8-62.2)	47.5 (28.3-71.1)
	Agidi (white maize, cooked)	Ogun	87.5	43.3 (14.5-67.1)*	6.25 (0.58-16.7)	9.57 (3.70-24.2)	17.7 (3.72-32.3)*
		Abuja	85.5	10.1 (6.07-15.5)	4.43 (2.74-8.19)	6.58 (5.26-7.44)	5.97 (3.67-9.45)
	Semovita (cooked)	Ogun	77.4	66.1 (27.9-112)	6.34 (4.20-10.2)	13.6 (10.7-14.5)	16.4 (13.8-20.9)
		Abuja	75.8	40.8 (30.6-51.2)	4.63 (2.07-8.82)	14.9 (9.19-21.0)	13.6 (7.34-21.9)
Legumes and legume products	Bean (Drum, plain, boiled)	Ogun	64.5	268 (83.9-471)	216 (66.2-419)	20.9 (12.9-27.6)	61.3 (16.9-93.2)
		Abuja	64.0	367 (221-495)	319 (155-443)	17.1 (9.80-29.2)	59.0 (35.1-84.2)
	Tofu (fried)	Ogun	45.5	152 (118-184)	524 (468-579)*	48.8 (47.6-50.1)	45.1 (40.5-48.0)
		Abuja	63.4	118 (109-126)	448 (435-462)	52.3 (48.1-53.6)	52.4 (49.2-55.5)*

Table 4.14: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Potassium	Sodium	Calcium	Magnesium
Fruits	Banana (ripe)	Ogun	74.7	252 (204-338)	8.48 (3.27-15.9)*	2.81 (0.880-3.67)	41.1 (31.5-53.7)
		Abuja	76.3	294 (270-320)	3.47 (3.24-3.79)	5.03 (3.62-7.42)*	43.3 (40.1-45.9)
	Cucumber	Ogun	96.7	101 (86.3-126)	2.34 (0.78-4.06)*	11.1 (10.7-11.5)	11.5 (7.39-17.1)
		Abuja	96.6	119 (102-150)	1.02 (0.79-1.23)	11.4 (11.2-11.6)	11.0 (9.28-13.1)
	Orange	Ogun	90.5	112 (83.3-199)	2.29 (1.58-3.13)*	16.5 (14.6-18.9)	12.6 (10.8-11.9)
		Abuja	88.5	137 (107-159)	0.81 (nd-2.12)	20.3 (14.0-25.1)*	14.5 (10.3-19.8)
	Pawpaw	Ogun	88.2	119 (84.8-141)	4.46 (2.68-6.43)*	14.3 (13.5-14.9)	15.0 (7.58-20.6)
		Abuja	90.9	99.5 (78.9-108)	1.91 (1.51-2.75)	16.6 (13.7-18.3)	8.58 (5.74-11.5)
	Pineapple	Ogun	82.9	74.2 (52.1-94.3)	4.04 (2.52-5.53)*	9.07 (8.09-10.7)	21.9 (16.4-25.7)
		Abuja	86.3	93.7 (55.6-109)	1.36 (1.07-1.74)	11.9 (6.54-14.6)	24.9 (21.7-29.0)
	Water melon (fruit without seed)	Ogun	92.7	83.7 (61.0-121)	3.29 (1.66-5.89)	4.07 (3.60-4.76)	10.0 (5.38-15.1)
		Abuja	94.1	91.9 (77.7-99.7)	2.21 (1.13-4.02)	5.18 (3.77-6.49)*	7.72 (5.20-9.66)
	Apple (wine)	Ogun	83.7	87.9 (72.5-105)	3.60 (2.64-5.25)	4.30 (3.48-4.98)	7.46 (4.42-12.0)
		Abuja	83.9	80.6 (70.6-88.8)	2.76 (2.14-3.29)	5.06 (3.69-6.41)	11.9 (4.38-24.0)
	Apple (green)	Ogun	84.7	79.9 (50.0-116)*	3.70 (2.19-6.67)*	2.44 (1.65-3.32)	7.04 (3.57-13.1)*
		Abuja	84.8	57.4 (56.5-58.3)	1.26 (1.06-1.46)	3.10 (2.14-3.76)	4.09 (3.47-4.70)
	Pear apple	Ogun	82.2	93.7 (82.1-105)*	4.06 (3.27-4.86)*	9.50 (8.0-11.1)	18.4 (15.0-21.9)*
		Abuja	86.2	70.0 (69.0-71.1)	2.61 (2.41-2.81)	11.8 (11.5-12.1)*	11.0 (10.8-11.1)
Leafy and fruity vegetables	Garden egg (light yellow)	Ogun	92.2	146 (143-148)	3.02 (3.00-3.03)*	6.08 (5.12-7.66)	15.0 (14.8-15.1)
		Abuja	93.0	153 (132-162)	2.02 (1.08-3.47)	6.44 (5.07-8.07)	16.3 (13.8-20.4)
	Garden egg (green)	Ogun	91.7	153 (128-187)	3.61 (2.47-5.69)*	6.63 (4.42-6.80)	20.4 (16.9-22.8)
		Abuja	92.0	174 (161-187)	2.03 (1.90-2.16)	6.07 (4.78-7.15)	21.2 (19.3-23.2)

Table 4.14: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Potassium	Sodium	Calcium	Magnesium
Leafy and fruity vegetables	Cabbage (raw)	Ogun	91.8	215 (212-218)	5.20 (4.75-5.66)*	16.2 (15.1-18.2)	17.6 (17.3-18.0)
		Abuja	93.4	201 (152-233)	3.70 (2.59-5.05)	14.9 (13.6-21.3)	13.9 (11.4-21.4)
Condiments and soups	Locust bean (raw)	Ogun	61.2	48.7 (48.2-49.1)	577 (576-578)	66.5 (56.4-76.6)	62.3 (61.4-63.1)
		Abuja	67.4	31.2 (15.7-51.8)	478 (103-957)	72.2 (56.3-84.4)	69.0 (49.8-101)
	Stew (for rice, with vegetable oil)	Ogun	67.6	274 (137-489)	704 (267-1200)	9.43 (5.90-16.2)	22.7 (11.5-40.3)
		Abuja	76.8	271 (96.4-468)	898 (473-1580)	10.6 (7.95-24.7)	35.1 (21.4-50.3)*
	Stew (with palm oil)	Ogun	73.1	204 (199-209)	411 (401-422)	10.7 (6.65-13.8)	13.7 (13.6-13.8)
		Abuja	83.0	273 (239-296)	688 (660-706)*	11.9 (9.62-20.2)	27.6 (25.2-30.8)*
	Okra (plain)	Ogun	94.2	120 (118-121)	381 ((374-388)	24.4 (21.9-31.4)	18.3 (18.2-18.4)
		Abuja	88.8	134 (132-137)*	433 (431-436)*	28.2 (23.7-39.2)	33.1 (32.6-33.6)*
	Okra soup	Ogun	93.2	99.7 (89.0-118)	360 (331-412)	35.1 (28.7-42.3)	19.4 (18.5-21.0)
		Abuja	93.2	90.9 (90.0-91.8)	445 (406-484)	42.5 (31.2-57.4)	19.5 (18.3-20.8)
	Ugwu soup (with egusi)	Ogun	68.5	184 (153-290)	456 (344-637)	54.5 (41.2-94.4)	76.6 (64.8-83.1)
		Abuja	68.7	204 (189-226)	457 (422-488)	60.9 (55.6-84.3)	125 (120-133)*
Beef, poultry and eggs	Beef (boiled, in stew)	Ogun	65.7	193 (121-265)	360 (339-372)	16.3 (14.8-23.6)	28.8 (25.2-30.7)
		Abuja	65.4	204 (135-252)	551 (312-850)*	20.3 (13.9-31.2)	37.6 (24.8-55.1)
	Beef (fried, in stew)	Ogun	44.2	329 (257-418)*	565 (352-840)	19.7 (14.8-22.0)	32.3 (29.2-36.7)
		Abuja	55.5	238 (231-245)	552 (540-565)	24.5 (18.3-27.6)	46.4 (45.9-46.9)*

Table 4.14: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of macrominerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Potassium	Sodium	Calcium	Magnesium
Beef, poultry, eggs	Ponmo (raw)	Ogun	78.1	8.49 (4.07-10.8)*	15.6 (11.5-19.9)	5.67 (3.11-7.14)	6.45 (3.81-9.46)
		Abuja	81.4	3.09 (2.57-3.60)	15.8 (15.3-16.3)	6.22 (5.66-6.79)	6.66 (6.30-7.03)
	Chicken thigh (fried	Ogun	52.4	286 (283-291)*	236 (153-281)	33.1 (28.0-38.3)	31.7 (29.1-35.0)
		Abuja	32.7	197 (178-217)	554 (535-573)*	37.5 (33.1-43.9)	53.2 (49.6-56.8)*
	Turkey wing (fried)	Ogun	47.9	282 (268-305)	291 (268-315)	37.9 (31.6-40.9)	43.3 (39.6-46.7)
		Abuja	39.1	242 (241-244)	763 (657-869)*	41.6 (36.4-45.8)	59.2 (53.8-64.7)*
	Egg (boiled)	Ogun	74.0	170 (161-177)*	153 (135-170)	45.2 (38.8-48.4)	15.4 (14.3-16.5)
		Abuja	76.0	103 (96.8-109)	150 (143-158)	50.8 (45.5-58.3)	19.5 (19.2-19.7)*
Fish	Shawa (fried)	Ogun	40.6	718 (567-869)*	319 (267-377)	61.4 (55.6-68.5)	69.7 (48.9-95.8)
		Abuja	41.7	582 (573-591)	323 (312-334)	58.6 (55.1-62.5)	68.6 (67.0-70.2)
	Cray fish (roasted)	Ogun	9.0	1080 (1070-1090)*	1390 (1370-1410)*	159 (135-187)	485 (477-492)*
		Abuja	12.6	748 (704-791)	917 (762-1070)	180 (150-213)	381 (358-395)
	Panla (roasted)	Ogun	74.0	281 (164-409)	391 (339-446)	34.5 (30.3-44.7)	120 (117-124)
		Abuja	36.0	829 (819-838)*	581 (576-587)*	40.8 (32.7-46.9)	132 (129-136)*
Oil seeds	Ground nut (roasted)	Ogun	2.25	831 (822-841)*	571 (555-587)*	44.7 (42.4-46.9)	203 (198-207)
		Abuja	3.36	671 (523-830)	300 (222-505)	46.2 (40.6-48.5)	299 (196-375)*
	Ground nut (boiled)	Ogun	39.2	343 (281-394)	366 (336-447)*	41.3 (40.9-43.9)	130 (114-140)
		Abuja	42.2	305 (258-335)	197 (110-296)	44.7 (38.6-49.5)	170 (148-209)*
	Coconut (fresh)	Ogun	41.5	363 (323-398)*	22.0 (16.5-30.7)	15.1 (10.2-22.8)	86.6 (75.5-101)
		Abuja	42.3	259 (170-314)	26.9 (20.3-36.4)	18.0 (12.4-24.0)	111 (90.4-132)*

Values are presented as mean (min.-max.); nd – not detected (value is below limit of detection (LOD)). LOD (mg/100 g sample) = 0.207, 0.251, 0.178 and 0.162 for K, Na, Ca and Mg, respectively.

* Significantly higher than the mean value available for other location at $p < 0.05$.

4.20 Comparison of mean concentrations of microminerals in some Nigerian foods analysed 'as consumed' in Ogun State and Abuja

The differences between the mean concentrations of Cu, Mn, Fe and Zn in 46 similar food samples separately collected from Ogun State and Abuja are presented in Table 4.15. Nine food groups were also involved in this comparison, which are exactly as those involved in the comparison highlighted above for some macrominerals. Only 14 food samples from Ogun State and Abuja cutting across all the food groups, except legumes and legume products and oil seeds, had their mean Cu contents being significantly different ($p< 0.05$) from each other. The mean Mn levels of 22 food samples were significantly different ($p< 0.05$) when the 46 samples from Ogun State an Abuja were compared. These food samples included 8 out of the 9 food groups involved in this comparison; 'oil seeds' group was the one excluded. Similarly, 19 and 22 food samples had their mean contents of Fe and Zn, respectively being significantly different ($p< 0.05$) between samples individually collected from Ogun State and Abuja. All food groups, except the oil seeds, had one or more samples with different mean Fe and Zn levels. On a general note, within the samples that were significantly different from each other, the mean levels of Cu and Mn were higher in more samples from Abuja than Ogun State, whereas mean Fe and Zn contents were higher in more samples from Ogun State than Abuja.

Table 4.15: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' in Ogun State and Abuja

Food group	Individual food sample	Location	Moisture	Copper	Manganese	Iron	Zinc
Tubers, starches and their products	Fufu (cooked)	Ogun	69.4	0.147 (0.035-0.260)	0.234 (0.042-0.440)	0.966 (0.279-1.80)	0.253 (0.069-0.427)
		Abuja	66.5	0.108 (0.036-0.201)	0.106 (0.057-0.192)	0.603 (0.228-1.14)	0.183 (0.780-0.373)
	Sweet potato (fried)	Ogun	55.2	0.191 (0.187-0.195)	0.191 (0.189-0.193)	0.633 (0.625-0.642)	0.120 (0.116-0.124)
		Abuja	50.9	0.322 (0.273-0.372)*	0.729 (0.520-0.938)*	2.14 (1.55-2.72)*	0.485 (0.276-0.694)*
	Amala (yam powder, cooked)	Ogun	75.3	0.209 (0.118-0.433)	0.328 (0.102-0.473)*	2.55 (1.41-3.29)*	0.465 (0.276-0.714)*
		Abuja	77.8	0.234 (0.196-0.252)	0.128 (0.097-0.180)	1.22 (1.09-1.33)	0.278 (0.238-0.394)
	Pounded yam	Ogun	73.2	0.214 (0.196-0.231)	0.386(0.309-0.482)*	2.76 (2.25-3.51)*	0.435 (0.400-0.474)*
		Abuja	68.3	0.192 (0.145-0.283)	0.098 (0.086-0.111)	0.733 (0.520-1.09)	0.252 (0.197-0.363)
	Yam (white, fried)	Ogun	53.9	0.291(0.145-0.419)	0.295 (0.085-0.681)	2.95 (0.828-5.12)	0.585 (0.283-1.12)
		Abuja	55.5	0.305 (0.290-0.320)	0.306 (0.296-0.316)	1.45 (1.43-1.46)	0.550 (0.540-0.560)

Table 4.15: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Copper	Manganese	Iron	Zinc
Tubers, starches and their products	Eba (white gari)	Ogun	72.9	0.151 (0.095-0.313)	0.395 (0.102-0.605)*	2.30 (0.625-3.39)*	0.261 (0.096-0.583)
		Abuja	71.9	0.072 (0.065-0.078)	0.145 (0.128-0.161)	0.953 (0.866-1.04)	0.137 (0.122-0.152)
	Plantain (mature ripe, fried)	Ogun	41.5	0.251 (0.120-0.455)	0.175 (0.138-0.193)	2.27 (2.15-2.43)*	0.317 (0.209-0.433)
		Abuja	46.9	0.294 (0.272-0.316)	0.229 (0.217-0.241)*	0.716 (0.702-0.729)	0.821 (0.745-0.897)*
Cereals and cereal products	Rice (Long grain, boiled)	Ogun	69.5	0.256 (0.140-0.386)	0.237 (0.030-0.502)	1.98 (1.23-3.58)	0.449 (0.209-0.969)*
		Abuja	72.0	0.189 (0.111-0.324)	0.099 (0.056-0.177)	1.34 (1.06-1.90)	0.254 (0.164-0.450)
	Jollof Rice	Ogun	65.2	0.116 (0.082-0.167)	0.047 (0.016-0.102)	2.14 (1.95-2.43)	0.257 (0.230-0.273)
		Abuja	66.7	0.159 (0.126-0.183)	0.221 (0.205-0.235)*	2.09 (2.00-2.16)	0.407 (0.360-0.440)*
	Maize (yellow, roasted)	Ogun	44.3	0.418 (0.216-0.837)*	0.357 (0.154-0.823)	1.66 (1.02-3.31)	1.56 (1.28-2.06)
		Abuja	41.9	0.183 (0.120-0.258)	0.440 (0.304-0.613)	1.22 (0.862-1.80)	1.16 (0.776-1.53)

Table 4.15: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Copper	Manganese	Iron	Zinc
Cereals products	Tunwo (white maize, cooked)	Ogun	79.7	0.124 (0.119-0.129)*	0.268 (0.258-0.278)*	1.46 (1.34-1.57)*	0.539 (0.519-0.560)*
		Abuja	77.0	0.054 (nd-0.095)	0.048 (0.027-0.076)	0.643 (0.410-0.911)	0.16 (0.131-0.181)
	Bread (white)	Ogun	31.5	0.409 (0.244-0.641)	0.914 (0.410-1.65)	3.47 (1.29-6.74)	1.20 (0.640-1.75)*
		Abuja	29.3	0.409 (0.328-0.545)	0.764 (0.561-1.00)	2.00 (1.08-3.03)	0.797 (0.677-0.944)
	Agidi (white maize, cooked)	Ogun	87.5	0.166 (0.078-0.293)*	0.910 (0.037-0.190)	0.800 (0.239-1.70)	0.209 (0.110-0.431)
		Abuja	85.5	0.099 (0.046-0.144)	0.049 (0.024-0.086)	0.487 (0.144-0.798)	0.206 (0.153-0.292)
	Semovita (cooked)	Ogun	77.4	0.224 (0.107-0.411)	0.263 (0.175-0.429)	1.02 (0.452-1.86)	0.248 (0.121-0.443)
		Abuja	75.8	0.113 (0.067-0.154)	0.390 (0.216-0.635)	0.768 (0.353-1.25)	0.328 (0.182-0.552)
Legumes and legume products	Bean (Drum, plain, boiled)	Ogun	64.5	0.413 (0.356-0.448)	1.05 (0.640-1.38)	2.66 (2.01-3.25)*	1.33 (0.942-1.66)
		Abuja	64.0	0.318 (0.188-0.496)	0.555 (0.388-0.771)	1.48 (1.27-1.64)	1.09 (0.952-1.18)
	Tofu (fried)	Ogun	45.5	0.788 (0.663-0.944)	1.82 (1.56-2.13)*	6.44 (5.34-8.16)	2.33 (2.06-2.64)
		Abuja	63.4	0.701 (0.686-0.716)	1.33 (1.31-1.35)	9.06 (8.85-9.26)*	2.86 (2.64-3.07)*

Table 4.15: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Copper	Manganese	Iron	Zinc
Fruits	Banana (ripe)	Ogun	74.7	0.195 (0.118-0.341)	0.501 (0.173-1.07)	0.755 (0.290-1.25)	0.298 (0.188-0.521)
		Abuja	76.3	0.157 (0.124-0.177)	0.279 (0.146-0.588)	0.520 (0.291-0.645)	0.254 (0.234-0.272)
	Cucumber	Ogun	96.7	0.045 (0.031-0.079)	0.059 (0.028-0.117)	0.200 (0.106-0.273)	0.111 (0.092-0.133)*
		Abuja	96.6	0.035 (0.026-0.050)	0.049 (0.036-0.067)	0.167 (0.124-0.213)	0.090 (0.079-0.099)
	Orange	Ogun	90.5	0.090 (0.054-0.161)*	0.035 (0.031-0.045)	0.295 (0.042-0.591)	0.087 (0.024-0.254)
		Abuja	88.5	0.053 (0.031-0.095)	0.078 (0.023-0.116)*	0.218 (0.073-0.400)	0.054 (0.018-0.146)
	Pawpaw	Ogun	88.2	0.110 (0.033-0.198)	0.043 (0.026-0.057)	0.521 (0.272-0.723)	0.132 (0.075-0.192)
		Abuja	90.9	0.047 (0.031-0.070)	0.048 (0.022-0.059)	0.257 (0.104-0.441)	0.077 (0.064-0.086)
	Pineapple	Ogun	82.9	0.145 (0.058-0.267)	0.713 (0.327-1.03)	0.752 (0.423-1.13)	0.076 (0.037-0.118)
		Abuja	86.3	0.092 (0.048-0.139)	0.353 (0.172-0.455)	0.468 (0.221-0.976)	0.114 (0.045-0.194)
	Water melon (fruit without seed)	Ogun	92.7	0.062 (0.031-0.134)	0.036 (nd-0.060)	0.351(0.152-0.634)*	0.087 (0.054-0.113)
		Abuja	94.1	0.036 (0.021-0.046)	0.045 (0.029-0.059)	0.186 (0.130-0.244)	0.067 (0.050-0.094)

Table 4.15: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Copper	Manganese	Iron	Zinc
Fruits	Apple (wine)	Ogun	83.7	0.098 (nd-0.214)	0.078 (0.023-0.138)	0.582 (0.223-0.780)	0.126 (0.040-0.347)
		Abuja	83.9	0.126 (0.070-0.167)	0.215 (0.085-0.315)	1.09 (0.411-1.45)	0.125 (0.047-0.176)
	Apple (green)	Ogun	84.7	0.079 (nd-0.179)	0.068 (0.027-0.147)	0.411 (0.122-0.616)*	0.158 (0.044-0.299)*
		Abuja	84.8	0.090 (0.079-0.100)	0.068 (0.067-0.069)	0.093 (0.090-0.096)	0.031 (0.024-0.032)
	Pear apple (European pear)	Ogun	82.2	0.076 (0.047-0.105)	0.064 (0.055-0.073)	0.486 (0.422-0.549)*	0.284 (0.210-0.358)*
		Abuja	86.2	0.053 (0.051-0.054)	0.088 (0.081-0.095)*	0.316 (0.274-0.357)	0.145 (0.143-0.147)
Leafy and fruity vegetables	Garden egg (light yellow)	Ogun	92.2	0.130 (0.129-0.131)*	0.050 (0.048-0.051)	0.317 (0.312-0.321)*	0.175 (0.170-0.180)*
		Abuja	93.0	0.081 (0.064-0.101)	0.136 (0.088-0.209)*	0.228 (0.133-0.286)	0.101 (0.078-0.128)
	Garden egg (green)	Ogun	91.7	0.168 (0.122-0.230)	0.150 (0.091-0.228)	0.466 (0.278-0.690)	0.200 (0.154-0.254)
		Abuja	92.0	0.214 (0.188-0.239)	0.201 (0.188-0.215)	0.383 (0.367-0.399)	0.177 (0.172-0.182)
	Cabbage (raw)	Ogun	91.8	0.025 (0.017-0.029)	0.255 (0.244-0.265)	0.505 (0.490-0.520)	0.305 (0.291-0.318)*
		Abuja	93.4	0.037 (0.026-0.061)	0.199 (0.117-0.344)	0.351 (0.185-0.700)	0.166 (0.118-0.267)

Table 4.15: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Copper	Manganese	Iron	Zinc
Condiments, sauces and soups	Locust bean (raw)	Ogun	61.2	0.765 (0.758-0.772)	1.97 (1.90-2.04)	2.05 (1.97-2.12)	1.88 (1.84-1.91)
		Abuja	67.4	0.632 (0.312-0.882)	2.74 (1.87-3.77)	2.48 (0.534-4.97)	1.72 (1.23-2.17)
	Stew (for rice, with vegetable oil)	Ogun	67.6	0.118 (0.057-0.193)	0.055 (0.029-0.082)	3.92 (1.07-8.64)	0.226 (0.073-0.522)
		Abuja	76.8	0.165 (0.089-0.237)	0.306 (0.084-0.726)*	1.98 (1.20-3.18)	0.369 (0.172-0.700)
	Stew (with palm oil)	Ogun	73.1	0.077 (0.064-0.090)	0.072 (0.068-0.076)	1.32 (1.21-1.43)	0.312 (0.286-0.338)
		Abuja	83.0	0.237 (0.140-0.395)*	0.161 (0.118-0.213)*	2.20 (1.90-2.35)*	0.427 (0.392-0.455)*
	Okra (plain)	Ogun	94.2	0.174 (0.152-0.191)	0.197 (0.194-0.199)*	1.17 (1.08-1.25)*	0.207 (0.177-0.237)
		Abuja	88.8	0.282 (0.269-0.294)*	0.140 (0.135-0.145)	0.923 (0.903-0.943)	0.426 (0.420-0.431)*
	Okra soup	Ogun	93.2	0.065 (0.043-0.080)	0.241 (0.213-0.258)*	0.496 (0.464-0.532)	0.295 (0.225-0.335)*
		Abuja	93.2	0.060 (0.055-0.064)	0.182 (0.174-0.189)	0.909 (0.818-0.999)*	0.138 (0.116-0.159)
	Ugwu soup (with egusi)	Ogun	68.5	0.285 (0.134-0.561)	0.428 (0.193-0.666)	3.88 (2.03-7.20)	1.17 (0.684-1.43)
		Abuja	68.7	0.559 (0.343-0.704)*	0.921 (0.828-1.01)*	3.30 (3.12-3.48)	1.16 (0.776-1.27)

Table 4.15: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Copper	Manganese	Iron	Zinc
Beef, poultry and eggs	Beef (boiled, in stew)	Ogun	65.7	0.342 (0.279-0.448)	0.143 (0.112-0.189)	2.89 (2.73-3.16)	4.51 (2.49-7.09)
		Abuja	65.4	0.402 (0.249-0.523)	0.255 (0.124-0.441)	3.26 (2.16-4.98)	4.41 (2.15-6.76)
	Beef (fried, in stew)	Ogun	44.2	0.669 (0.448-1.06)	0.103 (0.066-0.153)	4.76 (1.76-7.12)	7.41 (4.36-13.2)*
		Abuja	55.5	0.557 (0.540-0.574)	0.152 (0.150-0.154)	4.34 (4.15-4.54)	2.94 (2.89-2.99)
	Ponmo (raw)	Ogun	78.1	0.218 (0.103-0.371)	0.040 (nd-0.099)	0.964 (0.314-1.35)	0.214 (0.139-0.241)
		Abuja	81.4	0.177 (0.163-0.190)	0.060 (0.053-0.067)	0.745 (0.704-0.785)	0.180 (0.164-0.195)
	Chicken thigh (fried)	Ogun	52.4	0.495 (0.422-0.608)	0.514 (0.412-0.595)	2.87 (2.18-3.39)	3.60 (3.29-3.99)
		Abuja	32.7	1.01 (0.897-1.13)*	0.844 (0.739-0.950)*	7.53 (6.35-8.71)*	4.84 (3.86-5.81)
	Turkey wing (fried)	Ogun	47.9	0.645 (0.589-0.722)	0.419 (0.325-0.523)	5.75 (5.40-6.33)	4.65 (3.49-5.24)
		Abuja	39.1	0.869 (0.824-0.915)*	0.582 (0.567-0.598)*	6.84 (6.09-7.59)	3.68 (3.56-3.79)
	Egg (boiled)	Ogun	74.0	0.285 (0.202-0.382)	0.036 (0.028-0.042)	1.76 (1.56-1.91)	1.21 (1.03-1.37)*
		Abuja	76.0	0.462 (0.429-0.495)*	0.081 (0.076-0.086)*	1.77 (1.60-1.93)	0.795 (0.718-0.871)

Table 4.15: Moisture content (g/100g) and comparison of concentrations (mg/100g edible portion on fresh weight basis) of microminerals in some Nigerian foods 'as consumed' in Ogun State and Abuja (continued)

Food group	Individual food sample	Location	Moisture	Copper	Manganese	Iron	Zinc
Fish	Shawa (fried)	Ogun	40.6	0.554 (0.280-0.852)	0.026 (nd-0.034)	2.37 (1.34-3.18)*	2.47 (1.42-3.78)*
		Abuja	41.7	0.436 (0.423-0.448)	0.055 (0.048-0.062)*	1.27 (1.22-1.32)	1.20 (1.12-1.29)
	Cray fish (roasted)	Ogun	9.0	2.40 (2.31-2.49)*	0.937 (0.934-0.940)*	16.0 (15.6-16.5)*	6.55 (6.24-6.84)*
		Abuja	12.6	2.03 (1.86-2.19)	0.510 (0.434-0.568)	9.39 (8.77-10.0)	5.08 (4.89-5.34)
	Panla (roasted)	Ogun	74.0	0.545 (0.495-0.612)	0.095 (0.076-0.113)	0.848 (0.616-1.02)	1.82 (1.51-2.20)
		Abuja	36.0	1.03 (0.985-1.06)*	0.177 (0.159-0.194)*	1.82 (1.73-1.91)*	2.54 (2.38-2.69)*
Oil seeds	Ground nut (roasted)	Ogun	2.25	0.822 (0.788-0.856)	1.13 (1.08-1.19)	8.96 (8.24-9.67)	3.35 (3.30-3.41)
		Abuja	3.36	1.26 (0.983-1.88)	2.20 (1.03-3.09)	7.56 (5.18-9.20)	3.32 (2.00-4.92)
	Ground nut (boiled)	Ogun	39.2	0.589 (0.406-0.760)	1.07 (0.858-1.24)	3.07 (1.65-3.94)	2.26 (1.99-2.47)
		Abuja	42.2	0.824 (0.771-0.854)	1.37 (1.05-1.93)	3.17 (1.81-5.08)	1.78 (1.55-2.16)
	Coconut (fresh)	Ogun	41.5	0.693 (0.453-0.880)	1.08 (0.862-1.38)	4.23 (3.95-4.48)	1.45 (1.38-1.63)
		Abuja	42.3	0.91 (0.815-1.08)	1.61 (1.26-2.00)	4.77 (4.07-5.48)	1.84 (1.42-2.60)

Values are presented as mean (min.-max.); nd – not detected (value is below limit of detection (LOD)). LOD (mg/100 g sample) = 0.016, 0.017, 0.014 and 0.015 for Cu, Mn, Fe and Zn, respectively.

* Significantly higher than the mean value available for other location at $p < 0.05$.

4.21 Moisture content and mean concentrations of cadmium and lead in some Nigerian foods analysed 'as consumed'

Table 4.16 presents the mean levels of contaminants (cadmium (Cd) and lead (Pb)) in 205 individual food samples representing 76 different food items in 10 food groups. These foods were sub-samples from those collected from sub-locations in Abuja. Cadmium was non-detectable in 26 out of the 76 different food items analysed at the level of 0.06 µg/100g sample. White bread had the highest mean Cd level of 3.23 µg/100g edible portion on fresh weight basis. Other foods highly contaminated with Cd were cray fish (2.90 µg/100g), yellow tiger nut (2.04 µg/100g), brown tiger nut (1.90 µg/100g), 'eko/agidi' (1.42 µg/100g), boiled chicken thigh (1.16 µg/100g) and plain drum bean (1.14 µg/100g). The food groups mostly contaminated with Cd were cereals and cereal products; beef, poultry and eggs; and fish.

On the other hand Pb was not detectable in 12 food items at the level of 0.44 µg/100g sample. The foods highly contaminated with Pb were fura (53.5 µg/100g), boiled yellow maize (44.3 µg/100g), 'fufu/akpu' (29.6 µg/100g), white bread (26.0 µg/100g), roasted yellow maize (23.7 µg/100g), roasted 'panla' (22.6 µg/100g), 'eko/agidi' (22.1 µg/100g), roasted ground nut (17.6 µg/100g), donkwa (14.5 µg/100g), white porridge bean (14.1 µg/100g), fried plantain (11.9 µg/100g) and 'masa' (10.6 µg/100g).

Table 4.16: Moisture content (g/100g) and concentrations(µg/100g edible portion on fresh weight basis) of cadmium and lead insome Nigerian foods 'as consumed'

Food Group	Individual food sample	N	Moist-ure	Cadmium	Lead
Tubers, starches and their products	Fufu (cooked)	5	66.5	0.272 (nd-0.570)	29.6 (5.76-55.3)
	Sweet potato (fried)	2	50.9	0.125 (0.100-0.130)	7.59 (7.56-7.62)
	Amala (yam powder, cooked)	3	77.8	0.194 (0.174-0.226)	nd
	Pounded yam (cooked)	2	68.3	0.118 (nd-0.174)	nd
	Yam (white, fried)	2	55.1	nd	6.97 (6.53-7.42)
	Eba (white gari, cooked)	2	71.9	0.117 (nd-0.174)	nd
	Plantain (mature ripe, fried)	2	46.9	nd	11.9 (11.7-12.1)
Cereals and cereal products	Rice (Long grain, boiled)	7	72.0	0.915 (nd-2.82)	9.36 (nd-21.9)
	Masa (rice cake, fried)	2	56.4	0.242 (0.223-0.261)	10.6 (9.90-11.4)
	Garogaro (rice, beans, boiled; along with fresh sauce)	2	67.1	0.191 (0.184-0.198)	4.74 (4.35-5.12)
	Maize (yellow, roasted)	3	44.3	1.04 (0.105-2.84)	23.7 (12.5-53.8)
	Maize (yellow, boiled)	2	72.7	1.02 (0.708-1.32)	44.3 (38.4-50.2)
	Tunwo (white maize, cooked)	2	79.7	0.600 (0.299-0.926)	3.52 (nd-7.88)
	Tunwo (rice, cooked)	2	77.0	0.175 (nd-0.273)	4.01 (2.49-5.65)
	Bread (white)	3	29.3	3.23 (1.03-6.29)	26.0 (nd-60.6)

Table 4.16: Moisture content (g/100g) and concentrations(µg/100g edible portion on fresh weight basis) of cadmium and lead insome Nigerian foods 'as consumed' (continued)

Food Group	Individual food sample	N	Moist-ure	Cadmium	Lead
Cereals and cereal products	Agidi (white maize, cooked)	4	85.5	1.42 (0.090-4.40)	22.1 (3.79-74.2)
	Semovita (cooked)	4	75.8	0.143 (nd-0.247)	8.83 (1.89-18.6)
	Donkwa	2	18.0	0.562 (0.423-0.722)	14.5 (nd-21.8)
	Fura	2	57.5	0.109 (nd-0.207)	53.5 (47.9-64.2)
Legumes and legume products	Bean pottage (White bean)	3	70.0	0.766 (nd-1.93)	14.1 (2.60-19.9)
	Bean (Drum, plain, boiled)	4	64.0	1.14 (nd-2.86)	5.72 (nd-8.32)
	Bean (White, plain, boiled)	2	60.9	0.090 (nd-0.110)	nd
	Moi-moi (White bean, cooked)	2	68.5	0.090 (nd-0.106)	2.96 (2.28-3.63)
	Okpa bean (boiled)	2	59.6	0.125 (0.110-0.130)	6.57 (6.29-6.85)
	Moi-moi (Okpa bean, cooked)	4	64.1	0.091 (nd-0.185)	3.77 (0.718-7.12)
	Kunu (gyada)	2	89.6	0.090 (nd-0.112)	0.903 (0.610-1.20)
Fruits	Banana (ripe)	3	76.3	0.569 (nd-1.17)	8.56 (nd-15.4)
	Carrot (raw)	2	88.1	0.128 (0.102-0.136)	3.12 (1.84-4.40)
	Cucumber	2	96.6	0.116 (nd-0.171)	2.66 (1.77-3.55)
	Orange	6	88.5	nd	0.676 (nd-1.84)
	Pawpaw	4	90.9	nd	1.49 (nd-3.84)
	Pineapple	4	86.3	nd	3.99 (0.720-6.09)
	Water melon (fruit without seed)	4	94.1	nd	1.99 (0.800-3.35)
	Guava	4	84.0	nd	3.79 (3.13-4.63)
	Date	2	10.0	nd	3.73 (3.22-4.25)
	Tiger nut (yellow, raw)	2	47.5	2.04 (1.94-2.15)	3.56 (3.48-3.65)
	Tiger nut (brown, raw)	2	14.5	1.90 (1.87-1.92)	2.17(2.09-2.25)

Table 4.16: Moisture content (g/100g) and concentrations(µg/100g edible portion on fresh weight basis) of cadmium and lead insome Nigerian foods 'as consumed' (continued)

Food Group	Individual food sample	N	Moist-ure	Cadmium	Lead
Fruits	Apple (wine)	2	83.9	0.198 (nd-0.313)	3.45 (nd-5.29)
	Apple (green)	2	84.8	nd	nd
	Apple (wine/green)	2	83.0	nd	nd
	Pear apple (European pear)	2	86.2	nd	0.680 (0.520-0.790)
Leafy and fruity vegetables	Garden egg (light yellow)	4	93.0	nd	1.52 (nd-3.04)
	Garden egg (green)	2	92.0	nd	2.12 (1.70-2.53)
	Green bell pepper (raw)	3	94.4	0.075 (nd-0.105)	6.06 (2.04-9.51)
	Green peas (raw)	4	74.7	0.076 (nd-0.122)	4.28 (nd-9.38)
	Green bean (raw)	2	92.1	nd	1.28 (nd-2.13)
	Cabbage (raw)	2	93.4	nd	3.64 (1.16-7.76)
Condiment, sauces and soups	Locust bean	4	67.4	0.155 (nd-0.255)	4.01 (nd-7.60)
	Stew (for rice, with vegetable oil)	8	76.8	0.137 (nd-0.255)	1.15 (nd-6.11)
	Stew (for tunwo masara)	1	81.9	nd	1.80 (1.68-2.05)
	Stew (with palm oil)	2	83.0	nd	nd
	Stew (for masa)	1	86.6	nd	0.979 (0.949-1.01)
	Groundnut soup	1	84.1	nd	1.30 (1.21-1.39)
	Water leaf soup	2	67.1	nd	nd
	Okra (plain)	2	88.8	0.096 (0.065-0.104)	nd
	Okra Soup	2	93.2	nd	nd
	Ugwu soup (with egusi)	2	68.7	0.102 (0.077-0.115)	1.87 (nd-3.30)
	Tete soup (with egusi)	3	74.2	nd	6.26 (nd-15.3)
	Efo riro (tete)	2	79.9	0.614 (0.558-0.670)	2.85 (2.53-3.16)
	Soko soup (with egusi)	2	70.2	0.219 (0.210-0.228)	2.14 (1.88-2.39)

Table 4.16: Moisture content (g/100g) and concentrations(µg/100g edible portion on fresh weight basis) of cadmium and lead insome Nigerian foods 'as consumed' (continued)

Food Group	Individual food sample	N	Moist-ure	Cadmium	Lead
Milk and milk products	Nunu	2	92.3	nd	1.83 (1.19-2.46)
	Cheese (local)	2	63.4	nd	7.88 (7.61-8.14)
Beef, poultry and eggs	Beef (boiled, in stew)	6	65.4	0.383 (nd-1.16)	4.62 (1.70-8.19)
	Beef (fried, in stew)	2	55.5	0.156 (0.145-0.167)	0.941 (0.896-0.986)
	Ponmo (raw)	2	81.4	nd	3.01 (2.63-3.40)
	Chicken thigh (boiled)	2	61.4	1.16 (0.306-2.01)	2.56 (nd-4.68)
	Turkey wing (boiled)	2	58.1	0.133 (0.104-0.146)	1.16 (nd-1.87)
	Egg (boiled)	2	76.0	nd	nd
Fish	Shawa (fried)	2	41.7	0.378 (0.362-0.394)	2.71 (2.59-2.83)
	Cat fish (roasted)	5	13.9	0.350 (nd-0.98)	6.31 (0.820-13.8)
	Cray fish (roasted)	2	12.6	2.90 (1.98-4.01)	nd
	Panla (roasted)	2	36.0	0.760 (0.624-0.896)	22.6 (20.2-25.0)
Oil seeds	Ground nut (roasted)	3	3.36	0.239 (0.13-0.42)	17.6 (10.1-24.3)
	Ground nut (boiled)	3	42.2	0.093 (nd-0.160)	7.34 (1.96-10.5)
	Cashew nut (roasted)	2	4.78	0.091 (0.071-0.110)	9.61 (3.83-20.5)
	Coconut (fresh)	2	42.3	0.110 (nd-0.159)	4.27 (2.63-5.91)

Values are presented as mean (min.-max.); N – number of samples; nd – not detected (value is below limit of detection (LOD)). LOD (µg /100 g sample) = 0.120 and 0.880for Cd and Pb, respectively.

4.22 Concentrations of serum copper, zinc and iron of participants disaggregated by main location

Table 4.17 shows the concentrations of copper, zinc and iron in the serum of study population in Ogun State and Abuja. Three hundred and thirty-six (336) volunteers, among the 2027 total population, participated in the blood donation for the heavy metal analyses. The serum copper, zinc and iron ranged from 40 to 250 μg/dL, 31 to 188 μg/dL and 32 to 229 μg/dL, respectively for the whole participants. Their mean values per location and overall, fell within the normal ranges specified for adult human subjects by World Health Organization (WHO) (WHO, 1996). The levels of chromium and nickel were not detectable at the levels of 4.0 and 10.0 μg/dL, respectively, in the serum of the whole volunteers. The levels of manganese, cadmium and lead were also not detectable at the levels of 3.0, 1.0 and 8.0 μg/dL, respectively, in the whole blood samples of the whole group.

4.23 Concentrations of serum copper, zinc and iron of participants disaggregated by sub-location

The levels of the detectable metals are reported in Table 4.18, disaggregated by location. This result further shows that the mean levels of copper in all the sub-locations fell within WHO normal range, except for that of participants in Dutsen Alhaji, which fell below. The mean levels of zinc were within WHO normal range for participants in all Ogun State sub-locations, except for those in Abeokuta-South that fell below. However, in Abuja, only the mean levels of zinc of participants from Kuje and Abaji were within normal WHO range; others' mean values were higher than the normal range. As for the serum levels of iron in the study population, the mean levels in all the sub-locations were within normal range of WHO, except for those from Ilaro community, which was below.

Table 4.17: Concentrations (µg/dL) of serum copper, zinc and iron of participants from Ogun State and Abuja

Metal	Location	Number of participants (n)	Mean ± SD (Min, Max)	Reference value[a]
Copper	Ogun	205	113.9 ± 37.1 (40.0, 250.0)	
	Abuja	131	97.8 ± 27.9 (50.0, 240.0)	
	All	336	107.6 ± 34.7 (40.0, 250.0)	80.0-140.0
Zinc	Ogun	205	96.0 ± 34.6 (31.0, 174.0)	
	Abuja	131	106.9 ± 41.6 (34.0, 188.0)	
	All	336	100.3 ± 37.8 (31.0, 188.0)	80.0-110.0
Iron	Ogun	205	89.3 ± 43.8 (32.0, 229.0)	
	Abuja	131	106.3 ± 31.7 (40.0, 222.0)	
	All	336	95.9 ± 40.3 (32.0, 229)	80.0-120.0

[a]WHO (1996)

Table 4.18: Concentrations (µg/dL) of serum copper, zinc and iron of participants from various locations in Ogun State and Abuja

Location	Number of participants (n)	Copper – Mean ± SD (Min, Max)	Zinc – Mean ± SD (Min, Max)	Iron – Mean ± SD (Min, Max)
OGUN	**205**	**113.9 ± 37.1 (40.0, 250.0)**	**96.0 ± 34.6 (31.0, 174.0)**	**89.3 ± 43.8 (32.0, 229.0)**
Ipokia	35	118.0 ± 53.2 (40.0, 230.0)	106.7 ± 42.0 (38.0, 173.0)	101.9 ± 74.3 (40.0, 229.0)
Ilaro	51	130.8 ± 31.3 (60.0, 190.0)	104.5 ± 33.1 (54.0, 174.0)	66.5 ± 29.6 (40.0, 141.0)
Abeokuta South	45	106.7 ± 31.1 (70.0, 220.0)	77.8 ± 25.1 (31.0, 152.0)	88.3 ± 28.6 (32.0, 201.0)
Ilisan	46	89.8 ± 19.3 (60.0, 140.0)	95.1 ± 34.6 (36.0, 160.0)	101.6 ± 36.5 (40.0, 165.0)
Ijebu-Ode	28	128.9 ± 31.1 (90.0, 250.0)	98.0 ± 30.8 (43.0, 143.0)	96.3 ± 29.4 (50.0, 201.0)
ABUJA	**131**	**97.8 ± 27.9 (50.0, 240.0)**	**106.9 ± 41.6 (34.0, 188.0)**	**106.3 ± 31.7 (40.0, 222.0)**
Kwali	43	93.0 ± 33.1 (50.0, 240.0)	120.7 ± 41.5 (38.0, 186.0)	98.4 ± 25.9 (45.0, 154.0)
Kuje	30	100.0 ± 31.3 (50.0, 200.0)	90.9 ± 40.2 (34.0, 188.0)	110.2 ± 34.0 (44.0, 167.0)
Abaji	37	110.5 ± 16.3 (80.0, 160.0)	96.6 ± 37.1 (34.0, 166.0)	118.2 ± 35.1 (40.0, 222.0)
Dutsen Alhaji	9	68.9 ± 11.7 (50.0, 80.0)	113.1 ± 51.4 (46.0, 168.0)	105.3 ± 18.3 (76.8, 132.4)
Karu	12	91.7 ± 8.3 (70.0, 100.0)	125.3 ± 31.0 (84.0, 176.0)	101.9 ± 74.3 (40.0, 229.0)
Reference value[a]		**80.0-140.0**	**80.0-110.0**	**80.0-120.0**

[a]WHO (1996)

4.24 Concentrations of serum copper, zinc and iron of participants disaggregated by main location and gender

Considering the gender of the study population, some dynamics can be further obtained from Table 4.19 as regards the concentrations of copper, zinc and iron in their serum samples. The male participants in both main locations had their mean serum levels of copper, zinc and iron being within normal WHO ranges. For the women, the mean metal levels were also within normal ranges except for the mean copper levels of those from Abuja, which fell below.

Table 4.19: Concentrations (µg/dL) of serum copper, zinc and iron of participants from Ogun State and Abuja disaggregated by gender

Gender	Location	Number of participants (n)	Copper – Mean ± SD (Min, Max)	Zinc – Mean ± SD (Min, Max)	Iron – Mean ± SD (Min, Max)
Men	Ogun	61	106.6 ± 25.2 (50.0, 160.0)	98.1 ± 33.3 (35.0, 174.0)	83.0 ± 34.5 (32.0, 160.0)
	Abuja	58	98.3 ± 30.4 (50.0, 200.0)	103.8 ± 39.4 (34.0, 180.0)	103.6 ± 30.2 (40.0, 167.0)
	All	119	102.5 ± 28.1 (50.0, 200.0)	100.9 ± 36.4 (34.0, 180.0)	93.0 ± 34.0 (32.0, 167.0)
Reference value[a]			**80.0-110.0**	**80.0-110.0**	**80.0-120.0**
Women	Ogun	144	116.9 ± 40.8 (40.0, 250.0)	95.2 ± 35.3 (31.0, 173.0)	91.9 ± 47.0 (40.0, 229.0)
	Abuja	73	97.4 ± 25.8 (50.0, 240.0)	109.5 ± 43.3 (34.0, 188.0)	108.5 ± 32.9 (44.0, 222.0)
	All	217	110.4 ± 37.6 (40.0, 250.0)	100.0 ± 38.7 (31.0, 188.0)	97.5 ± 43.4 (40.0, 229.0)
Reference value[a]			**110.0-140.0**	**80.0-110.0**	**80.0-120.0**

[a]WHO (1996)

4.25 Concentrations of serum copper, zinc and iron of participants disaggregated by age and gender

The study population was categorized into normal, low and high groups using the WHO normal range values. They have been so classified based on their age and gender as presented in Table 4.20. Considering all the age brackets, most of the women fell below the normal WHO range for copper, whereas most of the men were within the normal range stipulated. In most of the age brackets for both men and women, majority of the study population had serum zinc levels above the WHO normal range. As for levels of iron in the study population, higher percentage of men was within normal range compared with the women of the same age bracket.

Table 4.20: Concentrations (µg/dL) of serum copper, zinc and iron of participants from Ogun State and Abuja disaggregated by age and gender

Age (years)	Gender	Category by WHO reference values[a]	Copper		Zinc		Iron	
			Freq-uency	(%)	Freq-uency	(%)	Freq-uency	(%)
18-25	Men	Normal	19	55.9	14	41.2	10	29.4
		Low	4	11.8	9	26.5	14	41.2
		High	11	32.4	11	32.4	10	29.4
	Women	Normal	19	30.2	15	23.8	26	41.3
		Low	32	50.8	23	36.5	17	27.0
		High	12	19.0	25	39.7	20	31.7
26-35	Men	Normal	19	47.5	9	22.5	19	47.5
		Low	9	22.5	15	37.5	14	35.0
		High	12	30.0	16	40.0	7	17.5
	Women	Normal	20	23.0	25	28.7	35	40.2
		Low	51	58.6	32	36.8	36	41.4
		High	16	18.4	30	34.5	16	18.4
36-45	Men	Normal	16	55.2	10	34.5	15	51.7
		Low	4	13.8	8	27.6	10	34.5
		High	9	31.1	11	37.9	4	13.8
	Women	Normal	15	34.9	13	30.2	21	48.8
		Low	25	58.1	14	32.6	14	32.6
		High	3	7.0	16	37.2	8	18.6
46-55	Men	Normal	6	54.5	2	18.2	8	72.7
		Low	2	18.2	4	36.4	2	18.2
		High	3	27.3	5	45.5	1	9.1
	Women	Normal	7	33.3	5	23.8	8	38.1
		Low	12	57.1	8	38.1	9	42.9
		High	2	9.5	8	38.1	4	19.0
56-60	Men	Normal	1	25.0	1	25.0	1	25.0
		Low	1	25.0	1	25.0	2	50.0
		High	2	50.0	2	50.0	1	25.0
	Women	Normal	-	-	-	-	1	33.3
		Low	2	66.7	-	-	2	66.7
		High	1	33.3	3	100	-	-
61-70	Men	Normal	1	100	-	-	-	-
		Low	-	-	1	100	-	-
		High	-	-	-	-	1	100

[a] WHO Reference values: Copper: 80-110 µg/dL (men), 110-140 µg/dL (women); Zinc 80-110 µg/dL (all); Iron: 80-120 µg/dL (all).

CHAPTER FIVE

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

5.1 Discussion

5.1.1 Frequency distribution of participants in study sites

The two main locations studied were well represented in the survey (Figure 4.1). The total number studied far exceeded the minimum sample size calculated for the study. This further strengthens the reliability of the decisions taken from the outcome of survey.

5.1.2 Characteristics of food samples analysed

Several commonly consumed foods at various part of Nigeria can be found on the list presented in Table 4.1. This makes the results provided to be applicable to majority of the populace. The scientific name provided and the individualized food sampling technique will enhance adaptability at various parts of the country for dietary intake assessment as well as within the West-African sub region. The number of food samples and items analysed in this study is known to be the highest ever reported at a go in Nigeria, after the extensive reports of Onianwa *et al*. (2000 and 2001) on 80 Nigerian food items.

5.1.3 Recoveries of macrominerals and microminerals from food items

Quality Control (QC) is a major aspect of instrumental analysis that is essential to affirm the precision and accuracy of results so obtained. This was the essence of setting up recovery experiment as one key aspect of the 5 QC checks designed primarily for the metal analyses in the food samples. The experiment was set up at low and high levels of the metals to check the extent of accuracy at both extremes. The results were quite excellent with 94-108% recoveries across the various food groups (Table 4.3). These results put a high level of confidence on the myriad set of data

generated from the food samples relative to the macromineral, micromineral and heavy metal concentrations. This pattern of result is similar to the reports of Gimou *et al*. (2014) and Akinyele and Shokunbi (2015b), suggesting that the 'precautions' taken enhanced the quality of data reported.

5.1.4 Recovery experiments on whole blood and serum samples

Researches on heavy metals in especially human blood samples have been very challenging over the years. The limited resource earmarked for researches in developing countries like Nigeria is the major underlying factor for these related challenges. The challenges around this aspect of research are in two phases which are: a. getting standardized method and equipment for processing of the blood or serum samples before instrumental analysis; b. having access to highly sensitive instrument(s) for the analysis of the digest for specific metals. This is most likely one of the reasons why limited reports are actually available relative to Nigerian subjects. This background informed the design of the experiment to get a standardized digestion method for the heavy metal analysis, using simple equipment like hotplate and fume cupboard, along with readily available glass wares, filter paper and concentrated acids. The outcome was somewhat fruitful with a conclusion of nitric acid: hydrochloric acid (1:3) mixture as the overall best among the trials (Tables 4.3a and 4.3b). This acid mixture was used to process all the whole blood and serum samples of study participants.

5.1.5 Distribution of study participants by gender and religious practice

The gender distribution of the study population is somewhat fair (Figure 4.2). This seems to have been affected by the usage of primary health facility as the place of convergence in some communities. Furthermore, in some cases, household heads had gone to work by the time the location was being surveyed.

The two main religions (Christianity and Islam) practiced across Nigeria were well represented at both main locations (Ogun State and Abuja). They have both been cooperative in past surveys organized by the Government of Nigeria, non-governmental organizations and research groups as evidenced in reports (NPC and ICF Macro, 2009; NPC and ICF International, 2014; NNHS, 2015).

5.1.6 Distribution of study participants by highest level of education

Relative to the basic socioeconomic characteristics of the participants, majority (83% minimum) of the respondents were educated up to secondary school level in both locations (Figure 4.4). This accounted in part to the reason why the starting parts of the questionnaire was self-administered to most of them, while those having low level of education or no formal education were assisted by research assistants to enable appropriate participation in the survey.

5.1.7 Occupation of household heads and spouses

The occupation of an individual or household head tends to affect the level of income, purchasing power, food and nutrition security of such individual as well as the dependents. The occupation of participants in OgunState and Abuja reflects the area being surveyed. Most parts surveyed in Ogun State were urban or semi-urban in nature, thus most were white collar income earners as different from the semi-urban settings surveyed in Abuja, where large percentage were farmers. This distribution further affected their Socio-Economic Status (SES). The spouses that were civil servants, traders and private company workers in Ogun State and Abuja had great likelihood of adding resources to their households, thereby enhancing their household SES, food and nutrition security. In the case of spouses of participants in Abuja, majority being famers could positively affect the finance as well as dietary diversification of their households.

5.1.8 Socio-economic status of participants

The characteristics of the accommodation an individual reside and the functional items available within the same place tend to reflect the SES of such individual. So these items have been carefully rated and scored, and their cumulative scores have been used to classify the study population into low, middle and high SES (Figure 4.6). On the face value, Abuja is usually rated as a territory for high class members of the society. However, the outcome of this survey showed that it depends on which part of Abuja and the occupation of the individuals that determine status, to a large extent. The occupation of the household heads and spouses in Ogun State seemed to have

enhanced their SES. The urban and semi-urban nature of most of the territories studied in Ogun could have also enhanced the SES of the people.

It is somehow surprising to find Ilaro and Ilisan (classified as semi-urban) being the major contributors to the high SES of Ogun State participants (Figure 4.7). This could be due to the fact that most of the participants were actually of high classes in Ilaro and Ilisan. In the same vein, it might be that those that participated in Ijebu-Ode were of the lower class of that sub-location. On another hand, it was somehow surprising to find Kuje and Kwali as the major contributors to the low SES of Abuja; since they are also semi-urban areas.

5.1.9 Age distribution and Body Mass Index (BMI) of participants

The age distribution of participants revealed that most of them fell within the reproductive age (18-45 years – in the case of women) as well as the work force (18-60 years) of our nation (Table 4.4). Thus, their health status should be of concern to the public, especially if poor; as it indirectly translates to national productivity.

The prevalence of chronic under-nutrition (2.5 %) reported here is quite low compared with national statistics of 11% (NPC and ICF International, 2014) or 7.4% (NNHS, 2015) for women of reproductive age. Though the prevalence of chronic under nutrition in this study population is low, it is still of concern because majority of them were women (Table 4.5). So, it is needful to find means of helping those in this category as the prevalence can increase with time if appropriate care is not taken. Chronic malnutrition has great implications for morbidity and low productivity of individuals so affected. This condition is associated with higher prevalence of low birth weight (in women), predisposing to higher infant mortality (Maziya-Dixon*et al*., 2004). On the other hand, a cumulative of about 50% of the participants in Ogun State and Abuja were noted to be overweight or obese. This is of great concern as obesity is a significant risk factor for the development of several non-communicable diseases including cardiovascular disease, hypertension and stroke, diabetes mellitus, various forms of cancers and economic consequences (Stern, 1995; Popkin, 2002; Guh*et al*., 2009; Dee*et al*., 2014). Thus these people require urgent nutritional intervention to slow down and or reverse the trend quickly. The disaggregation of the BMI status by

gender and location was helpful to clarify that virtually all the communities were affected with the burden of malnutrition (under- or over nutrition).

5.1.10 Dietary diversities of participants in Ogun State and Abuja

Starchy staples; vitamin A rich fruits and vegetables; other fruits and vegetables; meat and fish; and dark green leafy vegetables were the food groups consumed by those classified to have low Dietary Diversity Score (DDS) (Figures 4.8 and 4.9); that is, those consuming foods from less than 6 food groups per day. Having higher percentage of Ogun State participants being with low DDS seems quite ironic, especially bearing in mind that this population had more people with higher SES (Figures 4.6 and 4.9). This somewhat connotes that high SES does not absolutely translate to food/nutrition security and or good nutritional health. Nutrition education might play a huge role in bridging this gap. This ironic trend is clearly displayed by results presented in Figures 4.7 and 4.10 for Ilaro, Ilisan and Dutsen Alhaji. Though a large proportion of them had high SES, a high percentage still had low DDS. However, Abeokuta-South and Kuje showed normal trend of high SES going along with low percentage being with low DDS. This is in line with the submissions of Ruel (2002) and Rashid*et al*. (2006). The other locations that their trends are not so obvious (such as Abaji) could have adopted some other coping strategies to influence their diet diversification.

5.1.11 Frequency of consumption of various foods by all participants

The frequency of consumption of the staples such as rice (49%), maize and its products (with pap/akamu having 27.2%), then cassava and its products (with garri having 17.6%) and wheat and its products (with white bread having 15.1%, noodles having 8.9% and biscuit having 21.1%) on daily or more than four times weekly consumption pattern (Table 4.6) shows that the foods were most preferred or most readily available and affordable. In a way, these foods count a lot towards the food and nutrition security of the participants. Relative to the DDQ, this same set of foods (starchy staples) were indicated as being consumed by over 98% of the participants a day prior to the day of interview. However, Food Frequency Questionnaire (FFQ) rating is far more robust and specific than the DDQ rating.

Furthermore, similar high frequency of fish (55.6%) and beef (37.8%) consumption, on daily or more than four times weekly consumption pattern; is notable in the DDQ result. These two food groups, though combined, had the fourth highest frequency of consumption in the DDQ result (Figure 4.8). The consumption pattern of dark green leafy vegetables, milk and milk products, eggs, legumes, nuts and seed were still reasonably correlated between the FFQ and DDQ results (Table 4.6).

Orange, water melon and banana were the highly preferred fruits, with orange being most preferred. Preference for vegetables also ranged from 'ewedu' having the highest, followed by 'ugu' and 'soko' and then 'tete/aleiho'.

5.1.12 Disaggregated frequency of consumption of various foods by participants in Ogun State and Abuja

The frequency of consumption of specific food in the two main locations was further clarified in the disaggregated results shown in Table 4.7. Some foods were more preferred in Ogun State whereas others were more preferred in Abuja. This showed some regional and or cultural differences between the two main population groups. This unique difference peculiar to each location could partly be the reason why more fruits and vegetables were consumed by participants in Abuja compared with those in Ogun State. The farming practices of majority of household heads and spouses in Abuja might also be a contributing factor to this dietary pattern, which eventually made Abuja to have overall lesser percentage of people classified with low DDS. Generally, Abuja participants seem to enjoy more of swallow foods along with the vegetables (Table 4.7).

5.1.13 Mean daily mineral and heavy metal intakes of study participants

The dietary mineral intake pattern of the study population shows that the foods analysed in the study cover most of the kinds of foods taken by them (Tables 4.8 and 4.9). Most of the foods tend to supply the appropriate levels of minerals to the participants, up to the RecommendedDaily Intake (RDI) levels for most of them. The level of potassium intake is generally low compared with RDI as reported in some other studies within (Ijarotimi and Keshinro, 2008) and outside Nigeria (Geleijnse*et al*., 2003; Głąbska*et al*., 2016) on adult populations.

Sodium intake by these adult populations is far beyond recommendation (especially for those in Ogun State) perhaps due to the increase in consumption of processed foods, which have high sodium contents. Furthermore, the large proportion of participants (44.5%) patronizing resturant services (a day prior to dietary recall data collection) can be a contributing factor; as most outlets as such greatly spice their foods with bullion cubes and similar items, in a stance to attract customers. High sodium consumption has been implicated as major risk factors for the development of hypertension (Eckel*et al*., 2014). Hypertension has been a major public health concern in developed countries and recently in developing countries, as it is one of the prominent risk factors for mortality, as well as stroke, heart, and renal diseases (Kalaitzidis and Bakris, 2010; James*et al*., 2014).

Report has shown that even a slight lowering of the mean blood pressure of a population can yield a downturn in cardiovascular morbidity and mortality (Chobanian*et al*., 2003). Thus, in an effort to effectively prevent and or manage hypertension, various organization including the American Heart, Lung and Blood Institute and European Society of Hypertension have recommended intakes of diet low in sodium and fat, and high intake of fruits and vegetables (Chobanian *et al*., 2003; Mancia*et al*., 2013; James *et al*., 2014). Thus there is an urgent need to advocate to the study population, especially those in Ogun State and by extension Nigerian adults to make deliberate efforts to reduce sodium intakes and increase potassium intakes, thereby their enhancing cardiovascular health. This is similar to the passionate plea of Noubiap*et al*. (2015) for a positive outcome in Sub-Saharan Africa.

The low levels of calcium seen in this report portrays that there may be some other significant sources of calcium not fully covered in the analysis of foods consumed by participants. Calcium intake from water was not also captured in the analysis and intake estimation, which could be another contributing factor to the low calcium estimated here.

The mean copper intake (Table 4.8) by participants in this study somewhat close to 2.64 mg/day reported by Onianwa *et al*. (2001) for Nigerian adults, 2.7 mg/day reported by Bowen (1981) for Germans and 1.5-3.1 mg/day (Bowen, 1981) for Britons; and very much higher than 1.51 mg/day reported by Lewis and Buss (1988)

for elderly Britons and 1.23 mg/day reported by Nagy (1987) for Hungarian adults. However, the value is lower than the 5.8 mg/day reported for Indians by Bowen (1981) and 8.0 mg/day reported by Udoessien and Aremu (1991) for Nigerian adults. The mean copper intakes were still very much sufficient across the locations and both genders (Tables 4.9 – 4.11).

Furthermore, the mean intake of zinc by Nigerian adults that participated in this study (Table 4.8) is similar to the reports of Lewis and Buss (1988) – 9.05 mg/day, Hussein and Bruggerman (1997) – 8.54 mg/day, Murphy*et al*. (1975) – 8.11 mg/day, and Mbofung and Atinmo (1980) – 7.3 mg/day for adults in Britain, Egypt, USA and Nigeria, respectively. On another hand, reports from adults in Canada (Bowen, 1981) – 11-20 mg/day, India (Bowen, 1981) – 16 mg/day, Nigeria (Udoessien and Aremu, 1991) – 18 mg/day, Nigeria (Onianwa *et al*., 2001) – 15.8, and USA (Pennington and Gunderson (1987) have shown very high values. The mean zinc intakes obtained from this study across locations and genders are all within the RDI (Tables 4.9 – 4.11).

The mean dietary intakes of sodium, copper, manganese and iron that were significantly higher ($p< 0.05$) for participants in Ogun State compared with those in Abuja (Table 4.9) is a reflection of the variation in the dietary patterns and practices of participants at the two main locations. It can be further deduced that the variation is a reflection of the differences in their frequency of consumption of various Nigerian foods (Table 4.7). Similar explanation could be for the mean dietary intake of calcium that is significantly higher ($p< 0.05$)among Abuja participants.

The comparison done relative to the ULs/PTDIs shows that the consumers are safe with respect to potassium, calcium, magnesium, copper, manganese, iron, zinc, cadmium and lead intakes (Tables 4.8 – 4.10) and majority of the participants at both locations are safe from metal intake toxicities.

Participants' mineral intakes were minimally affected by gender as reflected in the amounts consumed by participants in Ogun State and Abuja of both genders (Table 4.11). However, there seems to be some unique foods consumed by male participants in Abuja, which significantly increased ($p< 0.05$)their heavy metal intake; though the increased level was still within safety limit. It can be laudable to identify such food

component and educate such set of consumers on the need to reduce or eliminate such component, and thereby enhance safety.

5.1.14 Moisture content and mean concentrations of macrominerals in some Nigerian foods analysed 'as consumed'

The wide range of distribution of potassium (K) in Nigerian foods 'as consumed' reported in Table 4.12 is quite similar to several K contents of food reported in some parts of the globe; though some differences really exist with some of the food items (Chekri *et al*., 2012; Gimou *et al*., 2014). The levels of K in powdered milk reported here (14400 mg/Kg) is slightly higher than the value (12800 mg/Kg) reported by Gimou *et al*. (2014). However, the K content of the milk was still surpassed by that of some soft bony smoked fish such as stock fish and '*ebolo*' fish. Other rich sources of K include plantain chips, boiled walnut, cocoa product, cornflakes and date. These rich sources show Nigerian rich biodiversity and possible dietary support in the management of hypertension among inhabitants of Nigeria.

The myriads of food analysed indicated that the mean levels of sodium (Na) in prepared Nigerian foods vary widely from location to location. The high levels of Na in stews and soups from various food vendors and restaurants across the sub-locations make it very necessary to take extra caution while consuming foods outside the home. The low levels of Na in the fruits, leafy and fruity vegetables (except cole slaw), and most cereals and tubers normally prepared without addition of salt also provide a wide pool for the selection of food items in the appropriate management of high blood pressure. The lower levels of sodium in unprocessed foods than in processed food was also reported by Thompson*et al*. (2008) as well asTanase*et al*.(2011).

Daily dietary intake of calcium (Ca) is very essential for bone and teeth health as well as modulation of various biochemical pathways in humans. Apart from the stock fish (530 mg/100g) that is very high in Ca content, local foods like 'donkwa' (111 mg/100g) and boiled bread fruit (71 mg/100g) are noteworthy as they tend to compete favourably with milk (72.1 mg/100g) in Ca levels. The trend noticed here relative to milk and fish having high Ca contents was similarly reported by Leblanc *et al*. (2005) and Chekri *et al*. (2012). However, promotion of indigenous food items like donkwa and bread fruit will be useful especially at locations with low income pattern.

The nuts and several fish in Nigerian dishes have been notable to contain high levels of magnesium (Mg) as reported in Table 4.13. The Mg levels reported are also similar to those reported by other studies from various parts of the world (Jodral-Segado*et al*., 2003; Chekri *et al*. 2012). Peanuts and cashew nuts were also reported to be high in Mg contents (USDA, 2002).

5.1.15 Moisture content and mean concentrations of microminerals in some Nigerian foods analysed 'as consumed'

The mean levels of Cu obtained from this study showed that most cooked leafy vegetables are poor sources of this essential mineral (Table 4.14). Foods of animal origin such as beef, poultry and eggs, and cat fish as well as plant sources like boiled walnut, legume and legume products are excellent sources of Cu. These can be rightly combined in various menus to adequately nourish consumers. Considering these results manganese level is relatively higher in food items of plant origin, compared with those from animal origin. This potentiates the antioxidant capacities of the foods along with the phytochemicals present in them. Animal proteins are known to yield more bioavailable Fe compared with the plant sources, due to some inherent factors in plants (Byrd-Bredbenner *et al*., 2013). However, the various plants that can possibly supply Fe, if well combined can adequately nourish individual. Low income earners and strict vegetarians that need to make the most of the limited available resources will benefit greatly from these individually reported food contents of Fe, especially in the forms 'as consumed'. Zinc is abundant mainly in animal products. However, it is noteworthy that cashew nut and other nuts are rich in Zn.

5.1.16 Comparison of mean concentrations of macrominerals and microminerals in some Nigerian foods analysed 'as consumed' in Ogun State and Abuja

The outcome of comparison done between the metal contents of similar foods collected from Ogun State and Abuja showed some unique dynamics (Tables 4.15 and 4.16). Though several of our foods across the country with similar origins have very similar mineral contents, many also differ. The possible reasons for the differences include differences in agro-climatic conditions (soil composition, geographic and climatic variations), methods of preparation; types of container used for preparation;

and differences in the sources of water used. Furthermore, it can be deduced that Ogun soil viz-a-viz the food crops produced on it are richer in K, Na, Fe and Zn compared with that of Abuja. On the other hand, the soil viz-a-viz the food crops produced from Abuja are richer in Ca, Mg, Cu and Mn compared with that of Ogun State. Generally, legume crops and oil seeds showed minimal differences between Ogun State and Abuja. Further studies will be needful to clarify the realities of these specific variations.

5.1.17 Moisture content and mean concentrations of cadmium and lead in some Nigerian foods analysed 'as consumed'

The results obtained on Cd and Pb contents of the commonly consumed foods from Abuja show that our foods are somewhat contaminated (Table 4.17). However, the level of contamination with Pb (84.2%) is quite higher compared with that of Cd (65.8%). The types of food contaminated with Cd tend to give a bit of concern. White bread, cray fish 'eko/agidi', boiled chicken and drum bean, which are regularly consumed by some on daily basis; poses a risk of bioaccumulation in consumers, over a given period of time. Thus, it will be expedient to re-evaluate the food processing vessels, equipment and protocol to clearly establish the source of contamination as soon as possible, for consumer safety.

Lead is known to induce reduced cognitive development and intellectual performance in children and increased blood pressure and cardiovascular disease in adults (Commission of the European Communities, 2002). Blood lead level had been well associated with the consumption of spinach, oyster, potatoes and seeweed. Transfer of Pb to plant had been notable from the top soil, where Pb is usually retained (DePieri*et al*., 1997; ATSDR, 2005). In the case of Pb, which had higher rate and level of contamination in the food, much more attention is needful to quickly trace and perhaps eliminate the major sources of this contaminant in the food chain. This will on a long run enhance the safety of all, as health hazards are being minimized. Lead contamination could have been from the farming equipment and tools, or farm inputs such as fertilizers and pesticides, or water used in cooking, or pots used in cooking, or a part contribution from all of these factors.

5.1.18 Concentrations of serum copper, zinc and iron in participants blood disaggregated by location, age and gender

The mean serum copper (Cu) levels (Table 4.17) in this study per location (113.9 µg/dL – Ogun, 97.8 µg/dL – Abuja) and overall (107.6 µg/dL) are within normal range values (80-140 µg/dL) of WHO for healthy adults (WHO, 1996). The values reported here are slightly above those reported by Schuhmacher*et al*. (1994) (84.1 µg/dL) and Clark *et al*. (2007) (91.1 µg/dL) but similar to the report of Rahil-Khazen*et al*. (2000) (108.9 µg/dL). These mean Cu levels were somewhat dependent on gender, with men having 102.5 µg/dL and women having 110.4 µg/dL (Table 4.19). Previous studies cited here reported the trend of having higher serum Cu in women. The use of oral contraceptives was suggested to have partly contributed to this trend (Rahil-Khazen *et al*., 2000). A variation of mean Cu levels was seen across the sub-locations studied (Table 4.18). This may be due to the varied proportion of gender represented. It might also be due to the long-term dietary practice of the participants from these sub-locations. The classification relative to WHO normal range as presented in Table 4.20 helps to put individuals that participated in this study in a sharper focus. Majority of the women fell below the benchmark (110-140 µg/dL) set by WHO for normal adult women.

The overall mean serum Zn was 100.3 µg/dL (Table 4.17). This value varies widely (77.8-125.3 µg/dL) across sub-locations studied (Table 4.18). However, it is not affected by gender difference (Table 4.19). The overall mean serum Zn is slightly above the values reported by Rahil-Khazen *et al*. (2000) (86.9 µg/dL) and Clark *et al* 2007 (81.0 µg/dL); but below the report of Schuhmacher *et al*. (1994) (113 µg/dL). However, it falls within the range (80-110 µg/dL) set by WHO (WHO, 1996). The Zn status of the study population is a reflectionof their food consumption pattern. Over 90% of the total participants reported to have consumed meat or fish previous day to their DDQ survey (Figure 4.8). The dietary pattern viz-a-viz serum Zn status seem to positively affect majority of the study population, across various age brackets: most of them either have normal or high serum Zn status (Table 4.20). Low serum zinc levels induced by rapid growth, pregnancy and lactation can lead to zinc deficiency if these increased needs are not met in the specific individuals involved(Maziya-Dixon *et al*., 2004).

The overall mean level of iron (Fe) was 95.9 µg/dL, a value which widely varied (66.5-118.2µg/dL) across sub-locations and insignificantly ($p > 0.05$) across gender (Tables 4.17 – 4.19). The lack of significant difference across gender deviates from previous reports in which women had significantly lower ($p < 0.05$)serum Fe compared with men of similar age range (Zacharski*et al*., 2000; Clark *et al*., 2007). The deviant trend noted in this study was somewhat resolved when the serum Fe levels were compared between men and women across various age brackets (Table 4.20). More women were noted to have low serum Fe compared with men of the same age. The overall mean Fe level reported falls within the ranges – 78.2-117.3 µg/dL and 61.5-167.6 µg/dL – reported by Iyengar (1998) and MedlinePlus (2016), respectively as well as that of WHO (1996) – 80-120 µg/dL. However, the mean serum Fe value obtained in this study is slightly above 84.9 µg/dL reported by Clark *et al*. (2007) and far below 119.6 µg/dL reported by Rahil-Khazen *et al*. (2000). These differences could be due to racial differences, variations in dietary patterns of the studied groups of people in this report compared with other above highlighted reports or a contribution of both factors.

5.1.19 Concentrations of serum chromium and nickel,blood manganese, cadmium and lead of participants

The levels of chromium (Cr) and nickel (Ni) were not detectable at the levels of 4.0 and 10.0 µg/dL, respectively, in the serum of the whole volunteers. Since the allowable range by WHO is 0.014-0.015 and <0.1 µg/dL, respectively for Cr and Ni, it cannot be concluded whether the individuals surveyed are safe or not. Similarly, the levels of manganese (Mn), cadmium (Cd) and lead (Pb) were also detectable at the levels of 3.0, 1.0 and 8.0 µg/dL, respectively, in the whole blood samples of all participants. According to by WHO, the stipulated safety levels for Mn, Cd and Pb are 0.8-1.2, 0.03-0.12 and 5.0-15.0 µg/dL, respectively (WHO, 1996). Thus it can only be said that the individuals are somewhat safe relative to Pb toxicity. Such conclusion cannot be drawn for Mn or Cd toxicity in the participants. The sensitivity of the flame atomic absorption spectrometer (FAAS) used for the analyses of these heavy metals in blood samples was a constraint of this study.

5.2 Conclusions

Digestion of blood or serum samples with nitric acid: hydrochloric acid (1:3) mixture can enable an accurate determination of heavy metals from the digested samples. Analysis of the various metals in the food samples produced excellent recoveries, which further increases the reliability of the results reported from food samples.

The knowledge of the occupation of the participants was useful to better understand their dietary patterns and dynamics around their Socio-Economic Status (SES). The location of individuals does not absolutely determine their SES.

About 50% of the study population were over nourished (overweight and obese), seemingly portraying effect of nutrition transition or other nutrition-related problems; and giving lots of concerns due to the related risk factors and likely future economic implication for management of such people.

High SES does not absolutely translate to food/nutrition security and/or good nutritional status. Nutrition education might play a huge role in bridging the gap.

The mean daily intakes of the studied minerals and heavy metals by the participants showed that most of them consume appropriate amount of the minerals and somewhat low levels of the heavy metals. Most of the participants had very high intake of sodium and low intake of potassium and calcium in their diets. Therefore there is an urgent need educate the participants, especially those within Ogun State on the need to reconsider their choices of food consumed in a way to drastically lower sodium intakes and improve on potassium and calcium intakes.

The mean serum copper, zinc and iron levels of the study population fell within the normal range stipulated by World Health Organization. However, disaggregation of the data revealed various interesting dynamics relative to differences in age, gender and location of participants. The levels of chromium and nickel (in serum); manganese, cadmium and lead (in blood) were not detectable in specimens of the volunteers. Considering the sensitivity of the FAAS used for the analysis of the serum and blood samples of participants, only safety relative to lead can be assured.

Commonly consumed Nigerian foods are very rich in potassium, sodium, calcium, magnesium, copper, manganese, iron and zinc. However, they also contain contaminants in the form of cadmium and lead widely distributed. This portrays an urgent need to trace and identify major sources of the contaminants in the food processing chain, in an effort to enhance safety of consumers.

The levels of minerals in Nigerian foods are not exactly the same across the nation. Agro-climatic conditions (geographic, climatic and soil variations); methods of preparation; types of container used for preparation; and differences in the sources of water used, among others could account for the differences in mineral composition of Nigerian foods. The individual food analyses are thus essential when estimating the true intake of the population.

The fact that the food items were analysed and presented 'as consumed' widens the adaptability of the results obtained for use in Nigeria Food Composition Table and mineral intake assessments.

5.3 Recommendations

1. Participants and by extension Nigerian adults should be strongly encouraged to improve on the amounts of fruits and vegetables consumed on daily basis in an effort to improve the potassium intakes. Salt intakes should be drastically reduced as well to control the very high sodium intakes currently observed.
2. There is a great need to mobilize efforts to get the attention of major stakeholders at the national level to sponsor the analysis of Nigerian foods, especially to fill up the current data gaps and get a truly national Food Composition Table (FCT) inaugurated.
3. More nutritionists should be trained and encouraged to research on specific data gaps that need filling, bearing in mind that we dearly need the accurate information.There is a need to similarly analyse the levels of vitamins in commonly consumed Nigerian foods, on a large scale.
4. There is a need to make more concerted efforts in analysing other food items consumed in Nigeria which were not covered in this study, especially those commonly consumed in the south-eastern and south-southern Nigeria.

5. There is a need to develop software program that can integrate these new data and previous ones easily in a way to carry out dietary intakes assessment of individuals as well as populations within the country.
6. Commonly consumed Nigerian foods are very rich in potassium, sodium, calcium, magnesium, copper, manganese, iron and zinc. However, they also contain contaminants in the form of cadmium and lead widely distributed. This portrays a need to design more specific studies to trace and identify major sources of the contaminants in the food processing chain, so as to devise effective strategies to minimize or possibly eliminate them and thereby enhance safety of consumers.

5.4 Contribution to knowledge

Considering the outcomes of this study, the following contributions to knowledge are notable:

1. The study has reported the widest coverage of Nigerian foods on the concentrations of macrominerals, microminerals and heavy metal (especially as consumed) as at date.
2. The high quality data provided from this study can be easily adapted in the compilation of the Nigerian Food Composition Table of which these set of data are greatly lacking.
3. This study has provided empirical data on the perspective that Nigerian adults are consuming far more than needed sodium and less than required potassium, which calls for urgent intervension to forestall future health harzards.
4. This report has also established a strong positive relationship between the food frequency questionnaire and dietary diversity questionnaire in the evaluation of the dietary pattern of adult population.
5. The location of individuals does not absolutely determine their Socio-Economic Status (SES). High SES does not absolutely translate to food/nutrition security and/or good nutritional health status. Nutrition education might play a huge role in bridging the gap.
6. This study established a viable wet digestion method for the analysis of metals in whole blood and serum samples.

7. The study provided data on the heavy metal contents of the blood samples of large number of Nigerian adults across wide locations.
8. Generally, the data supplied in this study will go a long way in alleviating the challenges of nutritional epidemiologists, especially those interested in Nigerian adults. The data provided will greatly assist in food safety analysis, which can by extension enhance health conditions and facilitate the export of some agricultural produce from Nigeria.

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APPENDICES

Appendix 1. Dietary Pattern Survey Questionnaire