| Serial No. | UGWU, JOSHUA IZUCHUKWU  
PG/MSC/12/62460 |
|-----------|-------------------|
| Author 1  | UGWU, JOSHUA IZUCHUKWU  
PG/MSC/12/62460 |
| Author 2  | UGWU, JOSHUA IZUCHUKWU  
PG/MSC/12/62460 |
| Author 3  | UGWU, JOSHUA IZUCHUKWU  
PG/MSC/12/62460 |
| Title:    | DIETARY INTAKE AND MICRONUTRIENT STATUS OF  
SCHOOL CHILDREN IN ENUGU- SOUTH L.G.A., ENUGU  
STATE, NIGERIA |
| Keyword:  | DEPARTMENT OF MEDICAL BIOCHEMISTRY |
| Category: | FACULTY OF BASIC MEDICAL SCIENCE |
| Publisher:| |
| Publication Date: | |
| Signature: | Ebere Omeje |

Digitally Signed by: Content manager’s Name
DN : CN = Webmaster’s name
O= University of Nigeria, Nsukka
OU = Innovation Centre
DIETARY INTAKE AND MICRONUTRIENT STATUS OF SCHOOL CHILDREN IN ENUGU- SOUTH L.G.A., ENUGU STATE, NIGERIA.

BY

UGWU, JOSHUA IZUCHUKWU
PG/MSC/12/62460

A PROJECT SUBMITTED TO THE DEPARTMENT OF MEDICAL BIOCHEMISTRY FACULTY OF BASIC MEDICAL SCIENCE UNIVERSITY OF NIGERIA ENUGU CAMPUS

APRIL, 2016
DIETARY INTAKE AND MICRONUTRIENT STATUS OF SCHOOL CHILDREN IN ENUGU- SOUTH L.G.A., ENUGU STATE, NIGERIA.

BY
UGWU, JOSHUA IZUCHUKWU
PG/MSC/12/62460

A PROJECT SUBMITTED TO THE
DEPARTMENT OF MEDICAL BIOCHEMISTRY
FACULTY OF BASIC MEDICAL SCIENCE
UNIVERSITY OF NIGERIA
ENUGU CAMPUS

IN PARTIAL FULFILMENT OF THE REQUIREMENTS
FOR THE AWARD OF MASTER OF SCIENCE (M.Sc.)
DEGREE IN MEDICAL BIOCHEMISTRY

RESEARCH SUPERVISOR
PROF. I. E. EZEAGU

SPECIAL AREA OF STUDY
MEDICAL BIOCHEMISTRY

APRIL, 2016
DEDICATION

I dedicate this work to Almighty God for his favour over me throughout my studies, and also to my sponsors, siblings, course mates and well wishers.
ACKNOWLEDGMENT

I wish to thank my project supervisor, Prof. I. E. Ezeagu from whom I have learnt a lot. Also I appreciate him for his fatherly love, care and advice.

My special thanks also goes to the head of the Department, Dr. (Mrs.) J. E. Ikekpazu and the entire staff of the Department of Medical Biochemistry for their care and guidance.

I acknowledge with gratitude the analytical assistance of Mr. Ekoh Solomon of Solex Lab. Garriki Awkunanaw. Enugu.

Finally, I acknowledge the immense support of my parents late Chief and Mrs. S. N. Ugwu.
ABSTRACT

This study sought to assess the dietary intake and serum Zinc, Iron and Copper status of primary school children aged 5-13 years living in Enugu-South Local Government Area of Enugu State, Nigeria. This study is a community-based cross-sectional study, adopting multistage random sampling techniques. Dietary intakes of the micronutrients were assessed using the 24 hours dietary recall. The micronutrient intakes of the children were evaluated using dietary requirement intake (DRI) as a reference. Two milliliters (2ml) of non-fasting venous blood were taken from the children for serum micronutrient analysis. Three hundred and thirty (330) children were analyzed for serum micronutrient status; 155 (47%) were male while 175 (53%) were females, with their mean age 8±1.09: The mean micronutrient intakes of the subjects were 4.98 ± 3.7, 4.53 ± 1.63, and 0.42 ± 0.20 mg/d for Fe, Zn and Cu respectively. Only the male group aged 5-9 years met 100% of the DRI for Zn while the 5-9 years females, 10-13 years males and 10-13 years female did not meet up with the DRI for Zn, Fe and Cu. The mean serum micronutrients of the total children were 63.16 ± 18.06, 62.27 ± 17.3 and 69.9 ± 14.99µg/dl for Fe, Zn and Cu respectively. Of the 330 children studied, 32%, 43% and 23% of them seems to deficient in Fe, Zn and Cu respectively. The food intakes of the children did not supply the recommended DRI for school children. There is therefore an urgent need to educate the public on good eating habits and the need for diversification of diets with fruits, vegetables and animal products in order to ensure adequate intake of these essential micronutrients.
### TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Contents</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Title page</td>
<td>i</td>
</tr>
<tr>
<td>Certification</td>
<td>ii</td>
</tr>
<tr>
<td>Dedication</td>
<td>iii</td>
</tr>
<tr>
<td>Acknowledgement</td>
<td>iv</td>
</tr>
<tr>
<td>Abstract</td>
<td>v</td>
</tr>
<tr>
<td>Table of content</td>
<td>vi</td>
</tr>
<tr>
<td>List of Tables</td>
<td>x</td>
</tr>
<tr>
<td>List of Figures</td>
<td>xi</td>
</tr>
</tbody>
</table>

#### CHAPTER ONE

1.0 Introduction - - - - - - - - - - 1
1.1 Iron - - - - - - - - - - 2
1.2 Zinc - - - - - - - - - - 3
1.3 Copper - - - - - - - - - - 4
1.4 Objective - - - - - - - - - - 5
1.4.1 Specific Objective 5

#### CHAPTER TWO

2.0 Literature Review - - - - - - - - - - 6
2.1 Iron - - - - - - - - - - 7
2.1.1 Functions of Iron - - - - - - - - - - 8
2.1.1.1 Iron as Oxygen Carriers - - - - - - - - - - 8
2.1.1.2 Iron in Electron Transport - - - - - - - - - - 8
2.1.1.3 Iron as Antioxidant and Pro-oxidant - - - - - - - - 9
2.1.1.4 Iron in DNA synthesis - - - - - - - 9
2.1.2 Iron Requirements - - - - - - - 9
2.1.3 Iron Absorption - - - - - - - 10
2.1.4 Iron Deficiency - - - - - - - 12
2.1.4.1 Causes of Iron Deficiency - - - - - - - 13
2.1.4.2 Effects of Iron Deficiency - - - - - - - 13
2.1.5 Food Sources - - - - - - - 14
2.2. Zinc - - - - - - - 14
2.2.1 Functions of Zinc - - - - - - - 15
2.2.1.1 Catalytic Role - - - - - - - 15
2.2.1.2 Structural Role - - - - - - - 15
2.2.1.3 Regulatory Role - - - - - - - 16
2.2.2 Zinc Deficiency in Children - - - - - - - 16
2.2.2.1 Impaired growth and Development - - - - - - - 16
2.2.2.2 Impaired Immune System Function - - - - - - - 17
2.2.2.3 Diarrhea - - - - - - - 17
2.2.2.4 Pneumonia - - - - - - - 18
2.2.3 Food Sources - - - - - - - 18
2.3 Copper - - - - - - - 18
2.3.1 Some of the physiological Copper - - - - - - - 19
2.3.1.1 Energy Production - - - - - - - 19
2.3.1.2 Connective Tissue Formation - - - - - - - 19
2.3.1.3 Iron Metabolism - - - - - - - 19
2.3.1.4 Central Nervous System - - - - - - - 20
2.3.1.5 Melanin Formation - - - - - - - 20
<table>
<thead>
<tr>
<th>Section</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.3.1.6 Antioxidant Function</td>
<td>20</td>
</tr>
<tr>
<td>2.3.1.7 Regulation of Gene Expression</td>
<td>21</td>
</tr>
<tr>
<td>2.3.2 Absorption Distribution of Copper</td>
<td>21</td>
</tr>
<tr>
<td>2.3.3 Dietary Sources of Copper</td>
<td>22</td>
</tr>
<tr>
<td>2.3.4 Copper Deficiency</td>
<td>22</td>
</tr>
<tr>
<td>2.3.4.1 Clinical Symptoms of Copper Deficiency</td>
<td>22</td>
</tr>
<tr>
<td><strong>CHAPTER THREE</strong></td>
<td></td>
</tr>
<tr>
<td>3.0 Methodology</td>
<td>25</td>
</tr>
<tr>
<td>3.1 Study Subjects</td>
<td>25</td>
</tr>
<tr>
<td>3.1.1 Study Location</td>
<td>25</td>
</tr>
<tr>
<td>3.1.2 Sample Design</td>
<td>25</td>
</tr>
<tr>
<td>3.2 Ethical Consideration</td>
<td>25</td>
</tr>
<tr>
<td>3.3 Dietary Intake</td>
<td>26</td>
</tr>
<tr>
<td>3.4 Blood Analysis</td>
<td>26</td>
</tr>
<tr>
<td>3.4.1 Preparation of Sample Containers</td>
<td>26</td>
</tr>
<tr>
<td>3.4.2 Digestion of Sera</td>
<td>27</td>
</tr>
<tr>
<td>3.5 Instrumentation</td>
<td>27</td>
</tr>
<tr>
<td><strong>CHAPTER FOUR</strong></td>
<td></td>
</tr>
<tr>
<td>4.0 Results</td>
<td>28</td>
</tr>
<tr>
<td>4.1 Micronutrient Composition of Major Foods and Drinks Consumed by Subjects</td>
<td>29</td>
</tr>
<tr>
<td>4.2 The Serum Micronutrient Indices of Subjects</td>
<td>32</td>
</tr>
</tbody>
</table>
CHAPTER FIVE

5.0 Discussion       - - - - - - - - - - 37
5.1 Conclusion       - - - - - - - - - - 49
Reference            - - - - - - - - - - 50
# LIST OF TABLES

<table>
<thead>
<tr>
<th>Contents</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Table 1: Gender and Age Distribution of Subjects</td>
<td>28</td>
</tr>
<tr>
<td>Table 2: Percentage distribution of food types consumed by children in 24 hours</td>
<td>29</td>
</tr>
<tr>
<td>Table 3: Micronutrient Composition of Major Foods and Drinks</td>
<td>30</td>
</tr>
<tr>
<td>Table 4: Mean Intake Micronutrient of Subjects and Percentage of DRI met</td>
<td>31</td>
</tr>
<tr>
<td>Table 5: Mean Serum Micronutrient Level of Subjects</td>
<td>32</td>
</tr>
<tr>
<td>Table 6: Micronutrient Daily Intakes and the mean Serum Concentration</td>
<td>33</td>
</tr>
<tr>
<td>Table 7: Frequency Distribution of Serum Fe Level</td>
<td>34</td>
</tr>
<tr>
<td>Table 8: Frequency Distribution of Serum Zn Level</td>
<td>34</td>
</tr>
<tr>
<td>Table 9: Frequency Distribution of Serum Cu Level</td>
<td>35</td>
</tr>
</tbody>
</table>
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Contents</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Figure 1:</strong> Distribution of Daily Meal Intake</td>
<td>28</td>
</tr>
<tr>
<td><strong>Figure 1:</strong> Prevalence of Micronutrients Deficiency</td>
<td>36</td>
</tr>
</tbody>
</table>
CHAPTER ONE

1.0 INTRODUCTION

Micronutrients are nutrients that are only needed by the body in minute amounts, which are involved in the production of enzymes, hormones and other substances, helping to regulate growth activity, development and the functioning of the immune and reproductive systems (FAO 2002).

Micronutrient malnutrition is a condition that results from eating diets that lack one or more of micronutrients, that is required by the body for proper growth and development (Allen et al. 2006).

Micronutrient deficiencies have been a major nutritional problem in developing countries like Nigeria and have adversely affected people’s health, performance and income, thereby becoming major impediments to economic development (Bowley 2008).

This condition usually occurs when people cannot afford to diversify their diets with adequate amount of fruits, vegetables or animal source foods that contains large amount of micronutrients. These usually occur with people based mostly on monotonous cereal or fiber based diets (FAO 2002).

Nutritional deficiencies have become more prevalent following economic stress and food insecurities faced by populations in these countries. Most at risk groups include children less than 5 years of age, adolescents, women of childbearing age, particularly the pregnant and lactating, refugees and victims of famine (Dairo et al. 2009).

Micronutrient deficiencies can exist in populations even where the food supply is adequate in terms of meeting energy requirements. In these situations, people are not considered "hungry" in the classical sense, but their diets may be grossly deficient in one or more micronutrients and
they are not aware. It is for these reasons that micronutrient deficiencies have been referred to as "hidden hunger" (Kennedy 2004).

Essential micronutrients includes; iodine, iron, zinc, calcium, selenium, Copper, fluorine and vitamins A, B6, B12, B, B2, B3, and C, D, E (Tulchinsky 2010).

The most recent estimates from FAO indicate that 840 million people do not receive enough energy from their diets to meet their needs. The overwhelming majority of these people - 799 million - live in developing countries. The global toll of people affected by micronutrient deficiency is estimated to be even higher and probably exceeds two billion. Primarily deficiencies of iodine, iron, vitamin A and zinc significantly contribute to chronic diseases and are the major cause of morbidity and mortality in these countries. In addition to the more obvious clinical manifestations, micronutrient malnutrition is responsible for a wide range of non-specific physiological impairments leading to reduced resistance to infections, metabolic disorders and delayed or impaired physical and psychomotor development (Allen et al. 2006).

School aged children are at an increased risk of micronutrient deficiency owing to their increased growth or metabolism, increased energy expenditure combined with decreased meal frequency, reduced maternal attention and parasitic infections (Akeredolu et al. 2011). Improving school children’s nutrition will improve their cognitive function and linear growth.

Nutritional programs in resource poor settings mainly focus on children under the age of five years. This has resulted in limited information on nutritional and micronutrient status of school aged children.
1.1 IRON
Iron deficiency is the most common and widespread nutritional disorder in the world (Onyezili et al. 2003). As well as affecting a large number of children and women in developing countries. Iron deficiency anemia is the most common micro-nutrient problem in the world as it affects more than 2 billion people globally (Onyezili et al. 2003). Over 30% of the world’s population are anemic, many due to iron deficiency. In resource poor areas this is frequently exacerbated by infectious diseases like malaria, HIV/AIDS, hookworm infestation, schistosomiasis and other infections that contribute to the high prevalence of anemia in some area (Onyezili et al. 2003).
Iron deficiency which can lead to anemia, a condition in which the number of red blood cells or their oxygen-carrying capacity is insufficient to meet the physiological needs of the organism which is associated with fatigue, weakness, dizziness and drowsiness. Iron deficiency anemia is associated with adverse health conditions including permanent behavioral and cognitive impairments. Therefore, early detection and prompt treatment are necessary to prevent these complications. Prevalence of iron deficiency anemia varies between countries, affecting 5.4% children in Spain, 30.8% and 22.3% under-five children in Brazil and Nigeria respectively (Munoz et al. 1995).

1.2 ZINC
Zinc is one of the trace mineral essential to human nutrition and metabolism, participating in all biochemical pathways and playing multiple roles in the gene expression, cell development and replication (Hambridge 2000).
Zinc deficiency is largely related to inadequate intake or absorption of zinc from diet such as animal sources, which have the highest zinc content compared to plant foods. Because of these inadequate intakes billions of people are at risk of zinc deficiency. More than 400,000 children
die each year due to zinc deficiency (FAO/WHO 2004). An estimated 17.3% of the global population is at risk of inadequate zinc intake (Maziya et al. 2004). The prevalence of zinc deficiency in sub-Sahara Africa is 50%. According to the 2001-2003 Nigerian Food Consumption and Nutrition Survey, the national prevalence of zinc deficiency among under five was 21% (Maziya et al. 2004). Studies among school children 5-13 years of age from Lagos state Nigeria put the prevalence of zinc deficiency at 21% (Akeredolu et al. 2011).

1.3 COPPER

Copper is one of the essential trace elements in humans, and disorders associated with its deficiency and excess have been reported (Aoki et al. 1996) Menkes (kinky-hair) disease is well-known to be associated with copper deficiency due to an inherited disorder of copper transport from the intestine metabolism, and Wilson disease (hepatolenticular degeneration) is a well-known inherited disorder of cellular copper transport resulting in copper accumulation (Culotta et al. 2000). Acquired copper deficiency is mainly attributable to nutritional deficiency, and may be seen in malnourished low-birth-weight infants, newborns, and small infants. Copper deficiency has also been reported to develop after gastrointestinal surgery, intractable diarrhea, and prolonged parenteral or enteral nutrition. Data are scarce on the level of copper deficiency worldwide. In Lagos Nigeria 32.1% of 85 primary school children were shown to be deficient in copper (Akeredolu et al. 2011), the incidence and prevalence of copper deficiency following roux-en-y gastric bypass surgery of 152 patients at Emory Bariatric Center was determined to be 9.6% and 18.8% respectively (Gletsu- Miller et al. 2012). 4.1% of 295 primary school children from rural community of Ebonyi State, Nigeria were shown to be copper deficient (Ugwuja et al 2015).
This work is aimed at providing information on serum Fe, Zn and Cu status of primary school children in Enugu-South LGA, in Enugu State, Nigeria

1.4 OBJECTIVE

The objective of this research is to assess the dietary intake and serum micronutrient status of primary school children aged 5-13 years in Enugu-South L.G.A.

1.4.1 Specific objectives

- To determine children’s serum Fe, Cu and Zn concentration.
- To determine children’s dietary Fe, Cu, and Zn intake.
- To established children’s feeding pattern.
CHAPTER TWO

2.0 LITRATURE REVIEW

The recent estimation from FAO indicates that 840 million people do not receive enough energy from their diets to meet their needs (FAO 2004). The majority of these people, 799 million – live in developing countries. Micronutrient deficiencies can also exist in population where the food supply is adequate in terms of meeting energy requirement. In these situations people are not considered ‘hungry’ in the classical sense, but their diets maybe grossly deficient in one or more micronutrients. These deficiencies often go unnoticed within the community in spite of their insidious effects on immune system functioning, growth and cognitive development. For these reasons micronutrients deficiencies are referred to as ‘hidden hunger’ (kennedy 2002).

Micronutrients are the essential vitamins and mineral required by human beings to stimulate cellular growth and metabolism (Brown 2002). The period of childhood between ages 4-13 years is characterized by continued physical growth and rapid cognitive, emotional and social development (Mahan 2008). Many children especially girls undergo their pubertal growth spurt between ages 4-13. This period of childhood precedes adolescence the transitional stage of development between childhood and adulthood. Due to increased growth or metabolism, the nutritional requirements of children are higher in proportion to body weight compared with adults (Brown 2002).

Good nutrition throughout childhood is important not only to support growth and cognitive development but also to establish healthy eating patterns that are associated with decreased risk of chronic conditions and diseases in adulthood, including; obesity, Type II diabetes, cardiovascular disease, metabolic syndrome and osteoporosis (Brody 1999). Deficiencies usually occur when the habitual diet lacks diversity or is overly dependent on a single staple food, as is the case with monotonous cereal or tuber based diets. Situations of food insecurity where
populations do not have enough to eat will also inevitably result in micronutrient deficiency (FAO 2002).

2.1 IRON

This is an essential component of hundreds of proteins and enzymes involved in various aspects of metabolism, including oxygen transport and storage, electron transport and energy metabolism, antioxidant and beneficial pro-oxidant functions, oxygen sensing and DNA synthesis. (Harris 1997).

Iron is stored in the body as ferritin, and serum level of ferritin is a good clinical indicator of iron status in children (Edelstein 2009). Iron-deficiency anemia in children leads to poor cognitive development, poor school achievement and behavioral problems (Thomas 2009). Several possible mechanisms link iron-deficiency anemia to altered cognition. For example, any cognitive benefit associated with iron supplementation could be possible due to changes in nerve myelination, which have been observed in iron deficient animals (Lozoff 2009). Iron has an important role in the development of the cells that produce myelin. The myelin sheath is the insulating layer of tissue comprised of lipids and proteins that surrounds nerve impulses. Iron is also important for enzymes involved in the synthesis of certain neurotransmitters and for DNA synthesis (Beard 2001).

The amount of bioavailable iron in food is influenced by the iron nutritional status of the individual and also by the form of iron (heme or non-heme). Individuals who are anemic or iron deficient absorbs a larger percentage of the iron they consume (especially non-heme iron) than individuals who are not anemic (Harris 1997). Iron found in meat, poultry and fish is more readily absorbed. Although heme iron generally accounts for only 10-15% of the iron found in the diet, it may provide up to one third of total absorbed dietary iron (Harris 1997).
2.1.1 FUNCTIONS OF IRON

2.1.1.1 Iron as Oxygen Carrier

Most of the iron in the body is present in the erythrocytes as haemoglobin, a molecule composed of four units, each containing one heme group and one protein chain. The structure of haemoglobin allows it to be fully loaded with oxygen in the lungs and partially unloaded in the tissues (eg. in the muscles). The iron containing oxygen storage protein in the muscles, myoglobin, is similar in structure to haemoglobin but has only one heme unit and one globin protein chain (FAO/WHO 2004).

2.1.1.2 Iron in Electron Transport

Several iron containing enzymes, the cytochromes, also have one group and one globin protein chain. These enzymes act as electron carriers within the cells and their structures do not permit reversible loading and unloading of oxygen. Their role in the oxidation metabolism is to transfer energy within the cell and specifically in the mitochondria.

Other key functions of Fe containing enzymes is as cytochrome p450. Cytochromes are heme-containing compounds that have important roles in mitochondrial electron transport, therefore cytochromes are critical to cellular energy production and thus life. They serve as electron carriers during the synthesis of ATP, the primary energy storage compound in cells. Cytochrome P450 is a family of enzymes that functions in the metabolism of a number of important biological molecules, as well as the detoxification and metabolism of drugs and pollutants. Non-heme iron-containing enzymes, such as NADH dehydrogenase and succinate dehydrogenase, are also critical to energy metabolism (Yip1996), including the synthesis of steroid hormones and bile acids. It also detoxifies foreign substances in the liver and in signal controlling in some neurotransmitters such as the dopamine and serotonin systems in the brain. Iron is reversibly
stored within the liver as ferritin and hemosiderin, whereas it is transported between different compartments in the body by the protein transferrin.

### 2.1.1.3 Iron as an Antioxidant and Pro-oxidant

Catalase and peroxidases are heme-containing enzymes that protect cells against the accumulation of hydrogen peroxide, a potentially damaging reactive oxygen species (ROS), by catalyzing a reaction that converts hydrogen peroxide to water and oxygen. As part of the immune response, some white blood cells engulf bacteria and expose them to ROS in order to kill them. The synthesis of one such ROS, hypochlorous acid, by neutrophils is catalyzed by the heme-containing enzyme myeloperoxidase (Brody 1999).

### 2.1.1.4 Iron in DNA Synthesis

Ribonucleotide reductase is an iron-dependent enzyme that is required for DNA synthesis (Fairbanks 1999). Thus iron is required for a number of vital functions such as growth, reproduction, healing, and immune function.

### 2.1.2 IRON REQUIREMENTS

Iron is not actively excreted from the body in urine or in the intestines. Iron is only lost with cells from the skin and interior surfaces of the body, intestine, urinary tract and airways. The total amount lost is estimated at 14 mg/kg body weight/day. In children it is probably more correct to relate the losses to body surface. The new born infant has an iron content of about 250-300 mg (75mg/kg body weight). During the first two months of life, haemoglobin concentration falls because of the improved oxygen situation in the new born infant, compared with the intrauterine fetus, this leads to a considerable redistribution of iron from catabolized erythrocytes to iron
stores. This iron will cover the needs of the term infant during the first 4-6 months of life. The iron situation is much less favorable in the premature and low birth weight infants than in the term infant, extra supply of iron is therefore needed in these infants even during the first 6 months of life. In full term infant iron requirements will rise markedly after age 4-6 months and amount to about 0.7-0.9 mg/day during the remaining part of the first year these requirements are therefore very high, especially in relation to body size and energy intake.

In the first year of life, the full term infant almost doubles its total iron stores and triple its body weight the changes in body iron during this period occurs mainly during the first 6-12 months of life. Between 1 and 6 years of age the body iron content is again doubled. The requirements for absorbed iron in vitamins and children are very high in relation to their energy requirements. For example, in infants 6-12 months of age about 1.5 mg of iron need to be absorbed per 4.18 mg and about half of this amount is required up to age 4 years (FAO/WHO 2004).

2.1.3 IRON ABSORPTION

With respect to the mechanism of absorption, there are two kinds of dietary iron; heme iron and non-heme iron. In the human diet the primary sources of heme iron are the haemoglobin and myoglobin from consumption of meat for too long. Calcium is the only dietary factors that negatively influence the absorption of heme iron and so to the extent that it influences absorption of non-heme iron. Reducing substances (i.e substances that keep iron in the ferrous form) must be present for iron to be absorbed. The presence of meat, poultry and fish in the diet enhance iron absorption. Other foods contain factors such as ligands that strongly bind ferrous irons and subsequently inhibit absorption of iron. Examples are phytates and certain iron-binding polyphenols. Phytates are found in all kinds of grains, seeds, nuts, vegetables, roots and fruits.
Chemically, phytates are inositol hexaphosphate salt and are a storage form of phosphates and minerals.

Ascorbic acid is the most potent enhancer of non-heme iron absorption. Synthetic vitamin C increases the absorption of iron to the same extent as the native ascorbic acid in fruits, vegetables and juices. If meals contain many inhibitors of iron absorption, more of vitamin C should be in the meal preferably more than 25mg (Hallberg 1997).

The body has three unique mechanisms for maintaining iron balance and preventing iron deficiency and iron overload. The first is the continuous re-utilization of iron from catabolized erythrocytes in the body. The iron is released and delivered to transferrin in the plasma, which brings the iron back to red blood cell precursors in the bone marrow or to other cells in different tissues. Regulation is the synthesis of transferrin receptors on the cell surface. This system for internal iron transport not only controls the rate of flow of iron to different tissues according to their needs but also effectively prevents the appearance of free iron and formation of free radicals in the circulation.

The second mechanism is the access of the specific storage ferritin which can store and release to meet excessive iron demands. This iron reservoir is especially important in the third trimester of pregnancy.

The third mechanism involves the regulation of absorption of iron from the intestines, with increased iron absorption in the presence of decreasing body iron stores and decrease iron absorption when iron stores increase (Hallberg 1997).
2.1.4 IRON DEFICIENCY

Worldwide, the highest prevalence of iron deficiency is found in infants, children, adolescent and women of childbearing age, especially pregnant women. In developing countries the iron situation is very critical in many groups, especially in the weaning period. Iron nutrition is of great importance for the adequate development of the brain and other tissues such as muscles, which are finally differentiated early in life (FAO/WHO 2004).

Iron deficiency is defined as an absence of iron stores combined with signs of an iron deficient erythropoiesis, implying that in a state of iron deficiency there is an insufficient supply of iron to various tissues. This occurs at a serum ferritin level <50 µg/dl, iron can then no longer be mobilized from iron stores, insufficient amount of iron will be delivered to transferrin the circulating transport protein for iron. The uptake of iron seems to be related both to transferrin saturation and the number of transferrin receptors on the cell surface. There is a marked diurnal variation in the saturation of transferrin because the turnover rate of iron in plasma is very high. This fact makes it difficult to evaluate the iron status from single determinations of transferrin saturation.

The use of serum ferritin has improved the diagnostic accuracy of iron deficiency. It is the only simple method available to detect early iron deficiency (FAO/WHO 2004).

A report from a research work conducted by Ekwochi et al on the ”prevalence of iron deficiency anaemia in anaemic under five children in Enugu, South-East Nigeria”, shows that 34.3% of the 312 children had iron deficiency anaemia, which is also high among the males (Ekwochi et al. 2014).

Also in a similar work by Akeredolu et al on “iron, zinc and copper malnutrition among primary school children in Lagos, Nigeria”, showed that iron deficiency rate was 34.6% of the 200 primary school children (Akeredolu et al. 2011).
2.1.4.1 Causes of Iron Deficiency

Nutritional iron deficiency implies that the diet cannot cover physiological iron requirements. Worldwide this is the most common cause of iron deficiency. In many tropical countries, infestations with hook worms lead to intestinal blood losses that may be considerable in clinical practice. A diagnosis of iron deficiency must always lead to a search for pathological causes of blood loss (tumors in the gastrointestinal tract of uterus, especially if uterine bleedings have increased or changed in regularity) patients with achlorhydria absorbed dietary iron less well (a reduction of about 50%) and patients who have undergone gastric surgery, especially if the surgery was extensive may eventually develop iron deficiency, because of impaired iron absorption (FAO/WHO 2004).

2.1.4.2 Effects of Iron Deficiency

The relationship between iron deficiency and brain function is of great importance for the choice of strategy in combating iron deficiency. Several structures in the brain have a high iron content of the same magnitude as observed in the liver. In human about 10% of brain iron is present at birth, at the age of 10 years the brain has only reached half its normal iron content and optimal amounts are first reached at the age of 20-30 years.

In populations with long-standing iron deficiency, a reduction of physical working capacity has been demonstrated by several groups, with improvement in working capacity after iron administration.

Iron deficiency also negatively influences the normal defense system against infection. The cell-mediated immunological response by the action of T lymphocytes is impaired as a result of a reduced formation of these cells. This in turn is due to a reduced DNA synthesis depending on the
function of these cells because Ribonucleotide reductase requires a continuous supply of iron for its function (Lozoff 2007).

2.1.5 FOOD SOURCES

The amount of iron in food (or supplements) that is absorbed and used by the body is influenced by the iron nutritional status of the individual and whether or not the iron is in the form of heme. Because it is absorbed by a different mechanism than non-heme iron, heme iron is more readily absorbed and its absorption is less affected by other dietary factors. Individuals who are anemic or iron deficient absorb a larger percentage of the iron they consume (especially non-heme iron) than individuals who are not anemic and have sufficient iron stores (Lynch 1997). Heme iron comes mainly from hemoglobin and myoglobin in meat, poultry and fish. Plants, dairy products, meat, and iron salts added to foods and supplements are all sources of non-heme iron.

2.2 ZINC

This is present in all body tissues and fluids. It is an essential component of large number of enzymes. Zinc is an essential trace element for all forms of life. The significance of zinc in human nutrition and public health was recognized relatively recently. Clinical zinc deficiency in humans was first described in 1961, when the consumption of diets with low zinc bioavailability due to high phytic acid content was associated with "adolescent nutritional dwarfism" in the Middle East (Prasad 1998). Since then, zinc insufficiency has been recognized by a number of experts as an important public health issue, especially in developing countries.
2.2.1 Functions of Zinc

Numerous aspects of cellular metabolism are zinc-dependent. Zinc plays important roles in growth and development, the immune response, neurological function, and reproduction. On the cellular level, the function of zinc can be divided into three categories: (1) catalytic, (2) structural, and (3) regulatory (Cousins 2006).

2.2.1.1 Catalytic Role

Zinc was first shown to be required for the growth of the mold Aspergillus niger by Raulin in 1869. Since then, zinc has been demonstrated to be essential for the growth, development and differentiation of all types of life, including microorganisms, plants and animals. After iron, zinc is the second most abundant trace metal in the human body. An average 70-kg adult human contains 2.3 g of zinc. The first zinc metalloenzyme, carbonic anhydrase II (CAII), was discovered in 1940 by Keilin and Mann. Since then, over 300 zinc enzymes covering all six classes of enzymes and in different species of all phyla have been discovered (Christianson 1991, Coleman 1992). In most cases, the zinc ion is an essential cofactor for the observed biological function of these metalloenzymes. Furthermore, the biological functions of zinc, which are versatile and observed in many tissues, are most often associated with proteins (LPI 2015).

2.2.1.2 Structural Role

Zinc plays an important role in the structure of proteins and cell membranes. A finger-like structure, known as a zinc finger motif, stabilizes the structure of a number of proteins. For example, copper provides the catalytic activity for the antioxidant enzyme copper-zinc superoxide dismutase (Cu/ZnSOD), while zinc plays a critical structural role (Betts 1994). The
structure and function of cell membranes are also affected by zinc. Loss of zinc from biological membranes increases their susceptibility to oxidative damage and impairs their function.

2.2.1.3 Regulatory Role

Zinc finger proteins have been found to regulate gene expression by acting as transcription factors (binding to DNA and influencing the transcription of specific genes). Zinc also plays a role in cell signaling and has been found to influence hormone release and nerve impulse transmission. Zinc has been found to play a role in apoptosis (gene-directed cell death), a critical cellular regulatory process with implications for growth and development, as well as a number of chronic diseases (Bracey 1994).

2.2.2 Zinc Deficiency in Children

Zinc deficiency is a major public health concern and has been estimated to affect more than 2 billion people in less developed countries (Tuerk 2009). Children are at increased risk for zinc deficiency, which can lead to delayed physical growth, impaired immunity, and possibly to delayed mental development.

2.2.2.1 Impaired Growth and Development

Significant delays in linear growth and weight gain, known as growth retardation or failure to thrive, are common features of mild zinc deficiency in children. In the 1970s and 1980s, several randomized, placebo-controlled studies of zinc supplementation (5.7 mg/day) in young children with significant growth delays resulted in increased growth rates compared to placebo. Although the exact mechanism for the growth-limiting effects of zinc deficiency are not known, research
indicates that zinc availability affects cell-signaling systems that coordinate the response to the growth-regulating hormone, insulin-like growth factor-1 (IGF-1) (MacDonald 2000).

2.2.2.2 Impaired Immune System Function

Adequate zinc intake is essential in maintaining the integrity of the immune system, specifically for normal development and function of cells that mediate both innate (neutrophils, macrophages, and natural killer cells) and adaptive (B-cells and T-cells) immune responses (Prasad 2009). Moreover, zinc plays a structural role in the antioxidant enzyme, Cu/ZnSOD. Zinc deficiency adversely affects a number of immune functions, resulting in decreased production of certain cytokines; reduced activation of zinc-dependent enzymes and transcription factors; and decreased activity of thymulin, a zinc-dependent thymic hormone important for T-cell function (Prasad 2008). Consequently, zinc-deficient individuals are known to experience increased susceptibility to a variety of infectious agents.

2.2.2.3 Diarrhea

It is estimated that diarrheal diseases result in the deaths of over 1.8 million children under the age of five years of age in developing countries annually (Boschi-Pinto 2008). The adverse effects of zinc deficiency on immune system function are likely to increase the susceptibility of children to infectious diarrhea and persistent diarrhea contributes to zinc deficiency and malnutrition. Research indicates that zinc deficiency may also potentiate the effects of toxins produced by diarrhea-causing bacteria like *E. coli* (Wapnir 2000). Zinc supplementation in combination with oral rehydration therapy has been shown to significantly reduce the duration and severity of acute and persistent childhood diarrhea and to increase survival in a number of randomized controlled trials (Fischer 2007).
2.2.4 Pneumonia

Zinc supplementation may also reduce the incidence of lower respiratory infections, such as pneumonia. A pooled analysis of a number of studies in developing countries demonstrated a substantial reduction in the prevalence of pneumonia in children supplemented with zinc (Bhutta 1999). However, it is not clear whether supplemental zinc, in conjunction with antibiotic therapy, is beneficial in the treatment of pneumonia (Basnet 2012).

2.2.3 Food Sources of Zinc

Shellfish, beef, and other red meats are rich sources of zinc; nuts and legumes are relatively good plant sources of zinc. Zinc bioavailability (the fraction of zinc retained and used by the body) is relatively high in meat, eggs, and seafood because of the relative absence of compounds that inhibit zinc absorption and the presence of sulfur-containing amino acids (cysteine and methionine) that improve zinc absorption.

2.3 Copper

This is an essential micronutrient that is required for plant, animal, and human health. It is also required for the normal functioning of aerobic microorganisms. Copper is incorporated into a verity of proteins and metalloenzymes which perform essential metabolic functions, the micronutrient is necessary for the proper growth, development, and maintenance of bone, connective tissue, brain, heart, and many other body organs. Copper is involved in formation of red blood cells, the absorption and utilization of iron, the metabolism of cholesterol and glucose and the synthesis and release of life- sustaining proteins and enzymes. These enzymes in turn
produce cellular energy and regulate nerve transmission; blood clothing and oxygen transport (Bertinato et al. 2004).

2.3.1 SOME OF THE PHYSIOLOGICAL FUNCTIONS OF CUPPER:

2.3.1.1 Energy Production

The copper-dependent enzyme, cytochrome c oxidase, plays a critical role in cellular energy production by catalyzing the reduction of molecular oxygen (O\textsubscript{2}) to water (H\textsubscript{2}O), cytochrome c oxidase generates an electrical gradient used by the mitochondria to create a high energy proton gradient required for adenosine triphosphate (ATP) synthesis. This copper enzyme is particularly abundant in tissues of greatest metabolic activity including heart, brain, and liver (Uauy et al. 1998).

2.3.1.2 Connective Tissue Formation

Lysyl oxidase uses lysine and hydroxylysine found in collagen and elastinas substrates for posttranslational processing to produce the cross-linking of collagen, which are essential for the formation of strong and flexible connective tissue. The action of lysyl oxidase helps maintain the integrity of connective tissue in the heart and blood vessels and also plays a role in bone formation (Turnlund 2006).

2.3.1.3 Iron Metabolism

The Multi-copper oxidases (MCO) or ferroxidases, have the capacity to oxidize ferrous iron (Fe\textsuperscript{2+}) to ferric iron (Fe\textsuperscript{3+}), the form of iron that can be loaded onto the protein transferring for transport to the site of red blood cell formation. The MCO family comprises of Ferroxidase I, also called ceruloplasmin, is the predominant copper protein in plasma and may also have...
antioxidant functions, (which represents ~90% of plasma copper), defects in ceruloplasmin function produce cellular iron accumulation, and a result that supports its ferroxidase role. The membrane bound ceruloplasmin and two proteins called Hephaestin and Zyklofen found in the intestine and the placenta respectively (Vashchenko et al. 2013). Mice that do not express ceruloplasmin have normal copper metabolism but abnormal iron accumulation in the liver (Meyer et al. 2001). Similarly, individuals lacking ceruloplasmin display iron overload in selected tissues, including liver, brain, and retina (Kono 2012). This supports the idea that the ferroxidase activities of ceruloplasmin are essential to the flux of iron in the body. Moreover, the fact that iron mobilization from storage sites is impaired in copper deficiency supports the role of MCO in iron metabolism (Thackeray et al. 2011).

2.3.1.4 Central Nervous System

A number of reactions essential to normal function of the brain and nervous system are catalyzed by cuproenzymes. Such as (a) Dopamine β- hydroxylase catalyze the conversion of dopamine to the neurotransmitter, norepinephrine (Harris et al. 1997). (b) The myelin sheath which is made of phospholipids depends on cytochrome c oxidase activity for its synthesis.

2.3.1.5 Melanin Formation

Cuproenzyme, tyrosinase is required for the formation the pigment melanin. Melanin is formed in cells called melanocytes and plays a role in the pigmentation of the hair, skin, and eyes (Turnlund 2006).

2.3.1.6 Antioxidant Function

The superoxide dismutase (SOD) functions as an antioxidant by catalyzing the conversion of superoxide radicals to hydrogen peroxide, which can subsequently be reduced to water by other
antioxidant enzymes. Two forms of SOD contain copper: copper/zinc SOD which is found within most cells of the body, including red blood cells. And extracellular SOD which is a copper containing enzyme found at high levels in the lungs and low levels in plasma. The ceruloplasmin can function as antioxidants in two different ways: Free copper and iron ions these are powerful catalysts of free radical damage. By binding copper, ceruloplasmin prevents free copper ions from catalyzing oxidative damage. The ferroxidase activity of ceruloplasmin facilitates iron loading onto its transport protein, transferrin, and may prevent free ferrous ions (Fe^{2+}) from participating in harmful free radical generating reactions.

2.3.1.7 Regulation of Gene Expression

Cellular copper levels may affect the synthesis of protein by enhancing or inhibiting the transcription of specific genes. Copper may regulate the expression of genes by increasing the level of intracellular oxidative stress. A number of signal transduction pathways are activated in the expression of genes involved in the detoxification of reactive oxygen species (Mattie et al. 2008).

2.3.2 Absorption Distribution of Copper

Copper absorption occurs primarily in the small intestine. Some absorption may occur in the stomach where the acidic environment promotes copper solubility by dissociation from copper-containing macromolecules derived from dietary sources such as amino acids (Harris 1997; Turnlund 1999), and is transported via the portal vein to the liver, bound to albumin, for uptake by liver parenchymal cells. Biliary copper excretion is adjusted to maintain balance. Copper is released via plasma to extra hepatic sites where up to 95 percent of the copper is bound to ceruloplasmin (Turnlund, 1999).
2.3.3 Dietary Sources of Copper

The most current set of recommendations by the Food and Nutrition Board, used the dietary reference intake (DRIs) to reflect the food intakes of a society. In 2001, the Food and Nutrition Board issued a DRI for Cu as 440µg/day for children 4-8 years and 700µg/day for children 9-13 years (Trumbo et al. 2001). Copper is widely distributed in foods. The accumulation of copper in plants is not affected by the copper content of the soil in which they grow. Organ meats, seafood, nuts, and seeds are major contributors of dietary copper (Pennington et al. 1995). Wheat bran cereals and whole grain products are also sources of copper.

2.3.4 Copper Deficiency

Frank copper deficiency in humans is rare, but has been found in a number of special conditions. Acquired copper deficiency is mainly attributable to nutritional deficiency, and may be seen in malnourished low-birth-weight infants, newborns, and small infants (Linder et al. 1996). Copper deficiency has also been reported to develop after gastrointestinal surgery, intractable diarrhea, and prolonged parenteral or enteral nutrition (Prohaska 2012). Menkes (kinky-hair) disease is well-known to be associated with copper deficiency due to an inherited disorder of copper transport from the intestine metabolism, and Wilson disease (hepatolenticular degeneration) is a well-known inherited disorder of cellular copper transport resulting in copper accumulation (Turnlund 2006).

2.3.4.1 Clinical Symptoms of Copper Deficiency

The clinical symptoms associated with copper deficiency are extremely diverse. Hematological abnormalities (Harris 1997).
1. **Microcytic hypochromic anemia**: This is attributable to a decrease in the ferroxidase activity of ceruloplasmin (Cp) and reduced iron oxidation. When anemia is noted in low-birth-weight infants, patients with chronic diarrhea, and patients receiving prolonged enteral or parenteral nutrition, copper deficiency must be suspected in addition to iron deficiency. Granulocyte maturation disorder in the bone marrow and vacuolation in neutrophils are observed.

2. **Bone lesions in Copper Deficiency States**: Rachitic-like or scorbutic-like changes (enlargement of the epiphyseal area and changes in the margin) are observed in the bones of extremities (Thackeray *et al.* 2011). They may be accompanied by osteoporosis and occipital horn formation after adolescence. These are attributable to functional impairment of copper-requiring enzymes, such as ascorbate oxidase and lysyl oxidase, associated with copper deficiency.

3. **Vascular lesions**: Menkes disease is characterized by tortuosity and winding of arteries and increased capillary fragility (Linder *et al.* 1996). Caution must be exercised to avoid prolonged copper deficiency in humans, since this may lead to abnormal vascular tortuosity and increased capillary fragility.

4. **Central nervous system disorder and convulsion**: Reports of central nervous system disorder and convulsion associated with secondary copper deficiency are rare, but they are characteristic features of Menkes disease (Thackeray *et al.* 2011). Progressive Menkes disease can be fatal. Prolonged copper deficiency may cause degeneration of the cerebrum and cerebellum (numerous copper requiring enzymes are present in the brain, such as dopamine-hydroxylase and cytochrome c oxidase), associated with slowing of mentation and muscular
rigidity, as well as hemorrhagic changes due to increased capillary fragility. In children, hypotonia is often observed.

5. Hair Abnormalities: Change of hair texture, namely, kinky-hair, may be observed in children with Menkes disease. Hair changes are, however, considered rare in cases with secondary copper deficiency (Turnlund 2006). On the other hand, the possibility of changes in the hair should be borne in mind in cases of prolonged copper deficiency. The copper content of the hair and nail is decreased in cases of copper deficiency.

Except in newborns, low-birth-weight infants, and small infants, serum copper levels may be interpreted as follows: 60 to 80µg/dl, mild decrease; 40 to 60µg/dl, moderate decrease; 40µg/dl or less, marked decrease. In addition, information regarding the copper content of the hair and nails, and a study of the urinary copper excretion and copper balance would be useful.
CHAPTER THREE

3.0 METHODOLOGY

3.1 Study Subjects

The children 6-13 years attending public primary schools in Enugu- South local Government area of Enugu State, South- Eastern part of Nigeria as of the time of this study, there are (8841) pupils in the local Government.

3.1.1 Study Location

Enugu - South Local Government Area, Enugu State.

3.1.2 Sample design

This was a community- based cross sectional study carried out among school children by adopting a multistage random sampling technique in Enugu State. Enugu State has 17 Local Government Areas (LGAs). Enugu South was randomly selected. Enugu South has forty two (42) public primary schools with population of 8,841 pupils as at the time of this research. These schools are distributed in the LGA wards. Four wards were randomly selected in the L.G.A. A school was selected from each of the wards to give a total of four schools. One hundred pupils per school were objectively selected, to give a total of four hundred (400) pupils that will participate in the research.

3.2 Ethical Considerations

1. Ethical approval was obtained from the State Ministry of Health, Enugu State.

2. Ethical approval was also obtained from the University of Nigeria Teaching Hospital, Ittuku- Ozalla, Enugu State.

3. Permission was obtained from Enugu State Universal Basic Education Board (ENSUBEB).
4. Head Teachers of the selected schools and the Parents Teachers Association (PTA), and the School Based Management Board (SBMB), were sensitized about the project and its importance to children. Thereafter the parents that consented to be part of the research were given an informed consent form to fill and submit.

3.3 Dietary Intake

The dietary intakes of the respondents were assessed using the 24-hour dietary recall protocol. Subjects were asked to recall and describe all foods, drinks and snacks (including amount) eaten in the previous 24 hours. Portion sizes were established using standard household measures quantified in grams. The micronutrient intakes of the subjects were evaluated using dietary requirement intake (DRI).

3.4 Blood Analyses

The pupils that consented to be part of the work were recruited, and 2 mls of non-fasting venous blood sample were taken by a certified laboratory scientist that was recruited for the work. Venous blood was drawn into sterile non contaminated tubes. All tubes were kept in dark cool box (0-4°C) and transported to parasitology laboratory of Enugu State Teaching Hospital. The sera were separated from cells and stored at 0°C until analysis.

3.4.1 Preparation of Sample Containers

All glassware, sample containers and Teflon beakers were thoroughly washed with non-ionic detergent solution to ensure all dirt were removed followed by rinsing with tap water until free of detergents. The sample containers soaked in 10% (v/v) nitric acid for 48 hours and finally rinsed with distilled water and dried in the oven at 70°C prior to analysis.
3.4.2 Digestion of Sera

Digestion of biological material is very important for trace element determination. The conventional wet Acid method of Memon et al. (Memon et al 2007) was adopted. Accurately 0.5 ml of serum was taken into pyrex test tube separately. To this was added 3 ml of freshly prepared mixture of concentrated nitric acid and hydrogen peroxide (HNO₃- H₂O₂) (2:1 v/v) and stood for 10 minutes. The test tubes were covered with cotton wool and then digested at 60 °C for 1 hour in a water bath. The digests were cooled and the precipitates were separated, the filtrates were diluted with distilled water to 4 ml. The worked up samples were stored in polyethylene container at 4°C prior to AAS analysis.

3.5 Instrumentation

Atomic Absorption Spectrophotometry (AAS) (Agilent Technologies Analytical 200 series AA Model 240fsAA (UK)) was used to determine the levels of the trace metals in the digested matrices.
CHAPTER FOUR

4.0 RESULT

Table 1  Gender and Age Distribution of School Children

<table>
<thead>
<tr>
<th>Age group</th>
<th>Male (n = 155)</th>
<th>Female (n = 175)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 – 9</td>
<td>83</td>
<td>93</td>
<td>176</td>
</tr>
<tr>
<td>10 – 13</td>
<td>72</td>
<td>82</td>
<td>154</td>
</tr>
<tr>
<td>Total</td>
<td>155</td>
<td>175</td>
<td>330</td>
</tr>
</tbody>
</table>

Figure 2 Distribution of Daily Meal Intake of children.


Table 2: Frequency distribution of food types consumed by children of Enugu-South L. G. A. in 24 hours (n = 991 meals).

<table>
<thead>
<tr>
<th>Food Type</th>
<th>Frequency</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cereals</td>
<td>255</td>
<td>38.9</td>
</tr>
<tr>
<td>Tuber &amp; plantain</td>
<td>198</td>
<td>30.2</td>
</tr>
<tr>
<td>Legumes</td>
<td>81</td>
<td>12.3</td>
</tr>
<tr>
<td>Fats &amp; Oil</td>
<td>64</td>
<td>9.8</td>
</tr>
<tr>
<td>Soups</td>
<td>135</td>
<td>20.6</td>
</tr>
<tr>
<td>Animal products</td>
<td>56</td>
<td>8.5</td>
</tr>
<tr>
<td>Vegetables</td>
<td>120</td>
<td>18.3</td>
</tr>
<tr>
<td>Fruits</td>
<td>14</td>
<td>2.1</td>
</tr>
<tr>
<td>Dairy</td>
<td>36</td>
<td>5.5</td>
</tr>
<tr>
<td>Others</td>
<td>32</td>
<td>4.9</td>
</tr>
</tbody>
</table>

4.1 Micronutrient Composition of Major Foods and Drinks Consumed by subjects

The major food taken by at least 10% of the respondents are tuber based vegetables, cereals, legumes and soup. Micronutrient values of the food intakes were estimated using published data on the major foods taken.
Table 3 Micronutrient Composition of Major Foods and Drinks

<table>
<thead>
<tr>
<th>Dishes/ Drink</th>
<th>Iron (mg/100g)</th>
<th>Zinc (mg/100g)</th>
<th>Copper (mg/100g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice &amp; Tomatoes stew</td>
<td>7.43</td>
<td>8.46</td>
<td>0.98</td>
</tr>
<tr>
<td>Rice &amp; Banga Soup</td>
<td>28.76</td>
<td>3.39</td>
<td>0.89</td>
</tr>
<tr>
<td>Noodle</td>
<td>5.52</td>
<td>3.01</td>
<td>0.85</td>
</tr>
<tr>
<td>Garri &amp; Egusi Soup</td>
<td>5.20</td>
<td>4.41</td>
<td>0.15</td>
</tr>
<tr>
<td>Jellof Rice</td>
<td>7.22</td>
<td>5.62</td>
<td>0.92</td>
</tr>
<tr>
<td>Garri &amp; Okro Soup</td>
<td>4.14</td>
<td>4.31</td>
<td>0.12</td>
</tr>
<tr>
<td>Akpu &amp; Ora Soup</td>
<td>3.95</td>
<td>5.31</td>
<td>0.23</td>
</tr>
<tr>
<td>Pap &amp; Sugar</td>
<td>1.10</td>
<td>1.18</td>
<td>0.03</td>
</tr>
<tr>
<td>Okpa</td>
<td>2.27</td>
<td>3.33</td>
<td>0.15</td>
</tr>
<tr>
<td>Garri &amp; Better leaf soup</td>
<td>4.36</td>
<td>4.7</td>
<td>0.15</td>
</tr>
<tr>
<td>Akara</td>
<td>2.85</td>
<td>3.77</td>
<td>0.17</td>
</tr>
<tr>
<td>Bread</td>
<td>4.57</td>
<td>2.05</td>
<td>0.37</td>
</tr>
<tr>
<td>Chocolate tea &amp; Milk</td>
<td>4.09</td>
<td>2.39</td>
<td>0.11</td>
</tr>
</tbody>
</table>


The mean micronutrient intakes of the subjects are 4.98±3.70, 4.53±1.63, and 0.42±0.20 mg/d for Fe, Zn and Cu respectively. Table 4 shows the percentage of the dietary requirement intake (DRI) met by the subjects.
Table 4 Mean Micronutrient intakes of school children and Percentage of DRI met.

<table>
<thead>
<tr>
<th>Micronutrient</th>
<th>Sex (Age)</th>
<th>Mean intakes (mg/day ± SD)</th>
<th>% DRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fe</td>
<td>M (5-9)</td>
<td>3.94±0.96</td>
<td>39.40</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>5.71±5.40</td>
<td>71.40</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>5.48±4.87</td>
<td>54.80</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>4.80±2.50</td>
<td>60.00</td>
</tr>
<tr>
<td>Zn</td>
<td>M (5-9)</td>
<td>5.02±1.70</td>
<td>100.40</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>4.14±1.60</td>
<td>89.20</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>4.46±1.87</td>
<td>51.75</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>4.49±1.63</td>
<td>56.10</td>
</tr>
<tr>
<td>Cu</td>
<td>M (5-9)</td>
<td>0.25±0.14</td>
<td>62.50</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>0.51±0.12</td>
<td>72.85</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>0.32±0.19</td>
<td>80.00</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>0.58±0.26</td>
<td>82.50</td>
</tr>
</tbody>
</table>

F: Female, M: Male; Figures in parenthesis indicate age bracket

DRI for:

**IRON**  
5-9 years=10mg  
10-13 years = 8.0mg

**ZINC**  
5-9 years= 5.0mg  
10-13 years=8.0mg

**COPPER**  
5-9 years = 0.4 mg  
10-13 years = 0.7 mg

Source: Food and Nutrition Board (FNB 2001).
The males 5-9 years met 100% of the DRI for Zn, but not for Fe and Cu. None of the males 10-13 years met 100% of DRI for Fe, Zn, and Cu. Also none of the females 5-9 and 10-13 years met 100% of DRI for Fe, Zn and Cu.

4.2 The Serum Micronutrient Indices of Subjects

Table 5: Mean serum Micronutrient Level of School children (n= 330) in Enugu-South

<table>
<thead>
<tr>
<th>Micronutrients</th>
<th>Sex (Age)</th>
<th>Mean serum (µg/dl±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fe</td>
<td>M (5-9)</td>
<td>57.55±17.66</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>63.61±19.14</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>67.48±18.63</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>64.00±16.03</td>
</tr>
<tr>
<td>Zn</td>
<td>M (5-9)</td>
<td>61.91±16.99</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>61.36±18.13</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>57.93±16.83</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>67.88±16.53</td>
</tr>
<tr>
<td>Cu</td>
<td>M (5-9)</td>
<td>62.33±16.38</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>64.36±13.17</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>74.43±16.41</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>76.05±14.16</td>
</tr>
</tbody>
</table>

M: Male, F: Female; Figures in parenthesis indicate age bracket.

Normal Range: Fe = 50 - 120 µg/dl, Zn = 60 - 110 µg/dl, Cu = 70 - 150 µg/dl. (FAO, WHO 2004)

Table 5 shows the mean serum micronutrient levels of the subjects, the mean serum Fe for the subjects is 63.16±18.06 µg/dl. There is no statistical difference between the different age groups.
(P> 0.05). The female 5-9 years had the highest mean serum Fe level (67.48±18.63µg/dl), while the 5-9 years male group had the lowest mean serum Fe level (57.55±17.66µg/dl).

The mean serum Zn level for the subjects was 62.27±17.31µg/dl. There is no statistical difference between the different age groups in serum Zn level (P> 0.05). The males 5-9 years group had the highest serum Zn level (67.88±16.53µg/dl), while the female 5-9 years group had the lowest serum Zn level (57.93±16.83µg/dl).

The mean serum Cu level of the subjects was 69.29±14.99µg/dl. There is no statistical difference between the groups in serum Cu level (P> 0.05). The females 10-13 years had the highest mean Cu level (81.70±14.16 µg/dl), while the 5-9 years male group had the lowest mean serum Cu level (62.33±16.38µg/dl).

Table 6: Micronutrient Daily Intakes and the mean Serum level of Enugu-South children

<table>
<thead>
<tr>
<th>Micronutrients</th>
<th>Sex (Age)</th>
<th>Mean intake (mg/d±SD)</th>
<th>Mean serum (µg/dl±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fe</td>
<td>M (5-9)</td>
<td>3.94±0.96</td>
<td>57.55±17.66</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>5.71±5.40</td>
<td>63.61±19.14</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>5.48±4.87</td>
<td>67.48±18.63</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>4.80±2.50</td>
<td>64.00±16.03</td>
</tr>
<tr>
<td>Zn</td>
<td>M (5-9)</td>
<td>5.02±1.70</td>
<td>61.91±16.99</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>4.14±1.60</td>
<td>61.36±18.13</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>4.46±1.87</td>
<td>57.93±16.83</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>4.49±1.63</td>
<td>67.88±16.53</td>
</tr>
<tr>
<td>Cu</td>
<td>M (5-9)</td>
<td>0.25±0.14</td>
<td>62.33±16.38</td>
</tr>
<tr>
<td></td>
<td>M (10-13)</td>
<td>0.51±0.12</td>
<td>64.36±13.17</td>
</tr>
<tr>
<td></td>
<td>F (5-9)</td>
<td>0.32±0.19</td>
<td>74.43±16.41</td>
</tr>
<tr>
<td></td>
<td>F (10-13)</td>
<td>0.58±0.26</td>
<td>76.05±14.16</td>
</tr>
</tbody>
</table>

M: Male, F: Female; Figures in parenthesis indicate age bracket
Table 7: Frequency distribution of serum Fe level (n= 330) of Enugu-South school children.

<table>
<thead>
<tr>
<th>Range (µg/dl)</th>
<th>5-9 years</th>
<th>10-13 years</th>
<th>Total subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M/F</td>
<td>M/F</td>
<td>(n= 330)</td>
</tr>
<tr>
<td>0-49</td>
<td>40 (48%) / 25 (27%)</td>
<td>18 (25%) / 24 (29%)</td>
<td>107</td>
</tr>
<tr>
<td>50-120</td>
<td>43 (52%) / 68 (73%)</td>
<td>54 (75%) / 58 (71%)</td>
<td>223</td>
</tr>
<tr>
<td>&gt;120</td>
<td>0 / 0</td>
<td>0 / 0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>83/93</td>
<td>72/82</td>
<td>330</td>
</tr>
</tbody>
</table>

M: Male, F: Female. Figures in parenthesis indicate the percentages.

Normal range: 50-120 µg/dl (FAO, WHO 2004).

Table 8: Frequency distribution of serum Zn level (n= 330) of Enugu-South school children.

<table>
<thead>
<tr>
<th>Range (µg/dl)</th>
<th>5-9 years</th>
<th>10-13 years</th>
<th>Total subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M/F</td>
<td>M/F</td>
<td>(n= 330)</td>
</tr>
<tr>
<td>0-50</td>
<td>28 (34%) / 45(48%)</td>
<td>38(53%) /33 (40%)</td>
<td>142</td>
</tr>
<tr>
<td>60-110</td>
<td>55 (66%) /48 (52%)</td>
<td>36(47%) /49 (60%)</td>
<td>188</td>
</tr>
<tr>
<td>&gt;110</td>
<td>0/0</td>
<td>0/0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>83/93</td>
<td>72/82</td>
<td>330</td>
</tr>
</tbody>
</table>

M: Male, F: Female. Figures in parenthesis indicate the percentages.

Table 9: Frequency distribution of serum Cu level (n = 330) of Enugu-South school children.

<table>
<thead>
<tr>
<th>Range (µg/dl)</th>
<th>5-9 years</th>
<th>10-13 years</th>
<th>Total subjects (n= 330)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M/F</td>
<td>M/F</td>
<td></td>
</tr>
<tr>
<td>0-69</td>
<td>25 (30%) / 16 (17%)</td>
<td>20 (28%) / 16 (20%)</td>
<td>77</td>
</tr>
<tr>
<td>70-150</td>
<td>58 (70%) / 77 (83%)</td>
<td>52 (72%) / 66 (80%)</td>
<td>253</td>
</tr>
<tr>
<td>&gt;150</td>
<td>0 / 0</td>
<td>0 / 0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>83 / 93</td>
<td>72 / 82</td>
<td>330</td>
</tr>
</tbody>
</table>

M: Male, F: Female. Figures in parenthesis indicate the percentages.

**Normal range:** 70-150 µg/dl (FAO, WHO 2004).
Figure 3  Prevalence of Micronutrients Deficiency among Enugu-South school children.
CHAPTER FIVE

5.0 DISCUSSION

The period of childhood between ages 4 and 13 years is characterized by continued physical growth and rapid cognitive, emotional, and social development (Lucas et al. 2008). Many children especially girls undergo their pubertal growth spurt between ages 4 and 13. Table 1 showed the gender and age distribution of the children. The males who were between the ages of 5 to 9 years were eighty three (83) and the females who were between the ages of 5 to 9 years were ninety three (93). The nutritional requirement of children is higher in proportion to body weight compared with adults, this is because inadequate intake of micronutrients can impair growth and development in children. Only 233 (70%) of the children took three serving meals per day, 165 (15%) took two serving meals per day, 20 (6%) took one serving meal per day and 28 (9%) took up to four serving meals per day (Fig.1). This could be attributed to poverty and food insecurity in households as was observed by Abah et al. 2015 in North-Central Nigeria. Insufficient calorie consumption is often associated with micronutrient malnutrition (FAO 2004). Food insecurity exists when people, at all times have no physical and economic access to sufficient, safe and nutritious food to meet their dietary needs and food preferences for an active and healthy life (FAO, 2002). Following the result of the daily meal intake of the children, it can be inferred that 68 (21%) of the children do not have adequate calorie for their active and healthy life. Food insecurity can affect children’s health and brain development years before they enter a classroom, such children often are cognitively, emotionally and physically lag behind their food-secure peers. Studies have shown that food insecurity harms children’s health in a variety of ways such as mental retardation, learning difficulties, compromised immune systems, low work
capacity (Gundersen et al. 2011, Nord 2009). Also food-insecure young children are nearly twice as likely to be in fair or poor health when compared to food-secure young children and significantly more likely to be hospitalized (Cook et al. 2004). Food-insecure children are also more likely to suffer from common illnesses such as stomachaches, headaches, and colds when they reach preschool age (Alaimo et al. 2001). The stress that family hardships, like food-insecurity, place on young children physically alters the development of crucial brain structures controlling memory and psychosocial functioning (Knudsen et al. 2006). Early childhood is the narrow window during which one builds the basic capacity to learn and interact productively with their peers, disrupting this brief period diminishes children’s ability to acquire complex school skills as they grow, and later job skills (Lozoff et al 2000). Examining the role of food insecurity in cognitive outcomes showed that food-insecure 6-11 year-olds scored lower than their food-secure peers on a measure of child intelligence and was more likely to have seen a child psychologist (Ashiabi 2005). The same study also found that these children had a harder time getting along with others and more likely to have repeated a class and had lower arithmetic and general achievement test scores than food-secure children in the same age group (Ashiabi 2005). The children in this present study, who were malnourished, were more prone to micronutrient defects such as mental retardation, learning difficulties, compromised immune systems, and stunting.

As shown in Table 2, the main diet taken by these children were mostly from plant sources (Table 2). Only 56 (8.5%) of them took animal source foods. The implication is that the micronutrients (Fe and Zn) in the diets will not be completely bioavailable for absorption, because of the presence of phytates (Samuel et al. 2010, Abah et al. 2015). The micronutrient composition of the major foods and drinks consumed by the children were used to estimate their
daily micronutrient intakes (Table 3) this was obtained from published data. The result of the micronutrient intake of the children shows, that the daily Fe intake of the children ranged from 1.54-22.56 mg/d with a mean of 4.98 ± 3.70 mg/d and none of the different age groups met 100% of the DRI for Fe (Table 4). Fe deficiency is obvious since only 8.5 percent of the children consumed animal source foods and only 2.1 percent took fruits, which contains ascorbic acid that aids in non-heme iron absorption in the GIT. Plant source of Fe are mostly non-heme iron, which are not completely absorbed because of the chelating effects of plants phytic acids on the micronutrient, impairing the absorption of Fe and may make it unavailable or in short supply for absorption, thereby promoting Fe deficiency. Dietary Fe intake therefore could not meet their physiological requirements (Schlemmer et al. 2009).

The result of the serum Fe quantification shows that, the serum Fe concentration of the children ranged between 31 and 108 µg/dl, with a mean of 63.16 µg/dl (Table 5). The serum Fe tolerable value for school aged children is 50-120 µg/dl. The result of this study shows that the mean serum Fe of the children is higher than the lower cut-off value for Fe (50µg/dl). This value is because the children that had normal value tend to have high Fe intake values compared to those that are deficient. Out of 330 children, one hundred and seven (107) of them were deficient, while two hundred and twenty three (223) of them were normal. This may have contributed to their mean value being normal.

The mean serum Fe level obtained for school children in this study is lower than the mean serum Fe level of 69.84 µg/dl, found among school children in Lagos, Nigeria (Akeredolu et al. 2011) and 328.19 µg/dl, reported for school children living in North West Ethiopia (Amare et al. 2012). The disparity in the serum Fe can be associated with the dietary Fe intake of the children. The
children in Lagos which had their main dietary intake for iron as 10.66 ± 12.44 mg/d (106% of DRI) were not deficient in Fe (Akeredolu et al. 2011).

The prevalence of Fe deficiency in the children is 32.4% (Fig. 2) and is higher than 19.8% prevalence rate among the school children in Lagos (Akeredolu et al. 2011), and also lower than the 34.3% prevalence rate of Fe deficiency among anaemic under-5 children in Enugu south, Nigeria (Ekwochi et al. 2014). The Fe intakes of the children compared with their serum level shows that the males 5-9 years met only 39.4% of DRI for Fe, while the females met only 54.8% of the DRI for Fe (Table 6). Therefore the estimated dietary Fe intake of the 5-9 years children were reflected in their serum Fe level. The females 10-13 years had a higher mean serum Fe level (64.00±16.03 µg/dl) than the males 10-13 years (63.61±19.14 µg/dl). But the older males (10-13 years) had a higher dietary Fe intake meting 71.4% of the DRI, while the females 10-13 years met only 60% of the DRI for Fe. Comparing the micronutrient intake of this group (10-13years) with their serum Fe shows that their Fe intake did not appear in their serum level. It can therefore be inferred that the intake of iron-containing diets by most of the males 10-13 years on the day of sample collection was higher than that on the day the 24 hours recall interview was conducted, or that they may be infested with intestinal worms such as hook worm, which causes physiologic Fe loss or that there was a degree of exaggeration or underreporting during the interview. As shown in Table 7 and Figure 2, the males 5-9 years were mostly deficient in serum Fe, because 40 of them were Fe deficient with a prevalence rate of 48%, while the females 5-9 years had 25 of them deficient in Fe and a prevalence rate of 27%. Twenty four (24) of the females 10-13 years were deficient in Fe with a prevalence rate of 29%, while among the males 10-13 years 18 of them were deficient in Fe with and the prevalence of 25%. This result is in agreement with the report on the prevalence of Fe deficiency in anaemic under five children in
Enugu State, which shows that Fe deficiency was higher among the males than the females (Ekwochi et al., 2014). Also a similar study conducted in Lagos, Nigerian, shows that the males 9-13 years had a higher prevalence rate of Fe deficiency than the females (Akeredolu et al., 2011), which is contrary to this study.

The serum Fe level of the children was higher among the males across the age than the females, this could be attributed to the fact that many children, especially girls who undergo their pubertal growth spurt between ages 10 and 13 and may be undergoing their monthly menstruation which contributes to physiologic Fe loss. The females 5-9 years had a higher mean serum Fe level (67.48±18.63 µg/dl), than the males 5-9 years (57.55±17.66 µg/dl).

The male children, especially the 5-9 years were mostly at risk of Fe deficiency in Enugu – South which could lead to anemia and may expose them to illnesses thereby affecting their performance at school. The females 5-9 years had a higher serum Fe level than the females 10-13 years, the difference in the serum Fe concentration of the female pupils suggests that the older females may have been menstruating, which increases the risk of iron lost in the system. The implication here will be that more of the females (10-13 years) and may predispose them to iron deficiency. Pregnant girl with iron deficiency anemia is prone to have pregnancy complications and its related problems (Ugwuja et al. 2011). The low level of serum iron concentration in this study, especially with the males indicates that Fe deficiencies will be obvious, which may lead to iron deficiency anemia and is associated with reduced immunity, impaired mental development, physical coordination skills and impaired school achievement in older children. It also lowers resistance to disease and weakens a child’s learning ability and physical stamina. It slows mental and motor development and reduces work performance if not attended to (UNICEF 1998).
Inadequate iron diet is by far the major cause of anemia. It also can occur as a result of parasitic infections, inherited disorders, and deficiencies of other micronutrients (Draper 1996). Parasitic infections includes; malaria and helminthes (notably, hookworm). Malaria causes the destruction of red blood cells and hookworms cause blood loss. Inherited disorders that can leave an individual vulnerable to anemia include sickle cell anemia. A much common cause of anemia results from deficiencies of other nutrients, such as folate a B-complex vitamin (Draper 1996).

The far reaching effects of Zn deficiency in children led to the assessment of the dietary intake and serum Zn level of the school children. Although zinc deficiency is largely related to inadequate intake or absorption of zinc from the diet, excess losses of zinc during diarrhea may also contribute to Zn deficiencies. Tables 3 showed that majority of the children’s food were derived from plant sources, which supplied inadequate amount of Zn needed by the body for its optimum functions.

The mean daily Zn intake of the children ranged from 1.25-8.46 mg/d with a mean of 4.53 ± 1.63 mg/d (Table 4). Only the males 5-9 years group met 100% of the DRI for Zn. Zinc seems deficient since the majority of the food ingested by this children where cereals and tuber crops. These foods contain phytic acids, and other potent inhibitor that inhibits the absorption of Zn from the GIT (Samuel et al. 2010). Zinc supply from the meal may be unavailable for uptake into the system due to the chelating effects of these inhibitors.

The serum Zn levels of the children ranged between 31 and 107µg/dl, with a mean value of 62.27±17.31 µg/dl. This value is adequate using the lower cut-off value of 60µg/dl (Table 5). The mean serum Zn values obtained in this study is lower than the mean serum Zn, reported for school children in South African (66.4µg/dl) (Samuel et al. 2010). Also in a similar study conducted on school children living in Northwest Ethiopia mean Zn level was 86.40µg/dl.
(Amare et al. 2012), showing that Zn deficiency was not severe among school children. School children in North Central Nigeria had a mean serum Zn concentration 22.4µg/dl (Abah et al. 2015), which is very low when compared to the Zn concentration obtained in this study. Since the micronutrient intake of individuals contributes to the serum micronutrient levels, the micronutrient intake of the children in this study could have been affected by the type of meal taken in Nigeria. A report on the Zn intake and the serum Zn concentration of the children in Lagos showed that their mean Zn intake met 92% of the DRI for Zn, and their serum zinc concentration as 84.58µg/l (Akeredolu et al. 2011), linking serum concentration as a reflection of meal intake. The dietary Zn intake of the South African children is 4.6mg/day, while their mean serum concentration was within the normal range (Samuel et al. 2010).

The prevalence of Zn deficiency among the school children was 43.6% (Figure 2), using the cut-off value for serum Zn concentration below 60µg/dl (Hortz et al. 2003). This value is higher than the 20% prevalence rate set by the International Zinc nutrition consultative group (IZiNCG), as an indicator of maximum Zn prevalence rate of public health concern (IZiNCG 2007). It is also higher than 21% found among school children in Lagos, Nigeria (Akeredolu et al. 2001) and of 46% and 47% prevalence rate found among school children in South Africa and Northwest Ethiopia respectively (Samuel et al. 2010, Amare et al. 2012). A study in North Central Nigeria reported a prevalence rate of 99.2% (Abah et al. 2015). There is therefore a high prevalence of Zn deficiency among school aged children in Nigeria, more especially in Northern Nigeria.

The frequency distribution of Zn deficiency among the children in this study (Table 8 and Fig. 2), shows a high level of Zn deficiency among the females 5-9 years and a prevalence rate of 48% (45 children), compared to the males 5-9 years who had 34% (28 children) deficiency rates. The males 5-9 years had a higher serum Zn level (61.91 ± 16.99µg/dl), than the females 5-9
years (57.93 ± 16.83µg/dl). Comparing this with the dietary Zn intake of the children reveals that the females had a lower zinc intake meeting only 51.75% of the DRI for zinc, while the males 5-9 years group had intake up to 100% of the DRI. The daily Zn intake of the male 5-9 years was not reflected in their serum Zn, because their serum Zn concentration is within the normal range (60-120µg/dl). It can be inferred that the total Zn in their food was not absorbed due to the presence of phytates. The females 10-13 years had a higher serum Zn level (67.88 ± 16.53µg/dl), than the males 10-13 years (61.36 ± 18.13 µg/dl). This was also not reflected in their Zn intake since the male 10-13 years met 89% of DRI for Zn and the females 10-13 years met only 56% of the DRI, there might be a possibility of underreporting during the interview. The males 10-13 years were more at risk of Zn deficiency than the females because the females who were deficient were 33 (40%), when compared to the males that had only 38 (53%) who were deficient (Table 8).

Zinc plays an important role in growth and development, the immune response, neurological function, and reproduction, so its deficiency may lead to episodes of sickness, stunting and delayed onset of puberty among the males. This study does not agree with the report that boys have greater vulnerability to Zn deficiency than the girls (Cavan et al. 1993). However reports showed that 13% boys and 14% girls were Zn deficient in Iran (n = 350) (Sherif et al. 1999) and that 51% boys and 58% girls were Zn deficient in Sri Lanka (n = 400) (Hettiarachchi et al. 2006). This study demonstrated the existence of Zn deficiency among school aged children and adolescence in developing countries. The serum Zn of the subjects shows a deficiency rate high above normal and the mean Zn intakes did not met the DRI for Zn except for the males 5-9 years. The implication will be a risk of growth retardation, delayed onset of puberty, impaired immunity against diseases, abnormal cognitive development and poor performance at school.
(Maureen 1998). However this could be prevented by advocating for foods that are rich in Zn, such as red meats and shellfish especially for the older subjects.

Deficiency of Cu can affect all age group. Cu is required with Fe for the synthesis of hemoglobin. The DRI of the children shows that the Cu requirement of the subjects increased as the age increased among the males and the females. The major meals taken by these children were mostly cereal and tuber based (Table 3), which do not supply adequate amount of Cu required by the body. Dietary sources of copper includes; organ meats, sea foods, nuts, and seeds, which were lacking in the diet of the school children. The Cu intake results showed that none of the children in the different age groups met the DRI for Cu as shown in Table 4. The implication is that the children will be deficient in Cu. The dietary intake of the children ranged between 0.04 and 1.15mg/d with a mean of 0.31mg/d. The results showed that the females 5-9 years group had higher Cu intake (0.32±0.19) than the males 5-9 years (0.25±0.14) and the females 10-13 years group had higher Cu intake (0.58±0.26) than the males 10-13 years (0.51±0.12). Overall, the females of the groups consumed foods high in Cu than the males. The intake of Cu will manifest in the serum level, therefore the male groups will obviously be deficient in Cu.

The result of the serum Cu level of the children shows that, their Cu level ranged between 28 and 98 µg/dl, with a mean of 69.29±14.99µg/dl (Table 5). The mean serum Cu shows that the children were deficient in Cu, based on the cut–off value for serum Cu concentration (70µg/dl). The males 5-9 years group were the most deficient in Cu (62.33±16.38µg/dl), while the females 10-13 years had the highest Cu level (76.05±14.16µg/dl).

The mean serum Cu level of the children reflects their Cu intake, which shows deficiency existing mostly among the males of different groups. The reason is likely to be, because the
females of the groups had appreciable higher intake of Cu than the males as shown in Table 4. A similar study in Ethiopia showed that the children who were normal had a Cu concentration of 200µg/dl, while those that were severely and mildly stunted had their Cu concentrations as 152.55 and 186.89µg/dl respectively (Amare et al. 2012). This value is higher than the result obtained in this study. It was observed that the water and diets of the children in Ethiopia were very high in Cu indicating that the mineral contents of the soil can accumulate in the plants and also in the water of a particular environment. Thus mineral from the soil can contribute to Cu deficiency or its fortification (Amare et al. 2012). Another study carried out among malnourished children and well feed children showed that, the serum Cu concentration of the malnourished children were lower than that of the well feed group (Ugwuja et al. 2007). This indicates further that, the dietary intake of Cu reflected in the serum concentration.

Comparing the micronutrient dietary intakes and the mean serum concentration of Cu in the children shows a similarity between the Cu intake and the serum levels (Table 6). The males of 5-9 years met 62.5% of Cu DRI and the serum concentration of 62.33µg/dl, while the females of 10-13 years that met 82.50% of the DRI for Cu, had a serum concentration of 76.05µg/dl.

The prevalence of Cu deficiency among the groups indicates that 23.3% (77) of all the children were Cu deficient (Table 9 and Fig. 2). The males 5-9 years had a deficiency rate of 30% (25 children) and the females 5-9 years had a deficiency rate of 17% (16 children). The males 10-13 years had a deficiency rate of 28% (20 children) and the females 10-13 years had a deficiency rate of 20% (16 children). The result shows that the males of all the age groups were more at risk of Cu deficiency than the females (Akeredolu et al. 2011). A study in Lagos showed that 32.1% (n = 200) of all the children were Cu deficient and that the males 5-8 years were mostly deficient. In another study on school aged children at North-West Nigeria involving five states
showed that all the children were deficient in Cu (Ebiloma 2013). The low serum concentration of Cu in this study is an indication that copper was present at low level in the children’s diet probably due to the level of the element in the soil or that copper was poorly available for absorption.

Prolonged deficiency of Cu may lead to bone lesions, which may be accompanied by osteoporosis and oceital horn formation after adolescence in these children. Therefore Cu intake should be encouraged and confectionary foods fortified with Cu, which will alleviate the deficiency of these children in this region.

Generally interactions between trace elements have long been recognized (Ajayi 2005). An intriguing interaction appears to exist between copper, zinc and iron in absorption and utilization (Ajayi 2005). Supplementation of Fe has been reported to affect bioavailability of Zn and Cu in Fe deficiency anemia by inter-element competition in the bowel, while on the bioavailability of Cu and Fe are affected by Zn supplementation (Ajayi 2005). It has been established that Fe deficiency results in increased Cu levels in the liver (Turnlun 2006) while severe Cu deficiency causes changes in Fe metabolism, leading to anemia because Cu is an essential component in the formation of feroxidase 1. Deficiency of Cu induces a dramatically decrease in feroxidase activity which in turn prevents the mobilization of Fe from stores by being oxidized from +2 to +3 and its incorporation into hemoglobin therefore causing an accumulation of Fe in the liver (Smith et al. 1998). The micronutrients deficient in this study might be an indication that they were present at low supply in the diet of the subjects or were poorly bioavailable.

Other important micronutrients for further research includes, vitamin A which has a vital role in maintaining eye health and vision, growth, immune function and survival of children (Sommer et al. 1996). Vitamin A deficiency is the most important cause of preventable blindness in young
children. Incidence of morbidities, especially episodes of respiratory infection, diarrhea, measles and childhood mortality are closely associated with vitamin A deficiency (Vijayaraghavan 2006).

Iodine functions as a component of thyroid hormones, which play a vital role in the regulation of metabolic processes such as growth and energy expenditure. It is essential to the normal development of the foetal brain and the nervous system. It may protect against the effect of radioactivity. It regulates the effect of oestrogen on breast tissue. It is a component of a healthy connective tissue (Ekweagwu et al. 2008). However, the Universal Salt Iodization (USI) program has greatly reduced the risk of iodine deficiency diseases among the Nigerian population (UNICEF 2007).

These deficiencies can lead to serious health problems, including reduced resistance to infectious diseases, blindness, lethargy, reduced learning capacity, mental retardation and in some cases, to death. Therefore efforts should be intensified towards the education and awareness on proper nutrition especially among the rural population.
5.1 CONCLUSION

The food intakes of school children did not supply adequate amount of micronutrients needed for a healthy body.

Based on the dietary intake and serum micronutrient concentration the result shows that micronutrient deficiency exists among the school children. This can lead to adverse effect on their maturation and performance at school.

There is therefore, urgent need to educate the public on good eating life style and the importance of diversification of diets. Also a nutrition education program should be put in place in other to combat the micronutrient deficiency in this Enugu-South L.G.A and beyond.
REFERENCE


Linus Pauling Institute at Oregon State University lpi.oregonstate.edu


