

**LEVELS OF CHROMIUM IN SOME SELECTED MEAT  
PRODUCTS, FRUITS AND DRINKS IN ZARIA  
AND ENVIRONS**

**BY**

**MUSA GARBA ABDULLAHI**

**MARCH, 2008.**

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**A THESIS SUBMITTED TO THE POST GRADUATE SCHOOL  
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OF THE REQUIREMENT FOR THE AWARD OF THE DEGREE OF  
MASTER OF SCIENCE IN PHARMACEUTICAL CHEMISTRY.**

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**MARCH, 2008**

## **DECLARATION**

I hereby declare that the work reported in this thesis was carried by me under the supervision of Dr. (Mrs.).M.T. Bakare Odunola and Professor Garba Magaji, both of the Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmaceutical Sciences, Ahmadu Bello University Zaria. The work has never been presented in any previous application for higher degree. The work of other investigators are acknowledged and referred to accordingly.

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## CERTIFICATION

This thesis entitled level of chromium in some selected Nigerian Meat products, Fruits and Drinks by MUSA GARBA ABDULLAHI meets the regulations governing the award of the Degree of MASTER OF SCIENCE of Ahmadu Bello University, Zaria and is approved for its contribution to knowledge and Literary presentation.

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## **DEDICATION**

I dedicate this work to my late Father, Alhaji Garba Asaya who nurtured my dream, my Mother Saudatu who assisted me with prayer all through, my family and the general Ummah.

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## **ABSTRACT**

Chromium has been known to be a micronutrient for mammals for more than four decades. But progress in elucidating the role of Chromium has proceeded slowly. However, recent studies have shown a potential role of Chromium in maintaining proper Carbohydrate and lipid metabolism at the molecular level. This study quantitatively determined the level of chromium in meat products, fruits and drinks. Sample (Meat products, fruits and Drinks), were purchased from Zaria market and environs for the study. A 5.0 gm quantity of each of the samples was treated to a wet digestion method. Concentration of Chromium in the samples was determined using TAS 990 Intec Roma model of atomic absorption spectrophotometer (AAS). The results showed that, significant ( $p < 0.05$ ) levels of Chromium were detected in all the meat products and at level higher than the 0.002 mg/l FAO guideline and the 0.02-0.52 mg/l, WHO report of level of chromium in meat products. Highest level of chromium  $0.865 \pm 0.001$  mg/l (Mean  $\pm$  S.E.M) was detected in Chicken, while lowest level of Chromium  $0.013 \pm 0.000$ mg/L (Mean  $\pm$  S. E.M) in watermelon. Chromium was detected in all the samples except Lipton tea. .

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## ABREVIATIONS

NIDDM	=	Non Insulin Dependant Diabetes Mellitus.
G.D.M	=	Gestational Diabetes Mellitus
MODY	=	Maturity Onset Diabetes Mellitus of the young.
ml	=	Milliliter.
ROS	=	Reactive Oxygen Species.
PPM	=	Parts per million.
AAS	=	Atomic Absorption Spectrophotometer.
S.E.M	=	Standard Error of the Mean.
N.R.C.F.N.B	=	National Research Council, Food and Nutrition Board
WHO	=	World Health Organization.
$\beta$	=	Beta.
IDDM	=	Insulin dependent Diabetes mellitus.
G T F	=	Glucose tolerance factor.
G.C.D.W.Q.	=	Guidance for Canadian drinking water Quality.
F.D.A	=	Food drug Administration.
$\mu$	=	Microgram.
ESSADI	=	Estimated Safe and adequate daily dietary intake.
F.N.B.N.A.S.	=	Food and nutrition Board of United State National Academy of Science.
P	=	Probability
ND	=	Not Detected.

## CHAPTER ONE

### INTRODUCTION

#### 1.1 CHROMIUM.

Chromium (Cr) is a metal with atomic number 24, and an atomic mass of 52 Daltons. It occurs in nature chiefly as a chrome-iron ore. Studies have shown that chromium exist in several valence states. The most common valence state that it exists are trivalent and hexavalent. The trivalent State of chromium is the one that is mostly found in food, while the hexavalent-compound are recognized as toxic and are ``that good food sources of chromium include whole grains, cereals spices (black pepper and thyme), Mushrooms, brown sugar, brewers yeast, coffee, tea, wine and Meat products (Merz,1993). Fruits and vegetables are generally poor sources of chromium as are most refined food(Stearns *etal*, 1995).Studies have shown that Chromium is an essential trace element in human nutrition and it plays an important role in carbohydrate metabolism (Anderson, 1998). A strong link has been observed between Chromium and Insulin in carbohydrate metabolism. (Anderson, 1998). Chromium has been found to be

involved in the production of insulin and the release of glucose's energy from cells. It is normally referred to as master regulator of insulin, which is involved in protein, carbohydrate and fat metabolism (Anderson,1998).Chromium deficient rat had impaired glucose tolerance, subsequently, it was found that patient receiving long term total parenteral Nutrition (TPN) without Chromium , developed glucose intolerance, weight loss and peripheral neuropathy. These symptoms were reversed when patients were given intravenous Chromium chloride (Wolf *et al*, 1974). Chromium studies have shown that, the chromium deficient animals, malnourished children, diabetic patients and elderly receiving total parenteral (TPN) respond to inorganic chromium with improved glucose tolerance. The dietary intake of Chromium is approximately 25µg/day (F.D.A.,2006).The Food and Nutrition Board of United State National Academy of Science (F.N.B.N.A.S.) has recommended the following estimated safe and adequate daily dietary intake( ESSADI) values for Chromium:-

Age (Years)	ESSADI Micrograms (µg)
0-0.5	10-40
0.5-1	20-60
1-3	20-80
4-6	30-120
7- Adult	50-200.

90% Americans don't get enough chromium and 60% are diabetic or hypoglycemic (FDA, 2006, Anderson, 1989 and Anderson, *et al*, 1977).It was reported in 1985 that chromium level is decreased with age and it is important to replace it on daily bases as it was reported that one of chromium main function is to regulate sugar metabolism, even if sugar is absent from a diet, chromium is still necessary because, foods are essentially reduced to simple sugar (FDA, 2006).

Consumption of refined foods include simple sugars exacerbate the problem of insufficiency dietary Chromium, because these foods are not only low in dietary Chromium but also increase its loss from the body (Leads, 2002).

## 1.2 Mechanisms of Action of Chromium.

Chromium may have glucose –regulatory activity. It may also have hypercholesterolemic and anti-atherogenic activities (Vincent 2000).Mechanisms of action involves increase of insulin binding, increased insulin receptor number and increased insulin receptor phosphorylation. It was observed that chromium stimulate protein kinase activity of rat adepococyte in the presence of insulin. It also inhibits phosphotyrosine phosphatase.The activation by chromium of insulin receptor kinase activity and the inhibition of insulin-receptor tyrosine phosphates would lead to increased phosphorylaion of the insulin receptor which is associated with increased insulin sensitivity (Vincent, 2000).It was also observed that chromium may decrease hepatic extraction of insulin and improve glucose tolerance by such a Mechanism (Anderson *et al*, 1987).

### 1.3 Metabolism of Chromium

Studies have shown that chromium is absorbed through both the gastrointestinal and respiratory tract. The amount absorbed differs in each system and it depends on the form of chromium (Towill, 1978). Chromium is poorly absorbed from 0.1%- 1.2 %, where as 25% of glucose tolerance factor (GTF) a chromium complex necessary for normal glucose tolerance is absorbed. It was reported in 1979 that natural chromium complex in the diet seem to be more available for absorption than simple salt (Fribag, 1979). It was reported that tissue chromium levels in rats exposed for one year to hexavalent chromium in drinking water level of 25mg/litre were approximately nine (9) times higher than the leveling tissue of rats similarly exposed to trivalent chromium (Mackenzie, 1958). Chromium is distributed in human tissue in variable low concentrations, the levels in tissue other than the lungs decline with age. The largest stores of chromium in man are skin, muscle and fats. The tissue levels of chromium are function of sex, age and geographical location (Guideline for Canadian drinking water quality (G.C.D.W.Q.) (1978).

Studies carried out in 1999 revealed that, following absorption, chromium is bound to transferrin and albumin. It is transported primarily by transferrin.

In vivo administration of chromium ion to mammals orally or by injection resulted in the appearance of chromium ion in the iron-transport protein transferrin. It was found that chromium is distributed in various tissues of the body but appear to have preference for bone, spleen, liver and kidney (Morris *et al*, 1999).

Pharmacokinetic studies indicated that chromium is distributed into four different compartments that have rapid, medium, slow and very slow turn-over respectively. Bone, spleen, liver and kidney appear to contain all four compartments. Studies have shown that the half life of the rapid compartment is less than one day that of the medium compartment is approximately one week, while the slow compartment from 7-12 weeks. The half-life of chromium in the compartment which appears to have turn-over of most slowly is approximately one year. This compartment is probably related to long term tissue deposition (Morris *et al*, 1999).It was reported that most of an undigested

dose of chromium is excreted in the feces. Chromium that has been absorbed is excreted mainly in the urine. Little excretion occurred via the biliary route (Mackenzie, 1958).

#### 1.4 Toxicity of Chromium

It was observed that chromium supplements are generally well tolerated, but there were few reports of adverse reactions particularly with the use of chromium picolinate. A case of interstitial nephritis was reported to occur five months after a subject received a six-week course of 600 µg of chromium in the form of chromium picolinate daily (Young *et al*, 1999).

Another report describe anemia, thrombocytopenia, hemolysis, liver dysfunction, renal failure and weight loss after the use of 1,200-2,400µg of chromium picolinate daily for four to five months (Wasser *et al*,1997).There were two invitro reports that high concentrations of chromium picolinate is carcinogenic (Ceculli *etal*,1998). It was also observed that long-term usage of chromium picolinate, at a dose higher than 200µg daily could be hazardous (Anderson, 1997). It has not been demonstrated conclusively that chromium picolinate course renal failure but some have speculated that the grater absorbability of picolinate

form of chromium may, when use at higher dose (200 $\mu$ g) and long term increase the incidence of any possible effects (Anderson,1997).

## **1.5 SCOPE AND OBJECTIVES**

Diabetes has been recognized since antiquity, and treatment of various efficacy have been known in various regions since the Middle ages and legend for much longer. Pathogenesis of diabetes has only been understood experimentally since 1990 (Weiss and Sumpio, 2006).Despite the availability of treatment, diabetes has remained a major course of death.

The scope of this work is to determine the level of Chromium in some selected meat products (fish, eggs, chicken and beef), fruits (apple, carrot, orange, pineapple, watermelon and banana) and drinks (Nescafe, fanta, coca-cola, Lipton tea, zobo-drinks and Tap-water) in Zaria and environs. Atomic absorption spectroscopic method would be employed using standard procedures.

The objectives of the study is determine the level of Chromium in the above samples and to create awareness on the level of Chromium in each sample in relation to the United State

recommended estimated safe daily dietary intake (F.D.A., 2006). Since in vivo, number of Chromium level decreases in individual with increase in age, pregnancy, strenuous exercise, infection, physical trauma and other forms of stress (Anderson, R.A. 1986, Ding et al 1998 and Davies *et al*, 1997).

It is hoped that a document would be produced to enlighten the general public and the medical practitioners on the importance of this element in the management of diabetes mellitus, thus contributing to the management of diabetes mellitus.

## **CHAPTER TWO**

### **LITERATURE REVIEW**

#### **2.1.0 Diabetes Mellitus**

Diabetes Mellitus is a metabolic disorder characterized by hyperglycemia (High blood sugar) and other signs, as distinct from a single disease or condition. The World Health Organization in 1990 recognizes three main forms of diabetes:-

1. Type I
2. Type II and
3. Gestational diabetes.

It was reported that all these types of diabetes have similar signs, symptoms and Consequences but different causes and population distribution (W.H.O 1990).

Type I diabetes Mellitus is usually due to autoimmune destruction of the pancreatic  $\beta$ -cells that produce insulin.

Studies have shown that type 2 diabetes Mellitus is characterized by tissue -wide insulin resistance and varies widely; it may sometimes progresses to loss of  $\beta$ - Cell function (W.H.O. 1990).

Gestational diabetes Mellitus is similar to type 2 diabetes, in that it involves insulin resistance. The hormone of pregnancy causes insulin resistance in those women that are genetically predisposed to developing this condition. It was reported in 2006 that type 1 and 2 diabetes Mellitus are incurable chronic condition have been treatable, since insulin is medically available in 1921, but the gestational diabetes Mellitus is reported to have resolved with delivery (Stuebe *et al*, 2005).

### **Types of Diabetes Mellitus**

#### 2.1.1 Type I – Diabetes Mellitus.

Type I – Diabetes Mellitus is formally known as insulin dependent diabetes (IDDM), childhood diabetes, is characterized by loss of insulin – producing  $\beta$ - cells of the islets of langerhans of the pancreas leading to a deficiency of insulin (Shapiro,2006). It should be noted that no documentation on the measure to be taken against types 1 diabetes mellitus. It was reported that, most of the victim of this type of diabetes are otherwise healthy and are of a healthy weight when onset occurs. It was also stated that diet and exercise can't reverse or

prevent this type of diabetes mellitus. Sensitivity and responsiveness to insulin are usually normal, especially in the early stages. Types I diabetes comprise up to 10% of total cases in the North America and Europe, though this varies with geographical location (American Diabetes Association (ADS) (2005). This type of diabetes mellitus can affect children or adults but was traditionally termed “juvenile diabetes” because it represents a majority of cases of diabetes affecting children. The most common causes of  $\beta$ -cell loss leading to types I diabetes is autoimmune destruction, accompanied by antibodies directed against insulin and islet cell proteins. It was reported in 2006, that the principal treatment of type 1 diabetes is replacement of insulin. Without insulin, ketosis and diabetes ketoacidosis can develop and coma or death will result (Razari *et al*, 2006).

### **2.1.2 Type 2 Diabetes Mellitus**

Studies have shown that a type 2 diabetes mellitus was previously known as adult onset diabetes, maturity - onset diabetes or non-insulin dependent diabetes mellitus (NIDDM).

This is also because of the fact that, there is defective insulin secretion and insulin resistance or reduced insulin sensitivity (defective responsiveness of tissue to insulin), which almost certainly involves the insulin receptor in the cell membrane. In the early stage, the pronominal abnormality is reduced i.e. reduced insulin sensitivity characterized by elevated level of insulin in the blood. At this stage, hyperglycemia can be reversed by a variety of measure and medications that improve insulin sensitively or reduce glucose production by the liver, but as the disease progress, the impairment of insulin secretion worsens, and therapeutic replacement of insulin often become necessary (Weiss and Sumpio,2006). There are numerous theories as to the exact cause and mechanism for this resistance but central obesity, (Fat concentrated around the waist in relation to abdominal organs, note it seems, subcutaneous fat) in known to predispose for insulin resistance, possibly due to its secretion of adipokins (a group of hormone) that impair glucose tolerance (Knowler *et al*, 2002).

Studies have shown that obesity was found in approximately 90% of developed World patient diagnosed with type 2

diabetes. Other factors may include aging and family history, although there was a report in the decade that, type 2 diabetes mellitus has increasingly begun to affect children, and adolescent patients can be diagnosed with the sign and symptoms of types 2 diabetes Mellitus (W.H.O 1990). However, severe complications including Coronal Artery Disease and vision damage can result from unnoticed types 2 diabetes (Weiss and Sumpio, 2006).

It was reported in 1990 that there could be nerve damage and micro-regular damage which may cause erectile dysfunction, poor healing of wounds particularly of the feets can lead to gangrene which may require amputation (W.H.O. 1990).

### **2.1.3 Gestational Diabetes Mellitus**

Gestational diabetes mellitus is the type of diabetes that involves a combination of inadequate insulin secretion and responsiveness resembling type 2 diabetes in several respects. Studies have shown that it is normally developed during pregnancy and may improve or disappear after delivery. Although it may be transient or may damage the health of the

mother or fetus. About 20 – 50% of women with gestational diabetes develop type 2 diabetes mellitus later in life (Stuebe *et al*, 2005).

It was observed that gestational diabetes mellitus (GDM) occurs in about 2-5% of all pregnancies. It is temporary and fully treatable but, if left untreated it may cause problems with pregnancy, such as macrosomia (high birth weight), fetal malfunction and congenital heart diseases. It requires careful medical supervision during the pregnancy (Stuebe *et al*, 2005).

#### **2.1.4 Genetics**

It was reported in 2006, that both type 1 and 2 diabetes mellitus are at least partly inherited. Type I diabetes appears to be triggered by some (mainly viral) infections, or in a less common group, by stress or environmental factors (such as exposure to certain chemicals or drugs). Studies have shown that there is a genetic element in individual susceptibility to some of these triggers, which has been traced to particular HLA genotypes (i.e. genetics self identifiers used by the immune system). However even those who are susceptible to type I diabetes mellitus seems to require an environmental trigger. A small

proportion of people with type I diabetes carry a mutated gene that causes maturity onset diabetes of the young (MODY) (Gerstein *et al*, 2006).

### 2.1.5 Management of Diabetes Mellitus

Diabetes is a chronic diseases and emphasis is on managing short term as well as long-term diabetes. Studies have shown that there is an important role for patient education, nutritional support, self glucose monitoring, as well as long term glycemic control .A scrupulous control is needed to help reduce the risk of long term complications. In addition given the associated higher risk of cardiovascular disease, life style modifications must also be implemented to control blood pressure and cholesterol by exercising more, smoking cessation and consuming an appropriate diet (Alder, 2000). It was reported in 2004, that type I diabetes mellitus is due to the failure of one of the cell type of a single organ with a relatively simple function (i.e. the failure of the islets of langerhans) has lead to the study of several possible schemes to cure diabetes (Vinik *et al*, 2004). Studies have also been shown that in contrast, type 2 diabetes is more complex with fewer prospects of a curative measure, but further of underlying mechanism of insulin resistance may make a cure possible. Correcting insulin

resistance may provide a cure for type 2 diabetes (Bahijri and Mufti, 2002).

It was reported in 2004 that only type 1 diabetes who have received a kidney pancreas transplant (when they have developed diabetes nephropathy) and become insulin-independent may be considered “cure” from diabetes (Vinik *et al*, 2004). It has also been reported that transplants of exogenous beta cells were performed experimentally on both mice and humans, but this measure is not yet practical in regular clinical practice (Shapiro, 2006).

Type 2 diabetes can be prevented in many cases by making changes in diet and increasing physical activity (Lindstrom, *et al*, 2006 and Knowler,2002). American Diabetes Association in 2006, recommended maintaining a healthy weight, getting at least 2½hrs exercise per week, not too much fat intake, and eating a good amount of fiber and whole grain (ADA) (2006).

## 2.2 METABOLISM OF CARBOHYDRATE

Insulin is the principal hormone that regulates uptake of glucose into most cells from blood (Primarily muscle and fat cells, but not central nervous system cells), deficiency of insulin play a central role in all forms of diabetes mellitus. Much of the carbohydrate in food is converted within a few hours to the monosaccharide glucose, the principal carbohydrate found, in blood, examples of such carbohydrates are fruit sugar (fructose) that is use as cellular fuel, but is not converted to glucose, and does not participate in the Insulin/ glucose metabolic mechanism. Additionally, the carbohydrate celluloses not converted to glucose as human and many animals have no digestive pathway capable of handling cellulose.

Insulin is released into the blood by beta cells ( $\beta$ -cells) in the pancreas in response to rising levels of blood glucose (e.g. after a meal). Insulin enables most body cells (about 2/3 is the usual estimate, including muscle cells and adipose tissue) to absorb glucose from the blood for use as fuel for conversion to other needed molecules or for storage. It is also the principal control signal for conversion of glucose (the basic sugar used

for fuel) to glycogen for internal storage in liver and muscle cells. Reduced glucose level results both in the release of Insulin from the  $\beta$ - cells and in the reverse conversion of glucose, when glucose level falls, although only glucose thus recovered by the liver re-enter the blood stream, as muscle cells lack the necessary export mechanism (Vinik *et al*, 2004).

Higher insulin levels increases many anabolic (building up) processes such as cell growth and duplication, protein synthesis and fat storage. If the amount of insulin available is insufficient, if cells respond poorly to the effects of insulin (Insulin resistance or insensitivity) or if the insulin itself is defective, glucose will not be handled properly by body cells (about 2/3 require it) or stored appropriately in the liver and muscle. The net effects are persistent high levels of blood glucose and poor protein synthesis.

### **2.3 EFFECT OF CHROMIUM IN HUMAN HEALTH.**

Chromium is said to be a naturally occurring lustrous metallic element found in rocks, soil, volcanic, dust and gases and also in animals and plants. Metallic chromium is mined for use in steel and other metal products. Many chromium containing

compounds are used for planting, manufacturing paints and dyes, tanning leather and preserved wood. Less toxic forms are used to make flooring materials, video and audio recording tapes and copy machine toner. The most common forms of chromium are the metallic form, trivalent chromium (iii) and hexavalent chromium (vi). The hexavalent chromium is known as toxin, mutagen and carcinogen. Its cell penetration is 1000 times more than trivalent form; it enters erythrocytes and binds to the globin fraction hemoglobin where it is reduced to the trivalent form. It was reported in 1992 that hexavalent chromium is known as industrial toxins. Acute exposure to high concentration can cause irritation of mouth, nose, throat and lungs leading to nasal ulcer, asthma and bronchitis. It also causes skin irritation and allergy; it may also lead to kidney and liver damage (Baruthin, 1992). The element is recognized by world Health Organization (WHO) as a human carcinogen on prolonged exposure affecting particularly skin and respiratory tract (WHO, 1990).

The hexavalent chromium at 10mg/kg of body weight will result in liver necrosis and death in man, lower dose will cause

irritation of the gastrointestinal mucosa (Kaufman, 1970). The mechanism of carcinogenesis is however, not clearly known, but it was reported that the reactive Oxygen species (ROS) mediated reaction may play a role. There is also evidence that, intercellular reduction of chromium (vi) to chromium (v) plays fundamental role in DNA damage, leading to carcinogenesis. Fortunately, hexavalent is not present in significant quantities in the food chain. Toxic effects have been observed in rats and rabbits, when their drinking water contained more than 5mg of hexavalent chromium per liter (G.C.D.W.Q) (1978). It was also reported in some studies that up to 25mg / liter produced no ill effects. There was also a study of effects of hexavalent chromium and cholesterol on the development of atherosclerosis in rabbits appear consistent with the hypothesis that chromium (+6) inhibits the development of experimentally induced arteriosclerosis (Novakova, 1974). Chromium is also found to be significantly importance in altering the immune response by immuno- stimulatory or immuno- suppressive process as shown by its effects on T and

$\beta$ -lymphocytes, microphages, cytokine production and the immune response that may induce hypersensitivity reaction.

It was also reported that chromium(+3) supplementation increases muscle gain and fat loss associated with exercise and improves glucose metabolism and serum lipid profile in patients with or without diabetes (Campbell *et al*, 2002). Chromium losses are also increased during pregnancy and as a result of strenuous exercise, infection, physical training and other form of stress (Anderson, 1986). Studies have shown that, low chromium concentrations and associated impairment in insulin, glucose and lipid metabolism many also result in increased cardiovascular risk, (Horlick, 1994). Evidence for the essentiality of Chromium (+3) in human being came as a result of Patients receiving total parenteral nutrition , who have developed diabetics symptoms that were refractory to insulin but reversed by addition of Chromium.(Brown *et al*, 1988).

## 2.4 ATOMIC ABSORPTION SPECTROPHOTOMETRY

Atomic absorption spectrophotometry (AAS) is a classical technique for the determination of metals that are dissolved or suspended in a solution. The solution can be pure water, dilute or concentrated acids or organic solvents. The metals can be alkalis, alkaline earth's, transition metals, heavy metals, semi-metals and even some refractory metals.

The basics method is quite simple and fairly rapid, allowing trace metals in the fractional parts per million (ppm) range and major components in the percentage to be determined accurate and precisely.

Atomic absorption spectrophotometry is the measurement of optical radiation by atoms in the gaseous state and it is based on the ability of "excited" atom of an element to absorb energy from wavelengths of light of the same frequency as the elements. The ground state atoms in vapour state are first produced by spraying solution containing metallic elements into a flame of specific characteristics. The ground state atoms in the flame are excited by passing resonance radiation of the same frequency as the element from an external light source

through the flame. This creates a decrease in the initial signal energy, and this difference is proportional to the concentration. Each element has its own series of specific resonance wavelengths. These wavelengths will have specific characteristics for sensitivity, noise and linearity. Sensitivity and noise will determine the limits of detection for that element. Linearity will define the range in which the calibration curve will be accurate. The optical system is set up with a hollow cathode lamp for the element of interest (Analyte), with appropriate slit and wavelengths selected for this element. A solution of a known concentration of the analyte (standard) is aspirated and the absorbance reading noted. An unknown sample is aspirated the absorbance reading is rationed to the standard and a sample concentration is interpolated from the intensity of the reading.

## CHAPTER THREE

### MATERIALS AND METHODS

#### 3.1.0 MATERIALS

##### 3.1.1. Samples used.

###### **Meat products :**

Eggs,

Fish,

Beef and

Chicken.

###### **Fruits**

Banana,

Apple,

Orange,

Water melon,

Mango

Pineapple

Carrot

### **Drinks**

Tap- water

Coca-cola= Reg. No. 01- 7079. Coca-cola Bottling Company,  
Lagos, Nigeria.

Fanta = Reg. No. 01- 7416 Coca-cola Bottling Company,  
Lagos, Nigeria =

Sprite =Reg. No. 01- 7415 Coca-cola Bottling Company, Lagos,  
Nigeria.

Zobo-drinks

Nescafe. Reg. No 01-5079 (Manufactured by Nestle)

Lipton tea. Reg, No 01-0132 (Manufactured by Unilever Nigeria  
PLC).

#### **3.1.2 Solvents/Reagents.**

Distilled Water, Soil Lab. Kaduna.

Hydrogen peroxide (50%). BDH. England.

Concentrated trioxonitrate (v) acid.BDH .Analar grade, VWR  
International Ltd, Poole BH151 TD, England.

Concentrated Hydrochloric acid Breckland Scientific Supply  
11and 13, Brunel way, the ford Norfolk IP24 IHP U.K.

Chromium metal Strip (99.99%) Intec Roma –Rome.

### 3.1.3. **Equipment and Glassware.**

Atomic Absorption Spectrophotometer (TAS 990 Model) with its accessories. Manufacture by Intec Roma, Rome.

Water bath Model BJE 750A (Gallenkam).

Fume cardboard.

Analytical weighing balance Type H35AR Meter Gallen kamp

Fume cardboard. ASALAIR Model

Crucibles.

Measuring cylinders. 100ml, 1000 ml

Polyethylene bottles- 100 ml.

Conical flask, 250 ml.

Filter paper (Whatman Maidstone No 14)

Glass rods.

Volumetric flasks, 100 ml.

Hotplate, Stuart Scientific model.(pyrex)

Beaker, 50ml, 100ml and 250 ml.

Pipette, 1ml

### 3.2.0 **METHODS.**

#### 3.2.1. **Sample Collection.**

Fresh fruits, meat products and drinks were purchased from Zaria City, Tudun Wada, Samaru, Sabon Gari and Danmagaji markets. All the fruits were ripe. 50.0 gm of each item was purchased for the research. The fruits were all peeled prior to homogenization and processed to represent the fruits as consumed. They were all chopped and digested for analysis.

#### 3.2.2 **SAMPLE PREPARATION AND TREATMENT**

Samples were prepared in five forms using a wet digestion method (Miller- Ihli, 1988). 5.0 gm of each of the samples of homogenate was weighed and placed in a climax- test tube and 5.0 ml of concentrated trioxo-nitrate (v) acid was measured and added. Each of the homogenate was heated at 80 degree Celsius over night and treated with 5.0 ml Of 50 % hydrogen peroxide drop-wisely and heated at 100 degree Celsius for several hours repeating the peroxide until samples digest were cleared. The minimum amount of peroxide never exceeded 5.0 ml. Digest were filtered and diluted to final volume of 100 ml .0 and finally stored in cleaned polythene bottles until analysis.

The digested samples solutions were subsequently analyzed for Chromium content using flame atomic spectrophotometer (Intec Roma 990 model). Analysis was carried out at the most sensitive spectral line of Chromium (356.9 nm).

### **3.2.3 PREPARATION OF STANDARD SOLUTION OF CHROMIUM.**

A 1.0 gram quantity of Chromium metal was dissolved in 1ml concentrated hydrochloric acid with gentle heating and cool and finally made-up to volume in 1000 ml flask with distilled water to produce 1000.0 mg/L.

Six working standard solutions were prepared by series of dilution from the original stock solution that was prepared earlier on. Each of the diluted solution was aspirated into the atomic absorption spectrophotometer (AAS) and the absorbance recorded in each case. A plot of the concentration (mg/liter) against the corresponding absorbance gave the calibration curve for Chromium.

### **3.3.1 QUANTITATIVE DETERMINATION OF CHROMIUM.**

The Chromium content of the digested samples were determined using atomic absorption spectrometric method (Miller- Ihli,1988).The atomic absorption spectrophotometer was equipped with hollow cathode lamp of Chromium . The instrument was switched on and allow to stabilize for 10 minutes. The spectral line for Chromium was set at 357.9 nm and band pass 0.2 nm. The lamp current was set up at 7mA. The flame type was Air/ acetylene. The flame was switched on and allows to stabilize for 10 minutes. The instrument was adjusted as to achieve the sensitive line for the element to be analyzed. The system was flushed with distilled water .The samples were aspirated one after the other. The system was flushed with distilled water after each sample was aspirated and before the next one.

### **3.3.2 Statistical analysis of results.**

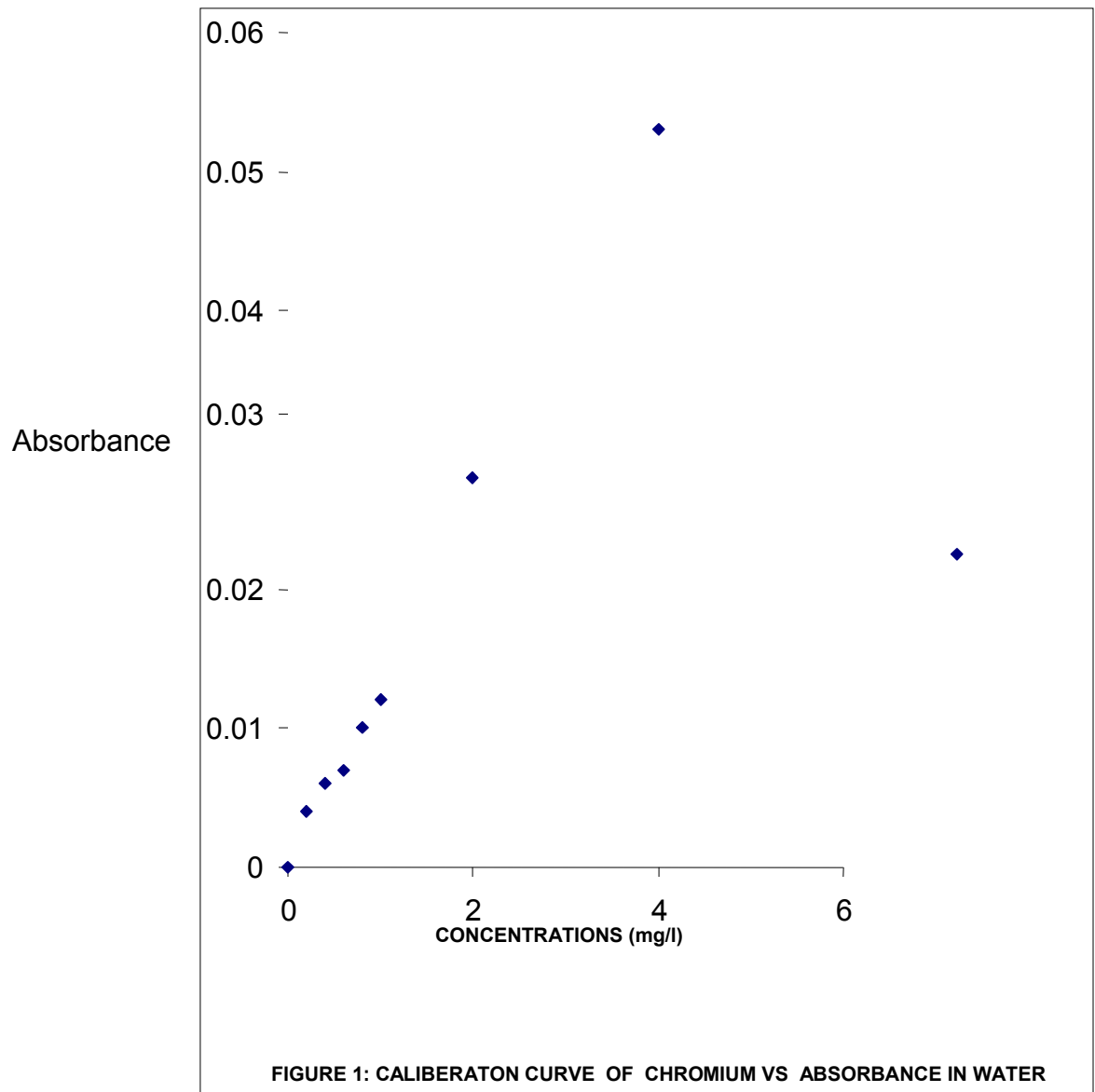
The data obtained were statistically analyzed using analysis of variance (ANOVA). The P values less than 0.05 were considered statistically significant.

## CHAPTER FOUR.

### RESULTS

#### 4,1a Calibration Curve.

Seven working Standard solutions were prepared by serial dilution (0.200, 0.400, 0.600, 0.800.1.000.2.000 and 4.000) mg/l from the 1000.0 mg/l stock solution of chromium. Each of the solutions were aspirated into the atomic absorption spectrophotometer five times and the absorbance determined. A plot of the mean concentrations (mg/l) against mean absorbance gave the calibration curve (**Fig.1**).The plot was used to generate the concentrations of chromium in meat products, fruits and different drinks.



#### **4.2 Concentration of Chromium in different meat products.**

The result obtained for the levels of Chromium in different meat products are shown in Table 1.

The highest concentration of Chromium was detected in Chicken ( $0.865 \pm 0.001 \text{ mg/l}$ ) while the lowest level of Chromium was determined in fish ( $0.052 \pm 0.001 \text{ mg/l}$ ).

Table 1: Mean Chromium Level in meat products.

S/N	SAMPLES	(Mean $\pm$ S.E.M)
1	Beef	0.770 $\pm$ 0.001
2	Fish	0.052 $\pm$ 0.001
3	Chicken	0.865 $\pm$ 0.001
4	Eggs	0.046 $\pm$ 0.000

### **Concentration of Chromium in Different Fruits.**

4.3 The results obtained for the levels of Chromium in different fruits are shown in Table 2.

The highest level of Chromium was detected in apple ( $0.077 \pm 0.001$  mg/ l), while the lowest concentration was detected in water melon ( $0.013 \pm 0.000$  mg/l).

Table 2: Mean Chromium Level in different fruits.

S/N	SAMPLES	MEAN $\pm$ S.E.M
1	Watermelon	0.013 $\pm$ 0.000
2	Pineapple	0.057 $\pm$ 0.000
3	Carrot	0.065 $\pm$ 0.000
4	Apple	0.077 $\pm$ 0.001
5	Mango	0.043 $\pm$ 0.000
6	Orange	0.054 $\pm$ 0.000
7	Banana	0.074 $\pm$ 0.000

### **Concentration of Chromium in different Drinks.**

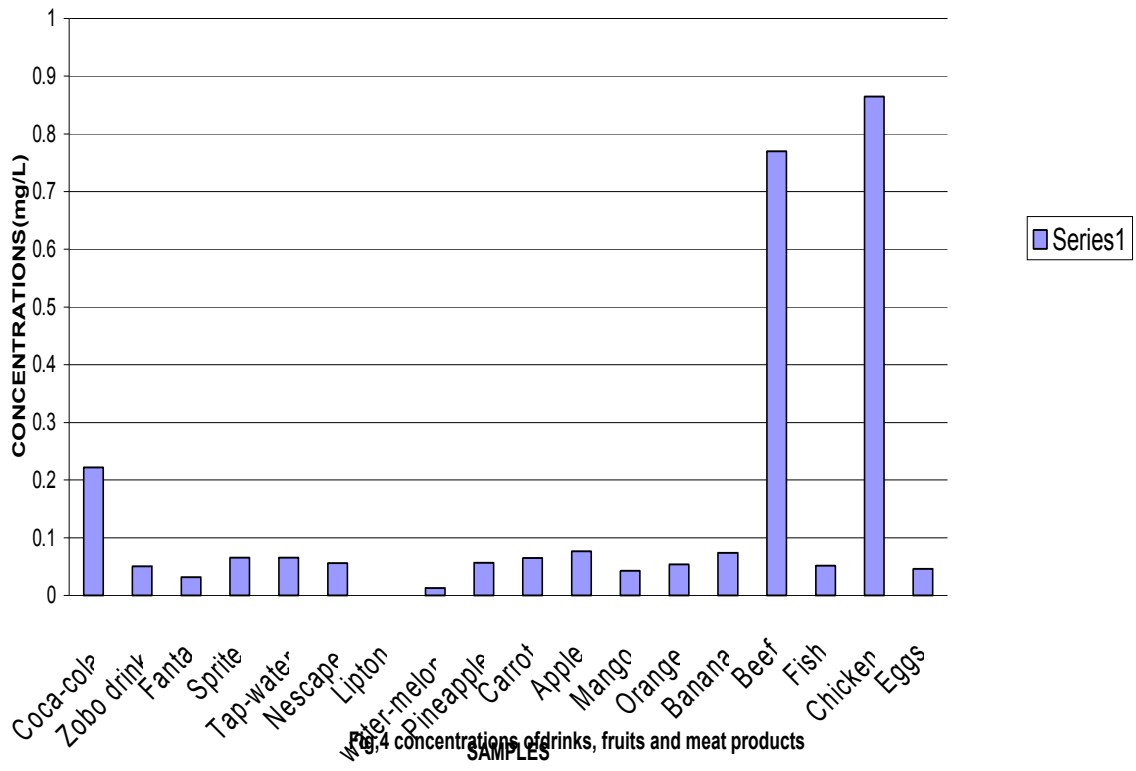
The results obtained for the levels of Chromium in different drinks are shown in Table 3.

The highest level of Chromium ( $0.222 \pm 0.000$  mg /l) was detected in Coca-cola, while the lowest concentration of Chromium was detected in Fanta ( $0.032 \pm 0.000$  mg /l).

Table 3: Mean Chromium level in different drinks.

S/N	SAMPLES	(Mean $\pm$ S.E.M)
1	Coca- cola	0.222 $\pm$ 0.001
2	Zobo- drinks	0.051 $\pm$ 0.000
3	Fanta	0.032 $\pm$ 0.000
4	Sprite	0.066 $\pm$ 0.001
5	Nescafe	0.056 $\pm$ 0.001
6	Lipton	ND
7	Tap water	0.066 $\pm$ 0.001

ND= Not detected.



The analysis of variance (ANOVA) of chromium levels among the three categories of food samples were presented in Tables 4.

**Table 4: Descriptive Statistics for the three categories of food samples.**

Category of food samples		N	Mean	Std. Deviation	Std. Error.	Significant
Meat product.	Beef	5	0.771	0.001	0.006	0.000
	Fish	5	0.53	0.009	0.004	
	Chicken	5	0.863	0.002	0.009	
	Eggs	5	0.046	0.008	0.004	
Fruits	Water melon	5	0.013	0.008	0.004	0.000
	Pineapple	5	0.061	0.009	0.042	
	Carrot	5	0.066	0.008	0.004	
	Apple	5	0.077	0.001	0.004	
	Mango	5	0.043	0.001	0.004	
	Orange	5	0.054	0.004	0.002	
	Banana	5	0.750	0.001	0.004	
Drinks	Coca- cola	5	0.223	0.02	0.001	0.000
	Zobo drinks	5	0.051	00.001	0.000	
	Fanta	5	0.031	0.012	0.001	
	Sprite	5	0.066	0.002	0.007	
	Tap water	5	0.658	0.001	0.004	
	Nescafe	5	0.055	0.001	0.006	
	Lipton tea	5	0.000	0.000	0.000	

## CHAPTER FIVE

### 5.0 Discussion.

Since low dietary Chromium has been associated with many disorders including diabetes mellitus, premature birth and intrauterine growth retardation and in male, it has been linked with infertility (Barnes and Bradley, 1994, Bradley and Bennett, 1995). It could be argued that dietary correction with foods containing high levels of chromium should be a choice for the management of these disorders. The results showed that, significant levels of Chromium were detected in all the meat products when compared with levels of chromium in fruits and drinks. The results agreed with the report that of Mertz in 1993 that meat product are good source of chromium, while lower concentrations were detected in eggs.

From the results chromium were detected in chicken fruits and drinks. A Strong link has been observed between chromium and Insulin in carbohydrate metabolism (Anderson, 1989).

Dietary intervention of source of these samples which are high in chromium level and low in cholesterol will help in the management of diabetes. Since the method adopted (Miller-ihli, 1988) in the determination of chromium is specific for chromium (iii), environmental factors may not influence the concentration of chromium determined in the samples.

## **CHAPTER SIX.**

### **6.0 SUMMARY, CONCLUSION AND RECOMMENDATIONS.**

#### **6.1 Summary.**

Atomic absorption spectrophotometer (A.A.S) was used to determine the chromium concentrations in meat products, fruits and drinks. From the studies, high levels of chromium were detected in all the samples except lipton tea.

The findings inferred the beneficiary use of meat products, fruits and drinks instead of chromium formulated drugs.

#### **6.2. Conclusion**

The research findings revealed that all the samples analyzed contained significant levels of chromium except Lipton tea. Fruits such as banana, apple, and pineapple should be used as a dietary supplement to replace chromium formulated drugs.

#### **6.3 Recommendations.**

- The Medical Practitioners should lay more emphasis on the inclusion of samples analyzed which contain high chromium levels such as fruits {banana, apple, pineapple}, and meat products that have low cholesterol level in the dietary management of diabetes.
- The blood levels of chromium in diabetic patient should be determined and compared with the glycaemic response.
- The general public should be enlightened on the importance of these food samples that contain significant amount of chromium

in relation to its importance in the management of diabetes mellitus.

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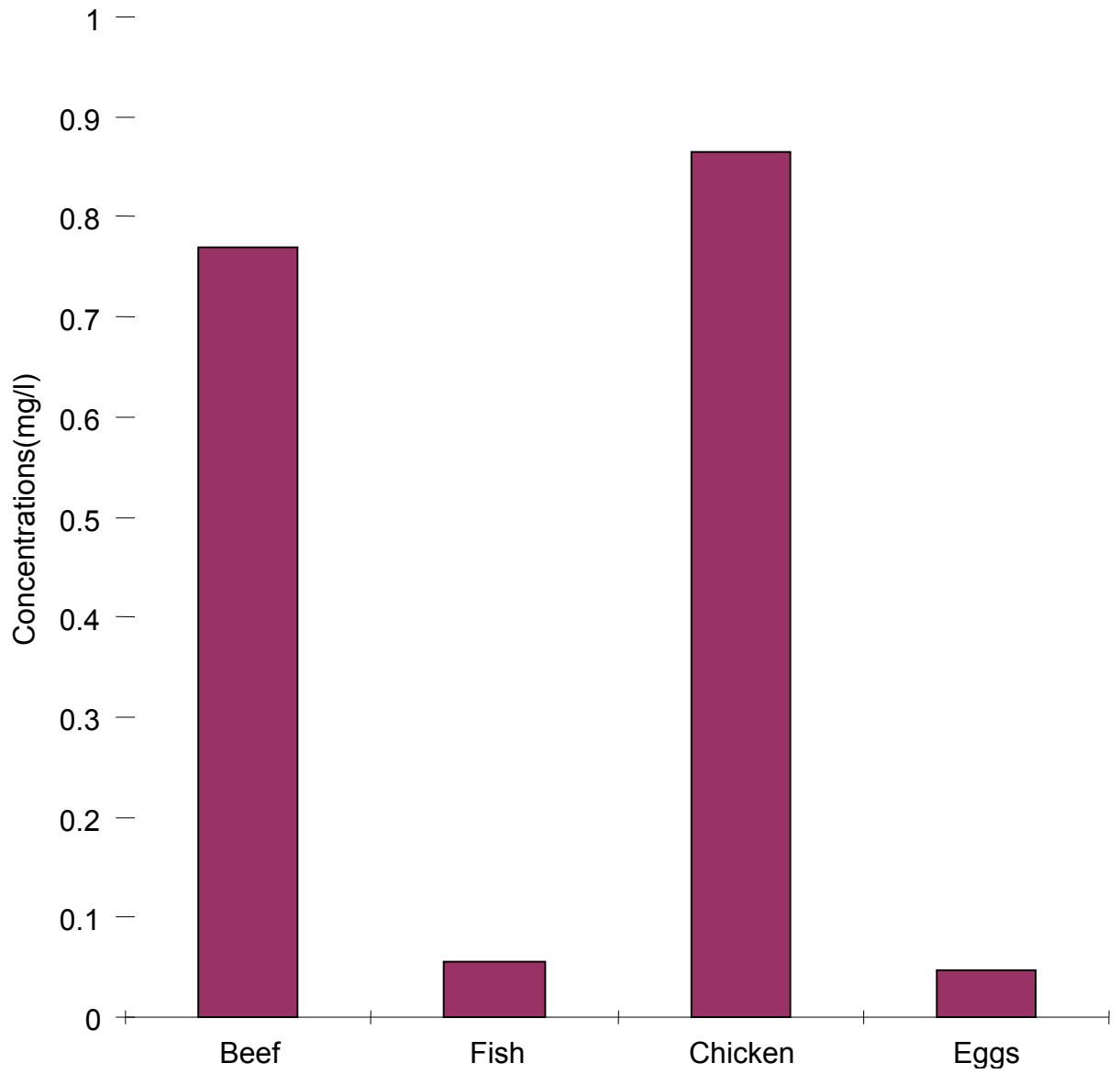
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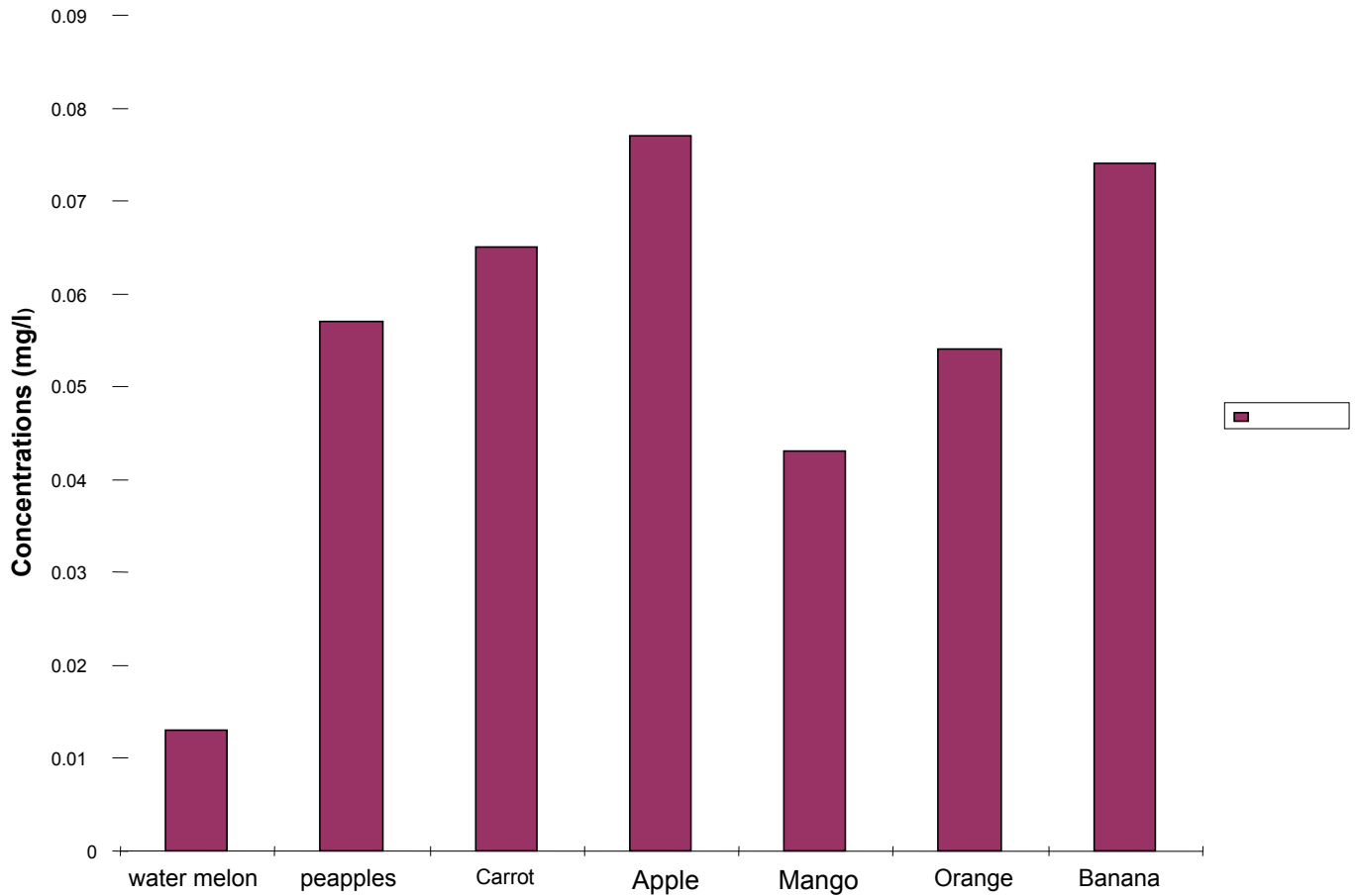
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APPENDIX 1: LEVELS OF CHROMIUM IN DIFERENT MEAT PRODUCTS IN ZARIA AND ENVIRONS.



# APPENDIX 11: LEVELS OF CHROMIUM IN DIFERENT FRUITS IN ZARIA AND ENVIRONS.



# PPENDIX 111: LEVELS OF CHROMIUM IN DIFERENT DRINKS IN ZARIAND ENVIRONS.

