**THE EFFECT OF BUCCHOLZIA CORIACEA ON RENAL FUNCTION INDICES AND OXIDATIVE STRESS IN SUCROSE FED PREGNANT RATS AND THEIR OFFSPRINGS**

**BY**

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**A RESEARCH WORK SUBMITTED TO THE DEPARTMENT OF CHEMICAL SCIENCE FACULTY OF EDUCATION, GODFREY OKOYE UNIVERSITY IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR AWARD OF B.Sc IN BIOCHEMISTRY**

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**JULY,2018**

**CERTIFICATION PAGE**

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**APPROVAL**

This is to certify that this research work "The Renal Effect of Buccholzia Coriacea On Renal Function Indices And Oxidative Stress In Sucrose Fed Pregnant Rats And Their Off springs by **Agu Lazarus Chidiebere** in the Department of Chemical science has been examined and approved as meeting the requirements for the award of Bachelor of Science (BCH) degree in, Faculty of Faculty and applied Sciences, Godfrey Okoye University, Enugu State.

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**DEDICATION**

This work is dedicated to the Almighty God

**ACKNOWLEDGEMENT**

I am most indebted to my wonderful supervisor, Mrs. Amanda Okolie, for her encouragement and support towards the success of this project. May the Almighty God increase her in wisdom and greatness forever .

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**ABSTRACT**

This study examined the nephrotic effect of *Buchholzia coriacea* formulated diet in sucrose-fed pregnant rats and their offspring. *Buchholzia coriacea (B. coriacea)* (family, *capparidaceae*) is a perennial plant commonly known as wonderful kola. Seeds were obtained from ogbete main market, Enugu state, identified, dried and grounded using a miller machine. 10% of the seed powder was used to formulate their diet. Thirty (240) adult female and six (6) adult male albino rats (180-250 g) were used in this study. High sucrose (20%) were given via drinking water to animals before, during and after pregnancy. Group A; Control normal was administered distilled water, Group B received sucrose + 10% *Buchholzia Coriacea* formulated diet (BCFD), Group C; untreated rats were administered sucrose and distilled water while Group D received 10% *Buchholzia Coriacea* formulated diet (BCFD) only. All animals were sacrificed following overnight fast by anaesthetic dos e of diethyl ether and cervical dislocation. Blood samples were obtained through cardiac puncture for the analysis of biochemical parameters. Results were represented as mean ± standard error of mean. SUC (20%) (untreated) significantly elevated urea levels (p<0.05); Creatinine (p<0.05) and LPO of the Kidney (p<0.05); (when compared with normal control group. However, following treatment with BCFD, the Urea, Creatinine and MDA level in treated rats were significantly reduced (p<0.05) when compared with the untreated group. Interestingly, all offspring showed reduced Urea level, Creatinine level and low MDA level in the Kidney except for the negative control whose offspring showed significant increase (p<0.05) in the parameters when compared with the normal group. In conclusion, this research suggests that *buchholzia coriacea* seeds has protective effect on the damage induced by high sucrose diet on the kidney of pregnant rats as well as their offsprings.

**TABLE OF CONTENTS**

Title page

Certification

Approval i

Dedication ii

Acknowledgement iii

Abstract v

Table contents vi

List of figures vii

**CHAPTER ONE: INTRODUCTION**

1.1 Background of Study 1

1.2 Statement of the problem 2

1.3 Objective of the Study 3

1.4 Significant of the study 3

CHAPTER TWO: INTRODUCTION

* 1. Introduction of Wonderful Kola 4

2.1.1 Use of wonderful kola 5

2.1.2 Taxonomy of wonderful kola 5

2.2 Hyperglycemia 6

2.2.1Causes of hyperglycemia 6

2.2.2 Signs and symptoms of Hyperglycemia 7

2.2.3 Tests That Diagnose Hyperglycemia 7

2.2.4 Treatment for hyperglycemia 8

2.2.5 Complication of hyperglycemia 8

2.2.6 Nephrons 9

2.2.7 Renal corpuscles 9

2.2.8 Renal tubule 9

* 1. Oxidative Stress In Kidney 9

2.4 Maternal Malnutrition and its Effect on Off springs 11

2.4.1 Over Nutrition 12

2.4.2 Effects of on the off springs 12

2.2.3 Causes of material malnutrition 13

2.2.4 Health risks for the baby 13

2.2.5 Prevention 14

2.5 Dietary Sucrose 15

2.5.1 Hydrolysis of sugar 16

2.2.5 Synthesis and Biosynthesis of sucrose. 16

2.6 The Effect of Sucrose on the kidney: 17

2.7 Diabetes Mellitus 18

* + 1. Mechanism 18

2.7.2 Types of diabetes 19

2.7.2 Type 1 diabetes 19

2.7.3 Type 2 diabetes 19

2.7.4 Gestational diabetes 20

2.7.5 Pathophysiology 20

2.7.6 Type 2 diabetes 21

2.7.7 Blood glucose monitoring 22

2.7.8 New oral agents 22

* 1. Metabolic Syndrome 24

2.8.1 Symptoms of Metabolic Syndrome 24

2.9 Oxidative Stress in Metabolic Syndrome 25

2.9.1 Oxidative stress 26

2.9.2 Oxidative stress in cardiovascular disease: 26

2.9.3 Oxidative Stress in Aging: 26

2.9.4 Oxidative Stress in Diabetes Mellitus: 27

2.10 Kidney Function Test 27

CHAPTER THREE: MATERIALS AND METHODS

3.1 Materials 30

3.1.1 Chemical and Reagents 30

3.1.2 Equipment 30

3.1.3 Biological Materials 30

3.2 Methods 30

3.2.1.2 Collection of Animal. 30

3.2.2 Handling of Animals 30

3.2.1: Preparation of diets 31

3.2.2: Biochemical Analyses 33

3.2.3 Statistical Analysis 31

CHAPTER FOUR: RESULT

4.1: Kidney Function Tests 38

4.2 Lipid Peroxidation (Kidney) 39

CHAPTER FIVE: DISCUSSION, CONCLUSION, RECOMMENDATIONS.

5.1 Discussion 41

REFERENCES 43

**LIST OF FIGURES**

Kidney function chat

Lipid per oxidation chat

**CHAPTER ONE**

**INTRODUCTION**

**1.1 Background of Study**

Malnutrition during pregnancy creates health risks for both the pregnant woman her developing fetus. Not eating enough or eating too much can create health risks, for both the pregnant woman and her developing fetus over nutrition during pregnancy is common in developed countries.

Many woman experience increased appetite during pregnancy. This occurs partly because their hormonal balance is altered, but also because the growing fetus removes food by products from their blood. It is also possible for woman to consume too much during pregnancy and gain excessive weight.

Woman who over consume during pregnancy increase their risk of obesity i.e. woman who gain excessive weight during pregnancy often fail to lose weight after childbirth and risk becoming overweight or obese, pre-exclaims psiai.e a condition which occurs in late pregnancy and s characterized by high levels of protein in the urine, hypertension and excessive fluid in tissues, gestational diabetes. Macros Omnia (fetus over- growth) can occur because a pregnant woman over consumers for example, excessive transfer of glucose and other nutrients can occur in pregnant woman with diabetes. As the nutrition a fetus receives in the womb pregnancies the metabolic system to function later in life over nourished fetuses have an increased risk of obesity and associated metabolic condition 5 such as type 2 diabetes later in life.

There is also evidence of an increased risk of the infant experiencing polycythemias the abnormally high red blood cell concentration seizures.

Eating too much sugar during pregnancy could affect your child’s intelligence and memory (American Journal of preventative medicine) consuming too much sugar acids gain weight. This added to me naturally increasing pregnancy weight, can lead to obesity, which can complicate the delivery. It also leads to containing sugars can lead to the fatty liver syndrome. It can also affect the fetal metabolism in life ( B.Sc Pharm MD and Erick Yoshida, MD MHSc FRCPC). It increases the risk of preeclampsia, high sugar, high sugar intake influences the risk of preeclampsia in pregnant women (EUR JCLIN NUTR. 2012) Aug. 66(8) 920-5 doi:10.103/ESCN 2012.61. EPUB 2012 Jun 20).

Wonderful kola also known as (buchholzia seeds is quickly gaining popularity as it seems to be a cure to almost all ailments. It’s look stands out from the normal kola as it look like a root and not a fruit like the others.

This plant grows in different states including Congo, Nigeria, and others. This plant is known as evergreen, and its seeds are widely used in medicine. However, more frequently it is used in herbal medicine rather than traditional.

**1.2 Statement of the problem**

The consumption of added sugars (sucrose) over the last 200 years has increased exponentially and parallels the creased prevalence of chronic kidney disease. Data for animals and humans suggest that the consumption of added sugars leads to kidney damage and related metabolic derangements that increase cardiovascular risk. Importantly, the consumption of added sugars has been sound to induce insulin resistance and increase uric acid in humans, both of which increase the conversion of glucose and fructose via the payola pathway. The payola pathway has recently been implicated in the contrition and progression of kidney damage.

To mitigate this serious complications and a negative outcome of kidney disease, the plant Buccholzia coriacea can be used as therapeutic option readily available to cure the damage caused by high sucrose.

**1.3 Objective of the Study**

The general of the, this study was to evaluate the biochemical/metabolic effect of sucrose on a sucrose fed pregnant rat and their offspring’s. The specific objectives are to

1. to induce the rats with sucrose
2. Carryout biochemical assays including, serum urea nitrogen and lipid per oxidation, serum creatinine.

**1.4 Significant of the study**

This study will increase our understanding on the renal effect of buccholzia coriacea on the kidney function indices of a sucrose fed pregnant rats. It is hope that constituent compounds present in buchholzia coriacea would aid further scientific investigation while contributing to the drug discovery and development.

**CHAPTER TWO**

* 1. **Introduction of Wonderful Kola**

Wonderful kola also called buchholziacoriacea was named after R. W. buchholziawho collected the plants in Cameroon in the late 19th century. Young leaves of the plant buchholziacoriacea are used in a gruel poultice for were and boils. In Gabon pounded back of the plant buchholziacoriacea is used as a lotion against scabies, fruit of the plant buchholziacoriacea as an anthelmintic. The seed of buchholziacoriacea has medicinal values.

These seeds gave the plant a name wonderful kola nut because of its usage in traditional medicine. The seeds are covered in purple aril which are Child in Ivory coast and has a pungent taste. It is used to treat a has a pungent taste. It is an ever green under story tree of lowland rain forest, up to 20 meters high accruing in West Africa, From Gruinea to West and east Cameroon and in Gabon. The tree is found in the southern past of Nigeria, Ghana and Liberia. The pulped bark is applied to the chest to treat chest pains and also boils.Wonderful kola is a wonderful herb with incredible healing power hence it is known in Yoruba land in the western part of Nigeria as “Obi Awogbaarun” meaning kola that can cure two hundred diseases).

It is known in the world as memory not as it enhances memory, it cleans blood, facilitates learning ability and strengthens nervous system. It is very rich in vitamin K.

It is found mainly in the Western region of Nigeria and it is very much available in commercial quality in the areas it can be found. It is used in preparation of local herbal therapy for the treatments of various ailments such as cough, typhoid, ulcer diabetes etc.

2.1.1 **Use of wonderful kola**

1. Wonderful kola a lot of active ingredients that can treat and curve many diseases like memory loss, cough, chest pain, waist pain, irregular menstruation, mile, malaria weak erection, hypertensions , premature aging, dysentery, headache, mental mal functioning minor burns, scars, scleroderma, skin conditioner, varicose veins, wound healing, rheumatism, blood diseases, congestive heart failure culinary tract infections, renal diseases etc.

2. wonderful kola also improves blood circulation strengthens veins and capillaries, it purifies blood, it is used for HIV/AIDS treatment, eczema, epilepsy, insanity, hypochondria, hair loss, immures system boosting (cleansing and nourishing) skin conditioner, convulsions, elephantiasis, psoriasis.

3. It combats stress and depression increase libido. It scar formation following surge from anepisiotomies following vaginal delivery of a new born and treatment of external fistulas. It contains tritters periods which strengthens the skins and increases the concentration of oxidizing wounds.

4. It also restores inflamed tissues by increasing blood supply.

**2.1.2 Taxonomy of wonderful kola**

**-** Family: capparacaeJuss

**-** Order: brassicalesbromhead

**-** Class: Eguissetopsida C Agardh

**-** Sub Class: Magnoliidae nor akextakht.

**-** Specite: Coriaceae. Discription

The plant Buchholziacoriamea is a shrub or medium sized tree, evergreen with a dense leaves arranged spirally and clustered at the end of the branches and conspicuous cream white flowers in racemes at the end of the branches.

The back of the plant buchhoziacoriacea is smooth, blackish- brown or dark green. Slashes are deep red turning dark brown, (Akpayung et al (1995) and (Awonters et al (1995).

**2.2 Hyperglycemia**

Hyperglycemia is the medical term describing an abnormally high blood glucose blood sugar level. It can be measured using a glucometer, a small device that allows frequent monitoring of blood glucose levels without the need for a doctor’s office or laboratory.

Hyperglycemia or high blood sugar is a hallmark sign of diabetes both type and type 2 diabetes and pre diabetes. The normal ranges for blood glucose measurements can vary slightly among different laboratories but in genital a fasting early am before breakfast glucose level is considered normal if it is between 70- 100 mg/dl. Glucose level may rise slightly above this range following a meal random blood glucose measurements are usually lower than 12.5mg/dl.

**2.2.1Causes of hyperglycemia**

A number of medical conditions can cause hyperglycemia, but the most common by far is diabetes mellitus. In diabetes, blood glucose levels this either because there is an insufficient amount of insulin in she body or the body cannot use insulin well. Normally, the pancreas releases insulin after a meal so htat the cells of the body can utilize glucose for fuel. This keeps blood glucose levels in she normal range.

Type I diabetes is responsible for about of all causes of diabetes and results from damage to the insulin secreting cells of the pancreas. Types 2 diabetes is far more common and is related to the body’s inability to effectively use insulin.

Other medical conditions that can cause the condition include.

1. Pancreatitis (inflammation of the pancreas)
2. Pancreatic cancer
3. Hyperthyroidism (overactive thyroid gland)
4. Cushing’s syndrome (elevated blood cortisol level
5. Unsual tumors that secrete hormones, including glucagonoma,
6. Severe stress on the body, such as heats attack, stroke, trauma, or severe illnesses, can temporally lead to hyperglycemia
7. Taking certain medications, including prednisone, estrogens, beta-blockers, glucagon, oral contraceptives, phenothiazines, and others, can elevate blood glucose levels.

**2.2.2 Signs and symptoms of Hyperglycemia**

In addition to having elevated levels of glucose in the blood, people with Hyperglycemia often have glucose detected in their urine (Glycosuria). Ordinarily urine contains no glucose because it is reabsorbed by the kidneys.

Another symptom is increased thirst and a frequent need to urinate.

* Headaches
* Hardness
* Blurred vision
* Hunger
* Trouble with thinking or concentrating

Hyperglycemia can lead to damage to organs and tissues. It can also cause nerve damage, vision problem and damage to the blood vessels and kidneys.

2.2.3 **Tests That Diagnose Hyperglycemia**

There are different kinds of blood tests that can diagnose Hyperglycemia. These include:

1. Random blood glucose: This test reflects the blood sugar level at a given point in time. Normal values are generally between 70 and 125 my idea.
2. Fasting blood glucose: This is a measurement of blood sugar level taken in the early morning prior to eating or drinking anything since night before. Normal fasting blood glucose levels are less than 100 mg/dl. Levels are less than 100 mg/dl up to 125 mg/dl suggest pre diabetes.
3. Oral glucose tolerance test:This is a test the measures blood glucose levels at given time point after a dose of sugar is consumed. This test is most commonly used to diagnose gestational diabetes.
4. Glycohemoglobin A/C is a measurement of glucose that is bound to red blood cells and provides an indication about blood sugar level over the past 2 to 3 months.

**2.2.4 Treatment for hyperglycemia**

Mild or treatment Hyperglycemia may not need medical treatment depending upon the cause.

1. People with mild elevated glucose or predicable can often lower their glucose levels by incorporating diet and lifestyle changes.
2. Insulin is the treatment of choice for people with type diabetes and for life threatening increases in glucose levels.
3. People with type 2 diabetes may be managed with a combination of different oral and injectable medications. Some people with type 2 diabetes also take insulin.

**2.2.5 Complication of hyperglycemia**

Long term complication of prolonged Hyperglycemia or high blood sugar can be severe. These occur in people with diabetes and are worse when the condition is poorly controlled. The long term complications of diabetes tend to develop slowly over time some of the complications of hyperglycemia in poor controlled diabetes are:

* Heart and blood vessel disease, that can increase the risk of heart attack, stroke, and peripheral artery disease
* Poor kidney function eventually leading to kidney failure.
* Nerve damage, what can lead to burning tingling, pain and changes in sensations?

In untreated hhyperglycemia, a condition called ketoacidosis may develop because the activity of *hormone sensitive* lipase

The organ function: The kidney performs many crucial functions including.

1. Maintaining overall fluid balance

2. Regulating and filtering measures from blood

3. Filtering waste materials from good, medications and toxic substances

4. Creating hormones that help produce red blood cells, promote bone health, and regulate blood pressure

**2.2.6 Nephrons**

Nephrnons are the most important part of each kidney. They take in blood, metabolize nutrients and help out waste products from filtered blood each products from filtered blood.

2.2.7 **Renal corpuscles**

After blood enters a nepron, it goes into the renal corpuscle, also called a malpithian body. The renal corpuscle contains two additional strictures.

The glomerulus: This is a cluster of capillaries that absorb protein from blood traveling through the renal corpuscle.

The bowman capsule: the remaining fluid called capsular urine,passes through the bow man capsule in the renal tubules.

2.2.8 **Renal tubule**

Proximal convoluted tubule. This section absorbs water, sodium, and glucose back into the blood.

* Loop of Henle : this section further absorbs potassium, chloride, and sodium into the blood
* Distal convoluted tubule: This section absorbs, more sodium into the blood and takes in potassium and acid

HILUM

* Renal artery: this brings oxygenated blood from the heart to the kidney for filtration
* Renal vein: This carries filtered blood from the kidneys back to the heart
  1. **Oxidative Stress In Kidney**

Patients with chronic kidney disease (CKD) have high incidence rates of cardiovascular disease and malignancy. Several factors contribute to these conditions. Structural characteristics in (CKD), loss of renal energy, and uremia result in an imbalance between free radical production and antioxidant defenses.Also, CKD patients usually have multiple cardiovascular risk factors like diabetes mellitus, dyslipidemia, and hypertension. These conditions are associated with oxidative stress, which can trigger the inflammaetory process and accelerate renal injury progression. There are some clinical biomarkers to ducted oxidative stress and antioxidant status in CKD patients.

Antioxidant therapies may be beneficial in reducing oxidative stress, lowering uremic cardiovascular toxicity, and improving survival. Therefore their rooks in CKD patients have been evaluated in several studies as a new target for therapeutic intervention.

Oxidative stress is increased in patients with renal impairment as a result of increased oxidant activity and reduced antioxidant capacity, and this is increased in a granted manner with increasing renal dysfunction.

Inflammation which is also present in CKD, further amplifies the oxidant generation process. [ModaresiA,etal.Iran J Kidney Dis.2015.]

2.4 **Maternal Malnutrition and its Effect on Off springs**

Material malnutrition focuses attention on women as mothers, on their nutritional a status as it relates to the bearing and nurturing of children.

Material malnutrition is a condition that results from pregnant women or mother breast feeding babies eating a diet in which nutrients are either not enough or are too much such diet the diet causes health problems.(malnutrition at Dorland’s Medical Distionary.),(Fact for life(PDF)(4th ed. New York: United Nation Children’s Fund.2010) It may involve calories, protein, carbohydrates, vitamins or minerals (Fact for life(PDF)(4th ed. New York: United Nations Children’s Fund.2010). Not enough nutrients is called under nutrition while too much is called over nutrition. (Yound,E.M.(2012).Food and development.Abingdon,oxon:Routledge.pp.36-38.) Malnutrition is often used to specifically refers to under nutrition where an individual mothers is not getting enough calories, protein, or micronutrients.(Essentials of international Health. Jones and Bartlett Publishers.2011.p.194),(Young, E.M.(2012).Food and development.Abingdon,Oxon:Routledge.pp.36-38.)

There are two main types of material malnutrition which are

1. Under nutrition
2. Over nutrition

Under nutrition: Undernourishments is most often due to not enough high quality food being available to eat.

A lack of breast feeding may contribute, as May a number of infectious diseases such as gastroenteritis’s, pneumonia, malaria, and measles. There are two main types of under nutrition: protein energy malnutrition and dietary deficiencies. Protein energy malnutrition has two severe forms: marasmus (a lack of protein and calories) and kwashiorkor (a lack of just protein). (Young, E.M. (2012).food and development.Abingdon,oxon: Routledge.pp.36-38.)

A common micronutrient deficiency includes. A lack of iron, iodine and vitaminA.(Young,E.M.(2012).foodanddevelopment.Abingdon,oxon:Routledge.pp.36-38.)During pregnancy, due to the body’s increased need, deficiencies may become more common.

**2.4.1 Over Nutrition**

Over nutrition caused by overeating is also a form of malnutrition. It is a category of disease caused by over nutrition can result to obesity and being overweight . In some countries, over nutrition in the form of obesity is beginning to present within the same communities as under nutrition.

From the moment that a woman conceive she holds the responsibility of following a healthy diet in large quantities to support the growth of an entire life inside her womb.

**2.4.2 Effects of on the off springs**

If a pregnant woman is malnourished it is understandable that the in the mother’s womb is not receiving enough nutrients,. In other words, the nutrients and trace mineral essential for developing a whole life are not provided in sufficient amount. As a consequence the baby will exhibit poor growth rate and law weight.

The general effect of malnutrition on the body are:

* Weak immune system
* Greater risk to illness
* Low stamina level
* Babies of low birth weight
* Such children are prone to retarded growth
* Less coordination
* Poor vision
* Premature delivery

2.2.3 **Causes of material malnutrition**

Material malnutrition during pregnancy may be a result of the following factors

1. Lack of a nutrition’s diet in cow income families
2. Painful teeth or mouth condition that may affect ability to consume food
3. Following an unhealthy diet due to lack of knowledge
4. Loss of appetite due to other health condition such as chromic infections, depression etc.
5. Use of certain medications that may interfere with nutrient absorption
6. Diarrhea, nausea, and vomiting may also cause malnutrition
7. Inadequate intake of nutrients and calories that does not meet the increased demands of pregnancy.

**2.2.4 Health risks for the baby**

According to a study, in utero-malnutrition could adversely affect the growth of the baby in the early years. It can also increase his risk of suffering from obesity, diabetes, and other metabolic complication like liver diseases.

Micronutrient deficiency during pregnancy could adversely affect the baby in the following ways micronutrient deficiency during pregnancy could adversely affect the baby in the following ways **.**

* Iodine cy, spastic diplopia, mysoedematexus cretinism, etc. it can also increase infadeficiency can cause congenital abnormalities, neuroticallyevetinism, mental deficiennt mortality risk
* Low zinc levels can cause fatal growth retardation and congenital abnormalities
* Vitamin D deficiency can lead to rickets in the fetus
* A deficiency of floated can cause neural tube defect in the infant
* Calcium deficiency can lead to poor fetal skeletal development
* Low iron levels in the mother’s body can cause fetal growth retardation

Material under nutrition can make a child prove to the following health complications in the congruent.

* Renal disfunction
* Cardiovascular issues life hypertension, atherosckrosis stroke and coronary heart disease
* Osteoporosis
* Organ disfunction of tests ovaries, brain heart liver and final antes use etc.
* Negatively affect mental development and school performance of a child.

**2.2.5 Prevention**

Having a balanced diet can help you enjoyed a safe pregnancy. Consume a lot of fruits and vegetables to gain vitamins minerals and fiber. Include healthy protein sources like fish, eggs, pulses, beans, and poultry in your diet. Also, add starch food like cornmeal, pasta, noodles, bread and potatoes to meet your increased ear hydraterequirements.

You may ask your physical for health supplements during pregnancy. Material malnutrition during pregnancy happens due to insufficient intake of nutrition. By simply following a healthy diet and life style, you can protect your precious baby from malnutrition and various health complication.

**2.5 Dietary Sucrose**

Source is common table sugar. It is a disaccharide, a molecule composed of the two monosaccharide’s, glucose and fructose sucrose is produced naturally in plants, from which table sugar is refined. It had formula.

Appearance –white solid

Density – 1.587 g/cm3, solid

Melting point- 200g/l (2.5c0)

For human consumption, sucrose is extracted, and refined, from either sugar cane or sugar beet. Sugar mills are located where sugar cane is growth to crush the cane and produce raw sugar which is shipped around the world for refining into pure sucrose. Some sugar mills also process the saw sugar into pure sucrose guar beet factories are in colder dimates where the beet is grown and process the into the beets directly into defined sugar.

The sugar refining process involves washing the raw sugar crystals before dissolving them into a sugar syrup which is filtered and then passed over carbon to remove any residual colour. The by now clear sugar syrup is them concentration by boiling under vacuum and crystalized as the final purification process to produce crystal of pure sucrose. These crystals are clear sugar is often an added ingredient in food productionand food recipes.

Physical and chemical properties, in sucrose, the components glucose and fructose are linked via an either bond between on the glucoses subunit and C2 on the functions unit. The band is called a glycosideslinkage glucose exists predominantly as two isomeric pyra noses (A and B), but only one of those ferms links to the fructose.

Fructose itself exists as a mixture of fua-noses, each of which having A and B I source but only one particular B Isomers, but only one particular isomer lick to the glycosyl unit what is notable about sucrose is that unlike most disaccharides, the glycoside bond is formed between the reducing end of one and the nonproducing and of the other. This leakage inhibits further bonding to other saccharide units. Since it cantons no numeric hydroxyl groups it is classified as a non-reducing sugar.

**2.5.1 Hydrolysis of sugar**

Hydrolysis breaks the glycosidic bond converting sucrose into glucose and fructose hydrology is, however, so slow that solutions of fructose can sit for years with negligible change. If the enzyme sucrose is added, however, the reaction will proceed rapidly. Hydrolysis can also be accelerated with acids, such weak acids likewise, gastric acidity converts sucrose to glucose and fructose during digestion, the bond between them being an actual bond fructose during digestion, the bond between them being an actual bond which can be broken by an acid.

**2.2.5 Synthesis and Biosynthesis of sucrose.**

The biosynthesis of sucrose proceeds via the precursors UDP glucoseand fructose 6- phosphate, synthase. The energy for the reaction is gained by the cleavage of undine phosphate sucrose is formed by plants and cyanobacteria but not by other organisms sucrose is found naturally in many food plants along with the monosaccharide fructose. In many fruits, such as pineapple and apricot, sucrose is the main sugar. In other such as grapes and pears, fructose is the main sugar.

Sources: In nature, sucrose is present in may plants, and in particular their roots, fruits and nectars because it serves as a way to store energy, primarily from photosynthesis.

Honeybees are especially important because they accumulate sucrose and produce honey, important foods stuff all over the world. The carbohydrates in hone itself primarily consist of fructose and glucose with trace amounts of sucrose only. As fruits ripen, their sucrose content usually rise sharply.

**2.6 The Effect of Sucrose on the kidney:**

Sucrose is a disaccharide containing two monosaccharide’s glucose and fructose sugar. Sucrose may play a role in chronic kidney disease. Giving sucrose to rats causes kidney swelling and injury. It increases the abnormal loss of protein in the urine.

Excessive sucrose intake should be considered an environmental toxin. There is increasing evidence that fructose is associated with renal disease.

Sucrose are also known to induce renal hyper trophy and tubule interstitial disease in rats. The mechanism may involve two central past ways. First, the rise in uric acid in response to uric acid may cause an afferent alteriolopathy resulting in glomerular hypertension. Second, sucrose may also be filtered into the urine where it is taken up in the segment of the proximal tubule, leading to local intraceuular generation of uric acid with oxidative stress and local inflammation.

The administration of sucrose to rats with reduced renal function can accelerate the progression of renal disease, resulting in worse protein uria, glomenulosclerosis, and tubulointerstitialfibrosis .sucrose intake also hmparis calcium absorption and reduces 25- of vitamin D and 1, 25- dih- droxy. Vitamin D levels in this model. Furthermore, the intake of sugary soft drinks in humans is associated with increased prevalence of albuminurial.

Intake of sucrose causes a reduction in insplamimatory markers.

High blood glucose also called blood sugar can damage the blood vessel in your kidney when the blood vessels are damaged, they don’t work as well.

High level of blood sugar (sucrose) can cause the breakdown of filtering system. It can damage the kidneys and cause them to fail. Thus eliminating their ability to filter out waste, which over time can lead to kidney disease (nephropathy).

Excess glucose in the blood stream can cause the kidneys to filter too much blood over time, this extra work puts more pressure on the nephrons, which often results in them losing their vital filtering ability. The losing of their vital filtering ability is as a result of the damage of the tiny blood capillaries inside the kidneys.

This loss of function can cause useful protein, such s albumin the main protein in the blood to leak out of the kidney and into the urinary system.

**2.7 Diabetes Mellitus**

Diabetes mellitus is not a single disorder; it represents a series of metabolic Condition associated with hyper glycaemia and caused by defects in intuition secretion and for insulin action

* + 1. **Mechanism**

Diabetes mellitus (DM) is a set of related disease in which the body cannot

regulate the amount of sugar in the blood. The blood delivers glucose to pump the body with energy to perform all of a person’s daily activities. The liver converts the food a person eats into glucose. The glucose is then released into the blood stream.

In a healthy person, the blood glucose levels is regulated by several hormones, primarily insulin is produced by the pancreas, a small organ between the stomach and liver. The pancreases also makes other important enzymes released directly into the gut that helps digest food. Insulin allows glucose to move out of the blood into cells throughout the body where it is used for fuel. People suffered diabetes either do not produce enough insulin (Type diabetes) or cannot use inulin properly (types diabetes) or both.

In diabetes glucose in the blood cannot move efficiently the cells, so blood glucose levels become high. This not only starves all the cells that need the glucose for fuel but also harms certain organs and tissues exposed to the high glucose levels.

**2.7.2 Types of diabetes**

There are two main types of diabetes type I and type 2 with their clinical relevance

**2.7.2 Type 1 diabetes**

Types 1 diabetes is an autoimmune disease in which the B- cells of the pancreas do not produce sufficient insulin, a hormone which helps use blood sugar (glucose), for energy. The cell became starved of energy and there will be excess of glucose in the blood. This is the followed by life threatening conditions of hyperglycemia, high blood sugar. When hypoglycemia develops cells do not get enough glucose and patients suffer of confusion, loss consciousness, and coma.

**2.7.3 Type 2 diabetes**

Type 2 diabetes mellitus is a complex endocrine and metabolic disorder. The interaction between several genetic and environmental factors results in a heterogeneous and progressive disorder with variable degrees of insulin resistance and pancreatic – cell dysfunction. Overweight and obesity and major Contributors to the development of insulin resistance and impaired glucose tolerance. When B Cells have not longer able to secrete sufficient insulin to overcome insulin resistance, impaired glucose tolerance progresses to type 2 diabetes. Abnormalities in hormones such as reduced 5 of the incretion glucagon- like peptide, (GLPI), hyper glucose agonaemia, and raised concentrations of other counter- regulatory hormones also contribute to insulin resistance, reduced insulin secretion, and hyper glycaemia in type 2 diabetes overweight and obesity contribute to insulin resistance through several pathways including an imbalance in the concentrations of hormone’s eg. Increased lepton, reduced adiponectiumand increased glucagon, increased concern tratuinsof cytokines egtumour necrosis factor &in terleukin, suppressors of cytokine signaling inflammatory signals, and possible retinal binding protein current alterations in – Cell function often include a period of compensatory hyper insulin acmes with abnormal secretory dynamic when insulin secretion is no sufficient to overcome insulin resistance, glucose intolerance progresses to type 2 diabetes.

The decline in - cell function seems to involve chromic hyperglycemia (glucotoxicity, chromic exposure to non- esterifies fatty acids (lip toxicity), oxidative stress, inflammation and amyloid formation patients with type 2 diabetes usually have pancreatic & cell disfunction that results in creased or no suppressed) glucagon secretions.

**2.7.4 Gestational diabetes**

Gestational diabetes mellitus (GDM) is defined as any abnormal

carbohydrate intolerance that or is first recognized. Pregnancy. It does not exclude the possibility that unidentified glucose intolerance have preceded the pregnant state GDM complicates approximately. Which accounts for more than 2,00,000 cases per year. A recent study from India by sessile et all reported the incidence of GDM as 18. 9%. The clinical importance of GDM lies in the fact that it is associated with significant material and fetal morbidity in she present review we discuss about the pathophysiology screening, Diagnosis, Complications and various management issues partaning to GDM

**2.7.5 Pathophysiology**

Type I diabetes: TIDM is the result of a combination of genetic and environmental influences. It most commonly results from autoimmune destruction of insulin producing B- Cells in the pancreas. Sever birth proposed that one or more environmental factors such as enter viruses, dietary factors or toxins, might trigger the development of T- cell dependent autoimmunity in genetically susceptible individuals. Autoimmunity manifested by detectable antibodies to ICAS512/1A-2, insulin autoantibody (IAA) end glutamic acid decarboxylase (GAD) Insulitis with gradual B cell destructionleads to pre diebetes and finally to over DM. These patients are susceptible to other autoimmune diseases, such as Hashimoto’s thyroidis,celiac disease, Addison’s disease,and myasthenia gravis

Forty genetic loci have been associated with TIDM by a genomic association study and a number of genetic loci in the major his to compatibility (HLA) region are associated with increased susceptibility to developing TIDM, Including the Alleles DR 3/4 , DQ 0300/0302, DR 4/4, and DQ 0300/0302 The risk of TIDMCS approximately 5% if here is an affected first degree relative and slightly higher if the affected parent is the father rather than the mother. To date, international trials have failed to delay the onset or prevent TIDM in those genetically at reverse the progression of TIDM EG )Faitriainet, TRIGR).

**2.7.6 Type 2 diabetes**

Chronic fuel surfeits is the primary pathogenic event that drive development of type 2 diabetes genetically and epigenetically susceptible people many chorionic ally over nourished and overweight or obese individual, however, do not develop diabetes at all or develop it very late in life.

They remain resistant to type 2 diabetes and safely partition excess calories to subcutaneous adipose tissue (SAT) rather than to the heart, skeletal, muscle, liver, and islet cells owing to the following mechanisms successful, islet – cell compensation main tenancies of near normal blood nutrient concentrations, development of minimal insulin resistance, increased expansion of SAT relative to visceral adipose tissue (VAT), and limited increase in liver fat In this way key organs of the body avoid nutrient induced damage susceptible over nourished individuals develop type 2 diabetes owing to the failure of these adapted responseto safely dispose of the fuel surfeits. The following metabolic defects are initial to the development

- cells to compensate for the fuel surfeits, increased glucagon secretion and reduced in certain response, impaired expansion of SAT, hypoadiponectinaemia, and inflammation of adipose tissue, increased endogenous glucose production, and development of principal insulin resistance importantly, the fuel surfeits is not safely disposed of elsewhere. The use where “is less healthy VAT and ectopic” storage in organs, such as the liver, heart skeletal tissue damage worsening islet – cell function can lead to the necessary insulin therapy.

**2.7.7 Blood glucose monitoring**

Children and adolescents to monitor blood glucose at least four times per day (before each meal and at bed time). Maintenance of a blood glucose log book is essential to follow patterns and to make appropriate dose adjustments continuous glucose monitoring technologies have been developed and are increase singly been used clinical care as an adjunct to intermittent monitoring. Hbalc is a measure of glycaemic control over the previous 4el2 weeks week more heartily toward the most Lower HBALC values have been associated with fewer and delayed micro vascular and macro vascular complications. The goal of diabetes management should be to maintain to lowest possible HbAK without server or prolonged hypoglycemia or hyperglycemia.

Treatment options for elderly diabetics

Insulin

Incretions

**2.7.8 New oral agents**

INSULIN

Insulin is the most effective antidiuretic medication when closed appropriately. The projective decline of – cell function with advance age means that the majority if elderly diabetics will require insulin eventually. However, insulin has been underutilized in elderhydiabetics due to concern about hypoglycemia and complexity of administration. Nevertheless, insulin should not be perceived as the last resort in the elderly. At the last time, laboratory value eg blood glucose and HbAlc, should not be the only quire starting insulin. Before- insulin, assessment of psychosocial condition, functional, and cognitive condition, functional and cognitive status of patients is essential for the safe and effective use of insulin. Duapor imitations include inability, to self-administer due to poor vision, impaired cognitions potential for hypoglycemia.

Incret-0 GLP -1 and GLP, are short – lived gut hormones. They enhance the synthesis and release of insulin from pancreatic cells. These incretion hormones are released in response to oral glucose and other nutrient and increase insulin seer sun, attenuate post prandial glucagon secretion and hepatic gluconeogenesis with resulting improvement in post prandial glycaemia. Both GLP and GCP- large rapidly metabolized by the enzyme dipeptide – 4 (DDP-4) resulting in short masma – lives. Incretion based type is a good choice for elderly diabetics because of its low risk of hypoglycemia, a fairly benign side effect profile, and its glucose dependent mechanism of action which units hypoglycemia.

**New Oral Agent**

Bile acid sequestrates: Coleseveram is a bile acid binding resin approved for treatment of hypercholesterolemia. It can also lower blood glucose and can be used in DM treatment. It lowers HbAlc 0.5% when added to met form in, sulfonylurea or insulin. The major side effect of Colesevelam is constipation, this it should be avoided in patents with gastroparesis or other gastrointestinal motility disorder, in patient after major gastrointestinal surgical procedures, and in other to risk for bowel obstruction. The adverse effects include: elevated serum triglycerides and possible malabsorption of fat soluble vitamins.

There are no data in the elderly.

Bromocriptine: bromocriptine (cycloset), a sympatholytic dopamine D2 receptor agonist, was FDA- approved from DM treatment.The dose range is 1.6- 4 mg taken with food in the morning within two hours of awakening. It is used as monotherapy or in combination therapy with insulin and oral agents and a favourable safety profile and tolerability. Its effect on blood glucose may be due to action in the central nervous system. The mechanism of heating of bromocriptine in diabetes has not been clearly elucidated. Side effects include nausea, fatigue orthostatic hypotension head ach. Its efficacy in glycogen control is modest HbLc reduction of 0.1-04 there are no data in the elderly

* 1. **Metabolic Syndrome**

Metabolic syndrome is a cluster of metabolic risk factors that came together in a single individual. These metabolic factors include insulin, resistance, hypertension, cholesterol abnormalities, and an increased risk for blood clotting.

Metabolic syndrome is considered to be a risk factor for cardiovascular deseases and types 2 diabetes.

Metabolic syndrome is also known as syndrome X, insulin resistance syndrome, or dysmetabolic syndrome.

**2.8.1 Symptoms of Metabolic Syndrome**

1. Abdominal obesity : a waist circumference of 102cm (40m) or more in men and 88cm (35inclues) or more in women
2. Serum triglycerides 15o mg/de or above
3. HDL cholesterol 40mg/dl or lower in men and 50mg/dl or lower in women
4. Blood pressure of 130/85 or more
5. Fasting blood glucose of 100mg/dl or above.

**2.9 Oxidative Stress in Metabolic Syndrome**

Oxidative stress is essentially imbalance between the production of free radical and the ability of the body to counteract or detoxify their harmful effects through neutralization by antioxidants.

What are free radicals, a free radical is an oxygen containing molecule that has one or more unpaired electron, making it highly reactive with other molecules.

However, free radicals can chemically interact with cell components such as DNA, protein or lipid and steal their electrons in order to become stabilized. This in turn, destabilizes the cell component molecules which then seek and teal an election from another molecule therefore triggering a large chain of free radical reactions.

Oxidative stress occurs when excess oxygen radicals are produced in cells, which could overwhelm the normal antioxidant capacity. When the concentration of reactive species is not controlled by internal defense mechanisms such as antioxidants (tocopherols) ascorbic acid and ghtathionic) or enzymes involved in oxygen radical scavenging (catalase, peroxidase, and superoxide dismuhase SODI, oxidative damage occurs to proteins, lipids, and DNA, which could lead to cytotoxicity, genotoxicity, and even earcinogenesis when damaged (cululated) cells can proliferate oxidative stress could result from the following.

1. The presence of xenobiotics (ii) Radiation

Metabolic syndrome is often characterized by oxidative stress, a condition in which an imbalance results between the production and activation of Reactive oxygen species. Reactive oxygen species can best be described as double edged swords.

**2.9.1 Oxidative stress**

Is an enhanced susceptibility of biological molecules and membranes to reaction with ROS.

**2.9.2 Oxidative stress in cardiovascular disease:** Cardiovascular disease is the single largest cause of mortality in the population. Cardiovascular disease is a global tem used for a range of disease, which include Islamic heart disease (CHD), cerebrovascular disease (CCVD) and other related diseases, for example myocardial infection Excess free radicals are thought to initiate atheroscerosis by damaging blood vessel walls. LDL- cholesterol has long been implicated in the development of heart disease

However LDC only poses a threat after oxidant by free radicals, as is reported to migrate across the endothelial membrane into the arterial wall. These oxidized components attract macrophages, which absorb and deposit cholesterol within the cell to form what has been referred to as “formation of an atherosclerotic lision.(EL\_Gebali HH et al.).

which can result in blockage of blood vessels. Interruption of the blood supply causes severe pain, known as angina pectoris, and may eventually cause death of the cardiac tissue.

**2.9.3 Oxidative Stress in Aging:**

Aging is a process, which involves the accumulation of changes, which can be attributed to genetic defects, the environment, disease and also the in born aging process. Many pathological condition, are associated with this process, including the development of diabetes and macular degeneration.

Aging is thought to be caused by free radical reaction, generated in the mitochondria, which accumulate with age. Free radicals cause the progressive oxidation of protein and lipid components in the cell membranes and also activate phospholipases, proteases and endonucleases increased lipid peroxization has been implicated in the aging process followed by and induction of detoxification enzymes. Wrinkle formation in the skin has also been attributed to long- term exposure to oxidative damage light, resulting in the production of free radicals (Genova ML. et al.2001).

**2.9.4 Oxidative Stress in Diabetes Mellitus:**

Diabetes mellitus is a common disorder, caused by hyperglycemia resulting from a deficiency in insulin secretion or action.

Diabetes mellitus is associated with increased risk of complications including retinopaphy, kidney failure, nerve damage, circulatory problems, heart disease and stroke. Animal studies have suggested that reactive radicals contribute to the destruction of pancreatic islet cells in the pathogenesis of insulin dependent diabetes mellitus.

Toxic amount of reactive oxygen intermediates are released by endothelial cells and intermediates are released by endothelial cells and infiltrating macrophages during islet inflammation. Islet cell are thought to have deficient defense system against free radical attack making them susceptible to reactive oxygen intermediates, leading to the destruction of the cells. Impaired ascorbic acid metabolism has also been implicated in diabetes. Acerbate is required for the generation of vitamin E in vivo and may be oxidized to dehydroascorbic acid and which can disrupt cell structures and act as a neurotoxin.(Gueri b.et al 2001).

**2.10 Kidney Function Test**

Kidney function test or renal profile is used to screen people who are at risk of developing kidney disease or to monitor the condition of kidney which is already diagnosed with kidney disease.

Those with symptoms which may indicate kidney disfunction such as blood in urine, painful urination, frequent urges to urinate, high blood pressure, selling of hands and feet are required to take renal function test. Kidney tests are very important for people who have diabetes, high blood pressure or heart disease.

**Types of Kidney Tests**

To test your kidney function, your doctor will order a set of tests that can estimate your glomerular filtration rate (GFR). Your GFR tells your doctor how quickly your kidneys are clearing waste from your body.

**Urinalysis:**

A urinalysis screens for the presence of protein and blood in the urine. There are many possible reasons for protein in your urine, not all of which are related to disease.

**Serum creatinine test:**

The blood test examines whether creatinine is building up in your blood. The kidneys usually completely filter creatinine from the blood high level of creatinine suggests a kidney problem

**Blood urea nitrogen (BUN)**

The blood urea nitrogen (BUN) test also checks for waste products in your blood. BUN tests measure the amount of nitrogen in the blood. Urea nitrogen is a breaker down product of protein.

Normally, the kidneys filter out this waste, and urinating removes it from the body.

BUN levels tend to increase when the kidneys or live are damaged. Having too much urea nitrogen in the blood can be a sign of kidney or liver problems.

**CHAPTER THREE**

**METHODOLOGY**

This chapter introduce the material and methods that were used in this study.  
**3.1 Materials**

Glucose strips, glass wares, animal cages, stainless mates, hand towels, razor blade, later eg coves, syringes, sample collection tubes blood collection tubes.

**3.1.1 Chemical and Reagents**

TCA (Trichlroacetic acid), HCl, potassium chloride, petroleum ether, Na2 HPO4, Sodium Chloride DTMB, H2O2, CuSO4, fobricreugent, BSA c borinesermin albumin), metaphosphoric acid,tris base.

**3.1.2 Equipment**

Glucometer, electrical weighing balance, centrifuge, pipette, beaker

**3.1.3 Biological Materials**

Fresh seeds of buchholziacoriacea, 30 albino wister rats (23 femal and 7 male), sucrose

**3.2 Methods**

Collection and identification of samples

Collection of plant material fresh seeds of bucholziacoricca were bought from Ogbaete market, Enugu State metroporist.

The seeds were out into small bits and sun dried. It was grounded into power with an electric mill machine and stored

**3.2.1.2 Collection of Animal**.

Twenty-three adult female and seven adult male albino rats (Wistar strains), weighing between 180-250g were purchased from the animal house; University of Nigeria Nsukka, Enugu State.The rats were housed in the experimental animal handling facility of Godfrey Okoye University and fed with commercially available rat pelleted diet (Ladokun feeds, Nigeria) and water *ad libitum* throughout the period of acclamitization. Virgin female albino rats were randomly assigned to three dietary groups. The diet assigned to an animal before conception was also given throughout the gestation and lactation period. All mice had free access to water throughout the study. We refer to the adult offspring born to dams fed the BCFD diet during gestation and lactation as “BCFD offspring,” and to the adult offspring born to dams fed sucrose diet as “negative offspring.” While offspring’s that received sucrose + BCFD were called sucrose + BCFD offspring’s. Offspring were fasted and euthanized the next day by petroleum ether inhalation and cervical dislocation. Blood was collected by cardiac puncture, and kidney was collected, snap-frozen in ice cold sucrose, and stored at 80°C for later analysis. Urea and Creatinine and lipid per oxidation were determined with the use of an  
autoanalyzer (Konelab 20, Thermo Electron) and lipid peroxidation was performed on the organ.

EXPERIMENTAL DESIGN

The animals were divided into 4 groups of 6 rats each as follows:

* Group A served as the normal group
* Group B served as the sucrose and wonderful kola
* Group C served as sucrose
* Group D served as wonderful kola.

The rate ate normal good after two weed before the introduction of the wonderful kola and sucrose.

**3.2.1: Preparation of diets**

The seeds of *B. coricea* were washed and cleaned thoroughly to remove all detritus; it was dried in the oven (40oC) for two weeks and ground into powder using a miller grinder. 10g of the ground powder was mixed with the rat pellet and served as the BCFD.

**Preparation of** Sucrose **Solution**

Sucrose solution (20%) was prepared daily by dissolving 20 g of Sucrose in 100 ml of tap water. The animals were fed with the 20% Sucrose solution *ad libitum* throughout the period of the experiment. The control groups received normal drinking water *ad libitum* throughout the period of the experiment.

**3.2.3 Collection of Samples**

Blood samples were collected by a snip act at the tail vein and blood sugar level was measured with the accu-check glucometer with strips. The strips were inserted one at a time in the glucometer

PRINCIPLE

When blood is dropped on the red sugared sport on the test strips inserted inside the glucometers, glucose in the blood reacts with the chemical reagents on the test strip.

Glucometer test strip is based double sequential enzyme reaction in which an enzyme, glucose oxidase (God) Converts glucose to hydrogen peroxide and glucoronic acid while peroxidase oxidizes the dye in the test strip to produce a color. The blood glucose level in mildly will be displayed on the screen after 15 seconds. The reduction equation are shown below.

Glucose + O2Glucose oxidase glucose + H2O2

H2O2 +dye peroxidases oxidized does + H2 O2 +H2O

Preparation of the sucrose and wonderful kola for administration

20g of sucrose in 100 ml of water in the sucrose group 20g of sucrose in 100ml of water, 20g of wonderful kola in 80g of feed group in the sucrose and wonderful kola group 20 of wonderful kola in the wonderful kola group.

**3.2.21 Statistical Analysis**

Results were represented as mean ± standard error of mean (SEM) of triplicate readings. Differences between groups was determined by one-way analysis of variance (ANOVA) using statistical package for social sciences (SPSS, version 21.0) followed by Post hoc testing performed for inter group comparisons using the least significance difference (LSD). Significance level was set at p< 0.05.

**BIOCHEMICAL ANALYSES**

DETERMINATION OF CREATININE

Creatinine level was determined using direct endpoint according to Henry (1974) as described in randox commercial kit

Principle :

Creatinines to form a colour complex, which absorbs at 510nm, The rate of formation of colour is proportional to the creatine concentration in the sample. In the endpoint method the difference in absorbance measurements after colour formation yields a creatinine value corrected for interfering substances

Reagent:

Picsic acid (10MM), sodium borate (10mm), sodium hydroxide and surfactant (240mm, creatinine standard (50mglde).

Reagent preparation

Combine equal volumes of picric acid reagent and creatinine buffer reagent and mix well

PROCEDURE

Reagent blank, standard, control and sample test tubes were labeled

2.3 out of working reagent were pipetted into the into the tubes

3.0 iml (100nl) of smaple was transferred to its respective tubes, distilled water to reagent blank and mix

4 all the tubes were placed in 37c0 heating bather for 15minutes.

5 the spectrophotometer was at 510 and zeroed the instrument with the reagent blank the absorbance of all tubes were read and recorded. Creatinine value were calculated

**Calculations**

The creatinine value of unknown (sample) is determined by comparing its absorbance change with that of a known standard

=A sample x concentration of standard

Mylde A standard

DERMINATION OF UREA

Urea level was determined using urease. The ammonia is then measured photometrically by berthelot reaction

Urea+H20 Urease 2NH3 + CO3

NH3+ hypochlorite + phenol indophenol

Reagent

EDTA(RI)(116Mmolk), sodium nitroprusside (6mmoll), sodium mitroprusside (6mmoll), urease (igll), Diluted phenol (Rs) (rommoll), Diluted sodium hypochlorite (R3) (27mmoll), sodium hydroxide (o.4N), area standard preparation of reagents.

r.i sodium nitroprusside (ri) and urease (Ri) solution: transfer the contents of rialRia into bottle Rib and mux gently RS. Phenol:

dicute contents of bottle Rs with 66 mol of distilled water, Rinse bottle thoroughly and mix R3. Sodium hypochlorite.

Dilute contents of bottle R3 with 750ml of distilled water rinse bottle thoroughly and mix.

Procedure:

Test tubes were labeled a blank, standard and sample and pietted into testubeas follow

|  |  |  |  |
| --- | --- | --- | --- |
|  | blank | Standard | Sample |
| Sample | - | - | 10m |
| Standard (cal) | - | 10ul | - |
| Distilled water | 10ml | - | - |
| Reagent 1 | 100ul | 100ul | 100ul |

The above was immediately mixed and in cubated at 37 c for 10 minutes

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Reagent 2 | 250ml | 250ml | 250ml | 250ml |
| Reagent 3 | 25ml | 25ml | 25ml | 25ml |

The above was immediately mixed and incubated at 370 (for 15 minutes the absorbance of the sample (Asmaple) and standard was read against the blank

Calculation

Urea concentration As ample x standard concentrate

A standard c mmollc)

Urea concentration Asample x standard concentrate

A standard

DETERMINATION OF LIPID PER OXIDATION

The level of oxidative damage on the tissue was estimated by guanti flying the amount of fluiobarbitutic acid reactive substances (TBARS) present in the sample following the method described by varnay and kale (1990)

Principle

Malondiadehyde (MDA) which is produced from the peroxidation of membrane fatty acid and food products under acidic condition reacts with chromogenic reagent, 2 tfiobarbituric acid (tba) to give off a pink coloured complex with maximum absorbance at 532nm.

pageants

1. Trichloroacetic acid (TCA 30%): TCA (ag) was dissolved in distilled water and made up to 30 ml with same
2. Thiobarbtituric acid (0.75%): This was prepared hydessolving 0.225g of thiobarbituric acid (TBA) in O IM Hcl and made up to 30ml with
3. Tris-kcl buffer (0.15m,PH7.4): kcl (112g) and 2.36g of tris base were dissolved separately in distilled water and made up to 100 ml with same and PH was then adjusted to 7.4.

PROCEDURE

In a tube containing 0.4ml of the home genate (cytospine) 1.6ml of tris – kcl buffer was added followed by 0.5 ml of 30% TCA. Then, 0.5ml of 0.75% TBA was added and placed in a water bath for 45 minutes at 80c. it was removed and plased on ice and separated using a centrifuge at 300g, using a pipette, the clear supernatant was collected and the absorbance was measured against a reference blank of distilled water at 5.32nm. the level of NIDA was determined according to the method of adam – vizi and seregi (1982).

**CHAPTER FOUR**

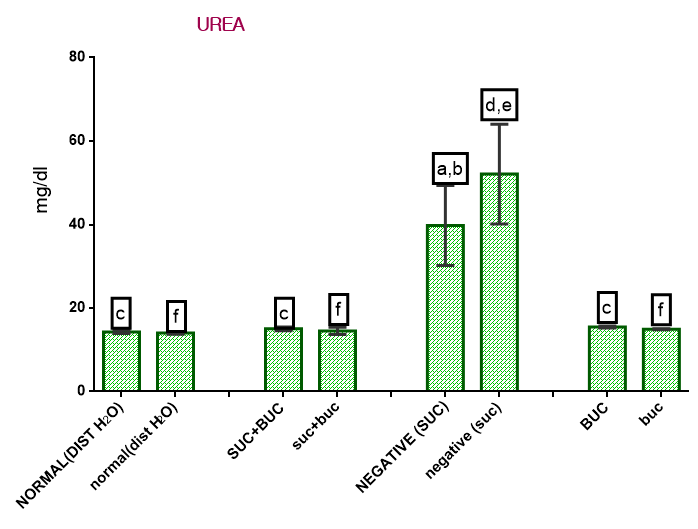


Figure 1:

Effects of buccholzia coriacea on serum Urea levels of normal and sucrose induced pregnant Wistar rats and their offspring’s. Results represented as Mean±SEM. n= 3. ap< 0.05 when compared with normal control group. bp< 0.05 when compared with control SUC + BCFD group. cp< 0.05 when compared with negative control group. dp< 0.05 when compared with control normal group (offspring). ep< 0.05 when compared with control SUC + BCFD group (offspring). fp< 0.05 when compared with control negative group (offspring). BCFD: *b.coriacea formulated diet,* SUC: Sucrose, DIST H2O: Distilled Water.

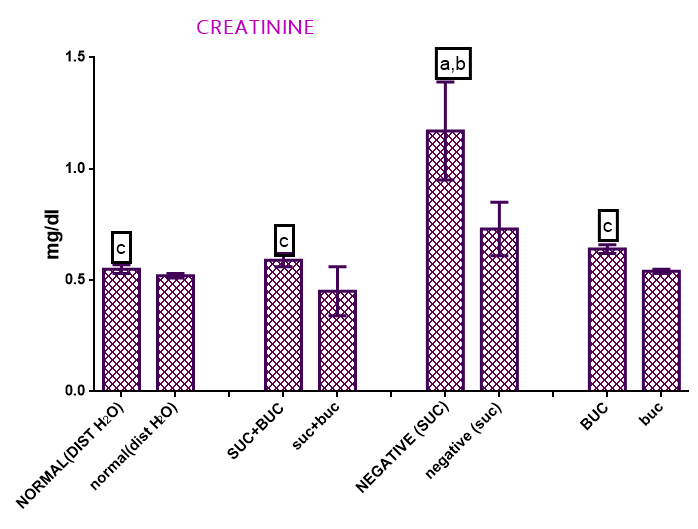


Figure 2:

Effects of *buccholzia coriacea* on serum creatinine levels of normal and sucrose induced pregnant Wistar rats and their offsprings. Results represented as Mean±SEM. n= 3. ap< 0.05 when compared with normal control group. bp< 0.05 when compared with control SUC + BCFD group. cp< 0.05 when compared with negative control group. dp< 0.05 when compared with control normal group (offspring). ep< 0.05 when compared with control SUC + BCFD group (offspring). fp< 0.05 when compared with control negative group (offspring). BCFD: *b.coriacea formulated diet,* SUC: Sucrose, DIST H2O: Distilled Water.

**4.2 Lipid Peroxidation (KIDNEY)**

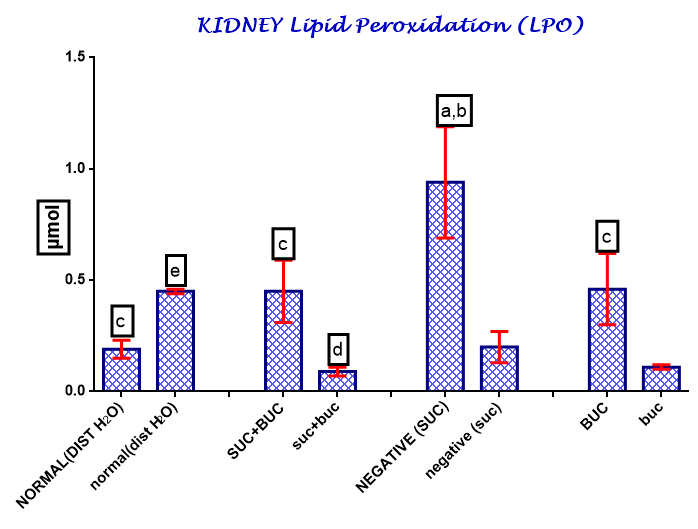


Figure 3:

Effects of *buccholzia coriacea* on Kidney lipid per oxidation of normal and sucrose induced pregnant Wistar rats and their off springs. Results represented as Mean±SEM. n= 3. ap< 0.05 when compared with normal control group. bp< 0.05 when compared with control SUC + BCFD group. cp< 0.05 when compared with negative control group. dp< 0.05 when compared with control normal group (offspring). ep< 0.05 when compared with control SUC + BCFD group (offspring). fp< 0.05 when compared with control negative group (offspring). BCFD: *b.coriacea formulated diet,* SUC: Sucrose, DIST H2O: Distilled Water.

**CHAPTER FIVE**

**DISCUTION AND CONCLUTION**

From figure 1: above, it was find out that the serum urea level of the treated group (group 2) rats did not have a significant increase (p>0.05) compared with the normal group. This is because group 2 rats were fed with buccholzia coriacea. It means , that the healing propertiesof buccholzia coriacea was able to reduce the damage induced by sucrose.

Furthermore, there was a significant increase (p<0.05) of serum urea of the untreated group (group 3) rats compared with the normal groups.

This is because the group 3 was not fed with buccholzia coriacea therefore the damage from sucrose was not controlled thereby allowing the serum urea to increase more than the p value. Also the last group has no significant increase in its urea level.

From figure 2 above, the creatinine level of the second group(Treated group) did not have a significant increase (p>0.05) compared with the normal group. This was because buccholzia coriacea was given to them that was able to control the effect of the sugar on the renal function. Secondly, it is observed that the creatinine level of the group 3(untreated group) was significantly high (p<0.05) compared with the normal group. This was because they were not fed with buccholzia coriacea and the sucrose induced damage allowed serum creatinine to increase.The fouth group also no significant increase (p>0.05) which mean that they was no damage of the renal function.

From figure 3 above, The untreated group (group 3) rats had a significant increase(p<0.05) lipid per oxidation levels measured as malondialdehyde(MDA) in the kidney when compared with the normal control group. Also, MDA levels significantly reduced in the second group (treated group) when compared with normal control group. The group four rats showed lowered (p>0.05) MDA level when compared with normal group.

CONCLUSION:

In conclusion, from the findings in the result buccholzia coriacea was able to control the renal damage induced by the high intake of sucrose. This is because they was no significant increase of serum creatinine,serum urea and lipid per oxidation in the treated group compared to the normal group but there was a significant increase (p<0.05) of serum creatinine level, urea level and lipid per oxidation in the untreated group.

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