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Evaluation of the Antidiabetic Effect of Ethanol Extract of the Unripe Fruit Peel of Musa Paradisiaca (Linn) on Alloxan Induced Hyperglyceamic Albino Rats

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ABSTRACT

Diabetes Mellitus is a major public health problem that is associated with persistent high blood levels that can lead to life threatening complications such as retionopathy, neuropathy and nephropathy. The use of orthodox medicine for managing diabetes is often expensive and is associated with toxic effect. Therefore, the need for drugs from natural sources which are more effective, affordable and less toxic. In this study, the anti-hyperglycemic activity of ethanol whole plant extract of Musa paradisiaca was evaluated in alloxan-induced diabetic rats. The ethanol extract of Musa paradisiaca (250 mg/kg, 500 mg/kg and 1000 mg/kg) were administered to three groups each contain five rats each). The other two groups were administered water and Insulin (0.75 IU/Kg). The results showed that the ethanol extract of the peel of Musa paradisiaca has significant hypoglycemic activity which justifies its traditional use. The phytochemical screening revealed the presence of carbohydrate, saponnins, alkaloida, glycosides and flavonoids. The acute toxicity studies revealed that the plant is safe at dose ≥ 5000 mg/kg.

Keywords: Antidiabetic activities, *Musa paradisiaca*, Alloxan induced hyperglycemia Albino rats

INTRODUCTION

Background of the study

Diabetes mellitus is a chronic metabolic disorder of hyperglycemia which is characterized through disturbances in carbohydrate, protein and fat metabolism resulting from absolute or relative insulin deficiency with disorder in organ system (WHO, 1999). There are three (3) classifications of diabetes namely; Type I diabetes, Type II diabetes, and Gestational diabetes. Type I diabetes which is also known as juvenile diabetes or insulin-dependent diabetes. In this type of diabetes, the pancreas produces little or no insulin (Papadakis et al, 2017). Type II diabetes is also known as adult-onset diabetes which a form of diabetes characterized by high blood sugar, insulin resistance and relative lack or insulin (NIDDK, 2016). Thirdly Gestational diabetes is a condition in which a woman who has no diabetes develops high blood sugar level during pregnancy. This type of diabetes results in in few symptoms. In general, it increases risk of pre-eclampsia, depression, and requiring a Caesarean section (NIDDK, 2016). Insulin is a peptide hormone which is produced by beta cells in the pancreatic islets. It is considered to be the main anabolic hormone of the body. However, lack of insulin can lead to development of diabetes as well as inability to adequately respond to insulin (Voet et al, 2011).

Diabetes mellitus (DM) is since long considered a pandemic and is increasing very rapidly all over the world, and is expected to disproportionately affect the developing world more than the developed world. Even in the early 1900s, Diabetes mellitus was considered a rare disease in the African by Dr. Cook. (kengne *et al*., 2005). However, there are convincing evidence to indicate an upward trend of diabetes mellitus in the continent. (Kengne *et al*., 2005).

Diabetes is thought to affect 1% of people in rural Africa and 5% to 7% of people in urban sub-Saharan Africa (Kengne *et al.*, 2005). With 158 million people, Nigeria is the most populous country in Africa and makes up one-sixth of the continent's total population. Nigeria has 398 recognized ethnic groups and a diverse

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

population, with around 50% of its citizens living in cities (UN, 2012). Out of the three kinds of diabetes mellitus, type II diabetes is the most frequently reported type and makes up between 90% and 95% of all cases in most endocrine clinics. Although there are conflicting findings from different endocrine centers, the recorded prevalence rates of T1DM, which are all hospital-based, range from 0.1/1000 to 3.1/1000. (Ugege *et al*., 2013). For the diagnosis of DM, the World Health Organization (WHO) 1999 criteria apply (ADA, 2009) and the most often used test for DM diagnosis is the fasting plasma glucose test, which is more practical than the oral glucose tolerance test, which is not easily repeatable. The World Health Organization suggested the use of the glycosylated hemoglobin test to diagnose type II diabetes in non-pregnant individuals. A cut-off level of ≥ 6.5% (≥ 48 mmol/mol) was established (WHO, 2011). In order to guarantee that the results obtained from various assays are comparable and trustworthy, the International Federation of Clinical Chemistry standardized tests for measuring HbA1c when it is used for diagnosis. (Mostafa *et al.*, 2010).

Insulin and oral glucose-lowering medications, as well as complementary and alternative medicine in certain cases, make up the pharmacological treatment of diabetes mellitus. About a quarter of people with type II diabetes are on insulin therapy alone or in conjunction with oral glucose medications, as effective insulin use in the management of glycaemia is still difficult in underdeveloped nations like Nigeria. (Kuku *et al.*, 2012).

Review of some Nigerian studies on Diabetes Mellitus

In a study titled "Prevalence of diabetes mellitus and impaired glucose tolerance in a group of urban adults in Nigeria". (Olatunbosun *et al.*,1998), reported in Nigeria that the prevalence of diabetes ranged from low level of 0.8 % among adults in rural highland dwellers to over 7 % in urban Lagos with an average of 2.2 % nationally.

Statement of the problem

Currently, injectable insulin for type I and type II diabetes and traditional oral antidiabetic medications for type II diabetes are the only available treatments for diabetes mellitus. Drug management is currently the cornerstone of diabetes mellitus treatment for all disease types, which has been a major global concern. Orally effective antidiabetic agents are needed, particularly for type I treatment, as insulin is now the only accessible and effective type I antidiabetic agent, although only in injectable form. Additionally, a cure rather than merely management is required.

Diabetes mellitus is a chronic disease with an increasing prevalence worldwide. It constitutes a significant health and socioeconomic burden for patients and the health care systems. According to the WHO, there were 150 million diabetic patients worldwide by the year 2000, with a projection of 221 million people in 2010 and 300 million in 2025. The International Diabetes Federation (IDF) estimates that over 5 million people suffer from the disease in Africa and the number is expected to increase to 15 million by 2025 (IDF, 2006).

The Scope of Study

The scope of the study is to evaluate the preventive and the curative potential use of the ethanol extract of the unripe fruit peel of *Musa Paradisiaca,* in a alloxan induced diabetes mellitus and to determine the short-term adverse effect of the extract using rats and to establish scientific evidence in support of the folkloric claim by traditional medicine practitioners.

Significance of the study

Results obtained by this study will help to further other studies on *Musa Paradisiaca* in diabetes mellitus.

Result obtained from this study may further help to justify the folkloric claim that the unripe peel of *Musa Paradisica* is used in alleviating diabetes mellitus.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

Aim and objective of study

Aim

The aim of this study is to determine the antidiabetic effect of the ethanol extract of the unripe peel fruit of *Musa Paradisica* in alloxan induced diabetes mellitus in rats.

Objective

To determine the phytochemical constituents of the unripe peel extract of *Musa Paradisiaca*

Determine the acute toxicity of ethanol extract of the unripe peel of *Musa Paradisiaca*

To compare the antidiabetic effect of the unripe peel of *Musa Paradisiaca* extract to that of the standard conventional antidiabetic injectable (insulin) using the standard experimental procedure.

Study Of Research Question

Does the ethanol extract of the unripe peel of *Musa Paradisiaca* have antidiabetic effect?

Can the antidiabetic effect of the ethanol extract of the unripe fruit peel of *Musa Paradisiaca* be compared to that of injectable agent (insulin)?

LITERATURE REVIEW

Scientific classification

Scientific name: Musa Paradisiaca (Linn)
Kingdom: Plantae
Division: Tracheophyta
Class: Magnoliopsida
Family: Musaceae
Genius: Musa
Specie: Musa X paradisiaca L. (pro sp.)
Part used: Unripe fruit peel
Common names
English: Plantain
Hausa: Agade
Igbo: Abrika
Yoruba: Boli

Figure 1: A picture of a plantain tree

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

Traditional/ Ethnobotanical

One of the most significant tropical commercial fruits is the plantain. Both the rich and the poor like its inexpensive, high-energy treat. *Musa paradisiaca* is its biological name. It is a member of the Musaceae family. Since ancient times, people have used plantains for a variety of purposes, including food, medicine, religious ceremonies, festivals, and customs. One of the most often consumed fruits is the plantain. Every portion of the plantain plant has therapeutic uses. Plantains provide a lot of health benefits. The fruit, which is rich in iron, is preferred by anemic patients; it has moderate laxative properties, helps to maintain cardiovascular health, protects against strokes and ulcers, and helps to reduce water retention. Antioxidant, antibacterial, anticancer, antidiabetic, and antiulcerogenic qualities are all present in plantains. Along with being a well-known edible fruit and food, it is also high in vitamins and minerals. The flower and peel of this plant is used to treat ulcers, dysentery, and bronchitis and the unripe fruit is good food for diabetics. The astringent ashes of the unripe plantain leaves and peel are used to treat malignant ulcers as well as diarrhea and dysentery (Kumar *et al.,* 2012).

Diabetes Mellitus

Since ancient times, people have been aware of diabetes. The word "diabetes," which means "a siphon" in Greek, was originally used by the physician Aretaeus in the 5th century to refer to the condition as a "melting down of flesh and limbs into urine." The term "mellitus," which is Latin for "honey," was introduced in the 17th century, but Indian physicians in the 5th century BC recorded the sweet, honey-like taste of urine in polyuric patients (madhu meha, or "honey urine") that drew ants and other insects. Diabetes was described in two kinds as early as the 5th century AD: one in older, thicker people and another in thinner, short-lived ones (Karamanou *et al.,* 2016).

Classification of Diabetes Mellitus

The great majority of people with diabetes fall into one of two main categories: Type I diabetes mellitus, which is brought on by an absolute or nearly absolute lack of insulin, or type II diabetes mellitus, which is marked by insulin resistance and insufficient insulin secretion to compensate. Furthermore, gestational diabetes is the term used to describe women who acquire diabetes during pregnancy. Lastly, there are numerous rare and varied forms of diabetes that can be brought on by medications, infections, endocrinopathies, pancreatic damage, and genetic abnormalities.

Type 1 Diabetes Millitus: The autoimmune loss of the beta-cells in the pancreas causes type I diabetes (Pihoker *et al.,* 2005). 90% of people have markers of immunological destruction of the beta-cell at the time of diagnosis, such as antibodies to the islet cell (ICAs), to glutamic acid decarboxylase (GAD65), tyrosine phosphatases IA-2 and IA-2b, ZnT8, and insulin auto-antibodies (IAAs). If a person has only one positive marker, they may turn negative, but the more positive indicators a person has, the higher their chance of type I diabetes. A 75% likelihood of getting diabetes within the next 10 years is linked to two positive antibodies (Skylar *et al.,* 2017). Although it typically affects kids and teenagers, this type of diabetes can strike anyone at any age.

Type II Diabetes Mellitus: Insulin resistance and, initially at least, a relative lack of insulin secretion are the hallmarks of type II diabetes (Defronzo, 1998). Although the plasma insulin concentration (both fasting and meal-stimulated) is often elevated in absolute terms, it is insufficient to sustain normal glucose homeostasis "relative" to the degree of insulin resistance. (Defronzo, 2004). However, as time passes, beta cell loss worsens and insulin insufficiency gets worse. The majority of individuals at risk for type II diabetes, or those with both impaired fasting glucose and impaired glucose tolerance, already have a significant loss, nearly 80% of the pancreatic capacity to secrete insulin, according to more recent, advanced analyses of the beta-cell response and regulation (Defronzo, 2009). A small percentage of people with type II diabetes have severe insulinopenia at diagnosis, but their insulin sensitivity is normal or very close to normal (Banerji *et al.,* 1992).

Gestational Diabetes Mellitus: During pregnancy, glucose intolerance is the initial sign of gestational diabetes mellitus (GDM). The third trimester of pregnancy is when GDM typically first appears in women. The woman

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

should undergo an oral glucose tolerance test and be reclassified as having diabetes, normal glucose tolerance, impaired glucose tolerance, or impaired fasting glucose at least six weeks following the end of the pregnancy. About 8–9% of pregnancies are complicated by gestational diabetes; however, among groups at high risk for type II diabetes, the rates may double (Desisto *et al.,* 2010). Since treatment would lower perinatal morbidity and death, clinical diagnosis is crucial.

Pathophysiology of Diabetes Mellitus

With the exception of smooth muscle, where insulin functions through IGF-1, insulin is the primary hormone that controls the uptake of glucose from the blood into the majority of the body's cells, particularly the liver, adipose tissue, and muscle. Therefore, a key factor in all types of diabetes mellitus is either an insulin shortage or an insensitivity of the insulin receptors (ADA, 2014). Three primary processes provide the body with glucose: the breakdown of glycogen (glycogenolysis), the storage form of glucose found in the liver; intestinal absorption of meals; and gluconeogenesis, the body's production of glucose from non-carbohydrate substrates. (Shoback *et al.,* 2011). Insulin is essential for controlling the body's glucose levels. Insulin can promote the transport of glucose into muscle and fat cells, prevent the breakdown of glycogen or the process of gluconeogenesis, and promote the storage of glucose as glycogen.(Shoback *et al.,* 2011) When blood glucose levels rise, usually after eating, beta cells (β-cells), which are located in the pancreatic islets of Langerhans, release insulin into the blood. About two-thirds of the body's cells utilize insulin to take in glucose from the blood and use it as fuel, to change it into other molecules that are needed, or to store it. Reduced insulin release from beta cells and the conversion of glycogen to glucose are the outcomes of lower glucose levels. The primary regulator of this process is the hormone glucagon, which functions in the opposite manner to insulin (Berret *et al.,* 2012).

Risk factors of Diabetes Mellitus

The type of diabetes determines the risk factors. Type I diabetes mellitus risk factors include:

Environmental factors: Circumstances such as exposure to a viral illness likely play some role in typeI1 diabetes.

Family history: Your risk increases if a parent or sibling has type 1 diabetes.

The presence of damaging immune system cells (autoantibodies): Sometimes family members of people with type I diabetes are tested for the presence of diabetes autoantibodies. If you have these autoantibodies, you have an increased risk of developing type I diabetes. But not everyone who has these autoantibodies develops diabetes.

Geography: Certain countries have higher rates of diabetes Mellitus (Ferri , 2018).

Type II diabetes mellitus risk factors include:

Weight (Obesity): The fattier tissue you have, the more resistant your cells become to insulin.

Inactivity (Lack of Exercise): The less active you are, the greater your risk. Physical activity helps you control your weight, uses up glucose as energy and makes your cells more sensitive to insulin.

Family history: Your risk increases if a parent or sibling has type II diabetes.

Age: Your risk increases as you get older. This may be because you tend to exercise less, lose muscle mass and gain weight as you age. But type II diabetes is also increasing among children, adolescents and younger adults (Ferri, 2018).

Gestational diabetes can occur in pregnant women. Certain women are more vulnerable than others. The following are risk factors for gestational diabetes:

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

Age: Women older than age 25 are at increased risk.

Family or personal history: Your risk increases if you have prediabetes — a precursor to type II diabetes — or if a close family member, such as a parent or sibling, has type II diabetes. You're also at greater risk if you had gestational diabetes during a previous pregnancy, if you delivered a very large baby or if you had an unexplained stillbirth.

Weight: Being overweight before pregnancy increases your risk (Ferri, 2018).

Signs and symptoms

Unintentional weight loss, polyuria (increased urine), polydipsia (increased thirst), and polyphagia (increased appetite) are the hallmark signs of uncontrolled diabetes (Cooke *et al*., 2008). In type I diabetes, symptoms might appear quickly (weeks or months), but in type II diabetes, they often appear considerably more slowly and may be mild or nonexistent (2019, WHO). Although they are not unique to diabetes, a number of additional symptoms can indicate the start of the condition. These include blurred vision, headaches, fatigue, delayed wound healing, and itchy skin, in addition to the ones mentioned above. Long-term elevated blood glucose levels can result in the absorption of glucose by the eye lens, changing its shape and altering eyesight. Diabetic retinopathy can potentially result in long-term vision loss.

Diagnosis of Diabetes Mellitus

Recurrent or persistently elevated blood sugar is a hallmark of diabetes mellitus, which can be diagnosed by any of the following:

Fasting plasma glucose level ≥ 7.0 mmol/L (126 mg/dL)

Plasma glucose ≥ 11.1 mmol/L (200 mg/dL) two hours after a 75gram oral glucose load as in a glucose tolerance test (OGTT)

Symptoms of high blood sugar and casual plasma glucose ≥ 11.1 mmol/L (200 mg/dL)

Glycated hemoglobin (HbA1C) ≥ 48 mmol/mol (≥ 6.5 DCCT %) (WHO, 2011).

If there are no clear signs of elevated blood sugar, a positive result should be verified by repeating any of the aforementioned procedures on a different day. It is preferable to measure a fasting glucose level because of the ease of measurement and the considerable time commitment of formal glucose tolerance testing, which takes two hours to complete and offers no prognostic advantage over the fasting test. Two fasting glucose readings above 7.0 mmol/L (126 mg/dL) is currently defined as diagnostic for diabetes mellitus (Saydah *et al.*, 2001).

Management of Diabetes Mellitus

The goal of managing diabetes is to prevent low blood sugar by maintaining blood sugar levels as close to normal as possible. Dietary adjustments, physical activity, weight loss, and the use of the right drugs (insulin, oral medicines) can typically achieve this.

Nonpharmacological Management

Diet: Maintaining your blood glucose level within the range depends on your food choices, portion sizes, and timing of eating. The best evidence for improving glycemia in people with diabetes has been found when overall carbohydrate intake is reduced. Low or very-low carbohydrate diets are a good option for people with type II diabetes who are unable to meet glycemic targets or where cutting back on anti-glycemic medications is a top priority. For diabetes patients, eating foods like fruits (banana, apple, etc.), eggs, fish, and dairy products (nonfat or low fat), such as yogurt, etc., is beneficial (Rockefeller, 2015).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

Exercise: Exercise lowers cardiovascular risk factors, helps people lose weight, enhances blood glucose control in people with type II diabetes, and enhances overall health. Frequent exercise can either prevent or postpone the onset of type II diabetes. People with type I diabetes can also benefit greatly from regular exercise in terms of their cardiovascular fitness, muscle strength, insulin sensitivity, and other health outcomes.

Pharmacological management of Diabetes Melliutus

Because their bodies are unable to manufacture enough or any insulin at all, patients with type I diabetes mellitus need to get direct injection of insulin. Diabetic therapy for type II diabetics includes any feasible mix of weight loss, exercise, and diet, depending on the patient. After making lifestyle changes, patients who still have poor control over their diabetes are usually put on oral hypoglycemics. Examples include:

Sulphonylureas: In patients with remaining β-cell function, these medicines lower blood glucose by promoting insulin production from pancreatic β-cells. Each has a different half-life and duration of action, and they are all highly absorbed. They include the first-generation agents like chlorpropamide, tolbutamide, tolazamide, and acetohexamide, the second-generation agents like glyburide and glipizide. The third-generation agents like glimepiride. Sulphonylureas work best for patients who were diagnosed with type II diabetes before the age of 40, had the disease for less than five years before starting medication therapy, and had a fasting blood glucose level of less than 3000 mg/L (Ngugi *et al.,* 2012).

Biguanides: These drugs increase the sensitivity of insulin by decreasing hepatic gluconeogenesis (primary effect), increasing skeletal muscle glucose uptake, reducing plasma triglycerides and LDL-Cholesterol levels and increasing peripheral insulin sensitivity (secondary effect). They don't make you gain weight or raise your insulin levels. They don't result in hypoglycemia when taken alone. Metformin which was initially developed from the medicinal herb *Galega officinalis*, is an example of a biguanide. It is used to treat type II diabetes mellitus either alone or in conjunction with sulfonylureas (Ngugi *et al.,* 2012).

α-Glucosidase Inhibitors (α-Gis): For the treatment of type II diabetes, α-glucosidase inhibitors, such Acarbose and Miglitol, are recommended either alone or in conjunction with sulfonylureas. By preventing the breakdown of complex carbohydrates and delaying the absorption of monosaccharides from the gastrointestinal system, these substances lower postprandial glucose levels. They inhibit the action of α-glucosidase, the enzyme responsible for digesting carbohydrates, in the intestine, thus delaying and attenuating postprandial blood glucose peaks. They can be used alone or in combination with insulin, metformin, thiazolidinediones, or sulfonylureas. To reduce side effects, they are taken with food (Ngugi *et al.,* 2012).

Thiazolidinediones: Thiazolidinediones are a special class of "insulin sensitizers" that increase the absorption of glucose by skeletal muscle. They boost insulin sensitivity in the liver and muscles, which lowers plasma triglyceride levels. However, these reductions are linked to weight gain and elevated LDL cholesterol (Ngugi *et al.,* 2012).

Meglitinides: These drugs are short-acting insulin secretagogues. They open calcium channels and promote insulin release via acting on the ATP-dependent potassium channels in pancreatic β-cells. Among these is the medication repaglinide, a derivative of benzoic acid and the first non-sulfonylurea. Meglitinide was introduced in early 1998. Repaglinide's mode of action and adverse effect profile are similar to those of sulfonylureas (Ngugi *et al.,* 2012).

Insulin therapy: When glycemic control is not excellent at the highest dosages of oral medicines, insulin is added to the oral drug. For individuals with recently diagnosed type II diabetes, some diabetologists prefer to start insulin therapy. Hypoglycemia and weight gain are frequent side effects of insulin therapy. There is also a higher chance of atherogenesis with intensive insulin therapy. To encourage glucose utilization, patients with type I diabetes need to take insulin for the rest of their lives (Ngugi *et al.,* 2012).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

Alloxan Monohydrate

When given to rodents and many other animal species, the toxic glucose analog alloxan specifically kills the beta cells in the pancreas, which produce insulin. This results in insulin-dependent diabetes mellitus (also known as "alloxan diabetes") in these animals, which resembles human type I diabetes. Because alloxan preferentially accumulates in beta cells through uptake via the GLUT2 glucose transporter, it is selectively toxic to insulin-producing pancreatic beta cells (Lenzen, 2008). In a cyclic reaction with its reduction product, dialuric acid, alloxan produces reactive oxygen species (ROS) when intracellular thiols are present. Free radicals produced during this redox process are what cause alloxan's beta cell toxicity. According to studies, alloxan does not cause diabetes in people. Others discovered that children with and without Type I diabetes have significantly different amounts of alloxan plasma (Mrozikiewicz *et al.*,1994). Alloxan is used to cause diabetes in experimental animals because it specifically destroys the beta-cells in the pancreas that produce insulin. This most likely happens as a result of the compound's selective uptake because of its structural resemblance to glucose and the beta-cell's extremely effective uptake mechanism (GLUT2) (Szkudelski, 2001).

MATERIALS AND METHODS

Materials

The materials used include: unripe peel of Musa paradisiaca, naso-gastric (NG) tube, test-tube, beakers, petri-dishes, measuring cylinder, round conical flask/round bottom flask, filter paper, weighing balance, filter paper, pestle and mortar, funnel, rotary evaporator, spatula, syringes (1ml, 5ml), analytical weighing balance, hand gloves, animal feed.

Reagents/drugs used

The reagents and drugs used in this research include the following; ethanol, alloxan monohydrate, distilled water and insulin.

Plant collection and identification

The unripe *Musa paradisiaca* fruit was collected in March, 2020 from Enugu metropolitan city, Nigeria. The green peel was removed from the plantain fruit and allowed to air dry in a well-ventilated room for two weeks. The peel was identified and authenticated by a Taxonomist Professor S.S. Sanusi in the Department of Biological Science, Faculty of Science, University of Maiduguri, Borno State, Nigeria.

Preparation of plant extract

The dried peel of *Musa paradisiaca* was size reduced using pestle and mortar in which 512 g of the powder plant material was obtained and, put in a container and macerated for 24 hours with 250 ml of ethanol. It was stirred again and filtered through a filter paper lined funnel into a conical flask. The residue was macerated again with 250 ml of ethanol for 24 hours in order to increase the yield of extract. Then finally, the crude extract was obtained by concentrating the filtrate with a rotary evaporator. All extract obtained was put inside a container and stored in a desiccator.

Phytochemical screening

2 g of the extract was subjected to phytochemical screen to test for presence of the following secondary metabolites: carbohydrate, tannins, alkaloid, flavonoids, saponins and glycosides as described by Trease and Evans(2002), Brain and Turner(1975), Markham (1982), Sofowora (1993).

Test for carbohydrate

General test (molish's): Two drops of molish's reagent was added to the extract dissolved in distilled water. This was followed by addition of 1ml of concentrated tetraoxosulphate (VI) acid (H_2SO_4) by the side of the test tube, so that the acid forms a layer beneath the aqueous layer. The mixture was then allowed to stand for

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

two minutes and then diluted with 5ml of distilled water. Formation of red or dull violet colour at the interface of the layers showed a positive test (Trease and Evans, 2002).

Test for reducing sugar (fehling's test): 0.2g of the extract was dissolved in distilled water and filtered. The filtrate was heated with 5ml equal volume of Fehling solution A and B. Formation of a red precipitate cuprous oxide (Cu_2O) indicate the presence of reducing sugar (Trease and Evans, 2002).

Test for monosaccharide (Barfoed's test): One milliliter of Barfoed's reagent is added to the extract in a test tube and heated on a water bath for 2 minutes. A red precipitate of cuprous oxide is an indication of the presence of monosaccharides (Trease and Evans, 2002).

Test for Tannins

0.5g of the extract was dissolved in 3ml distilled water. The mixture was filtered and the resultant filterate is used for the following test: To 2ml of the filterate few drops of 10% ferric chloride solution are added and occurrence of a blue-black, green or blue-green precipitate shows the presence of tanninsThe filterate of the extract was boiled with 3 drops of 10% HCL and 1 drop of methanol and a red precipitate is taken as an evidence of the presence of tannins.To 2ml of the filterate, a mixture of equal volumes of 10% lead ethanoate was added. Formation of a white precipitate is an indication for the presence of tannins (Trease and Evans, 2002).

Test for alkaloids

0.5g of the extract was stirred with 5ml of 10% aqueous hydrochloric acid on a water bath and then filtered. The filtrate was taken and divided into three portions in the test tube each:

Dragendoff's test: To the first portion, three drops of Dragendoff's reagent are added. A positive result is obtained with the formation of an orange-red precipitate.

Mayer's test: To the second portion, 3 drops of Mayer's reagent was added. A positive result was obtained with the formation of a buff-coloured precipitate as an indication of the presence of alkaloids.

Wagner's test: To the last portion, 3 drops of Wagner's reagent was added. A positive result was obtained with the formation of a dark-brown precipitate as an indication of the presence of an alkaloid (Brain and Turner,1975).

Test for Flavanoids

Shinoda's Test: 0.1g of magnesium chip and a few drops of concentrated HCL was added to the extract dissolved in ethanol, warmed and then filtered. The reaction mixture gives a rose red colouration or red to purple colour, indicating the presence of flavonoids (Markham,1982).

Ferreic Chloride Test: The extract was boiled with distilled water and then filtered. Few drops of 10% ferric was added to 2ml of the filtrate. The appearance of a green-blue or violet colouration was an indication of the presence of phenolic hydroxyl group (Trease and Evans, 2002).

Sodium Hydorxide Test: 0.5g of the extract was dissolved in distilled water and filtered. 2ml of 10% of aqueous sodium hydroxide was added to the filtrate to produce a yellow color. A change in color from yellow to colorless on addition to dilute HCL is an indication for the presence of flavonoids (Trease and Evans, 2002).

Test for Saponins

1g of the extract was boiled with 10ml distilled water filtered, fitered and the filterate is used for the following test below:

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

Frothing test: 3ml of the filtrate was mixed with 5ml distilled water in a test tube. The test tube is stoppered and shaken vigorously for five minutes. The occurrence of a foam column that is persistent for over 5 minutes denotes the presence of saponin (Sofowora, 1993).

Fehling's solution test: 2ml of the filtrate was mixed with 2.5ml of equal volumes of fehlings solution A and B and then heated. The appearance of a brick-red precipitate is taken as the presence of saponin glycosides (Trease and Evans, 2002).

Test for glycosides

0.2g of the extract is put in a test tube and 5ml of dilute sulphuric acid was added and then boiled on a water bath for 10-15 minutes followed by cooling and neutralization with 20% potassium hydroxide. The mixture was then divided in two portions.

To the first portion 5ml of a mixture of fehlings solution A and B was added and boiled. The appearance of a brick red precipitate is an indication of the release of reducing sugar as a result of hydrolysis of glycoside.

To the second portion, 3ml of ferric chloride solution was added and the appearance of a green to blue color is an indication of release of phenolic glycosides.

Experimental animal acclimatization

Adult albino rats of both sexes weighing 78-306g were used for both the acute toxicity studies (LD_{50} determination) and the hypoglycemic effect amounting to the total number of forty-two (42) rats in all. These rats were purchased from the animal house section of the Faculty of Pharmacy, University of Maiduguri, Borno State. The rats were maintained in standard wire meshed iron cages in Pharmacology and Toxicology Laboratory of the Faculty of Pharmacy. The animals were kept in iron cages at standard condition of temperature, light and humidity for a period of two weeks to allow them acclimatize to laboratory condition. These animals were allowed free access to drinking water and standard livestock feed.

Acute toxicity studies (LD_{50} determination)

The acute toxicity of the ethanol extract of unripe peel of *Musa paradisiaca* was determined using standard conventional procedure described by Lorke(1983) in this study, one route of administration was considered, that is, oral route, this comprised 2 phases which include:

Phase I:

The rats were divided into three groups of six rat. The rats were then treated with the ethanol extract of *Musa paradisiaca* at doses 10 mg/kg, 100 mg/kg and 1000 mg/kg body weight orally.

Phase II:

Three doses level were based on the result of phase I after 24 hours for the oral route. Six rats were given 3 doses of the *Musa paradisiaca* extract1600 mg/kg, 2900 mg/kg and 5000 mg/kg respectively. These rats were then observed for 24 hours for signs of toxicity and death after which the LD_{50} (acute toxicity) was reported as appropriate.

Induction of Diabetes and Animal Treatment

Diabetes was induced in overnight fasted rats left for 24hours. Alloxan was then injected intraperitoneally at dose of 150 mg/kg and left for 48 hours. Then their blood glucose level was measured to ascertain if diabetes is successfully induced or not. Then glucose level was taken again at 1 hour, 3 hour, 6 hour, 9 hour, 24 hour and 48 hours respectively. Hyperglycemia was confirmed by the elevated glucose levels in animals using glucometer.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

The animal grouping

Thirty (30) rats were divided into six groups, each consisting of five (5) rats. The extract was dissolved in water and administered orally. The rats were divided into following groups:

GROUP 1: Control group (Untreated rats)

GROUP 2: Diabetic control group (Alloxan untreated rats)

GROUP 3: Positive group (Alloxan rats treated with 0.75 IU/Kg insulin)

GROUP 4: Alloxan rats treated with 250 mg/kg body weight of *Musa paradisiaca* unripe peel ethanol extract

GROUP 5: Alloxan rats treated with 500 mg/kg body weight of *Musa paradisiaca* unripe peel ethanol extract

GROUP 6: Alloxan rats treated with 1000 mg/kg body weight of *Musa paradisiaca* unripe peel ethanol extract

Statistical analysis

The data generated during the study were express in Mean ± Standard Error of Mean (SEM) and analyzed using SPSS student t-test and $P<0.05$ was considered significant.

RESULT ANALYSIS

Phytochemical screening of ethanol extract of the unripe peel of *Musa paradisiaca*

The phytochemical screening of compounds found in *Musa paradisiaca* unripe peel indicates the presence of alkaloids, flavonoids, tannins, glycosides, saponins and carbohydrate. (Table 1)

Acute toxicity study of ethanol extract of the unripe peel of *Musa paradisiaca*

The acute toxicity of ethanol unripe peel extract of *Musa paradisiaca* in rats was carried out at 10 mg/kg, 100 mg/kg and 1000 mg/kg in phase I in which no death was recorded. In phase II, a dose of 1600 mg/kg, 2900 mg/kg and 5000 mg/kg was administered and no death was recorded. This can be said that the acute toxicity of ethanol unripe peel extract of *Musa paradisiaca* is greater than 5000 mg/kg orally (Table 2).

Table 1: Result of preliminary phytochemical screening

S/No	Phytochemical	Test	Inference
1	Carbohydtate	Molish test	+
		Barffoed test	–
		Fehlings test	+
2	Glycoside	Sulphuric acid	+
		Fehling solution	+
		Ferric Chloride	–
3	Tannin	Ferric Chloride	–

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

		HCL and Methanol	–
4	Alkaloid	Dragendoff's test	+
		Mayer's test	–
		Wagner test	+
5	Flavonoid	Shinoda's test	+
		Ferric Chloride	+
		Sodium Hydroxide	–
6	Saponin	Frothing Solution	–
		Fehling's solution test	+

Acute oral toxicity studies

Table 2 shows the study of oral acute toxicity of ethanol unripe peel extract of *Musa paradisiaca*

Experimental phase	Dose(mg/kg)	Observation
Phase I	10	0/2
	100	0/2
	1000	0/2
Phase II	1600	0/2
	2900	0/2
	5000	0/2
LD_{50} ≥ 5000mg/kg		

Physical Observation and Mortality

The toxicity study resulted to no mortality of treatment rats and no toxic effect was observed throughout the 14 days study period. Physical observation of the treated rats throughout the study indicated none of them showed signs of toxic effect such as changes on skin and fur, eye and mucus membrane, behavior pattern, tremors, salivation, diarrhea, sleep and coma.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

Table 3: Effect of ethanol unripe peel extract of *Musa paradisiaca* on glucose levels in alloxan induced diabetic rats

Groups	Baseline glucose level(mg/dl)	Glucose level after 48hrs of alloxan induction(mg/dl)	1hr	3hrs	6hrs	9hrs	24hrs	48hrs
Normal control	107.41±4.11	208.22±6.12	86.80±3.07	98.40±4.08	89.80±3.73	85.20±1.93	94.20±5.142	92.20±4.49
Diabetic control	121.31±9.5	418.11±71.40	565.80±21.37	573.20±17.17	573.20±17.08	574.40±16.38	585.80±14.20	596.80±3.20
Positive control	103.50±5.20	512.39±49.91	62.40±2.89	63.20±1.59	86.00±1.82	98.60±6.93	295.60±54.25	481.80±42.62
MP(250 mg/kg)	112.00±4.31	594.21±71.32	573.00±22.68*	567.00±21.73*	563.40±22.39*	548.60±26.94*	518.40±29.53*	487.20±29.29
MP(500 mg/kg)	109.71±2.21	447.91±86.41	490.20±54.45*	481.20±53.08*	475.80±53.31*	442.00±50.38*	342.60±63.14@	286.80±52.89*@
MP(1000mg/kg)	101.27±4.85	391.11±79.69	505.20±41.65*	467.00±49.46*	451.60±45.58*@	408.60±45.18*@	370.00±33.18@	290.40±31.28*@

Results were expressed as mean±SEM, MP= *Musa paradisiaca*, n=5, One way ANOVA, * = $p<0.05$ (significant compared with insulin). @ = $p<0.05$ (significant with diabetic control)

Glucose level for Treatments (mg/dl)

Normal control rats without alloxan

From the control group (Group 1), the result showed that the basal glucose level is 107.41±4.1 mg/dl. At one hour (1 hr) the blood glucose level was 86.80±3.07 mg/dl. There was a noticeable increase in glucose level to 98.40±4.0 8mg/dl after 3 hrs, there was a drop in glucose level to 89.80±3.73mg/dl after 6hrs, there was a drop in glucose level to 85.20±1.93 mg/dl after 9 hrs, there was a noticeable increase in glucose level to 94.20±5.142 mg/dl after 24 hrs and finally a drop in glucose level to 92.20± 4.49 mg/dl after 48 hrs was observed (Table 3).

Alloxan without treatment (Diabetic control)

Group 2 represents rats induced with Alloxan without treatment. The analysis using the table (figure 4.3) shows that the basal glucose level of 121.31±9.50 mg/dl. There was an increase in glucose level of

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

418.11±71.4 mg/dl after 48 hrs post alloxan induction, then at 1hr after after 48hrs post alloxan induction the glucose level increase to 565.80±21.37 mg/dl, 573.20±17.17 mg/dl at 3 hrs, 573.20±17.08 mg/dl at 6hrs, then an increase in glucose level was observed to 574.40±16.38 mg/dl at 9hrs, an increase in glucose level was observed to 585.80 14.20 mg/dl at 24 hrs, then the glucose level increase to 596.80 3.20 mg/dl at 48 hrs (Table 3)

Post treatment with insulin (as standard)

Since the alloxan-induced diabetes is a model of type I diabetes mellitus, insulin was used as a standard anti-diabetic drug. To compare the efficacy of ethanol unripe peel extract of *Musa paradisiaca* at different doses (250 mg, 500 mg, and 1000 mg, PO) with insulin (0.75 IU/kg, IP), their time-dependent effects on blood glucose were assessed. Group 3 represents rats (n=5) induced with alloxan and treated with insulin (0.75 IU/kg, IP). The average basal glucose level in this set of rats was determined to be 103.50±5.20 mg/dl. Administration of alloxan(150 mg/kg, IP) increased glucose level to 512.39±49.91 mg/dl after 48 hours. Following one hour administration of insulin at 0.75 IU/kg, IP, there was a decrease in glucose level to 62.40±2.89 mg/dl. The glucose level was found to slightly increase to 63.20±1.59 mg/dl after 3hours (Table 3).

Effect of *Musa paradisiaca* unripe peel extract at 250 mg/kg

The average basal glucose level was 112.00±4.3 mg/d. On administration of alloxan(150 mg/kg, IP) the glucose level increased to 594.39±71.32 mg/dl at 48 hours. Following the administration of *Musa paradisiaca* unripe peel extract (250 mg/kg, PO) there was a decrease in glucose level to 573.00±22 mg/dl at 1 hour after the administration of the extract. At 3 hours there was a decrease in glucose level to 567.00±21.73 mg/dl. Similarly, there was a decrease in glucose level to 563.40±22.39 mg/dl at 6 hours after administration of the extract, then there was a decrease in glucose level to 548.60±26.94 mg/dl at 9 hours after the administration of the extract and subsequently decrease to 518.40±29.53 mg/dl after 24 hour of extract administration and ultimately decrease to 487.20±29.29 mg/dl after 48 hours of extract administration (Table 3).

Effect of Musa paradisiaca unripe peel extract at 500 mg/kg

The effect of ethanol *Musa paradisiaca* unripe peel extract (500 mg/kg) on alloxan(150 mg/kg) induced diabetic rats (n=5). The average basal glucose level was 109.71±2.21 mg/dl. On administration of alloxan(150 mg/kg, IP) the glucose level increased to 447.91±86.41 md/dl at 48hours. Following the administration of ethanol *Musa paradisiaca* unripe peel extract (500 mg/kg, PO), there was a decrease in glucose level to 490.20±54.45 mg/dl at 1hour after administration of the extract. Similarly there was a decrease in glucose level to 481.20±53.08 mg/dl at 3 hours after administration of the extract, a decrease to 475.80±53.31 after 6hours of administration of the extract, a decrease to 442.00±50.38 after 9 hours of administration of the extract, then there was a decrease in glucose level to 342.60±63.14 mg/dl after 24 hours of administration of the extract and finally a decrease in glucose level to 286.80±52.89 mg/dl after 48hours of administration of the extract (Table 3).

Effect of *Musa paradisiaca* unripe peel extract at 1000 mg/kg

The effect of ethanol unripe peel extract of *Musa paradisiaca*(1000 mg/kg, PO) on alloxan(150 mg/kg) induced diabetic rat (n=5). The average basal glucose level was 101.27±4.85 mg/dl. On administration of alloxan(150mg/kg, IP) the glucose level increased to 391.11±79.69 mg/dl at 48 hours. Following the administration of ethanol unripe peel extract of *Musa paradisiaca*(1000 mg/kg, PO), there was an increase in glucose level to 505.20±41.65 mg/dl at 1 hour after administration of the extract. There was a decresase in glucose levels to 467.00±49.46 mg/dl at 3 hours after the administration of the extract, then there was a decrease in glucose level to 451.60±45.58 mg/dl at 6 hours after the administration of the extract, a decrease in glucose level to 408.60±45.18 mg/dl was observed at 9 hours after the administration of the extract. Then there was a decrease in glucose to 370.00±33.18 mg/dl at 24 hours after the administration of the extract and finally decrease to 290.40±31.28 mg/dl after 48 hours of administration of extract (Table 3).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VII July 2025

DISCUSSION, CONCLUSION AND RECOMMENDATION

Discussion

Appropriate experimental animal models have provided important information on metabolic, genetic, and environmental risks of diabetes and helped to scrutinize the molecular mechanisms underlying the development, progression, and therapeutic control of this disease (Potenza *et al.*, 2011). This study was carried out to evaluate the hypoglycemic effect of ethanol unripe peel extract of *Musa Paradisiaca* (250 mg/kg, 500 mg/kg and 1000 mg/kg) on alloxan induced rats. The LD_{50} greater than 5000 mg/kg suggest that the extract have low toxicity when administered orally as previously reported by (Adaku et *all*., 2020) and none of them showed signs of toxic effect such as changes on skin and fur, eye and mucus membrane, behavior pattern, tremors, salivation, diarrhea, sleep and coma. The presence of some phytochemical constituent detected in the present study agrees with a report of (Adaku *et al.*, 2020) in which similar phytochemical constituent was found present i.e. alkaloid, flavonoid, saponin, glycosides and carbohydrate. These phytochemicals have been said to influence physiological activities in the body.

The result of the present study shows hypoglycemic effects of ethanol unripe peel extract of *Musa paradisiaca* showed statistically significant decrease ($P< 0.05$) at doses 0f 250 mg/kg, 500 mg/kg and 1000 mg/kg respectively is in agreement with the study carried out by (Adaku *et al.*, 2020) . The maximum decrease in blood glucose level was produced by 500 mg/kg dose.

The results of the present study in which ethanol unripe peel extract of *Musa paradisiaca* showed a significant decrease ($P< 0.05$) in blood glucose level compared with the diabetic control is in agreement with a study carried out by (Adaku *et al.*, 2020).

Conclusion

With respect to the outcome of this research study, the ethanol unripe peel extract of *Musa paradisiaca* contain a lot of phytochemical constituents which may be responsible for the observed hypoglycemic activity. This simply justify the local use of this plant in management of hyperglycemia

Recommendations

Further investigations are needed to elucidate the mechanism action of the chemical constituents of the unripe peel extract responsible for the observed pharmacological activities.

Further studies aimed at isolation, purification and characterization of the active compound(s) responsible for the hypoglycemic activity are hereby encouraged.

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