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# COMPARATIVE STUDY OF THE EFFECTS OF AQUEOUS AND ETHANOLIC LEAF EXTRACTS OF *Musa paradisiaca* L. (Musaceae) ON SOME BIOCHEMICAL PARAMETERS IN RATS

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**ABSTRACT**

The present work was undertaken to evaluate the effects of the aqueous and ethanolic leaf extracts of *M. paradisiaca* on some biomolecules in rats. Five groups of albino rats were respectively treated orally with 250 and 1000mg/kg of ethanolic extract, 250 and 1000 mg/kg of aqueous extract, and saline water daily for 28 days. Blood was collected on the 29th day. Blood serum concentrations of some key liver and kidney marker enzymes and biomolecules were evaluated. The results show that the aqueous extract significantly affected the levels of Aspartate transaminase, Alanine aminotransaminases, High density lipoproteins, total protein and globulins, while the ethanolic extract did not. Neither the aqueous nor ethanolic extract affected the serum level of total lipids and triglyceride. Both extracts however significantly affected the concentrations of $Na^+$, $K^+$ and $Cl^-$. The results of this work indicate the likely hepatotoxicity of the 250 mg/kg aqueous extracts. But both ethanolic and aqueous extracts gave an indication of nephrotoxicity, with 250 mg/kg aqueous extract producing the greatest effect.

**KEYWORDS**: *Musa paradisiaca*, nephrotoxicity, leaf extract.

## INTRODUCTION

*Musa paradisiaca* is a member of Musaceae family and is popularly known as plantain. The plant is widely distributed in the southern part of Nigeria, West and East Africa, Malaysia, Cameroon and Southern parts of United States [1, 2]. The plant is rich in potassium and vitamins B6 and C. several oligosaccharides comprising fructose, xylose, galactose, glucose and mannose are contained in the plant [3]. It is made up of 20% starch (fresh weight) and 1% proteins. The plant contains about 180 aromatic substances, including isopentyl acetate which is the principal aroma bearer. *Musa paradisiaca* is known to have medicinal activity. It is recommended for treatment of urinary stone in Ayurverdic medicine. It has been reported that the stem dissolve pre-formed bones and prevent the formation of stones in the urinary bladder of rats. It is also used in nervous affectations like epilepsy, hysteria and in dysentery and diarrhea [3]. The root is said to have aphrodisiac property and is used for impotency in men. The root sap mixed with honey is also used to treat enlarged prostate. The stem mixed with *Talinum triangulare* leaves is reported to treat measles [4]. Various species of *Musa* have been shown to possess hypoglycemic properties. The flowers and roots of *M. sapientum* showed hypoglycemic effect on normal fasting rabbits [5]. Hydroxylanigorufone, a constituent of the plant showed to be a potential cancer chemo preventive agent [6]. The fruit peel extracts showed mutagenic effect in the peripheral blood cells of Swiss albino mice [7]. In southern Nigeria various parts of *M. paradisiaca* are used as hypoglycemic agents. Unfortunately, in spite of the wide consumption of this plant, literature search gave only scanty information on the toxicity or otherwise of the leaf of this plant. Since both the ethanolic and aqueous extracts of the plant are used, this work was therefore undertaken to assess the effects of both the ethanolic and aqueous extracts of the plant on some biochemical parameters, in order to determine which of the extracts is safer for consumption.

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## MATERIALS AND METHODS

**Collection and Identification of Plant Materials:** The leaves of *M. paradisiaca* were collected from Uyo, Akwa Ibom state, Nigeria in June 2007. The plant was identified and authenticated by Dr (Mrs.) U. Eshiet, a taxonomist in the Department of Botany and Ecological studies, University of Uyo, Nigeria. Specimen voucher was deposited at the Herbarium of the Department of Pharmacognosy and Natural Medicine, University of Uyo, Nigeria.

**Extraction and Drying of Plant Materials:** The leaves were chopped into bits and pounded with mortar and pestle and divided into two portions. The first portion was macerated with distilled water while the second portion was macerated with 96% ethanol for 48 hours. The extracts were filtered using glass wool. The filtrates were then concentrated in vacuo with a rotary evaporator and dried in a desiccator containing silica gel (self indicating) to obtain the aqueous and ethanolic extracts, respectively.

**Animals:** Albino rats (Wistar strain) of both sexes weighing 120±20.0g obtained from the animal house of Nigeria Institute of Trypanosomiasis Research (NITR) Vom, Jos, Nigeria were used. They were kept in the animal house of the University of Uyo under standard conditions of temperature (25 ±2.5$^{o}$C), 12 hour light and 12 hour dark cycle in steel cages. They had free access to water and food (Guinea feed pellets from Edo state, Nigeria). The University guidelines on animal ethics were followed in handling of the animals.

**Administration of Extracts**: Twenty five rats were divided into five equal groups. Groups A, B, C, and D received 250 of the aqueous extract, 1000mg/kg of the aqueous extract, 250 of ethanolic extract, and 1000mg/kg of ethanolic extracts, respectively, once daily through the oral route for 28 days. Group E which served as control received saline water only.

**Collection of Blood**: On the 29$^{th}$ day, blood was collected from the heart of the overnight fasted rats under chloroform anaesthesia. The blood collected was allowed to clot and centrifuged to obtain the serum..

**Estimation of Biomolecules**: Standard methods were used to evaluate the concentrations of Alanine and Aspartate aminotransaminases (ALT and AST), alkaline phosphatase (ALP), conjugated bilirubin, total proteins, albumin, globulin, creatinine, urea, glucose, total lipids, triglycerides, high density lipoproteins:
Estimation of Glucose: This was done using Glucose oxidase method [8].
Alanine Transaminase: The method involves the monitoring of the concentration of pyruvate hydrazone formed with 2,4-dinitrophenylhydrazine [9].
Aspartate Transaminase: The principle of the method involves monitoring of oxaloacetate hydrazone formed with 2,4-dinitrophenylhydrazine [9].
Alkaline phosphatase (Phenolphthalein monophosphate method): This is based on the principle that serum serum alkaline phosphatase hydrolyzes a colourless substrate of phenolphthalein that results in phosphoric acid and phenolphthalein at alkaline pH values. The pinkly coloured product is measured colorimetrically at 550nm.
Triglycerides: This involves the enzymatic colorimetric test of glycerol phosphate oxidase method [10].
Creatinine: Modified Jaffe's method [11] was used. Creatinine which is a hydride of creatine reacts with alkaline sodium picrate to form a red complex which can be determined photometrically.
Total and Conjugated Bilirubin: This is based on colorimetric method by Jendrassik and Groff [12]. Inorganic ions: $Na^{+}$ and $K^{+}$ concentrations were determined using reagent set [13] and $Cl^{-}$ concentration was determined using the mercuric nitrate method [14].

## STATISTICAL ANALYSIS

The data obtained were analyzed using One-Way Analysis of Variance (ANOVA) and Scheffe's post test.

## RESULTS

Results obtained are expressed in Tables 1 and 2. 250 mg/kg of the aqueous extract significantly increased AST and ALT activities, while 1000 mg/kg decreased the activities of the enzymes. Neither 250 nor 1000 mg/kg of the ethanolic extract significantly affected the activities of AST and ALT. 250 and 1000 mg/kg of both the aqueous and ethanolic extracts significantly reduced the activity of ALP (Table 1). 250 mg/kg aqueous extract gave the highest reduction. 250 mg/kg of the aqueous extract also increased the levels of total proteins and globulins but 1000mg/kg aqueous extract did not. Ethanolic extract had no significant effect on the levels of proteins. The level of albumin was neither affected by the aqueous nor ethanolic extract (Table 1).

Both the aqueous and ethanolic extracts elevated glucose concentration significantly. While 250 mg/kg aqueous extract raised the concentration of conjugated bilirubin, 1000 mg/kg of both the aqueous and ethanolic extracts decreased the concentration of conjugated bilirubin. The levels of total lipids and triglycerides were not affected by either aqueous or ethanolic extracts. Both the 250 and 1000 mg/kg of aqueous extracts increased HDL. But in the case of the ethanolic extract, 250 mg/kg reduced while 1000 mg/kg elevated the level of HDL.

Apart from 250 mg/kg aqueous extract which elevated the urea level, 1000 mg/kg aqueous extract and both 250 and 1000 mg/kg ethanolic extract decreased the concentration of urea. Both the aqueous and ethanolic extracts lowered creatinine level. Aqueous extract reduced Sodium ion concentration. However, while 250 mg/kg ethanolic extract elevated Sodium ion level, 1000 mg/kg ethanolic extract lowered the concentration. In the case of Potassium ion, both the aqueous and ethanolic extracts decreased the concentration except 250 mg/kg aqueous

extract which elevated Potassium ion level. Only 250 mg/kg aqueous extract elevated Chloride ion concentration significantly.

## DISCUSSION

Liver disease is the most important cause of increased ALT activity and a common cause of increased AST activity. In hepatocellular injury or necrosis they leak into the circulation and raise the serum level of the enzymes. The major diagnostic use of AST is in myocardial infarction, while ALT is used in viral hepatitis and acute pancreatitis [15]. The effect of extracts of *M. paradisiaca* on the activities of AST and ALT are not consistent. While 250 mg/kg aqueous extract increased activity of AST by 115%, 1000 mg/kg extract reduced the activity by 58 %. A similar trend was obtained on the effect of aqueous extract on the activity of ALT (348 % increase and 57 % decrease by 250 and 1000 mg/kg dose, respectively). On the contrary, ethanolic extract did not affect the activities of AST and ALT. The serum level of total proteins, bilirubin and albumin could also give an indication of liver damage. Hyperbilirubinemia is indicative of impairment of liver function. 1000 mg/kg dose of both aqueous extract caused hyperbilirubinemia. These results show that aqueous extract affected liver function. The low dose of the aqueous extract (250mg/kg) showed a higher indication of hepatotoxicity than the high dose of 1000 mg/kg. The reason for this observation is not clear. Unfortunately, literature search for necessary information on the effect of the leaf of this plant on these biomolecules yielded no positive results. Neither the low nor high dose of the ethanolic extract showed a clear indication of toxicity to the liver. Increased level of ALP is associated with a variety of bone and liver disorders [16]. Both aqueous and ethanolic extracts decreased the activity of ALP, implying that the extracts might have caused some disorders in bone tissue.

Neither the aqueous nor ethanolic extract affected the concentration of total protein, globulin, and albumin, except 250 mg/kg of aqueous extract which increased the level of total protein and globulin by 22 and 34%, respectively. This may also be a consequence of liver disorder.

Hyperlipidemia is one of the most important preventable risk factors for coronary heart disease. Clinical signs of this condition are an increase in the fasting serum cholesterol level or the fasting triglyceride level or both. Neither the aqueous nor ethanolic extract affected the concentration of total lipids and triglycerides. While both 250 and 1000 mg/kg aqueous extracts and 1000 mg/kg ethanolic extract increased HDL level, only 250 mg/kg ethanolic extract reduced HDL level. These results indicate that both the aqueous and ethanolic extracts did not show detrimental effect of the lipid level, except 250 mg/kg aqueous extract whose effect needs to be investigated further.

Both the aqueous and ethanolic extracts elevated serum glucose concentration to similar extent, with the lower dose of 250 mg/kg giving the higher concentration in both cases. This result is consistent with the report that the stem juice of the plant had a hyperglycemic effect in normoglycemic rats [17]. Serum urea, creatinine and electrolytes ($Na^+$, $K^+$ and $Cl^-$) are indices of renal function [18]. Reduction of serum urea, creatinine, $Na^+$ and $K^+$ concentration by both the aqueous and ethanolic extracts indicates kidney dysfunction.

The results of this work show that a low dose of the aqueous extract (250mg/kg) seemed to be hepatoxic while a high dose (1000mg/kg) did not. Also, both the aqueous and ethanolic extracts of the leaf of *Musa paradisiaca* may cause kidney dysfunction when consumed for a prolonged period. 250 mg/kg of the aqueous extract seemed to give the highest toxic effect, while 250 mg/kg of ethanolic extract posed the least threat. In conclusion, a low dose of the ethanolic rather than the aqueous extract of the leaf of *M. paradisiaca* is suggested for use.

**Table 1: Effects of the aqueous and ethanolic leaf extracts of *M. paradisiaca* on some enzymes and proteins in rats**

| Parameter | Aqueous (250mg/kg) | Aqueous (1000mg/kg) | Ethanolic (250 mg/kg) | Ethanolic (1000 mg/kg) | control |
|---|---|---|---|---|---|
| **Aspartate transaminase(U/L)** | 74.8* ± 2.06 | 14.6* ± 0.80 | 30.65± 0.49 | 27.0 ± 0.01 | 34.8 ± 1.94 |
| **Alanine transaminase(U/L)** | 50.0* ±0.02 | 4.82* ± 0.04 | 9.68 ±0.12 | 9.48 ± 0.19 | 11.2 ± 0.08 |
| **Alkaline phosphatase(U/L)** | 81.4* ±0.80 | 148.4* ± 0.49 | 150.2*± 0.40 | 126.4* ±3.20 | 281.2 ± 0.40 |
| **Total protein (g/L)** | 93.0* ±1.55 | 70.6 ± 0.80 | 70.4 ± 0.49 | 65.6 ±2.87 | 76.2 ± 0.98 |
| **Albumin(g/L)** | 45.6 ±2.80 | 35.2 ± 3.49 | 41.4 ±0.80 | 35.2 ±0.75 | 41.8 ±1.33 |
| **Globulin(g/L)** | 42.0 ±0.02* | 31.8 ± 0.40 | 26.6 ±2.80 | 30.8 ±0.40 | 31.4 ±0.80 |
| **Conjugated Bilirubin (Umol/L)** | 7.4 ±0.49* | 2.5 ± 0.04* | 4.74 ±0.32 | 2.42* ±0.16 | 4.68 ±0.27 |

**Mean ± SD n = 5 * $p < 0.05$**

**Table 2: Effects of the aqueous and ethanolic leaf extracts of *M. paradisiaca* on some biomolecules and inorganic ions in rats**

| Parameter | Aqueous (250 mg/kg) | Aqueous (1000 mg/kg) | Ethanolic (250 mg/kg) | Ethanolic (1000 mg/kg) | control |
|---|---|---|---|---|---|
| **Glucose (mg/dl)** | 81.4* ± 0.49 | 76.2* ±0.98 | 82.6*±0.80 | 72.0 *±0.63 | 46.6 ±0.49 |
| **Total Lipids (mmol/L)** | 1.7 ±0.35 | 1.5 ±0.11 | 1.2 ±0.08 | 1.1 ±0.06 | 1.8 ±0.13 |
| **Triglycerides (mmol/L)** | 1.06±0.08 | 1.04±0.08 | 0.79±0.98 | 0.92 ±0.04 | 0.98 ±0.98 |
| **High Density Lipoproteins(mg/kg)** | 0.83*±0.63 | 0.57* ±0.75 | 0.25* ±0.63 | 0.47* ±0.49 | 0.36 ±0.49 |
| **Urea(mg/dL)** | 58.1* ±0.13 | 13.5* ±0.33 | 19.9* ±0.01 | 24.1* ±1.60 | 34.9 ±0.04 |
| **Creatinine(mmol/L)** | 1360.6*±0.80 | 210.2* ±0.40 | 750..0* ±0.01 | 808.6* ±0.80 | 1035.2 ±2.14 |
| **$Na^+$(mmol/L)** | 181.0* ±0.89 | 170.8* ±1.17 | 251.0* ±1.55 | 171.2* ±0.40 | 202.4 ±0.80 |
| **$Cl^-$ (mmol/L)** | 100.0* ±0.49 | 94.0 ±0.01 | 91.0 ±0.01 | 90.8 ±0.40 | 94.6 ±1.20 |
| **$K^+$(mmol/L)** | 7.28*±0.16 | 4.66* ±0.17 | 5.74* ±0.08 | 5.82* ±0.12 | 6.16 ±0.08 |

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