Use of unripe plantain (Musa paradisiaca) in the management of diabetes and hepatic dysfunction in streptozotocin induced diabetes in rats

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Abstract: Aim: This study aims to investigate the effect of unripe plantain (Musa paradisiaca) on markers of hepatic dysfunction in streptozotocin induced diabetic rats. Methods: Blood glucose; relative liver weight (RLW); relative kidney weight (RKW); relative heart weight (RHW); relative pancreatic weight (RPW); serum and hepatic serum aspartate transaminase (AST), alanine transaminase (ALT), and alkaline phosphatase (ALP); serum amylase, lipase, total, and conjugated bilirubin; and chemical analysis of the test feed were determined using standard techniques. Results: The diabetic rats had significant alteration \((P < 0.05)\) of blood glucose; RLW; RKW; RPW; serum and hepatic AST, ALT, and ALP; serum total and conjugated bilirubin; and serum lipase activities compared with nondiabetic while these parameters were significantly improved \((P < 0.05)\) in the rats fed unripe plantain. There were no significant differences \((P > 0.05)\) in the RHW of the rats in the three groups, as well as significant decreases \((P < 0.05)\) in the amylase levels of the diabetic rats compared with the nondiabetic, but there was nonsignificant increase \((P > 0.05)\) in the amylase levels of the rats fed unripe plantain compared with the nondiabetic rats. The test and standard rat feeds contained considerable amount of proteins, carbohydrates, fats, phenols, and crude fiber. Conclusion: Amelioration of acute pancreatitis by unripe plantain could play a key role in its management of diabetes and related complications.

Keywords: diabetes, unripe plantain, streptozotocin

Introduction

Diabetes mellitus is a global metabolic disease that affects essential biochemical pathways in the body resulting to complications. Excess hepatic glycogen accumulation is seen in 80% of diabetic patients due to impaired glycogen synthesis and this could be a leading cause of liver disease in diabetics. Patients showing solely excessive glycogen deposition may exhibit hepatomegaly and liver enzyme abnormalities which are improved with sustained glucose control [1].

Though synthetic drugs have been useful in the management of this disease, their use is limited by the side effects associated with them as well as the enormous cost they pose on the economies of developing nations. Moreover, these therapies only partially compensate for metabolic derangements seen in diabetics and do not necessarily correct the fundamental biochemical lesions [1]. Therefore, the need for alternative therapies cannot be overemphasized.

Plantain is cultivated in many tropical countries of the world, and it is known to be rich in iron, fiber, vitamins, minerals, and serotonin [2].

In folklore medicine, unripe plantain is useful in the management of diabetes, treatment of anemia, and liver disorders (independent of diabetes) [3, 4].

Although the antidiabetic potentials of unripe plantain on animal models have been reported [5, 6], the biochemical basis of its folkloric use in the management of diabetes and liver dysfunctions has not been fully investigated. In addition, there is paucity of information in literature on the effect of unripe plantain on hepatic dysfunction arising from diabetes or other sources (alcohol, viral hepatitis or demographic factors). Since the
incidence of hepatic complications arising solely from diabetes mellitus is gradually on the increase and though it is most prevalent in patients of type 2 diabetes, results obtained from this study could provide an insight into the prospects of unripe plantain in the management of hepatic dysfunction arising from type 2 diabetes.

This research was therefore setup to study the effect of unripe plantain on blood and urinary glucose, serum amylase, lipase, body weights, relative liver, kidney, pancreas and heart weights, serum total and conjugated bilirubin, serum and hepatic aspartate amino transaminase (AST), alanine amino transaminase (ALT) and alkaline phosphatase (ALP) activities in streptozotocin induced diabetic rats.

Materials and Methods

Plant materials

The unripe plantain (*Musa paradisiaca*) variety, locally known in the Eastern part of Nigeria as seed plantain was obtained from Umuahia main market in August, 2013. It was identified by Mr. Ibe of the Forestry Department, Michael Okpara University of Agriculture, Umudike, Nigeria (MOAU). The plant was deposited in the herbarium of MOUAU for authentication.

Reagents/chemicals

The streptozotocin (STZ) that was used was obtained from Sigma and Aldrich Chemical Company, United Kingdom. The bilirubin, amylase and lipase assay kits used were purchased from Biosystems, Barcelona, Spain. The AST, ALT, and ALP kits used were products of Randox.

Processing of the plant material

The samples were properly peeled, soaked in water for about 10 min, washed, and oven dried at 70 °C to constant weight and processed to flour. The processed flour was pelleted and oven dried at 80 °C to constant weight before it was fed to the rats.

Animal experiments

Selection of animals

Forty-eight male albino rats of the Wistar strain (141–243 g) obtained from the animal house of the Department of Biochemistry, University of Nigeria, Nsukka, Enugu State, Nigeria, were used for the study. The animals were kept in metabolic cages in the animal house of the Department of Biochemistry, MOUAU. They were acclimatized for 2 weeks to their diets prior to the commencement of the experiment and were maintained under a constant 12-h light and dark cycle and at room temperature. All animal protocols were approved by the ethical committee of MOUAU which was in line with the principles of laboratory animal care as designated by the National Institute of Health’s Principles [7].

Induction of diabetes

After 2 weeks of acclimatization, freshly prepared solution of streptozotocin (0.1 g dissolved in 5 mL of freshly prepared sodium citrate buffer 0.1 M, pH 4.5) was injected intraperitoneally to forty-two of the rats at a dosage of 65 mg/kg body weight at fasting state while six of the remaining rats served as the nondiabetic group and received standard rat pellets. Blood was collected from the tail vein, and blood glucose concentration was analyzed in the STZ-treated rats prior to the commencement of the dietary feeding using a blood glucose meter (Double G glucometer, USA) and subsequently, twice in a week, throughout the duration of the experiment. The STZ-treated rats with fasting blood glucose levels >200 mg/dL, after 12 days of induction of STZ and with evidence of glycosuria, were considered to be diabetic and used for the study.

Experimental procedure

The STZ-treated rats with stable diabetic condition were then divided into 2 subgroups (groups 2 and 3) comprising of six animals per group while the nondiabetic group formed the first group:

*Group 1.* Normal rats fed standard rat pellets (nondiabetic control)

*Group 2.* Diabetic control rats fed standard rat pellets

*Group 3.* Diabetic rats fed unripe plantain pellets (81%)

Their diets and water were both administered *ad libitum* for 28 days, after which, the rats were stunned by blow and sacrificed and their blood samples were collected in non-anticoagulant tubes for serum assay of liver and kidney function parameters. The liver, kidney, and heart were also collected and weighed. The body weights of the rats were recorded on a daily basis using an electronic weighing balance (Model Scout Pro, Ohaus Corporation, USA), and the percentage change in weight was calculated as:

\[
\text{Percentage change in weight} = \frac{\text{Final weight} - \text{Initial weight}}{\text{Initial weight}} \times 100.
\]
Similarly, the percentage change in fasting blood glucose was calculated as:

\[
\text{Percentage change in fasting blood glucose (FBG)} = \frac{\text{Final FBG} - \text{Initial FBG}}{\text{Final FBG}} \times 100.
\]

The relative tissue weights were expressed in g/100 g and were calculated as:

\[
\begin{align*}
\text{Relative liver weight (g/100 g)} &= \frac{\text{Total liver weight}}{\text{Final body weight}} \times 100, \\
\text{Relative kidney weight (g/100 g)} &= \frac{\text{Total kidney weight}}{\text{Final body weight}} \times 100, \\
\text{Relative pancreatic weight (g/100 g)} &= \frac{\text{Total pancreatic weight}}{\text{Final body weight}} \times 100, \\
\text{Relative heart weight (g/100 g)} &= \frac{\text{Total heart weight}}{\text{Final body weight}} \times 100.
\end{align*}
\]

**Determination of marker enzyme activities in the liver**

After weighing, the liver was quickly washed with ice-cold physiological saline and stored at −20 °C until analyzed. Ten percent homogenate (w/v) of the liver was prepared in 150 mM KCl using a homogenizer at 4 °C and centrifuged for 15 min at 4 °C [10], and the supernatant was analyzed for hepatic AST, ALT, and ALP.

**Determination of biochemical parameters in the sera**

The serum AST, ALP and ALT levels of the rats were determined spectrophotometrically at 540 nm using Randox kits method as described by Gutmann and Bergmeyer [9]. The total and direct (conjugated) bilirubin levels of the rats were determined using the Biosystems diagnostic kits, and the principle was based on the reaction of the serum bilirubin with diazo reagent to form a colored complex which is measured at 540 nm with a spectrophotometer [10]. The serum amylase and lipase levels of the rats were also determined using Biosystems diagnostic kits using the methods described by previous researchers [10, 11].

**Preparation of the extract of the feeds for total phenolic assay**

Six grams each of the flours of unripe plantain and standard rat feed were soaked in 60 mL of water and left overnight. The mixture was filtered (Whatman No. 1) and centrifuged at 3000 × g for 10 min for the assay of the phenolic contents of the flour.

**Chemical analysis of the flours**

The total phenolic contents of the extracts of the flours were determined using the Folin–Ciocalteu method as described by Singleton et al. [12]. Gallic acid was used as the standard, and results were expressed in mg gallic acid equivalents (GAE)/100 g. The percentage protein, fat, and crude fiber contents of the flours of the test and standard rat feeds were determined using the methods of AOAC [13].

**Statistical analysis**

Data was subjected to analysis using the Statistical Package for Social Sciences (SPSS), version 17.0. Results were presented as the means ± standard deviations. One-way analysis of variance (ANOVA) was used for comparison of the means. Differences between means were considered to be significant when \( P < 0.05 \).

**Results**

The administration of STZ at a dosage of 65 mg/kg body weight to the rats of group 2 and 3 produced stable diabetic condition within 12 days in most of the experimental rats. Intake of unripe plantain by the dia-
betic rats of group 3 resulted in 159.52% decreases in blood glucose compared with the diabetic control rats (Table I).

The diabetic rats had elevated levels of glucose and protein in their urine samples by the 1st and 2nd weeks of the experiment. However, by the last week of experimentation, the rats fed unripe plantain had decreased levels of these parameters in their urine samples (Table II).

There were no significant differences (P > 0.05) in the relative heart weights of all the rats in the 3 groups (Table III).

The diabetic control rats had significant increase (P < 0.05) in their relative liver and kidney weights but significant decrease in their relative pancreatic weights compared with the non diabetic rats (Table I). Feeding of unripe plantain to the diabetic rats of group 3, resulted in significant (P < 0.05) amelioration of their relative liver, kidney, and pancreatic weights compared with the diabetic control rats.

The feed composition that was given to the group 3 diabetic rats comprised of 81% unripe plantain flour, 9% soya bean flour, 4% vitamin mixture, 2% salt, and 4% groundnut oil.

The diabetic rats fed unripe plantain recorded 24.91% loss of weight, compared with diabetic control rats that recorded 45.36% loss of weight unlike the nondiabetic rats that recorded 13.42% gain in weight (Table IV).

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### Table II

<table>
<thead>
<tr>
<th>Groups</th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
<th>Week 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>Prot: Nil</td>
<td>Trace</td>
<td>30 mg/dL</td>
<td>30 mg/dL</td>
</tr>
<tr>
<td>Gluc: −ve</td>
<td>−ve</td>
<td>−ve</td>
<td>−ve</td>
<td>−ve</td>
</tr>
<tr>
<td>Group 2</td>
<td>Prot: 30 to 100 mg/dL</td>
<td>30 to 100 mg/dL</td>
<td>30 to 100 mg/dL</td>
<td>30 to 200 mg/dL</td>
</tr>
<tr>
<td>Gluc: Trace to +</td>
<td>2+</td>
<td>2+</td>
<td>2+ to 3+</td>
<td></td>
</tr>
<tr>
<td>Group 3</td>
<td>Prot: 30 to 100 mg/dL</td>
<td>30 to 100 mg/dL</td>
<td>Trace</td>
<td>30 mg/dL</td>
</tr>
<tr>
<td>Gluc: Trace to +</td>
<td>Trace to +</td>
<td>−ve</td>
<td>−ve</td>
<td></td>
</tr>
</tbody>
</table>

Prot = protein; Gluc = glucose; −ve = negative or absent; +ve = positive

### Table III

<table>
<thead>
<tr>
<th>Groups</th>
<th>Relative heart weight</th>
<th>Relative liver weight</th>
<th>RKWT</th>
<th>RPWT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>0.42 ± 0.07&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.49 ± 0.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.73 ± 0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.54 ± 0.12&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group 2</td>
<td>0.45 ± 0.03&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.21 ± 0.13&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.99 ± 0.06&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.36 ± 0.03&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group 3</td>
<td>0.44 ± 0.04&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.23 ± 0.77&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.72 ± 0.13&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.48 ± 0.03&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SD. <sup>a,b</sup>Means with different superscripts (column) are significantly different (P < 0.05). RKWT – relative kidney weight; RPWT – relative pancreatic weight

### Table IV

<table>
<thead>
<tr>
<th>Groups</th>
<th>Initial weight</th>
<th>Final weight</th>
<th>Percentage change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>175.75 ± 24.58&lt;sup&gt;a&lt;/sup&gt;</td>
<td>203.00 ± 13.04&lt;sup&gt;c&lt;/sup&gt;</td>
<td>13.42 (increase)</td>
</tr>
<tr>
<td>Group 2</td>
<td>169.20 ± 28.52&lt;sup&gt;a&lt;/sup&gt;</td>
<td>116.40 ± 15.63&lt;sup&gt;a&lt;/sup&gt;</td>
<td>−45.36 (decrease)</td>
</tr>
<tr>
<td>Group 3</td>
<td>183.00 ± 32.49&lt;sup&gt;b&lt;/sup&gt;</td>
<td>146.50 ± 37.46&lt;sup&gt;c&lt;/sup&gt;</td>
<td>−24.91(decrease)</td>
</tr>
</tbody>
</table>

Values are means ± SD. <sup>a–c</sup>Means with different superscripts (column) are significantly different (P < 0.05)

### Table V

<table>
<thead>
<tr>
<th>Groups</th>
<th>AST</th>
<th>ALT</th>
<th>ALP</th>
<th>Total bilirubin</th>
<th>Conjugated bilirubin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>15.56 ± 13.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>12.96 ± 11.21&lt;sup&gt;a&lt;/sup&gt;</td>
<td>35.65 ± 6.14&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.55 ± 0.09&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.37 ± 0.12&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group 2</td>
<td>41.90 ± 12.33&lt;sup&gt;c&lt;/sup&gt;</td>
<td>25.11 ± 17.56&lt;sup&gt;b&lt;/sup&gt;</td>
<td>55.30 ± 2.41&lt;sup&gt;c&lt;/sup&gt;</td>
<td>1.36 ± 0.20&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.78 ± 0.16&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group 3</td>
<td>29.61 ± 4.57&lt;sup&gt;b&lt;/sup&gt;</td>
<td>11.68 ± 9.85&lt;sup&gt;a&lt;/sup&gt;</td>
<td>47.99 ± 11.06&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.67 ± 0.14&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.47 ± 0.16&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are means ± SD. <sup>a–c</sup>Means with different superscripts (column) are significantly different (P < 0.05). AST, ALT, ALP – U/L; bilirubin – mg/dL
There were significant increases ($P < 0.05$) in the serum AST, ALT, ALP, total, and conjugated bilirubin contents of the diabetic control rats compared with the nondiabetic rats but significant decreases ($P < 0.05$) in the serum AST, ALT, ALP, total, and conjugated bilirubin contents of the diabetic rats fed unripe plantain compared with the diabetic control rats (Table V).

There were significant decreases ($P < 0.05$) in the hepatic AST, ALT, and ALP activities of the diabetic control rats compared with the nondiabetic rats but significant increases ($P < 0.05$) in the hepatic AST, ALT, and ALP activities of the diabetic rats fed unripe plantain compared with the diabetic control rats (Table VI). In addition, there were no significant differences in the hepatic AST activities of the diabetic rats fed unripe plantain compared with the nondiabetic rats (Table VI).

There were significant ($P < 0.05$) increases in the serum amylase activities but significant decreases ($P < 0.05$) in the serum lipase activities of the diabetic control rats compared with the nondiabetic rats (Table VII). There were nonsignificant increases ($P > 0.05$) in the serum amylase activities of the diabetic rats fed unripe plantain compared with the nondiabetic rats but significant increases in the serum amylase activities of the diabetic rats fed unripe plantain compared with the diabetic control rats. The serum lipase activities of the diabetic

| Table VI | Liver function parameters in the liver of rats (U/L) |
| Groups | AST | ALT | ALP |
| Group 1 | 24.68 ± 12.54$^b$ | 21.68 ± 2.78$^c$ | 47.38 ± 5.14$^b$ |
| Group 2 | 12.12 ± 2.27$^a$ | 12.03 ± 0.36$^c$ | 18.77 ± 2.46$^a$ |
| Group 3 | 28.61 ± 8.96$^b$ | 16.76 ± 3.99$^b$ | 55.66 ± 18.87$^c$ |

Values are means ± SD. $^{a-c}$Means with different superscripts (column) are significantly different ($P < 0.05$).

| Table VII | Serum amylase and lipase activities of rats (U/L) |
| Groups | Amylase | Lipase |
| Group 1 | 109.28 ± 20.93$^b$ | 11.60 ± 6.10$^c$ |
| Group 2 | 81.44 ± 2.24$^a$ | 45.85 ± 8.46$^b$ |
| Group 3 | 116.94 ± 13.64$^b$ | 21.60 ± 13.51$^c$ |

Values are means ± SD. $^{a-c}$Means with different superscripts (column) are significantly different ($P < 0.05$).

![Fig. 1.](image) Chemical composition of unripe plantain pellets. Phenol – mg gallic acid equivalent/g (mg GAE/g)
control rats were significantly increased ($P < 0.05$) compared with the nondiabetic rats. Intake of unripe plantain by the diabetic rats of group 3 resulted in significant decrease ($P < 0.05$) of their serum lipase activities compared with the diabetic control rats (Table VII).

Proximate analysis of the unripe plantain incorporated feed and standard feed showed that the unripe plantain incorporated feed contained 55.60 ± 0.16% carbohydrates, 14.68 ± 0.40% protein, and 17.59 ± 0.19% fat while the standard rat feed contained 31.55 ± 2.62% carbohydrates, 15.00 ± 0.78% protein, and 7.24 ± 1.20% fat.

Phenolic and crude fiber analysis of the unripe plantain incorporated and standard rat feed revealed that the unripe plantain incorporated feed contained 7.50 ± 1.73 mg gallic acid equivalent/g of sample and 6.77 ± 0.05% crude fiber while the standard rat feed contained 0.12 ± 0.04 mg gallic acid equivalent/g of sample and 10.00% crude fiber (Fig. 1).

**Discussion**

The normalization of blood glucose to the extent that was observed in this study suggests the antihyperglycemic actions of unripe plantain.

The excretion of large amounts of glucose by the last week of experimentation in the urine samples of the diabetic control rats was an indication that their renal threshold for glucose was exceeded while the absence of glucose by the last week of experimentation in the urine samples of the diabetic rats fed unripe plantain further affirms the antidiabetic potentials of unripe plantain.

The development of diabetic nephropathy is characterized by a progressive increase in urinary protein and a late decline in glomerular filtration rate, leading to end stage renal failure [14]. Thus, the considerable amount of proteins in the urine samples of the diabetic control rats by the last week of experimentation is a pointer of progression towards the development of renal complication while the trace amount of proteins in the urine samples of the diabetic rats fed unripe plantain further confirms the antidiabetic potentials of unripe plantain.

The development of diabetic nephropathy is characterized by a progressive increase in urinary protein and a late decline in glomerular filtration rate, leading to end stage renal failure [14]. Thus, the considerable amount of proteins in the urine samples of the diabetic control rats by the last week of experimentation is a pointer of progression towards the development of renal complication while the trace amount of proteins in the urine samples of the diabetic rats fed unripe plantain by the last week of experimentation, suggests the ability of unripe plantain to ameliorate glomerular complication in diabetics.

The nonsignificant changes in the relative heart weights of the rats in the three groups investigated can be attributed to the nonsusceptibility of the heart to STZ attack as the heart expresses GLUT 4 transporter [14].

Liver hypertrophy (hepatomegaly) is a well recognized complication of diabetes with a reported frequency of 40–70% [1]. Liver hypertrophy (increased liver weight) as observed in the diabetic control rats may be due to hypoinsulinemia-induced increased triacylglycerol accumulation in the liver as alternative glucose precursors since liver glycogen is usually depleted in STZ-induced diabetic rats [15] or it could be as a result of the susceptibility of the liver to STZ challenge while the decrease in the relative liver weights of the diabetic rats fed unripe plantain, indicates the ameliorating actions of unripe plantain on diabetic liver hypertrophy arising from uncontrolled diabetes.

The renal hypertrophy of the diabetic control rats suggests their progression to the development of renal pathology [16] while the decrease in the relative kidney weights of the diabetic rats fed unripe plantain suggests the ability of unripe plantain to ameliorate renal hypertrophy.

The decrease in the relative pancreatic weight of the diabetic untreated group suggests disruption of the pancreatic β-cells [17] while the increase in the relative pancreatic weights of the diabetic rats fed unripe plantain may suggest regeneration of the pancreatic β-cells since the pancreas contains stable cells that have the capacity to regenerate [18].

The loss of weight by the diabetic control rats is simply due to increased protein catabolism as a result of hyperglycemia [19] while the improvement in the body weights of the diabetic rats after feeding of unripe plantain could be attributed to reduction of hyperglycemia by unripe plantain.

Bilirubin is excreted by the liver, and any interference with the normal liver function affects its rate of conjugation and excretion [20], thereby, making bilirubin a good marker of liver function and bile excretion status.

The increase in the total and conjugated bilirubin levels of the diabetic control rats indicates defect in the normal liver function of the rats of this group while the reduction in the total and conjugated bilirubin levels of the diabetic rats fed unripe plantain, suggests the ability of unripe plantain to enhance liver function.

Measurement of enzymic activities of aminotransferases (AST and ALT) and phosphatases is of clinical and toxicological importance as changes in their activities are indicative of tissue damage by toxicants or in disease conditions [10, 21, 22].

The study indicates an increase in the level of the diagnostic enzymes (AST, ALT, and ALP) in the serum of streptozotocin diabetic rat models which is attributable to the toxicity of STZ to tissues that express GLUT 2 transporter such as the hepatocytes and renal tubular cells [23].

The effect of streptozotocin on the levels of diagnostic enzymes in the liver has remained unraveled. While some authors reported increased activities of AST, ALT [24, 25], and ALP [21, 26] in the liver of alloxan and streptozotocin diabetic rat models, some others reported no alteration in the levels of these enzymes in the liver of diabetic rats [22]. In a study carried out by Sing et al. [8], they reported a nonsignificant decrease in the activities of AST and ALT in the liver of streptozotocin diabetic rat models but a significant increase in...
the activity of ALP in the liver of streptozotocin diabetic rat models.

The decreased hepatic AST, ALT, and ALP activities of the diabetic control rats as observed in this study lend credence to earlier works carried out by El-Demerash et al. [27] who reported decreased hepatic AST, ALT, and ALP activities in diabetic rat models. This decrease could be as a result of the leakage of these enzymes from the liver cytosol into the blood stream [28] which gives an indication of the hepatotoxicity of streptozotocin. However, treatment of the diabetic animals with unripe plantain for 28 days was able to increase the activities of the above enzymes indicating the ability of unripe plantain to repair liver damage as used in folk medicine.

Acute pancreatitis is a pathological condition resulting from inflammation of the exocrine pancreas with resultant elevation of the pancreatic enzymes – amylase and lipase. Acute pancreatitis has also been implicated in the etiology of diabetes [29].

The decreased serum amylase activities of the diabetic control rats as observed in this study is attributed to the inhibition of STZ on Ca and Mg homeostasis and amylase gene expression [30] while the increased amylase activities of the nondiabetic rats may be related to fluctuations in the rates of amylase synthesis and its secretion by secretory glands [31, 32].

Although the diabetic rats fed unripe plantain had higher serum amylase levels compared with other groups of rats investigated, the nonsignificant increase compared with the nondiabetic rats suggests that the increase may not necessarily be as a result of pancreatic dysfunction. Such increase may be due to a random effect [31, 33].

Pancreatic lipase is a more specific marker of pancreatic dysfunction than pancreatic amylase [29], and it is also employed in human and animal model studies to evaluate natural products for their usefulness as antiobesity and antidiabetic agents [34].

Elevation of the pancreatic lipase activities of the diabetic control rats as observed in this study establishes a case of acute pancreatitis for the rats of this group. The progression to diabetic state for the rats of this group suggests that their exocrine tissues may have been extensively damaged by the STZ administered. However, the reduced pancreatic lipase activity of the diabetic rats fed unripe plantain is an indication of the ameliorating action unripe plantain on acute pancreatitis as playing a key role in its management of diabetes [35].

The recommended daily allowance for fat in diabetic patients is 15–20% of the total calorie [36]. Crude fat provided about 17.59% of the total calories of the unripe plantain incorporated feed and 7.24% of the total calories of the standard rat feed. The implication is that while the fat content of the standard rat feed failed to meet the daily requirement, the fat content of the unripe plantain incorporated feed did.

Crude protein provided about 15% of the total calories of the standard rat feed and about 14.68% of the total calories of the unripe plantain incorporated feed. The recommended daily allowance for proteins in diabetic patients is 15–20% of the total calories [36]. Thus, while the range of proteins in the standard rat feed met with this requirement, the range of proteins in the unripe plantain incorporated feed did not. It is worth noting that, despite containing large amount of proteins, consumption of the standard rat feeds by the diabetic control rats did not translate into weight gain which is a confirmation that the loss of weight by the diabetic control rats despite eating highly proteinous diet was as a result of uncontrolled hyperglycemia while the gain in weight after feeding of unripe plantain incorporated feed resulted from glycemic control.

Total carbohydrates provided approximately 55.60% of the total calories for the unripe plantain incorporated feed and 31.55% of the total calories for the standard rat feed. Although the carbohydrate contents of the unripe plantain incorporated feed met the required standard for diabetics (50–70% of total calories), the carbohydrate contents of the standard rat feed did not.

Dietary fiber is the indigestible carbohydrate in the diet. Dietary fiber has also been reported to inhibit pancreatic lipase activity [36]. However, it was observed that the crude fiber contents of the standard rat feeds were higher than that of the unripe plantain incorporated feed.

Phenolics have received considerable attention because of their potential antioxidant activity and effects on carbohydrate metabolism involving the inhibition of α-glucosidase and α-amylase, the key enzymes responsible for digestion of dietary carbohydrates to glucose. Furthermore, a positive correlation between the phenolic content of plants and their respective antidiabetic activities has been reported [37].

It is therefore plausible to assume that the higher phenolic content of the unripe plantain incorporated feed compared with the standard rat feed, could be one explanation for the higher antidiabetic action shown by unripe plantain compared with the standard rat feed in this study.

Conclusion

The study indicates the potential of unripe plantain in the management of renal and liver complications arising from diabetes mellitus.

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References