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Research Article

Insights of Cardiovascular Pharmacology Research

Phytochemical, Cardio and Renal protective potentials of Peeled and Unpeeled tuber of *Ipomoea batatas* Lams (Sweet potato) Supplemented Diet on Streptozotocin - Induced Diabetic Albino Rats

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Abstract

This study investigated the phytochemical composition, cardio and renal protective potentials of Ipomoea batatas (sweet potato) peeled and unpeeled tuber in streptozotocin – induced diabetic male albino rats. The GC-MS analysis showed the presences of n-Hexadecanoic acid (19.38%) in the peeled tuber and (14.13%) in the unpeeled tuber as the highest. 2-Ethylacridine was the least in the tuber (0.46%). Isopropylhydroxylamine, 2- Bromoethyl methyl sulphide and 1-tetradecyl acetate are least in abundance in the unpeeled tuber. A total of 45 assumed healthy male albino rats were randomly distributed into nine groups of 5 animals each.. Diabetes was induced in groups 2-9 through intraperitoneal injection of streptozotocin (60mg/kg). The animals were fed diets supplemented with different percentages (10%, 20%, 30%) of sweet potato peeled and unpeeled tuber combinations respectively for 21 days. The group 1 (normal control); group 2 (diabetic fed normal rat pellets), group 3 (diabetic, treated with standard drug metformin and glibeneclamide at 50mg/mL and 0.5mg/mL). Groups 4-6 and 7-9 were fed the peeled tuber and unpeeled tuber supplemented diet respectively. After 21 days the animals were sacrificed, blood collected by cardiac puncture and analyzed for cardiac and renal biomarkers. .The test groups fed sweet potato supplemented diets (peeled tuber) at 10%-30% inclusion showed a significant ($P<0.05$) reduction in the LDH and CPK, restored electrolyte balance, (Na^+, K^+, Cl^-). Bicarbonate (HCO_3^-) compare to the negative control. The unpeeled tuber test groups showed a more significant ($P<0.05$) reduction in the LDH and CPK when compared to the negative control. The supplemented diet had therapeutic effect and showed cardio and renal protective potentials

Keywords: Food, Heart, Kidney, Nutraceuticals

1. Introduction

Diabetes is rapidly emerging as one of the most pressing global health crises, with its prevalence soaring to unprecedented levels. It is a chronic metabolic disorder characterized by elevated levels of blood glucose, resulting from either insufficient insulin production by the pancreas or the body's inability to effectively utilize insulin. This condition is classified primarily into two types: Type 1 diabetes, which is often diagnosed in childhood and results from autoimmune destruction of insulin-producing beta cells, and Type 2 diabetes, which typically develops in adulthood and is associated with insulin resistance and relative insulin deficiency [1].

This number is projected to rise significantly, with estimates suggesting that 643 million individuals will be affected by 2030 and 783 million by 2045 if effective preventive measures are not implemented [2,1]. The increasing prevalence of diabetes has profound implications for public health, as it is one of the leading causes of morbidity and mortality worldwide. Diabetes was responsible for nearly 1.37 million deaths in 2017 alone, making it one of the top ten causes of death globally [3].

Diabetes mellitus (DM) management primarily relies on conventional treatments, including lifestyle modifications, oral hypoglycemic agents, and insulin therapy. However, these approaches have notable limitations that can hinder effective diabetes control and patient outcomes. One of the primary challenges is the high cost associated with diabetes management, which can be prohibitive for many patients, particularly in low- and middle-income countries. For instance, the financial burden of insulin therapy and other medications can lead to treatment non-adherence, exacerbating the disease's complications and overall health outcomes [4]. This financial burden can deter patients from adhering to their treatment regimens, ultimately worsening their health outcomes.

Over time, having too much glucose in the blood can cause serious problems. It can damage the eyes, kidneys, and nerves. Diabetes can also cause heart disease, stroke and even the need to remove a limb. Pregnant women can also get diabetes, called gestational diabetes. Moreover, conventional therapies often fail to address the underlying pathophysiology of Type 2 diabetes effectively. Many treatments focus solely on lowering blood glucose levels without considering the broader metabolic implications, such as cardiovascular health and kidney function [5].

Recent studies have highlighted that intensive glycemic control does not always correlate with reduced risk of cardiovascular events or renal complications in diabetic patients. For example, trials like the Action to Control Cardiovascular Risk in Diabetes (ACCORD) and Veterans Affairs Diabetes Trial (VADT) showed that intensive glycemic control did not significantly reduce cardiovascular events in patients with long-standing diabetes [6,7].

Among such potential dietary interventions is *Ipomoea batatas* (sweet potato). *Ipomoea batatas* has garnered attention as a nutrient-rich food with significant nutritional and medicinal value Sweet potatoes are rich in essential nutrients, making them a valuable addition to the diet [8]. They contain substantial amounts of carbohydrates, vitamins, and minerals. Specifically, sweet potatoes are known to be a good source of vitamin A, vitamin C, manganese, and dietary fiber [9]. Dietary modification remains a cornerstone in the comprehensive management of diabetes mellitus, acting alongside pharmacotherapy, physical activity, and lifestyle changes to achieve glycemic control and mitigate complications [10]. Medical nutrition therapy (MNT), when individualized, can significantly improve metabolic parameters, including fasting glucose, glycated hemoglobin (HbA1c), and lipid profiles [11]. Functional foods, particularly those rich in dietary fiber, antioxidants, resistant starch, and phytochemicals, have garnered increasing attention for their potential to modulate glucose absorption, enhance insulin sensitivity, and suppress postprandial glycemic excursions [12].

In diabetic animal models, supplementation with sweet potato flour has been shown to significantly reduce fasting blood glucose levels, improve lipid profiles, and enhance antioxidant enzyme activities, indicating its potential as a dietary adjunct in diabetes management [13]. Furthermore, the utilization of whole flours, rather than isolated extracts, mirrors real-world dietary patterns and offers a synergistic matrix of nutrients and phytochemicals that may provide greater bio efficacy than single-compound interventions [14]. Given the increasing emphasis on food-based approaches to chronic disease prevention and management, exploring the biochemical effects of different sweet potato flours in diabetic conditions is both timely and necessary This study therefore is to investigate the phytochemical composition of *Ipomoea batatas* (sweet potato) peeled and unpeeled flours, their potential protective effects on cardio and renal biomarkers in streptozotocin – induced diabetic albino rats.

2. Materials and Methods

- **Chemicals and Reagents**

Chemicals used in this work were of analytical grade and products of Sigma Chemical Company, Mayer and Baker Ltd, Fluker, BDH, Randox and TECO Diagnostics, USA

- **Collection, authentication and preparation of *Ipomoea batatas* (sweet potato) peeled and unpeeled flour material**

The Ipomoea batatas (Sweet Potato) tuber was bought from Afia Ebi Market, in Ututu, Arochukwu LGA of Abia State, Nigeria. The tuber was authenticated at the Department of Plant Science and Biotechnology of Abia State University, Uturu. Voucher specimens were kept at the departmental herbarium with number: ABSU/ DPSBH/BCM/2025/063. The sweet potato tuber was washed, and the peeled and unpeeled samples were cut into smaller pieces and sun dried. The dried samples (peeled and unpeeled tuber) were milled into coarse flours and stored at room temperature until used.

- **Phytochemical analysis and bioactive composition of *Ipomoea batata* peeled and unpeeled tuber.**

Sweet Potato (*Ipomoea batatas*) oil was subjected to GC-MS analysis. The analysis was conducted using Agilent 7890A-5975C GC-MS system. A HP5-column (30m x 0.25mm x 0.25µm), operating in electron impact mode at 70eV was used. The carrier gas is ultra-pure helium at a flow rate of 1.0 mL/min and a linear velocity of 37 cm/s. The injector temperature was set at 250^{0}C. The initial oven temperature was set at 110^{0}C which was programmed to increase to 280^{0}C at the rate of 1^{0}C/min with a hold time of 7 minutes at each increment. Injections of 0.5 µL was made in the split less mode with a split ratio of 10:1. The mass spectrometer is operated in the electron ionization mode at 70 eV. The bioactive compounds were identified by direct comparison of the retention times and mass spectral data and fragmentation pattern with those in the National Institute of Standards and Technology (NIST) library.

➢ Experimental Design

A randomized experimental design was employed using Streptozotocin (STZ)-induced diabetic rats to evaluate the biochemical effects of different sweet potato supplemented diets. The animals were randomly divided into nine groups of 5 animals per group, with each group receiving specific treatments as outlined below:

Group 1 - Normal control

Group II - Diabetic group (fed normal rat pellets)

Group III - Diabetic Group (Treated with standard drug Glucophage and glanil).

Group IV – Diabetic - Fed 10% supplemented diet of whole potato tuber (unpeeled)

Group V – Diabetic - Fed 20% supplemented diet of whole potato tuber (unpeeled)

Group VI – Diabetic - Fed 30% supplemented diet of whole potato tuber (unpeeled)

Group VII – Diabetic - Fed 10% supplemented diet of potato tuber (peeled)

Group VIII – Diabetic - Fed 20% supplemented diet of potato tuber (peeled)

Group IX – Diabetic - Fed 30% supplemented diet of potato tuber (peeled)

➢ Formulation of Sweet Potato Supplemented Diet Feed

1. 90gm of normal rat feed mixed thoroughly with 10gm of *Ipomoea batatas* to get 10%
2. 80gm of normal rat feed mixed thoroughly with 20gm of *Ipomoea batatas* to get 20%
3. 70gm of normal rat feed mixed thoroughly with 30gm of *Ipomoea batatas* to get 30%

➢ Experimental Animals A total of forty five (45) apparently assumed healthy maleAlbino rats (9-10weeks old) weighing between (160 - 180 g) was procured from the animal house, Department of Physiology, College of Medicine, University of Nigeria Nsukka. The rats were kept in the animal house at the Department of Biochemistry, Abia State University, Uturu. The animals were allowed to acclimatize for 14 days under standard laboratory conditions prior to the commencement of this investigation with free access to commercial feed (growers feed from Vita feed Nig. Ltd), and clean drinking water ad libitum. United States guidelines for experimental animals use (Guide for Care and Use of laboratory animals (GCULA) 2011) were strictly adhered to throughout the study [15].

➢ Ethical Clearance

Ethical Committee of Animal Care Use of the Faculty of Biological Sciences in conjunction with directorate of research and publication of Abia State University, Uturu Nigeria gave clearance for the use of these animals. Ref: ABSU/DRP/EC/BCM/264

➢ Induction of Diabetes

Animals from group 2 - 9 were fasted overnight prior to the induction of diabetes. They were administered Intra-peritoneal (i.p.) (60mg/kg body weight) Streptozotocin (STZ) α-anomer (Sigma-Aldrich, USA). After 48 hours of streptozotocin administration, blood was collected from the tails of the animals using **Tail Flick Method** to test for blood glucose concentration to establish elevated blood glucose level using the fine test glucometer (ACCUCHEK ACTIVE). Rats that had fasting blood glucose level of 150mg/dL and above were considered diabetic rats.

➢ Preparation of Drugs

Glucophage (Metformin) 500mg and Glanil (Glibenclamide) 5mg solutions were prepared by crushing the tablets in a glass mortar seperately and was dissolved in 10ml of distilled water to give 50mg/ml and 0.5mg/ml stock solutions. Glucophage and glanil were orally administered to the animals.

➢ Route of Administration

In the test groups, (group 2) the rats were administered the standard drugs (Glucophage and Glanil) through oral route using a gavage tube, the rest of the groups were fed different supplemented diets of Ipomoea batatas (sweet potato). All the animals were allowed free access to food and water.

➢ Blood Glucose Determination (Taofik and Anthony, 2014) [16]

Blood glucose level was determined using the glucose enzymatic test kit (Accu-Chek Active) the test was done on day 1, 5, 10, 15 and 20 through the Tail Flick Method of blood collection. 0.2mLs of blood was collected from the tail after mild anesthesia using ether. Animals were fasted for 16-18 hours prior to blood collection.

➢ Collection of Blood Samples

On the 21th day, the animals were starved overnight, anaesthetized with chloroform and sacrificed. Blood from each animal was collected through cardiac puncture.. The blood samples was put into a plain sample bottle and was allowed to stand for 2 minutes to clot and was further spun in a centrifuge to get the serum, it was used for the measurement and evaluation of the biochemical assays.

➢ Determination of Some Cardiac Parameters

• Determination of Lactate Dehydrogenase (LDH) Activity

The LDH activity was determined by the method of Uchendu et al., (2024), as outlined in the LDH-P assay kits [17].

• Principle

The reaction system was as follows: Pyruvate + NADH + H^+ → L-lactate + NAD^+

Kit Components:

i. Reagent A: Vial containing NADH-0.2 mmol/L

ii. Reagent B: Tris buffer solution - 80mM, pH 7.2, containing pyruvate (1.6 mmol/L) and sodium chloride (200 mmol)

• Procedure

Reagent A (3mL) was reconstituted and pre-incubated for a few minutes. After incubation, it was added to the sample, which was mixed simultaneously for 30 seconds using a stopwatch for accuracy. Absorbance readings were taken at 1, 2, and 3 minutes for the first reading. The average absorbance per minute (ΔA/min) was determined by subtracting each reading from the previous one and averaging the values. Then 50µL of the sample was added to Reagent B at 30-37°C and mixed thoroughly, following the procedure as indicated in Reagent A.

Calculation: LDH (U/L) = ΔA / min × factor

• Determination of Creatine Phosphokinase (CPK) also known as Creatine Kinase (CK)

.Creatine kinase -myocardial Bound (CK-MB) Determination: Creatine kinase- myocardial bound was estimated by the methods of Gerhardt et al; (1977), as contained in the Abnova assay kit [18].

• Principle

Creatine kinase assay kit was based on enzyme coupled reactions in which creatine phosphate and ADP was converted to creatine and ATP by CK, the generated ATP is used to phosphorylate glucose by hexokinase to generate glucose-6-phosphate, which is then oxidized by NADPH in the presence of glucose-6-phosphate dehydrogenase. The NADPH produced was measured at 340nm, which is proportional to the activity of the CK in the sample.

Kit components

Assay buffer	12ml
Substrate solution	1.0ml
Enzyme mix	120ml
Calibrator	150ml

All reagents were stored at -20oC

• Procedure

Each reaction well was added and mixed with 10ml substrate solution. The 100ml assay buffer and 1ml enzyme were mixed at room temperature. The non-haemolysed sample was stored at -50^{0}C.Water was added into the separate wells of a clear bottom 96-well plate along with 10ml of sample and was incubated at room temperature. The fully activated CK after 20mins by glutathione in substrate solution was read as follows: Read 0.0340nm at 20mins and again at 40mins, the value was read calorimetrically.

$$CK\ (u/L) = \frac{CD340mins\text{-}OD20mins}{CDcalibrator\text{-}ODH2O} \times 650$$

➢ Determination of Renal Function Parameters

• The serum concentrations of Creatinine and Urea were determined using auto-analizer (Biosystem A25 Random Access Analyzer) as contained in the kit (Natelson et al .,1951)

❖ Urea Concentration [19]

The contents of vial R1a were transferred into bottle R1b and mixed gently to form reagent R1. The contents of bottle R2 were diluted with 660 ml of distilled water, and the contents of bottle R3 were diluted with 750 ml of distilled water. Three test tubes labeled "test," "blank," and "standard" were set up. Approximately 100 µl of R1 was pipetted into each test tube, and 10 µl of the test sample was added to the test tube labeled "test." The same volume of standard reagent and distilled water were added to the test tubes labeled "standard" and "blank," respectively. The mixtures in the test tubes were incubated at 37°C for 10 minutes, after which 2.5 ml of reagent R2 and an equal volume of R3 were added to each test tube. The contents of each tube were mixed immediately and incubated at 37°C for 15 minutes. The absorbance of the test and sample were read after zeroing with the blank. The urea concentration in the sample was calculated using the following formula:

$$\text{Urea concentration in mg/dl} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{concentration of standard}$$

Where concentration of standard = 78.73 mg/dl

• Determination of creatinine concentration Fabiny and Ertingshausen (1971) [20]

Equal volumes of R1a and R1b were mixed to form the working reagent. Two test tubes labeled "test" and "standard" were set up. Exactly 2 ml of the working reagent was transferred into each test tube, and 0.2 ml (200 µl) of the sample was added to the test tube labeled "test" and the same volume of standard reagent to the standard. The contents of each test tube were mixed, and after 30 seconds, absorbance A1 for both the standard and test were read. After another 2 minutes, absorbance A2 for both the standard and test were read. The absorbance for the test and standard were calculated as:

Absorbance of test or sample = $A_2 - A_1$

Absorbance of standard = $A_2 - A_1$

$$\text{Creatinine concentration in mg/dl} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{concentration of standard}$$

Where concentration of standard = 2.05 mg/dl

Serum concentrations of Sodium, Potassium, Hydrogen carbonate, and Chloride were determined using auto-analizer (EasyLyte Plus Analyzer). The manufacturer's instructions for the entire biochemical test were strictly adhered to.

• Determination of Sodium Concentration

Three test tubes labeled "test," "standard," and "blank" were set up, and 1 ml of filtrate reagent was pipetted into each test tube. Exactly 50 µl of the sample was added to the test tube labeled "test," the same volume of standard reagent to the standard, and the same volume of distilled water to the blank. All test tubes were shaken vigorously and centrifuged at high speed for 10 minutes to obtain a supernatant. The test proceeded with the supernatant only. Another set of three test tubes labeled "test," "standard," and "blank" was set up, and 1 ml of acid reagent was pipetted into each of the tubes. Then 50 µl of the supernatant was added to their corresponding test tubes and mixed. The same volume of color reagent was added to all the tubes and mixed. The spectrophotometer was zeroed with distilled water, and the absorbance of the content of each test tube was read and recorded. The sodium concentration in the sample was calculated using the formula:

$$\text{Sodium conc. in mEq/L} = \frac{\text{Abs. of blank} - \text{Abs. of test}}{\text{Abs. of blank} - \text{Abs. of standard}} \times \text{Concentration of standard}$$

Where concentration of standard = 150 mEq/L

• Determination of Chloride Concentration

Three test tubes labeled "test," "standard," and "blank" were set up, and 1.5 ml of chloride reagent was pipetted into each tube. Then, 10 µl of the sample was added to the test tube labeled "test," the same volume of the standard reagent to the standard, and the same volume of distilled water to the blank. The test tubes were shaken and incubated at 25°C for 5 minutes. The absorbance of the content of each test tube was read at a wavelength of 500 nm after zeroing with the blank. The chloride concentration in mEq/L was calculated using the formula:

$$\text{Chloride concentration in mEq/L} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{concentration of standard}$$

Where concentration of standard = 100 mEq/L

• Determination of Potassium Concentration

Three test tubes labeled "test," "standard," and "blank" were set up, and 1 ml of chloride reagent was pipetted into each tube. Then, 10 µl of the sample was added to the test tube labeled "test," the same volume of the standard reagent to the standard, and the same volume of distilled water to the blank. The test tubes were shaken and allowed to stand at 25°C for 3 minutes. The absorbance of the content of each test tube was read at a wavelength of 500 nm after zeroing with the blank. The potassium concentration in mEq/L was calculated using the formula:

$$\text{Potassium conc. in mEq/L} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{concentration of standard}$$

Where concentration of standard = 4 mEq/L

• Determination of Bicarbonate Concentration

Carbon dioxide (CO_2) reagent was reconstituted with the volume of CO_2^- free water indicated on the vial label and mixed by gentle inversion 5-6 times. Three cuvettes labeled "test," "standard," and "blank" were set up, and 1 ml of the reagent was pipetted i`nto each tube. All tubes were incubated for 3 minutes at 37°C. Exactly 10 µl of the sample was added to the cuvette labeled "test," and the same volume of the standard reagent and distilled water to the standard and blank, respectively. The absorbance was read at 340 nm, and the bicarbonate concentration was calculated using the formula:

$$\text{Bicarbonate conc. in mmol/L} = \frac{\text{Abs of blank} - \text{Abs of test}}{\text{Abs of blank} - \text{Abs of standard}} \times \text{Conc. of standard}$$

Where concentration of standard = 30 mmol/L

• Statistical Analysis

The results of the biochemical experiments were presented as mean ± SEM (standard error of mean). We used the one-way analysis of variance (ANOVA) to determine the degree of significance with probability levels below 0.05 ($p < 0.05$) as significant.

3. Results

Tables showing results of study

Serial No.	Compound name	Molecular formula	Molecular weight (g/mol)	Retention time (min)	Quantity/Peak area (%)
1	Oxetane, 2,2- dimethyl	$C_5H_{10}O$	86.073165	1.749	1.88
2	2,3-Butanediol	$C_4H_{10}O_2$	0.0680795	1.830	0.87
3	2,3-Butanediol	$C_4H_{10}O_2$	0.0680795	1.876	1.24
4	2-Furanmethanol	$C_5H_6O_2$	98.0367794	2.210	0.93
5	1,5-Pentanedithiol	$C_5H_{12}S_2$	136.038042	2.337	1.44
6	2-Amino-3-methyl-1-butanol	$C_5H_{13}NO$	103.0997143	2.643	1.10
7	cis-3-Methyl-2-n-propylthiophane	$C_8H_{16}S$	144.097272	2.978	1.31
8	Butanoic acid	$C_7H_{12}O_3$	144.078644	3.139	1.15
9	1,2,3,4-Butanetetrol	$C_4H_{10}O_4$	122.057909	3.462	0.78
10	Uracil, 1-methyl	$C_5H_6N_2O_2$	126.0429273	3.705	1.46
11	2-Propanamine	$C_4H_{10}N_2O$	102.079313	4.103	1.15
12	4H-Pyran-4-one, 2,3-dihydro-3,5-dihydroxy-6-methy	$C_6H_8O_4$	144.042258	4.172	3.59
13	Catechol	$C_6H_6O_2$	110.0367794	4.530	3.34
14	5-Hydroxymethylfurfural	$C_6H_6O_3$	26.031694	4.766	2.94

15	Butanedioic acid	$C_6H_{10}O_5$	162.052823	4.887	1.59
16	1-Methyl-2-dimethyl(isopropyl) silyloxycyclohexane	$C_{12}H_{26}OSi$	214.175292	5.378	1.71
17	β-D-Glucopyranose	$C_{12}H_{22}O_{11}$	342.11621	6.935	0.83
18	Di(n-hexyl)sulfone	$C_{12}H_{26}O_2S$	234.165351	7.558	0.80
19	n-Hexadecanoic acid	$C_{16}H_{32}O_2$	268.24023	9.693	19.38
20	2-cyclohexanone	$C_{10}H_{14}O_2$	166.099	9.872	0.68
21	Pentadecafluorooctanoic acid	$C_{26}H_{37}F_{15}O_2$	666.255	10.668	0.75
22	9,12-Octadecadienoic acid	$C_{18}H_{32}O_2$	280.240	11.043	6.94
23	Octadecanoic acid	$C_{18}H_{36}O_2$	284.271	11.222	2.13
24	Hexahydropyridine,	$C_{12}H_{17}NO_2$	207.125	12.133	0.51
25	Glycerol 1-palmitate	$C_{19}H_{38}O_4$	330.277	13.079	4.09
26	2-Ethylacridine	$C_{15}H_{13}N$	207.104	13.443	0.46
27	1,3,12-Nonadecatriene	$C_{19}H_{34}$	262.266	13.835	2.97
28	4-tert-Butylphenol	$C_{13}H_{22}OSi$	222.143	14.366	1.06
29	2-Methyl-7-phenylindole	$C_{15}H_{13}N$	207.104	15.894	1.21
30	2-Ethylacridine	$C_{15}H_{13}N$	207.104	16.598	6.41
31	4-(4-Hydroxyphenyl)-4-methyl-2-pentanone	$C_{15}H_{24}O_2Si$	264.154	16.823	1.88
32	Indole-2-one	$C_{11}H_{13}NO_3$	207.089	16.933	2.70
33	β-Sitosterol	$C_{29}H_{50}O$	386	17.256	18.24
34	2-Methyl-7-phenylindole	$C_{15}H_{13}N$	207.104	17.723	1.09
35	Tris(tert-butyldimethylsilyloxy) arsane	$C_{18}H_{45}AsO_3Si_3$	468.189	18.848	1.39

Table 1a: GC-MS Spectral Analysis of the Chemical Constituents of Unpeeled Tuber Oil Extract of *Ipomoea batatas*

The GC-MS spectral analysis of the chemical constituents of n-Hexane seed oil extract of *Ipomoea batatas* revealed the details of their peak number, molecular formula, molecular weight, retention time and quantity or peak area. The result showed the presence of thirty-five (35) chemical components. The result revealed that n-Hexadecanoic acid which has antioxidants, anti-inflammatory, metabolic modulations properties (19.38%) is the most abundant chemical in the plant with a retention time and % composition of 9.693 minutes and 19.38 respectively. The result further revealed that 2-Ethylacridine which has anticancer and antioxidant properties is the least abundant chemical with a retention time and % composition of 13.443 minutes and 0.46% respectively. Other notable bioactives included β-sitosterol (18.24%), 9,12-octadecadienoic acid (6.94%), 2-ethylacridine (6.41%), and glycerol 1-palmitate (4.09%), catechol, 5-hydroxymethylfurfural, 4H-pyran-4-one derivatives, and indole-based compounds with known pharmacological relevance.

Serial No.	Compound name	Molecular formula	Molecular weight (g/mol)	Retention time (min)	Quantity/Peak area (%)
1	Cyclopropanecarboxylic acid	$C_6H_6O_2$	86.036	1.784	0.88
2	o-Isopropylhydroxylamine	C_3H_9NO	75.068	1.870	0.64
3	2-Thiazolamine	$C_3H_6N_2S$	102.025	2.355	0.94
4	Propanoic acid	C_2H_5COOH	74.078	2.649	0.82
5	Carbamic acid	$C_4H_8N_2O_3$	132.053	3.134	0.69
6	Thymine	$C_5H_6N_2O_2$	126.042	3.716	0.93
7	2,3-dihydro-3,5-dihydroxy-6-methyl	$C_6H_8O_4$	144.042	4.183	2.26
8	Catechol	$C_6H_6O_2$	110.036	4.535	1.33

9	5-Hydroxymethylfurfural	$C_6H_6O_3$	126.031	4.749	5.26
10	2-Bromoethyl methyl sulphide	C_3H_7BrS	153.945	6.156	0.64
11	Cyclohexanol	$C_8H_{13}NO_3$	171.089	7.149	1.26
12	4-methyl-1-(2,3,4,5-tetrahydro-5-methyl[2,3'-bifuran]-5-yl)	$C_{15}H_{22}O_3$	250.156	7.558	3.96
13	Octyl tetracosyl ethe	$C_{32}H_{66}O$	466.511	8.135	0.66
14	1-Tetradecyl acetate	$C_{16}H_{32}O_2$	256.240	8.885	0.64
15	D-Mannonic acid,	$C_6H_{10}C_6$	178.047	9.497	0.70
16	n-Hexadecanoic acid	$C_{16}H_{32}O_2$	256.240	9.716	14.13
17	Cyclotetradecane	$C_{14}H_{28}$	196.219	10.673	0.91
18	9,12-Octadecadienoic acid	$C_{18}H_{32}O_2$	280.24	11.060	4.29
19	Octadecanoic acid	$C_{18}H_{36}O_2$	284.27	11.233	2.99
20	Glycerol 1-palmitate	$C_{19}H_{38}O_4$	330.27	13.079	2.00
21	E,Z-1,3,12-Nonadecatriene	$C_{19}H_{34}$	262.26	13.835	2.70
22	1,2-Bis(trimethylsilyl)benzene	$C_{12}H_{22}Si_2$	22.12	15.900	0.69
23	1,4-Bis(trimethylsilyl)benzene	$C_{12}H_{22}Si_2$	222.12	16.448	0.89
24	Campesterol	$C_{28}H_{48}O$	400.37	16.598	3.50
25	1,2-Bis(trimethylsilyl)benzene	$C_{12}H_{22}Si_2$	222.12	16.823	1.80
26	Benzenepropanoic acid, 4-hydroxy-, methyl ester	$C_{10}H_{12}O_3$	180.07	16.944	11.10
27	γ-Sitosterol	$C_{29}H_{50}O$	414.38	17.267	11.28
28	2-Aminobenzothiazol-6-carboxamide	$C_8H_7N_3OS$	193.03	17.723	4.99
29	Methyltris(trimethylsiloxy)silane	$C_{10}H_{30}O_3Si_4$	310.12	18.121	0.65
30	Thymol, TMS derivative	$C_{13}H_{22}OSi$	222.14	18.462	1.35
31	1H-Indole-2-carboxylic acid, 6-(4-ethoxyphenyl)-3-methyl-4-oxo-4,5,6,7-tetrahydro-, isopropyl ester	$C_{21}H_{25}NO_4$	355.17	18.635	0.67
32	Carbonic acid, monoamide, N-(3,4-dimethoxyphenethyl)-, butyl ester	$C_{15}H_{23}NO_4$	281.16	18.848	8.82
33	2-Ethylacridine	$C_{15}H_{13}N$	207.10	19.113	3.98
34	Methyltris(trimethylsiloxy)silane	$C_{10}H_{30}O_3Si_4$	310.12	19.361	0.77
35	3-Amino-7-nitro-1,2,4-benzotriazine 1-oxide	$C_7H_5N_5O_3$	207.03	19.956	0.88

Table 1b: GC-MS Spectral Analysis of The Chemical Constituents of Peeled Tuber Oil Extract of *Ipomoea batatas*

The GC-MS spectral analysis of the chemical constituents of peeled tuber oil extract of *Ipomoea batatas* revealed the details of their peak number, molecular formula, molecular weight, retention time and quantity or peak area. The result showed the presence of thirty-five (35) chemical components. The result revealed that n-Hexadecanoic acid which has antioxidants, anti-inflammatory, metabolic modulations properties is the most abundant chemical in the plant with a retention time and % composition of 9.716 minutes and 14.13 respectively. The result further revealed that Isopropylhydroxylamine, 2-Bromoethyl methyl sulphide, 1-Tetradecyl acetate are the least abundant chemical with a retention time and % composition of 1.87, 6.15 and 8.88 minutes and 0.64% for the three compounds respectively.

Groups	Treatment	LDH (mg/dl)	CPK (u/l)
1	Normal Control	139.80 ± 4.35^{a}	129.40 ± 4.24^{a}
2	Negative Control (Not treated)	437.00 ± 10.45^{e}	372.00 ± 7.88^{e}
3	Positive Control (Standard Drug)	198.00 ± 8.60^{b}	162.60 ± 2.62^{b}
4	10% peeled tuber	292.20 ± 15.00^{d}	249.40 ± 7.75^{d}
5	20% peeled tuber	272.80 ± 6.01^{d}	254.00 ± 5.63^{d}
6	30% peeled tuber	272.60 ± 4.11^{d}	244.00 ± 8.49^{d}
7	10% unpeeled tuber	231.40 ± 3.71^{c}	189.40 ± 5.22^{c}
8	20% unpeeled tuber	244.80 ± 6.43^{c}	206.20 ± 7.85^{c}
9	30% unpeeled tuber	198.00 ± 8.65^{b}	193.00 ± 5.96^{c}

Table 2: Effect of *Ipomoea batatas* supplemented diets on Cardiac biomarkrs LDH (mg/dl) CPK (u/l) in diabetic rats

Values are expressed as mean ± standard deviation (n=3). Means with different superscript letters (a, b, c, d) within the same column are significantly different from each other ($p < 0.05$). LDH, Lactate Dehydrogenase; CPK, Creatine Phosphokinase

In table 2, the negative control group (untreated diabetic rats) showed a significant ($P<0.05$) elevation of LDH and CPK levels which signifies elevated cardiac stress in diabetes that predispose to cardiovascular complications when compared to the normal control, while treatment with the standard anti-diabetic drug significantly ($P<0.05$) reduced these enzyme levels of LDH and CPK, indicating its cardio protective potential. The test groups of sweet potato supplemented diets (peeled tuber) at 10%-30% inclusion showed a significant ($P<0.05$) reduction in the LDH and CPK compare to the negative control, while the unpeeled tuber test groups showed a more significant ($P<0.05$) reduction in the LDH and CPK when compared to the negative control.

Groups	Treatment	Urea (mg/dl)	Creatinine (mg/dl)	Na^{+} (mEq/L)	K^{+} (mEq/L)	Cl^{-} (mEq/L)	HCO_3^{-} (mmol/L)
1	Normal Control	17.45 ± 0.53^{a}	0.67 ± 0.01^{a}	130.22 ± 0.59^{c}	4.58 ± 0.04^{c}	87.88 ± 0.71^{bc}	19.48 ± 0.06^{a}
2	Negative Control (Not treated)	32.54 ± 0.85^{c}	1.07 ± 0.03^{a}	126.20 ± 0.51^{a}	4.23 ± 0.03^{a}	85.82 ± 0.33^{a}	19.78 ± 0.12^{b}
3	Positive Control (Standard Drug)	17.65 ± 0.64^{a}	0.70 ± 0.02^{a}	130.88 ± 0.46^{c}	4.58 ± 0.02^{c}	89.16 ± 0.43^{c}	19.70 ± 0.05^{ab}
4	10% peeled tuber	21.97 ± 0.72^{b}	0.75 ± 0.02^{a}	129.62 ± 0.38^{bc}	4.42 ± 0.05^{b}	87.22 ± 0.42^{ab}	19.66 ± 0.04^{ab}
5	20% peeled tuber	22.06 ± 0.91^{b}	0.74 ± 0.02^{a}	129.58 ± 0.72^{bc}	4.42 ± 0.05^{b}	86.96 ± 0.49^{ab}	19.68 ± 0.06^{ab}
6	30% peeled tuber	21.24 ± 0.55^{b}	0.73 ± 0.02^{a}	128.70 ± 0.60^{b}	4.45 ± 0.03^{b}	87.06 ± 0.52^{ab}	19.64 ± 0.05^{ab}
7	10% unpeeled tuber	20.25 ± 0.32^{b}	0.80 ± 0.03^{a}	129.22 ± 0.58^{bc}	4.50 ± 0.06^{bc}	87.90 ± 0.32^{bc}	19.64 ± 0.05^{ab}
8	20% unpeeled tuber	20.79 ± 0.27^{b}	0.76 ± 1.79^{a}	129.80 ± 0.33^{bc}	4.48 ± 0.04^{bc}	88.28 ± 0.47^{bc}	19.64 ± 0.06^{ab}
9	30% unpeeled tuber	21.32 ± 0.60^{b}	0.78 ± 0.02^{a}	130.54 ± 0.29^{bc}	4.53 ± 0.02^{bc}	87.88 ± 0.47^{bc}	19.62 ± 0.10^{ab}

Table 3: Effect of *Ipomoea batatas* supplemented diets on Renal FUNCTION Biomarkers in Diabetic Rats

Values are expressed as mean ± standard deviation (n=3). Means with different superscript letters (a, b, c, d) within the same column are significantly different from each other ($p < 0.05$). Urea, Creatinine, Na^{+}, Sodium ion; K^{+}, Potassium ion; HCO_3^{-}, Bicarbonate; Cl^{-}, Chloride ion.

In table 3, the negative control group (untreated diabetic rats), showed a significant ($P<0.05$) increase in urea and creatinine when compare to the normal control group, indicating impairedkidney function. Treatment with the standard drug (Group 3) effectively restored these parameters urea and creatinine to near-normal levels. Likewise, sweet potato-based diets (Groups 4–9) significantly ($P<0.05$) improved urea and creatinine levels compared to the negative control, suggesting nephro-protective properties. Electrolyte imbalances was observed in the negative control group characterized by decrease Na^{+}, Cl^{-} and K^{+}, reflects disrupted electrolyte homeostasis, which can lead to complications such as dehydration, muscle weakness, and cardiac issues. Treatment with sweet potato supplemented diets, particularly the 30% unpeeled tuber group, significantly ($P<0.05$) restored electrolyte balance, (Na^{+}, K^{+}, Cl^{-}). Bicarbonate (HCO_3^{-}) relatively remained stable, it had no significant ($P<0.05$) effect across the groups.

4. Discussion

The phytochemical profile of *Ipomoea batata* tuber and peel oils reveals a rich matrix of fatty acids, sterols, furans, phenolics, and heterocyclic compounds with known antioxidant, anti-inflammatory, and hypoglycemic activities. Notably, n-hexadecanoic acid, β-sitosterol, linoleic acid (9,12-octadecadienoic acid), and campesterol are implicated in improving insulin sensitivity and modulating lipid profiles [21,22]. β-sitosterol and γ-sitosterol, which are abundant in both tuber and peel extracts, have previously been shown to exert insulin-mimetic effects, enhance glucose uptake, and inhibit α-glucosidase activity, making them promising candidates in managing type 2 diabetes [23]. The presence of catechol, 5-hydroxymethylfurfural, and 4H-pyranone derivatives further supports the antioxidant capacity of the sweet potato supplemented diets, which may protect pancreatic β-cells from oxidative stress-induced damage [24].

Lactate Dehydrogenase (LDH) and Creatine Phosphokinase (CPK) are key biomarkers of cardiac tissue damage and general cell injury [25]. LDH is an intracellular enzyme involved in the interconversion of lactate and pyruvate in glycolysis and gluconeogenesis [26]. It is present in nearly all body tissues, but elevated serum LDH levels typically indicate tissue damage, especially of the heart, liver, skeletal muscles, and red blood cells [27]. In the context of cardiac injury, LDH is released into the blood following myocardial cell membrane damage, making it a useful marker of myocardial infarction or diabetic cardiomyopathy [25]. CPK (also known as Creatine Kinase, CK) is an enzyme involved in energy metabolism, catalysing the conversion of creatine and ATP to phosphocreatine and ADP [28]. It is most abundant in heart muscle (CK-MB), skeletal muscle (CK-MM), and the brain (CK-BB). Elevated serum CPK, particularly CK-MB, is a well-established marker of cardiac muscle damage, such as myocardial infarction or cardiac stress in diabetes [29,30]. Both LDH and CPK have been used extensively to assess cardiac injury, especially in conditions like diabetes-induced cardiomyopathy, where hyperglycaemia-induced oxidative stress damages cardiac myocytes [31,32].

The current study demonstrated significant changes in LDH and CPK levels across treatment groups. In the normal control group, LDH (139.80 ± 4.35 mg/dl) and CPK (129.40 ± 4.24 u/l) levels were within normal physiological ranges, indicating intact myocardial cell integrity and normal cardiac enzyme activity [33]. In contrast, the negative control group (untreated diabetic rats) showed significantly elevated LDH (437.00 ± 10.45 mg/dl) and CPK (372.00 ± 7.88 u/l) levels. These marked increases reflected substantial cardiac tissue injury and myocardial cell membrane disruption associated with hyperglycaemia-induced oxidative stress and inflammation in diabetes [31,32]. Such elevations were consistent with previous findings in diabetic cardiomyopathy models, where oxidative stress and metabolic disturbances result in cell membrane damage and enzyme leakage [34]. Treatment with the standard anti-diabetic drug significantly reduced these enzyme levels (LDH: 198.00 ± 8.60 mg/dl, CPK: 162.60 ± 2.62 u/l), indicating its cardioprotective potential, likely through improved glycaemic control and attenuation of oxidative stress [35].

The groups treated with sweet potato tuber-only diets at 10–30 % inclusion also exhibited significant reductions in LDH and CPK compared to the negative control. For example, the 20 % peeled tuber group recorded LDH (272.80 ± 6.01 mg/dl) and CPK (254.00 ± 5.63 u/l). Sweet potato tubers are known to contain bioactive compounds, such as ferulic acid and catechins, which possess potent antioxidant and anti-inflammatory effects [36,37]. These properties likely reduced oxidative stress, resulting in attenuation of myocardial injury [38,39]. Notably, the unpeeled tuber combination groups showed even more pronounced protective effects, particularly the 30 % unpeeled tuber group, with LDH and CPK levels comparable to those in the standard drug group. This synergistic effect might be attributed to the higher polyphenol and flavonoid content in the peel, which has been demonstrated to provide additional cardioprotective benefits [40,41]. These bioactive compounds likely acted synergistically to stabilize cardiac cell membranes, prevent oxidative damage, and suppress inflammation [42].

Renal function is crucial in diabetes management, as diabetic nephropathy is a common and severe complication [43]. In this study, key markers such as urea, creatinine, and electrolytes (Na^+, K^+, Cl^-, and HCO_3^-) were assessed to evaluate the impact of sweet potato-based diets on renal health in diabetic rats.

Urea and creatinine are waste products filtered by the kidneys, and their elevation typically signals impaired renal filtration [44]. In the negative control group (untreated diabetic rats), both urea (32.54 mg/dl) and creatinine (1.07 mg/dl) were significantly ($P<0.05$) elevated compared to the normal control group (17.45 mg/dl and 0.67 mg/dl, respectively). This indicated a decline in glomerular filtration rate (GFR) and suggested renal dysfunction due to the hyperglycaemic and oxidative stress environment in diabetes [45]. Interestingly, treatment with the standard drug (Group 3) effectively restored these parameters to near-normal levels (17.65 mg/dl urea, 0.70 mg/dl creatinine). Likewise, sweet potato-based diets (Groups 4–9) significantly improved urea and creatinine levels compared to the negative control. The 10% unpeeled tuber group, for example, had urea (20.25 mg/dl) and creatinine (0.80 mg/dl) values closer to the normal group, suggesting preserved renal filtration function. These observations highlighted the potential nephro-protective effects of sweet potato unpeeled tubers, which may be linked to their high polyphenol and fibre content, known for mitigating oxidative stress and inflammation in diabetic kidneys [46].

Electrolyte homeostasis is vital for normal renal and cardiovascular function. Diabetes often disrupts these balances through osmotic diuresis and renal tubular damage [47]. In this study, the negative control group displayed significant electrolyte imbalances. Sodium (126.20 mEq/L) and chloride (85.82 mEq/L) were notably lower than in normal controls (130.22 mEq/L and 87.88 mEq/L, respectively), reflecting impaired renal reabsorption processes. Potassium (4.23 mEq/L) also showed a downward trend. Treatment with sweet potato-based diets, particularly the 30% unpeeled tuber group, helped restore electrolyte balance. Sodium (130.54 mEq/L), chloride (87.88 mEq/L), and potassium

(4.53 mEq/L) in this group approached normal control values, indicating improved tubular handling and ion transport capacity. This suggested that sweet potato-derived bioactive compounds may protect renal tubular integrity, thereby normalizing electrolyte reabsorption and secretion.

Bicarbonate (HCO_3^-) levels remained relatively stable across all groups (~19.6 mmol/L), suggesting that acid-base regulation was not severely affected by diabetes in this model. Nonetheless, maintenance of bicarbonate levels in treatment groups highlighted the ability of sweet potato-based diets to support renal function and acid-base balance in the diabetic state. Overall, the administration of sweet potato tuber and peel in various concentrations ameliorated diabetic-induced renal dysfunction, as evidenced by normalized urea, creatinine, and electrolyte profiles. This renoprotective effect likely stems from the rich antioxidant profile of sweet potato, which helps mitigate renal oxidative stress and inflammation [48,49].

5. Conclusion

The results from this study shows that sweet potato is rich in phytochemicals. Most of these photochemical are bioactive and have been reported to have pharmacological effects. This study has shown the diets supplemented with Ipomoea batatas (sweet potato) unpeeled and peeled tuber effectively improved the cardiovascular and renal biomarkers in the test animals compared to the diabetic animals placed on normal rat feeds. From the phytochemical analysis, Octadecadienoic acid, benzotrieazine oxide and propanamine were identified and reported to have cardiovascular health effects. 2-Bromoethyl methyl sulphide and Catechol were also identified which have, antioxidant and pro-oxidant effects, and Neuro-protective effects, which also has effect on renal and kidney health. The unpeeled sweet potato tuber supplemented diets showed significantly higher ameliorating effects on the parameters assayed than the peeled tuber. We therefore recommend the processing and consumption of sweet potato unpeeled to get a maximum of the health benefits especially for the management of diabetes and its complications.

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