Profile of *Morinda lucida* Leaf Fractions on Blood Glucose and Lipids in Normal and Alloxan-Induced Hyperglycaemic Rats

Adejuwon Adewale Adeneye
Department of Pharmacology, Faculty of Basic Medical Sciences, Lagos State University College of Medicine, Ikeja, Lagos, Nigeria

**ABSTRACT**

**Background:** Ethanol decoction made from the fresh leaves of *Morinda lucida* Benth. is highly valued in the local management of diabetes mellitus among the Nigerian herbalists. Of the different solvent fractions (diethyl ether, chloroform, butanol and the residue) made from the crude ethanol leaf extract of *Morinda lucida* (MLE). **Objective:** the present study aims to determine and evaluate the most effective antihyperglycaemic and antihyperlipidaemic fraction in normal and alloxan-induced diabetic rats for 14 days using various solvents successively and determining the secondary metabolites/phytochemicals in the effective solvent fractions of MLE. **Material and Methods:** MLE was successively partitioned in diethyl ether, chloroform and butanol and 50 mg kg⁻¹ of each of these fractions was administered to normal and alloxan-induced hyperglycaemic rats for 14 days. The effect of each fraction was on Fasting Blood Glucose (FBG) on the 1st, 8 and 15th day post-treatment was evaluated using glucose monitoring system. The effect of the fractions on serum triglyceride and total cholesterol was also determined using standard procedures. In addition, qualitative phytochemical analysis was conducted in MLE, and MLE₆ using standard procedures. **Results:** In the normal and alloxan-induced diabetic rats, oral treatment with MLE and MLE₆ resulted in significant (p<0.05, p<0.01 and p<0.001) time-dependent lowering of FBG, serum triglyceride and total cholesterol in the treated rats with MLE, producing the most significant (p<0.001) antihyperglycaemic and antihyperlipidaemic effects. Qualitative phytochemical analysis of MLE, showed the presence flavonoids, alkaloids and anthraquinones while that of MLE₆ showed the presence of flavonoids, alkaloids, tannins and saponin. **Conclusion:** Results of this study shows MLE to be the most effective antihyperglycaemic and antihyperlipidaemic fraction of all the solvent fractions of MLE tested.

**Key words:** *Morinda lucida* ethanolic leaf extract, solvent fractions, alloxan-induced hyperglycaemic rats, antihyperglycaemic and antihyperlipidaemic effects

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**INTRODUCTION**

Diabetes Mellitus (DM) remains the most common endocrine disorder of carbohydrate metabolism with over 170 million adult sufferers estimated to have been affected worldwide and about two-thirds of this figure residing in the developing countries¹. This estimate is expected to double by the year 2030¹². In view of the growing prevalence of the disease, there is an increasing interest towards developing integrated approaches in the prevention and management of DM by exploring the potentials offered by the indigenous herbal therapies³. Herbal drugs are prescribed widely because of their effectiveness, relatively low cost and based on the general belief that they are free of intolerable side-effects⁴. Also, World Health Organization⁵ has directed further evaluation of the traditional practices and methods (including herbal practices) of managing the disease. In compliance with this directive, many indigenous medicinal plants are currently been screened and evaluated for their antihyperglycaemic potentials. In African herbal medicine, there are various native plants that are employed in the local management of DM, one of which cuts across the West African region is *Morinda lucida* Benth.

*Morinda lucida* Benth., belonging to the family Rubiaceae, is a tropical West Africa rainforest tree with the English name "Brimstone tree". It is known as Sangogo or Bondoucou alongua (in Cote d’Ivoire), Twi, Kôn krônâ or Ewe anak (in Ghana), Ewe amake or Atak ake (in Togo) and Orouwó or ruwó amongst the Yoruba tribe (South-West Nigeria)⁶. Amongst the Igbo (South-East Nigeria), it is locally known as "Huka" and "Eze-ogu". Decoctions made from the plant leaves, stem bark and roots are highly valued in the local DM management⁷. The leaves and stem bark of *Morinda lucida* are used as purgative, emetic and diuretic⁸. In Nigeria, a mixture of fresh leaves of *Morinda lucida*, *Monodora charantia*, *Vernonia amygdalina* and *Dalbergia welwitschii* are
ground together into a fine paste before mixing it with native black soap for bathing a cure for DM. In South-west Nigeria, fresh leaves of the plant are macerated in fresh palm wine and the filtrate taken orally for blood sugar control in suspected diabetic patients. Earlier, Olajide et al. reported the antidiabetic effect of the aqueous leaf extract of *Marinda lucida* in streptozotocin-induced hyperglycaemic rats. We also reported the antihyperglycaemic activity of the crude ethanolic leaf extract of *Marinda lucida* in normal and alloxan-induced hyperglycaemic rats. Bearing in mind the fact that identifying and isolating the effective extract fractions and isolating active fractions of a crude extract may prove better therapeutically and probably less toxic than its crude form, the present study aims to evaluate the antihyperglycaemic profile of the solvent fractions of the ethanolic leaf extract of *Marinda lucida* Benth in normal and alloxan diabetic rats for 14 days.

**MATERIALS AND METHODS**

**Plant collection and authentication:** Two kilogram of fresh leaves of *Marinda lucida* were collected from the same site as previously reported by Adeneye and Agbaje on the outskirts of Low Cost Housing Estate, Oke-Afa, Isolo, Lagos State, Nigeria in the months of October–November, 2009. Plant identification and authentication has earlier been done.

**Extract preparation:** Five hundred gram of *Marinda lucida* fresh leaves were exhaustively extracted in 1 L of 50% ethanol (Aldrich Chemical Co., USA) for 2 h using Soxhlet extraction procedure. The Soxhlet extractive was filtered using a piece of white cotton cloth and the filtrate obtained was completely dried into an aromatic green-brown solid residue over a water-bath. This procedure was repeated three more times. The residues obtained were pooled and stored in water- and air-tight container and kept refrigerated at -4°C until required for experiment.

**Solvent partitioning:** Fifty gram of the extract was completely deuterated in 100 mL of double-distilled water. The deuterated solution was then solvent partitioned in a 5 L burette using between 1 L to 1.5 L of different partitioning solvents (diethyl ether, chloroform, and butan-1-ol) in the order of their increasing solubility gradients. The fraction obtained with each partitioning solvent was concentrated *in vacuo* using rotary evaporator (Büchi Rotavapo® Model R-215, Switzerland) with Vacuum Module V-801 EasyVac® (Switzerland) set at a revolution of 70 rpm and a temperature of 35°C. The “extract residue” and the concentrate of each fraction were completely air-dried in an aerated oven preset at 35°C. The residues left after oven drying were then weighed. This procedure was repeated for 5 more times and each residue was pooled together and stored in clean and dry, water and air-proof containers and preserved in the refrigerator until required for experimentation.

**Preliminary Phytochemical analysis of MLEₙ and MLEₖ:** The presence of saponins, tannins, alkaloids, flavonoids, anthraquinones, glycosides and reducing sugars were determined by the simple and standard qualitative and quantitative methods described by Tread and Evans and Sofowora. The simple quantitative analysis of the extract was based on the intensity of the colour change. Briefly described, the qualitative phytochemical analysis of MLEₙ and MLEₖ was determined as follows:

**Tannins:** Two hundred milligram of each of the solvent fraction was dissolved in 10 mL of distilled water and then filtered. A 2 mL of filtrate was pipetted into a test tube after which 2 mL of 15% FeCl₃ was added and resultant colour change was observed. Blue-black presence indicated the presence of tannins.

**Alkaloids:** Two hundred milligram of the plant material was extracted with 200 mL of methanol for 20 min on a water bath and then filtered. To 2 mL of the cold water extract in different tubes, was added 6 drops of different alkaloids reagents, namely: Dragendorff’s or Mayer’s or Wagners’s reagent. Presence and colours of any precipitate were noted. Creamish precipitate or brownish-red precipitate or orange precipitate indicated the presence of respective alkaloids.

**Cyanogenic glycosides:** Two hundred milligram of the solvent fraction was placed in each of 3 different test tubes labeled A, B and C, respectively. The solvent fraction in test tubes A and B were moistened with 5 mL of water, while that in test tube C was left dry. Three pieces of freshly prepared sodium picrate paper were inserted into the mouth of each tube and stoppered. Test tube B was placed in a water bath while test tube A and C were kept at room temperature. After 30 minutes the colour of the picrate papers in each of the test tube were observed and recorded.

**Cardiac glycosides**

**Kedde’s test for lactone ring in cardiac glycosides:** Five hundred milligram of the solvent fraction was dissolved in 10 mL of methanol. To 2 mL of this, 1 mL of a solution of 2% of 3, 5-dinitrobenzoic acid in methanol and 1 mL of 5.7% sodium hydroxide were added. The result was recorded after 5 min.
Liebermann-Burchard reaction for steroidal/triterpenoidal nucleus: 500 mg of the dried solvent fraction was dissolved in 2 mL of acetic anhydride and allowed to cool. With the test tube inside ice pack and slanted at an angle of about 45°, 2 mL of concentrated tetroxosulphate (VI) acid was carefully poured by the side of the test tube. Colour obtained was noted. Blue-green ring indicated the presence of terpenoids.

Keller-Kiliani test for de-oxy sugars in cardiac glycosides: 50 mg of the solvent fraction was dissolved in 2 mL chloroform. Tetroxosulphate (VI) acid was added to form a layer and the colour at interphase recorded.

Legal test: The solvent fraction was dissolved in pyridine and 5 drops of 2% sodium nitroprusside together with 4-5 drops of 20% sodium hydroxide were added. Deep colour indicated the presence of cardenolides.

Salkowski’s test: 200 mg of the solvent fraction was dissolved in 2 mL of chloroform. Concentrated tetroxosulphate (VI) acid was carefully added to form a lower layer. A reddish-brown colour at the interface indicated the presence of a steroidal ring (i.e. aglycone portion of the cardiac glycoside).

Experimental animals: Healthy young adult male albino Wistar rats (120-150 g) used in this study were obtained from Zoology Department of the University of Ilorin, Kwara State, Nigeria. The rats were housed in polypropylene cages and handled in accordance with international principles guiding the Use and Handling of experimental animals. Rat feed (Ladokun Feeds, Ibadan, Nigeria) and tap water were provided ad libitum. The rats were maintained at an ambient temperature between 25-28°C, humidity of 56±5% and 12 h day/night photoperiod.

Oral treatment of normal rats with MLE fractions: To identify the biologically active fraction(s) of MLE, 50 mg kg⁻¹ of the diethyl ether fraction, chloroform fraction and butanol fraction each was constituted in 10 mL of 5% Tween-20 in distilled water was orally administered to each rat in each treatment group for 14 days. Thirty-six, young inbred adult male white albino Wistar rats (120-140 g) were randomly allotted to 6 groups. The rats were then fasted overnight for 12-14 h but had free access to drinking water. The basal fasting blood glucose of each rat was first determined using One Touch Basic Blood Glucose Monitoring System (LifeScan Inc., Milpitas, California, U.S.A.). This was then followed by the following treatments:

Group I: Rats were orally administered 10 mL kg⁻¹ of 5% Tween-20 in distilled water. Group II: Rats were orally treated with 20 mg kg⁻¹ of metformin while. Group III-VI: Rats were orally treated with a single daily dose of 50 mg kg⁻¹ of diethyl ether (MLE₉), chloroform fraction (MLE₈), and butanol fraction (MLE₇) and the “extract residue” (MLE₆), respectively, for 14 days. The effect of each extract fraction on the blood glucose concentration was then determined on the 15th day after an overnight fasting of the treated rats.

Induction of alloxan-hyperglycaemia in rats and their oral treatment with MLE fractions: Following a 24-h fast, rats were made hyperglycaemic by injecting each rat with a single intraperitoneal dose of 120 mg kg⁻¹ of alloxan monohydrate (Sigma Chemical Company, St. Louis, U.S.A.) dissolved in 3 mM of freshly prepared cold citrate buffer (pH 4.5). The baseline fasting blood glucose was first determined before alloxan treatment. Six hours after alloxan injection, rats were orally infused with 20% Dextrose (Unique Pharmaceuticals, Sango-Otta, Ogun State, Nigeria) at an oral dose of 10 mL kg⁻¹ so as to prevent the onset of fatal hypoglycaemia which often accompanies administration of alloxan as a result of acute massive pancreatic release of insulin. Gradual onset of hyperglycaemia was confirmed on the 3rd day post-induction but a steady hyperglycaemic state was achieved by the 5th day post-allocaxan treatment. By the 5th day, rats with fasting blood glucose of equal or greater than 200 mg dL⁻¹ were considered hyperglycaemic. Thirty-seven of the alloxan-treated rats had the fasting blood glucose concentration over 200 mg dL⁻¹ while the remaining three rats had spontaneous resolution of their hyperglycaemia.

Six normal and thirty-five alloxan-induced hyperglycaemic rats were randomly allocated into 7 treatment groups as follows:

- **Group I:** Consists of normoglycaemic rats which were orally treated with 10 mL kg⁻¹ of 5% Tween 20 dissolved in distilled water
- **Group II:** Consists of alloxan-induced hyperglycaemic rats orally treated with 10 mL kg⁻¹ of 5% Tween 20 in distilled water
- **Group III:** Consists of alloxan-induced hyperglycaemic rats orally pre-treated with 20 mg kg⁻¹ of metformin (Glucophage®, Hoechst Marion Roussel Limited, Mumbai, India) dissolved in 5% Tween 20 in distilled water
- **Groups IV-VII:** Consist of alloxan-induced hyperglycaemic rats that were orally pre-treated with 50 mg kg⁻¹ of diethyl ether, chloroform, butanol fractions and the “extract residue”, respectively. All treatments lasted 14 days after which the fasting blood glucose was determined
Bioassays: FBG was determined on one Touch Basic Blood Glucose Monitoring System using the glucose oxidase method of Trinder\(^{27}\). Serum triglyceride and total cholesterol were determined using standard test kits.

Data analysis: Results were expressed as mean±S.E.M. of six observations. Statistical analysis was done using two-way analysis of variance followed by post-hoc test, Student-Newman-Keuls test on SYSTAT 10.6. Statistical significance were considered at p<0.05, p<0.01 and p<0.001.

RESULTS

Extraction: The crude ethanolic extraction of *Marina huidra* fresh leaves gave a yield of 15.5% while that of solvent fractionation of MLE with diethyl ether, chloroform and butanol gave yield of 2.1, 15.6 and 20.0%, respectively, and 62.3% for the "extract residue".

Effect of MLE fractions on FBG, serum triglyceride and total cholesterol in normal rats: Daily oral treatment of normal Wistar rats with 50 mg kg\(^{-1}\) of MLE, and MLE\(_{s}\) resulted in significant (p<0.05, p<0.01 and p<0.001) time-dependent lowering of FBG in the treated rats with MLE, inducing the most significant (p<0.001) hypoglycemic effect over MLE\(_{s}\) and metformin, particularly, on the 15th day post-treatment (Table 1). However, oral treatment with MLE\(_{s}\) and MLE did not produce any appreciable changes in the FBG levels (Table 1). In a similar pattern, MLE, and MLE\(_{s}\) produced significant time-dependent (p<0.05, p<0.01 and p<0.001) lowering of the serum total cholesterol and triglyceride in the treated rats, with MLE, producing the most significant (p<0.001) hypolipidaemic effect when compared to that of MLE\(_{s}\) and metformin (Table 1). Again, MLE\(_{s}\) and MLE produced no significant (p>0.05) changes in the serum total cholesterol and triglyceride of the treated rats (Table 1).

**Table 1: Effect of *Marina huidra* leaf fractions on FBG, TG and TC in normal rats**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>FBG (mg dL(^{-1})) on Day</th>
<th>TG (mg dL(^{-1})) on Day</th>
<th>TC (mg dL(^{-1})) on Day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>1 Day 1</td>
<td>1 Day 8</td>
<td>1 Day 15</td>
</tr>
<tr>
<td>I</td>
<td>58.8±2.8</td>
<td>58.7±4.3</td>
<td>59.0±3.6</td>
</tr>
<tr>
<td>II</td>
<td>60.2±2.2</td>
<td>58.6±1.2(^{a})</td>
<td>47.5±2.2(^{a})</td>
</tr>
<tr>
<td>III</td>
<td>59.8±8.8</td>
<td>58.5±2.0</td>
<td>58.8±1.5</td>
</tr>
<tr>
<td>IV</td>
<td>56.7±3.0</td>
<td>56.2±1.9(^{a})</td>
<td>41.0±1.7(^{b})</td>
</tr>
<tr>
<td>V</td>
<td>56.0±4.3</td>
<td>51.5±2.2(^{a})</td>
<td>47.5±5.0(^{a})</td>
</tr>
<tr>
<td>VI</td>
<td>59.0±3.3</td>
<td>57.8±1.8</td>
<td>58.8±1.8</td>
</tr>
</tbody>
</table>

\(^{a}\) and \(^{b}\) represent significant decreases at p<0.05, p<0.01 and p<0.001, respectively, when compared to baseline value on day 1 and when compared to Group I values on days 1, 8 and 15. \(^{a}\) and \(^{b}\) represent significant decreases at p<0.05, p<0.01 and p<0.001 when compared to Group values. Group I: 10 mL kg\(^{-1}\) of 5% Tween-20 in distilled water. Group II: 20 mg kg\(^{-1}\) of metformin in 5% Tween-20 in distilled water. Group III: Single daily oral dose of 50 mg kg\(^{-1}\) of diethyl ether fraction (MLE). Group IV: Single daily oral dose of 50 mg kg\(^{-1}\) of chloroform fraction (MLE\(_{s}\)). Group V: Single daily oral dose of 50 mg kg\(^{-1}\) of butanol fraction (MLE\(_{b}\)). Group VI: Single daily oral dose of 50 mg kg\(^{-1}\) of the "extract residue" (MLE\(_{e}\)).

Effect of MLE fractions on FBG, serum triglyceride and total cholesterol in alloxan-induced hyperglycaemic rats: Single intraperitoneal injection with 120 mgkg\(^{-1}\) of alloxan monohydrate resulted in significant (p<0.001) hyperglycaemia by the 5th day post-induction (Table 2). However, oral daily treatment with 20 mg kg\(^{-1}\) of metformin, 50 mg kg\(^{-1}\) of MLE, and MLE\(_{s}\) resulted in significant (p<0.05, p<0.01 and p<0.001) time-dependent hypoglycaemic effect in the alloxan-induced hyperglycaemic rats, with MLE, producing the most significant hypoglycaemic effect on the 8th and 15th day of treatment (Table 2). In a similar pattern, hyperglycaemia induction with alloxan was associated with significant (p<0.001) elevation in the serum triglyceride and total cholesterol concentrations (Table 2). Also, oral treatment with 50 mg kg\(^{-1}\) of MLE caused the most significant (p<0.001) reductions in the serum triglyceride and total cholesterol levels compared to those induced by 20 mg kg\(^{-1}\) metformin and 50 mg kg\(^{-1}\) MLE\(_{s}\).

**Table 2: Effect of *Marina huidra* leaf fractions on FBG, TG and TC in normal and alloxan-induced hyperglycaemic rats**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Pre-induction FBG (mg dL(^{-1}))</th>
<th>Day 1</th>
<th>Day 8</th>
<th>Day 15</th>
<th>TG (mg dL(^{-1}))</th>
<th>TC (mg dL(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>58.7±2.7</td>
<td>59.3±1.7</td>
<td>58.7±2.1</td>
<td>58.3±3.0</td>
<td>93.7±5.4</td>
<td>89.8±5.1</td>
</tr>
<tr>
<td>II</td>
<td>59.3±2.9</td>
<td>250.0±7.3(^{a})</td>
<td>259.3±8.2(^{a})</td>
<td>276.8±12.2(^{a})</td>
<td>204.8±12.2(^{a})</td>
<td>170.0±16.5(^{a})</td>
</tr>
<tr>
<td>III</td>
<td>59.7±3.1</td>
<td>265.7±14.9(^{a})</td>
<td>151.2±14.9(^{a})</td>
<td>115.2±9.2(^{a})</td>
<td>116.8±13.5(^{a})</td>
<td>105.8±13.5(^{a})</td>
</tr>
<tr>
<td>IV</td>
<td>59.5±3.5</td>
<td>254.3±5.8(^{a})</td>
<td>260.3±11.0(^{a})</td>
<td>252.2±11.9(^{a})</td>
<td>182.5±6.8(^{a})</td>
<td>148.7±8.0(^{a})</td>
</tr>
<tr>
<td>V</td>
<td>59.7±2.7</td>
<td>252.2±6.9(^{a})</td>
<td>140.5±8.0(^{a})</td>
<td>107.5±9.5(^{a})</td>
<td>106.8±18.7(^{a})</td>
<td>96.2±16.2(^{a})</td>
</tr>
<tr>
<td>VI</td>
<td>58.8±2.6</td>
<td>250.8±6.3(^{a})</td>
<td>207.0±10.7(^{a})</td>
<td>153.2±14.6(^{a})</td>
<td>141.8±13.2(^{a})</td>
<td>128.8±8.4(^{a})</td>
</tr>
<tr>
<td>VII</td>
<td>60.1±7.5</td>
<td>252.6±10.3(^{a})</td>
<td>237.7±9.1(^{a})</td>
<td>231.2±6.8(^{a})</td>
<td>173.5±11.8(^{a})</td>
<td>145.2±8.2(^{a})</td>
</tr>
</tbody>
</table>

\(^{a}\) and \(^{b}\) represent significant increases at p<0.05, p<0.01 and p<0.001, respectively, when compared to pre-induction FBG values and Group I values while \(^{a}\) and \(^{b}\) represent significant decreases at p<0.05, p<0.01 and p<0.001, respectively, when compared to Group II. Values on days 1, 8 and 15. Group I: 10 mL kg\(^{-1}\) of 5% Tween-20 in distilled water + normal rats. Group II: 10 mL kg\(^{-1}\) of 5% Tween-20 in distilled water + alloxan hyperglycaemic rats. Group III: 20 mg kg\(^{-1}\) of metformin in 5% Tween-20 in distilled water + alloxan hyperglycaemic rats. Group IV: Single daily oral dose of 50 mg kg\(^{-1}\) of diethyl ether fraction (MLE) + alloxan hyperglycaemic rats. Group V: Single daily oral dose of 50 mg kg\(^{-1}\) of chloroform fraction (MLE\(_{s}\)) + alloxan hyperglycaemic rats. Group VI: Single daily oral dose of 50 mg kg\(^{-1}\) of butanol fraction (MLE\(_{b}\)) + alloxan hyperglycaemic rats. Group VII: Single daily oral dose of 50 mg kg\(^{-1}\) of the "extract residue" (MLE\(_{e}\)) + alloxan hyperglycaemic rats.
Preliminary phytochemical analysis of MLE and MLE: Preliminary phytochemical analysis of the two biologically active solvent fractions of MLE (MLE and MLE) showed the presence of flavonoids, alkaloids and anthraquinones only in MLE, and flavonoids, alkaloids, tannins and saponin in MLE.

DISCUSSION

In the present study, diethyl ether, chloroform, butanol and “extract residue” fractions of the crude ethanolic extract of Marinda lucida fresh leaves were evaluated for their anti-hyperglycaemic and antihyperlipidaemic profile in normal and alloxan-induced hyperglycaemia models. Alloxan, like its counterpart, streptozotocin has been frequently used to induce either type 1 or type 2 diabetes. Alloxan selectively accumulates in the pancreatic β-cells via the GLUT2 glucose transporter, and to this target mitochondrial DNA leading to impairment of mitochondrial signaling function and consequent induction of β-cell apoptosis through several mechanisms including caspases activation and Reactive Oxygen Species (ROS) production. The ROS mediates the cytotoxic action with the increase in cytosolic Ca2+ concentrations, leading to rapid β-cells destruction which results in hypoinsulinemia and eventual hyperglycaemia. In this study, alloxan-induced DM was most effectively controlled with repeated oral treatment with 50 mg kg−1 of MLE, as measured by significant lowering of the FBG, serum triglyceride and total cholesterol levels. These observations suggest that the phytochemicals responsible for the anti-hyperglycaemic activity of ethanolic crude extract of Marinda leaf are mostly partitioned into chloroform. Although, the possible antihyperglycaemic mechanism(s) of this fraction was not investigated in the present study, hyperinsulinemia mechanism is most unlikely since literature has it that for overt DM to become established up to 70% pancreatic β-cells must have been destroyed. Thus, it is most likely that effective glycemic control was achieved via increased action on cellular glucose uptake or intestinal glucose uptake inhibition. Similarly, alloxan is known to induce hyperlipidaemia in diabetic animals via increased mobilization of free fatty acids from the peripheral fat deposits. Since MLE, significantly lowered the serum triglyceride and total cholesterol levels in the treated rats, it may either be inhibiting mobilization of free fatty acids from the peripheral deposits or increasing their deposition into the peripheral tissues. Another significant finding of this study is the results of the phytochemical analysis of MLE, and MLE. Phytochemical analysis of MLE showed the presence of flavonoids, alkaloids, tannin and saponin while MLE showed the presence of flavonoids, alkaloids and anthraquinones. Previous independent studies have shown the crude Marinda lucida leaf extract to be rich in flavonoids, alkaloids, tannins and saponin. Also, oruwacin and anthraquinones, anthraquinones and anthraquinols identified as oruwal and oruwalo, morindin (glycoside) and tannins have also been isolated from Marinda lucida leaves. Literature has equally reported the biological activities of alkaloids and flavonoids to include hypoglycaemia, hypolipidaemia, hypoglycaemia, hypotension among other biological effects. Thus, the observed antihyperglycaemic and antihyperlipidaemic effect observed in this study could be attributed to the presence of flavonoids, alkaloids which were common denoting phytochemicals in both tested solvent fraction (MLE and MLE). Although the quantitative analysis of the phytochemicals in MLE and MLE was not undertaken in the present study, it is however, plausible that the concentrations of flavonoid and alkaloids could be higher in MLE than in MLE, in view of the fact that MLE exhibited more significant antihyperglycaemic and antihyperlipidaemic effects than MLE.

CONCLUSION

Overall, results of the present study showed the chloroform fraction of MLE (MLE) to be the most effective fraction in glucose and lipids homeostasis in both normal and alloxan-induced diabetic rats. Further studies geared towards isolating and characterizing the antihyperglycaemic and antihyperlipidaemic compounds in MLE, will be required in the nearest future.

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