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Evaluation of the Anti-hyperlipidemic Activity of Ethanolic and Aqueous Extracts of *Solanum melongena* in Wistar Rats

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Authors' contributions

This work was carried out in collaboration between all authors. Authors JNK, SS and JGN designed the study, wrote the protocol, managed literature searches, conducted the experimental procedures and wrote the first draft of the manuscript. Author JGN analyzed and presented the data. The final draft manuscript was revised by authors JNK and JGN. All authors read and approved the final manuscript.

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ABSTRACT

Hyperlipidemia is characterized by hypercholesterolemia and/or hypertriglyceridemia and can lead to obesity, arteroscelerosis and hypertension among others. Although different types of oral hypolipidemic agents are available for treatment of the condition, there is a growing interest in plant-based remedies due to the side effects and high cost of conventional antihyperlipidemic drugs. This study evaluated the antihyperlipidemic effects of ethanolic and aqueous extracts of *Solanum melongena* (Egg plant) fruits in Wistar albino rats. Fresh *S. melongena* fruits were bought from the market. Both aqueous and ethanolic fruit extracts were prepared. The study rats were induced with hyperlipidemia by feeding them on a high fat diet for 3 weeks. The extracts were administered daily, orally for 2 weeks to the rats at doses of 250 and 500 mg/kg b.wt. Atorvastatin (4 mg/kg) and distilled water, administered orally, were used as positive and normal control respectively. After 14 days, blood samples were collected in non-heparinised vacutainers by cardiac puncture. Serum levels of LDL, HDL, total cholesterol and triglycerides were determined

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using a Cobas 6000 automatic analyzer (Roche Diagnostic, USA). Phytochemical components of the extracts were determined qualitatively. There was a significant ($p<0.05$) increase in blood total cholesterol and triglycerides in all experimental animals following feeding with a high fat diet, with a significant reduction ($p<0.05$) in LDL at post treatment when compared with pretreatment phase. All extract doses caused weight decrease in all treatment groups when compared to the normal control group. The 500 mg/kg dose of the ethanolic extract hand the greatest ($p<0.05$) anti-hyperlipidemic activity. *S. melongena* activity may be attributed to multiple mechanisms of phytochemicals present.

Keywords: Hyperlipidaemia; hypercholesterolemia; hypertriglyceridemia; high fat diet; LDLs; HDLs; total cholesterol; triglycerides; Solanum melongena.

1. INTRODUCTION

At least 2.8 million people worldwide die each year as a result of hyperlipidemia [1]. The global prevalence of hyperlipidemia has nearly doubled between 1980 and 2008. For example in 2008, 14% of women in the world were hyperlipidemic compared to 8% in 1980 [2]. The same study estimated that 205 million men and 297 million women over 20 years were hyperlipidemic worldwide- more than half a billion adults. In Africa, Eastern Mediterranean and South Eastern Asia the prevalence of the condition in women roughly doubled that in men. The prevalence of hyperlipidemia is also rapidly increasing among children. In Africa the prevalence of hyperlipidemia is increasing despite high levels of under nutrition. Hyperlipidaemiais a growing problem in young adults (18-30 years) in Uganda and 53.2% of the affected individuals are in Kampala. Data shows that 10.4% of the adult population in Kampala is affected while the prevalence in Kamuli is 10.2% [3].

Compared to males, females are more likely to be hyperlipidemic (2.9% vs1.8%). Residing in the city, alcohol consumption, smoking, non-engagement in sports activities, commuting to school by vehicle and being from a rich family are significantly associated with hyperlipidemia [4]. Hyperlipidemia and obesity are associated with hypertension and diabetes mellitus, coronary heart disease, ischemic stroke and increased risk of breast, colon, prostate, kidney and bladder cancer [5]. Hyperlipidemia is also associated with increased mortality. There is increased risk of morbidity for people with BMI 25-29.9 and severe risk of morbidity for BMI greater than 30 [6].

Interventions used in control and treatment of hyperlipidemia include pharmacological and non-pharmacological approaches. The non-pharmacological approaches include weight reduction, sodium restriction, increased aerobic exercise, cessation of alcohol consumption and illicit drug use prohibition. The pharmacological approaches include the use of allopathic and traditional medicine. The allopathic (conventional) medicines include diuretics, angiotensin receptor blockers, Angiotensin converting enzyme inhibitors, calcium channel blockers, *beta* adrenergic blockers, *alpha* adrenergic blockers, NSAIDs and statins [7]. However these drugs are expensive and not easily available to the rural and poor people [2]. They also possess some side effects which some individuals may not tolerate and this is coupled with the long distance which people travel in order to access the drugs. As a result, most of the individuals have now resorted to traditional medicinal plants that are cheap, easily accessible and believed to have fewer side effects compared to allopathic drugs [8]. Several medicinal plants are reportedly used in management of hyperlipidaemia and these include *Adenatherapavonina* [9]; *Aarpagophytum procumbens* [10]; *Cantella asiatia*, *Catharanthus roseus*, *Bambusa vulgaris* and *Camellia sinensis* [11]; *Nigella sativa* [12]; *Solanum lycopersicum* [13] *and Solanum melongena* [14]. Furthermore, *S. melongena* has been reported to also have analgesic, anti-inflammatory and anti-pyretic activity [15], as well as anti-amnesic effects [16]. Therefore, this study evaluated further the anti-hyperlipidemic effect of *S. melongena* fruits, a plant used for weight reduction in many communities in Africa.

2. MATERIALS AND METHODS

2.1 Plant Materials

Fresh fruits of *S. melongena* were bought from Nakasero market, one of the major fresh vegetable markets in Kampala in February, 2014. Good fruit specimens were selected and a valid sample was submitted to the Botany Herbarium

of Makerere University for authentication. Samples were taken to the Pharmaceutical and Toxicology lab of the College of Veterinary Medicine, Animal Resources and Biosecurity (COVAB) of Makerere University for extract preparation.

2.2 Preparation of the Extracts

The fresh fruits of *S. melongena* were separated from the pulps, chopped into small pieces and oven-dried at 60ºC for 36h with turnover instances to a constant weight. The dried sample was pulverized in a coarse powder manually using mortar and pestle. For the ethanolic extract, serial extractions were done using 500g of powder soaked in 1000 ml of 95% ethanol in Erlenmeyer flasks for 3 days with occasional shaking. The mixture was decanted and filtered using What man No.1 filter paper into a Buchner funnel. This process was repeated for 3days to increase the extract yield. The filtrate was concentrated by use of a rotary evaporator to a constant volume, which was later dried at 50ºC in an oven to obtain a thick paste, stored at 4ºC in a refrigerator. For the aqueous extract, 500g of powder were steamed for 20minutes. The mashed mixture was squeezed manually using clean goselin cloth. The filtrate was put in mouth-wide kidney disc, oven-dried at 55ºC for 36h into a thick paste which was stored in bijous bottle at 4ºC for later use. Stock solutions (100 mg/ml) of extracts were prepared by dissolving 0.5g of powder in 5ml of distilled water. Dimethyl sulphoxide was used in proportions of 1:9 to water to dissolve the ethanol extract.

2.3 Experimental Animals

Wistar albino rats (1:1 male to female ratio) were procured from Makerere University, College of Veterinary Medicine Animal Resources and Biosecurity animal houses. Healthy rats with clean-white and smooth coat were selected and for females, only non-nulliporous ones were chosen. The weights of the rats (90-200g) were determined using an electronic weighing balance. The animals were allowed one week to acclimatize in the lab prior to the study and maintained under standard laboratory conditions of natural light and darkness, room temperature, relative humidity and rodent pellets (Engaano Millers, Kampala). Water was provided *ad libitum* until the termination of the study.

2.4 Preparation of a High Fat Diet

A high fat diet with high proportion of cholesterol was prepared. The constituents of the diet were lard 38%; normal rodent pellets 29%; dextrose 13%; milk powder 15%; egg yolk 4% and multivitamin 1%.Rodent pellets were crushed into powder and then added to the other constituents in a mixing bowl. All components were mixed together, with distilled water added gradually to convert the homogenous mix into a fairy hard paste. The uniformly mixed paste was molded using a small diameter plastic pipe to obtain the shape of ordinary pellets. The pellets were then put on absorbent paper and placed in an oven at 60ºC overnight to dry.

2.5 Experimental Set Up

Fifty two (52) rats were randomly allocated to 6 groups and fed on the high fat diet to induce hyperlipidemia. Feeding with this diet continued throughout the experiment. However, the rats in the normal control group were only fed on normal rodent pellets. Twenty one (21) days after commencing on this diet, the rats in groups A-D were then administered with the extracts at doses shown in (Table 1), while those in groups E and F (normal and positive controls respectively) were administered with distilled water or atorvastatin. All treatments were done orally, daily for 14 days using gavage tubes. Treatment doses were based on earlier study on *S. melongena* [15] and were calculated for each animal and per dose group as described by Ndukui et al. [17] using the equation:

$$\text{Dosing volume (mls)} = \frac{\text{Dose (mg/kg)} \times \text{Animal body weight (kg)}}{\text{Concentration of stock (mg/ml)}}$$

2.6 Serum Lipid Profile and Body Weight Determination

Blood samples were collected from four randomly selected rats per group on day 0 to obtain baseline lipid profiles (before starting the high fat diet); day 21 (to verify induction of hyperlipidemia) and day 35 (after 2 weeks of daily administration of the extracts or atorvastatin). The rats were enclosed in a square glass container and anesthetized by inhalation using cotton wool soaked in 99.9% diethyl ether. Blood was collected into clearly labeled non-heparinised vacutainers by cardiac puncture with a 2 ml hypodermic needle inserted at a 35° angle at the base of the sternum after disinfecting the site with an alcohol swab. Serum was prepared from the blood samples and serum levels of LDL, HDL, total cholesterol and triglycerides determined using a Cobas 6000 automatic

analyzer (Roche Diagnostic, USA). The body weights of the rats were also determined on days 0, 21 and 35 of the experiment.

2.7 Qualitative Determination of the Phytochemicals in the Extracts

The phytochemical composition of the extracts was qualitatively determined with minor modification as described by Harborne [18]. The aqueous extract was hydrolyzed for some of the assays.

2.7.1 Alkaloids

Samples (0.5g) of each extract were defatted with 5% ethyl ether in test tubes for 15 minutes and extracted for 20 minutes with 5ml HCl on a boiling water bath. The resulting mixtures were then be centrifuged at 3000 rpm for 10minutes. To 1 ml of the filtrate a few drops of Mayer's reagent were added followed by 1ml of Dragendorff's reagent. The intensity of turbidity of the two samples was observed under UV light at 365nm.

2.7.2 Tannins and saponins

For tannins, 0.5gof powder was weighed, boiled in 20ml of distilled water in a test tube and filtered. A few drops of 0.1% ferric chloride were added to the test tubes and observed for formation of brownish-green or blue-black coloration. For saponins, 2g of powder were boiled in 20ml of distilled water in a water bath and filtered. Ten (10) ml of each filtrate were mixed with 5ml of distilled water and shaken vigorously for stable and persistent froth. The frothing was mixed with 3 drops of olive oil and shaken vigorously and observed for emulsion formation.

2.7.3 Cardiac and steroid glycosides

For cardiac glycosides, 5 g of powder were weighed and 2 ml of glacial acetic acid containing 1 drop of ferric chloride solution added. Addition of 1 ml of conc. H_2SO_4 leads to formation of a brown ring of the interface indicating a deoxy-sugar of cardenolide. A violet ring may appear below the brown ring while a greenish ring forms gradually throughout the thin layer. For steroid glycosides, the extracts in labeled crucibles were evaporated to dryness. The residues were dissolved in glacial acetic anhydride. Each solution was transferred to a dry test tube. Using a pipette, conc. H_2SO_4 was added at the bottom of each tube. A reddish brown ring at the separating level of two liquids showed the presence of steroid glycosides.

2.7.4 Flavanones and mucilages

For flavanones, 5 ml of each extract were evaporated in test tubes to dryness and the residues in each tube were dissolved in 50% methanol by heating. Metal Mg and 5 drops of conc. HCl were added and presence of an orange solution indicated presence of flavanones. For mucilages, 2ml of extract were gradually added in test tubes followed by 10ml of acetone, ethanol and finally methylene blue. The occurrence of a violet blue precipitate denoted the presence of mucilages.

2.7.5 Glucides, anthracyanosides and coumarins

For glucides, 2ml of extract were transferred to different test tubes and evaporated to dryness. Three drops of conc.H_2SO_4were added and after5 minutes, furfural or hydro methyl furfural was formed. Then 4 drops of each extract saturated with thymol were added. The occurrence of a red precipitate denoted the presence of glucides. For anthracyanosides, to 4ml of extract, 1ml of 10% HCl was added followed by ammonium hydroxide solution to form a red solution. Presence of coumarins was determined by evaporating 3ml of extract to dryness. The residues were dissolved in hot water, cooled and the solutions divided into two portions. One set of tubes served as reference while others were made alkaline with 0.5ml ammonia solution. The occurrence of an intense fluorescence under UV light at 365 nm of the long wavelength indicated the presence of coumarins.

2.8 Data Analysis

Body weight, LDL, HDL, triglycerides, total cholesterol values were expressed as means±SEM. The differences in these values for the different doses and groups were determined with a two way ANOVA and student t-test. Bonferroni's multiple comparison test was used to ascertain mean differences among the treatment groups and considered statistically significant at $p<0.05$.

Table 1. Selected treatment groups and doses

Group	Treatment	Dose rates	No. of animals
A	Aqueous extract	250mg/kg	8
B	Aqueous extract	500mg/kg	8
C	Ethanol extract	250mg/kg	8
D	Ethanol extract	500mg/kg	8
E	Distilled water (normal control)	10ml/kg	12
F	Atorvastatin (positive control)	4mg/kg	8

2.9 Ethical Considerations

This study was approved by the Ethical Review Committee of the College of Veterinary Medicine, Animal Resources and Biosecurity of Makerere University. Laboratory animals were handled according to animal handling guidelines as per NACLAR declaration.

3. RESULTS

Percent yields were 10.2% and 38.3% for the ethanolic and aqueous extract respectively. The ethanolic extract had a higher concentration of tannins, flavanones and saponins. The aqueous extract had higher concentrations of cardiac glycosides, mucilages and glucides (Table 2).

Table 2. Phytochemical composition of test extracts of *Solanum melongena*

Phytochemical	Ethanolic extract	Aqueous extract
Alkaloids	++	_
Tannins	+++	+
Saponins	++	_
Cardiac glycosides	+	+++
Flavanones	+++	_
Anthracerosides	++	++
Coumarins	+	_
Steroid glycosides	+	_
Mucilages	_	++
Glucides	_	+

Key: +++ strongly present; ++ moderately present; + weakly present; - absent

3.1 Effect of the Test Extracts on the Weight of the Animals

The high fat diet caused increase in body weight in all treatment groups (represented by day 21 in Table 3). Thereafter, the test extracts and Atorvastatin caused decrease in body weight in all treatment groups when compared to the pre-treatment, post-hyperlipidemia induction weight. However, this decrease was only significant ($p<0.05$) for the group treated with the 500mg/kg dose of the ethanolic extract. The weight changes in other treatment groups were not significant ($p>0.05$). It is evident that the extracts had an effect of decreasing body weight as well as levels of serum total cholesterol, LDL and triglycerides as well as increasing HDL levels.

3.2 Antihyperlipidemic Activity of the Extracts

There was a significant ($p<0.05$) increase in blood total cholesterol and triglycerides levels in the test animals after feeding them with a high fat diet. Following treatment with both extracts, there was a significant reduction ($p<0.05$) in LDL in all groups post treatment when compared with the pre- treatment LDL levels. Both extracts at both doses caused a significant reduction ($p<0.05$) in LDL values just like atorvastatin. The increase in HDLs was not significant ($p>0.05$) in all treatment groups following treatment with the extracts (Table 4).

4. DISCUSSION

The high fat diet induces hyperlipidemia because increased intake of non-esterified cholesterol by rats results into increased absorption via the intestines. This raises blood total cholesterol and triglycerides which are transported to the liver through the hepatic portal vein. The excess total cholesterol in the liver enhances production of LDLs through the LDL receptors by stimulating synthesis of VLDL which is converted to LDL through the action of endothelial cell-associated lipoprotein lipase [6]. This explains increase in body weight, triglycerides, LDL and total cholesterol after the hyperlipidemia inducement phase (3 weeks of high fat diet).

This study showed a significant reduction ($p<0.05$) in serum LDL in all the treatment groups for both extracts post treatment when compared with pre-treatment levels. Both extracts at 250 mg/kg and 500mg/kg doses caused significant reduction in LDL; indicating the hypocholesterolemic activity of the extracts.

Table 3. Effects of the crude extracts of *S. melongena* on body weight of the rats

Treatment group	Body weight in g (mean±SEM)			
	Day 0 (baseline)	Day 21 (after induction)	1 week post treatment with extracts	2 weeks post treatment with extract
Group A(250mg/kg)aqueous	123.9±11.50	149.1±6.31	133.0±12.59	95.30±3.04^{*c}
Group B (500mg/kg) aqueous	117.2±9.33	129.4±6.18	113.8±7.72	91.85±7.50^{*c}
Group C(250mg/kg)ethanolic	119.1±15.35	130.0±12.08	117.0±9.67	99.93±6.06
Group D (500mg/kg) ethanolic	123.4±3.12	161.6±11.20^{*a}	143.2±10.95^{*b}	121.8±9.37^{*b}
Normal control E	110.2±4.66	113.2±6.99	108.0±4.30	109.2±4.52
Positive control F(atorvastatin)	123.1±11.74	152.8±16.22	133.7±3.81	117.1±3.91

Key: n=8; a - significant increase in body weight after induction when compared with day 0; b* - significant reduction in body weight when compared to day 21 after induction; *c-Significant reduction in body weight when compared to day 21 after induction;*

Table 4. Blood lipid parameters (mmol±SEM) for the treatment groups

Treatment group	Normal control (E)	250 mg/kg aqueous (A)	500 mg/kg aqueous (B)	250 mg/kg ethanolic (C)	500 mg/kg ethanolic (D)	Positive control (F)
Total cholesterol						
Pre-treatment	2.12±0.20	2.48±0.17^{*a}	2.47±0.08^{*a}	2.34±0.06^{*a}	2.20±0.25^{*a}	2.54±0.32^{*a}
Post-treatment	2.11±0.05	2.31±0.15	2.25±0.05^{*c}	2.21±0.12	2.17±0.16	2.16±0.39
HDL						
Pre-treatment	1.55±0.12	1.46±0.14	1.44±0.16	1.51±0.08	1.47±0.17	1.51±0.07
Post-treatment	1.66±0.05	1.57±0.31	1.58±0.09	1.63±0.05^{*d}	1.63±0.15	1.65±0.05^{*d}
LDL						
Pre-treatment	1.0±0.04	0.53±0.08	0.61±0.04	0.29±0.03	0.58±0.07	0.45±0.04
Post-treatment	0.07±0.03	0.28±0.06^{*b}	0.25±0.06^{*b}	0.24±0.03	0.21±0.04^{*b}	0.13±0.04^{*b}
TGs						
Pre-treatment	1.0±0.04	2.48±0.48^{*a}	1.34±0.26^{*a}	1.47±0.10^{*a}	1.12±0.08	1.23±0.26
Post-treatment	1.0±0.12	1.68±0.27^{*c}	1.34±0.05	1.20±0.10^{*c}	1.03±0.09	1.01±0.03

Key: n=4; Pretreatment after 3 weeks (21 days), post treatment after two weeks (14 days) of hyperlipidemic induction; Bonferroni's multiple comparison test used to analyze for significant changes between treatment groups. p<0.05;^{*a}-significant increase in the study parameter when compared with normal control group at pre-treatment level; ^{*b}- significant reduction in study parameters when compared with normal control group post treatment; ^{*c}- significant reduction in the study parameters when comparing pre-treatment with post treatment of a given study group; ^{*d}- significant increase in the study parameters when comparing pre-treatment with post treatment study groups using non parametric student t-test

During metabolism and synthesis of cholesterol in the body, hyperlipidaemia is often a stimulus for increased LDL synthesis [19]. However, *S. melongena* extracts seem to inhibit this hepatic production of LDL. The overall reduction in blood total cholesterol levels caused an overall weight loss in all treatment groups with a significant loss at a dose rate of 500 mg/kg of the ethanolic extract. The increase in HDL levels in all treatment groups although not significant resulted into reverse cholesterol transport were the HDLs returned total cholesterol to LDLs in the liver. The body gets rid of excess cholesterol and triglycerides by converting them to bile acids or excretion of free cholesterol [19]. This was indicated by reduction in levels of total cholesterol although not significant.

There was a significant increase in levels of triglycerides in all treatment groups at pre-treatment phase. Triglycerides are part of dietary total cholesterol; therefore a rise in blood total cholesterol directly increases the levels of triglycerides in blood [6]. The decrease in the levels of triglycerides by atorvastatin was due to the inhibition of excess triglyceride production by the liver through the HMG CoA pathway and increased excretion in feces together with bile salts [4]. While atorvastatin enhances cholesterol and triglyceride excretion through bile salts in faeces and hinders total cholesterol deposition in peripheral body tissues [6], the mode of action of the *S. melongena* extracts is unknown. It could however be probably attributed to the phytochemicals in the plant.

Studies by Odetola and Akinloye [20] showed that treatment of hyperlipidaemic rabbits with some *S. melongena* extracts for 6 weeks caused a significant decrease in total cholesterol, LDL, triglycerides but a significant increase in HDL levels. The difference could be explained by the differences in metabolic requirements and rate of rats and rabbits, the longer treatment phase (6 weeks) for the previous study could have had an additional effect on lipid parameters. Differences in geographical locations could also affect phytochemical components of the plant.

The demonstrated activity of *S. melongena* could be due to the reported antihyperlipidemic action of mucilages, glucides, cardiac glycosides and coumarins [21]. Mucilages reportedly reduce intestinal absorption of total cholesterol and triglycerides. Coumarins have blood thinning ability that increases blood flow in the veins, this increases blood transportation of cholesterol and triglycerides to metabolizing organs. Cardiac glycosides increase cardiac output [22]; the increased blood circulation increases cholesterol, triglyceride and oxygen transport to the organs. Glucides protect the cell membranes, and act as catalysts in the provision of molecules of immunity [23]; the cell membrane protection protects body organs from the adverse effects of plagues and catalyses enzyme controlled reactions responsible for cholesterol and triglyceride metabolism [24].These actions may be responsible for the observed effects on lipid parameters.

The results of this study have demonstrated that *S. melongena* extracts have antihyperlipidemic and body weight reducing effects. High levels of total cholesterol in blood especially LDL lead to formation of plagues in vessels and reduction in their diameter. This predisposes to hypertension and related cardiovascular ailments [24].

5. CONCLUSION

This study underlines the potential use of this plant as a source of alternative remedies for the management of obesity and hyperlipidemia especially at the highest dose of 500mg/kg of both aqueous and ethanolic extract. Therefore, inclusion of *Solanum melongena* in the diet of hyperlipidemic individuals could probably have medicinal values and prophylactic effects against these two non-communicable conditions in humans.

CONSENT

Not applicable.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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