**ANTIHYPERLIPIDEMIC POTENTIAL OF *Scoparia dulcis* LEAF AQUEOUS EXTRACT IN ALLOXAN-INDUCED DIABETIC RATS**

**ABSTRACT**

This study was to investigate the anti-hyperlipidemic potential of aqueous extract of*Scoparia dulcis*in alloxan induced wistar albino rat. Diabetes mostly results from abnormal insulin synthesis and it negatively affects the metabolism of carbohydrates, proteins, fats, electrolytes, and water. *Scoparia dulcis* is a medicinal plant that exert its antihyperlipidemic effect by reducing the synthesis of cholesterol in the liver, lowering the absorption of lipids from the intestine, and potentially increasing the breakdown of lipids in addition to its antidiabetic effect. The aqueous extract of the leaf was obtained by immersing the leaf in distilled water at 500g in 1500 litres of water for 24 hours. The acute toxicity was conducted in two phases. In the first phase, nine animals were grouped into three of three rats per group and administered 10ml of normal saline, 100 and 1000 mg/kg of aqueous extract of *Scoparia dulcis* orally and monitored for twenty-four hours for signs of toxicity like, tremor, restlessness, dizziness and death. In the second phase, 10 groups (IV-XIII) of one rat each were given oral dose of aqueous extract of *Scoparia dulcis* at 750, 1000. 1250, 1500, 1750, 2000, 2250, 2500, 3500 and 5000 mg/kg respectively and monitored for twenty-four hours for signs of toxicity. The result revealed that the administration of the extract of *Scoparia dulcis* orally in rats did not produce any behavioural sign of toxicity, there was no death recorded at all the doses tested up to 5000 mg /kg in the rats. The analysis of variance result of the 11 days lipid profile study revealed that the group of rats treated with the aqueous extract of *Scoparia dulcis* revealed that there was a non-significant reduction (p>0.05) in Total Cholesterol (TC), Triglyceride (TG), Low density lipoprotein (LDL) and High-density lipoprotein (HDL) when compared with the diabetic untreated group. Thus, this research revealed that the aqueous extract of*Scoparia dulcis*has antilipidemic effects and holds potential as an alternative therapy for its management.

Keywords: *Scoparia dulcis*; antihyperlipidemic; aqueous; toxicity; extract; cholesterol

INTRODUCTION

Diabetes is an epidemic endocrine metabolic disorder. It is distinguished by incessant hyperglycemia and hyperlipidemia as a result of interference in carbohydrate and lipid metabolism, a pathological process that may include deficiency in insulin action or/and insulin secretion (Offor et *al*., 2024).

The primary cause of myocardial infarction is atherosclerosis and it is typified by the development of fatty plaques that block blood flow in the arteries; this is brought on by elevated blood lipid concentrations (hyperlipidemia), cholesterol (hypercholesterolemia), and triglyceride levels (hypertriglyceridemia). The risk of dying from atherosclerotic illnesses may be decreased by identifying and preventing hyperlipidemia. (Santos *et al*., 2018). “With the renewed and increased interest in trado-medicine coupled with emerging side effects with the application of orthodox medications, attention is being channeled in promoting and redesigning herbal products with the intent of harnessing both their nutritive and medicinal efficacy for societal welfare. Medicinal plants are more affordable and have less side effects compared to synthetic drugs, and they are more effective in treatment of diabetes mellitus” (Kooti et al., 2016).

*Scoparia dulcis*, also known as sweet broomweed possesses a significant antihyperlipidemic effect in rats with alloxan-induced diabetes, meaning it can help lower elevated levels of lipids (like cholesterol and triglycerides) in the blood of diabetic rats induced by alloxan treatment; this is attributed to its potential to regulate lipid metabolism and potentially prevent complications associated with high cholesterol levels in diabetes (Pari and Latha, 2004). According to Oliveira *et al*. (2015), flavonoids and saponins are the key substances to decrease cholesterol levels. Thus, the presence of these chemical compounds can explain the hypolipidemia and hypocholesterolemia activity of the extract of *Scoparia dulcis*.

**METHODOLOGY**

**Site for the Research**

The study was carried out in the Zoology Research and Development Center of the Department of Zoology located at Nnamdi Azikiwe University, Awka, Anambra State.

**Procurement of Experimental Animals**

A total of 75 adult male albino rats aged 2 – 3 months, weighing 160-200g were used for the experiment. The animals were allowed to get acclimatized with the environment one week before the commencement of the experiment. They were fed throughout the research period with water and vital growers’ chick mash pellets. The *experiment* lasted for 11 days. The research was conducted according to the Nnamdi Azikiwe University-Animal Research Ethical guidelines.

**Collection and Identification of the Medicinal Plant (*Scoparia dulcis*)**

Fresh leaves of *Scoparia dulcis* was collected from Pharm. Opi Eze research farm in Amansea, Awka-North Local Government Area, Anambra State. Thereafter, it was taken to the herbarium of the Botany Department of Nnamdi Azikiwe University, Awka, Anambra state for identification and authentication by a plant taxonomist. It was given the herbarium number: NAUH- 225a.

**Preparation of Plant Materials**

The aqueous leaf extract of *Scoparia dulcis* was obtained with the method of Joselin *et al.* (2020) with modifications. The dried leaves of the *Scoparia dulcis* were ground into powder using electric grinder and then immersed in distilled water at 500g in 1500 litres of water for 24 hours. Thereafter, the mixture was filtered with Whatman no. 1 (125mm) filter paper to obtain aqueous extract of the leaf. The filtrate was concentrated with a rotary evaporator to remove the water until it is in pellet form. The extract was then put into several test tubes, stopped with a cork to prevent air and contaminants and refrigerated at +2 - +8oC.

**Induction of Diabetes mellitus**

The blood glucose levels of all the animals were taken just before the administration of the alloxan using glucose test strips and a glucometer. Diabetes was induced by a single intraperitoneal injection of freshly prepared alloxan monohydrate dissolved in 1 ml normal saline to the overnight fasted normal glycaemic experimental albino rats. After the induction, all the rats were allowed free access to food and water. The animals were allowed to rest for 48 hours when sustained hyperglycaemia is expected (Balamash *et al.,* 2018). An animal was considered diabetic when their blood glucose level was ≥ 200 mg/dl (IDF, 2021). Two days after alloxan administration, blood samples were obtained from the tips of the rat’s tail (rat tail vein puncture method) and the fasting blood glucose levels was determined using glucose test strips and Fine test glucometer to confirm diabetes. This gives a digital result within 45 seconds.

**Preparation of Glibenclamide Suspension**

Glibenclamide tablet was crushed and mixed with 20 ml of sterile distilled water. The mixture was kept in an ultrasonic water bath for 45 minutes until a homogenous mixture is obtained. The suspension was administered orally to the induced diabetic rats at 5mg/kg body weight.

**Acute toxicity study**

The toxicity test was carried out on albino rats in accordance with the protocol devised by Lorke (1983). In the first phase, nine animals were grouped into three of three rats per group and administered 10ml of normal saline (control), 100 and 1000 mg/kg of aqueous extract of *Scoparia dulcis* orally and monitored for twenty-four hours for signs of toxicity like, tremor, restlessness, dizziness and death. In the second phase, 10 groups (IV-XIII) of one rat each were given oral dose of aqueous extract of *Scoparia dulcis* at 750, 1000, 1250, 1500, 1750, 2000, 2250, 2500, 3500 and 5000 mg/kg respectively and monitored for twenty-four hours for signs of toxicity.

**Experimental design**

This experiment was carried out using a complete randomized block design. A total of 75 adult male wistar albino rats (*Rattus norvegicus*) was used. After acclimatization which lasted for 1 week, the rats were weighed using an analytical weighing balance and randomly distributed into five (5) treatment groups (T1-T5) with each group containing 5 rats. Each treatment group were replicated three times.Treatment 1 (T1) contained non-diabetic rats. In this group, they were given normal feed (growers' chick mash) and 2ml/kg of distilled water was orally administered using gastrointestinal cannular. This was the normal control group. Treatment 2 (T2) was induced with an intraperitoneal injection of alloxan at 150mg/kg and not treated (positive control). Treatment 3 (T3) was induced with an intraperitoneal injection of alloxan at 150mg/kg and treated with 200mg/kg dosage of the leaf extract of *Scoparia dulcis*. Treatment 4 (T4) was induced with an intraperitoneal injection of alloxan at 150mg/kg and treated with 600mg/kg dosage of the leaf extract of *Scoparia dulcis*. Treatment 5 (T5) was induced with an intraperitoneal injection of alloxan at 150mg/kg and treated with oral administration of gilbenclamide at a dose of 5mg/kg.

**Treatments**

Treatment 3 (T3) and Treatment 4 (T4) were given the aqueous leaf extract of *Scoparia dulcis* once in 24 hours through oral gavages using canular for 11 days during the experimental period at a fixed time of 10:00 am. Treatment 5 (T5) was given glibenclamide at 5mg/kg body weight. Fasting blood glucose levels were checked by collecting a blood sample from the tail vein every 3 days and determined using fine test glucometer and glucose test strips (code 25) at 8:00 am before treatment and feeding.

**Collection of Blood Sample**

At the end of the experiment, 11th day, the fasting blood glucose level of all the animals were taken, the animals were weighed and the animals were anaesthetized using chloroform and bled by cardiac puncture. The blood sample was collected in specimen bottles, allowed to clot and the serum was then centrifuged at 4000rpm. The bottles were immediately capped and preserved in a bowl of ice blocks. Later the blood was refrigerated at 4° C for about three hours and allowed to defrost before the analysis proper.

**LIPID PROFILE TEST PROCEDURES**

1. **Total Cholesterol**: The method of Allain *et al*., 1974 was adopted. The cholesterol content of the serum was measured at 546nm using a UV-Visible spectrophotometer.

Principle of Assay: The cholesterol was determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase.

$$Cholesterol+H\_{2}0 cholestreol esterase \rightarrow Cholesterol+Fatty Acid$$

$$Cholesterol+ O\_{2} cholesterol esterase \rightarrow Cholesterol-3-one+ H\_{2 }0\_{2} $$

$$2H\_{2 }0\_{2}+Phenol+4-Aminoantipyrine peroxidase \rightarrow Quinoneimine+ H\_{2}0$$

**Procedure**: A randox Total cholesterol kit was used. The serum (10µL) was mixed with 1000µL of cholesterol reagent and allowed to stand for 10mins at room temperature after which the total cholesterol content was measured using a spectrophotometer.

1. **Triglyceride**: The method described by Fossati and Prencipe (1982) was used. The triglyceride content of the serum was measured at 546nm using a UV-Visible spectrophotometer.

**Principle of Assay**: A randox triglyceride kit was used. This was determined after enzymatic hydrolysis with lipases.

 $Triglyceride+ H\_{2 }$0 $lipases \rightarrow Glycerol+fatty acid $

$$Glycerol-3-ATP Glycerol kinase \rightarrow Glycerol-3-Phosphate$$

$$Glycerol-3-Phosphate+0\_{2} Glycerol-3-Phoshate Oxidase \rightarrow Dihydroxyacetonephosphate+H\_{2}0\_{2}$$

$$2H\_{2}0\_{2}+4-Aminophenazone+4-chlorophenol POD \rightarrow Quimomneimine+HCL+4H\_{2}O$$

**Procedure**: The serum (10µL) was mixed with 1000µL of Triglyceride reagent and allowed to stand for 10mins at room temperature after which the Triglyceride content was measured using a UV-visible spectrophotometer.

1. **High density lipoproteins cholesterol (HDL-c):** This was carried out according to the method described by Warnick *et al*., 1982.This was determined by measuring the amount of cholesterol remaining in the serum after precipitation of LDL, VLDL and Chylomicron by the addition of phosphotungstic acid and magnesium chloride. The HDL content was measured as the remaining cholesterol in the sample solution after precipitation.

Procedure: The serum (200µL) was mixed with 500µL of Randox HDL reagent and allowed to stand for 10mins at room temperature. It was centrifuged at 4000rpm for 10mins. The supernatant (100µL) was mixed with 1000µL of cholesterol reagent and allowed to stand for 10mins after which the HDL content was measured using a UV-Visible spectrophotometer.

1. **Low density lipoproteins (LDL-c)**: This was determined for each of the samples by using the Friedewald formula: $LDL=Total cholesterol-\left(\frac{Triglyceride}{5}\right)-LDL$

**STATISTICAL ANALYSIS**

Using the Statistical Package for Social Science software for windows version 25, data on the lipid profile levels were reported as Mean ± SEM and compared among groups with One Way ANOVA Tests at (P<0.05) significant levels.

**RESULTS**

**Acute Toxicity Profile of the Aqueous Leaf Extracts of *Scoparia dulcis***

The oral median lethal dose (LD50) values of the aqueous leaf extracts of *Scoparia dulcis* were estimated to be greater than 5000 mg/kg in rats as shown in table 1.

**Table 1: Acute toxicity of the aqueous extract of *Scoparia dulcis***

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Phases | Serial number | Groups | Number of animals | Dosage | Mortality | Symptoms |
| Phase 1 | 1 | Group I | 3 | Normal saline (Control) (10ml/kg) | 0/3 | Nil |
|  | 2 | Group II | 3 | 100mg/kg | 0/3 | Nil |
|  | 3 | Group III | 3 | 500mg/kg | 0/3 | Nil |
| Phase 2 | 4 | Group IV | 1 | 750mg/kg | 0/1 | Nil |
|  | 5 | Group V | 1 | 1000mg/kg | 0/1 | Nil |
|  | 6 | Group VI | 1 | 1250mg/kg | 0/1 | Nil |
|  | 7 | Group VII | 1 | 1500mg/kg | 0/1 | Nil |
|  | 8 | Group VIII | 1 | 1750mg/kg | 0/1 | Nil |
|  | 9 | Group IX | 1 | 2000mg/kg | 0/1 | Nil |
|  | 10 | Group X | 1 | 2250mg/kg | 0/1 | Nil |
|  | 11 | Group XI | 1 | 2500mg/kg | 0/1 | Nil |
|  | 12 | Group XII | 1 | 3500mg/kg | 0/1 | Nil |
|  | 13 | Group XIII | 1 | 5000mg/kg | 0/1 | Nil |

**Lipid Profile**

**Table 2: Average lipid profile of the various experimental groups**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Groups | Total cholesterol (mg/dl) | Triglycerides (mg/dl) | High density lipoproteins (HDL) | Low density lipoproteins (LDL) |
| Normal control | 168.98±8.75 | 129.85±10.10 | 46.85±6.05 | 107.39±18.48 |
| Diabetic untreated | 202.22±12.66 | 138.67±1.21 | 50.44±6.36 | 139.97±12.11 |
| 200mg/kg extract group | 201.80±7.60 | 134.97±2.85 | 49.31±0.57 | 120..67±16.49 |
| 600mg/kg extract group | 198.81±4.35 | 138.40±2.50 | 40.35±0.45 | 133.34±0.30 |
| Gilbenclamide | 183.56±7.50 | 141.61±0.00 | 45.40±4.97 | 115.47±3.61 |

**Total cholesterol**

Changes in lipid metabolism are one of the symptoms of onset of diabetes mellitus. Figure 1 below shows the average total cholesterol levels of the various groups after the experimental period. From the results, the untreated group had the highest total cholesterol level (202.22±12.66 mg/dl) followed by the extract groups while the normal control had the least (168.98±8.75 mg/dl) though the differences were not significant (p>0.05) when all the groups were compared p≥0.05.

**Figure 1: Bar chart showing average total cholesterol levels of the various groups after the experimental period.**

**Triglycerides**

Figure 2 below shows the average triglyceride levels after the experimental period.The standard drug group had the highest triglyceride levels (141.61±0.00 mg/dl) while the normal control group had the least (129.85±10.10 mg/dl). The treatment groups all had increased triglyceride levels after onset of diabetes mellitus. The analysis of variance revealed that there was no significant difference with the triglyceride of the various groups (p>0.05). This is also as a result of reduced insulin inhibition of lipolysis and reduced activity of lipoprotein lipase, thus the increase in triglyceride levels.

**Figure 2: Bar chart showing average triglyceride levels after the experimental period.**

**High density lipoproteins (HDL)**

Figure 3 shows the average HDL levels after the experimental period. There was no significant difference in HDL levels when all the groups were compared with the 600mg/kg extract group having the least HDL levels (40.35±0.45 mg/dl) and the untreated having the highest HDL levels (50.44±6.36 mg/dl). Onset of diabetes may not have any significant effect on HDL but might affect LDLs. Also, the 200 mg/kg extract group had the highest HDL levels when compared with the control, 600mg/kg and gilbenclamide groups, thus its potential to increase good cholesterol levels at such doses.

**Figure 3: Bar chart showing average HDL levels after the experimental period**

**Low density lipoproteins (LDL)**

Figure 4 below shows the LDL (Bad cholesterol) levels of the various groups. The diabetic untreated group had the highest LDL levels while the control group had the least (107.39±18.48 mg/dl) which was significantly lower than the rest of the groups. The non-significantly increased LDL levels in the treatment groups show a healing state since they were still lower than that of the untreated group.

**Figure 4: Bar chart showing average LDL levels of the various groups after the experimental period.**

**DISCUSSION**

“Hyperlipidemia is a metabolic disorder that constitutes a crucial risk factor of atherosclerosis and cardiovascular diseases” (Jiang *et al*., 2021). “It has been demonstrated that insulin deficiency in diabetes mellitus leads to accumulation of lipids such as total cholesterol and triglycerides in diabetic patients” (Luciani *et al*., 2024).

“The median lethal dose (LD50) value is used for assessing the safety margin of substances and it often gives an idea of the toxic level of a chemical substance or compound. It provides the prospect for discovering and maximizing the clinical benefits of test compounds” (Ahmed *et al*., 2019). The oral median lethal dose (LD50) values of the aqueous leaf extracts of *Scoparia dulcis* were estimated to be greater than 5000 mg/kg in rats. The administration of extract of *Scoparia dulcis* orally in rats did not produce any behavioural sign of toxicity, there was no death recorded at all the doses tested up to 5000 mg /kg in the rats. This is in agreement with the study of Ahmed at al. (2019) and Coulibaly *et al*. (2020). This suggests that the extract is practically non-toxic.

The result of the lipid profile study revealed that the group of rats treated with the aqueous extract of *Scoparia dulcis* showed a non-significant reduction in Total Cholesterol (TC), Triglyceride (TG), Low density lipoprotein (LDL) and High-density lipoprotein (HDL) when compared with the diabetic untreated group. The result is supported by the findings of Jiang *et al*. (2021) who reported that in diabetic rats treated with 200 mg/kg of aqueous extract of *Scoparia dulcis*, their serum cholesterol, low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), triglycerides, were reduced, but in contrast with their report on high-density lipoprotein (HDL) levels, which were elevated. This result is in contrast with the observation by Adebiyi *et al*. (2021) who stated that there was also a significant increase (p<0.05) in total cholesterol, triglyceride, low density lipoprotein cholesterol though they reported that there was a significant decrease in HDL-cholesterol which supports this result. Santos *et al*. (2018) also reported that acute treatment with ethanolic extract of *Scoparia dulcis* (500mg/kg) was effective to reduce total cholesterol; however, the extract did not present significant reduction in triglycerides levels. The observation of Sarkar *et al*. (2020) for 6 weeks on streptozotocin diabetic rats which were administered with aqueous extract of the *Scoparia dulcis* orally resulted in reduction of serum and tissue cholesterol, triglycerides, free fatty acids, phospholipids and very low-density lipoprotein and low-density lipoprotein cholesterol levels also supports the result. This result can be attributed to the findings by Oliveira *et al*. (2015) who stated that flavonoids and saponins are the key substances to decrease cholesterol levels. Thus, the presence of these chemical compounds in *Scoparia dulcis* can explain the hypolipidemia and hypocholesterolemia activity of the extract.

**CONCLUSION**

The primary cause of myocardial infarction is atherosclerosis and it is typified by the development of fatty plaques that block blood flow in the arteries; this is brought on by elevated blood lipid concentrations (hyperlipidemia), cholesterol (hypercholesterolemia), and triglyceride levels (hypertriglyceridemia). This study revealed the antilipidemic potential of the aqueous extract of *Scoparia dulcis* at varied dosage on diabetic-induced rats and therefore can be used as adjunctive therapy to subjugate diabetes.

**DISCLAIMER (ARTIFICIAL INTELLIGENCE)**

Author(s) hereby declares that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators were used during writing or editing of manuscripts.

**ETHICAL APPROVAL**

The Animal Research Ethical Committee of Nnamdi Azikiwe University, Awka, Anambra state approved this study and it was assigned the reference number; NAU/AREC/2024/0092.

**COMPETING INTERESTS**

Authors have declared that no competing interests exist.

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