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ANTIDIABETIC EFFECT OF THE AQUEOUS EXTRACT OF LECANIODISCUS CUPANIOIDES PLANCH. (SAPINDACEAE) LEAVES ON ALLOXAN-INDUCED HYPERGLYCEMIA IN RATS

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ABSTRACT

This study evaluates the antihyperglycemic effect of the aqueous leaf extract of *Lecaniodiscus cupanioides* (EAq Lc) in Wistar rats made diabetic by alloxan. The animals were divided into five groups and treated orally with the extract at doses of 250 and 500 mg/kg, compared to a control group receiving glibenclamide (100 mg/kg). The results show a significant reduction in blood glucose, serum urea, and serum creatinine, as well as normalization of liver markers (ASAT, ALAT, PAL). At a dose of 500 mg/kg body weight, the extract reduced blood glucose levels by 81.68% compared to those of the diabetic control rats, urea by 58.9%, creatinine by 57.96%, aspartate aminotransferase (ASAT) by 42.34%, and alkaline phosphatase (ALP). The extract at 250 mg/kg bw also reduced alanine aminotransferase (ALT) by 68.57% compared to the diabetic control. Thus, the aqueous extract of Lecaniodiscus cupanioides (EAq Lc) regenerated pancreatic β-cells and restored hepatic and renal markers.

KEYWORDS: *Lecaniodiscus cupanioides*, antihyperglycemic, renal and hepatic markers.

INTRODUCTION

Diabetes is a chronic metabolic condition characterized by persistent hyperglycemia, resulting from a deficiency in insulin secretion or insulin action. According to the WHO, more than 422 million people currently live with diabetes, and this number continues to rise, particularly in developing countries.[1] Conventional treatments, such as sulfonylureas or metformin, have limitations, including side effects and reduced long-term efficacy.[2] In this context, medicinal plants represent a valuable source of bioactive compounds. Several recent studies have shown that phytoconstituents such as flavonoids, alkaloids, and terpenoids exert antidiabetic effects by improving insulin sensitivity, stimulating pancreatic secretion, or inhibiting intestinal glucose absorption.[3,4]

Lecaniodiscus cupanioides (Sapindaceae), widely used in traditional African medicine, is recognized for its antidiabetic, antioxidant, and anti-inflammatory properties.[5] However, few experimental studies have explored its antihyperglycemic activity. This study aims to evaluate the effect of the aqueous extract of Lecaniodiscus cupanioides on blood glucose levels and hepatic and renal biochemical parameters in alloxan-induced diabetic rats.

MATERIALS AND METHODS

Plant Material

The plant material consists of leaves of *Lecaniodiscus cupanioides* Planch (Sapindaceae). The leaves of Lecaniodiscus cupanioides were collected in December 2024 at the University of Man, located 7 kilometers from the city of Man, near the village of Kassiapleu to the west, in the Tonkpi region of Côte d'Ivoire. Identification was performed by the National Center for Floristics (CNF) at Félix Houphouët-Boigny University in Cocody.

Animal

The animal subjects consisted of albino *Rattus norvegicus* rats of the Wistar strain weighing between 170 g and 220 g. These animals were 10 to 16 weeks old and had free access to water and food consisting of pellets supplied by the company "Ivograin." The animals were housed in the central laboratory at the University of Man under a 12-hour light/12-hour dark photoperiod. They were cared for in accordance with good laboratory practices.[6] The various experimental protocols were followed in accordance with the European Council's animal welfare protocols under Directive 2012/707.[7]

Pharmacodynamic substances and chemicals

Distilled water, an aqueous extract of *Lecaniodiscus cupanoides*, diethyl ether (VWR International, Leuven, Belgium), and glybenclamide (reference compound) were used in this study.

Preparation of the aqueous extract of *Lecaniodiscus cupanioides*

The leaves were washed with distilled water and then dried at room temperature (22–24°C). They were then ground into a powder using an electric grinder (GM 300, RETSCH brand). A quantity of powdered leaves of Lecaniodiscus cupanioides Planch (Sapindaceae) (100 g) was macerated in 1 L of distilled water, then homogenized under magnetic stirring for 24 h. This process prevents oxidation or enzymatic hydrolysis. The macerate was squeezed through a square of sterile cloth and filtered successively through cotton to obtain a clear filtrate. The filtrate is slowly dried in an oven (AGROLAB TCN 115) at 50 °C. This yields the aqueous extract of Lecaniodiscus cupanioides (EAq Lc). The resulting powder is stored in a sterilized, hermetically sealed jar.

Antihyperglycemic effect of EAqLc in alloxan-induced hyperglycemia in treated rats

The antihyperglycemic effect of EAqLc was evaluated using the method described by.[8] Thirty (30) rats were randomly divided into 5 groups of 6 animals each. Prior to the experiment, the rats' blood glucose levels were measured using blood samples taken from the rat's tail. Sixteen (16) hours after the rats were fasted, an intraperitoneal injection of alloxan monohydrate dissolved in sterile saline at a dose of 180 mg/kg was administered. The rats were administered a 50 mg/mL D-glucose solution via gavage at a rate of 10 mL/kg body weight over the following 3 days. After the 3-day period, blood glucose levels were measured. Rats with an increase in fasting blood glucose of 1 mmol/L or more were considered hyperglycemic and were included in the study. All rats received their respective treatments orally via a gastric tube. Animals in Group 1 (healthy, untreated controls) and Group 2 (diabetic, untreated controls) received distilled water. Rats in Groups 3 and 4 received, orally, EAqLc at doses of 250 and 500 mg/kg body weight, respectively, in accordance with the doses prescribed by traditional therapists (two to three glasses of the extract decoction per day) and the[9] acute toxicity limit. Rats in group 5 (negative control) were force-fed glibenclamide at 100 mg/kg body weight. measurements of the study parameters were taken on day 0, day 4, and day 20.

Blood Collection from Rats

Three (3) blood samples were collected at the start of the experiment (Day 0), on Day 4 (D4), and on Day 20 (D20) of the experiment. Approximately 3 ml of blood was collected in red or sterile dry tubes, and after centrifugation, serum was obtained for biochemical analysis (RAYTO-RT 7600S; China). These samples were collected during the experiment from rats that had been fasted for 16 hours and anesthetized with diethyl ether. Blood was collected by puncture at the retroorbital sinus of the eye.

Measurement of blood glucose, serum liver, and kidney markers

Blood collected in dry tubes was centrifuged at 3,000 rpm for 10 minutes to obtain serum. Biochemical parameters were analyzed using an automated analyzer (RAYTO-RT 7600S; China). Measurements of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin, and direct bilirubin were performed. The various assays were performed using the LABKIT reagent according to the methods (kinetic, enzymatic, and colorimetric) described by,[10;11]

Statistical Analysis

The results are expressed as the mean followed by the standard error of the mean (M±SEM). Statistical analysis was performed using GraphPad Prism 8.01 software (San Diego, California, USA). One-way analysis of variance (ANOVA1) followed by Turkey's comparison test was used to demonstrate differences between the treatment and control groups. Values are considered significant at $p < 0.05$.

RESULTS

Effect of an aqueous extract of Lecaniodiscus cupanoides on capillary blood glucose levels in rats

Table 1 shows the changes in capillary blood glucose levels during alloxan-induced hyperglycemia in rats treated with EAq Lc. Capillary blood glucose levels in the rats prior to alloxan injection ranged from 0.70 ± 0.30 g/dL to 0.97 ± 0.05 g/dL. Three days after injection, blood glucose levels increased significantly, rising from 1.22 ± 0.77 to 5.75 ± 0.02 g/dL.

On day 20, no significant variation in capillary blood glucose levels was observed in diabetic rats. Treatment with the aqueous extract of Lecaniodiscus cupanoides (EAq Lc) resulted in a significant reduction in blood glucose levels. At a dose of 250 mg/kg, blood glucose levels decreased from 5.24 ± 0.35 g/dL on Day 4 to 1.03 ± 0.08 g/dL on Day 20, representing an

80.34% reduction; and at a dose of 500 mg/kg, it decreased from 5.27 ± 0.25 g/dL on Day 4 to 0.96 ± 0.02 g/dL on Day 20, corresponding to an 81.68% reduction. Glibenclamide (100 mg/kg), used as a reference, resulted in a significant reduction in alloxan-induced blood glucose levels, with a percentage reduction of 85.30%. **(Table 1).**

Table 1: Changes in capillary blood glucose levels during alloxan-induced hyperglycemia in rats treated with EAq Lc.

Doses (mg/kg body weight)	Capillary blood glucose (g/dL) / Percentage increase or decrease		
	Day 0	Day 4	Day 20
Healthy controls (10 mg/kg body weight)	0.70 ± 0.30	1.22 ± 0.07	1.06 ± 0.05
Diabetic controls (10 ml/kg body weight)	0.97± 0.09	5.75 ±0.02 ### **(371.3%)**	5.24 ± 0.16 ### **(394.33%)**
EAq Lc 250	0.92 ± 0.04	5.24 ± 0.35### **(329.51%)**	1.03 ± 0.08*** **(80.34%)**
EAq Lc 500	0.86 ± 0,03	5.27 ± 0.25### **(331.96%)**	0.96 ± 0.02*** **(81.68%)**
Gly 100	0.76 ± 0.03	4.99 ± 0.13### **(309.02%)**	0.77 ± 0.03*** **(85.30%)**

p < 0.001; n = 5; significant difference in capillary blood glucose levels between the alloxan-induced hyperglycemic groups and the healthy control group on the same day. ***p < 0.001; n = 5; significant difference in capillary blood glucose levels between the EAq Lc-treated groups and the diabetic control group on the same day.

Where: EAq Lc: *Lecaniodiscus cupanoides* aqueous extract; Gly: Glibenclamide

Effect of the aqueous extract of Lecaniodiscus cupanoides on renal parameters

- **Effect of the aqueous extract of EAq Lc on urea concentration**

Urea concentration ranged from 0.30 ± 0.05 to 0.86 ± 0.03 g/L on day 4. In diabetic controls, a significant decrease in urea concentration was observed, from 0.81 ± 0.01 to 0.73 ± 0.02 g/L, representing a percentage change of 114.7%. Following oral administration of EAq Lc, a significant variation in urea concentration was observed compared to the control group. Thus, the urea concentration in rats treated with EAq Lc at the doses studied (500; 250 mg/kg body weight) ranged from 0.79 ± 0.06 to 0.83 ± 0.03 g/L on day 4 and from 0.3 ± 0.02 to 0.46 ± 0.08 g/L, representing respective percentages of 58.90% and 36.98% on day 20. Glibenclamide (100 mg/kg) causes a significant reduction in urea concentration, which is 0.86 ± 0.03 g/L on day 4 and 0.33 ± 0.05 g/L on day 20, representing a rate of 54.79% **(Table 2).**

- **Effect of the EAq Lc aqueous extract on creatinine concentration**

Administration of EAq Lc causes a significant change in creatinine concentration compared to the control group. In the control group of rats, creatinine concentration ranged from 4.10 ± 1.48 to 11.3 ± 0.31 g/dL, representing a 175.61% increase, while in those treated with EAq Lc, it ranged from 9.93 ± 0.92 to 10.3 ± 1.03 g/dL on day 4. On day 20, a significant decrease in creatinine concentration was observed in all rats **(Table 2).**

Table 2: Changes in serum renal markers during alloxan-induced hyperglycemia in rats treated with EAq Lc.

Days	Doses (mg/kg body weight)	Serum renal markers / Percentage increase or decrease	
		Urée (g/dl)	Créatinine (g/dl)
Day 4	Healthy controls (10 mg/kg body weight)	0.30 ± 0.05	4.10 ± 1.48
	Diabetic controls (10 ml/kg body weight)	0.81 ± 0.01$^{###}$ **(170%)**	11.3 ± 0.31$^{##}$ **(175.61%)**
	EAq Kp 250	0.83 ± 0.03$^{##}$ **(176,66%)**	9.93 ± 0.92$^{#}$ **(142.19%)**
	EAq Kp 500	0.79 ± 0.06$^{#}$ **(163.33%)**	10.3 ± 1.03$^{#}$ **(151.22%)**
	Gly	0,86 ± 0,03$^{##}$ **(186.66%)**	10,0 ± 1,25$^{#}$ **(143.90%)**
Day 20	Healthy controls (10 mg/kg body weight)	0.34 ± 0.03	3.97 ± 0.79
	Diabetic controls (10 ml/kg body weight)	0.73 ± 0.02$^{###}$ **(114.7%)**	10.3 ± 2.27$^{#}$ **(159.44%)**
	EAq Kp 250	0.46 ± 0.08* **(36.98%)**	4.77 ± 0.61* **(53.69%)**
	EAq Kp 500	0.3 ± 0.02*** **(58.90%)**	4.33 ± 0.88* **(57.96%)**
	Gly	0.33 ±0.05*** **(54.79%)**	4.30 ± 0.47* **(58.25%)**

# p < 0.05; ## p < 0.01; ### p < 0.001; n = 5; significant difference in urea and creatinine levels between the alloxan-induced hyperglycemic groups and their respective healthy control groups on the same day. *p < 0.05; ** p < 0.01; *** p < 0.001; n = 5; significant difference in urea and creatinine levels between the groups treated with EAq Lc and their respective diabetic control groups on the same day.

Where: EAq Lc: Aqueous extract of *Lecaniodiscus cupanoides*; Gly: Glibenclamide.

Effect of the aqueous extract of *Lecaniodiscus cupanoides* on liver parameters

Effect of EAq Lc on aspartate aminotransferase (AST) levels

The ASAT levels in the control group of rats were 132.2 ± 2.73 and 310 ± 47 U/L, whereas those in rats treated with EAq Lc and glibenclamide ranged from 294 ± 16.3 to 390 ± 29.4 U/L on day 4. A significant variation was observed, with a decrease at the doses studied ranging from 143 ± 6.41 to 174 ± 10.8 U/L on day 20 **(Table 3).**

Effect of EAq Lc on alanine aminotransferase (ALT) levels

After 20 days of the experiment, the ALAT levels in rats administered EAq Lc at doses of 250 and 500 mg/kg body weight decreased significantly compared to the control group. These levels ranged from 55.3 ± 2.60 to 106 ± 2.3 U/L (Table 3).

Effect of EAq Lc on alkaline phosphatase (ALP) levels

On day 20, the alkaline phosphatase level in the control group of rats was 35.1 ± 4.32 U/L. A significant decrease was observed in rats administered EAq Lc at doses of 500 and 250 mg/kg body weight, ranging from 550 ± 209 U/L to 712 ± 9.70 U/L. As for glibenclamide, it decreased from 913 ± 112 to 281 ± 19.1 U/L (Table 3).

Table 3: Changes in serum liver markers during alloxan-induced hyperglycemia in rats treated with EAq Lc.

Days	Doses (mg/kg body weight)	Serum liver markers / Percentage increase or decrease		
		ASAT(U/L)	ALAT(U/L)	PAL(U/L)
Day 4	Healthy controls (10 mg/kg body weight)	132 ± 2.73	91 ± 6.08	341 ± 45.4
	Diabetic controls (10 ml/kg body weight)	310 ± 47.4$^{\#\#}$ **(134.84%)**	197 ± 8.66$^{\#\#}$ **(116.48%)**	1061 ±90.1$^{\#\#}$ **(211.14%)**
	EAq Kp 250	326 ± 32.3$^{\#\#}$ **(146.97%)**	178 ± 14.7$^{\#\#}$ **(95.60%)**	912 ± 36.1$^{\#}$ **(167.44%)**
	EAq Kp 500	390 ± 29.4$^{\#\#\#}$ **(195.45%)**	196 ± 15.5$^{\#\#}$ **(115.38%)**	1072 ± 141$^{\#\#}$ **(214.37 %)**
	Gly	294 ± 16.3$^{\#\#}$ **(122.72%)**	181 ± 9.33$^{\#\#}$ **(98.90%)**	913 ± 112$^{\#}$ **(167.75%)**
Day 20	Healthy controls (10 mg/kg body weight)	132 ± 1.45	95,7 ± 16.8	351 ±43.2
	Diabetic controls (10 ml/kg body weight)	248 ± 28.1$^{\#\#}$ **(87.87%)**	176 ± 8.84$^{\#\#}$ **(83.90%)**	860 ± 87.8$^{\sharp}$ **(145.01%)**
	EAq Kp 250	174 ± 10.8 **(29.83%)**	55.3 ± 2.60*** **(68.57%)**	712 ± 9.70 **(17.21%)**

	EAq Kp 500	143 ± 6.51** **(42.34%)**	106 ± 2.6** **(39.77%)**	550 ± 209 **(36.04%)**
	Gly	144 ± 11.0** **(41.93%)**	79.0 ± 5.20*** **(55.11%)**	282 ±19.1* **(67.20%)**

# p < 0.05; ## p < 0.01; ### p < 0.001; n = 5; significant difference in AST, ALT, and PAL levels between the alloxan-induced hyperglycemic groups and their respective healthy control groups on the same day. *p < 0.05; ** p < 0.01; *** p < 0.001; n = 5; significant difference in AST, ALT, and PAL levels between groups treated with EAq Lc and their respective diabetic control groups on the same day.

Where : EAq Lc : *Lecaniodiscus cupanoides* aqueous extract ; Gly: Glibenclamide ; ASAT : Aspartate aminotransferase; ALAT : Alanine aminotransferase; PAL : Alkaline phosphatase.

DISCUSSION

Intraperitoneal injection of alloxan over a 3-day period in rats caused a significant increase ($p<0.001$) in blood glucose levels. Alloxan disrupted carbohydrate metabolism, as evidenced by hyperglycemia and decreased insulin levels. Indeed, alloxan acts through a cytotoxic effect on pancreatic β-cells mediated by the production of free radicals and by a disruption of the homeostasis of free cytosolic Ca2+ in Langerhans β-cells. Thus, alloxan causes a deficit in insulin secretion, resulting in severe hyperglycemia.[12] After 14 days of treatment, EAq Lc significantly reduced ($p<0.001$) blood glucose levels during the 3-day alloxan-induced hyperglycemia. This result suggests that EAqLc may have acted to restore β-cells in the islets of Langerhans and that EAqLc may have acted like glibenclamide by blocking the ATP-K+ pump, thereby preventing glucose entry into the islet cells during nutrient absorption. Furthermore, some researchers claim that alloxan-induced hyperglycemia may be reversible, due to the relatively rapid and frequent spontaneous regeneration of the pancreas in the presence of this reagent.[12] This finding confirms that EAqLc regenerated the pancreas after three days of hyperglycemia induction via intraperitoneal injection of alloxan at 180 mg/kg body weight. Our results corroborate those of[13], who demonstrated that Nigella sativa reduced hyperglycemia and restored pancreatic function following alloxan-induced diabetes in rats. After three days of hyperglycemia induced by alloxan at a dose of 180 mg/kg body weight, urea and creatinine levels increased significantly ($p<0.001$) compared to those in the control group. Indeed, elevated levels of these two parameters are associated with renal dysfunction in diabetic patients, resulting from the toxicity of alloxan or high glucose on the kidneys (nephropathy).[14;15] Several studies have demonstrated the nephrotoxicity of alloxan

and glucose on the kidneys of animals.[16;17] Furthermore, elevated levels of renal serum markers and certain electrolytes are believed to be linked to impaired renal function. Additionally, creatinine is known as an important marker of kidney function, particularly glomerular filtration.[18] Serum creatinine and urea levels are elevated in patients with renal insufficiency, particularly those with reduced glomerular filtration. In the early stages of kidney damage, an increase in serum urea levels generally precedes the increase in serum creatinine observed in chronic kidney disease.[19] However, after 14 days of treatment, EAq Lc significantly reduced urea and creatinine levels. This suggests that this extract corrected all dysfunctions related to glomerular filtration and any tissue damage that might have occurred following intraperitoneal injection of alloxan. Thus, EAq Lc protected the kidneys against the toxicity of alloxan and hyperglycemia. Our results corroborate those of.[5] These authors demonstrated that the aqueous extract of Phyllostachys aurea (Poaceae) at various doses did not cause hypoglycemia, but antihyperglycemic activity was observed.

Injection of alloxan at 180 mg/kg body weight into the intraperitoneal cavity for 3 days in rats caused a significant increase ($p<0.001$) in aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP). Indeed, these biochemical markers allow for the assessment of liver cell function.[20] During the 3 days of hyperglycemia induction, alloxan caused an elevation of these markers in serum, indicating liver damage and dysfunction.[21;22] Oral administration of EAqLc resulted in a significant decrease in these levels. This suggests that EAqLc may protect liver function and promote the conversion of glucose into glycogen. Our results corroborate those of.[5] These authors demonstrated that the aqueous extract of Phyllostachys aurea (Poaceae) at various doses did not cause hypoglycemia, but antihyperglycemic activity was observed.

CONCLUSION

The aqueous extract of *Lecaniodiscus cupanioides* (EAq Lc) regenerated pancreatic β-cells and restored liver and kidney markers. The aqueous extract of Lecaniodiscus cupanioides thus appears to be a promising alternative in the management of diabetes and its complications.

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