

Toxicological Evaluation and Hypoglycemic Effects of *Solanum aethiopicum* Fruit Extracts in Wistar Rats

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Abstract

Introduction: Dietary strategies for diabetes management traditionally prioritize foods with low or moderate glycemic indices to attenuate postprandial glucose excursions. Nevertheless, glycemic responses remain highly variable, prompting growing interest in bitter-tasting foods and their bioactive constituents as potential dietary interventions for improving glycemic control and metabolic outcomes. **Objective:** This study aimed to evaluate the toxicity and hypoglycemic activity of *Solanum aethiopicum* fruits consumed in the Democratic Republic of the Congo. **Materials and Methods:** An experimental randomized study was conducted using Wistar rats. Animals were allocated into six groups for LD₅₀ determination and five groups for the evaluation of hypoglycemic activity. The hypoglycemic potential of *Solanum aethiopicum* was assessed using the oral glucose-induced hyperglycemia method. LD₅₀ and serum biochemical parameters, including urea, creatinine, AST, ALT were determined using standard analytical methods. **Results:** The median LD₅₀ of *Solanum aethiopicum* extracts exceeded the highest dose administered in this study (5000 mg/kg body weight), suggesting a low toxicity profile. Mean blood glucose levels varied significantly over the experimental period (0 - 180 min), $p < 0.05$. A significant difference was also observed between the effects of *Solanum aethiopicum* extracts and glibenclamide ($p < 0.05$), suggesting that the extracts do not exert direct hypoglycemic effects. Furthermore, no significant

alterations were detected in the biochemical parameters evaluated, including urea, creatinine, AST, and ALT, irrespective of the administered dose. **Conclusion:** The results of this study indicate that *Solanum aethiopicum* fruits are non-toxic and do not possess direct hypoglycemic activity.

Keywords

Solanum Aethiopicum, Hypoglycemic Activity, LD₅₀, Biochemical Parameters

1. Introduction

Diabetes mellitus represents a major and rapidly growing global public health challenge. According to the World Health Organization [1] and the International Diabetes Federation [2], the disease has reached pandemic proportions, with the number of affected individuals increasing from approximately 30 million worldwide in 1985 to 366 million in 2011. More recent estimates indicate that 536.6 million adults aged 20 - 79 years were living with diabetes in 2021, with similar prevalence in men and women and the highest prevalence observed among individuals aged 75 - 79 years [3]. If current trends persist, the global burden of diabetes is projected to exceed 550 million cases by 2030 and continue rising through 2045, with particularly marked increases expected in urban populations [2].

Diabetes is a major global health challenge associated with substantial morbidity and mortality arising from numerous complications. It remains one of the leading causes of death worldwide, accounting for millions of deaths each year [4]. In 2019, diabetes directly caused an estimated 1.5 million deaths, with nearly half occurring before the age of 70 years. Beyond its direct effects, diabetes substantially increases the risk of other life-threatening conditions, including chronic kidney disease and cardiovascular diseases. Hyperglycemia alone has been implicated in approximately 20% of cardiovascular-related deaths, underscoring the far-reaching health burden of diabetes [5]. The burden of diabetes is increasing rapidly in low- and middle-income countries, particularly in sub-Saharan Africa. In this region, the number of people living with diabetes was estimated at 14.7 million in 2011, including more than 737,000 cases in the Democratic Republic of the Congo [6]. By 2021, the diabetic population in the DRC had risen to approximately 1.9 million adults, with the disease accounting for more than 34,000 deaths [7]. Earlier studies reported a diabetes prevalence of 7% among adults in Kinshasa [8]. However, the true burden of the disease is likely higher, as diabetes remains substantially underdiagnosed and many patients rely on private healthcare facilities and traditional medicine, which are often not captured in official health statistics [9].

Effective management of diabetes is critical for preventing or delaying the onset of disease-related complications. Contemporary management strategies adopt a multidisciplinary approach that integrates pharmacological interventions, including insulin and oral antidiabetic agents, with dietary modification, regular physi-

cal activity, and routine medical follow-up [10]. In addition, international guidelines underscore the importance of continuous monitoring through regular clinical consultations and periodic comprehensive assessments, which are essential for optimizing glycemic control, improving clinical outcomes, and enhancing patients' quality of life [11] [12].

In parallel, medicinal plants are increasingly being investigated as complementary strategies for diabetes management. The WHO has identified more than 400 plant species traditionally used for the treatment of diabetes mellitus; although, only a limited number have been subjected to rigorous pharmacological and clinical [13]. Ethnopharmacological studies in the Maghreb region have documented a rich diversity of antidiabetic medicinal plants, including *Citrullus colocynthis*, *Berberis vulgaris*, and *Trigonella foenum-graecum* [14].

In the Democratic Republic of the Congo (DRC), traditional medicine remains an integral component of healthcare practices. For example, extracts from the fruit of *Raphia gentiliana* De Wild, a bitter-tasting plant traditionally used for diabetes management, have been reported to exhibit hypoglycemic activity [15]. This traditional use has contributed to a widespread perception among Congolese populations, particularly in Kinshasa, that bitter-tasting foods confer health benefits for individuals with diabetes.

However, this assumption remains poorly supported by scientific evidence, highlighting the need for rigorous experimental evaluation of commonly consumed bitter fruits and medicinal plants. In this context, the present study investigates the toxicity and hypoglycemic potential of *Solanum aethiopicum*, a widely consumed fruit in the Democratic Republic of the Congo, using an experimental Wistar rat model.

2. Materials and Methods

2.1. Materials

Study Setting

This experimental study was conducted at the animal facility of the National Institute of Biomedical Research (INRB) and the Biochemistry and Hematology Laboratory, Faculty of Pharmaceutical Sciences, University of Kinshasa (UNIKIN), Kinshasa, Democratic Republic of Congo.

2.2. Biological Materials

2.2.1. Experimental Animals

A total of 25 adult Wistar rats (male and female), an outbred albino strain widely used in biomedical research, were used in this study.

Animals were randomly assigned and acclimatized under standard laboratory conditions with ad libitum access to a standard commercial diet and water. The sample consisted of 25 adult male and female Wistar rats divided into groups. These 25 rats were first used to evaluate hypoglycemic activity and were subsequently kept for two weeks or one month for toxicology testing and biochemical analysis. The rats were fed a cake made from standard complete livestock feed.

Their average weight was 204 g before extract administration and 195 g after extract administration.

Rats were individually identified using non-toxic body marking applied to different anatomical sites (head, tail, back, and flank). Body weight was measured to determine dosing, assess toxicity, perform oral glucose tolerance tests, and evaluate changes in body mass throughout the experiment.

Blood samples were collected from the tail vein for glycemic measurements and from cardiac puncture for biochemical analyses.

2.2.2. Plant Material



Fresh fruits of *Solanum aethiopicum*

Lyophilized *Solanum aethiopicum*

Fresh fruits of *Solanum aethiopicum* were purchased from Rond-Point Ngaba market in Kinshasa, *Democratic Republic of Congo*. Taxonomic identification was conducted at the Herbarium of the National Institute for Agronomic Study and Research (INERA), Faculty of Sciences, University of Kinshasa. Lyophilized whole-fruit extracts were subsequently prepared and used for all experimental procedures. The *Solanum aethiopicum* samples were prepared by cleaning the edible part of the fruit, cutting it into pieces, and placing them in an Erlenmeyer flask.

After adding distilled water, the mixture was brought to a boil, filtered, allowed to cool, and then transferred to trays.

Subsequently, the extract was frozen at -18°C and placed in a freeze-dryer for 48 hours at -55°C ; the freeze-dried product was stored in a dry place.

The freeze-drying yield was 1.89%, based on the weight of the fresh *Solanum aethiopicum* sample (P1) = 2300 g and the weight of the freeze-dried *Solanum aethiopicum* sample (P2) = 43.48 g.

3. Methods

This randomized experimental study conducted over a two-month period from September 12 to November 12, 2022.

3.1. Acute Toxicity Study (LD₅₀ Determination)

3.1.1. Principle

Acute oral toxicity was evaluated by administering graded doses of *S. aethiopicum*

extract via oral gavage. Animals were monitored for mortality and clinical signs of toxicity for 7 days following treatment. Dose selection was based on OECD acute oral toxicity guidelines and focused on the dose range corresponding to moderate toxicity (0.5 - 5 g/kg body weight).

3.1.2. Experimental Procedure

Lyophilized fruit extract was dissolved in distilled water to prepare a series of concentrations of 5000 mg/kg, 3750 mg/kg, 2500 mg/kg, 1250 mg/kg, and 650 mg/kg. Animals were divided into six groups:

Group I: 5000 mg/kg (n = 5)

Group II: 3750 mg/kg (n = 5)

Group III: 2500 mg/kg (n = 4)

Group IV: 1250 mg/kg (n = 4)

Group V: 650 mg/kg (n = 4)

Control group: distilled water (n = 3)

Following a 24 h fasting period, the extracts were administered orally via an orogastric gavage tube at doses normalized to body weight.

Animals were observed continuously at 30-minute intervals the first 8 hours, reassessed at 24 hours, and monitored daily thereafter for 7 days. Mortality and clinical signs of toxicity were systematically recorded throughout the study period. 2.1.3 LD₅₀ calculation.

The median lethal dose (LD₅₀) was calculated using the Kärber and Behrens-Tainter method and expressed as mg/Kg body weight according to the following equation:

$$DL_{50} = DL_{100} - \sum \frac{ab}{c}$$

where:

DL₁₀₀ = lowest dose causing 100% mortality

a = difference between consecutive doses

b = mean number of deaths between two doses

c = number of animals per group

Σ = sum of all intervals

3.2. Evaluation of Hypoglycemic Activity

3.2.1. Animal Grouping and Treatment

After acclimatization and a 24-hour fasting period, rats were randomly assigned to five groups (n = 5 per group):

Group 1: *S. aethiopicum* extract (500 mg/kg)

Group 2: extract (250 mg/kg)

Group 3: extract (125 mg/kg)

Group 4: glibenclamide (5 mg/kg; positive control)

Group 5: distilled water (negative control)

3.2.2. Test Substances

Glucose solution: 4 g/kg (oral load)

Test extract: *S. aethiopicum* (125 - 500 mg/kg)

Reference drug: glibenclamide (5 mg/kg)

Vehicle: distilled water

A fixed administration volume of 2 mL was used for a 200 g rat.

3.2.3. Oral Glucose Tolerance Test (OGTT)

The hypoglycemic activity was evaluated using an oral glucose-induced hyperglycemia model. Following a fast, baseline blood glucose levels were measured prior to oral glucose administration (4 g/kg body weight). Immediately after glucose loading, the test substances were administered according to the experimental group allocation. Blood glucose concentrations were subsequently determined at 30, 60, 120, and 180 minutes post-administration.

3.2.4. Blood Glucose Measurement

Blood glucose concentration were measured using a Sinocare® glucometer based on the glucose oxidase method coupled with a potassium ferricyanide-mediated redox reaction [16].

Blood samples were obtained from the tail vein, and glucose concentrations were expressed as mg/dL.

3.3. Biochemical Analysis

3.3.1. Blood Collection

Following a 24-hour fasting period, animals were anesthetized using ketamine hydrochloride at a dose adjusted to body weight. Blood samples were collected via cardiac puncture and centrifuged at 1000 rpm for 10 minutes. The resulting serum was separated and stored until biochemical analysis.

3.3.2. Method for the Analysis of Biochemical Parameters

Serum aspartate aminotransferase (AST/TGO), alanine aminotransferase (ALT/TGP), urea, and creatinine concentrations were determined using standard spectrophotometric assays. AST and ALT: kinetic IFCC method (340 nm)

Urea: enzymatic Berthelot reaction (546 nm)

Creatinine: Jaffé reaction (490 nm) [17].

3.4. Statistical Analysis

Statistical analyses were conducted using SPSS version 21 (IBM Corp., Armonk, NY, USA). Results are reported as mean $\pm$ SD. Group differences were evaluated using one-way ANOVA with Bonferroni post hoc correction, while paired Student's *t*-tests were used to assess changes in body weight. A significance level of $p < 0.05$ was adopted throughout.

4. Results and Discussion

4.1. Results

4.1.1. Toxicity Results

A total of 25 Wistar rats were included in this study. The sex distribution (**Table**

1), showed a predominance of females (17/25, 68%) relative to males (8/25, 32%).

Table 1. Distribution of Wistar rats by sex.

SEX	FREQUENCY	PERCENTAGE
Female	17	68
Male	8	32
TOTAL	25	100

A total of 25 Wistar rats were included in this study. The sex distribution (**Table 1**), showed a predominance of females (17/25, 68%) relative to males (8/25, 32%).

Table 2. Determination of the lethal dose (LD₅₀) of **Solanum aethiopicum** extracts.

DOSES ADMINISTERED (mg/Kg)	DEATH	SYMPTOMATIC DISORDERS
5000	0	None
3750	0	None
2500	0	None
1250	0	None
650	0	None

As shown in **Table 2**, no mortality or treatment-related clinical signs of toxicity were observed in rats administered *Solanum aethiopicum* extract at doses ranging from 650 to 5000 mg/kg. The absence of mortality across all treatment groups (0/25) indicates that the medianLD₅₀ exceeds 5000 mg/kg, suggesting that the extract exhibits very low acute toxicity.

Table 3. Comparison of mean weight before and after dose administration.

Descriptive parameters (n = 25)						
				Mean	Standard deviation	
Paired sample		Front weight (g)		204.00	30.00	
		Weight after (g)		195.00	29.64	
t-Test						
Pair of differences				T	Df	p < 0.05
Average	Standard deviation	IC95%				
		Li	Ls			
9000	10.626	4614	13.386	4235	24	0000

No statistically significant differences in body weight were observed before and after treatment among the experimental groups (**Table 3**).

4.1.2. Glycemia Results

Table 4. Description of blood glucose levels over the observation period.

Time in minutes	N	Mean	Standard deviation	95% confidence interval for the mean		Fisher	P < 0.05
				Lower bound	Upper bound		
0	25	59.12	12.882	53.80	64.44	41.765	0.00
30	25	127.32	27.049	116.15	138.49		
60	25	116.24	25.948	105.53	126.95		
120	25	74.88	27.990	63.33	86.43		
180	25	67.76	21.316	58.96	76.56		

Blood glucose levels varied significantly over time following glucose administration ($F = 41.765$, $p < 0.05$), as detailed in **Table 4**. Mean glycemia increased sharply from baseline (59.12 ± 12.88 mg/dL) to 30 minutes (127.32 ± 27.05 mg/dL), followed by a progressive decline at 60 minutes (116.24 ± 25.95 mg/dL), 120 minutes (74.88 ± 27.99 mg/dL), and 180 minutes (67.76 ± 21.32 mg/dL).

Table 5. Comparison of the difference in means between the times, pairwise.

TIMES (i)	TIMES (j)	Difference of means	p < 0.05	Signification	95% confidence interval	
					Bi	Bs
0	30	-68.200*	0.000	***	-87.37	-49.03
	60	-57.120*	0.000	***	-76.29	-37.95
	120	-15.760	0.204	NS	-34.93	3.41
	180	-8.640	1.000	NS	-27.81	10.53
30	0	68.200*	0.000	***	49.03	87.37
	60	11.080	1.000	NS	-8.09	30.25
	120	52.440*	0.000	***	33.27	71.61
	180	59.560*	0.000	***	40.39	78.73
60	0	57.120*	0.000	***	37.95	76.29
	30	-11.080	1.000	NS	-30.25	8.09
	120	41.360*	0.000	***	22.19	60.53
	180	48.480*	0.000	***	29.31	67.65
120	0	15.760	0.204	NS	-3.41	34.93
	30	-52.440*	0.000	***	-71.61	-33.27
	60	-41.360*	0.000	***	-60.53	-22.19
	180	7.120	1.000	NS	-12.05	26.29
180	0	8.640	1.000	NS	-10.53	27.81
	30	-59.560*	0.000	***	-78.73	-40.39
	60	-48.480*	0.000	***	-67.65	-29.31
	120	-7.120	1.000	NS	-26.29	12.05

1) Bonferroni test; the difference is significant at the 5% level; 2) ***: significant; 3) NS: not significant; 4) Bi: Lower bound; 5) Bs: Upper bound.

Pairwise comparisons using the Bonferroni test (**Table 5**) revealed significant differences primarily between the early (0 - 60 min) and late (120 - 180 min) time points, indicating a pronounced time-dependent glycemic response to oral glucose loading.

Table 6. Description of blood glucose levels in relation to *Solanum* extracts, with Glibenclamide as a reference sample and distilled water as a control.

Samples	N	Mean	Standard deviation	95% confidence interval for the mean		Fisher	P < 0.05
				Lower bound	Upper bound		
Distilled water	25	92.76	34.049	78.71	106.81		
Glibenclamide	25	65.56	31.027	52.75	78.37		
E125	25	92.04	34.278	77.89	106.19	3.754	0.006
E250	25	98.08	33.910	84.08	112.08		
E500	25	96.88	39.047	80.76	113.00		

Significant differences were also observed among treatment groups ($F = 3.754$, $p = 0.006$), as shown in **Table 6**. Treatment with glibenclamide resulted in a marked reduction in blood glucose levels compared with distilled water and all extract-treated groups.

Table 7. Comparison of the difference between means across pairwise samples.

Samples	Solution (J)	Difference of means	p < 0.05	Signification	95% confidence interval	
					Bi	Bs
Distilled water	Glibenclamide	27.200	0.003	**	-0.75	55.15
	E125	0.720	1.000	NS	-27.23	28.67
	E250	-5.320	1.000	NS	-33.27	22.63
	E500	-4.120	1.000	NS	-32.07	23.83
Glibenclamide	Distilled water	-27.200	0.003	**	-55.15	0.75
	E125	-26.480	0.007	**	-54.43	1.47
	E250	-32.520	0.021	**	-60.47	-4.57
	E500	-31.320	0.034	***	-59.27	-3.37
E125	Distilled water	-0.720	1.000	NS	-28.67	27.23
	Glibenclamide	26.480	0.007	**	-1.47	54.43
	E250	-6.040	1.000	NS	-33.99	21.91
	E500	-4.840	1.000	NS	-32.79	23.11
E250	Distilled water	5.320	1.000	NS	-22.63	33.27
	Glibenclamide	32.520	0.010	***	4.57	60.47
	E125	6.040	1.000	NS	-21.91	33.99
	E500	1.200	1.000	NS	-26.75	29.15
E500	Distilled water	4.120	1.000	NS	-23.83	32.07
	Glibenclamide	31.320	0.034	***	3.37	59.27
	E125	4.840	1.000	NS	-23.11	32.79
	E250	-1.200	1.000	NS	-29.15	26.75

1) Bonferroni test; the difference is significant at the 5% level; 2) ***: significant; 3) NS: not significant; 4) Bi: Lower bound; 5) Bs: Upper bound.

In contrast, administration of *Solanum aethiopicum* extract at doses of 125, 250, and 500 mg/kg did not significantly reduce blood glucose levels relative to the control group, as confirmed by Bonferroni pairwise comparisons (**Table 7**).

4.1.3. Determination of Certain Biochemical Parameters

Serum biochemical parameters are summarized in **Table 8**. Urea, creatinine, AST, and ALT levels in treated animals remained within, or were close to, established physiological reference ranges.

Table 8. Comparison of biochemical parameters against reference ranges.

BIOCHEMICAL PARAMETERS	VALUES FOUND	PHYSIOLOGICAL VALUES (Janvier Laboratories, 2012)	
		Male	Female
UREA (mg/dl)	31.28 ± 8.58	52.00 ± 7.00	55.00 ± 1.00
CREATININE (mg/dl)	0.93 ± 0.13	4.91 ± 0.37	4.76 ± 0.46
TGO (U.I/l)	73.23 ± 7.95	93.00 ± 20.00	89.00 ± 17.00
TGP (U.I/l)	25.72 ± 3.54	32.00 ± 5.00	30.00 ± 8.00

Serum biochemical parameters are summarized in **Table 8**. Urea, creatinine, AST, and ALT levels in treated animals remained within, or were close to, established physiological reference ranges.

Table 9. Comparison of biochemical parameters.

ANOVA						
		Sum of squared deviations	Df	Mean squared deviation	F	p < 0.05
UREA	Between variance group	543.023	5	108.605	1.683	0.187
	Résidual variance	1226.017	19	64.527		
CREA	Between variance group	0.161	5	0.032	2.463	0.070
	Résidual variance	0.249	19	0.013		
TGO	Between variance group	9537.248	5	1907.450	0.794	0.567
	Résidual variance	45651.542	19	2402.713		
TGP	Between variance group	6767.291	5	1353.458	0.836	0.540
	Résidual variance	30752.269	19	1618.540		

Overall comparisons among experimental groups revealed no significant differences in urea, creatinine, AST, or ALT concentrations ($p > 0.05$), as shown on **Table 9**, indicating that administration of the extract did not induce detectable hepatic or renal toxicity under the experimental conditions.

Table 10. Comparaison des paramètres biochimiques par rapport au sexe.

ANOVA						
PARAMETERS		Sum of squared deviations	Df	Mean squared deviation	F	p < 0.05
UREA	Between variance group	3.930	1	3.930	0.051	0.823
	Résidual variance	1765.110	23	76.744		
CREA	Between variance group	6472.744	1	6472.744	4.795	0.039
	Résidual variance	31046.816	23	1349.862		
TGO	Between variance group	5990.152	1	5990.152	2.800	0.108
	Résidual variance	49198.638	23	2139.071		
TGP	Between variance group	0.000	1	0.000	0.001	0.981
	Résidual variance	0.410	23	0.018		

Sex-based analysis showed no significant differences in urea, AST, or ALT levels (Table 10). In contrast, creatine concentrations differed significantly between male and female rats ($p = 0.039$), although all values remained within physiological limits.

5. Discussion

5.1. Toxicity of *Solanum aethiopicum*

In the present study, oral administration of *Solanum aethiopicum* extract at doses of 650 - 5000 mg/kg produced neither mortality nor observable clinical signs of toxicity in 22 Wistar rats. Accordingly, the median lethal dose (LD_{50}) was estimated to be greater than 5000 mg/kg, indicating that the extract possesses low acute oral toxicity under the conditions of this study.

5.2. Hypoglycemic Activity of *Solanum aethiopicum*

Following oral glucose administration, blood glucose concentrations in Wistar rats increased markedly, reaching a peak at 30 minutes with a mean value of 127.32 ± 27.05 mg/dL (95% CI: 116.15 - 138.49 mg/dL). Thereafter, glucose levels decreased progressively to 116.24 ± 25.95 mg/dL at 60 minutes and 67.76 ± 21.31 mg/dL at 180 minutes (Table 4).

The overall mean blood glucose concentration during the experimental period was 89.1 ± 36.1 mg/dL (95% CI: 82.68 - 95.45 mg/dL), indicating a clear time-dependent regulation of glucose homeostasis following oral glucose loading. These findings are broadly consistent with previous reports on plant-derived hypoglycemic agents. For example, Masunda *et al.* demonstrated that extracts of *Raphia gentiliana* significantly reduced blood glucose levels approximately two hours after administration. In the present study, however, the decline in blood glucose was evident as early as 60 minutes, suggesting a more glycemic response. This observation is in agreement with physiological evidence showing that post-

prandial blood glucose typically peaks within 30 - 60 minutes and gradually returns toward baseline within 60 - 120 minutes after carbohydrate ingestion [18].

Among the treatment groups, the lowest mean blood glucose concentration was recorded in the glibenclamide-treated group (65.56 ± 31.03 mg/dL), followed by the group receiving 125 mg/kg of *S. aethiopicum* extract (92.04 ± 34.28 mg/dL) (Table 6).

Statistical analyses revealed a significant effect of time on blood glucose concentrations ($p < 0.05$), indicating substantial temporal variation throughout the 180-minute observation period (Table 5).

Furthermore, significant differences were also observed among treatment groups ($p < 0.05$), as shown in Table 7. Nevertheless, the hypoglycemic effect of *S. aethiopicum* extract was less pronounced than that of glibenclamide, suggesting that the extract does not exhibit glucose-lowering activity comparable to the reference drug under experimental conditions employed.

This finding contrasts with the study of Masunda *et al.* (2014), who reported hypoglycemic activity of *Raphia gentiliana* comparable to glibenclamide. Likewise, while Achyut *et al.* [19] observed significant reductions in blood glucose in animals treated with *Aegle marmelos* extracts relative to controls, no significant differences were detected between the distilled water group and the *S. aethiopicum* extract-treated groups in the present study. These results suggest that *S. aethiopicum* extract exhibits limited or no direct hypoglycemic activity at the tested doses. In addition, a significant reduction in body weight was observed during the acute toxicity study ($p < 0.05$). This decrease is likely attributable to the 24-hour fasting period imposed prior to LD₅₀ determination rather than to a toxic effect of the extract itself.

5.3. Biochemical Parameters

The analysis of biochemical parameters, including urea, creatinine, AST (TGO), and ALT (TGP), showed no statistically significant differences among the treated groups ($p > 0.05$), as shown in Tables 8-10. These findings indicate that *S. aethiopicum* extract did not induce detectable hepatic or renal toxicity in Wistar rats under the experimental conditions employed.

The present results differ from those reported by Srikanth and Muralidharan [20], who observed significant dose-dependent alterations in biochemical markers following administration of *Sapindus emarginatus* extracts. Similarly, Achyut *et al.* [19] (2006) reported changes in biochemical parameters varied according to both dose and duration of treatment with *Aegle marmelos*. These discrepancies may reflect differences in plant species, phytochemical composition, extraction procedures, administered doses, and experimental designs.

Conversely, several studies have highlighted beneficial metabolic effects of medicinal plants. For example, Beddazekri and Ghemam [21] demonstrated that *Ajuga iva* and *Matricaria pubescens* significantly reduced blood glucose levels and improved body weight following repeated administration. Likewise, Djedioui and

Messareh [22] reported that *Inula viscosa* extract reduced blood glucose levels, improved hepatic function, and enhanced antioxidant status without causing adverse effects in healthy rats.

More recently, [23] showed that consumption of *S. aethiopicum* preserved pancreatic histoarchitecture, improved glycemic control, and reduced body weight in diabetic rats. Although the present study similarly observed a reduction in body weight in Wistar rats, no direct hypoglycemic effect was detected. This discrepancy may be attributed to differences in experimental conditions, particularly the form of plant material used and extraction procedures, as previous studies frequently employed fresh or differently processed fruits, whereas the present study utilized lyophilized extracts.

6. Conclusions

This study evaluated the acute toxicity and hypoglycemic potential of *Solanum aethiopicum* fruit extracts in Wistar rats using an in vivo experimental model.

The experimental design comprised acute toxicity testing for LD₅₀ determination, oral glucose tolerance testing to assess hypoglycemic activity, and biochemical analysis of serum urea, creatinine, AST, and ALT using standard laboratory methods.

The results showed that the LD₅₀ of *Solanum aethiopicum* fruit extract exceeded 5000 mg/kg body weight, indicating very low acute toxicity under the experimental conditions. However, the extract did not elicit a significant hypoglycemic effect in Wistar rats compared with the control groups. Furthermore, serum biochemical parameters remained within physiological ranges across all treatment groups, with no statistically significant differences observed, suggesting the absence of detectable hepatic or renal toxicity.

Overall, these findings indicate that *Solanum aethiopicum* fruit extract is well tolerated at the tested doses but does not exhibit substantial direct hypoglycemic activity in this experimental model.

Further investigations are needed to elucidate its antioxidant properties and underlying mechanisms of action, as well as evaluate its potential antidiabetic effects using alternative extraction methods and more clinically relevant experimental and human models.

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Generative Artificial Intelligence (AI) Disclosure Statement

During manuscript preparation, the authors used ChatGPT (OpenAI, free GPT) version solely as a language editing tool for limited rephrasing and improvement of the clarity of selected sentences. No AI tool was used to generate research content, perform data analysis, interpret results, or prepare references. The ideas, arguments, and intellectual content of the manuscript are entirely original to the authors. All AI-assisted edits were carefully reviewed, revised where necessary, and approved by the authors, who assume full responsibility for the accuracy and integrity of the final manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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