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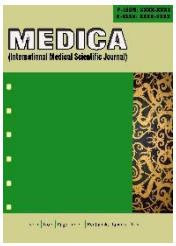

Potential of *Borassus Flabellifer* Linn Flower Extract as Antidiabetic: An Experimental Study in Alloxan-Induced Hyperglycemic Mice

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Abstract

Hyperglycemia resulting from pancreatic β-cell damage remains a major challenge in diabetes mellitus management, highlighting the need for effective bioactive agents to reduce blood glucose levels. This study aimed to assess the antihyperglycemic activity of *Borassus flabellifer* Linn extract in alloxan-induced mice and to compare the efficacy across different doses. A post-test only control group design was applied with five groups: three treatment groups receiving *B. flabellifer* extract (200, 400, and 600 mg/kgBW), a positive control, and a negative control (n=5 per group). Blood glucose levels were monitored for seven days after induction and analyzed using one-way ANOVA followed by Tukey HSD post hoc test. All three doses significantly reduced blood glucose levels compared to the negative control ($p<0.05$). The 600 mg/kgBW dose produced the largest absolute glucose reduction (261.6 mg/dL), while the 200 mg/kgBW dose achieved the best glucose normalization, reaching a final level of 109.4 mg/dL by day 7. These findings highlight a distinction between absolute reduction magnitude and degree of normalization that warrants careful interpretation. Due to the limited sample size (n=5 per group) and short observation period, these results should be considered preliminary. In conclusion, *B. flabellifer* Linn flower extract demonstrates antihyperglycemic potential across all tested doses. The 200 mg/kgBW dose showed the best glucose normalization, while the 600 mg/kgBW dose produced the largest absolute reduction. These preliminary results support its potential as a natural candidate for antidiabetic therapy and warrant further investigation into its mechanisms, phytochemical profile, and long-term safety.

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1. INTRODUCTION

Diabetes mellitus is a chronic metabolic disease characterized by hyperglycemia due to impaired insulin secretion or action (Anitha et al., 2023). The prevalence of diabetes continues to increase globally each year and has become one of the leading causes of morbidity and mortality (Peter et al., 2023). Prolonged hyperglycemia can trigger microvascular and macrovascular complications, thereby reducing the quality of life of patients (Tomic et al., 2022). Therefore, the search for safer, more effective, and more affordable alternative therapies has become an important priority in the development of health science (Abel et al., 2024).

Currently, pharmacological therapies such as metformin, sulfonylureas, and insulin are the standard treatment for diabetes (Farween et al., 2025). However, the use of these drugs is often accompanied by side effects, high costs, and limited access in certain areas (Dubey et al., 2025). These conditions have prompted the development of complementary therapy strategies based on natural resources, particularly medicinal plants that have long been used in traditional medicine (Narayanankutty et al., 2023). Phytotherapy is considered to have the potential to provide antidiabetic effects with lower toxicity (Abel et al., 2024).

One plant that attracts attention is *Borassus flabellifer* Linn, also known as siwalan or lontar, which grows widely in tropical regions including Indonesia (Rahman et al., 2021). Various parts of this plant have long been used by communities as food, beverages, and traditional medicine (Alam et al., 2022). Extracts from parts of the plant such as the roots, leaves, and flowers are known to contain bioactive compounds that have important biological activities, including antioxidant, anti-inflammatory, and antihyperglycemic properties (Rahman et al., 2021).

Bunga *B. flabellifer* Linn contains a number of secondary metabolites such as flavonoids, phenolics, tannins, and saponins, which are known to have therapeutic effects on improving glucose metabolism (Mohaideen & Srinivasan, 2024). These compounds have strong antioxidant capacity, which can neutralize free radicals and reduce oxidative stress, a mechanism that plays a major role in β-cell damage in the pancreas under hyperglycemic conditions (Tunit et al., 2022). Therefore, the flower part of this plant has great potential as a natural antidiabetic candidate.

Pancreatic β-cell damage can be modeled using diabetogenic agents such as alloxan (Mishra et al., 2023). Alloxan causes selective necrosis of β-cells through the formation of free radicals and the induction of oxidative stress (Rahman et al., 2021; Roy et al., 2024). The alloxan-induced mouse model is a commonly used choice in pharmacological research because it can clearly describe the pathological conditions of both type 1 and type 2 diabetes (Malayil et al., 2022). The use of this model can provide a comprehensive picture of the effectiveness of an extract in improving blood glucose profiles (Prasad et al., 2023).

The results of a study conducted by Natarajan and Elizabeth (2025) show that flavonoid-rich plant extracts can improve blood sugar levels, increase insulin sensitivity, and protect pancreatic β cells from oxidative damage (Natarajan & Elizabeth, 2025). Although other parts of the B. flabellifer Linn plant have been evaluated for their biological activity, research on the plant's flower extract as an antidiabetic agent is still limited. Therefore, more systematic scientific studies are needed to evaluate its potential.

The antidiabetic activity of an extract is generally determined by testing various doses to determine its biological effectiveness range (Banu et al., 2021). Dose evaluation is important because it can identify the relationship between increased extract concentration and the response of decreased blood glucose levels (Tusskorn et al., 2021). This approach provides a scientific basis for determining the optimal safe and effective dose to be developed in the advanced preclinical testing stage.

Experimental analysis of antidiabetic activity is usually conducted by measuring blood glucose levels before and after treatment, as well as monitoring changes in glucose levels over several days (Job et al., 2022). Repeated monitoring can reveal consistent patterns of effectiveness and provide strong evidence of the therapeutic potential of the extract. In addition, the presence of positive and negative control groups allows for objective comparison of the effectiveness of a test compound.

A study on the potential of *B. flabellifer* Linn flower extract in lowering blood glucose in alloxan-induced mice is expected to contribute significantly to the development of Indonesian herbal medicine raw materials. With strict methodological standards, this study can serve as a scientific basis for further exploration, including the isolation of active compounds and toxicity testing. This information is important to support the use of local plants as an alternative therapy for diabetes.

Based on the above background, this study aims to evaluate the antidiabetic activity of *B. flabellifer* Linn flower extract using an alloxan-induced mouse model. The results of this study are expected to fill the knowledge gap regarding the therapeutic potential of this plant flower, as well as provide an initial understanding of its effectiveness in lowering blood glucose levels at various doses. The findings of this study are an important contribution to the development of antidiabetic phytopharmaceuticals based on local biological resources.

2. METHOD

This study employed an in vivo laboratory experimental design using a post-test only control group design to evaluate the antihyperglycemic activity of *Borassus flabellifer* Linn flower extract in alloxan-induced diabetic mice. The experiment was conducted in the Pharmacology Laboratory of the University of Bima International MFH over a seven-day observation period. The study aimed to determine the effect of different doses of *B. flabellifer* Linn flower extract on blood glucose levels in experimental animals.

The experimental animals used were male Swiss Webster mice aged 8–10 weeks with body weights ranging from 25 to 30 g. All animals underwent a seven-day acclimatization period under controlled environmental conditions, including a temperature of 22–25°C, relative humidity of 50–60%, and a 12-hour light–dark cycle. During acclimatization, the animals received a standard laboratory diet and water ad libitum. Mice showing signs of illness, inactivity, or weight loss exceeding 10% during the acclimatization period were excluded from the study.

The flowers of *B. flabellifer* Linn were collected from healthy plants, air-dried at room temperature, and ground into a fine powder. Extraction was performed using the maceration method with 70% ethanol as the solvent. The resulting filtrate was concentrated using a rotary evaporator to obtain a thick extract, which was subsequently stored at 4°C until use.

Hyperglycemia was induced by intraperitoneal administration of alloxan monohydrate at a dose of 150 mg/kg body weight. Seventy-two hours after induction, blood glucose levels were measured using a digital glucometer from tail vein blood samples. Mice with fasting blood glucose levels of ≥200 mg/dL were considered hyperglycemic and included in the treatment phase of the experiment.

Twenty-five mice were randomly allocated into five experimental groups using a random number table, with five animals in each group. The negative control group received distilled water, whereas the positive control group received glibenclamide at a dose of 0.65 mg/kg body weight. Three treatment groups received *B. flabellifer* Linn flower extract at doses of 200, 400, and 600 mg/kg body weight, respectively. All treatments were administered orally using a gastric tube once daily for seven consecutive days. The sample size was determined based on common practice in preliminary pharmacological

studies; however, the relatively small number of animals may limit statistical power and should be considered when interpreting the findings. In addition, investigators were not blinded to treatment allocation, which may represent a potential source of bias.

Blood glucose levels were measured before alloxan induction, after induction (day 0), and daily from day 1 to day 7 of treatment. Blood samples were collected from the tail vein following an 8-hour fasting period. Blood glucose concentrations were expressed in mg/dL and used as the primary outcome measure to assess the antihyperglycemic effect of the extract.

Statistical analysis was performed using IBM SPSS Statistics version 26. Descriptive statistics were used to summarize blood glucose values in each experimental group. Differences among groups were analyzed using one-way analysis of variance (ANOVA), followed by Tukey's honestly significant difference (HSD) test for post hoc multiple comparisons. Statistical significance was established at a p-value of <0.05.

All experimental procedures were approved by the Research Ethics and Academic Integrity Committee (RE-AIC), University of Bima International MFH, under ethical approval number 037/KEPH/2025. All procedures involving animals were conducted in accordance with institutional ethical guidelines for the care and use of laboratory animals.

3. RESULTS AND DISCUSSION

The study aimed to evaluate the effectiveness of several treatment doses (200 mg/kgBW, 400 mg/kgBW, and 600 mg/kgBW) in lowering blood sugar levels in mice induced by alloxan. In general, all treatments showed a decrease in blood sugar levels within 7 days, but the magnitude differed between groups. The positive control group (generally given standard antidiabetic drugs) experienced a decrease in blood sugar levels from 342 mg/dL to 120 mg/dL on day 7. The total average decrease was 201 mg/dL, indicating the high effectiveness of the standard therapy used. The pattern of decrease occurred gradually and consistently from the first day of treatment (Figure 1). It should be noted that baseline blood glucose levels before alloxan induction differed significantly between groups ($p=0.003$), indicating that pre-existing inter-group variability may have influenced the magnitude of observed reductions and should be considered when interpreting results.

The negative control group (without treatment) showed only a small decrease from 179.2 mg/dL to 113.8 mg/dL, with a total decrease of 4.8 mg/dL. This decrease was minimal and likely caused only by the spontaneous physiological mechanisms of mice. This shows that without intervention, blood sugar levels remain high with insignificant changes. The group given a dose of 200 mg/kgBW showed a drastic decrease from 211.6 mg/dL to 109.4 mg/dL on day 7, with a total decrease of 228 mg/dL. This decrease was even greater than that of the positive control, indicating that a dose of 200 mg/kgBW has very strong antidiabetic potential. The pattern of decrease remained stable every day (Figure 1).

The 400 mg/kgBW dose group experienced a decrease in blood sugar levels of 173 mg/dL, from 247.8 mg/dL to 127.4 mg/dL. The decrease occurred gradually, but was not as significant as in the 200 mg/kgBW dose group or the positive control group. The effectiveness of this dose is in the moderate category. The 600 mg/kgBW dose group had the highest absolute decrease in blood sugar levels, namely 261.6 mg/dL, from 357.8 mg/dL to 207.6 mg/dL. However, despite the large total decrease, the final blood sugar level (207.6 mg/dL) was still higher than the other groups. This indicates that despite the large decrease, the blood sugar normalization effect at the 600 mg/kgBW dose was less than optimal. Possible toxicity effects or excessive biological responses caused the decrease to be less effective in the final phase (Figure 1).

All treatment groups showed a decrease in blood sugar levels after alloxan induction. The 200 mg/kgBW dose appeared to be the most effective dose because it produced a large decrease (228 mg/dL) and achieved a relatively low final level (109.4 mg/dL). The positive control still provided a strong decrease (201 mg/dL) as a standard comparison. The 600 mg/kgBW dose had the largest total reduction, but did not achieve normalization of blood glucose levels, making its effectiveness in restoring blood glucose to the normal range less than optimal. The negative control showed minimal changes, confirming the success of alloxan induction and the effectiveness of the treatment administered (Figure 1). These findings highlight an important distinction: while the 600 mg/kgBW dose produced the greatest absolute reduction in blood glucose, the 200 mg/kgBW dose achieved superior normalization of blood glucose to near-normal levels. The two metrics — absolute reduction and final normalization — should therefore be reported and interpreted separately.

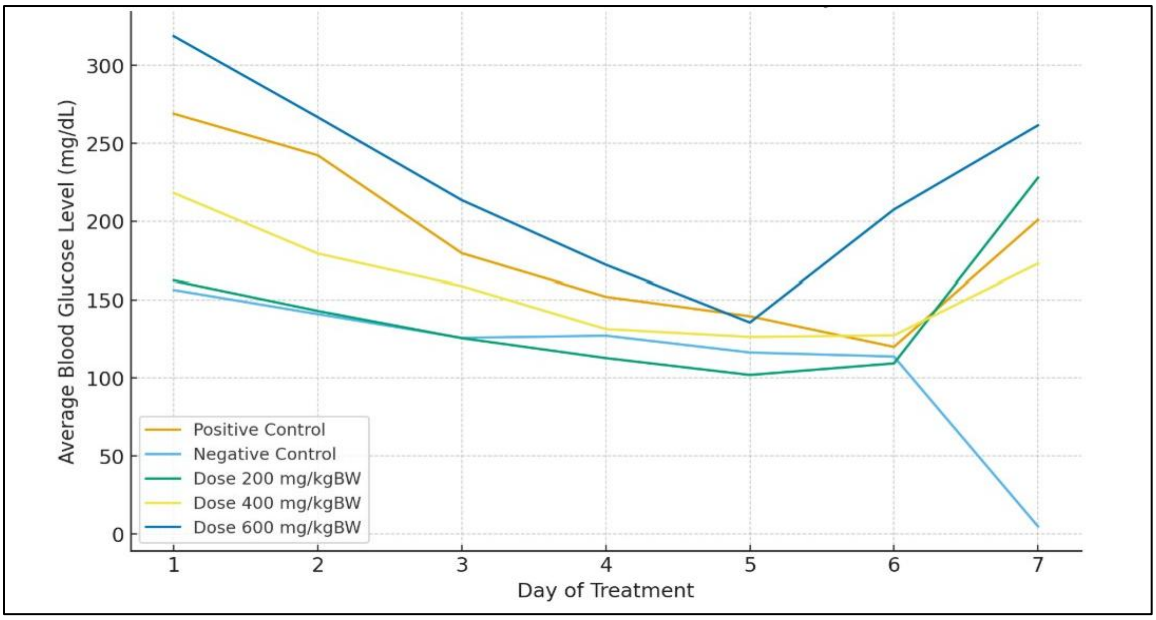

Figure 1. Average blood sugar level reduction measurements in mice

The results of the study show that there is a significant difference between the groups of mice before aloxan administration (p=0.003). This indicates that the baseline blood sugar levels between groups are not completely homogeneous. However, this difference can still be controlled because aloxan induction aims to cause relatively uniform hyperglycemia. In addition, the results of the study show very significant differences between groups after induction (p<0.001). This is in line with expectations because alloxan selectively damages pancreatic beta cells, thereby dramatically increasing blood sugar levels. This difference indicates the success of hyperglycemia induction before treatment (Table 1).

The results of the test on day 1 showed a significant difference between treatment groups on the first day (p=0.009). This means that various treatment doses began to show initial effects in lowering blood sugar levels compared to the negative control. On day 2, a p-value of 0.001 was obtained, indicating a highly significant difference. This shows that the treatment effect was more pronounced than on the previous day. The doses administered began to show a stronger difference in effectiveness (Table 1).

The third result shows a significant difference between groups (p=0.007). This change indicates that the blood sugar reduction response continues and the difference in

performance between doses and controls is becoming more consistent. On day 4, the differences between groups remained significant (p=0.018). This indicates that on day 4, all treatments had different effects on the dynamics of blood sugar reduction, and the effects of the intervention were still ongoing. On day 5, there were no significant differences between groups (p=0.170). This means that on this day, the decrease in blood sugar levels between groups tended to be uniform. This could be due to the metabolic adaptation of mice, the speed of blood sugar stabilization, or variations in response between individuals (Table 1).

On day 6, there were no significant differences (p=0.369). At this point, the decrease in blood sugar levels between groups was approaching a stable pattern, so that the difference in dosage did not produce a large enough variation in effect to be statistically significant. A significant difference was found on day 7 (p=0.015). This indicates that toward the end of the observation period, the treatment effects of each dose again showed noticeable differences. Some doses showed greater effectiveness in maintaining or continuing the decrease in blood sugar levels (Table 1).

Significant differences between groups were observed on almost all days of observation, except on days 5 and 6. The effects of treatment began to appear on the first day and became stronger on days 2 to 4. Physiological stabilization likely caused the differences to become insignificant on days 5 and 6. On the 7th day, the differences became significant again, indicating that the effects of specific doses were more consistent or stronger at the end of the period. ANOVA data confirmed that different doses had different effects in lowering blood sugar levels (Table 1).

Table 1. One-Way ANOVA Test Results for Mouse Groups Based on Treatment Days

Days		Sum of Squares	Mean Square	F	*p*
1	Between Groups	125069.840	31267.460	4.494	0.009*
	Within Groups	139165.600	6958.280		
	Total	264235.440			
2	Between Groups	97152.240	24288.060	6.669	0.001*
	Within Groups	72841.600	3642.080		
	Total	169993.840			
3	Between Groups	66321.040	16580.260	4.735	0.007*
	Within Groups	70031.200	3501.560		
	Total	136352.240			
4	Between Groups	28046.240	7011.560	3.854	0.018*
	Within Groups	36387.200	1819.360		
	Total	64433.440			
5	Between Groups	10773.040	2693.260	1.790	0.170
	Within Groups	30087.600	1504.380		
	Total	40860.640			
6	Between Groups	4627.200	1156.800	1.135	0.369
	Within Groups	20386.800	1019.340		
	Total	25014.000			
7	Between Groups	33281.360	8320.340	3.988	0.015*
	Within Groups	41724.400	2086.220		
	Total	75005.760			
Before alloxan induction	Between Groups	14506.640	3626.660	5.959	0.003*
	Within Groups	12172.800	608.640		
	Total	26679.440			
	Between Groups	344875.760	86218.940	18.166	<0.001*

After alloxan induction	Within Groups	94921.200	4746.060
	Total	439796.960	

**p<0.05*

Further analysis using Tukey's test was performed to determine specific differences between groups after the Oneway ANOVA test showed significant differences on most observation days. In general, the Tukey test results revealed that treatments with various doses produced different antihyperglycemic responses, with a consistent pattern showing that the 600 mg/kgBW dose was most effective in lowering blood glucose levels in mice (Table 2).

On the first to fourth days of observation, the 600 mg/kgBW dose group showed a significant difference compared to the negative control group ($p<0.05$), indicating a blood sugar-lowering effect that began to appear early in the treatment period. The 200 mg/kgBW dose also showed some significant differences when compared to the negative control, although these were not consistent on every day of observation. Meanwhile, the 400 mg/kgBW dose showed a moderate response that fell between the low and high doses. These findings indicate a relationship between increased dosage and increased biological effects, although this relationship is not always linear (Table 2).

Table 2. Results of the Tukey Test on the Mouse Group

Days	Mice Group	Mice Group	MD	SE	*p*	95% CI
1	600 mg/kgBW	400 mg/kgBW	110.000	52.757	0.265	-47.87-267.87
		200 mg/kgBW	146.200	52.757	0.078	-11.67-304.07
		Positive Control	15.800	52.757	0.998	-142.07-173.67
		Negative Control	178.600*	52.757	0.022	20.73-336.47
	400 mg/kgBW	600 mg/kgBW	-110.000	52.757	0.265	-267.87-47.87
		200 mg/kgBW	36.200	52.757	0.957	-121.67-194.07
		Positive Control	-94.200	52.757	0.409	-252.07-63.67
		Negative Control	68.600	52.757	0.694	-89.27-226.47
	200 mg/kgBW	600 mg/kgBW	-146.200	52.757	0.078	-304.07-11.67
		400 mg/kgBW B	-36.200	52.757	0.957	-194.07-121.67
		Positive Control	-130.400	52.757	0.137	-288.27-27.47
		Negative Control	32.400	52.757	0.971	-125.47-190.27
	Positive Control	600 mg/kgBW	-15.800	52.757	0.998	-173.67-142.07
		400 mg/kgBW	94.200	52.757	0.409	-63.67-252.07
		200 mg/kgBW	130.400	52.757	0.137	-27.47-288.27
		Negative Control	162.800*	52.757	0.041	4.93-320.67
	Negative Control	600 mg/kgBW	-178.600*	52.757	0.022	-336.47--20.73
		400 mg/kgBW	-68.600	52.757	0.694	-226.47-89.27
		200 mg/kgBW	-32.400	52.757	0.971	-190.27-125.47
		Positive Control	-162.800*	52.757	0.041	-320.67--4.93
2	600 mg/kgBW	400 mg/kgBW	100.600	38.168	0.101	-13.61-214.81
		200 mg/kgBW	156.600*	38.168	0.004	42.39-270.81
		Positive Control	49.800	38.168	0.691	-64.41-164.01
		Negative Control	162.400*	38.168	0.003	48.19-276.61
	400 mg/kgBW	600 mg/kgBW	-100.600	38.168	0.101	-214.81-13.61
		200 mg/kgBW	56.000	38.168	0.594	-58.21-170.21
		Positive Control	-50.800	38.168	0.676	-165.01-63.41
		Negative Control	61.800	38.168	0.503	-52.41-176.01
	200 mg/kgBW	600 mg/kgBW	-156.600*	38.168	0.004	-270.81--42.39

Days	Mice Group	Mice Group	MD	SE	*p*	95% CI
		400 mg/kgBW	-56.000	38.168	0.594	-170.21-58.21
		Positive Control	-106.800	38.168	0.074	-221.01-7.41
		Negative Control	5.800	38.168	1.000	-108.41-120.01
	Positive Control	600 mg/kgBW	-49.800	38.168	0.691	-164.01-64.41
		400 mg/kgBW	50.800	38.168	0.676	-63.41-165.01
		200 mg/kgBW	106.800	38.168	0.074	-7.41-221.01
		Negative Control	112.600	38.168	0.055	-1.61-226.81
	Negative Control	600 mg/kgBW	-162.400*	38.168	0.003	-276.61--48.19
		400 mg/kgBW	-61.800	38.168	0.503	-176.01-52.41
		200 mg/kgBW	-5.800	38.168	1.000	-120.01-108.41
		Positive Control	-112.600	38.168	0.055	-226.81-1.61
3	600 mg/kgBW	400 mg/kgBW	87.400	37.425	0.175	-24.59-199.39
		200 mg/kgBW	123.800*	37.425	0.026	11.81-235.79
		Positive Control	24.400	37.425	0.964	-87.59-136.39
		Negative Control	125.800*	37.425	0.023	13.81-237.79
	400 mg/kgBW	600 mg/kgBW	-87.400	37.425	0.175	-199.39-24.59
		200 mg/kgBW	36.400	37.425	0.864	-75.59-148.39
		Positive Control	-63.000	37.425	0.466	-174.99-48.99
		Negative Control	38.400	37.425	0.840	-73.59-150.39
	200 mg/kgBW	600 mg/kgBW	-123.800*	37.425	0.026	-235.79--11.81
		400 mg/kgBW	-36.400	37.425	0.864	-148.39-75.59
		Positive Control	-99.400	37.425	0.097	-211.39-12.59
		Negative Control	2.000	37.425	1.000	-109.99-113.99
	Positive Control	600 mg/kgBW	-24.400	37.425	0.964	-136.39-87.59
		400 mg/kgBW	63.000	37.425	0.466	-48.99-174.99
		200 mg/kgBW	99.400	37.425	0.097	-12.59-211.39
		Negative Control	101.400	37.425	0.088	-10.59-213.39
	Negative Control	600 mg/kgBW	-125.800*	37.425	0.023	-237.79--13.81
		400 mg/kgBW	-38.400	37.425	0.840	-150.39-73.59
		200 mg/kgBW	-2.000	37.425	1.000	-113.99-109.99
		Positive Control	-101.400	37.425	0.088	-213.39-10.59
4	600 mg/kgBW	400 mg/kgBW	54.800	26.977	0.288	-25.92-135.52
		200 mg/kgBW	88.000*	26.977	0.028	7.28-168.72
		Positive Control	34.000	26.977	0.717	-46.72-114.72
		Negative Control	87.800*	26.977	0.029	7.08-168.52
	400 mg/kgBW	600 mg/kgBW	-54.800	26.977	0.288	-135.52-25.92
		200 mg/kgBW	33.200	26.977	0.734	-47.52-113.92
		Positive Control	-20.800	26.977	0.936	-101.52-59.92
		Negative Control	33.000	26.977	0.738	-47.72-113.72
	200 mg/kgBW	600 mg/kgBW	-88.000*	26.977	0.028	-168.72--7.28
		400 mg/kgBW	-33.200	26.977	0.734	-113.92-47.52
		Positive Control	-54.000	26.977	0.301	-134.72-26.72
		Negative Control	-0.200	26.977	1.000	-80.92-80.52
	Kontrol Positif	600 mg/kgBW	-34.000	26.977	0.717	-114.72-46.72
		400 mg/kgBW	20.800	26.977	0.936	-59.92-101.52
		200 mg/kgBW	54.000	26.977	0.301	-26.72-134.72
		Negative Control	53.800	26.977	0.304	-26.92-134.52
		600 mg/kgBW	-87.800*	26.977	0.029	-168.52--7.08

Days	Mice Group	Mice Group	MD	SE	*p*	95% CI
	Negative Control	400 mg/kgBW	-33.000	26.977	0.738	-113.72-47.72
		200 mg/kgBW	0.200	26.977	1.000	-80.52-80.92
		Positive Control	-53.800	26.977	0.304	-134.52-26.92
5	600 mg/kgBW	400 mg/kgBW	40.800	24.531	0.477	-32.60-114.20
		200 mg/kgBW	59.400	24.531	0.150	-14.00-132.80
		Positive Control	20.200	24.531	0.920	-53.20-93.60
		Negative Control	45.000	24.531	0.383	-28.40-118.40
	400 mg/kgBW	600 mg/kgBW	-40.800	24.531	0.477	-114.20-32.60
		200 mg/kgBW	18.600	24.531	0.940	-54.80-92.00
		Positive Control	-20.600	24.531	0.915	-94.00-52.80
		Negative Control	4.200	24.531	1.000	-69.20-77.60
	200 mg/kgBW	600 mg/kgBW	-59.400	24.531	0.150	-132.80-14.00
		400 mg/kgBW	-18.600	24.531	0.940	-92.00-54.80
		Positive Control	-39.200	24.531	0.515	-112.60-34.20
		Negative Control	-14.400	24.531	0.975	-87.80-59.00
	Positive Control	600 mg/kgBW	-20.200	24.531	0.920	-93.60-53.20
		400 mg/kgBW	20.600	24.531	0.915	-52.80-94.00
		200 mg/kgBW	39.200	24.531	0.515	-34.20-112.60
		Kontrol Negatif	24.800	24.531	0.847	-48.60-98.20
	Negative Control	600 mg/kgBW	-45.000	24.531	0.383	-118.40-28.40
		400 mg/kgBW	-4.200	24.531	1.000	-77.60-69.20
		200 mg/kgBW	14.400	24.531	0.975	-59.00-87.80
		Positive Control	-24.800	24.531	0.847	-98.20-48.60
6	600 mg/kgBW	400mg/kgBW	9.200	20.192	0.990	-51.22-69.62
		200mg/kgBW	33.600	20.192	0.477	-26.82-94.02
		Positive Control	-4.000	20.192	1.000	-64.42-56.42
		Negative Control	19.200	20.192	0.873	-41.22-79.62
	400 mg/kgBW	600mg/kgBW	-9.200	20.192	0.990	-69.62-51.22
		200mg/kgBW	24.400	20.192	0.747	-36.02-84.82
		Positive Control	-13.200	20.192	0.964	-73.62-47.22
		Negative Control	10.000	20.192	0.987	-50.42-70.42
	200 mg/kgBW	600mg/kgBW	-33.600	20.192	0.477	-94.02-26.82
		400mg/kgBW	-24.400	20.192	0.747	-84.82-36.02
		Positive Control	-37.600	20.192	0.368	-98.02-22.82
		Negative Control	-14.400	20.192	0.951	-74.82-46.02
	Positive Control	600mg/kgBW	4.000	20.192	1.000	-56.42-64.42
		400mg/kgBW	13.200	20.192	0.964	-47.22-73.62
		200mg/kgBW	37.600	20.192	0.368	-22.82-98.02
		Negative Control	23.200	20.192	0.779	-37.22-83.62
	Negative Control	600mg/kgBW	-19.200	20.192	0.873	-79.62-41.22
		400mg/kgBW	-10.000	20.192	0.987	-70.42-50.42
		200 mg/kgBW	14.400	20.192	0.951	-46.02-74.82
		Positive Control	-23.200	20.192	0.779	-83.62-37.22
7	600 mg/kgBW	400 mg/kgBW	80.200	28.888	0.077	-6.24-166.64
		200 mg/kgBW	98.200*	28.888	0.021	11.76-184.64
		Positive Control	87.600*	28.888	0.046	1.16-174.04
		Negative Control	93.800*	28.888	0.029	7.36-180.24
	400 mg/kgBW	600 mg/kgBW	-80.200	28.888	0.077	-166.64-6.24

Days	Mice Group	Mice Group	MD	SE	*p*	95% CI
		200 mg/kgBW	18.000	28.888	0.970	-68.44-104.44
		Positive Control	7.400	28.888	0.999	-79.04-93.84
		Negative Control	13.600	28.888	0.989	-72.84-100.04
	200 mg/kgBW	600 mg/kgBW	-98.200*	28.888	0.021	-184.64--11.76
		400 mg/kgBW	-18.000	28.888	0.970	-104.44-68.44
		Positive Control	-10.600	28.888	0.996	-97.04-75.84
		Negative Control	-4.400	28.888	1.000	-90.84-82.04
	Positive Control	600 mg/kgBW	-87.600*	28.888	0.046	-174.04--1.16
		400 mg/kgBW	-7.400	28.888	0.999	-93.84-79.04
		200 mg/kgBW	10.600	28.888	0.996	-75.84-97.04
		Negative Control	6.200	28.888	0.999	-80.24-92.64
	Negative Control	600 mg/kgBW	-93.800*	28.888	0.029	-180.24--7.36
		400 mg/kgBW	-13.600	28.888	0.989	-100.04-72.84
		200 mg/kgBW	4.400	28.888	1.000	-82.04-90.84
		Positive Control	-6.200	28.888	0.999	-92.64-80.24
Before alloxan induction	600 mg/kgBW	400 mg/kgBW	25.600	15.603	0.490	-21.09-72.29
		200 mg/kgBW	-10.200	15.603	0.964	-56.89-36.49
		Positive Control	-29.800	15.603	0.344	-76.49-16.89
		Negative Control	36.800	15.603	0.168	-9.89-83.49
	400 mg/kgBW	600 mg/kgBW	-25.600	15.603	0.490	-72.29-21.09
		200 mg/kgBW	-35.800	15.603	0.188	-82.49-10.89
		Positive Control	-55.400*	15.603	0.015	-102.09--8.71
		Negative Control	11.200	15.603	0.950	-35.49-57.89
	200 mg/kgBW	600 mg/kgBW	10.200	15.603	0.964	-36.49-56.89
		400 mg/kgBW	35.800	15.603	0.188	-10.89-82.49
		Positive Control	-19.600	15.603	0.720	-66.29-27.09
		Negative Control	47.000*	15.603	0.048	0.31-93.69
	Positive Control	600 mg/kgBW	29.800	15.603	0.344	-16.89-76.49
		400 mg/kgBW	55.400*	15.603	0.015	8.71-102.09
		200 mg/kgBW	19.600	15.603	0.720	-27.09-66.29
		Negative Control	66.600*	15.603	0.003	19.91-113.29
	Negative Control	600 mg/kgBW	-36.800	15.603	0.168	-83.49-9.89
		400 mg/kgBW	-11.200	15.603	0.950	-57.89-35.49
		200 mg/kgBW	-47.000*	15.603	0.048	-93.69--.31
		Positive Control	-66.600*	15.603	0.003	-113.29--19.91
After alloxan induction	600 mg/kgBW	400 mg/kgBW	164.800*	43.571	0.009	34.42-295.18
		200 mg/kgBW	72.200	43.571	0.481	-58.18-202.58
		Positive Control	148.200*	43.571	0.021	17.82-278.58
		Negative Control	350.600*	43.571	0.000	220.22-480.98
	400 mg/kgBW	600 mg/kgBW	-164.800*	43.571	0.009	-295.18--34.42
		200 mg/kgBW	-92.600	43.571	0.248	-222.98-37.78
		Positive Control	-16.600	43.571	0.995	-146.98-113.78
		Negative Control	185.800*	43.571	0.003	55.42-316.18
	200 mg/kgBW	600 mg/kgBW	-72.200	43.571	0.481	-202.58-58.18
		400 mg/kgBW	92.600	43.571	0.248	-37.78-222.98
		Positive Control	76.000	43.571	0.431	-54.38-206.38
		Negative Control	278.400*	43.571	0.000	148.02-408.78
		600 mg/kgBW	-148.200*	43.571	0.021	-278.58--17.82

Days	Mice Group	Mice Group	MD	SE	*p*	95% CI
	Positive Control	400 mg/kgBW	16.600	43.571	0.995	-113.78-146.98
		200 mg/kgBW	-76.000	43.571	0.431	-206.38-54.38
		Negative Control	202.400*	43.571	0.001	72.02-332.78
	Negative Control	600 mg/kgBW	-350.600*	43.571	0.000	-480.98--220.22
		400 mg/kgBW	-185.800*	43.571	0.003	-316.18--55.42
		200 mg/kgBW	-278.400*	43.571	0.000	-408.78--148.02
		Positive Control	-202.400*	43.571	0.001	-332.78--72.02

**The mean difference is significant at the 0.05 level.*
mg/kgBW: milligrams per kilogram of body weight; p: p-value; MD: Mean Difference; SE: Standard Error; CI: Confidence Interval

On the fifth and sixth days, the Tukey test results showed no significant differences between groups. This condition indicates that the physiological response of mice to treatment underwent a stabilization or adaptation phase, thereby increasing inter-individual variability and reducing statistical sensitivity to the detection of differences. However, on the seventh day, patterns of difference reappeared significantly, particularly between the 600 mg/kgBW dose group and the negative control group as well as the 200 mg/kgBW dose group ($p < 0.05$). This indicates that the blood sugar-lowering effect at the highest dose reached a stronger stability towards the end of the observation period (Table 2).

Before alloxan induction, there were significant differences in baseline blood glucose between groups ($p=0.003$), indicating that groups were not fully comparable at the start. This pre-existing variability should be acknowledged as a limitation when interpreting treatment effects. After alloxan induction, very significant differences emerged between groups ($p < 0.01$), with the negative control group showing the highest blood sugar levels. The Tukey test confirmed that the 600 mg/kgBW group showed the largest absolute glucose reduction compared to the negative control (Table 2); however, the 200 mg/kgBW group achieved the lowest final blood glucose value (109.4 mg/dL), indicating superior normalization.

The post-hoc analysis results indicate that all doses of B. flabellifer flower extract produced antihyperglycemic effects. The 600 mg/kgBW dose yielded the greatest absolute blood glucose reduction, while the 200 mg/kgBW dose achieved the best glucose normalization by day 7. These two outcomes should be distinguished when drawing conclusions about dose optimality. These findings support the potential of B. flabellifer Linn flower extract as a candidate for a natural antidiabetic agent; however, mechanistic conclusions regarding β-cell protection and insulin sensitivity cannot be confirmed from blood glucose data alone and require further biochemical investigation.

The results of this study indicate that treatment at doses of 200 mg/kgBW, 400 mg/kgBW, and 600 mg/kgBW was able to reduce blood glucose levels in mice induced by alloxan. The ANOVA test showed significant differences between groups on most observation days. Tukey analysis confirmed a significant difference between the 600 mg/kgBW dose and the negative control at several observation points in terms of absolute glucose reduction. However, the 200 mg/kgBW dose achieved the lowest final blood glucose level (109.4 mg/dL), demonstrating superior normalization. These findings suggest that the relationship between dose and antihyperglycemic outcome is not straightforwardly linear and that higher doses do not necessarily yield better normalization.

The effectiveness of the 600 mg/kgBW dose is thought to be related to the bioactive content in the extract of *B. flabellifer* Linn or the compounds used. A study conducted by Malayil et al. (2022) revealed that phytochemical compounds such as flavonoids, alkaloids, polyphenols, and terpenoids have the ability to increase insulin sensitivity, reduce insulin resistance, and protect pancreatic β cells from oxidative damage caused

by alloxan (Malayil et al., 2022). A study by Singh and Rout (2023) reported that flavonoids play an important role in stimulating pancreatic β cell regeneration and reducing the levels of free radicals produced by alloxan (Singh & Rout, 2023). Aparnna et al. (2024) also showed that polyphenol-rich plant extracts can lower glucose levels by increasing glycolysis enzyme activity and suppressing gluconeogenesis (Aparnna et al., 2024). The consistent decrease in glucose at a dose of 600 mg/kgBW in this study supports this mechanism, primarily through increased insulin sensitivity and cellular protection.

The insignificant differences on days 5 and 6 may be due to the metabolic adaptation phase of mice to the treatment. This phenomenon has been reported in a study by Dash et al. (2024), which showed that blood glucose response in hyperglycemia models sometimes fluctuates in the middle of the observation period due to a temporary decrease in the responsiveness of β cells to phytotherapeutic agents (Dash et al., 2024). In addition, biological variability among mice can increase the standard error, thereby reducing statistical significance even though a downward trend remains physiologically apparent.

Interestingly, on day 7, the differences between groups were again significant, and the 600 mg/kgBW dose showed the strongest effect. The stability of the effects at the end of the observation period reflects the accumulation or positive adaptation process to the active compound. This is consistent with the report by Saravanan et al. (2023) that certain antioxidant compounds require time to reach optimal concentrations in tissues before producing maximum effects on blood glucose (Saravanan et al., 2023). Thus, the findings on day 7 reinforce the hypothesis that the antihyperglycemic effect of the treatment is progressive.

The drastic change in glucose levels after alloxan induction also supports the validity of the research model. Alloxan is known to work through a mechanism of free radical formation that causes damage to pancreatic β cells, leading to acute hyperglycemia. These findings are in line with the study by Sudiono and Susanto (2021), which reported that alloxan produces harmful reactive oxygen species that trigger β cell necrosis (Sudiono & Susanto, 2021). The high glucose levels in the negative control group after induction in this study reinforce that the hyperglycemia model was well established before the intervention was performed (Dzigbor et al., 2025).

Overall, the results of the study reinforce previous findings showing that bioactive compounds in *B. flabellifer* Linn extract can be effective antihyperglycemic agents. The effect observed at a dose of 600 mg/kgBW provides evidence that increasing the dose enhances the pharmacological effect without showing signs of toxicity during the observation period. The study findings can serve as a basis for further research focusing on molecular mechanisms, metabolite profiles, and long-term toxicity testing to ensure its safety for use.

The study has several limitations that should be noted. First, the number of mouse samples was relatively limited, which may affect the accuracy of estimating the effects between groups. Second, the study only focused on blood glucose parameters without measuring other supporting biomarkers such as insulin levels, oxidative stress, or pancreatic histopathology, which could provide a more comprehensive picture of the mechanism. Additionally, the relatively short observation period limits the ability to assess long-term effects and potential toxicity of the treatment, and the study did not evaluate individual response variations that may be influenced by genetic or metabolic factors.

Further research is recommended to expand the sample size and extend the duration of the intervention in order to evaluate the long-term stability of the antihyperglycemic effect. Evaluation of additional biochemical biomarkers such as insulin profile, malondialdehyde (MDA), antioxidant enzyme activity, and histopathological analysis of pancreatic β cells is also important to gain a deeper understanding of the molecular mechanisms. Additionally, subchronic and chronic toxicity testing and

exploration of a wider range of doses are needed to determine the safety limits and optimal efficacy. Initial clinical trials in humans can be the next step after safety and efficacy in animal models have been strongly verified.

4. CONCLUSION

The results of the study confirm that B. flabellifer Linn flower extract at doses of 200, 400, and 600 mg/kgBW was able to reduce blood glucose levels in alloxan-induced mice. Significant differences between groups were observed on most observation days. The 600 mg/kgBW dose produced the largest absolute blood glucose reduction, while the 200 mg/kgBW dose achieved the best glucose normalization by day 7 (final level: 109.4 mg/dL). These two outcomes represent different dimensions of antihyperglycemic efficacy and should not be conflated when determining the optimal dose.

The study findings reinforce evidence that bioactive compounds in the treatment are capable of protecting pancreatic β cells from oxidative damage and improving blood glucose regulation under alloxan-induced hyperglycemia. Overall, the results support the potential of B. flabellifer Linn flower extract as a natural antidiabetic candidate, with the optimal dose depending on whether the target outcome is maximum absolute reduction (600 mg/kgBW) or blood glucose normalization (200 mg/kgBW). These findings should be interpreted cautiously given the preliminary nature of the study, including limited sample size, short observation period, and lack of mechanistic or phytochemical characterization. Further research incorporating larger sample sizes, phytochemical profiling, repeated-measures analysis, toxicity testing, and mechanistic investigation is recommended before wider clinical application.

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