



Research Article

DETERMINING PARAMETERS FOR A PROTOCOL TO ASSESS THE BLOOD GLUCOSE-LOWERING ABILITY OF AQUEOUS GUAVA EXTRACT IN STREPTOZOTOCIN-INDUCED MICE

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ABSTRACT

*To assess the bioactive properties of guava leaf extract (*Psidium guajava* L.) in lowering blood glucose, in vivo experiments were designed, including parameters such as the concentration and dose of the extract, the dose of streptozotocin (STZ) used to treat mice, and the time points for administering the extract to STZ-induced mice. The aims of this study were to determine various factors related to the experimental procedure and to evaluate the effects of guava extract in a mouse model. Specifically, the study determined the optimal extract concentrations and examined physiological functions in mice, including body weight changes, oxidative stress through malondialdehyde (MDA) markers in the kidneys and pancreas of mice, blood glucose levels, and insulin and glucose tolerance. At different aqueous extract concentrations ranging from 100 mg/kg body weight (bw) to 800 mg/kg bw, physiological and metabolic abnormalities were observed in mice. There was no significant difference between the mice in the experimental and control groups. A single dose of STZ (100, 150, or 200 mg/kg bw) was used to induce prediabetic conditions in mice, characterized by symptoms such as hyperglycemia, loss of appetite, weight loss, insulin resistance, and impaired glucose tolerance. The results indicate that a dose of 150 mg/kg bw STZ effectively induced these prediabetic characteristics. Additionally, the survival rate of mice in this group was higher than that of mice treated with 200 mg/kg bw STZ.*

Keywords: guava leaf extract; prediabetic conditions

1. Introduction

Psidium guajava L. (guava) belongs to the Myrtaceae family and is commonly cultivated in tropical regions. In Vietnam, guava is grown across the country, with diverse varieties of different origins, such as Taiwanese Red Guava and Queen Guava. Various components of the plant, such as its fruits, bark, and leaves, contain bioactive compounds

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with potential therapeutic applications, including polyphenols, tannins, flavonoids, carotenoids, lycopene, ascorbic acid, and potassium. For instance, studies have demonstrated that extracts from guava fruits have antioxidant properties, inhibit lipid peroxidation in cellular membranes, induce cytotoxicity in cancer cells, and exhibit anti-inflammatory activity. Specifically, Jamieson et al. (2021) and Joshi et al. (2023) identified secondary metabolites in guava leaves, such as tannins, triterpenoids, and meroterpenoids, which play a crucial role in lowering blood glucose levels. Guava leaves are pruned annually to promote growth and optimize fruit yield in orchards.

Diabetes is a disorder of carbohydrate metabolism characterized by hyperglycemia and classified into type 1 and type 2. Type 2 diabetes develops gradually, often without noticeable symptoms, increasing the risk of serious complications. In addition to genetic predisposition and family history, factors such as an unhealthy diet, physical inactivity, obesity, metabolic disorders, and aging increase the risk of developing type 2 diabetes. Alongside a healthy lifestyle and a balanced diet, early screening and diagnosis are necessary to reduce the risks and consequences of this disease. Developing drugs with minimal side effects for prevention and treatment remains a challenge.

Animal experiments using models such as Swiss albino mice are designed to assess the properties of bioactive compounds or herbal extracts. Currently, STZ is widely used to induce diabetes in experimental animal models by selectively attacking pancreatic β -cells, leading to insulin deficiency and hyperglycemia. A single high dose of STZ (100–200 mg/kg bw) causes severe pancreatic β -cell injury and dysfunction, leading to insulin resistance. In contrast, a low-dose STZ injection (25–50 mg/kg) causes partial β -cell damage, impairing insulin secretion without complete β -cell destruction. After entering the β -cells via the GLUT2 glucose transporter, STZ undergoes glycolysis, self-oxidation, and free radical generation, triggering inflammatory responses, activating signaling pathways, and disrupting insulin synthesis, gradually changing blood glucose levels. The STZ-induced prediabetic model closely reflects the metabolic disruptions seen in early-stage disease, such as mild β -cell impairment, unstable blood glucose, and insulin resistance. Prolonged exposure results in irreversible damage to the pancreatic islets, characterized by diminished islet mass and chronic hyperglycemia.

Consequently, this study aims to establish the baseline parameters of this STZ-induced animal model to evaluate the protective efficacy of guava extract against prediabetic progression.

2. Materials and methods

2.1. Guava Leaf Extract

Mature leaves of the red guava (*Psidium guajava* L.) were collected from Binh Chanh District, Ho Chi Minh City. After being washed, the plant material was air-dried at room temperature in the Genetics and Evolution Laboratory. To ensure complete moisture

removal, the samples were further dehydrated in a Memmert UN110 oven (Germany) at 50–60 °C until a constant weight was achieved, after which the final dry mass was recorded.

The aqueous extraction process was adapted from the method described by Nguyen (2007). Specifically, 20 g of dried leaves were boiled in 500 mL of distilled water for 5 minutes, followed by continuous heating at 70–80 °C for an additional 3 hours. The resulting mixture was then centrifuged at 3,000 rpm for 10 minutes to eliminate solid residues. To obtain the concentrated extract, the solvent was thoroughly evaporated using a water bath set to 50–55 °C. Finally, the extraction yield was quantified, and the extract was stored at 4°C in the dark to preserve the stability of the bioactive compounds.

2.2. Animals

Male albino mice (*Mus musculus* var. *albino*) weighing 16–18 g (4–5 weeks of age) were acquired from the Institute of Biotechnology, Ho Chi Minh City, Vietnam. Prior to the main experiment, the mice were acclimatized for 1 to 2 weeks at the Genetics and Evolution Laboratory. The animals were grouped in standard cages (4 to 6 individuals per cage) and housed within a climate-controlled facility maintained at 26 ± 2 °C under a 12-hour light/dark cycle. The mice were supplied with a standard pellet diet (Rat Pro-01) equivalent to 5% of their body weight, distributed evenly between two daily feedings at 08:00 and 20:00.

2.3. Safety of Guava Leaf Extract in Mice

To evaluate the physiological safety of the extract under normal conditions, 30 mice were randomly assigned to five groups. The control group (Group 1) received no intervention. Guided by the dosage protocols established by Dai et al. (2012) and Pham et al. (2024), the experimental groups (Groups 2–5) received a 7-day regimen of a single daily oral administration of the extract at respective doses of 100, 200, 400, and 800 mg/kg body weight.

Body weight, blood glucose levels, and general behavior were recorded throughout the experiment. At the end of the study, the mice were humanely euthanized by cervical dislocation, and their kidneys and pancreas were collected for MDA quantification.

2.4. Body Weight

Metabolic disorders can impact physiological functions, often resulting in body weight fluctuations in mice. To monitor these variations, the animals were weighed daily prior to feeding, allowing for the analysis of the difference between their initial and final body weights.

2.5. Blood Glucose Levels

Blood glucose concentrations serve as a practical indicator of the extent of pancreatic cell dysfunction caused by either STZ or the tested extract. Prior to feeding, blood samples were collected from the tail vein using a 28-gauge lancet, and glucose levels were measured with a blood glucose meter (Sinocare, Germany). These measurements were systematically recorded before the experiment, throughout the study, and upon its completion.

2.6. Quantification of MDA Levels

MDA is a biomarker of lipid peroxidation in cell membranes, reflecting oxidative stress. In this study, MDA levels were determined by measuring optical density (OD) at 532 nm using a spectrophotometer. Metabolic disorders in the body can affect physiological functions, leading to changes in body weight in mice. To monitor these changes, the mice were weighed daily before feeding, and the difference between their initial and final body weights was recorded.

The kidneys and pancreas of the mice were collected, rinsed with 0.9% NaCl physiological saline solution, and weighed. The tissues were then homogenized under cold conditions (0–4 °C) in 1.15% KCl solution at a ratio of 1 g of tissue to 10 mL of solution and centrifuged at 5,000 rpm at 4 °C for 15 minutes to obtain the supernatant. A volume of 2 mL of kidney homogenate or 1 mL of pancreatic homogenate was incubated with 25 mM Tris-HCl buffer at a 2:1 ratio at 37 °C for 60 minutes. The reaction was terminated by adding 1 mL of 10% trichloroacetic acid (TCA) for kidney samples or 0.5 mL of 10% TCA for pancreatic samples. The mixture was then centrifuged at 10,000 rpm at 4 °C for 10 minutes, and the supernatant was collected. Next, 1 mL of 0.8% thiobarbituric acid (TBA, Merck) solution was added to the reaction mixture, incubated at 100 °C for 15 minutes, and then rapidly cooled. The optical density was measured at 532 nm using a spectrophotometer (Ultrospec 2100 pro).

2.7. Design of the STZ-Induced Diabetic Mouse Model

Before the experiment, 15 male mice (6–7 weeks old, weighing 20–30 g) were acclimatized within a controlled laboratory environment. Drawing upon the experimental design described by Brondum et al. (2005), the animals were randomly allocated into three groups ($n = 5$ per group). In accordance with the procedures outlined by Deeds et al. (2011), each group received an intraperitoneal STZ injection at one of the following doses: 100, 150, or 200 mg/kg of body weight.

Baseline body weights and initial blood glucose concentrations were recorded at the onset of the trial. Subsequently, insulin and glucose tolerance assessments were performed based on the framework established by Benedé-Ubieto et al. (2020). These evaluations required the mice to fast overnight for 6–8 hr before measurement, as described below.

After fasting, the mice in the three groups were administered STZ at the specified concentrations. Following injection, they were provided with a 10% sucrose solution and maintained on standard chow and water ad libitum. Physiological parameters, including appetite loss, reduced activity, aggression (fighting), and lethargy, were monitored. Additionally, body weight and blood glucose levels were measured at the 24-hour time point. Blood glucose fluctuations were monitored every three days for 7–8 days.

Mice exhibiting prediabetic symptoms, including hyperglycemia (>9 mmol/L), insulin tolerance, and increased blood glucose levels after glucose administration, were selected for the next experiment to evaluate the effectiveness of the extract in reducing blood glucose levels.

2.8. Insulin Tolerance Test

To determine the effectiveness of STZ-induced prediabetes in mice, insulin sensitivity was assessed following the method described by Benedé-Ubieto et al. (2020). After a 6–8-hr fast, blood glucose levels were measured using a tail vein blood sample, and body weights were recorded. Mice were then given a single intraperitoneal insulin dose of 1 U/kg body weight. Blood glucose levels were recorded at 30, 60, and 120 min after injection. The reduction in blood glucose levels over time indicated insulin resistance, a characteristic of prediabetic conditions.

2.9. Glucose Tolerance Test

The procedure for evaluating glucose tolerance in this study was based on the method described by Benedé-Ubieto et al. (2020). Similar to the insulin resistance test above, a single glucose dose of 2 g/kg body weight was administered to the mice after a 6–8-hr fast. Blood glucose levels were recorded using tail vein blood samples at 30, 60, and 120 min after administration. Persistently high blood glucose concentrations reflect compromised glycemic control, a primary feature of prediabetes.

2.10. Statistical Analysis

To assess the physiological effects of the guava leaf extract and the impact of STZ induction on the mice, data including body weight, blood glucose, and MDA levels were expressed as means and standard deviations. Statistical comparisons among the experimental groups were performed with a one-way analysis of variance (ANOVA) in SPSS (Version 27.0), with significance defined at $p < .05$. For data visualization, GraphPad Prism 9.5 was used to construct line graphs tracking body weight variations and scatter plots detailing MDA concentrations.

3. Results and discussion

3.1. Results

3.1.1. Safety of Guava Leaf Extract in Mice

Body Weight Changes

At lower concentrations of guava extract (100 mg/kg and 200 mg/kg bw), a slight increase in body weight was observed in the days following administration (Figure 1). The fluctuations in body weight in these groups were comparable to those in the control group ($p > .05$). By the end of the experiment, body weight increased to 27.87–31.23 g in the 100 mg/kg bw extract group and 27.59–29.9 g in the 200 mg/kg bw extract group, indicating no adverse effects on weight regulation. At higher concentrations of guava extract (400 mg/kg and 800 mg/kg bw), body weight tended to decrease by the fourth day of administration, with reductions of 1.27 g and 1.47 g, respectively, compared to the initial body weight.

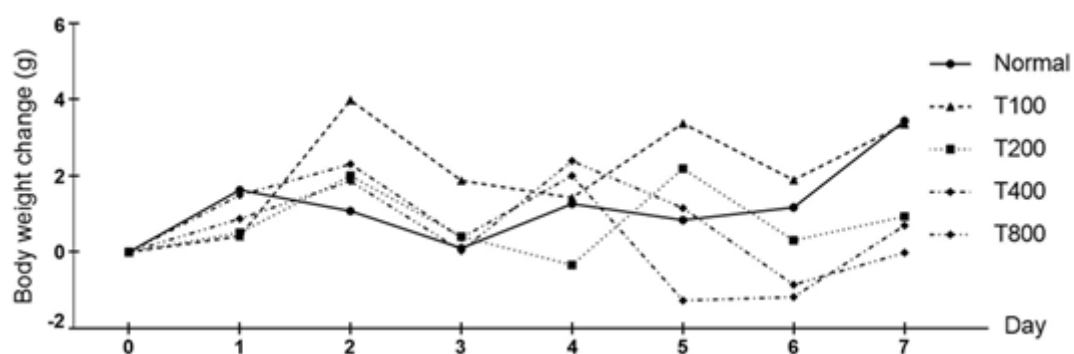


Figure 1. Body Weight Changes in Mice Given Guava Extract

Note. T100, T200, T400, and T800 represent body weight changes in mice given guava extract at 100, 200, 400, and 800 mg/kg bw, respectively, over a 7-day experiment.

Blood Glucose Levels

As shown in Table 1, normal mice exhibited stable glucose regulation, as indicated by a continuous decrease in blood glucose levels post-administration (from 8.20 mmol/L to 4.28 mmol/L, and further to 3.50 mmol/L). In contrast, the 100 mg/kg bw group showed a slight increase during administration (from 6.18 to 6.44 mmol/L) before decreasing to 4.32 mmol/L post-administration. A mild reduction was also observed in the 400 mg/kg bw group, where glucose levels declined from 6.84 mmol/L pre-administration to 4.80 mmol/L post-administration, a fluctuation comparable to that of the normal group ($p > .05$).

Table 1. Blood Glucose Levels in Mice Treated with Guava Leaf Extract

		Before administration	During administration	After administration
Normal	<i>M</i>	8.20	4.28a	3.50a
(<i>n</i> = 5)	<i>SD</i>	1.53	0.82	0.39
100 mg/kg	<i>M</i>	6.18	6.44	4.32
(<i>n</i> = 5)	<i>SD</i>	1.29	1.95	0.80
200 mg/kg	<i>M</i>	5.16	5.20	5.56
(<i>n</i> = 5)	<i>SD</i>	0.84	0.71	1.42
400 mg/kg	<i>M</i>	6.84	6.76	4.80
(<i>n</i> = 5)	<i>SD</i>	1.69	1.24	2.29
800 mg/kg	<i>M</i>	6.12	7.02	4.92
(<i>n</i> = 5)	<i>SD</i>	1.10	2.34	0.64

Note. a indicates $p < .05$ compared with the normal group before administration.

However, in the 800 mg/kg bw group, blood glucose levels increased during administration (from 6.12 to 7.02 mmol/L) before decreasing to 4.92 mmol/L post-administration. Meanwhile, the 200 mg/kg bw group exhibited only minor fluctuations over time. Statistical analysis indicated that the standard deviations in extract-treated groups were higher than in the untreated groups, suggesting variability in response to guava leaf extract administration.

MDA Levels in Kidney and Pancreatic Tissues

MDA levels in kidney cells are represented by the scatter plot in Figure 2A, while MDA levels in pancreatic cells are shown in Figure 2B. In kidney cells, the OD values measured at 532 nm using a spectrophotometer indicated no significant differences between the groups treated with various concentrations of the extract and the normal group ($p > .05$). These values remained stable without a downward trend (Figure 2A). The findings demonstrate that guava leaf extract positively influenced metabolic activity in the kidneys of mice.

Figure 2B depicts the regulation of MDA levels in pancreatic cells after administration of different concentrations of guava extract. Despite variations in responses, no clear dose-dependent trend was observed, indicating that the extract maintained stable metabolic effects ($p > .05$).

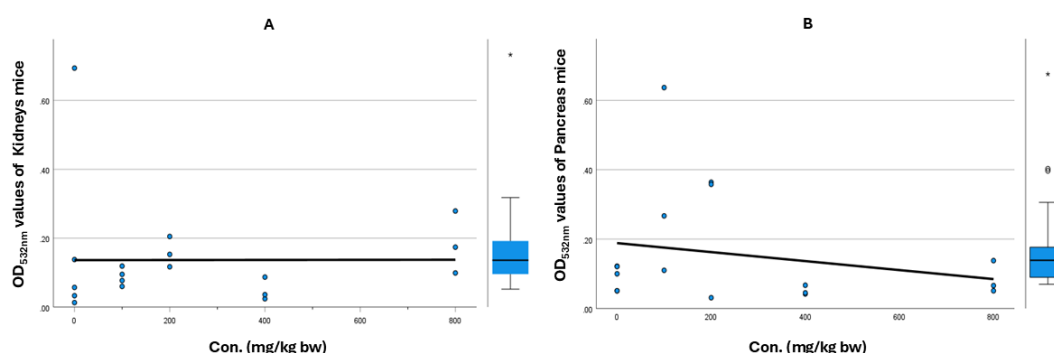


Figure 2. MDA Levels in Kidney (A) and Pancreatic (B) Cells

3.1.2. Effects of STZ on Metabolism in Mice

Body Weight Changes in STZ-Induced Mice

During the experiment, fluctuations in the body weight of mice were observed, indicating that STZ influenced metabolism and glucose regulation. For example, Figure 3 shows that weight gain occurred in the group of mice administered STZ at a concentration of 100 mg/kg bw. In contrast, at higher concentrations of 150 mg/kg bw and 200 mg/kg bw, body weight decreased by 1–2 g by the end of the experiment compared to the initial weight. Notably, a clear weight loss was observed in the 200 mg/kg bw STZ-induced group within the first 24 hours post-injection. While some weight recovery was observed in the following days, metabolic imbalances persisted, leading to further weight loss at the 8-day and 12-day time points. These findings demonstrate that STZ administration causes significant metabolic disturbances, impacting body weight regulation. Consistent with observations reported in the studies by Akbarzadeh et al. (2007) and Kancherla et al. (2016), the STZ-treated group of mice also exhibited a trend towards reduced body weight compared to the control group.

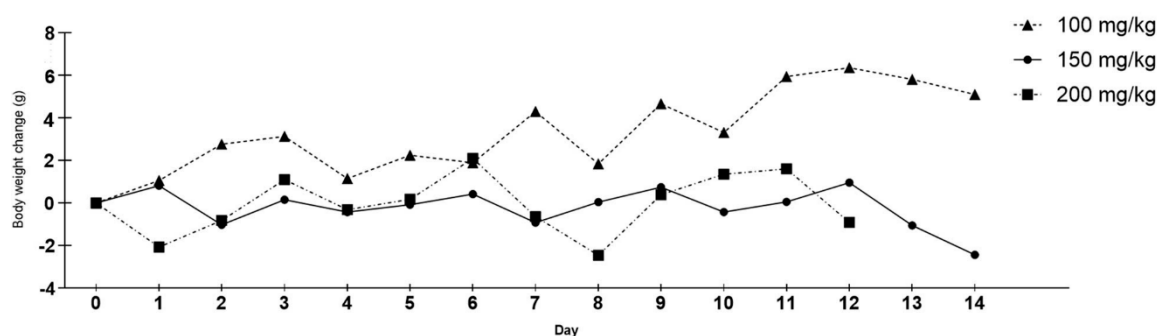


Figure 3. Body Weight Changes in Mice Given Different STZ Doses

Note. Triangles (▲), circles (●), and squares (■) represent body weight changes in mice given STZ at 100, 150, and 200 mg/kg bw, respectively, over a 14-day experiment.

Changes in Blood Glucose Levels in STZ-Induced Mice

After administration, blood glucose levels in mice fluctuated across all groups with various STZ concentrations. In the 100 mg/kg bw STZ-induced group, blood glucose levels decreased from 7.19 to 3.36 mmol/L by the eighth day, followed by a slight increase to 4.34 mmol/L on the following day. In summary, hyperglycemia was not observed in this group of mice (Table 2).

Table 2. Changes in Blood Glucose Levels in STZ-Induced Mice

Treatment		1-hour*	4-hour	1 st -day	3 rd -day	4 th -day	5 th -day	7 th -day	8 th -day	9 th -day	10 th -day	11 th -day	14 th -day	18 th -day
100 mg/kg (n = 8)	M	7.19		6.65			5.15 ^a		3.36 ^a		4.34 ^a		5.21 ^a	5.01 ^a
	SD	0.71		0.59			0.86		0.59		0.82		1.17	0.76
150 mg/kg (n = 7)	M	9.54	5.80	5.21	10.70	7.57	9.19	13.00	9.37	6.54		9.70		
	SD	1.53	1.72	1.37	4.89	6.33	6.10	6.36	4.53	1.31		2.75		
200 mg/kg (n = 6)	M	7.65	6.29	5.02	8.66	8.78		8.02		7.90				
	SD	0.47	2.29	0.90	5.34	5.77		4.76		5.10				

*: Blood glucose levels in mice were measured 1 hour before the experiment began.

^a: p -values < 0,05 when compared with the 100 mg/kg bw treatment group at 1 hour*.

In contrast, blood glucose levels in the 150 mg/kg bw STZ-induced mouse group tended to fluctuate significantly compared to those in the 200 mg/kg bw STZ-injected group. For instance, blood glucose levels decreased 4 hours post-administration, continued to decline in the following days, and then increased on the third day. The blood glucose levels were recorded at 10.70 mmol/L in the 150 mg/kg bw group and 8.66 mmol/L in the 200 mg/kg bw group, respectively. At that time, some individual mice had blood glucose levels exceeding 10 mmol/L. In the following days, blood glucose remained higher than at the time of the initial injection. At the high STZ dose of 200 mg/kg, the survival rate of mice was

approximately 83%, with one recorded fatality, indicating that this dose may cause significant toxicity. Despite the diverse STZ concentrations used in various studies, the increasing trend in blood glucose levels and survival rate observed in our murine model align with the findings of Bloch et al. (2006) and Graham et al. (2011).

Insulin Resistance and Glucose Tolerance in STZ-Induced Mice

Mice exhibiting blood glucose levels exceeding 9 mmol/L after STZ induction at a concentration of 150 mg/kg bw were selected for insulin tolerance measurement.

Table 3 shows the reduction in blood glucose levels over time following insulin administration in mice. The initial blood glucose level decreased to 10.26 mmol/L, followed by a further decline to 8.56 mmol/L at 30 minutes post-injection and 6.06 mmol/L at 120 minutes post-injection, indicating a progressive hypoglycemic response to insulin, characteristic of prediabetic conditions. These changes were not statistically significant ($p > .05$).

At the 60-minute interval, blood glucose concentrations remained persistently high, reflecting compromised insulin sensitivity; however, these values experienced a decline to 4.78 mmol/L at 120 minutes post-glucose administration, pointing to partial glucose disposal (Table 3). Notably, these overall fluctuations did not reach statistical significance ($p > .05$).

In alignment with the findings reported by Ghasemi and Jeddi (2022b) on STZ-treated rat models, the glucose tolerance test (GTT) conducted on mice similarly highlighted a marked deterioration in glycemic regulation (Table 3). Whereas Ghasemi and Jeddi's research primarily addressed deficiencies in insulin release, our insulin tolerance test (ITT) underscored a distinct drop in insulin sensitivity after STZ exposure. Ultimately, both studies confirm that STZ comprehensively disrupts glucose homeostasis by impairing both systemic insulin secretion and cellular sensitivity.

Table 3. Responses to Insulin and Glucose Administration

Time	Insulin tolerance			Glucose tolerance		
	<i>M</i>	<i>SD</i>	<i>p</i>	<i>M</i>	<i>SD</i>	<i>p</i>
0	10.26	3.50		10.26	3.50	
30 min	8.56	3.23	.923	10.27	3.11	.425
60 min	7.70	3.78	.602	8.30	2.59	.887
120 min	6.06	2.40	.211	4.78	1.44	.147

3.2. Discussion

The results demonstrate that guava leaf extract, administered at doses between 100 and 800 mg/kg body weight, actively modulates metabolic functions. This impact is reflected in body weight variations and altered oxidative stress markers, specifically measured via MDA levels in the kidneys and pancreas. Furthermore, the extract contributes to glycemic regulation in mice, which is shown by the observed shifts in blood glucose concentrations before and after treatment. Based on previous studies (Deguchi & Miyazaki, 2010; Dias et al., 2003), extracts derived from different parts of the plant (fruits, leaves, and stems) have demonstrated various bioactivities, including antioxidant, anticancer, and anti-inflammatory

effects. These effects may be explained by the presence of antioxidant compounds in the extract, such as flavonoids, tannins, and alkaloids. At doses of 400 and 800 mg/kg bw, the body weight changed, whereas at the dose of 200 mg/kg bw, body weight remained stable.

Based on the literature, the predicted optimal concentration range for blood glucose lowering in mice is between 100 and 200 mg/kg body weight. Therefore, building upon the preliminary safety and efficacy data, this research will systematically assess the hypoglycemic efficacy of guava leaf extract at a concentration of 200 mg/kg body weight in an STZ-induced experimental diabetic mouse model.

According to Lenzen (2008), after entering pancreatic β -cells via the GLUT2 transporter, STZ causes alkylation of macromolecules such as DNA and proteins, leading to molecular damage and activation of the poly(ADP-ribose) polymerase (PARP) enzyme. This activation results in a reduction of nicotinamide adenine dinucleotide (NAD⁺) and adenosine triphosphate (ATP) levels. Ultimately, this process leads to the destruction of pancreatic β -cells and a decrease in pancreatic tissue volume, contributing to dysregulated glucose metabolism in mice. Because of this, at a concentration of 200 mg/kg bw, STZ affects body weight and survival rate.

At a concentration of 150 mg/kg bw, STZ-treated mice maintained normal physiological functions, including sustained body weight gain, optimal survival rates, and no observable abnormal behaviors throughout the experiment. Furthermore, glucose and insulin tolerance tests verified that this cohort had entered a prediabetic state.

These observations are consistent with the recognized pathways of STZ-mediated β -cell toxicity, where reactive oxygen species (ROS) production—driven by glycosylation and auto-oxidation—triggers targeted cellular injury and metabolic disruption. Post-induction, glycemic values temporarily dropped during the initial 4 hours but rebounded significantly by day 3 or 4. Exogenous insulin challenges further revealed tissue responsiveness, reflecting a broader dysfunction in glucose homeostasis. Ultimately, this defect diminished cellular glucose uptake, culminating in chronic hyperglycemia.

Several study limitations must be noted. First, the extract's safety assessment relied on a restricted set of indicators (body weight, blood glucose, and MDA levels), highlighting the need for comprehensive biochemical and hematological analyses in future work. Second, the selected STZ concentration (150 mg/kg bw)—determined via clinical signs and tolerance tests—should be corroborated by direct measures of β -cell destruction, such as histopathological examination and endogenous insulin assays. Finally, as tolerance tests were only evaluated at a single post-induction time point, longitudinal monitoring is recommended to thoroughly delineate the disease progression in this experimental model.

4. Conclusion

Evaluation of different extract concentrations revealed that both the 100 and 200 mg/kg bw regimens preserved normal physiology in animal subjects. Notably, the 200 mg/kg bw dose was selected for further efficacy testing in STZ-treated groups. Meanwhile, an STZ dose of 150 mg/kg bw effectively induced prediabetes without causing lethal or abnormal outcomes. Therefore, this specific concentration was utilized to explore the glucose-lowering potential of the extract in the STZ-induced mice.

❖ **Conflict of Interest:** Authors have no conflict of interest to declare.

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**XÁC ĐỊNH MỘT SỐ THÔNG SỐ CỦA QUY TRÌNH THÍ NGHIỆM ĐÁNH GIÁ
KHẢ NĂNG HẠ GLUCOSE MÁU CỦA DỊCH CHIẾT LÁ ỔI
TRÊN CHUỘT ĐÁI THÁO ĐƯỜNG BỞI STREPTOZOTOCIN**

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TÓM TẮT

Để đánh giá khả năng hạ glucose máu của cao chiết lá ổi (*Psidium guajava* L.), các thí nghiệm *in vivo* được thiết kế với các thông số như nồng độ và liều lượng của cao chiết, liều lượng Streptozotocin (STZ) được sử dụng để gây tổn thương tế bào β -đảo tụy ở chuột. Vì vậy, nghiên cứu này nhằm mục đích xác định các yếu tố của quy trình thí nghiệm đánh giá hiệu quả của cao chiết trên mô hình chuột. Nghiên cứu đã xác định nồng độ cao chiết phù hợp và kiểm tra các chức năng sinh lý ở chuột, như sự thay đổi về trọng lượng cơ thể, stress oxy hóa thể hiện thông qua hàm lượng malondialdehyde (MDA) ở thận và tuyến tụy, nồng độ glucose máu, khả năng dung nạp insulin và glucose. Trong khoảng nồng độ cao chiết từ 100 mg/kg trọng lượng cơ thể (bw.) đến 800 mg/kg bw, kết quả không ghi nhận được các bất thường về hành vi, sinh lý. Không có sự khác biệt có ý nghĩa về mặt thống kê giữa chuột ở nhóm thử nghiệm và nhóm đối chứng. Với một liều duy nhất (100, 150 hoặc 200 mg/kg bw), STZ đã gây tổn thương tế bào β -đảo tụy ở chuột gồm các triệu chứng đặc trưng như sự tăng đường huyết, bỏ ăn, giảm trọng lượng, kháng insulin và suy giảm dung nạp glucose. Kết quả cho thấy ở liều 150 mg/kg bw, STZ có hiệu quả gây ra các đặc điểm đái tháo đường này. Ngoài ra, tỉ lệ sống sót của chuột trong nhóm này cao hơn so với những con được điều trị bằng 200 mg/kg bw STZ.

Từ khóa: dịch chiết lá ổi; mô hình tiền đái tháo đường