



EXTRACTION, PHYTOCHEMICAL SCREENING, AND BIOLOGICAL ACTIVITY OF *FICUS DALHOUSIAE* AS POTENTIAL ANTI- DIABETIC AGENT

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ABSTRACT

After going through many literature, we have designed a work to investigate herbal plant for the treatment of diabetes mellitus. Therefore we aimed to investigate effectiveness of *Ficus dalhousiae* in diabetes using *in vivo* models. From qualitative phytochemical screening it was observed that the extract possess numerous kind of chemicals such as alkaloid, carbohydrates, saponins, glycoside, fats and fixed oils, resins, phenols, tannins, diterpins, flavonoids, and proteins. *In vivo* antidiabetic activity of extract have been performed in STZ-induced diabetic model in rats. The antidiabetic potential have been determined by estimating different biochemical parameters such as body weight, serum glucose level, SGOT (AST), and SGPT (ALT) levels. The antidiabetic potential of the extract was comparable with that of Metformin, which is evidenced by decreased levels of blood glucose, SGOT, and SGPT. The extract was given to the animals at doses of 100 mg/kg (E1) and 200 mg/kg (E2). The decreased body weight was significantly ($p < 0.001$) improved in E1, E2, and metformin treated groups. The basal serum glucose level of animals of all groups was found to be statistically equivalent. NC group serum OGTT levels were observed at specific time intervals of 0 min, 30 min, 60 min, and 120 min along with the AUC curve. NC group level was 283 ± 5.3 , whereas DC group OGTT levels were considerably ($p < 0.001$) higher at 375 ± 4.9 mg/dl. Treatment with E1, E2, and metformin ($p < 0.001$), considerably reduced these elevated levels. The mean serum SGOT level of NC group was 58 ± 1.4 U/L, which significantly ($p < 0.001$) increased to 90 ± 1.2

U/L in DC group, the increased levels were significantly decreased in E1, E2, and metformin group ($p < 0.001$), when compared to DC group. Diabetes induction caused significant increase ($p < 0.001$) in SGPT levels from 53 ± 1.8 U/L to 98 ± 1.3 U/L, when compared to the NC group. The increased SGPT levels were significantly decreased in E1, E2, and metformin group ($p < 0.001$), when compared to DC group. Thus, our findings demonstrate that the *Ficus dalhousiae* possess significant antidiabetic activity in dose dependent manner and if optimize latter can be used clinically to overcome diabetes in a very convenient way.

Keyword: Streptozotocin, Metformin, *Ficus dalhousiae*

1. INTRODUCTION

1.1 Diabetes Mellitus

Diabetes mellitus is a chronic disorder of carbohydrates, fats and protein metabolism. A defective or deficient insulin secretory response, which translates into impaired carbohydrates (glucose) use, is a characteristic feature of diabetes mellitus, as is the resulting hyperglycemias. Diabetes mellitus (DM) is commonly referred to as a “sugar” and it is the most common endocrine disorder and usually occurs when there is deficiency or absence of insulin or rarely, impairment of insulin activity (insulin resistance). The International Diabetes Federation (IDF) estimates the total number of diabetic subjects to be around 40.9 million in India and this is further set to rise to 69.9 million by the year 2025.

Insulin and glucagon hormones both are secreted by the pancreas. Insulin is secreted by the beta (β) cells and glucagon is secreted by the alpha (α) cells both are located in the islets of Langerhan's. Insulin decreases the blood glucose level by the glycogenesis and transport glucose into the muscles, liver and adipose tissue. Neural tissue and erythrocytes do not required insulin for glucose utilization whereas alpha (α) cells plays an important role in controlling blood glucose by producing the glucagon and it increases the blood glucose level by accelerating the glycogenolysis.

In addition to increased risk of obesity, metabolic and cardiovascular disorders, and malignancy in future life of fetus after delivery. Type II diabetes mellitus comprises 80% to 90% of all cases of diabetes mellitus. Geographical variation can contribute in the magnitude of the problems and to overall morbidity and mortality. Moreover, people with diabetes who undertake moderate amounts of physical activity are at inappreciably lower risk of death than inactive persons. It is now well established that a specific genetic constitution is required for such an event to cause.

1.2 Etiology of Diabetes Mellitus

The word etiology is derived from Greek word “aetiologia”. Hence, etiology is defined as the science of finding causes and origins in which a disease is arise, It includes:

1. It is currently believed that the juvenile-onset (insulin dependent) form has an auto immune etiology.
2. Viruses may also play a role in the etiology of diabetes like coxsackieB.
3. Mumps and rubella viruses all have been shown to produce morphologic changes in the islet cell structure.

- The genetic role in the etiology of diabetes is controversial. Possibly a genetic trait makes an individual's pancreas more susceptible to one of the above viruses.

1.3 Causes of Diabetes Mellitus

Disturbances or abnormality in gluco-receptor of β cell so that they respond to higher glucose concentration or relative β cell deficiency. In either way, insulin secretion is impaired; may progress to β cell failure. The theory of principal in micro vascular disease leading to neural hypoxia, and the direct effects of hyperglycaemia on neuronal metabolism.

- Reduced sensitivity of peripheral tissues to insulin: reduction in number of insulin receptors, 'down regulation' of insulin receptors. Many hypersensitive and hyperinsulinaemic, but normal glycaemic; and have associated dyslipidaemic, hyperuricaemic, abdominal obesity. Thus there is relative insulin resistance, particularly at the level of liver, muscle and fat. Hyperinsulinaemic has been implicated in causing angiopathy.
- Excess of hyperglycaemia hormone (glucagon) etc./obesity; causes relative insulin deficiency—the β cells lag behind. Two theories have demonstrated abnormalities in nitric oxide metabolism, resulting in altered perineural blood flow and nerve damage.
- Other rare forms of diabetes mellitus are those due to specific genetic defects (type 3) like "maturity onset diabetes of young" (MODY) other endocrine disorders, pancreatectomy and gestational diabetes mellitus (GDM).
- Due to imbalance of specific receptor can cause diabetes mellitus. Some specific receptors are Glucagon-like peptide-1 (GLP-1) receptor, peroxisomes proliferator-activated (γ) receptor ($PPAR\gamma$), beta3 (β_3) adrenergic-receptor some enzymes like α glycosidase, dipeptidyl peptidase IV enzyme etc.
- Current research on diabetic neuropathy is focused on oxidative stress, advanced glycation-end products, protein kinase C and the polyol pathway.

2. MATERIAL AND METHODS

2.1 Collection of Plant Material and Authentication

The plant *Ficus dalhousiae* was collected and authenticated from Department of Botany, Sri Venkateswara University, Tirupati, India.

2.2 Preparation of Plant Material and Hydro-alcoholic Soxhlet Extraction

100 gm of crude powder of *Ficus dalhousiae* plant was subjected for Soxhlet extraction using hydro-alcoholic (30:70, water:ethanol) solvent. The extraction was carried out till the completion of 10 siphon cycle for at least 48 hours. It was observed that the solvent turned into the dark green color ensuring the isolation of maximum phytoconstituents from each plant. The extract then collected and the solvent was removed by simple evaporation at room temperature. The crude powder obtained from this process was further utilized for subsequent investigation.

2.3 Pharmacognostical evaluation

2.3.1 Colour: The untreated part of the drug was taken and colour of the drug was examined under sunlight.

2.3.2 Odour and Taste: A little amount of the drug was ingested, and the air was inhaled slowly and repeatedly over the substance to examine the odour. Additionally, a small amount of drug was placed on the tongue to evaluate the flavour of the drug.

2.3.3 Ash values:

Three gm of drug were weighed and burned in a crucible at a temperature not exceeding 45 °C until carbon-free, cooled, then weighed three times more until a steady weight was achieved for three consecutive readings. The percentage of ash was estimated using the air dried drug as a reference.

$$\text{Total Ash} = \frac{\text{Wt. of ash}}{\text{Wt. of drug}} \times 100$$

2.3.4 Extractive values:

5 gm of accurately weighed powdered drug was placed in a stoppered conical flask and 100 ml of 90% alcohol was added. The flask was shaken continuously for 6 hours in an electrical shaker and then left overnight for maceration. The flask was then carefully filtered and the filter was evaporated to dryness. The weight of the extractive was taken and the percentage calculated.

$$\text{Extractive values} = \frac{\text{Wt. of extractive}}{\text{Wt. of drug}} \times 100$$

2.4 Preliminary Phytochemical Screening

The crude drug sample was subjected for preliminary phytochemical screening by various qualitative tests to detect the presence of different class of phytoconstituents. The *Ficus dalhousiae* Plant extract acquire was subjected to the precursory phytochemical analysis following standard methods by Khandelwal and Kokate. The extract was screened to identify the presence of various active principles of alkaloids, glycosides, phenols, flavonoids, Terpenoids, Saponins, Steroids.

2.5 In vivo Antidiabetic Activity

2.5.1 Experimental Animals and Ethical Considerations

Swiss Albino mice of either sex weighing between 20 and 30 g were used. The mice were acclimatized to the animal house condition for 1 week before carrying out any experimental work. The mice were fed with normal animal pellet diet and water *at-libitum*. They were housed at standard housing conditions of temperature (23°C ± 12°C), humidity (45 ± 5%), and 12-h light and dark cycle.

2.5.2 Acute Toxicity Studies

Safety Studies for dose titration were carried out according to of the Organization for Economic Co-Operation and Development (OECD 425) Guideline on normal mice with three different doses of the tea bag solution. The fasted mice were fed with single dose of 500, 1000 and 2000 mg/kg body weight by oral route to three different

groups respectively. All the mice were keenly examined for 2 h to check any abnormalities in behavior of the animals and further continued to monitor and examine the mice for 24 and 72 h.

2.5.3 Induction of Chronic Diabetes & Experimental Design

Hyperglycemia was induced by a single intraperitoneal injection of freshly prepared streptozotocine (STZ) (55mg/kg body weight, in 0.1M citrate buffer (pH 4.5) to a group of overnight fasted rats. To control drug-induced hypoglycemia, a solution of 5% glucose overnight was given to rats. Hyperglycemia was confirmed on the third day after STZ injection by estimating glucose level by Glucometer. The rats having a glucose level of 300 mg/dl were used for the study.

On day 14 of post STZ injection, the diabetic rats were randomized based on their fasting blood glucose and regrouped into 7 groups, comprising 6 rats each. STZ-untreated rats served as non-diabetic control. Group I served as control. Group II served as diabetic control, group III and IV were treated with an oral dose of extract at dosage of 100 and 200 mL/kg respectively. Group V was served as a standard group that received Metformin 120mg/kg (Oral route). The rats were allocated to different treatment groups and were administered with different treatments for next 14 days as tabulated in table 2.1.

Table 2.1. Experimental design of the animal activity

Group ID	Group Details	Treatment (Dose & Route)	No.of animals
G1	Normal Control	Saline, 10ml/kg/day, Oral Route	6
G2	Diabetic control	Saline, 10ml/kg/day, Oral Route	6
G3	E1	100 mL/kg/day, Oral Route	6
G4	E2	200 mL/kg/day, Oral Route	6
G5	Diabetic + Metformin	Metformin, 120 mg/kg/day, Oral	6

2.5.4 The Body Weight and Blood Glucose Level

The body weight and blood glucose level were estimated in rats at 0, 7th, 14th, and 21st day of the treatment. The initial body weight were measured and compared with normal control rats. Estimation of blood glucose level before and after 3 weeks of treatment was done by Glucometer (One touch). All animals were monitored for body weight during the treatment period. Blood was collected from tip of the tail vein and fasting blood glucose levels were measured.

2.5.5 Estimation of SGOT and SGPT

Estimation of Serum Glutamate Oxaloacetate Transaminase (SGOT)

Aspartate transaminase is also known as L-aspartate-2-oxoglutarate aminotransferase, AST. This enzyme catalyzes the reversible transfer of amino groups between an amino acid, and α -keto acids are called aminotransferase or transaminases. One of the aminotransferases is Aspartate aminotransferase (AST), or the old

name was glutamic -oxaloacetic transaminase (GOT). This enzyme is distributed in all tissues (primarily all the tissues), but the highest concentration is found in the liver, heart, and skeletal muscles. AST (SGOT) concentration is more in heart >liver> muscles>kidneys>pancreas. This enzyme exists in two isoenzyme fractions: In the liver, it is present in cytosolic (cell cytoplasm), and the second form is mitochondrial. The intracellular concentration of the SGOT enzyme is 7000 times as compared to extracellular concentration. The cytoplasmic fraction isoenzyme is predominant in the serum.

Between L-Aspartate and SGOT catalyzes the transfer of amino group between L-Aspartate and alpha Ketoglutarate to form oxaloacetate and glutamate. The oxaloacetate formed reacts with NADH in the presence of malate Dehydrogenase to form NAD. The rate of oxidation of NADH to NAD is measured as a SGOT kit contains L1 (enzyme reagent) and L2 (Starter reagent).

Substrate start assay

0.1ml of sample was mixed with 0.8 ml of enzyme reagent and incubated at 350 C for 1 min and added 0.2 ml of starter reagent. Mixed well and read the initial absorbance change per minute ($\Delta A/\text{min}$).

Sample start assay

1.0ml of working reagent is incubated at 350 C for 1 min and added 0.1 ml of starter reagent. Mixed well and read the initial absorbance A0 and repeat the absorbance reading after every 1, 2 and 3 min. calculate the mean absorbance changer per minute ($\Delta A/\text{min}$).

$$\text{SGOT (ASAT) activity in U/L} = \Delta A/\text{min} \times 1546$$

Estimation of Serum glutamic pyruvic transaminase (SGPT)

This enzyme catalyzes the reversible transfer of amino groups between an amino acid, and α -keto acids are called aminotransferase or transaminases. One of the aminotransferases is Alanine aminotransferase (ALT), or the old name was glutamate-pyruvate transaminase (SGPT) which will catalyze the reversible transfer of the amino group between an amino acid and α -keto acid where vitamin B6 (pyridoxal phosphate) is the co-factor. ALT is an enzyme found predominantly in the liver. This enzyme is present in cytosolic and mitochondrial forms in the liver. The majority of the SGPT is present in the cytoplasm of liver cells. There is always a low level of this enzyme in the blood. This is a liver-specific enzyme of the transferases. The liver hepatocytes contain 3 to 4 times more AST (SGOT) than the ALT (SGPT). This is also normally found in bile, CSF, and saliva. It is not found in the urine unless there is a kidney lesion. Any damage to the liver cells, SGPT enzyme, is released, specifically for liver cell necrosis. First cytosolic SGPT is released, and in the case of necrosis, then mitochondria SGPT is released into circulation.

SGPT catalyzes the transfer of amino group between L-Alanine and α Ketoglutarate to form pyruvate and glutamate. The pyruvate formed reacts with NADH in the presence of lactate Dehydrogenase to form NAD. The rate of oxidation of NADH to NAD is measured as a decrease in absorbance which is proportional to the SGPT activity in the sample. SGPT kit contains L1 (Enzyme reagent) L2 (Starter reagent)

Substrate start assay

0.1ml of sample was mixed with 0.8 ml of enzyme reagent and incubated at 350 C for 1 min and added 0.2ml of starter reagent. Mixed well and read the initial absorbance A0 and repeat the absorbance reading after every 1, 2 and 3 min. calculate the mean absorbance change per minute ($\Delta A/\text{min}$).

Sample start assay

1.0 ml of working reagent is incubated at 350 C for 1 min and adds 0.1 ml of starter reagent. Mix well and read the initial absorbance A0 and repeat the absorbance reading after every 1, 2, and 3 min. calculate the mean absorbance change per minute ($\Delta A/\text{min}$).

$$\text{SGPT (ALAT) activity in U/L} = \Delta A/\text{min} \times 1546$$

3. RESULTS AND DISCUSSION

3.1 Result of Pharmacognostical Investigation of Hydro-alcoholic Extract

It was discovered that the extract had a percentage yield of 5.43 percent. It was discovered by qualitative screening that the bulk of the phytochemicals were present in the extract.

Table 3.1 The pharmacognostic evaluation of the crude extract

Parameters	Values (% dry weight basis)
Moisture content	4.3 ± 0.18
Total ash content	5.8 ± 0.15
Water soluble ash content	1.8 ± 0.12
Acid soluble ash content	1.1 ± 0.11
Hot ethanol extractable matter	8.4 ± 0.13
Cold-ethanol extractable matter	8.2 ± 0.12
Hot-water extractable matter	6.5 ± 0.21
Cold-water extractable matter	3.5 ± 0.12

Data represented as mean ± SEM (standard error mean); $n=6$.

3.2 Result of preliminary phytochemical screening of the Hydro-alcoholic extract

Table 3.2. The preliminary phytochemical screening of the extract

Name of Chemical Class	<i>Hydro-alcoholic extract</i>
Alkaloid	+++
Carbohydrates	++
Saponins	+
Glycoside	+
Fats and fixed oils	++
Resins	---
Phenols	++
Amino acids	---

Tannins	+++
Diterpins	+
Flavonoids	+++
Proteins	---

Whereas, + indicates present, ++ indicates moderately present, +++ indicates strongly present, --- indicates absent.

3.3 Result of *In vivo* Antidiabetic Activity

3.3.1 Effect on Body weights and Serum glucose level

The following table provide tabulations of the findings of the extract's antidiabetic efficacy when tested in *in vivo* conditions.

Table 3.3. Effect of extract on body weight and serum glucose levels

Day	Normal control	Diabetic control	F1	F2	Metformin
Body weight (g)					
0	220 ± 1.6	218 ± 1.2	219 ± 1.4	216 ± 1.2	215 ± 1.6
7	318 ± 1.9	298 ± 2.1 ^y	312 ± 1.9 ^c	310 ± 1.4 ^b	318 ± 0.9 ^a
14	330 ± 1.5	251 ± 2.2 ^a	317 ± 1.4 ^a	326 ± 1.3 ^a	357 ± 1.7 ^a
21	336 ± 2.3	238 ± 1.9 ^a	329 ± 2.3 ^a	330 ± 1.1 ^a	359 ± 1.8 ^a
Serum glucose (mg/dl)					
0	91 ± 1.3	98 ± 1.4	90 ± 1.6	89 ± 1.7	92 ± 2.1
7	98 ± 1.9	360 ± 1.3 ^a	290 ± 1.9 ^a	263 ± 2.3 ^a	230 ± 2.1 ^a
14	101 ± 1.9	365 ± 2.1 ^a	267 ± 2.2 ^a	238 ± 2.3 ^a	217 ± 2.7 ^a
21	113 ± 1.7	371 ± 2.2 ^a	178 ± 1.8 ^a	162 ± 1.9 ^a	130 ± 2.2 ^a

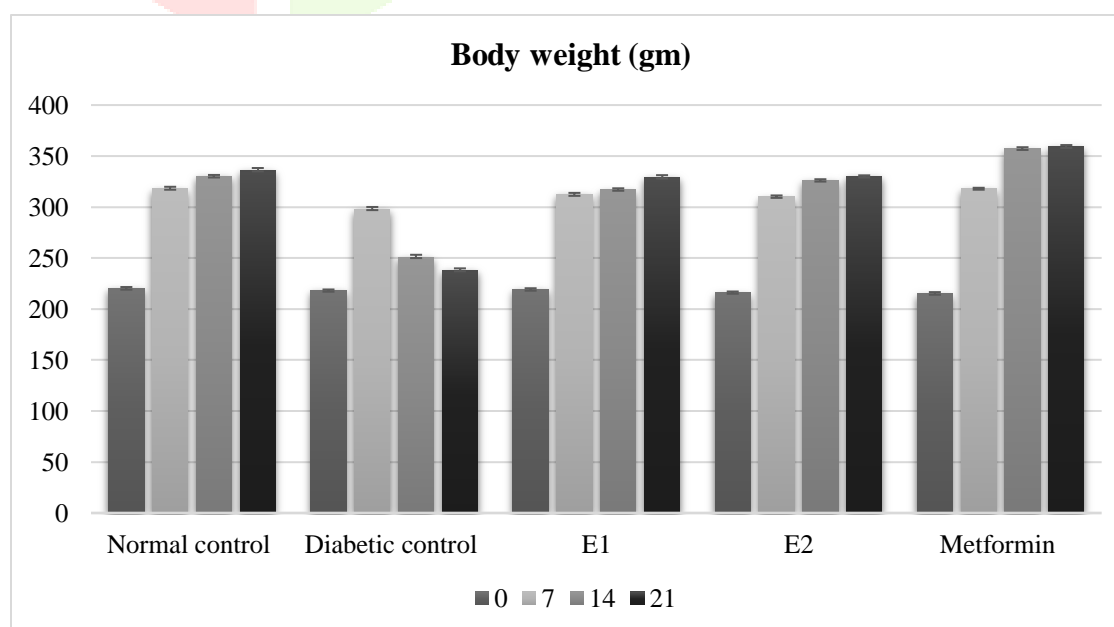


Figure 3.1. An effect of extract on body weight

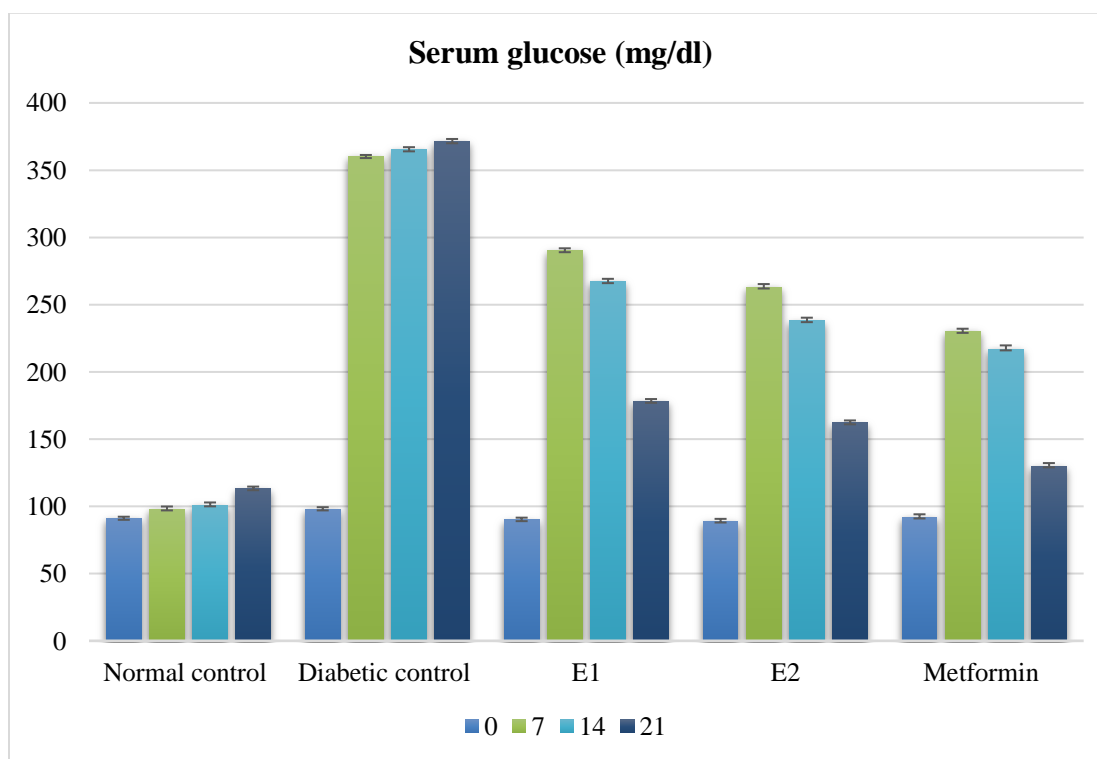


Figure 3.2. An effect of extract on serum glucose level

Hyperglycemia was induced by a single intraperitoneal injection of freshly prepared streptozotocine (STZ). Diabetes induction caused significant decrease in the body weight during the experimental period when compared to NC group. The mean body weights of NC group, at the end of the treatment period was found to be 336 ± 2.3 gm, this was significantly ($p < 0.001$) decreased to 238 ± 1.9 gm in DC group. The decreased body weight was significantly ($p < 0.001$) improved in E1, E2, and metformin treated groups. The basal serum glucose level of animals of all groups was found to be statistically equivalent. Over the course of the trial, the DC group had a considerable rise in blood glucose levels, indicating that they were diabetic. When comparing the DC to the NC group, the blood glucose levels of the DC group were considerably ($p < 0.001$) higher (from 98 ± 1.4 to 371 ± 2.2 mg/dl) at the conclusion of the research. The increased serum glucose level was significantly decreased with treatment of extract (at 100 and 200 mg/kg) and metformin ($p < 0.001$).

3.3.2 Result of Effect of extract on OGTT

The influence of extract on the OGTT is shown in Figure 3.3 and Table 3.4.

Table 3.4. Effect of extract on OGTT

Groups	0 min	30 min	60 min	120 min	AUC
Normal control	131 ± 4.1	176 ± 3.8	150 ± 2.2	132 ± 4.1	283 ± 5.1
Diabetic control	170 ± 2.2^a	220 ± 2.1^a	194 ± 2.2^a	182 ± 3.3^a	375 ± 4.9^a
E1	145 ± 2.9^a	177 ± 3.2^a	164 ± 2.6^a	151 ± 2.6^a	207 ± 4.5^a
E2	139 ± 1.4^a	174 ± 3.6^a	163 ± 2.6^a	149 ± 2.2^a	304 ± 4.1^a
Metformin	133 ± 2.1^a	165 ± 2.3^a	155 ± 2.5^a	135 ± 2.2^a	284 ± 3.9^a

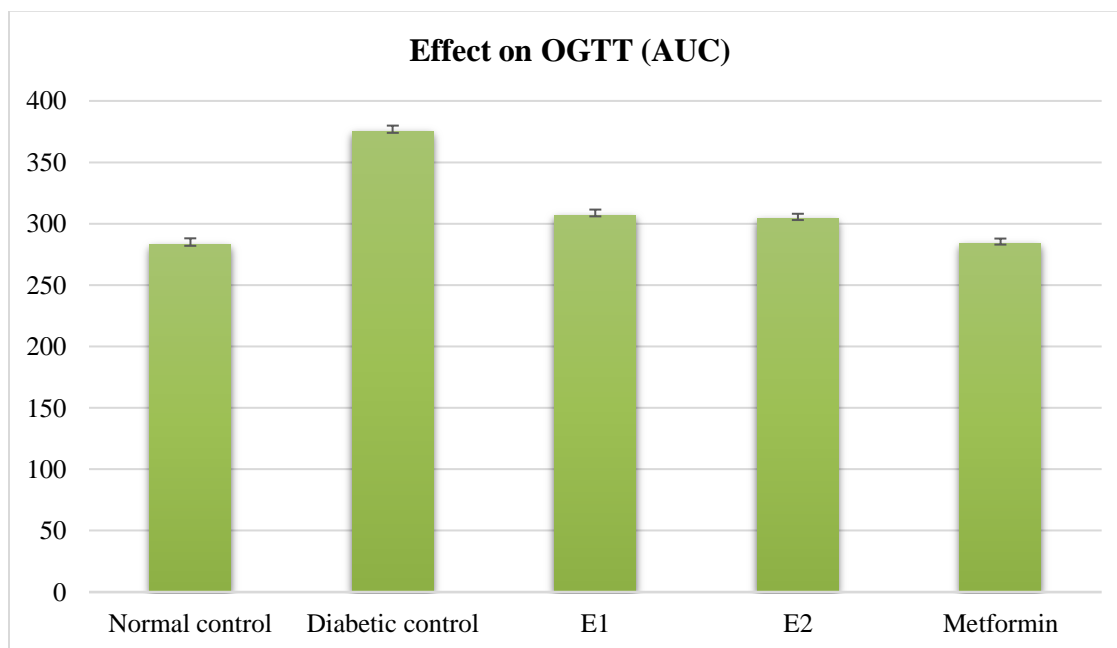


Figure 3.3. The effect of extract on OGTT (AUC)

NC group serum OGTT levels were observed at specific time intervals of 0 min, 30 min, 60 min, and 120 min along with the AUC curve. NC group level was 283 ± 5.3 , whereas DC group OGTT levels were considerably ($p < 0.001$) higher at 375 ± 4.9 mg/dl. Treatment with E1, E2, and metformin ($p < 0.001$), considerably reduced these elevated levels.

3.4 Result of Effect of extract on SGOT and SGPT

The influence of extract on the SGOT and SGPT is demonstrated in Figure 3.4 and Table 3.5.

Table 3.5. Effect of extract on SGOT and SGPT levels

Parameters	Normal control	Diabetic control	E1	E2	Metformin
SGOT (U/L)	58 ± 1.4	90 ± 1.2^a	70 ± 1.6^a	75 ± 1.4^a	69 ± 1.5^a
SGPT (U/L)	53 ± 1.8	98 ± 1.3^a	68 ± 1.5^a	71 ± 1.4^a	64 ± 1.1^a

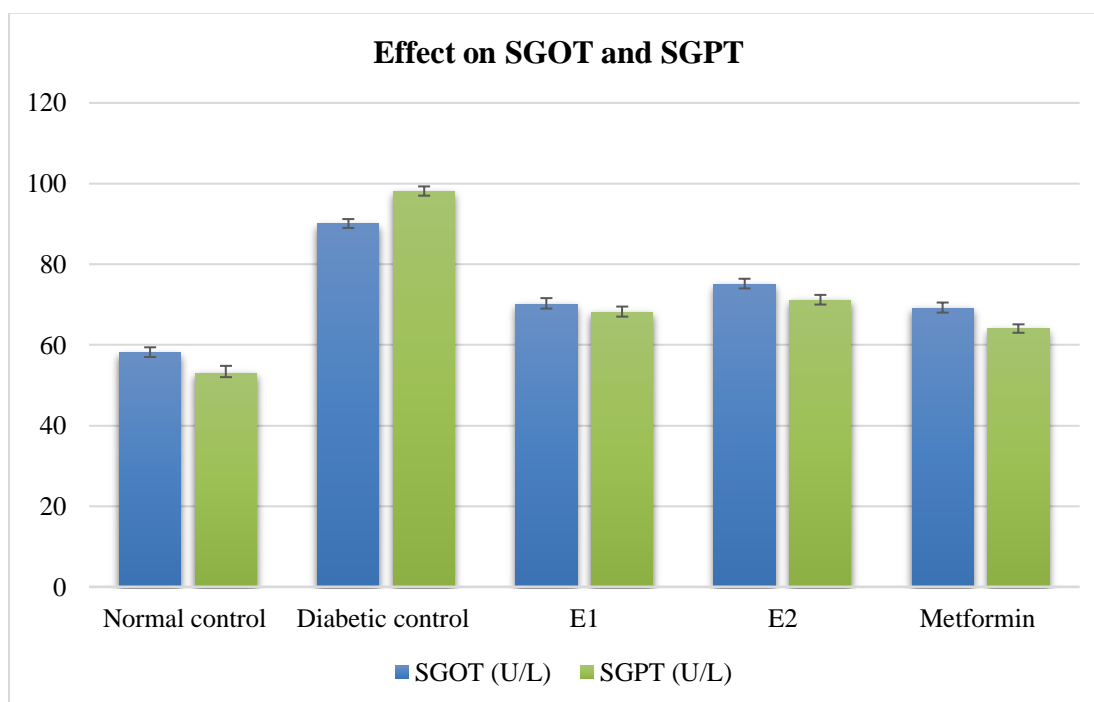


Figure 3.4. An effect of extract on SGOT and SGPT level

The mean serum SGOT level of NC group was 58 ± 1.4 U/L, which significantly ($p < 0.001$) increased to 90 ± 1.2 U/L in DC group, the increased levels were significantly decreased in E1, E2, and metformin group ($p < 0.001$), when compared to DC group. Diabetes induction caused significant increase ($p < 0.001$) in SGPT levels from 53 ± 1.8 U/L to 98 ± 1.3 U/L, when compared to the NC group. The increased SGPT levels were significantly decreased in E1, E2, and metformin group ($p < 0.001$), when compared to DC group.

CONCLUSION

Phytochemicals derived from plants may be used to address a wide range of underlying physiological issues. Due to their potency, purity, and cheap cost-effectiveness, herbal treatments have become more and more popular in recent years. Bioactive substances that are utilised in both conventional and contemporary medicine to treat and prevent disease are mostly sourced from plants. Humans have depended on remedies made from plants to treat a variety of ailments for a very long time. After reviewing the literature mentioned above, we have created a project to research herbal plants for the treatment of diabetes mellitus. For the investigation, we had picked *Ficus dalhousiae*. While the extract contains several different chemical classes, including alkaloids, glycosides, tannins, polyphenols, and terpenoids, they may all contribute to the antidiabetic effect by inhibiting different types of enzymes. As a result, we used *in vivo* models to examine the efficacy of *Ficus dalhousiae* in diabetes.

The extract was shown to include a wide variety of compounds, including alkaloids, carbohydrates, saponins, glycosides, lipids and fixed oils, resins, phenols, tannins, diterpins, flavonoids, and proteins, according to qualitative phytochemical screening. Rats with diabetes caused by STZ were used to test the extract's *in vivo* antidiabetic efficacy. By calculating several biochemical parameters, including body weight, blood glucose level, SGOT (AST), and SGPT (ALT) levels, the antidiabetic potential has been identified. The extract's antidiabetic

potential was equivalent to that of metformin, as seen by lower blood glucose, SGOT, and SGPT levels. Animals received dosages of 100 mg/kg (E1) and 200 mg/kg of the extract (E2).

A single intraperitoneal injection of newly produced streptozotocine (STZ) caused hyperglycemia. The experimental setup for the animal activity in which it was discovered that the basal body weight of animals in all groups was statistically equal. As compared to the NC group, the body weight significantly decreased throughout the study period after diabetes induction. The mean body weights of NC group, at the end of the treatment period was found to be 336 ± 2.3 gm, this was significantly ($p < 0.001$) decreased to 238 ± 1.9 gm in DC group. The decreased body weight was significantly ($p < 0.001$) improved in E1, E2, and metformin treated groups. The basal serum glucose level of animals of all groups was found to be statistically equivalent. Over the course of the trial, the DC group had a considerable rise in blood glucose levels, indicating that they were diabetic. When comparing the DC to the NC group, the blood glucose levels of the DC group were considerably ($p < 0.001$) higher (from 98 ± 1.4 to 371 ± 2.2 mg/dl) at the conclusion of the research. The increased serum glucose level was significantly decreased with treatment of extract (at 100 and 200 mg/kg) and metformin ($p < 0.001$).

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Plants have been investigated recently as potential treatments and preventative measures for illness. Herbal products are often preferred over synthetic ones due to their wide availability and the abundance of available empirical and accessible information on their traditional use. Modern scientific techniques, which may be used to validate claims of the therapeutic effects of herbs, may assist traditional medicine. Consequently, based on the aforementioned findings, we have drawn the conclusion that *Ficus dalhousiae* may be utilized therapeutically to treat diabetes mellitus. As a result, our results show that *Ficus dalhousiae* has strong antidiabetic action that is dosage dependent and, if optimized, may be utilized therapeutically to treat diabetes in a highly practical manner.

FUTURE PROSPECTIVE

Herbal medications have gained popularity in recent years due to their effectiveness, safety, and low cost. There are a variety of bioactive chemicals that may be found in plants, and both conventional and modern medicine use these substances to treat and prevent illness. Plant-based remedies have been used to treat a wide range of ailments for ages. Sometimes, herbal medicines are made from a variety of various substances, each of which is supposed to have a special effect that may include drowsiness, rejuvenation, or healing. In the recommended experiment, we observed significant antidiabetic activity at doses of 100 and 200 mg/kg of *Ficus dalhousiae*

extract. Our study demonstrates that the plant we selected has a potent anti-diabetic effect and might potentially be used therapeutically to treat diabetes with very minor modifications. Every endeavour also has an ultimate scope and an ultimate objective. We expect to shortly report on the *in vitro* and *in vivo* activity of several phytoconstituents isolated from this plant as a consequence of our ongoing study.

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